Post-cholecystectomy Bile Duct Injury

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To,

D.M.,
my patient who died
because of a bile duct injury
after undergoing laparoscopic
cholecystectomy.

Foreword by Henri Bismuth

Cholecystectomy is one of the most frequent operations performed by the GI surgeon.

For this reason, even if the main complication of the trauma of the bile duct is rare, i.e., less than one percent, it has serious consequences due to several factors:

1. It is usually on a young patient.
2. It is a benign disease.
3. It is directly the fault of the surgeon.

As a consequence, the management of this complication has to be perfect for there is one additional factor which makes this complication even more important for the surgeon—it must be considered that in front of the surgeon, there are not only the patient and the family but, very quickly, the lawyer.

These considerations make really important the book of Dr Vinay K. Kapoor (Fig. 1) on bile duct injury.

It is a very complete book detailing all the aspects of this surgical situation. Dr Vinay K. Kapoor adds to his personal experience, which is well recognized, a complete updated review of all that has been published on this topic.

I strongly recommend the lecture of this book not only to the specialist—the HPB surgeon—but also, and I must say above all, to the GI surgeon and, by extension, to all those who may be involved in the management of a bile duct injury.

Fig. 1 The Author (Vinay K. Kapoor) with Prof Henri Bismuth (Left) at International Hepato-Pancreato-Biliary Association (IHPBA) World Congress, Mumbai India 2008

Henri Bismuth
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Foreword by John L. Cameron

This publication by Professor Vinay K. Kapoor (Fig. 2) is an encyclopedia of bile duct injuries and iatrogenic benign biliary strictures. It covers anatomy, epidemiology, etiology, and classifications and provides definitions and information in terms of diagnosis and management, contains references, and states a variety of dos and don'ts. Techniques of repair and follow-up, and nonmedical issues such as costs, quality of life, and medico-legal, are also included. The author is obviously an experienced biliary surgeon, as his institution, the Sanjay Gandhi Post-Graduate Institute of Medical Sciences (SGPGIMS) at Lucknow in India, has managed more than 500 acute bile duct injuries as well as performed more than 700 repairs of a variety of iatrogenic benign biliary strictures in the last three decades. His Department of Surgical Gastroenterology maintains a prospective database that obviously allows easy access and evaluation of their data.

This book should be of value to surgical house officers, residents, fellows, and practicing surgeons who are interested in and take care of patients with biliary tract diseases.

Fig. 2 The Author (Vinay K. Kapoor) with Prof John L. Cameron (Left) at International Hepato-Pancreato-Biliary Association (IHPBA) World Congress, Geneva Switzerland 2018

John L. Cameron
Johns Hopkins Hospital
Baltimore, MD, USA

Foreword by Steven M. Strasberg

I am grateful to Dr Vinay K. Kapoor (Fig. 3) for asking me to write a brief foreword to his book on post-cholecystectomy bile duct injury. The main reason for being selected to write a foreword is that either the author knows the writer well or the writer is thought to be an expert on the subject of the book. I had a mild interest in bile duct injury during the open cholecystectomy era, but my interest rose sharply when I was appointed in 1992 to run a course to teach community general surgeons how to perform laparoscopic cholecystectomy. That course ran for 2 years and coincided with a sharp rise in referrals of patients with bile duct injuries, some of whom had been operated by the course attendees. These events were my entrée to working in this area for the past 25 years.

Many surgeons including Dr Vinay K. Kapoor have contributed to our understanding of the problem of bile duct injury, its prevention, and its treatment. Dr Vinay K. Kapoor is Senior Professor, Department of Surgical Gastroenterology, at the Sanjay Gandhi Postgraduate Institute of Medical Sciences in Lucknow, India. There he has accumulated considerable experience in the management of bile duct injuries. This book is the product of his experience and knowledge of the literature. The primary chapters cover the breadth of the subject understandably focusing on surgical aspects of the problem, but even the chapter on nonsurgical treatment is written by the author (Vinay K. Kapoor). To balance this personal approach, international experts were recruited to write commentaries on each chapter. These contributors comprise an international who's who in the field. The combination

of the chapters by Dr Vinay K. Kapoor and the commentaries by the experts provides a comprehensive summary of the field. The result will be of interest to trainees and hepato-pancreato-biliary (HPB) surgeons alike.

Fig. 3 The Author (Vinay K. Kapoor) with Prof Steven M. Strasberg (Left), and Prof Henri Bismuth (Center) at International Hepato-Pancreato-Biliary Association (IHPBA) World Congress, Geneva Switzerland 2018

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Preface

I performed a laparoscopic cholecystectomy on D.M., a 50-year-old otherwise healthy male. After an apparently uneventful operation and smooth postoperative recovery, he was discharged on day one. On day two, he developed some nonspecific symptoms (anorexia, nausea, and vomiting) and received symptomatic treatment. In view of no improvement in his condition, he came back to the emergency services a day later and was found to have tachycardia, icterus, abdominal distention, and some tenderness. Bile leak was suspected. Ultrasonography revealed minimal interloop fluid. Isotope hepato-biliary scan showed bile leak. Computed tomography did not reveal any major collection. At endoscopic retrograde cholangiography, common bile duct could not be canulated. Laparotomy revealed a small amount of bile in the subhepatic space—no obvious bile duct injury could be identified; lavage and drainage was done. He, however, developed severe sepsis and multiple organ dysfunction syndrome and died on day six.

That was when I realized that laparoscopic cholecystectomy is not a "minor" operation and that a bile duct injury can be fatal.

M.A., a pretty 19-year-old bright medical student and daughter of a doctor couple, underwent laparoscopic cholecystectomy for gallstone disease in 2006. The operation was performed by a very senior, richly experienced, and highly reputed surgeon of the town. Unfortunately, she had bile leak in the postoperative period. Endoscopic retrograde cholangiography showed complete transection of the common bile duct. She had to undergo percutaneous catheter drainage to let bile out. Sepsis, however, continued and laparotomy had to be performed for lavage and drainage of the peritoneal cavity. Hepatico-jejunostomy was performed 3 months later by a liver transplant surgeon. She developed severe pulmonary sepsis and required intensive care including ventilation but fortunately recovered. During the follow-up, she had repeated attacks of cholangitis due to an anastomotic stricture. Repeated attempts at percutaneous dilatation failed. She was then referred to us when investigations revealed right lobe atrophy. She then underwent right hepatectomy with a fresh hepatico-jejunostomy to the left hepatic duct in 2008. She had thus undergone repeated hospitalizations, several interventions, and four major operations. At a very tender age of 21, she had a very close shave with death. Her parents spent lakhs of rupees (and lost wages), her younger siblings suffered at school, and she herself had lost precious 6 months at the medical school. Even after more than a decade, she still runs the risk of having anastomotic failure.

Her case made me realize that a bile duct injury is not only a medical but a social and financial disaster also.

My lifetime experience with management of patients with bile duct injury and the efforts that have gone into writing this book will be worthwhile if it helps the reader to properly manage and save the life and improve the quality of life of just one patient who has sustained a bile duct injury during cholecystectomy.

Lucknow, India Vinay K. Kapoor

World Bile Duct Injury (BDI) Day

Global health community observes several specific days very year, namely World Health Day (7 April, to mark the anniversary of the founding of WHO in 1948), World Cancer Day (4 February), World Tuberculosis (TB) Day (24 March, to commemorate the date in 1882 when Dr Robert Koch announced his discovery of Mycobacterium tuberculosis, the bacillus that causes tuberculosis), World Kidney Day (second Thursday of March), World Malaria Day (25 April), World Hypertension Day (17 May), World Hepatitis Day (28 July), World Stroke Day (29 October), and even a Rare Disease Day (last day of February).

I propose that 12th April every year be observed as the *World Bile Duct Injury (BDI) Day*.

It was on April 12, 1953, that Anthony Eden, who succeeded Winston Churchill as the British Prime Minister (1955–1957), sustained a bile duct injury at (open) cholecystectomy. He had to undergo a total of as many as four operations, including a liver resection, but finally had to resign from his position because of health reasons related to the bile duct injury sustained at cholecystectomy.

I suggest that, on this day, every hospital, where cholecystectomies are performed, organize a continuing medical education (CME) or continuing professional development (CPD) program to emphasize the prevalence, importance, management, significance, and prevention of bile duct injury at cholecystectomy and promote the culture of a safe cholecystectomy.

Acknowledgments

My views on the management of bile duct injury (BDI) and benign biliary stricture (BBS) are a result of the huge departmental experience with a large number of patients with BDI referred to us and a large number of BBS repairs performed by us. I am grateful to my faculty colleagues in the Department of Surgical Gastroenterology (SP Kaushik, Rajan Saxena, SS Sikora, Ashok Kumar, Sujoy Pal, Anu Behari, RK Singh, Anand Prakash, Biju Pottakkat, Ashok Kumar II, Supriya Sharma, Ashish Singh, and Rahul Rai), Department of Medical Gastroenterology (Late SR Naik, G Choudhuri, VA Saraswat, Rakesh Aggarwal, UC Ghoshal, Samir Mohindra, Praveer Rai, Abhai Verma, Gaurav Pandey, and Amit Goel), Department of Radiology (RK Gupta, SS Baijal, Sheo Kumar, Hira Lal, and Rajnikant Yadav), and Department of Nuclear Medicine (BK Das, SK Gambhir, and PK Pradhan) at the Sanjay Gandhi Post-Graduate Institute of Medical Sciences (SGPGIMS), Lucknow India. Special thanks to Supriya and Rahul for reading the final proofs.

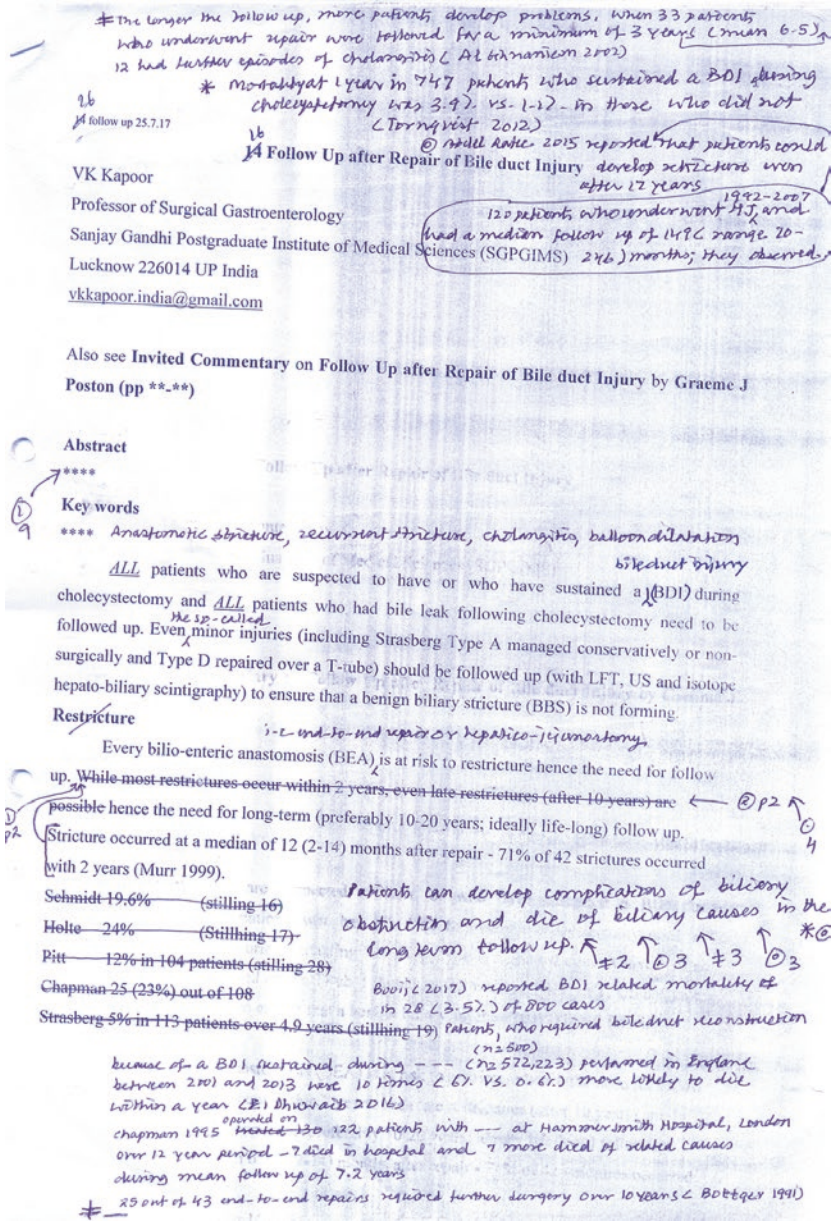
I am also grateful to SS Sikora, Vivek Singh, Anuj Sarkari, Biju Pottakkat, V Ranjit Hari, HM Lokesha, Joy Abraham, and Saurabh Galodha, our fellows who have prospectively collected, maintained, and analyzed the database of patients with bile duct injury and benign biliary stricture. Acknowledgments are also due to generations of fellows and residents (List on page xix) of my department who have looked after these patients in the last three decades.

I have been fortunate to have with me a pragmatic and humane physician, a skilled as well as safe surgeon, an intelligent yet unassuming scientist, a cooperative but critical coworker, and a reliable and dependable colleague in the form of Anu Behari who has shared with me the clinical, academic, and research responsibilities of our unit; many of the images used in this book are from patients admitted under her care in our unit.

I am thankful to my teachers and trainers (Late) Atm Prakash, Lalit K. Sharma, Tushar K. Chattopadhyay and Mahesh C. Misra at the All India Institute of Medical Sciences (AIIMS), New Delhi India.

Colleagues from the six continents, who are world-recognized authorities on the subject, readily accepted my invitation to write invited commentaries on the chapters written by me—I am indebted to all of them for their valuable comments.

Stalwarts of biliary surgery—Henri Bismuth, John L. Cameron, and Steven M. Strasberg—were kind enough to accept my request to write the forewords.



Follow Up after Repair of Bile duct Injury

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Also see **Invited Commentary** on **Follow Up after Repair of Bile duct Injury** by **Graeme J Poston** (pp **-**)

Abstract

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Key words

**** Anastomotic stricture, recurrent stricture, cholangitis, balloon dilatation, bile duct injury

ALL patients who are suspected to have or who have sustained a (BDI) during cholecystectomy and ALL patients who had bile leak following cholecystectomy need to be followed up. Even minor injuries (including Strasberg Type A managed conservatively or non-surgically and Type D repaired over a T-tube) should be followed up (with LFT, US and isotope hepato-biliary scintigraphy) to ensure that a benign biliary stricture (BBS) is not forming.

Restricture

Every bilio-enteric anastomosis (BEA) is at risk to restricture hence the need for follow up. ~~While most restrictures occur within 2 years, even late restrictures (after 10 years) are possible~~ hence the need for long-term (preferably 10-20 years; ideally life-long) follow up.

Stricture occurred at a median of 12 (2-14) months after repair - 71% of 42 strictures occurred with 2 years (Murr 1999).

~~Schmidt 19.6% (stilling 16)~~
~~Holte 24% (Stillhing 17)~~
~~Pitt 12% in 104 patients (stilling 28)~~
~~Chapman 25 (23%) out of 108~~
~~Strasberg 5% in 113 patients over 4.9 years (stillhing 19)~~

Naren Aggarwal, Teena Bedi, Rakesh Jotheeswaran, NS Pandian and Venkatesan Sathyapriya at Springer have been a big help and made my task easier by offering all possible logistic help.

Acknowledgments are also due to (Late) Ashish Agnihotri (database management), Anil Verma, Kumudesh Mishra, Sanjiv Singh, Priyanka Mishra, and Ram Lal (postal and telephonic follow-up of patients), Mithilesh Kumar Dwivedi, Ram Sanehi, and Pradeep Kumar (digital scanning of the images).

The final manuscript of this book has been made possible by my secretarial assistants Ajay Srivastava and KK Srivasatava who typed my almost illegible initial handwritten manuscripts and retyped the jumbled corrected typed scripts (Fig. 4) again and again, and yet again.

Fellows and Residents

I would like to thank my Fellows and Residents of Department of Surgical Gastroenterology, Sanjay Gandhi Post-Graduate Institute of Medical Sciences (SGPGIMS), Lucknow UP, India

Since 1989

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Abbreviations

ALP	Alkaline phosphatase
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
BBA	Bilio-biliary anastomosis
BBS	Benign biliary stricture
BDI	Bile duct injury
BEA	Bilio-enteric anastomosis
CBD	Common bile duct
CHD	Common hepatic duct
CT	Computed tomography
CTA	Computed tomography angiography
EBF	External biliary fistula
ENBD	Endoscopic naso-biliary drainage
EPT	Endoscopic papillotomy
ERC	Endoscopic retrograde cholangiography
GB	Gallbladder
GGTP	Gamma glutamyl transpeptidase
HDL	Hepato-duodenal ligament
HJ	Hepatico-jejunostomy
IHBRD	Intrahepatic biliary radical dilatation
JHH	Johns Hopkins Hospital
LHD	Left hepatic duct
MRA	Magnetic resonance angiography
MRC	Magnetic resonance cholangiography
MRI	Magnetic resonance imaging
PCD	Percutaneous catheter drainage
PH	Portal hypertension
PTBC	Percutaneous transhepatic biliary catheterization
PTBD	Percutaneous transhepatic biliary drainage
PTC	Percutaneous transhepatic cholangiography
RHA	Right hepatic artery
RHD	Right hepatic duct
SBC	Secondary biliary cirrhosis
SOJ	Surgical obstructive jaundice
UGIE	Upper gastrointestinal endoscopy
US	Ultrasonography

1 Surgical Anatomy of the Hepato-Biliary System

Vinay K. Kapoor

1.1 Gallbladder

Gallbladder (GB) is a pyriform organ lying on the undersurface of segments IV and V of liver. It has a fundus (the part protruding beyond the edge of the liver) (Fig. 1.1), body, and neck. The gallbladder neck narrows into the cystic duct at the infundibulum. Gallbladder neck often has an outpouching on its inferior border called Hartmann's pouch (Fig. 1.2). A large stone in the Hartmann's pouch may cause extrinsic compression of the common bile duct (Mirizzi's syndrome). Retraction of the gallbladder fundus elevates the liver to expose the subhepatic area and retraction of the gallbladder neck exposes the Calot's triangle. Repeated attacks of cholecystitis may cause fibrotic thickening of the gallbladder wall resulting in a small contracted thimble gallbladder which is difficult to hold and retract. The first part of the duodenum lies very close to the gallbladder; a cholecysto-duodenal fold (Fig. 1.3) of peritoneum may also be present. An attack of acute cholecystitis may cause the gallbladder to get adhered to the adjacent duodenum and colon; the gallbladder may even fistulate into these organs.

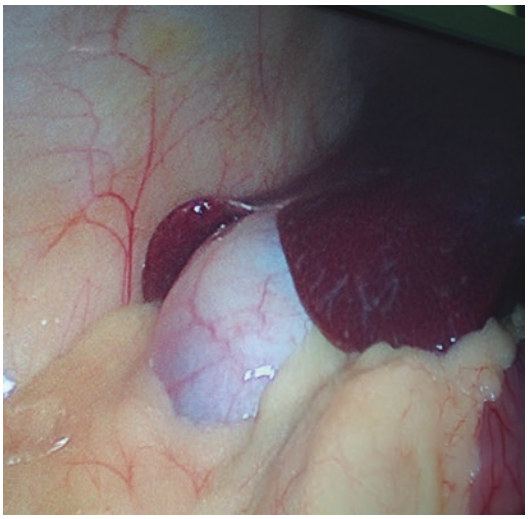

Fig. 1.1 Fundus of the gallbladder

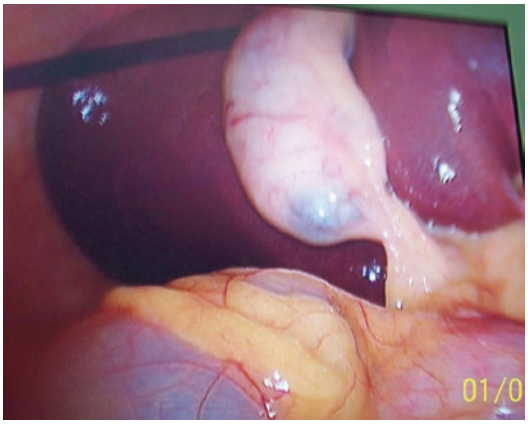

Fig. 1.2 Hartmann's pouch—an out pouching of the gallbladder neck

Please also see an Invited Commentary on Surgical Anatomy of the Hepato-biliary System by Daniel J Deziel (pp 9–10)

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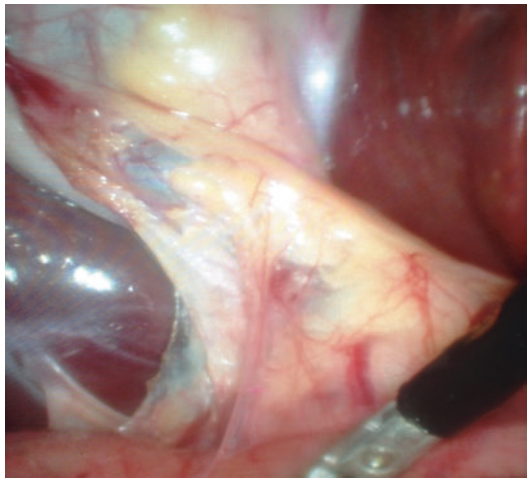

Fig. 1.3 Cholecysto-duodenal fold

1.2 Liver

The antero-superior surface of the liver is attached to the upper part of the anterior abdominal wall and the under surface of the anterior part of the diaphragm by the falciform ligament. Care should be taken when inserting the epigastric port so as not to cause injury to the falciform ligament which can result in bleeding. Ligamentum teres (round ligament) is the obliterated umbilical vein which lies in the free edge of the falciform ligament. The falciform ligament is attached to the inferior surface of the liver between segment IV and segment III. Ligamentum venosum is the obliterated ductus venosus which lies between the caudate lobe and the left lateral sector on the inferior surface of liver.

The postero-superior surface of the liver is attached to the diaphragm by the right and left coronary ligaments. The anterior layers of the coronary ligaments are continuous with the layers of the falciform ligament. The anterior and posterior layers of the coronary ligaments join to form the triangular ligaments; left triangular ligament is well formed. Hepato-renal ligament is the posterior layer of the right coronary ligament.

The surface of the liver is covered by a capsule which if torn can cause diffuse bleed from the exposed parenchyma. Based on the branches of the hepatic artery and portal vein, liver is divided into a larger (60%) right lobe and a smaller (40%) left lobe by the Cantlie's line on the inferior surface of the liver extending from the gallbladder fossa anteriorly to the inferior vena cava (IVC) fossa posteriorly. Hepatic veins do not follow lobar distribution—the middle hepatic vein lies in the Cantlie's line; right hepatic vein divides the right lobe into anterior and posterior sectors and the left hepatic vein divides the left lobe into medial and lateral sectors. There is no surface anatomical marking between right anterior and posterior sectors but the falciform ligament on the anterior surface and the umbilical fissure on the inferior surface demarcate left medial and lateral sectors.

Blood supply to the liver (about 1500 mL/min) is dual—from the hepatic artery (20–40%) and from the portal vein (60–80%). Normal liver can tolerate absence of the arterial blood supply, e.g., after injury, ligation, embolization, etc., without clinically significant deleterious effects.

1.3 CT Anatomy of Liver

On computed tomography (CT), liver sectors can be identified by the hepatic veins. Right posterior sector lies posterior to the right hepatic vein; right anterior sector lies between the right hepatic vein and middle hepatic vein; left medial sector (segment IV) lies between the middle hepatic vein and left hepatic vein; and left lateral sector (segment) lies posterior to the left hepatic vein. Sectors are divided into segments by portal veins. Right portal vein divides right posterior sector into segments VII (superior) and VI (inferior) and right anterior sector into VIII (superior) and V (inferior). Left portal vein divides left medial sector (segment IV) into subsegments A (superior) and B (inferior) and left lateral sector into segments II and III.

NOTE: *In Japan, the superior subsegment of segment IV is called IVB while the inferior subsegment is called IVA.*

1.4 Bile Ducts

The liver is divided into eight segments each having its own segmental bile duct (and hepatic artery and portal vein). Intrahepatic bile ducts, along with the branches of the hepatic artery and the portal vein, are enclosed in extensions of the Wallerian sheath (portal pedicles). Fortunately, the bile duct lies anteriorly and is easily exposed when this sheath is opened, e.g., for an intrahepatic cholangio-jejunostomy. Bile ducts of segments VI and VII unite to form the right posterior sectoral duct and those of segments V and VIII unite to form the right anterior sectoral duct inside the liver; these two sectoral ducts then unite to form the right hepatic duct (RHD) which has a short vertical largely intrahepatic course. Bile ducts of segments II, III, and IV unite variably to form the left hepatic duct (LHD) which has a long horizontal mainly extrahepatic course at the base of the quadrate lobe (segment IV) in a groove between the quadrate (segment IV) and the caudate (segment I) lobes. Hilum is a transverse fissure (slit) on the inferior surface of the liver between the base of segment IV (quadrate lobe) in front and segment I (caudate lobe) behind it. The hepatic artery and portal vein branches enter and the right and left hepatic ducts exit the liver at the hilum. The bilio-vascular pedicle at the hilum of the liver is called porta hepatis. At operation, the right anterior sectoral bilio-vascular pedicle is located in the gallbladder fossa—it has a vertical course (towards the right shoulder of the patient). The right posterior bilio-vascular pedicle is located in the Rouviere's sulcus (Fig. 1.4)—it has a horizontal course (towards the right elbow of the patient). Rouviere's sulcus is a useful but often ignored landmark [1]; it lies anterior to the caudate lobe to the right of the hepatic hilum and contains the right posterior sectoral portal pedicle. Dissection in the Calot's triangle during cholecystectomy should remain anterior to (in front of) the Rouviere's sulcus.

The union of right and left hepatic ducts in the porta hepatis is called the biliary ductal confluence. The caudate lobe drains directly by multiple small bile ducts into the left hepatic duct or

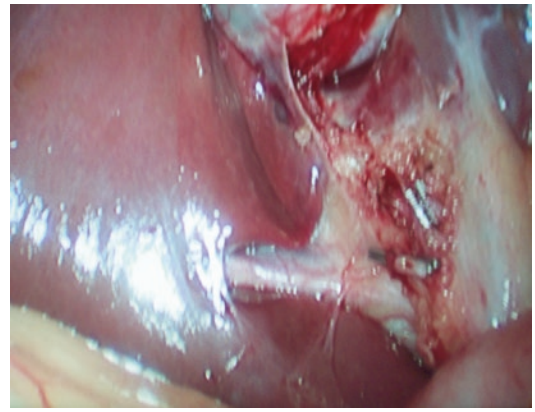

Fig. 1.4 Right posterior sectoral bilio-vascular pedicle in the Rouviere's sulcus

the biliary ductal confluence. Confluence of the right hepatic duct (RHD) and the left hepatic duct (LHD) is an important radiological landmark in the evaluation of a benign biliary stricture. If the biliary ductal confluence is patent (Bismuth Type I, II, and III biliary stricture), repair is easier and results are better. If the biliary ductal confluence is involved (Bismuth Type IV biliary stricture), repair is technically difficult and results are poorer. The right and left hepatic ducts unite outside the liver parenchyma in the hilum of the liver to form the common hepatic duct (CHD) which is joined by the cystic duct to form the common bile duct (CBD). All (even if the stricture is Bismuth Type I or II, i.e., a common hepatic duct stump is present) bilio-enteric anastomoses for benign biliary stricture should be performed at the hilum including the biliary ductal confluence (as the blood supply is richest here) and should be extended to the left hepatic duct—hilo-jejunostomy [2]. The cystic duct has irregular mucosal folds, called valves of Heister, which may make the passage of a catheter (for intra-operative cholangiography) or balloon (for dilatation of the cystic duct before trans-cystic duct choledocholithotomy) difficult. The common hepatic duct and the common bile duct run down vertically in the free edge of the lesser omentum (hepato-duodenal ligament) which contains these ducts to the right anterior, the hepatic artery to the left anterior, and the portal vein behind. The

right end of the base of the quadrate lobe marks the hilum of the liver and is another useful landmark during cholecystectomy [3]. At operation, the line joining the hilum of liver to the first part of the duodenum indicates the hepato-duodenal ligament; all dissection should remain to the right of the hepato-duodenal ligament. The common bile duct runs behind the first part of the duodenum and behind or through the head of the pancreas to be joined by the pancreatic duct to form a dilated common channel, the ampulla of Vater, which opens on the medial wall of the second part of duodenum at a nipple-like projection, the papilla of Vater surrounded by the sphincter of Oddi. The common bile duct, thus, has supraduodenal, retroduodenal, retro (or intra) pancreatic and intraduodenal parts.

1.5 Calot's Triangle

Calot's triangle (Fig. 1.5) is the most important area—*the sanctum sanctorum*—during cholecystectomy. The "surgical" Calot's triangle (also called hepato-cystic triangle) lies between the undersurface of the liver on the top, the common hepatic duct on the left and the cystic duct below. The cystic artery, along with the cystic lymph node of Lund which lies along it, usually lies in the Calot's triangle as it arises from the right hepatic artery and crosses the triangle to enter the gallbladder. Division of the peritoneum on the anterior and posterior surfaces of the Calot's triangle opens the triangle for dissection of the cystic artery.

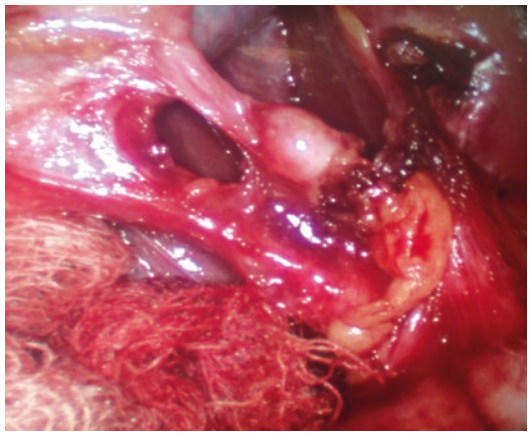

Fig. 1.5 Calot's triangle showing cystic duct, cystic artery and cystic lymph node

Repeated attacks of cholecystitis may cause inflammatory fibrosis in the Calot's triangle resulting in obliteration of the Calot's triangle and the gallbladder neck getting adherent to the common hepatic duct; in later stages, a cholecysto-choledochal fistula (Mirizzi's syndrome) may form.

1.6 Subvesical Ducts

There is a lot of confusion in the literature about the nomenclature of small bile ducts which are present in the gallbladder bed. They have been variously called subvesical ducts, cholecystohepatic ducts, and ducts of Luschka [4]. Usually, these ducts are small and get obliterated by the electrocautery or ultrasonic energy used for dissecting the gallbladder from its bed in the liver during cholecystectomy. An unnoticed duct in the gallbladder bed may get injured during dissection of the gallbladder from its bed in the liver and may cause bile leak in the postoperative period (Strasberg Type A bile duct injury). As many as 15% of 270 cases of bile duct injury were a consequence of an injury to these ducts [5].

The author is of the opinion that any bile duct present in the gallbladder bed should be called a subvesical duct. There are two types of subvesical ducts present in the gallbladder bed. One, small aberrant ducts which drain some (small) volume of the liver parenchyma around the gallbladder bed in segments IV and V into the gallbladder (they should rightly be called hepato-cholecystic NOT cholecysto-hepatic ducts; Fig. 1.6); they do not communicate with the intrahepatic bile ducts. Injury to these ducts results in small amount of transient bile leak which usually resolves on its own after the biloma has been drained; endoscopic intervention is not required (in fact, endoscopic intervention will not work as the injured open duct is not in communication with the main ductal system). The other type of subvesical ducts is small aberrant ducts

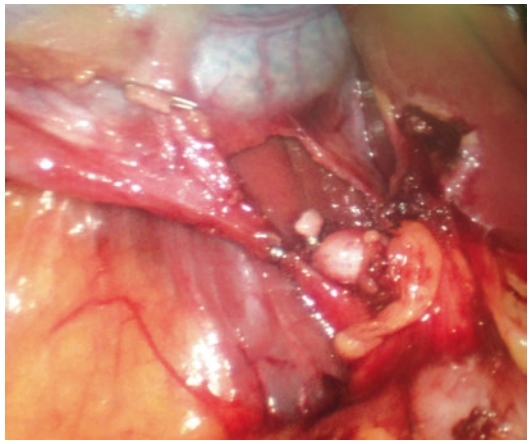

Fig. 1.6 Hepato-cholecystic duct in the Calot's triangle

which connect an intrahepatic bile duct to the gallbladder; these are the subvesical ducts originally described by Luschka. Injury to these ducts also results in postoperative bile leak which usually stops after endoscopic stenting because the injured open duct is in continuity with the main ductal system.

1.7 Hilar Plate

Most of the surface of the liver (except the bare area on the top) is covered by the visceral peritoneum, underneath which lies the Glisson's capsule. Hilar plate is the visceral peritoneum as it reflects from the inferior surface of the segment IV (quadrate lobe) of the liver to the hepatic hilum and then to the lesser omentum (gastrohepatic ligament) and the hepato-duodenal ligament. Hilar plate separates the liver parenchyma from the bilio-vascular pedicle in the hilum of the liver. Hilar plate continues with the cholecystic plate on the right and with the umbilical plate on the left. Cholecystic plate (Fig. 1.7) separates the gallbladder from the liver parenchyma in the gallbladder bed. Glisson's capsule continues as sleeve-like sheaths along the right and left portal pedicles into the liver parenchyma. The left bilioportal pedicle runs a horizontal course at the base of segment IV (quadrate lobe) and has longer extrahepatic length than the right pedicle. Hilar plate needs to be lowered (using sharp dissection)

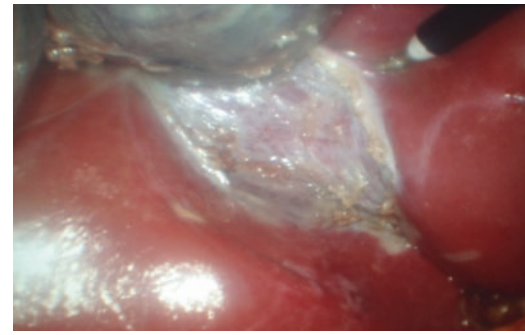

Fig. 1.7 Cholecystic plate

to expose the anterior surface of the left hepatic duct at the base of the segment IV (quadrate lobe) for performing a high (hilar) bilio-enteric anastomosis.

1.8 Aberrant (Anomalous) Anatomy

Anomalies are common in the anatomy of the bile ducts—both intrahepatic and extrahepatic—so common that some surgeons (including the Author) believe that there is nothing called "normal" biliary anatomy and all patients have variations of anatomy. During cholecystectomy, the anomalies of the extrahepatic biliary tree are important (cf. during hepatectomy, where anomalies of the intrahepatic biliary tree are more important). The surgeon should always keep in mind the common anatomical anomalies or variations (aberrations) of the biliary anatomy. Ignorance of these anatomical anomalies or variations may lead to their non-recognition/misinterpretation and an iatrogenic bile duct injury during cholecystectomy.

One of the common anomalies of the extrahepatic bile ducts which is of importance during cholecystectomy is an aberrant right subsegmental, segmental (usually V), or even sectoral (usually posterior) duct. These anomalous ducts lie in the Calot's triangle and join the common hepatic duct; they may rarely join the cystic duct or even the gallbladder. These ducts may be mistaken for the cystic duct and clipped and divided. If they are not identified during the operation, they can

get injured during dissection in the Calot's triangle—this will produce a Strasberg type B or C bile duct injury and Bismuth type V benign biliary stricture.

The common anomalies of the cystic duct are short or even absent (a sessile gallbladder opening directly into the common bile duct) cystic duct and a long tortuous cystic duct which crosses the common bile duct either in front or behind to open low on its left border. A short cystic duct results in a narrow Calot's triangle making the dissection difficult. It may result in inadvertent clipping of the lateral wall of the common hepatic duct or the common bile duct; in a sessile gallbladder, the common bile duct (especially if it is normal and undilated) may be mistaken for the cystic duct and dissected, clipped and excised (the classical laparoscopic bile duct injury). The long tortuous cystic duct may be fused with the common bile duct and attempts to dissect it along its entire length may cause injury to the common bile duct.

> It is safer to leave a few mm of the cystic duct than to remove or clip even one mm of the common bile duct.

The cystic duct also may be aberrant and join the right hepatic duct; in such a case, the right hepatic duct may be mistaken for the cystic duct and clipped and divided—this will result in an isolated right hepatic duct injury (left hepatic duct, common hepatic duct, and common bile duct are in continuity and are normal).

The gallbladder may be absent (agenesis) or double (duplication).

ANECDOTE: *A patient had symptoms suggestive of gallstones; ultrasonography (US) revealed "contracted gallbladder." At operation, the gallbladder was about to be grasped in an instrument when it was fortunately realized that it was actually the dilated common bile duct which looked like the gallbladder. The gallbladder fossa was empty. Postoperative isotope hepato-biliary scintigraphy confirmed the diagnosis of agenesis of gallbladder.*

1.9 Vascular Anatomy

Celiac axis (trunk) arises from the abdominal aorta on its anterior surface at T12-L1 level between the two crura of the diaphragm. It is only about 2 cm long; soon after its origin from the aorta, it divides into 3 branches—common hepatic artery, splenic artery, and left gastric artery. Common hepatic artery runs towards the right along the superior border of the proximal body of the pancreas. It gives off the gastroduodenal artery and then continues as the proper hepatic artery. Proper hepatic artery runs upwards in the free edge of the lesser omentum (hepatoduodenal ligament) lying to the left of the common bile duct and in front of the portal vein. Below the hepatic hilum it divides in a Y-shaped manner into a right and a left hepatic artery.

Right hepatic artery usually (80–90% of cases) runs behind the common bile duct but may (in 10–20% of cases) cross in front of the common bile duct to enter the hilum of the liver. A long tortuous right hepatic artery may form a Moynihan hump (Fig. 1.8) which lies in the Calot's triangle or even on the anterior surface of the neck of the gallbladder. The right hepatic artery may get injured during dissection in the Calot's triangle resulting in a major bleed, desperate attempts to control which may in turn cause a bile duct injury. An incomplete injury,

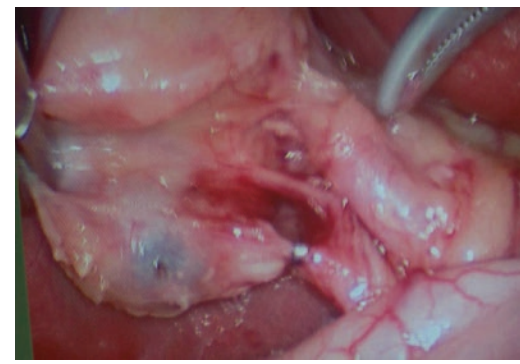

Fig. 1.8 Moynihan hump of the right hepatic artery

e.g., thermal, to the right hepatic artery may result in a pseudoaneurysm. An aberrant right hepatic artery arises from the superior mesenteric artery, instead of from the proper hepatic artery. It may be accessory (in addition to a normally placed right hepatic artery) or replaced (no normally placed right hepatic artery). The aberrant right hepatic artery travels on the right posterior aspect of the common bile duct behind the cystic duct. It may get injured during cholecystectomy resulting in profuse bleeding, attempts to control which may in turn cause a bile duct injury.

Cystic artery arises from the right hepatic artery. It runs in the Calot's triangle along the cystic lymph node of Lund. Dissection in the Calot's triangle should be kept to the right of the cystic lymph node in order to safeguard the common bile duct. In most cases, the cystic artery divides into two branches—anterior (Fig. 1.9) and posterior (Fig. 1.10)—before it enters the gallbladder. The posterior branch of the cystic artery lies inside the posterior peritoneal fold of the Calot's triangle and may get injured when this fold is opened. Cystic artery gives a small twig to the cystic duct which may get injured when a plane is being developed between the cystic artery and the cystic duct.

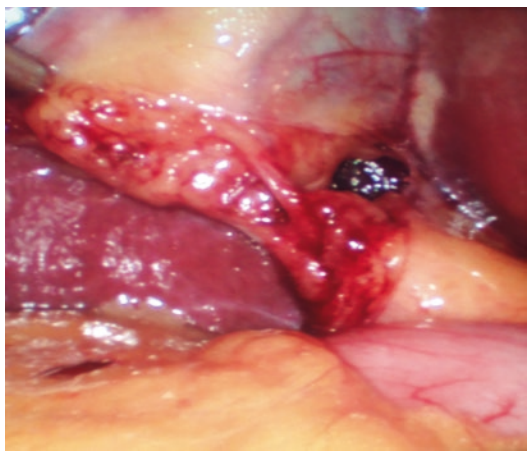

Fig. 1.9 Anterior branch of the cystic artery

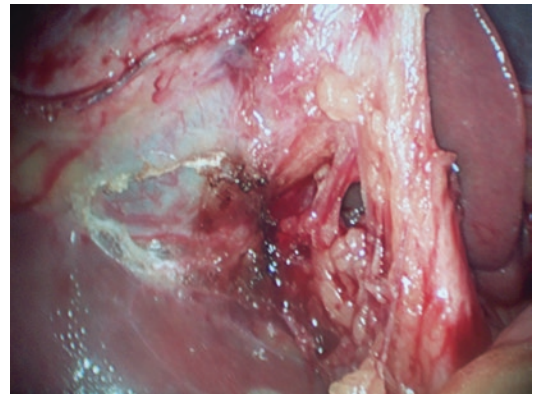

Fig. 1.10 Posterior branch of the cystic artery seen from behind

Common bile duct has an axial blood supply which comes from below as well as from above. The blood supply to the common bile duct comes from the hepatic artery (and its right and left branches), gastro-duodenal artery, anterior and posterior branches of the superior pancreatico-duodenal artery, and the cystic artery. The blood supply is two-thirds from below (gastro-duodenal and pancreatico-duodenal arteries) and one-third from above (cystic artery, and right and left hepatic arteries). The wall of the common bile duct usually has two longitudinal arteries running vertically at 3 and 9 o'clock positions with a periductal arterial plexus. Complete circumferential mobilization of the common bile duct, therefore, should not be done during common bile duct exploration as it may cause ischemia of the bile duct and may result in a delayed (after months or even years) benign biliary stricture without a bile duct injury and bile leak. Upper part of the common hepatic duct receives its blood supply from the caudate artery and medial subsegmental artery of segment IV via an arterial network (hilar plexus) present in the hilum of the liver inferior to the hilar plate—they should be preserved when lowering the hilar plate during hepatico-jejunostomy.

Superior mesenteric vein and splenic vein join at a right angle behind the pancreatic neck to

form the portal vein. Portal vein runs upwards in the free edge of the lesser omentum (hepatoduodenal ligament) lying behind the common bile duct and the proper hepatic artery. At the hepatic hilum it divides in a T-shaped manner into a right and a left portal vein.

A few cholecystic veins (Fig. 1.11) may drain from the gallbladder directly into the portal vein; few cholecysto-hepatic veins (Fig. 1.12) drain from the gallbladder into the intrahepatic branches of the portal vein.

The common bile duct is surrounded by peri- and epi-choledochal venous plexuses of Sappe which enlarge in presence of extrahepatic portal venous obstruction (EHPVO) causing portal biliopathy, an uncommon cause of benign biliary obstruction. Inadvertent injury to one of these veins during cholecystectomy may result in profuse and difficult to control, even fatal, bleeding due to high portal venous pressure in these patients.

CAUTION: *Cholecystectomy in presence of EHPVO is one of the most challenging procedures and should be attempted only by a very experienced surgeon preferably a few months after a porta-systemic shunt.*

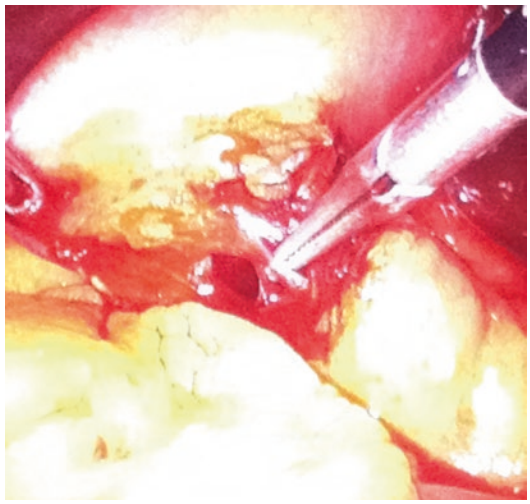

Fig. 1.11 Small cholecystic vein from the gallbladder draining directly into the portal vein

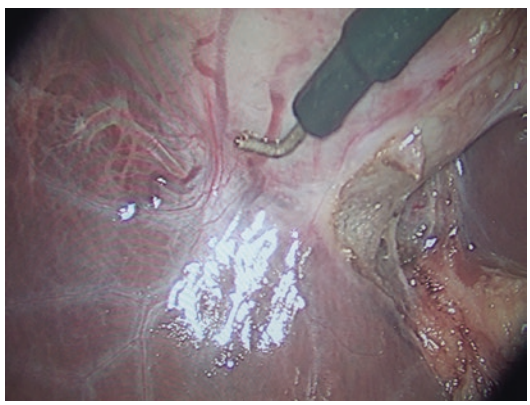

Fig. 1.12 Cholecysto-hepatic veins

1.10 Umbilicus

At the umbilicus, all fibro-aponeurotic layers of the parietes fuse into the umbilical scar. The nick for open insertion of the first trocar for laparoscopic cholecystectomy is made at the angular junction between the infraumbilical parietes (linea alba) and the umbilical scar.

1.11 Big Vessels

Major abdominal vessels, e.g., aorta, inferior vena cava, and iliac vessels, may be at a very small distance from the anterior abdominal wall in a thin built patient and are liable to injury by the Veress needle or the trocar (in case of blind insertion).

1.12 Abdominal Wall

The wound in linea alba after a midline laparotomy should be closed with a continuous suture. Subcostal incision should be closed in two layers—transversus, internal oblique and posterior rectus sheath as one layer and external oblique and anterior rectus sheath as the second layer. Incisions for 10 mm ports should be closed with a few (2 or 3) interrupted sutures to prevent a port site incisional hernia. Heavy (0 or 1) long acting absorbable suture, e.g., polydioxanone (PDS^{R}) should be used for closure. Non-absorbable sutures, e.g., polypropylene (Prolene^{R}) are not

preferred as they can be associated with stitch abscess/sinus, more so in a thin built patient with little subcutaneous fat.

1.13 Jejunum

For the preparation of a Roux-en-Y limb during hepatico-jejunostomy, the jejunum can be identified by locating the duodeno-jejunal junction to the right of the inferior mesenteric vein. Jejunum should be divided about 30 cm from the duodeno-jejunal junction so as to preserve the proximal jejunum, which has important absorptive functions, in the enteric limb.

Invited Commentary on Surgical Anatomy of the Hepato-Biliary System

Daniel J. Deziel

Our detailed understanding of the hepato-biliary anatomy comes from classic studies of cadaver dissection and resin casts, from studies of direct cholangiography, and from more contemporary imaging studies using 3D computed tomography (CT) and magnetic resonance imaging (MRI) reconstructions. Professor Kapoor has provided a succinct introductory overview of the salient anatomic features that have practical significance for performing safe operations including laparoscopic cholecystectomy. Since "typical" anatomy is only present in about one-half of individuals, one may quibble with the use of the term "aberrant" to describe the frequent variations that exist. Anatomic variations are present natively, and also result from the effects of inflammatory fibrosis and fusion.

The roughly triangular area bounded by the cystic duct and the gallbladder neck, the common hepatic duct, and the edge of the liver is the crucial region where bile duct and vascular injuries often occur during cholecystectomy. This is most properly referred to as the hepato-cystic triangle, rather than Calot's triangle, which is bound by the cystic artery (instead of the cystic duct). Calot's triangle is neither consistently present nor anatomically precise as the term is commonly used. We might advocate that "Calot's triangle" be deleted from the nomenclature, other than for historical purposes. But, alas, it is undoubtedly so well ensconced in the lexicon that it will continue to roll off of the tongues of surgeons.

One of the most common and most important variations in right duct anatomy occurs when the right anterior sectional (sectoral) duct and right posterior sectional (sectoral) duct do not join to form a main right hepatic duct. Rather, each of these ducts has a separate junction with the common hepatic duct, or even with the left hepatic duct. Some version of this occurs in one out of four individuals. When the right sectional (sectoral) ducts join the common hepatic duct independently, the distance between the junction of the lower right sectional (sectoral) duct with the common hepatic duct and the cystic duct junction is variable. A separate right posterior sectional (sectoral) duct tends to join lower down on the common hepatic duct than does a separate right anterior sectional (sectoral) duct. Hence, this posterior sectional (sectoral) duct can be in close proximity to the gallbladder and the cystic duct and it is particularly vulnerable to injury if it is not recognized. In 2% of individuals, or one out of 50 cholecystectomies, the cystic duct actually joins a separate right posterior sectional (sectoral) duct.

Professor Kapoor importantly emphasizes the presence of subvesical ducts which are the most common source of bile leak from the gallbladder bed following cholecystectomy. There have been somewhat different interpretations of the anatomic studies regarding these structures. Subvesical ducts are best understood as segmental or accessory segmental ducts that are located superficially under the Glisson's tunic in the gallbladder bed. They usually join the right anterior sectional (sectoral) duct or right hepatic duct or, occasionally, the common hepatic duct. True hepatico-cystic ducts have been described, but they are rare, certainly much rarer than the other subvesical ducts, which are common and may be present in up to one-third of individuals. None of these subvesical ducts are "ducts of Luschka." The German anatomist, Hubert von Luschka, did not describe ducts going directly from the liver

into the gallbladder (hepatico-cystic ducts) as is commonly misunderstood. Luschka described two tubular microscopic structures in the gallbladder wall that were present on both the peritoneal and the hepatic sides of the gallbladder; these were likely intramural glands and lymphatics.

There are two variations in cystic duct anatomy that are particularly dangerous for causation of bile duct injury during laparoscopic cholecystectomy. The first is when the cystic duct is fused to the common hepatic duct: the "hidden cystic duct." This can occur naturally when the structures share a common sheath. However, this more frequently is the result of inflammatory fusion. The second is when the cystic duct is "short," meaning that it is fused with the common bile duct. This can essentially result in a cholecystocholedochal fistula. Surgeons sometimes refer to the cystic duct as being "absent." Almost certainly, this situation is the result of inflammatory fusion. I have not found a study based on anatomic dissection that has described true "absence" of the cystic duct.

Variations in arterial anatomy add to the risk for bleeding or vascular injury. The cystic artery is characterized by anatomic diversity: it can branch at variable distances from the gallbladder wall, 25% individuals have multiple cystic arteries, 30% of cystic arteries arise from someplace other than the right hepatic artery, 10% of the time there is no cystic artery within the hepatocystic triangle (so that a true Calot's triangle does not exist). The right hepatic artery can be closely applied to the gallbladder for various lengths, either natively or due to inflammation.

When performing cholecystectomy, the cystic artery or its branches should be ligated and divided directly on the gallbladder wall. There are two reasons for this. First, to avoid compromising the right hepatic artery which may be in close proximity. Second, to avoid compromising either a hepatic arterial branch or a recurrent arterial branch to the common bile duct originating from the cystic artery. Professor Kapoor has highlighted the features of the blood supply to the extrahepatic bile ducts. There are anatomic variations in this pattern as well. On occasion, the marginal anastomotic vessels that anchor the epicholedochal plexus are essentially absent and an important component of the blood supply to the common bile duct is provided by an artery that feeds back from the cystic artery.

As a final comment, I would add the falciform ligament to the list of landmarks that are valuable for maintaining orientation during cholecystectomy. The common hepatic duct lies in the midplane of the liver between segments IV and V. If dissection is near the plane of the falciform ligament, which lies between segments III and IV, the surgeon is too far to the patients' left side and on the wrong side of the common bile duct.

For those who have interest, the vascular and ductal variations in biliary anatomy were elegantly detailed by the classic dissections of Nicholas A Michels in the 1950s and 1960s [6–8].

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Epidemiology of Bile Duct Injury

2

Vinay K. Kapoor

> Injuries to the bile ducts are unfortunately not rare and often turn out to be tragedies.
>
> Grey Turner [1]
>
> Statement made in 1944—holds true even today!

A bile duct injury will probably occur, at least once in the lifetime, in the hands of every surgeon who performs laparoscopic cholecystectomy.

WJ Mayo reported the first two cases of hepaticoduodenostomy for post-cholecystectomy bile duct injury (BDI) in the Annals of Surgery in 1905—23 years after the first report of (open) cholecystectomy by Carl Langenbuch in 1882. *What was done to the patients who sustained a BDI during cholecystectomy between 1882 and 1905 is any one's guess?*

2.1 Gallstone Disease and Cholecystectomy

Gallstone disease is common all over the world. In the USA, gallstone disease affects 10–15% of adult population; about 30 million persons suffer from gallstone disease. Cholecystectomy is the treatment of choice for symptomatic gallstone disease. It is one of the commonest surgical procedures performed by a general surgeon. About 750,000 cholecystectomies are performed in the USA (population 300 million), about 50,000 in the UK (population 60 million), about 17,000 in the Netherlands (population 16 million), about 12,000 in Sweden (population 10 million), and about 7,000 in Denmark (population 6 million) each year. Similar figures are not available for India but if the USA/ UK statistics are applied to India (population 1,200 million), it will translate into 3,000,000/1,000,000 cholecystectomies each year (Fig. 2.1). In northern India, gallstone disease is very common and stones form at an earlier age than in the West—prevalence of gallstone disease in women was 15% (31–40 years), 16% (41–50 years), and 29% (51–60 years) [2]. Majority of patients with bile duct injury and benign biliary stricture, therefore, are young, otherwise healthy and in the productive years of their life.

First open cholecystectomy was performed by Carl Langenbuch in Berlin, Germany on 15 July 1882. Laparoscopic cholecystectomy, introduced in the late 1980s, when Eric Muhe performed the first laparoscopic cholecystectomy under direct scope vision in 1985 and Philip Mouret using a video laparoscope in 1987, has become the "gold standard" (*although without much and strong evidence in the form of prospective randomized controlled trials to compare it with open cholecystectomy!*) for the management of

Also see Invited Commentary on Epidemiology of Bile Duct Injury by Bjorn Tornqvist (pp 16–17)

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Fig. 2.1 Three patients with post-cholecystectomy bile duct injury admitted at the same time in the Surgical Gastroenterology ward at the Sanjay Gandhi Post-Graduate Institute of Medical Sciences (SGPGIMS) Lucknow, Uttar Pradesh, India - the Author's workplace. Gallstone disease is very common in northern India and a large number of cholecystectomies are performed at primary and secondary level hospitals; SGPGIMS being a tertiary level biliary center gets many of these bile duct injuries referred to us

symptomatic gallstone disease—96% of cholecystectomies in Denmark being performed laparoscopically [3]. Indications for cholecystectomy have broadened and become more liberal and threshold for offering and accepting cholecystectomy has decreased after the introduction of laparoscopic cholecystectomy. The number of cholecystectomies increased by as much as 21% from 1988 to 1991 in the New York State in the USA [4]. Reduction in the threshold for surgery may have contributed to the increased rate of cholecystectomy [5]. Number of cholecystectomies performed in the Netherlands increased after the introduction of laparoscopic cholecystectomy—from 9,356 in 1994 to 15,942 in 2005 [6]. Pulvirenti [7] also observed broadening of indications for laparoscopic cholecystectomy in Italy during 1999–2008.

Cholecystectomy is associated with several complications—general (due to anesthesia and those common to any operation) and specific (to cholecystectomy); of the specific complications of cholecystectomy, BDI is the most important.

BDI is an uncommon (incidence about 0.1–0.2% in open cholecystectomy and 0.4–0.6% in laparoscopic cholecystectomy) but serious, dangerous and even potentially fatal complication of cholecystectomy as it leads to significant morbidity and may even cause mortality. Also, considering the large denominator (the total number of cholecystectomies performed), the absolute number of BDIs is big. Even the most conservative incidence of 0.5% of BDI during laparoscopic cholecystectomy would translate into as many as 3,750 BDIs every year in the USA. The annual report of Japanese Society of Endoscopic Surgery (JSES) mentions about 100–150 BDIs in Japan (population about 130 million) every year.

BDI occurred less frequently during open cholecystectomy than it is occurring now during laparoscopic cholecystectomy. Widespread use of laparoscopic cholecystectomy in the 1990s was associated with a dramatic increase in the

number of BDIs [8]. Morbidity during the open cholecystectomy era was reported to be about 0.2% in 42,474 patients [9] and 0.3% of 25,000 patients [10] (cf. 0.4–0.6% after the introduction of laparoscopic cholecystectomy). Only one major BDI was reported in New York State in 1980, before laparoscopic cholecystectomy was introduced; on the other hand, 158 incidents were reported during laparoscopic cholecystectomy between August 1990 and March 1993 [4].

2.2 Laparoscopic Cholecystectomy

BDIs are more frequent during laparoscopic cholecystectomy (0.4–0.6%) than during open cholecystectomy (0.1–0.2%). They are more often unrecognized during the operation, usually more complex, i.e., proximal/higher (closer to the hilum), more often associated with vascular injuries and more commonly associated with bile leak and thermal injury [11]. Hogan [12] reported 78 BDIs from Ireland (1992–2014); from 1992–2004 to 2005–2014, Strasberg Type E injuries increased from 4% to 23%, vascular injuries increased from 4% to 23% and attempted repair at the index hospital (*which is NOT recommended any way!*) increased from 16% to 35%.

2.3 Incidence

More or less similar BDI rates have been reported from several countries across the world (Table 2.1).

Very few Indian centers and surgeons have reported their BDIs, although, most certainly, BDIs must be occurring at almost every center and in the hands of most surgeons. Kaushik [39] reported an incidence of 0.6% in 1,233 laparoscopic cholecystectomies. Mir [40] reported 5 BDIs (1 CBD injury and 4 bile leaks) in 1,267 laparoscopic cholecystectomies performed in 3 non-teaching hospitals between 2001 and 2007 in Kashmir. Tantia [41] reported 52 (0.4%) BDIs in 13,305 laparoscopic cholecystectomies performed between 1992 and 2005. We had reported 57 BDIs (1.0%) in 5,782 cholecystectomies—10 minor (Type A), 25 major, and 22 unclassified injuries [42].

The introduction of single incision laparoscopic cholecystectomy (SILC) has resulted in an even higher risk of BDI during cholecystectomy. Li [43] reported 500 SILCs without any BDI and Lee [44] reported 817 SILCs with 2 (0.2%) CBD injuries and 3 (0.4%) cystic duct leaks, but a review of 2,626 SILCs reported in 45 studies (all including 20 or more patients) revealed a 0.7% rate of BDI [45]. Even greater caution needs to be exercised when proceeding with SILC and threshold for "conversion" to standard laparoscopic cholecystectomy should be very low.

2.4 Underreporting

The reported incidence rate depends on the definition of BDI—some reports including only those injuries which required surgical repair, whereas others include even minor bile leaks. It also varies depending upon the method of collection of data viz. interview, questionnaire, retrospective chart review, or prospective audit. de Reuver [46] commented that 0.4–0.5% incidence of BDI for laparoscopic cholecystectomy reported in most reviews is an underestimate. The true incidence of BDI is definitely higher than what is known as many BDIs are not reported; in a systematic review of 233 studies about laparoscopic cholecystectomy (2013–2016) only 89 (33%) were found to be reporting bile leak and only 75 (32%) reported about BDI [47]. Halbert [48] found only 125 (0.08%) BDIs in 156,958 laparoscopic cholecystectomies performed in the New York State (2005–2010) but the authors themselves observe that this low incidence could be due to failure to capture all BDIs in the international classification of diseases (ICD) and current procedural terminology (CPT) code. Incidence rates are higher in unselected state/nationwide cohorts/databases. The incidence of BDI in 1.5 million cholecystectomies performed in Medicare patients between 1992 and 1999 in

Table 2.1 Incidence rates of bile duct injury during cholecystectomy

Author/reference	Country (period)	BDI/cholecystectomy	%
Deziel [13]	USA	47/77,604	0.6
Gouma [14]	Netherlands		1.1
Adamsen [15]	Denmark (1992–1994)		0.7
Gigot [16]	Belgium	65/9959	0.6
Vecchio [17]	USA	561/114,005 major injury	0.5
		401/114,005 bile leak	0.35
Calvete [18]	Spain (1993–1998)	11/784	1.4
Csendes [19]	Chile (29 hospitals, 3 years)	74/25,007	0.3
Flum [20]	USA	7,911/1,570,361	0.5
Diamantis [21]	Greece (1991–2001)	13/2079	0.6
Nuzzo [22]	Italy (1998–2000)	235/56591	0.4
Waage [23]	Sweden	613/152,776	0.4
Karvonen [24]	Finland	32/3736	0.9
Downing [25]	USA	1124/377,424	0.3
Giger [26]	Switzerland	101/31,838	0.3
Harboe [3]	Denmark	39/20,307	0.2
∗Requiring reconstructive biliary surgery			
Harrison [27]	Florida, USA (1997–2006)/234,220 cases	0.25%	
Tornqvist [28]	Sweden (2005–2010)	747/51,041	1.5
Sheffield [29]	USA (2000–2009)	280/92,932	0.3
Sinha [30]	UK (2000–2009)	418,214	0.4 major
Yamashita [31]	Japan (1990–2007)	0.6–0.8%	
Rystedt [32]	Sweden (2007–2011)	174/55,134	0.3
El Nakeeb [33]	Egypt (2011–2016)	14/3269 bile leak	0.4
Palaz Ali [34]	Turkey (2014–2015)	920/308,481	0.3 major
Barrett [35]	USA (2011–2014)	741#/319,184	0.2 major
#BDI requiring operative intervention within 1 year			
Fong [36]	California USA (2005–2014)	BDI 1584/711,454	0.2
		Bile leak 3551/711,454	0.5
Kohn [37]	USA (2000–2015)	CBD injury 4/800	0.5
		Cystic duct leak 3/800	0.4
Pucher [38]	Pooled (global)	307,788 patients	0.3–0.5

the USA was 0.5% [20]. Overall incidence of BDI in 3,736 laparoscopic cholecystectomies performed in south-western Finland between 1997 and 2003 was 0.86%; Amsterdam Type B (major injury with bile leak) and Type D (complete transection or excision of CBD) injury being 0.38% [24]. The incidence of major BDI requiring reconstruction in Denmark in 2006–2009 was only 0.2% [3] but Adamsen [15] had earlier reported a high (0.74%) incidence in a nationwide prospective audit of 7654 laparoscopic cholecystectomies in Denmark (1991–1994).

2.5 Prevalence

BDI occurs in the hands of the majority of the surgeons—one-third of 1,661 US surgeons responding to a survey admitted of having caused at least one BDI [49]—"*another one-third would probably have one in their remaining surgical lifetime.*" As many as 60 of 114 surgeons in British Columbia, Canada reported an experience with BDI; average years in practice for 60 surgeons who reported an injury was 21 years while that for those who did not report any injury was

14 years [50]. In a survey of 316 heads of surgical units in Italy, 126/184 (69%) units which responded reported at least one BDI during 1988–2000 [22]. 60 out of 76 surgical departments in Sweden reported at least one BDI between 2010 and 2011 [32]. As many as 72% (269/372) surgeons in Japan, Korea, Taiwan, and the USA had a BDI or a "near miss" [51]. As many as 45% of 117 respondents to a questionnaire sent to the members of the Association of Upper GI Surgeons in the UK had experienced a BDI [52].

2.6 Training and Experience

Lack of adequate training and experience is one of the causes of BDI during laparoscopic cholecystectomy, but it is more than inexperience alone that causes BDI because BDIs occur in the hands of surgeons with enough experience and in centers with high volume also. Experience on the part of the surgeon does not offer complete protection against a BDI during laparoscopic cholecystectomy. Gigot [16] reported that 1/3rd of 65 BDIs in Belgium occurred in the hands of experienced surgeons. In Spain, 30% of BDIs were reported by surgeons after they had done 200 cases [18]. One-third of 704 BDIs, found during a survey in the USA, occurred in the hands of surgeons who had performed more than 200 laparoscopic cholecystectomies [49]. The so-called learning curve of laparoscopic cholecystectomy may extend well beyond 50 cases [53]. In British Columbia, Canada 61% surgeons experienced a BDI after 100 cholecystectomies [50]. BDIs have been reported from tertiary level teaching hospitals also. Twelve out of 200 BDIs reported by Sicklick [54] during 1990–2003 occurred during cholecystectomies performed at the Johns Hopkins Hospital, Baltimore, MD, USA itself; the denominator (number of cholecystectomies performed during this period), however, was not mentioned. Even experienced surgeons should, therefore, be careful while performing laparoscopic cholecystectomy.

> An "overconfident" surgeon doing an "easy" cholecystectomy "in record time" is actually a ripe setting for a BDI to happen.

It was thought that the initial reports of high incidence of BDI during laparoscopic cholecystectomy were because the surgeons performing these procedures were not trained for it during their residency period and learnt the procedure in "*over-the-weekend*" workshops. It was hoped that once laparoscopic cholecystectomy is introduced in the residency training programs, the rates of BDI will come down. Richardson [55] reported that the incidence of BDI in the UK decreased from 0.8% in 1990–1993 to 0.4% during 1995. Buanes [56] also reported a decreasing trend in the incidence of BDI in Norway. But by and large, the high incidence of BDI during laparoscopic cholecystectomy has remained unchanged over three decades. The incidence of BDI remained steady at 0.7% during 1991–1994 in a Danish nationwide database [15]. A nationwide survey which reported 613 (0.4%) BDIs in 152,776 cholecystectomies performed in Sweden (1987–2001) showed that the incidence of BDI has remained the same over various periods of time—1987–1990 (0.40%), 1991–1995 (0.32%), and 1996–2001 (0.47%) [23]. According to a Japan Society of Endoscopic Surgery (JSES) questionnaire survey, the incidence of BDI has remained largely unchanged, i.e., 1990–2001 (0.66%), 2002 (0.79%), 2003 (0.77%), 2004 (0.66%), 2005 (0.77%), 2006 (0.65%), and 2007 (0.58%) [31]. It was hoped that as time passes the incidence of BDI during laparoscopic cholecystectomy will decrease, but even after three decades of its introduction, the incidence of BDI during laparoscopic cholecystectomy has not decreased and has stabilized at around 0.5% (1 in 200).

> *Learning curve in laparoscopic cholecystectomy* [57], therefore, was *a myth.* [18]

Notwithstanding the universal acceptability of laparoscopic cholecystectomy as the gold standard of the management of symptomatic gallstone disease, BDI remains its Achilles' heel as it is now well established beyond doubt that the risk of BDI during laparoscopic cholecystectomy is

more than what it was during the open cholecystectomy era and this risk does not seem to be lessening with time and experience.

Invited Commentary on Epidemiology of Bile Duct Injury

BjörnTörnqvist

Cholecystectomy for gallstones is one of the most common surgical procedures and is considered a routine operation in modern surgery. Although a routine procedure, the consequences of accidental injuries to the bile ducts may have severe impact on health of afflicted patients, including mortality and considerable disability, and poses a major economic burden both to the individual patient and to the healthcare system at large.

The understanding of epidemiology of bile duct injury is essential, not only for researchers dealing with the challenges of rare outcomes, but perhaps even more for caregivers and surgeons in their daily work. The awareness of this rare but potentially disastrous complication of a common surgical procedure, often performed by inexperienced surgeons during their training, is fundamental for a safety and prevention orientated surgical community.

The Author (VKK) has made a thorough review of the literature addressing the epidemiology of bile duct injury and the challenges of interpreting the, to some extent, diverging results. A few comments regarding bile duct injury incidence and the impact of experience and training follow below.

More or less, all reported incidence figures of bile duct injuries suffer from systematic biases and comparing these incidences should be made with great caution. First of all, the definition of a bile duct injury may vary substantially as no single classification system has been globally accepted as standard. A majority of classification systems define post-operative cystic duct leaks and peripheral leaks as a bile duct injury, but these, often relatively numerous, lesions are rarely included in incidence calculations. Furthermore, the definition of a major lesion is often inconsistent between researchers and may vary according to the level and extent of the injury or the treatment required, with major injuries requiring surgical repair whereas minor injuries being handled with less invasive radiologic or endoscopic interventions. Secondly, as the Author (VKK) points out, the methodology of bile duct injury identification greatly affects incidence figures. As the International Classification of Diseases (ICD) lacks reliable complication coding for bile duct injuries, a majority of population based research uses the more accurate ICD-procedure codes, defining bile duct injuries as a cholecystectomy with a subsequent procedure code for bile duct repair. However, this method is highly dependent on uniform treatment patterns with a low frequency of non-surgical treatment options and reliable exclusion of malignant causes of bile duct repairs. Incidence calculations based on questionnaires or self-reported registers are, as mentioned, likely to suffer low response rates or underestimates of injury rates due to potential unwillingness of the reporting personal for local complications.

The most reliable and accurate incidence rates are likely to be found within prospectively collected high coverage quality registers with surgeon-independent registration of complications. A few registers meeting these criteria exist and with increasing number of included patients even rare outcomes such as bile duct injuries are being addressed precisely.

The impact of surgical training and experience on bile duct injury rates is indeed an important and complex issue; more so, since these are risk factors with a real potential for improvement. The Author's (VKK) thorough review of the literature concludes that bile duct injuries do occur in the hands of both inexperienced as well as well-trained surgeons. Even 200 or more cholecystectomies are no guarantee against severe complications, but on the other hand, a majority of cholecystectomies can safely be handled by trainee surgeons. This illustrates the great range of complexity within gallstone surgery. The majority of cholecystectomies are straightfor-

ward, with well-defined anatomical landmarks, and can be safely used for training within residency programs, with a reasonable learning curve. On the other hand, a difficult cholecystectomy can challenge the most experienced surgeon, requiring expertise, skills, and judgment to avoid a disaster. Patient selection is, thus, essential. Patients with known risk factors for difficult cholecystectomy and bile duct injury such as ongoing acute cholecystitis or severe chronic cholecystitis should be handled by, or under the guidance of, an experienced surgeon with optimized perioperative conditions.

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3 Mechanisms of Causation of Bile Duct Injury

Vinay K. Kapoor

Bile duct injury—a "surgeonogenic" disease. Title of Dr Jaipal Singh Memorial Oration of the Association of Surgeons of India (ASI) delivered by the Author (VKK) December 2015

Bile duct injury (BDI) (especially isolated BDI, i.e., with no other intra-abdominal injury) due to external accidental trauma (more often penetrating than blunt trauma) is very rare. Most (almost all) BDIs are iatrogenic. Most iatrogenic BDIs occur during cholecystectomy and some occur during common bile duct (CBD) exploration; we recently had a BDI during extended (radical) cholecystectomy for gallbladder cancer. A BDI may also occur during operations on the liver (e.g., hepatectomy, hydatid cyst removal), stomach and duodenum (e.g., distal gastrectomy and operations for bleeding duodenal ulcer), and pancreas (e.g., head coring for chronic pancreatitis). BDI caused during operations on the stomach, duodenum, and pancreas, however, is a low injury usually resulting in a Bismuth Type I benign biliary stricture. An aberrant (usually right) bile duct may get injured during excision of a choledochal cyst. The Author's (VKK) unit once had a referral from urology for a CBD injury following nephrectomy (Fig. 3.1). Percutaneous interventions, e.g., percutaneous transhepatic liver biopsy, percutaneous transhepatic cholangiography (PTC), percutaneous transhepatic biliary drainage (PTBD), may also cause injury to an intrahepatic bile duct; endo-

Fig. 3.1 Rare low BDI during nephrectomy. Commonest operation during which a BDI occurs is cholecystectomy

Also see Invited Commentary on Mechanisms of Causation of Bile Duct Injury by Miguel A Mercado (pp 33–34)

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scopic interventions, e.g., endoscopic stone removal, may also cause BDI and bile leak.

3.1 Causes of Bile Duct Injury

> Dangerous disease, dangerous anatomy and dangerous surgery are three main reasons for a bile duct injury during cholecystectomy [1].

Most texts mention aberrant anatomy, difficult pathology, bleeding, thermal injury, inexperience, and overconfidence (*in that order*) as the causes of BDI during cholecystectomy. The Author (VKK), however, believes that the causes of BDI during cholecystectomy (in order of their frequency and importance) are ignored or misidentified (and sometimes aberrant) anatomy, inexperience and/or overconfidence on the part of the surgeon, difficult pathology, bleeding, and thermal injury. Misinterpretation of biliary ductal anatomy, i.e., misidentification of the CBD as the cystic duct is the commonest etiological factor for BDI during laparoscopic cholecystectomy; aberrant anatomy and difficult pathology are less commonly responsible for the BDI.

> Aberrant biliary ductal anatomy is frequently blamed but is not usually responsible for majority of the bile duct injuries [2].

3.2 Visual Perception Error

Laparoscopic cholecystectomy has the disadvantages of lack of three-dimensional vision, absence of hand–eye coordination, and loss of haptic perception during the operation. The increased risk of BDI during laparoscopic (as compared to open) cholecystectomy is an inherent problem of the laparoscopic technique. Even with wider and increasing use of laparoscopic cholecystectomy, the risk of BDI has not decreased and it continues to occur even in the hands of well-trained, highly experienced, and high volume surgeons (See Chap. 2).

> BDI continues to be the Achilles heel of laparoscopic cholecystectomy.

Mistaking the CBD for the cystic duct is responsible for the majority of BDIs during laparoscopic cholecystectomy. A total of 252 laparoscopic BDIs were analyzed according to the principles of cognitive science of visual perception, judgment, and error. The primary cause of injury was a visual illusion in 97% cases; fault in technical skill was present in only 3%—errors of knowledge and judgment were contributory but not the primary causes of the BDI. Though 64 (25%) injuries were recognized by the surgeon during the operation, the reoperation was early enough to limit the injury in only 15 cases. As many as 61% of the injuries were class III where the CBD was erroneously misidentified as the cystic duct—this happens because of the heuristic nature (unconscious but firmly held assumption) of human visual perception—this illusion is persuasive and compelling as the surgeon continues to think that everything is "fine'" [3]. Sutherland [4] described it as surgeon spatial disorientation and cognitive map misplacement. Misidentification of the duct due to cognitive fixation was the cause of injury (transaction) in 42 (86%) of 49 patients [5]. The best example of this heuristic nature is the Kanizsa's triangle (Fig. 3.2)—the picture actually has 3 PCmans and 3 angles but the viewers perceive either a black-lined triangle or a white lined triangle while in fact there is no triangle

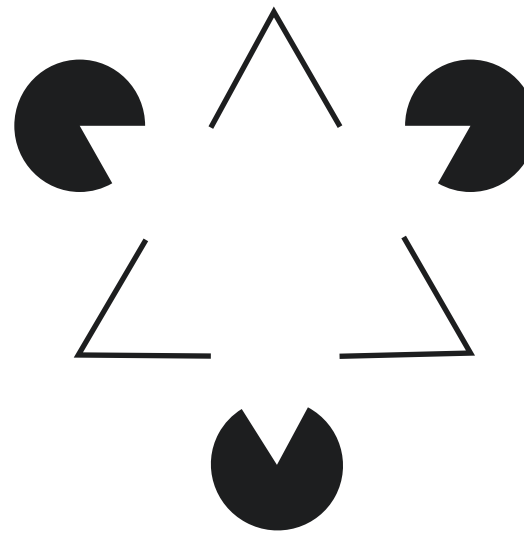

Fig. 3.2 Kanizsa's triangle—the picture actually has 3 PCmans and 3 angles but the viewers perceive either a black-lined triangle or a white lined triangle while in fact there is no triangle at all!

at all. Misidentification of the CBD, especially when it is undilated (normal sized), as the cystic duct results in dissection on the left side of the CBD and its circumferential dissection and mobilization which can interfere with and disrupt its axial blood supply from the hepatic artery in the hepato-duodenal ligament; even if the mistake is identified at this point and the CBD is not clipped and divided, the resultant ischemia of the CBD may cause a biliary stricture which presents late (after months or even years).

3.3 Anatomy

"Normal" biliary anatomy does not exist—every patient has her own individual biliary anatomy. Every general/laparoscopic/biliary surgeon must be aware of these variations of normal anatomy and keep them in mind during every cholecystectomy (See Chap. 1).

> Biliary anatomy is like the by lanes of Old Delhi India (or Istanbul Turkey) – the Author wonders whether any global positioning system (GPS) will ever be able to cover all of them and guide a tourist who has lost his way in there.

An absent cystic duct (sessile gallbladder) may be responsible for the CBD being mistaken for the cystic duct and dissected, clipped and divided.

A short cystic duct needs to be carefully handled. It results in a very narrow (acute angled) Calot's triangle providing very little space for dissection; the right wall of the common hepatic duct lies very close to the left wall of the gallbladder neck and can get injured during dissection in this narrow Calot's triangle. Even if the Calot's triangle has been dissected and cystic duct defined, there may be very little length of the cystic duct for application of 3 clips—the most proximal clip may, in such a situation, encroach on the lumen of the normal sized (undilated) CBD and cause its narrowing.

A long cystic duct running parallel and adjacent to the CBD may be a cause of BDI if desperate attempts are made to dissect this adherent cystic duct from the CBD and to remove the "entire" cystic duct.

A long tortuous cystic duct may wind (spiral) around (usually behind but sometimes in front of) the CBD and join it on its left side (Fig. 3.3)—attempts to dissect and remove the "entire" cystic duct may cause injury to the CBD.

The cystic duct may rarely join the right hepatic duct (Fig. 3.4) or one of the aberrant right sectoral or segmental (Fig. 3.5) ducts lying in the Calot's triangle which may then be mistaken for the cystic duct and clipped and divided.

An aberrant right segmental (usually of segment V), sectoral (usually right posterior), or even the main right hepatic duct can lie extrahepatically in the Calot's triangle (Fig. 3.6). It joins the common hepatic duct below the

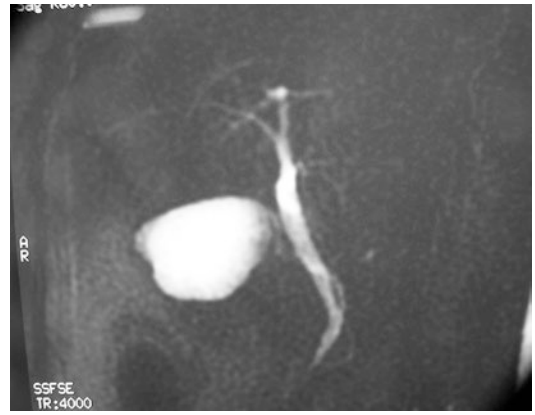

Fig. 3.3 Long tortuous cystic duct winding (spiraling) around the CBD and joining it on its left side

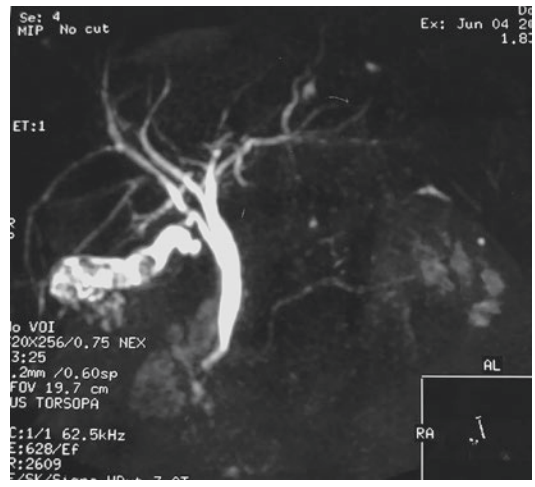

Fig. 3.4 Cystic duct joining the right hepatic duct

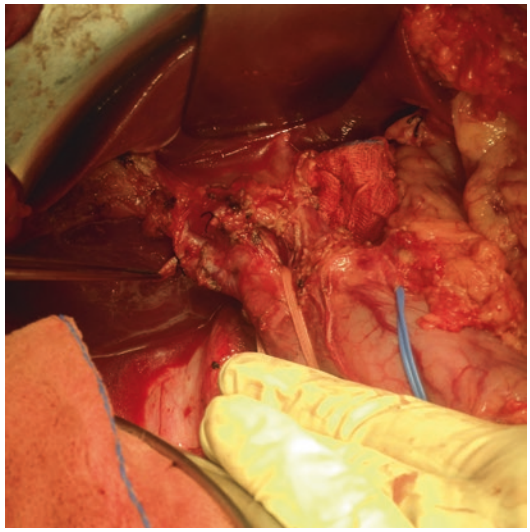

Fig. 3.5 Cystic duct joining the right posterior sectoral duct

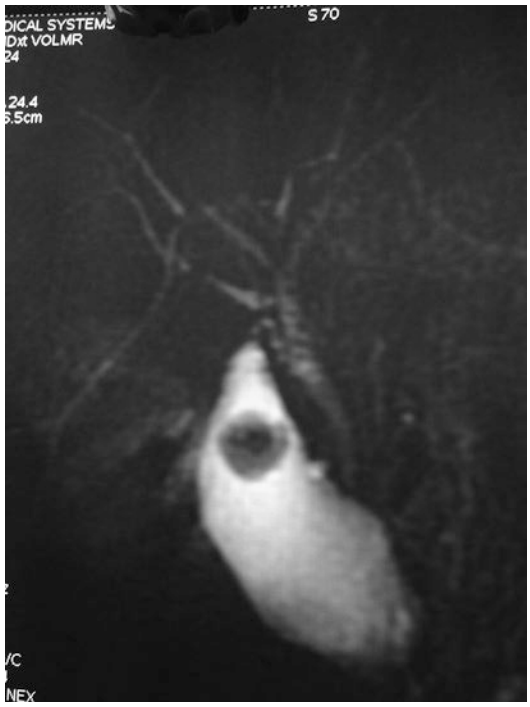

Fig. 3.6 Aberrant right duct in the gallbladder bed

biliary ductal confluence; rarely, it may join the cystic duct or even the gallbladder. It can then be mistaken for the cystic duct and divided or clipped and divided during laparoscopic cholecystectomy. The external biliary fistula in such cases will not respond to endoscopic stenting as the injured duct is isolated (Strasberg Type C BDI) and is not in continuity with the CBD.

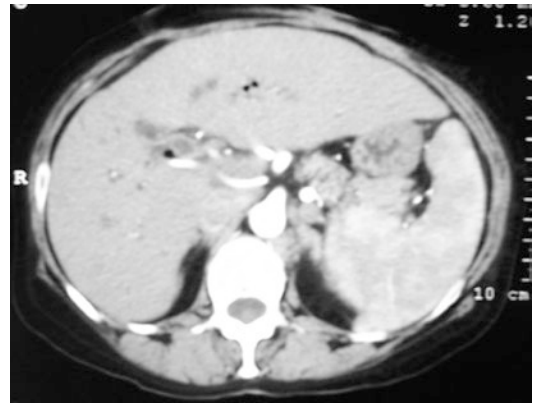

Fig. 3.7 Aberrant right hepatic artery from the superior mesenteric artery lying behind the CBD

An aberrant right hepatic artery originating from the superior mesenteric artery (Fig. 3.7) lies to the right of the CBD and posterior to the cystic duct—it may get injured resulting in profuse bleeding. Even a normally placed right hepatic artery may get injured if it has a tortuous course and lies in the Calot's triangle or even on the gallbladder neck (caterpillar turn or Moynihan's hump). Desperate attempts (with sutures, clips, or an energy source) to control this torrential bleeding from an injured right hepatic artery may cause a BDI.

3.4 Difficult Pathology

Various pathological conditions may make the cholecystectomy difficult in its various stages, e.g., entry into the peritoneal cavity, access to the right upper abdomen, handling and retraction of the gallbladder, exposure and dissection of the Calot's triangle, dissection of the gallbladder from its bed, etc.

Chances of BDI are more in a difficult case, e.g.,

1. Acute cholecystitis (Fig. 3.8) and its complications, e.g., empyema gallbladder, emphysematous or gangrenous cholecystitis,

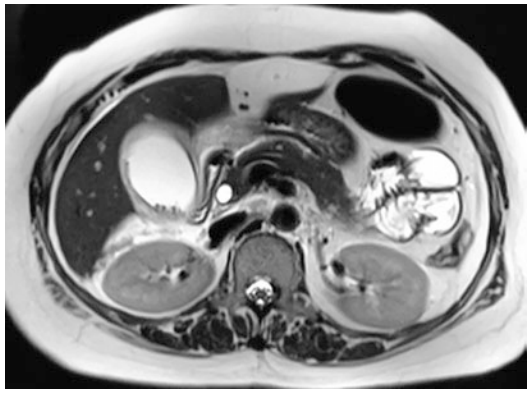

Fig. 3.8 MRI showing thickened edematous gallbladder wall suggestive of acute cholecystitis

gallbladder perforation. Acute cholecystitis is not a contraindication for laparoscopic cholecystectomy but cholecystectomy is certainly more difficult in the acute setting than in elective and should be performed by an experienced surgeon. In less experienced hands, risk of BDI is higher in patients undergoing emergency (middle-of-the-night) or early (the next morning) cholecystectomy for acute cholecystitis (cf. elective interval cholecystectomy 4–6 weeks after the acute attack has settled with conservative management).

A meta-analysis of 1625 patients in 16 reports (including 15 randomized controlled trials) revealed that early laparoscopic cholecystectomy, though associated with longer operating time, resulted in shorter hospital stay, fewer work days lost, and better quality of life (QoL) than delayed laparoscopic cholecystectomy; mortality, BDI, bile leak, or conversion rates were equal [6]. El-Dhuwaib [7] analyzed 572,223 laparoscopic cholecystectomies performed in England between 2001 and 2013—500 (0.09%) patients required bile duct reconstruction; risk of a bile duct injury requiring bile duct reconstruction was lower (OR 0.48, 0.30–0.76) if the patient did not have acute cholecystitis. The risk of BDI is less if the cholecystectomy is performed within the first 3–5 days of the onset of the acute attack when edema due to the inflammation may in fact help in dissection but is maximum during 7–14 days after the onset of the acute attack when the inflamed gallbladder is thickened and difficult to grasp, manipulate, and retract and tissues are edematous, vascular, and friable resulting in oozing which obscures vision and may increase the risk of BDI. The clips on the cystic duct may cut through the edematous friable wall or may loosen later as the inflammatory edema settles, thus causing bile leak (cystic duct blow out) in the postoperative period. The best evidence on timing of cholecystectomy for acute cholecystitis comes from the Swedish Registry for Gallstone Surgery (GallRiks). Between 2006 and 2014, 87,106 cholecystectomies were performed—15,760 (18%) for acute cholecystitis. BDI and 30-day and 90-day mortality rates were higher if time from admission to surgery exceeded 4 days than when it was 2 days. Patients operated on the day of admission also had higher BDI and 30-day and 90-day mortality rates, emphasizing the importance of optimizing the patient before surgery [8]. In case of difficulty, threshold for conversion to an open operation should be low. Conversion rates of laparoscopic cholecystectomy in acute cholecystitis were higher—23 (19%) out of 124 patients [9]. Even at open cholecystectomy, a surgical cholecystostomy may have to be performed if dissection in the Calot's triangle is difficult due to excessive inflammation. In a patient presenting after 5 days of onset of an attack of acute cholecystitis, which is not responding to conservative management (thus suggesting a complication, e.g., empyema), an US or CT guided percutaneous cholecystostomy can be performed.

It goes without saying that laparoscopic cholecystectomy for acute cholecystitis is not every surgeon's cup of tea and should be performed by an experienced surgeon.

2. Long-standing chronic fibro-atrophic (sclero-atrophic) cholecystitis (recurrent inflammatory fibrotic scarring leading to a small contracted (Fig. 3.9) shrunken thimble thick-

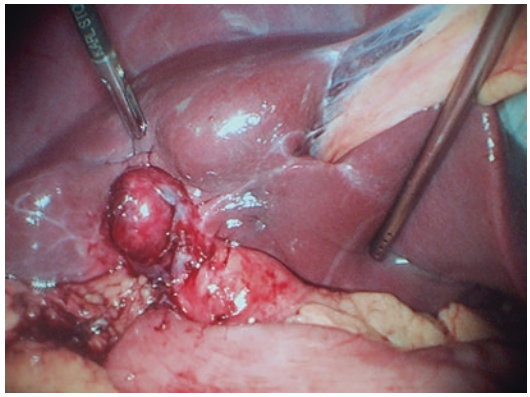

Fig. 3.9 Small contracted gallbladder caused by long-standing chronic fibro-atrophic (sclero-atrophic) cholecystitis

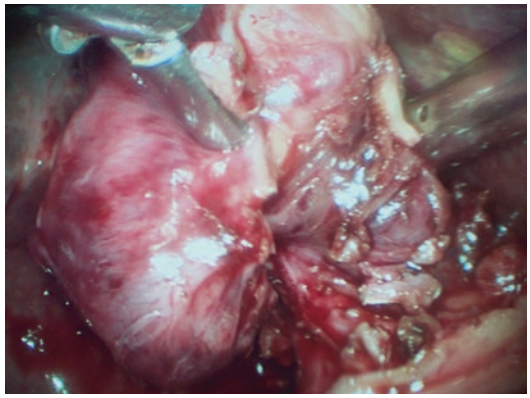

Fig. 3.10 Hidden cystic duct—there is no Calot's triangle

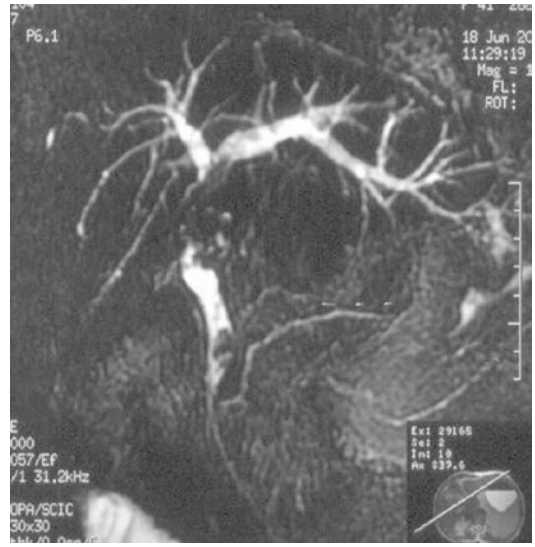

Fig. 3.11 MRC showing a large stone in gallbladder neck causing CBD compression—Mirizzi's syndrome

walled gallbladder (TWGB), which may even become intrahepatic, and resulting in fibrosed, shrunken and narrowed, even obliterated Calot's triangle). Dissection in the fibrosed scarred Calot's triangle may be difficult and forceful attempts to dissect may result in bleeding (from the cystic artery and/or the right hepatic artery) and injury to the common hepatic duct or the right hepatic duct. Partial cholecystectomy, leaving a part of the gallbladder neck behind, but removing all stones, is a safe option in such cases.

3. Strasberg [10] described the phenomenon of the "hidden cystic duct" (Fig. 3.10)—the surgeon uses the infundibular technique wherein the cystic duct is shown to flair (widen) to become the infundibulum of the gallbladder but what he thinks is the cystic duct is actually the CBD. Hidden cystic duct can happen in presence of a large stone impacted in the gallbladder neck, short/absent cystic duct, acute cholecystitis, and long-standing chronic cholecystitis.
4. A gallbladder tightly packed with stones is difficult to hold and retract; similarly, a large stone impacted in the gallbladder neck may make holding, manipulation, and retraction of the gallbladder neck difficult. One option is to open the GB, remove the stones (which are collected in a bag), and then hold the opened wall of the GB to retract it.
5. Mirizzi's syndrome (large stone in the gallbladder neck Fig. 3.11 or cystic duct which gets adherent to or may even fistulate into the CBD), if not diagnosed preoperatively or appreciated intraoperatively, may result in a major BDI, especially during laparoscopic cholecystectomy. It should be suspected if alkaline phosphatase (ALP) and/or gamma-glutamyl transpeptidase (GGTP) are elevated and US shows a large stone impacted in the gallbladder neck with intrahepatic biliary radical dilatation (IHBRD) and dilated common hepatic duct with normal CBD (mid CBD block). During operation, Mirizzi's

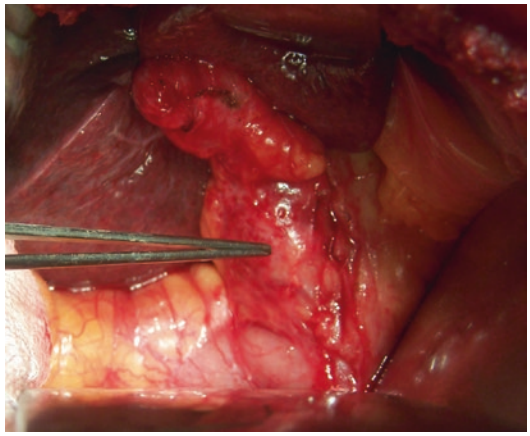

Fig. 3.12 Mirizzi's syndrome at operation—small gallbladder and CBD with large stone

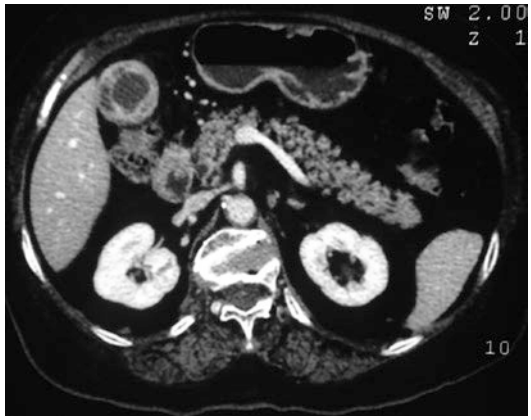

Fig. 3.13 Thick-walled gallbladder which turned out to be xantho-granulomatous cholecystitis (XGC) on histopathology

syndrome should be suspected if the Calot's triangle is obliterated (Fig. 3.12).

6. Xantho-granulomatous cholecystitis (XGC), a variant of chronic cholecystitis, resulting in a thick-walled gallbladder (TWGB), which is difficult to grasp and retract (Fig. 3.13).
7. Long-standing gall stone disease may result in complications, e.g., cholecysto-choledochal fistula, cholecysto-duodenal fistula (Fig. 3.14), cholecysto-colic fistula, choledocho-duodenal fistula which may make the cholecystectomy difficult.
8. Previous acute pancreatitis and prior endoscopic intervention, e.g., stenting, on the CBD.

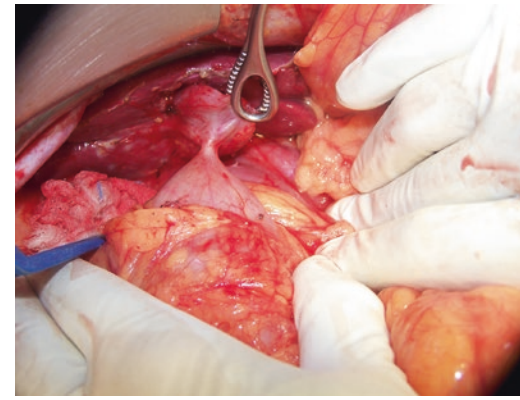

Fig. 3.14 Cholecysto-duodenal fistula

9. Cirrhosis and/or portal hypertension make the cholecystectomy difficult due to the presence of collaterals in the parietes (difficult entry into the peritoneal cavity), heavily vascularized adhesions (especially in presence of a previous laparotomy), firm stiff liver which is difficult to retract (and may even fracture on forceful retraction), presence of large, thin-walled, high-pressure collaterals in the Calot's triangle and around the gallbladder which can bleed profusely (and may even be fatal) and systemic coagulopathy. Presence of cirrhosis is not a contraindication for laparoscopic cholecystectomy but laparoscopic cholecystectomy in a cirrhotic has to be performed after adequate preparation (improvement of liver function, control of ascites and correction of coagulopathy) by an experienced surgeon. Collaterals in portal hypertension due to cirrhosis are mainly peripheral, i.e., esophago-gastric and in the splenic hilum but those in portal hypertension due to extrahepatic portal venous obstruction (EHPVO) are central, i.e., around the portal vein in the hepato-duodenal ligament, hepatic hilum and around the gallbladder (Fig. 3.15). Laparoscopic (or even open) cholecystectomy in presence of portal hypertension due to extrahepatic portal venous obstruction (EHPVO) should not be attempted unless a total shunt has been performed a few months ago to decrease the portal pressure.

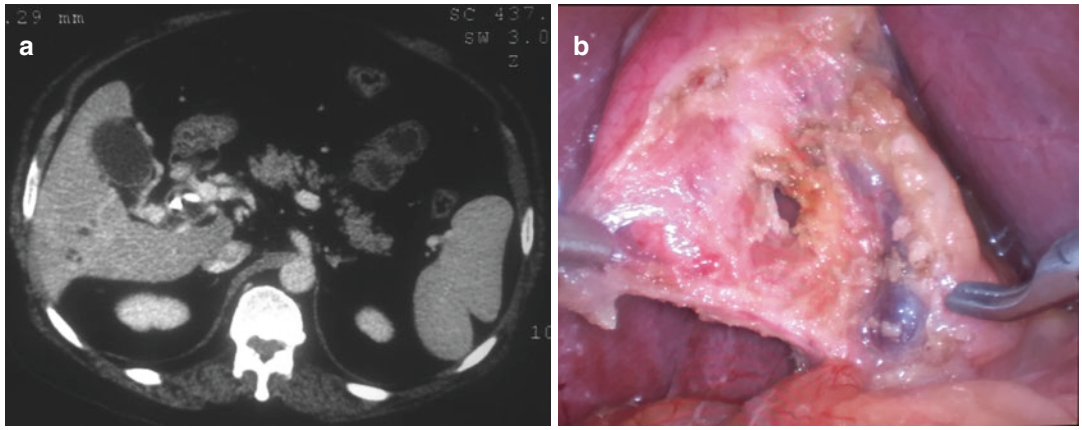

Fig. 3.15 Collaterals (**a**) around the gallbladder and (**b**) in the hepato-duodenal ligament in extrahepatic portal venous obstruction (EHPVO)

> High pressure thin walled collaterals of portal hypertension can prove to be the Waterloo of any surgeon who thinks he is a Napoleon.

A careful history and examination (elderly, male, obese, long duration of symptoms, repeated attacks of acute cholecystitis, history of previous acute pancreatitis, prior endoscopic stenting) and a good US (small contracted thick-walled gallbladder, air in the gallbladder, large stone impacted in the gallbladder neck, gallbladder packed with stones) can predict many of these situations so that a higher chance of conversion is anticipated and informed to the patient. Threshold for conversion from laparoscopic to open operation should be low in the above mentioned situations to avoid or at least reduce the risk of a BDI.

Strasberg [11] has described 4 error traps responsible for the occurrence of a BDI during cholecystectomy:

1. Obliteration of the Calot's triangle due to severe inflammation (chronic cholecystitis) which results in the gallbladder getting adherent to the CBD—even the infundibular (neck first) technique may cause injury to the common hepatic duct in such a case
2. Fundus down (first) technique may result in a major bilio-vascular injury
3. Aberrant right hepatic duct or right sectoral/segmental duct in the Calot's triangle
4. Cystic duct running parallel to the CBD

3.5 Patient

Gallbladders are usually more difficult in elderly patients than in the young. Cholecystectomy is generally more difficult in men than in women. Male gender is a strong predictor not only for conversion from laparoscopic to open cholecystectomy [12] but is also a risk factor for BDI [13]. Excessive fat in the hepato-duodenal ligament and in the Calot's triangle in obese patients makes identification of the structures (cystic lymph node, cystic artery, cystic duct, and CBD) difficult and may increase the risk of BDI. In a Nationwide (USA) Inpatient Sample (1998–2006) of 377,424 cholecystectomies with 1,124 (0.3%) BDIs, Asian race/ethnicity was a significant risk factor for BDI (odds ratio 2.3; 95% confidence interval 1.6–3.2) [14]. Similarly, Greenbaum [15] reported an increased risk of major BDI during laparoscopic cholecystectomy in Native Americans. This may be because of late presentation for treatment in these ethnic groups of patients.

BDI, and for that matter any complication of a surgical procedure, can (and does) occur in "*very important patients*" (VIPs) also.

> Anthony Eden, who succeeded Winston Churchill as the British Prime Minister (1955-1957), underwent open cholecystectomy on 12th April 1953. The official operative report did not mention anything amiss with the procedure. He had to be reex-

plored on 29th April. He was then flown to the USA where repair of a bile duct injury was performed by Richard Cattell of the Lahey Clinic on 10th June. He underwent a total of as many as 4 operations including a liver resection but finally had to resign from his position because of health reasons. He died on 5th March 1970.

It was a great tragedy that his career was savaged by a surgical error.

Braasch [16]

US Senator John Murtha, a Democratic Congressman, underwent a scheduled laparoscopic cholecystectomy on 28th January 2010 (after a previous hospitalization in December 2009, probably for an attack of acute cholecystitis) at the National Naval Medical Center in Bethesda, MD USA. It was described as a "routine minimally invasive surgery," but he required readmission to the Virginia Hospital Center, Arlington VA 3 days later—he died on 8th February 2010. It is alleged that the doctors had "hit his intestines."

http://www.medscape.com/viewarticle/716749

This could have been the small bowel (during the insertion of the first trocar), the duodenum or the transverse colon (adherent to an inflamed *gallbladder*).

3.6 Surgeon

Injuries to the bile ducts are nearly always the result of misadventures during operation.

Grey Turner [17]

When laparoscopic cholecystectomy was introduced in the 1980s, it was believed that the increased risk of BDI during laparoscopic cholecystectomy is a reflection of inadequate training and less experience and will decrease with proper training and more experience with the new technique—"the learning curve" [18]. This has not been proved to be so. Injuries occur (and will continue to occur) in the hands of well-trained and highly experienced surgeons also. More experienced surgeons may operate upon more and more difficult cases; this may be responsible for the fact that the incidence of BDI in their hands continues to remain high. Gigot [19] reported that 1/3rd of 65 BDIs in Belgium occurred in the hands of experienced surgeons. A survey of 1500 surgeons from Spain revealed that one-third of BDIs occurred after the first 200 cases [20]. One-third of 704 BDIs, found during a survey in the USA, occurred in the hands of surgeons who had performed more than 200 laparoscopic cholecystectomies [21]. Learning curve for laparoscopic cholecystectomy may extend well beyond 50 cases [22]. In British Columbia Canada, 61% surgeons experienced a BDI after 100 cholecystectomies [23].

A BDI can occur during a difficult cholecystectomy but a large number of BDIs occur during a so-described "easy," "routine," or "uncomplicated" lollipop or *laddoo* (an inexpensive and highly popular Indian sweet!) cholecystectomy because of dangerous surgery as a result of surgical misadventure or misperception, i.e., misidentification of the biliary ductal anatomy. The fact that the cholecystectomy was described by the referring surgeon to be an "easy" and "straightforward" cholecystectomy, therefore, should not rule out the suspicion of a BDI in an unsettled patient referred to a higher center for further management. In one-third of 704 BDIs in a US survey, the cholecystectomy was defined as "routine" by the operating surgeon [21]. In a nation-wide survey of 56,591 laparoscopic cholecystectomies performed in 184 hospitals in Italy (1998–2000), 235 (0.4%) BDIs were reported; no risk factor was present in 80% cases and in about half of these 235 cases, the cholecystectomy was described as "easy" [24].

3.7 Dangerous Surgery

Lack of proper and adequate training, inexperience, overconfidence, and disregard to the basic principles of surgery and the techniques of cholecystectomy are responsible for a large majority of BDIs during cholecystectomy. BDI rates continue to remain high even in countries with well-structured and properly regulated training programs and strict certification and accreditation processes; the rates are likely to be even higher in countries with poorly structured and ill-regulated training programs and lax certification and accreditation processes.

The classical laparoscopic cholecystectomy BDI occurs when the CBD, especially if it is undilated (normal sized), is mistaken for the

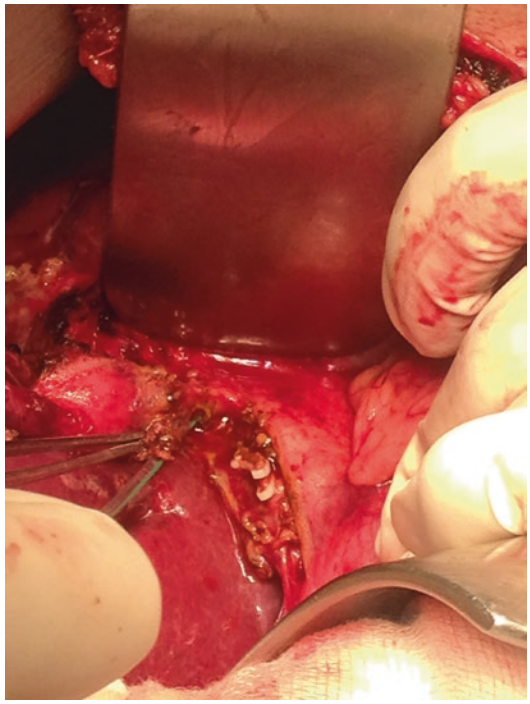

Fig. 3.16 Excision of a segment of CBD—the classical laparoscopic BDI

cystic duct and is clipped and divided. This is more likely to occur when the gallbladder neck is retracted upwards (instead of downwards)—this aligns the gallbladder neck, cystic duct and the CBD in one straight line and the CBD is then misidentified as the cystic duct. The injury, if recognized at this stage, results in transection only without its excision, i.e., there is no loss of a segment of the bile duct. Such an injury (transection, but without excision) may still be amenable to an end-to end repair. If the dissection proceeds further, the common hepatic duct is encountered which is also clipped and divided resulting in a segmental loss (excision) of the CBD (Fig. 3.16); such an injury is very often associated with an injury to the right hepatic artery also which was mistaken for the cystic artery. An end-to-end repair is not possible in such injuries without tension and a Roux-en-Y hepatico-jejunostomy is required for intraoperative repair.

Simplicity of the procedure may produce a false sense of security and produce complications. Risk taking behavior of the surgeon, i.e., a bold, speedy, impatient, or adventurous surgeon, is more likely to be associated with BDI during laparoscopic cholecystectomy [25]. Since cholecystectomy is a very commonly performed procedure, familiarity with the procedure sometimes breeds contempt and makes the surgeon a bit callous towards the operation. Some surgeons, especially those with large experience, sometimes develop a casual attitude towards cholecystectomy; this must be avoided. Overconfidence on the part of an experienced surgeon, especially in a straight forward (the so-called easy or routine) cholecystectomy, may be responsible for the classical laparoscopic BDI—CBD mistaken for the cystic duct and a segment excised. A hurried cholecystectomy for a simple (the so-called easy or routine) gallbladder by an overconfident surgeon is an ideal setting for a BDI. Overwork and stress may be responsible for some complications during any surgical procedure—Yaghoubian [26] reported decreased BDI during laparoscopic cholecystectomy in the era of the reduced 80-h resident work week.

3.8 Equipment

Laparoscopic cholecystectomy is a resource intensive, gadget driven technique—inadequate equipment viz. low definition camera, poor quality image, ill maintained (e.g., uninsulated) instruments may also be responsible for an inadvertent bile duct (or non-biliary—vascular or bowel) injury. In case bleeding occurs, suction will be used; non-availability of a high flow insufflator at such time will result in loss of pneumoperitoneum. Desperate blind attempts (with sutures, clips or an energy source) to control the bleeding in such a situation may then cause a BDI.

Electrocautery is a useful equipment but should be used judiciously as excessive use of high wattage current in the Calot's triangle can cause thermal injury to the right wall of the

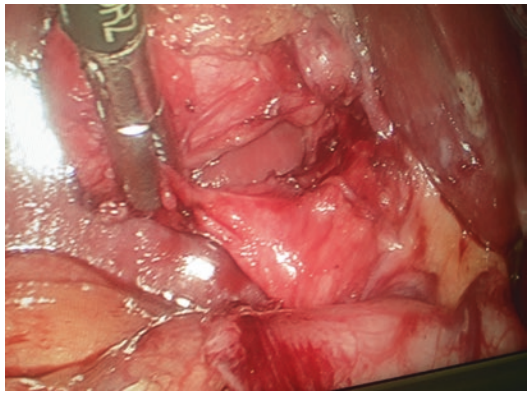

Fig. 3.17 Tenting of the CBD caused by excessive lateral traction on the gallbladder neck

CBD. These thermal injuries may not manifest in the early postoperative period but may cause delayed sloughing of the wall of the CBD resulting in bile leak after a few days (when the patient has been discharged home). Thermal injury to the blood supply of the CBD may also result in a delayed (after months or even years) ischemic stricture of the common bile with no history of bile leak in the postoperative period.

3.9 Technique

Excessive upward (superior, cranial, cephalad) traction on the gallbladder neck may align the cystic duct with the CBD in a straight line; the CBD, especially if it is normal sized (undilated) may then be misperceived (misidentified) as the cystic duct and dissected, clipped and divided (the classical laparoscopic BDI).

Excessive outward (lateral) traction on the gallbladder neck during clipping of the cystic duct can cause tenting of the CBD (Fig. 3.17) thus producing a camel hump on it; a clip applied on the cystic duct can then occlude a part of the circumference of the CBD which may then slough to result in a lateral hole in the CBD.

Deep dissection beyond the cystic plate into the liver parenchyma in the gallbladder bed may result in an injury to a peripheral sub segmental intrahepatic bile duct resulting in bile leak from the gallbladder bed.

3.10 Clips

Proper size clips should be used—200 (small) for the cystic artery and 300 (medium) or 400 (large) for the cystic duct— if a large clip is required (for a wide cystic duct), it must be doubly ensured that what is being thought of as the cystic duct is not the CBD.

> A wide cystic duct is the CBD, unless proved otherwise.

ANECDOTE: *One of the CBDs which was divided during laparoscopic cholecystectomy in our department was thought to be a wide cystic duct by the operating surgeon.*

If a clip is applied too close to the junction of the cystic duct and the CBD (Fig. 3.18), it can compromise the lumen of a normal sized (undilated) CBD.

> It is safer to leave a few mm of the cystic duct than to remove or clip even one mm of the CBD.

Clips must be properly applied so that they are not loose and do not come off; this is particularly so if the cystic duct is wide and thick-walled; a self locking clip, e.g., Hem-o-loc®

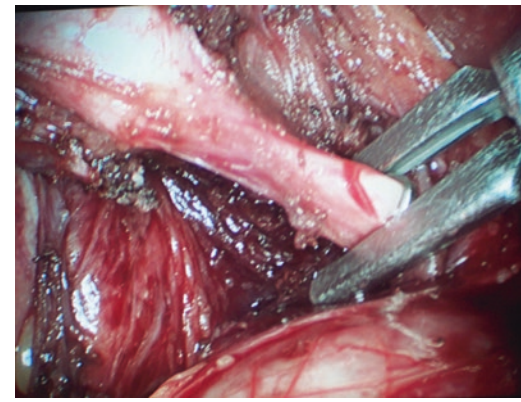

Fig. 3.18 Wrong application of the clip too close to the junction of the cystic duct and the CBD

(Weck) is more secure in such situations. A too tightly applied clip or scissoring (crossing of the two limbs) of the clip may cut through or cause pressure necrosis of a very thin-walled or acutely inflamed, edematous friable cystic duct and cause bile leak in the early postoperative period. A clip applied on a previous clip is bound to be loose. Metal clips can transmit electric current across—electrocautery should not be used on clips or clipped structures. Gauze should not be used after clips have been applied as the clip can get entangled in the threads of the gauze and come off.

A residual CBD stone may cause biliary obstruction and raised intrabiliary pressure letting even a properly applied clip to come off the cystic duct resulting in cystic duct blow out and bile leak in the early postoperative period.

3.11 Bleeding

The reflex response of a surgeon to a bleed during laparoscopic cholecystectomy is an attempt to control it with cautery or with application of clips but injudicious use of electrocautery or clips for control of bleeding in the Calot's triangle (Fig. 3.19), where the view is obscured by the bleed, is a recipe for BDI. A deep suture to control bleeding in the gallbladder bed, especially near the gallbladder neck, may cause injury to the intrahepatic segment V, right anterior sectoral or even main right hepatic duct.

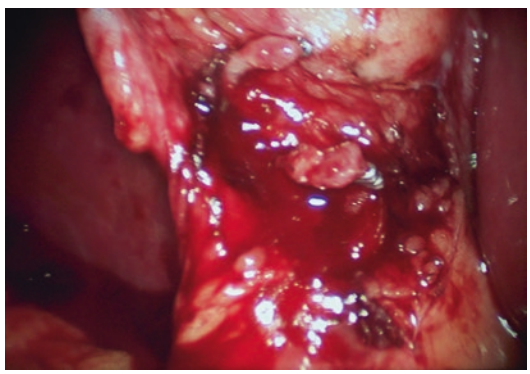

Fig. 3.19 Bleed in Calot's triangle—injudicious use of electrocautery or clips to control this bleed can cause BDI

3.12 Common Bile Duct Exploration

CBD exploration, especially of an undilated normal sized CBD, is the second most common (following cholecystectomy, the commonest) cause of BDI and consequent benign biliary stricture. Exploration of a normal sized (undilated) CBD to find out small stones may cause injury to its wall and result in a stricture**.** Such stones should be removed either endoscopically (preoperatively) or using the transcystic technique (during the cholecystectomy). Sutures applied to the CBD for closure of the choledochotomy may compromise its lumen and cause stricture. Excessive circumferential dissection and use of electrocautery around the CBD may cause ischemic injury as a result of interruption of its axial blood supply. This may not cause postoperative bile leak but may result in a late (after months or even years) benign biliary stricture. Such strictures are, however, usually low and incomplete (i.e., ductal continuity is present) and are easily amenable to endoscopic dilatation.

Forceful calibration or dilatation of the duodenal papilla with metal instruments, e.g., Bake's dilator, bougie, etc. during CBD exploration may cause injury and laceration leading to fibrosis and stricture. This low stricture with intact ductal continuity is, however, eminently suitable for endoscopic dilatation. The Author (VKK) strongly discourages use of any metal instruments to calibrate or dilate the CBD or the duodenal papilla; they do not have a place in today's surgical practice. If at all, a soft catheter or an infant feeding tube should be used for calibration and a balloon, e.g., Foley or Fogarty catheter for dilatation of the papilla.

3.13 Cystic Duct Blow Out

It is possible to have postoperative bile leak without an injury to the CBD—cystic duct blow out (Strasberg Type A BDI). This can occur

1. because of a loosely applied clip on the cystic duct (as a routine, two clips should be applied on the CBD side of the cystic duct so that at least one of them is tight and secure enough and works),
2. because of too tightly applied clips which cause pressure necrosis of a thin-walled or inflamed cystic duct
3. because of the clip becoming loose on a thick-walled cystic duct (a self locking clip, e.g., Hem-o-loc® (Weck) should be used in presence of a thick-walled/wide cystic duct),
4. because a retained (missed) CBD stone may impact at the papilla and increase the intrabiliary pressure resulting in cystic duct blow out and bile leak even after an uneventful cholecystectomy. The CBD stone can be picked up on ultrasonography (US) and confirmed on magnetic resonance cholangiography (MRC) or on endoscopic ultrasonography (EUS). The CBD stone should be removed endoscopically and a stent/naso-biliary drain placed to reduce the bile leak from the opened cystic duct.

3.14 Unrecognized

Majority of the BDIs remain unrecognized during the cholecystectomy (more so during laparoscopic cholecystectomy); a BDI is detected during laparoscopic cholecystectomy in only one-fourth to one-third of cases (See Chap. 9). In another one-fourth to one-third, it manifests in the early postoperative period as bile leak (See Chap. 10 or external biliary fistula (See Chap. 11). In the remaining cases, it may manifest later (after weeks or months) during the follow up as a benign biliary stricture (See Chap. 12).

The fact that the patient has gone home without any problems does not necessarily mean that the cholecystectomy was ‘safe’.

Invited Commentary on Mechanisms of Causation of Bile Duct Injury

Miguel A. Mercado

Bile duct injury is a tragedy both for the patient and the surgeon. Although many efforts have been made worldwide to prevent these injuries, their frequency remains constant. Several strategies have been developed—probably the most used and promoted is the critical view of safety of the Calot’s triangle developed by Strasberg as well as the culture of safety in which the value of changing the strategy of the operation (even laparoscopically) to partial cholecystectomy and conversion to open procedure is stressed. In my point of analysis, the only strategy effective to reduce the incidence of bile duct injuries is selecting the right patient for laparoscopic cholecystectomy. A good strategy is not to operate on patients with diagnosis of “biliary dyskinesia” and ultrasonography showing non-inflamed gallbladder with asymptomatic stones.

This chapter written by Prof. Kapoor, a worldwide recognized surgeon devoted to the treatment of bile duct injuries, exposes in a very nice dynamics the causes of bile duct injury. I can say that almost 99.9% (one can never say 100%) of the causes of bile duct injuries are very well discussed, resulting in a very well developed and useful chapter. Though not in order of frequency, each possibility of injury is nicely described. Also, some nowadays uncommon causes of injury, e.g., (technically wrong) placement of T tube are included. I have observed injuries that are the consequence of excessive traction of the Hartmann´s pouch in severely inflamed gallbladders, in which the complete bile duct is transected. In these cases, loss of substance (segment) of the common bile duct is the rule.

One of the most feared and non-presentable bile duct injuries is related to an anatomical variation in which the cystic duct joins an aberrant right hepatic duct. In these instances, the surgeon places the clips on the accessory duct even if he or she has followed the rules of the critical view of safety. Also in some instances, the surgeon does not detect this injury, even if he or she has done a cholangiography, because intrahepatic bile ducts in a part of the liver are not seen. Management of this type of injury is challenging as a bilio-enteric anastomosis of this type of duct has a high failure rate. Usually the duct is very small and does not have good blood circulation. Closing the duct and waiting for atrophy or secondary biliary cirrhosis of this part of the liver can be advised. Placement of a drain near the open duct can also be advised—this will create a small volume external biliary fistula; one can then wait for spontaneous closure. In my experience, some of these cases may need partial resection of the liver at a later stage. In these cases, ERC has no role (as the duct is isolated from the main biliary tree) and the external biliary fistula persists even after placement of an endoscopic stent.

Perhaps not to be discussed in this extraordinary chapter, injuries to the right hepatic artery deserve a short comment. If the complete right hepatic duct is sectioned (not considered in the Strasberg classification but in the Stewart-way classification as D) bilio-enteric anastomosis is needed (and very seldom) even liver resection. In my experience, if after removing all the clips at the time of repair, retrograde flow of the sectioned hepatic artery is obtained reconstruction of the divided artery is unnecessary.

Prof. Kapoor is to be congratulated. In my more than 25 years interest in operating upon and reading about the topic of bile duct injury repair, this is one of the most complete chapters on the topic.

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4 Tips and Tricks for Safe Cholecystectomy

Vinay K. Kapoor

NOTE: The Author (VKK) has addressed the issue of safe cholecystectomy in a separate publication Kapoor VK. Safe Cholecystectomy – A to Z (Foreword by John G Hunter). Lucknow: Shubham 2010: 1-128. ISBN 978-81-910315-0-8.

The book is available for free download at http://vkkapoor-india.weebly.com/uploads/1/4/6/7/1467272/safe_chole_a_to_z.pdf.pdf

The Author (VKK) recently spearheaded the formulation of Society of Endoscopic and Laparoscopic Surgeons of India (SELSI) Consensus Statement for Safe Cholecystectomy — Prevention and Management of Bile Duct Injury - Parts A and B published in the Indian Journal of Surgery 2019. https://doi.org/10.1007/s12262-019-01993-2 and https://doi.org/10.1007/s12262-019-01994-1.

European Association for Endoscopic Surgery (EAES) has published clinical practice guidelines for prevention and treatment of bile duct injuries during cholecystectomy [1].

The Society of American Gastrointestinal Endo Surgeons (SAGES) has set up a Safe Cholecystectomy Task Force (SCTF) which has launched a Safe Cholecystectomy Program to educate surgeons to reduce complications (especially bile duct injury during cholecystectomy) [2].

The Turkish HPB Surgery Association has published an expert consensus document for safe cholecystectomy [3].

Hori [4] has described an 8-point mandatory protocol for safe cholecystectomy.

Research Institute against Cancer of the Digestive System (IRCAD) made its recommendations including 7 statements on safe laparoscopic cholecystectomy [5].

Tokyo Guidelines (TG 18) have recently published safe steps in laparoscopic cholecystectomy for acute cholecystitis [6].

In spite of an increased risk of bile duct injury (BDI) as compared to open cholecystectomy, laparoscopic cholecystectomy has become the gold standard of management of symptomatic gallstones and is here to stay. A BDI is a major complication of a relatively simple operation—cholecystectomy. It is a serious complication as it can be dangerous, life threatening and even fatal. Management of a BDI is difficult. BDI may result in a complication for the surgeon also in the form of a medico-legal suit.

> Every surgeon must make every attempt to make every cholecystectomy safe.

Please read this Chapter along with Chap. 5.
Also see Invited Commentary on Tips and Tricks for Safe Cholecystectomy by Jose M Schiappa (pp 43–44)

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V. K. Kapoor (ed.), *Post-cholecystectomy Bile Duct Injury*, https://doi.org/10.1007/978-981-15-1236-0_4

4.1 Training

A surgeon should embark on laparoscopic cholecystectomy only after receiving adequate and proper training, preferably during residency.

4.2 Selection of Cases

In the beginning of his/her career, a surgeon should take young, thin built female patients with short duration of symptoms, no history of acute cholecystitis, jaundice/cholangitis or acute pancreatitis, and distended thin-walled gall bladder on ultrasonography (US).

4.3 Work Up

All patients scheduled to undergo cholecystectomy must have a good US (evaluating the gall bladder wall for any thickening and looking for any evidence of biliary obstruction in the form of intrahepatic biliary radical dilatation IHBRD) and complete liver function tests (LFT).

4.4 Equipment

Proper equipment viz. high definition camera, good quality monitor, high flow insufflator and properly insulated instruments are a must to perform a safe laparoscopic cholecystectomy and reduce the risk of BDI. A 30° telescope is a must to view the Calot's triangle from both in front (anterior) and behind (posterior). A good electrocautery is required but an ultrasonic scalpel is not mandatory for performing laparoscopic cholecystectomy.

4.5 Consent

A proper detailed (including a chance of conversion to open operation and a small risk of BDI) informed written consent must be obtained from all patients undergoing laparoscopic cholecystectomy.

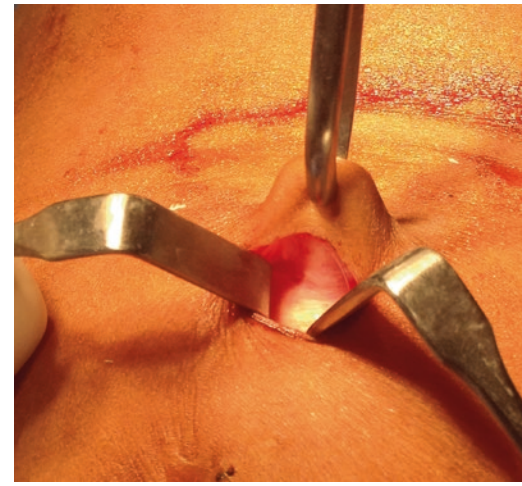

Fig. 4.1 Open technique of insertion of the first trocar

4.6 Access (Entry)

Open technique (using an infra-umbilical incision) of insertion of the first trocar (Fig. 4.1) is safer as compared to blind insertion of Veress needle and closed insertion of the first trocar. The patient should have evacuated her urinary bladder just before being shifted to the operation room.

4.7 Ports

The two working ports (epigastric and subcostal) should be so placed that the tips of the instruments introduced through them meet at the gall bladder neck at a right angle.

4.8 Landmarks

First part of the duodenum, segment IV (quadrate lobe) of liver, Rouviere's sulcus [7], Hartmann's pouch, and cystic lymph node are important and useful landmarks during cholecystectomy.

The first part of the duodenum should be retracted down (caudad) to view the supraduodenal part of the hepato-duodenal ligament—in a thin built patient, the bluish hue of the common bile duct (CBD) (Fig. 4.2) may be obvious. Hori [4] have described a U-shaped line from the right border

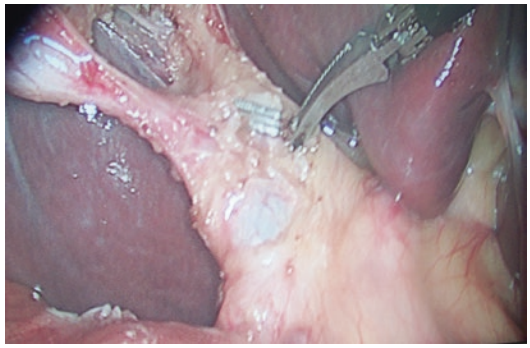

Fig. 4.2 Bluish hue of the common bile duct in thin built patient

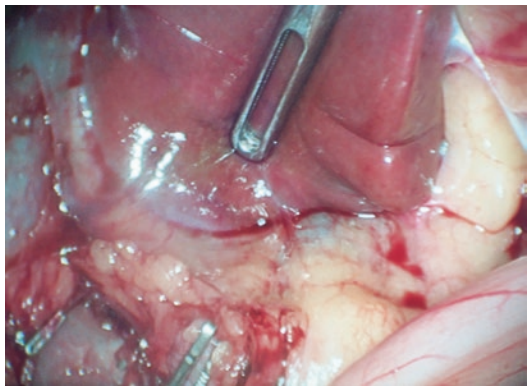

Fig. 4.3 Base of the quadrate lobe (segment IV) of the liver—hepato-duodenal ligament runs down from here to the first part of the duodenum

of the round ligament across the base of the quadrate lobe (segment IV) to the left border of the gall bladder—the bottom of this U identifies the hepatic hilum. The line joining the first part of the duodenum to the base of segment IV (quadrate lobe) identifies the hepato-duodenal ligament (Fig. 4.3); dissection should be kept to the right of it.

Rouviere's sulcus on the undersurface of the right lobe of liver is an important and useful landmark during laparoscopic cholecystectomy. It marks the position of the right posterior sectoral pedicle. Dissection in the Calot's triangle should be kept anterior to (in front of) the Rouviere's sulcus (Fig. 4.4).

Hartmann's pouch is an outpouching of the gall bladder neck—CBD lies to its left (Fig. 4.5).

Cystic lymph node (Fig. 4.6) in the Calot's triangle is a very important landmark to identify the cystic artery. If the dissection is kept lateral to (to the

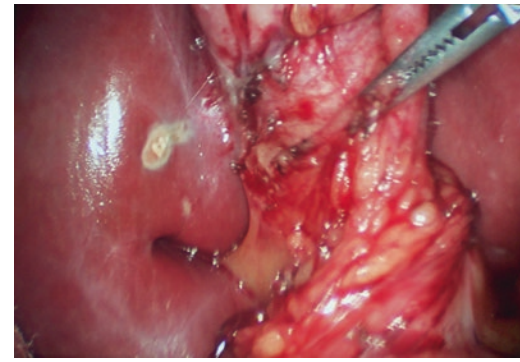

Fig. 4.4 Rouviere's sulcus marks the position of the right posterior sectoral pedicle. Dissection in the Calot's triangle should be kept anterior to (in front of) the Rouviere's sulcus

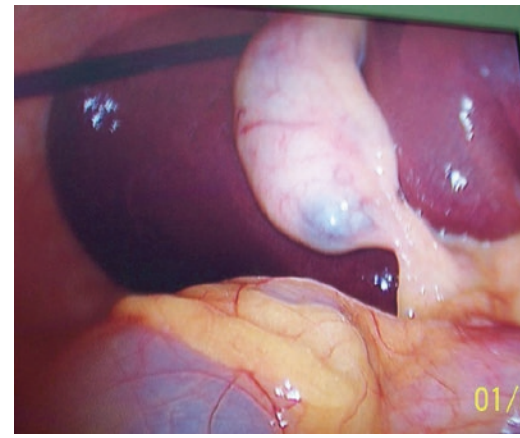

Fig. 4.5 Hartmann's pouch is an outpouching of the gall bladder neck—common bile duct lies to its left

right of) the cystic lymph node, it is very unlikely that damage will be caused to the CBD [8].

Pulsations of the proper hepatic artery should be looked for in the hepato-duodenal ligament—CBD lies to the right of the proper hepatic artery.

Sutherland [9] suggested the mnemonic B-SAFE for 5 subhepatic landmarks, viz. B: bile duct, S: Rouviere's sulcus, A: hepatic artery, F: umbilical fissure, E: enteric (duodenum) for correct placement of cognitive map to avoid a BDI cholecystectomy.

NOTE *Size of a duct does not differentiate between the cystic duct and the CBD; normal CBD can be just 3-4 mm in diameter and may be easily mistaken for the cystic duct.*

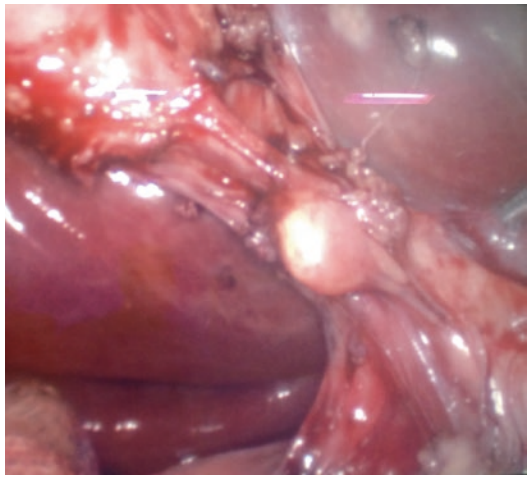

Fig. 4.6 Cystic lymph node in the Calot's triangle—dissection should be kept on the right of it

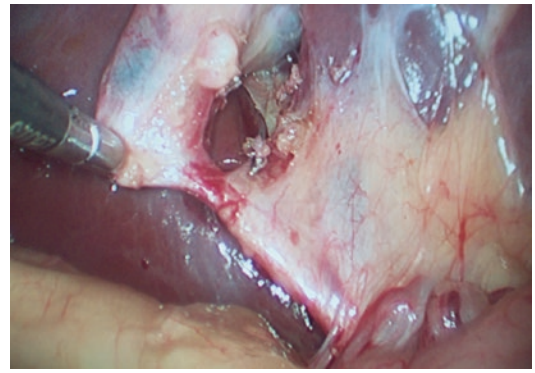

Fig. 4.8 Gall bladder neck should be retracted downwards and outwards (laterally) towards the right elbow of the patient in order to open the Calot's triangle

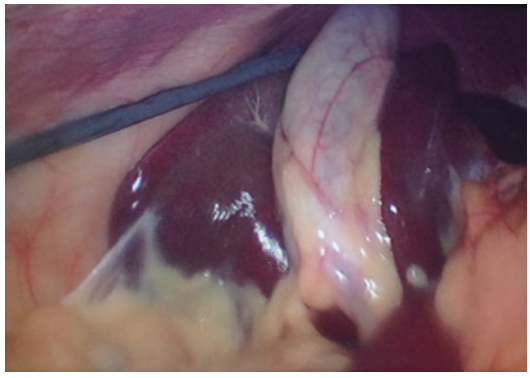

Fig. 4.7 Gall bladder fundus of the gall bladder should be retracted upwards (cranially) towards the right shoulder of the patient in order to retract the liver

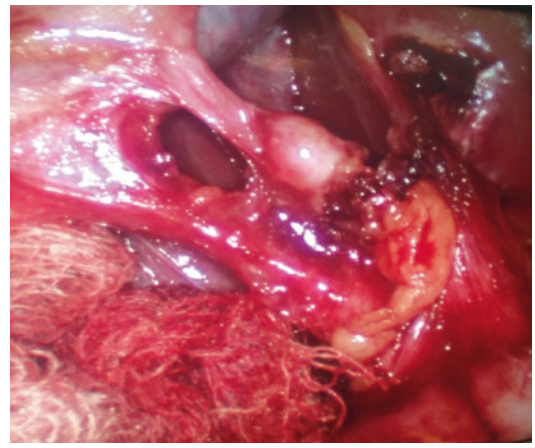

Fig. 4.9 Calot's triangle is bound by the inferior surface of liver, the common hepatic duct and the gall bladder neck

4.9 Retraction

Gall bladder fundus should be retracted upwards (cranially) towards the right shoulder of the patient in order to retract the liver (Fig. 4.7). Gall bladder neck should be retracted downwards and outwards (laterally) towards the right elbow of the patient in order to open the Calot's triangle (Fig. 4.8) and to place the cystic duct at a right angle to the CBD. Gall bladder neck has to be retracted upwards and medially towards the left shoulder of the patient in order to view the posterior surface of the Calot's triangle.

4.10 Calot's Triangle

The surgical Calot's triangle is bound by the inferior surface of liver, the common hepatic duct and the gall bladder neck—cystic duct (Fig. 4.9). It contains the cystic artery and the cystic lymph node. The first step is to open the peritoneum on the posterior (inferior) surface of the Calot's triangle—this widens the Calot's triangle. Peritoneum on the anterior (superior) aspect of the Calot's triangle should then be opened. Calot's triangle should be viewed from both overhead and underneath aspects—this can

easily be done with a 30° telescope. Blunt dissection (e.g., with the tip of the suction cannula) is recommended for dissection in a soft (normal) Calot's triangle.

4.11 Critical View of Safety

The concept of the critical view of safety was first described by Strasberg in 1995. It includes dissection in the Calot's triangle to free it of fatty, fibrous, and areolar tissue so that two and only two structures viz. cystic artery and cystic duct are seen to be attaching the gall bladder to the hepato-duodenal ligament (Fig. 4.10). As far as possible, the critical view of safety should be demonstrated [10]. The Author (VKK) prefers to dissect, isolate, and divide the cystic artery first—this opens the Calot's triangle further as cystic artery is a taut structure while the cystic duct has a tortuous course.

4.12 Flagging Manoeuvre

Flagging, i.e., turning the gall bladder infundibulum (neck) around is a helpful manoeuvre to show that the cystic duct and the CBD are seen as two separate structures.

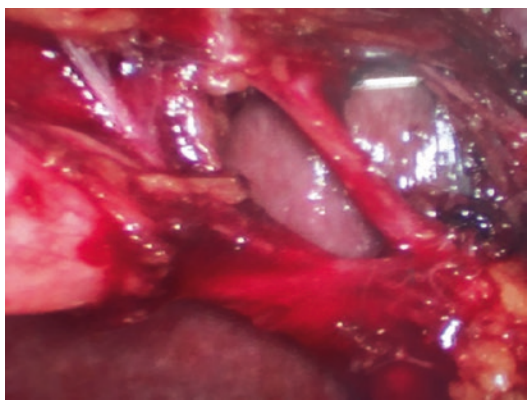

Fig. 4.10 Critical view of safety—two and only two structures viz. cystic artery and cystic duct should be seen to be attaching the gall bladder to the hepato-duodenal ligament

4.13 Hug the Gall Bladder

While doing cholecystectomy, the surgeon should stay close to (hug) the gall bladder neck—cystic duct junction (and not the cystic duct—CBD junction) [11]. Cystic duct should be demonstrated to be flaring (widening, funnelling) into the gall bladder neck (infundibulum). Cystic artery should also be divided close to (on) the gall bladder—to avoid injury to the right hepatic artery. This may mean dividing the anterior and posterior branches of the cystic artery separately. Another advantage is that even if the clip is loose, there is a remaining stump of the cystic artery which can then be controlled with another clip.

4.14 Clip

Cystic duct should be 'palpated' with a grasper for any stones which should be milked back into the GB or crushed into small fragments for spontaneous passage into the CBD and then across the papilla into the duodenum. Before firing/pressing the clip, it must be ensured that its tips are beyond the circumference of the structure being clipped, i.e., the cystic artery and the cystic duct and no other tissue/structure is being clipped. While applying the first clip on the cystic duct, traction on the gall bladder neck should be released to avoid the clip encroaching on a part of the circumference of the CBD. Double clips should always be applied on the patient (CBD) side of the cystic duct and the cystic artery for additional security.

4.15 Second Opinion

Before the cystic duct is clipped and divided it is advisable to take the opinion of an independent second observer [4] who is unbiased from the heuristic impression of the operating surgeon. The Author (VKK) has recently described in-vicinity 'colleaguography' (IVC) (Fig. 4.11) (opinion of a surgical colleague who is available in the vicinity of the operation room) as a universally available, easy to obtain and no cost alternative to intra-operative cholangiography (IOC) to prevent a BDI during laparoscopic cholecystectomy [12].

Fig. 4.11 The Author (VKK) (not scrubbed for the case) providing in vicinity 'colleaguography' to his fellows while they are performing laparoscopic cholecystectomy

4.16 Cystic Plate

Gall bladder should be dissected off its bed in the liver preserving the cystic plate on the gall bladder bed—the plane of dissection, thus, is between the gall bladder wall and the cystic plate. This is a bloodless plane and also avoids injury to a peripheral intrahepatic bile duct.

4.17 Extraction

A thin-walled gall bladder full of multiple small stones or an acutely inflamed gall bladder full of pus should be removed in a bag to avoid bile/pus and stone spill.

4.18 Closure

The aponeurosis (linea) of the 10 mm ports should be closed (with a long acting absorbable suture, e.g., polydioxanone PDS®) in order to prevent an incisional hernia (Fig. 4.12); at the 5 mm port sites, skin alone is closed.

4.19 Discharge

The patient should be discharged only if and when he/she is comfortable (with no or minimal pain), ambulant and tolerating oral diet; vitals are stable and the abdomen is normal (soft, non-distended and non-tender with normal bowl sounds). Some

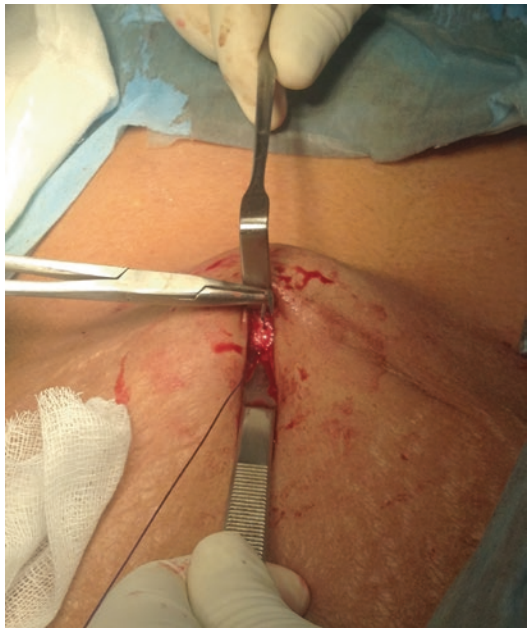

Fig. 4.12 10 mm ports should be closed to avoid an incisional hernia later

surgeons obtain liver function tests (LFT) again before discharge.

4.20 Follow-Up

Patients undergoing laparoscopic cholecystectomy should be instructed to return to the hospital/surgeon in case of any problem, e.g., fever, abdominal pain, jaundice, etc. during the early follow-up.

All attempts must be made and every precaution taken to ensure that the cholecystectomy becomes safe for the patient.

Invited Commentary on Tips and Tricks for Safe Cholecystectomy

Jose M. Schiappa

This is a very complete chapter on safe cholecystectomy. This being a subject of my special interest also, I believe I can still help readers by providing a few more 'Tips':

The fact—no doubt very correct—that most cholecystectomies in the world, nowadays, are done laparoscopically, this approach making it 'easier', does not mean that indications should be 'liberalised'. Only patients with symptomatic gall bladder stones should have the gall bladder removed.

As for training, it is never too little to say that laparoscopic surgery needs, absolutely, full and proper training. There are no excuses at this moment, regarding this issue: there are more than enough laparoscopic surgery courses, properly done and properly taught. Curricula of these courses are good and we have moved far away from the courses existing in the beginning of this type of surgery, which were done in 2 days and surgeons came back to their hospitals performing these surgeries.

For the work up of these patients, and as some readers will comment on it, let us consider the need of intraoperative cholangiography (IOC). IOC helps to clarify the biliary anatomy and it also helps, because of that, to minimise the gravity of iatrogenic BDI. It does not reduce the risk of BDI but there is a possibility that the injuries can be less harmful.

In 1997, Palazzo published a paper referring to the Risk Criteria for the Existence of Common Bile Duct (CBD) Stones; in there he calculated the possibility of a certain patient having CBD stones, depending on several factors:

1. Low Risk (CBD stones in 2–3% of cases)—if there was no history of stone migration, i.e., cholangitis or pancreatitis, if the liver function tests were normal and if the abdominal US showed a CBD diameter of less than or equal to 7 mm.
2. Medium Risk (CBD stones in 20–40% of cases)—patients having a history of stone migration, i.e., cholangitis or pancreatitis, having gamma-glutamyl transpeptidase (GGTP) and/or transaminases (ALT/AST), and/or alkaline phosphatise (ALP) twice the

normal value and/or if the abdominal US showed a CBD with a diameter 8–10 mm.
3. High Risk (CBD stones in 50–80% of cases)—patients having a recent history of jaundice, cholangitis or pancreatitis, having a rise to double or more of alkaline phosphatase and/or an abdominal US showing a CBD diameter of more than 10 mm.

Accordingly, I suggest that patients should follow this protocol:

1. If they belong to the low risk group—follow to laparoscopic cholecystectomy without any further investigations.
2. If they belong to the medium risk group—follow to laparoscopic cholecystectomy with a laparoscopic IOC
3. If they belong to the high risk group—follow to laparoscopic cholecystectomy after having ERCP (or having first a diagnostic MRCP or EUS, followed by therapeutic ERCP or with intraoperative exploration of the CBD)

So far, I have been happy with these options.

Let us now look at the other points of this chapter:

Equipment—it is very important to realise, once for all, that the leading surgeon is responsible both for the team's expertise and the quality and appropriateness of the equipment; the laparoscopic instruments need special attention, especially to their electric insulation. Specially the lack of insulation; sometimes, it is not clearly visible and when not appropriate it can cause serious problems.

Added to it, there is a recently found problem which is called "Inattention Blindness". When the surgeon has all his or her attention directed to a certain point in the operating field, many things around it become 'not visible'. This is called the 'cone of attention' and everything happening out of it is not visible to the surgeon; accidents may happen here without being noticed.

Regarding the informed consent, it is also quite important to note that—and big series show this—when there is conversion from laparoscopic to open procedure, there is a high rate of injuries in all these cases.

For access, I use, in general, the approach by Veress needle, having it placed in the Palmer space (in the left hypochondrium, below the last left ribs) after emptying of the stomach with a naso-gastric tube. It is also quite acceptable to adopt the open technique before starting the pneumoperitoneum.

The placement of ports has also to consider the build of each patient, considering, for instance, that, in general, men have the gall bladder more deeply placed. Ports shall be placed in a 'roundish' way, around the main working point.

As for the Critical View of Safety, I also try to have it shown every time. In the Netherlands, it seems to be even mandatory to get two photos of it, proving it has been obtained, one from the front and another from the back. Sometimes it cannot be obtained, and it does not in itself prevent iatrogenic injuries, but it is a good way to, at least, minimise the gravity of the BDI.

Flagging manoeuvre is very important to perform, every time, as it can take care of the "Hidden Cystic Duct Syndrome", where sometimes the view shows what can be understood as the cystic duct and has the common hepatic duct hidden behind the gall bladder.

Cutting the cystic artery first is wise, also because it is fragile and can get ruptured if the cystic duct is cut first.

Last comments have to do with the knowledge of possible anatomic anomalies—which can explain some injuries—and the care necessary to take if there is some unexpected haemorrhage. If dealt with without precautions, it can lead to wrong placement of control clips and lead to bile duct injury.

In the end, my recommendation is: this unfortunate situation and surgical disaster can happen to any surgeon, no matter how well trained, how experienced and how confident. In the unfortunate case that this disaster happens, do not forget the odds of success: repair done by the same team which caused the injury is successful in 11–17% of cases; if done by a specialist team the success rate is more than 90%, according to Stewart and Way [13] and Lillemoe [14].

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Prevention of Bile Duct Injury During Cholecystectomy

5

Vinay K. Kapoor

Bile duct injury (BDI) is a dreaded complication of laparoscopic cholecystectomy. Most major BDIs are avoidable—they can be prevented in majority of cases by paying proper attention to the biliary ductal anatomy (which, it must be remembered, is unique for each individual patient) (See Chap. 1), respecting the gallbladder (GB) pathology and using the correct technique of safe cholecystectomy (See Chap. 4). Every surgeon must make every effort to prevent a BDI during cholecystectomy. Cholecystectomy should not be considered and neglected as a small, minor operation and should not be taken lightly/casually; it should receive the attention it deserves as most BDIs occur during a so-called simple, straightforward, "lollipop" or "laddoo" (an inexpensive and popular Indian dessert) cholecystectomy.

> Surgery is not a race to be run in the shortest time— it should be like an unhurried leisurely refreshing evening walk.

Please read this chapter along with Chap. 4.
Also see Invited Commentary on Prevention of Bile Duct Injury During Cholecystectomy by L. Michael Brunt (pp 57–58)

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https://doi.org/10.1007/978-981-15-1236-0_5

5.1 Training

Adequate and proper training in laparoscopic surgery as a part of residency program, as opposed to learning laparoscopic surgery at weekend courses or workshops may offer some protection against BDI during laparoscopic cholecystectomy. While increasing evidence tends to overcome the learning curve, it must be remembered that high level of experience alone is not adequate to ensue successful performance of laparoscopic cholecystectomy [1].

5.2 Anticipate

One should "anticipate" (and be prepared for) a difficult cholecystectomy and a higher risk of a BDI in presence of known risk factors, e.g., elderly, obese, male, acute cholecystitis, long duration of symptoms, previous acute pancreatitis, associated common bile duct (CBD) stones, prior endoscopic intervention (stenting), palpable distended GB (mucocele, contracted and thick-walled GB (TWGB) on ultrasonography (US). A beginner may better refer these cases to a more experienced surgeon.

Laparoscopic cholecystectomy for acute cholecystitis should be performed by an experienced surgeon—it is NOT for everyone!

> Every surgeon who performs cholecystectomy should pray that a bile duct injury does not happen but must be prepared for it, in case it happens.

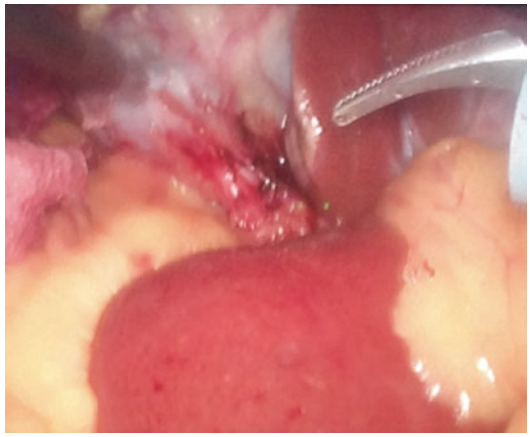

Fig. 5.1 Overhanging quadrate lobe (segment IV) of liver obscuring the view of the Calot's triangle—the surgeon should not hesitate to put an extra port to retract the overhanging quadrate lobe in order to get a clear unobstructed view of the Calot's triangle

5.3 Access

Many surgeons talk of reducing the number of ports (from four to three), but the Author (VKK) always uses four ports and is of the opinion that one should not hesitate to place even an extra (fifth) port in order to achieve better exposure of the Calot's triangle (by retracting an overhanging quadrate lobe Fig. 5.1) or to suck out blood and clear the view (in case of brisk bleeding). The surgeon should never hesitate to add the ports during laparoscopic cholecystectomy if needed; additional stab wounds are never "invasive" [1].

5.4 Adhesions

Omental/bowel adhesions to the parietes (following prior laparotomy) or to the GB (because of inflammation) should be carefully dissected using sharp dissection (scissors) to avoid bleeding or injury to vessels. A densely adherent duodenum (Fig. 5.2)/colon to the GB may indicate an underlying fistula.

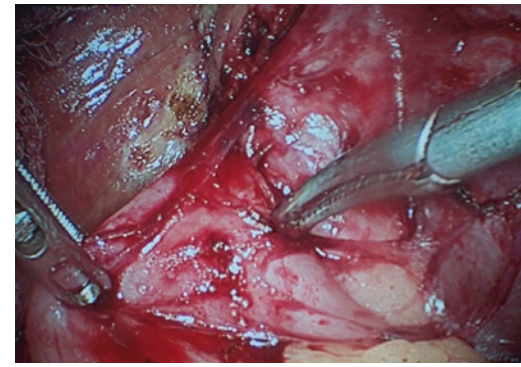

Fig. 5.2 Duodenum adherent to the gallbladder—presence of dense adhesions should raise the suspicion of a fistula

5.5 Gall Bladder

A tense distended GB, e.g., in mucocele, empyema, is difficult to hold and retract; it should be aspirated to decompress it (Fig. 5.3); this not only helps to hold the GB but also gains 1-2 mm extra length of the cystic duct.

5.6 Direction

Cystic duct travels in a horizontal direction (cf. CBD which has a vertical course) and joins the CBD at an angle. Extreme upward (cephalad) traction on the GB neck may, however, make the cystic duct align with the CBD when the CBD may be mistaken for the cystic duct and clipped and divided (classical laparoscopic BDI). Traction on the GB neck should, therefore, always be down (caudad) and out (lateral) so that the CBD does not get aligned with the cystic duct. This traction on the GB neck should be temporarily released (Fig. 5.4) when clipping the cystic duct to avoid tenting of the CBD (Fig. 5.5) and its partial clipping. Flush ligation or clipping of the cystic duct at its junction with the CBD should be avoided as it may encroach on the lumen of a normal sized (undilated) CBD.

> It is better to leave a few mm of the cystic duct than to remove or clip even one mm of the CBD.

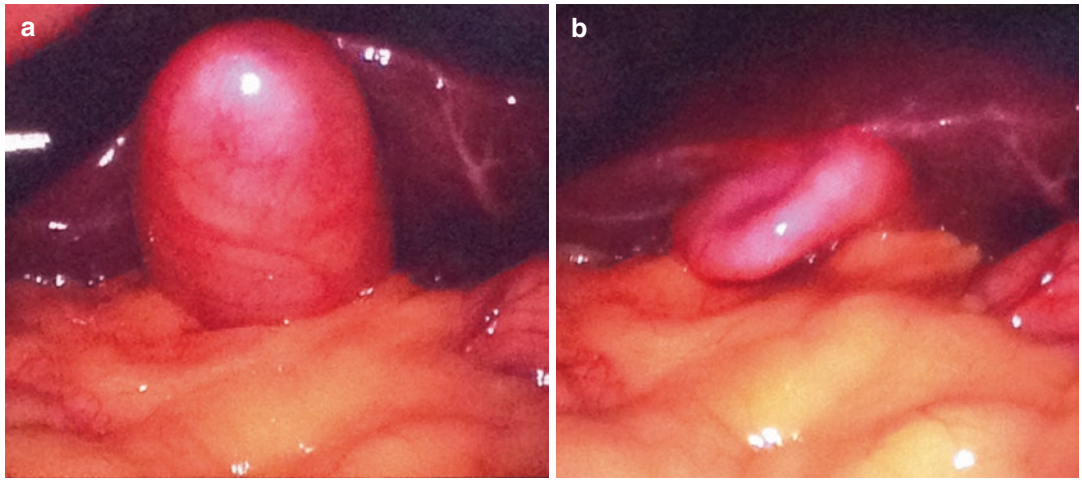

Fig. 5.3 (**a**) Tense distended gallbladder in mucocele—may be difficult to hold and retract; (**b**) gallbladder decompressed after needle aspiration—easier to hold now

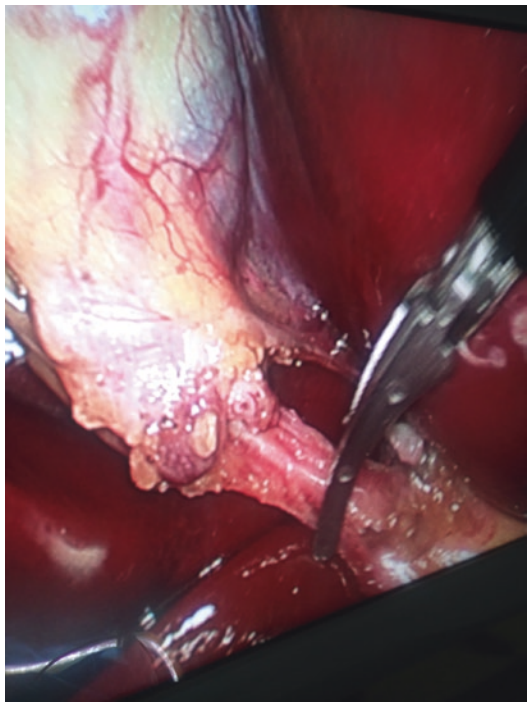

Fig. 5.4 Gallbladder neck retraction released before clips are applied on the cystic duct

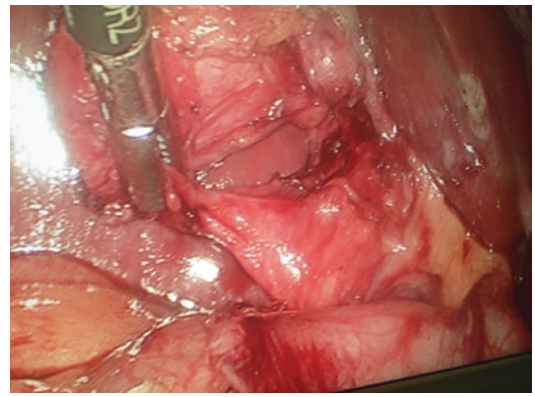

Fig. 5.5 Tenting of the common bile duct caused by excessive lateral traction on the gallbladder neck

5.7 Cystic Duct

Beware of a long vertical cystic duct (which runs parallel to the CBD and may spiral around either behind or in front of the CBD to open on its left side Fig. 5.6). A wide "cystic duct" or a vertical "cystic duct" may actually be the CBD. In case of doubt, intraoperative cholangiography (IOC), if available, can help to delineate the biliary anatomy.

Normally, the surgeon should encounter only two tubular structures in the Calot's triangle—the cystic duct and the cystic artery. A "third" tubular structure in the Calot's triangle should ring alarm bells.

If after dividing the cystic duct, another tubular structure is seen in the Calot's triangle, the surgeon must stop and think—is it that the first duct which was thought to be the "cystic duct" (which has already been divided) was in

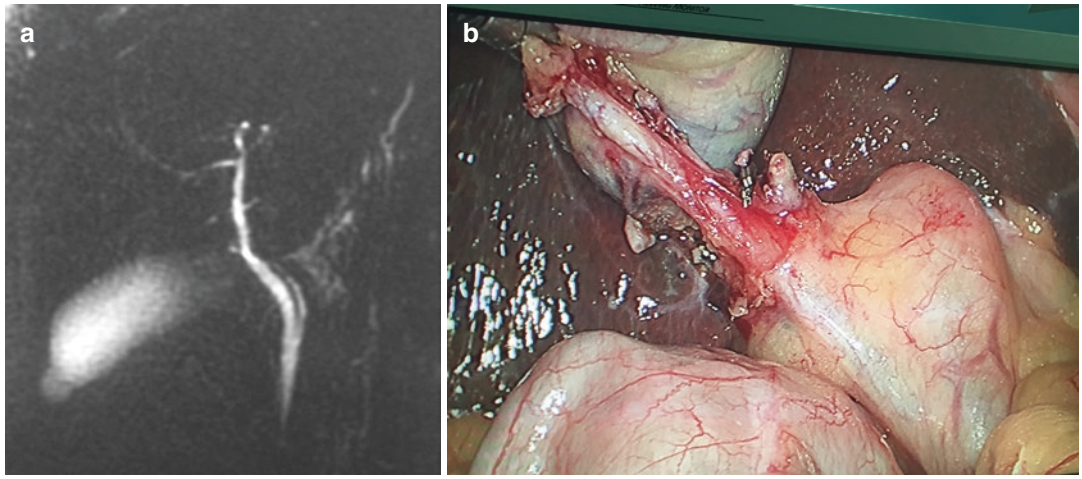

Fig. 5.6 (**a**) Long tortuous cystic duct on MRC; (**b**) at operation

fact the CBD and this tubular structure encountered now is the common hepatic duct (CHD). An IOC at this stage will delineate the biliary anatomy. If further dissection is stopped at this stage, an excision of the CBD can still be avoided (although, unfortunately, the CBD has already been divided). If the CHD and CBD are shown to be intact, the structure which has been divided was indeed the cystic duct and this "another duct" may be a cholecysto-hepatic duct or an aberrant right segmental or sectoral duct or even the main right hepatic duct (RHD).

5.8 Electrocautery

Electrocautery (diathermy) is a useful device for performing laparoscopic cholecystectomy but should be used judiciously and carefully to avoid thermal injuries to the bile duct, bowel (duodenum and colon) and the vessels (hepatic artery and portal vein).

Use of diathermy should be avoided in the Calot's triangle during cholecystectomy to avoid thermal injury to the bile ducts (and the hepatic artery). "Purists" avoid the use of cautery at all in the Calot's triangle. If at all it has to be used, it should be set at low wattage, small amounts of tissue should be picked and short bursts should be used. A hook with diathermy may be used to lift the peritoneum of the Calot's triangle. Since the area of work, i.e., Calot's triangle, is very small, the cautery pedal (button) should be in control of the operating surgeon (*and NOT an assistant!*) and should be activated only after the tissue to be cauterized is touched by/held in the instrument. The entire metal (conducting) part of the activated instrument should be in the visual field to ensure that it is not inadvertently touching any other organ/structure. Cautery should be used on tissues close to the GB, away from the CBD. Use of cautery on clipped structures should be avoided as metal clips can transmit electric current and cause thermal damage to the normal structures beyond the clips.

Blind, injudicious and excessive use of electrocautery should be avoided in case bleeding occurs in the Calot's triangle during cholecystectomy. Bleeding should first be temporarily controlled with pressure (using the GB itself or a gauze piece) and cautery should be applied only if the source of bleeding is clearly identified. Bipolar cautery results in less lateral thermal damage and should preferably be used, if at all, for control of bleeding in the Calot's triangle.

5.9 Clip

When the first (CBD side) clip is being applied on the cystic duct, it must be ensured that CBD is not getting tented (Fig. 5.5) and that the clip is not encroaching on the right lateral wall of the CBD—lateral traction on the GB neck should be released (Fig. 5.4) at the time of applying the first

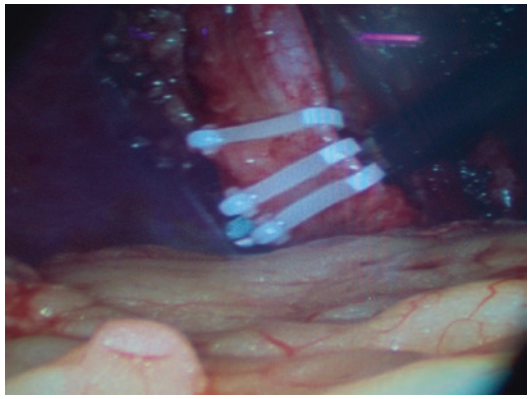

Fig. 5.7 Self locking clips are more secure than metal clips

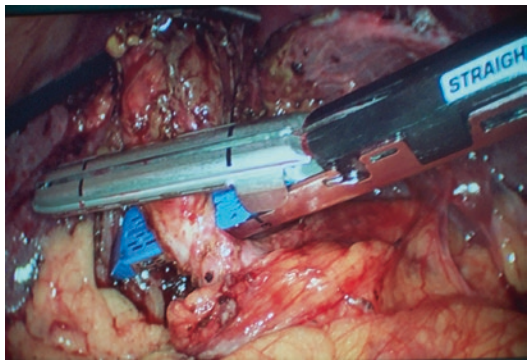

Fig. 5.8 Stapler being applied for a wide cystic duct

clip on the cystic duct. The clip should be applied perpendicular to the cystic duct (and not oblique, so that its ends do not encroach on the CBD.

Self locking clips, e.g., Hem-o-loc^{R} Weck (Fig. 5.7) are more secure than ordinary clips. It may not be safe to use metal clips on a wide, inflamed, or thick-walled cystic duct; it should be handled with a Hem-o-loc^{R} Weck, endoloop, suture, or stapler (Fig. 5.8).

Gauze should not be used after clips have been applied as the clip can get entangled in the threads of the gauze and may come off when the gauze is being removed.

5.10 IOC/POC

Intraoperative/peroperative/on-table cholangiography (IOC/POC/OTC), first described by Mirizzi in 1937, was used during the open cholecystectomy era to detect CBD stones. In the 1980s, it found a new use as a road map to prevent BDI during laparoscopic cholecystectomy.

IOC can help to delineate the biliary ductal anatomy if it is not clear or is aberrant. IOC/POC, however, is no replacement for a safe surgical technique.

IOC for detection of CBD stones can be selective, i.e., performed in moderate or high risk cases only but IOC for prevention of BDI has to be routine, i.e., performed in all cases in order to delineate the biliary ductal anatomy. Logistics of obtaining an IOC in every laparoscopic cholecystectomy may be difficult—very few centers in India perform routine IOC during laparoscopic cholecystectomy. Technical difficulties, e.g., valves of Heister offering resistance to the passage of the cannula in the cystic duct may make an IOC difficult; forceful pushing of the cannula into such a situation may result in avulsion of the cystic duct from the CBD causing a hole (lateral injury) in the right wall of the CBD. IOC is usually performed by making a nick in the anterior wall of the cystic duct and introducing a cannula into it. The performance of IOC itself may cause a BDI if the CBD is misidentified as the cystic duct and a nick is made into it. Even if the cystic duct has been correctly identified, the nick in the cystic duct may inadvertently extend into the CBD. Correct interpretation of the IOC also requires knowledge and training; there are several reports describing patients in whom IOC was performed but was not interpreted correctly and BDI still occurred. Way et al. [2] found that only nine out of 43 BDIs seen on IOC were correctly interpreted during the operation. Moreover, routine IOC is not cost effective because of the low incidence of BDI during laparoscopic cholecystectomy.

Eighty percent of BDIs were detected intraoperatively when IOC was obtained vs. only 45% when it was not [3]. A meta-analysis of 327,523 laparoscopic cholecystectomies reported in 40 case series found that the incidence of BDI decreased from 0.43% with selective use of IOC to 0.20% with routine use of IOC [4]. BDI occurred less frequently (2380/613,706; 0.39%) in patients in whom IOC was used than in those in whom IOC was not used (5531/956,655 l; 0.58%) [5]. Nuzzo [6] surveyed 184 surgical units in Italy which performed 56,591 laparo-

scopic cholecystectomies—CBD injury rate was 0.32% in the routine IOC group vs. 0.43% in the selective IOC group. BDI occurred in 0.21% of 37,533 patients who had IOC vs. 0.36% of 55,399 patients who did not have IOC during cholecystectomy [7]. Regular use of on-table cholangiogram (OTC) reduced the risk of a major BDI requiring bile duct reconstruction (n = 500) in 572,223 patients who underwent laparoscopic cholecystectomy in England between 2001 and 2013 [8]. Rystedt and Montgomery [9] reported 168 BDIs—IOC was performed in 93% cases; the injury was diagnosed intraoperatively in 92% cases, probably because of the IOC. In New York State, during 2000–2014, the rate of IOC during laparoscopic cholecystectomy decreased and this resulted in increased rate of BDI [10].

Debate, however, continues about the role of IOC to prevent BDI during laparoscopic cholecystectomy. Some studies [11–13] have shown that IOC reduces the risk of BDI during laparoscopic cholecystectomy, and is cost effective [14]. Massarweh and Flum [15], in a collective review, contended that evidence supporting IOC is strong and recommended that IOC should be considered a system level approach to avoid BDI during laparoscopic cholecystectomy. Sheffield [7], on the other hand concluded that IOC is not effective as a preventive strategy against BDI during cholecystectomy. Slim and Martin [16] also observed that IOC/POC may not necessarily prevent a BDI but may help in its intraoperative detection or at least reduce its extent. Routine IOC during every laparoscopic cholecystectomy, on the other hand, is not recommended by many reports [17]. The Author (VKK) has recently described in-vicinity "colleaguography" (IVC) (Fig. 5.9) (opinion of a surgical colleague who is available in the vicinity of the operation room) as a universally available, easy to obtain and no cost alternative to IOC to prevent a BDI during laparoscopic cholecystectomy [18].

5.11 Laparoscopic US

Laparoscopic US (in addition to detecting CBD stones) may help to delineate the biliary ductal anatomy also during laparoscopic cholecystectomy and may reduce the risk of BDI. No major BDI was reported in 1381 patients who underwent laparoscopic cholecystectomy with laparoscopic US [19]. Cost, availability, and expertise, however, limit its use during every laparoscopic cholecystectomy.

5.12 Near Infrared Fluorescent Cholangiography

Intraoperative dynamic (real time) fluorescent cholangiography using a fluorescent imaging system after intravenous injection of indocyanine green (ICG) has been described as an alternative to IOC [20, 21]. Direct injection of ICG into the GB has also been reported recently [22].

5.13 Neck First

Demonstration of the critical view of safety (CVS) may not be possible in patients with fibrosed/obliterated Calot's triangle due to long standing chronic cholecystitis (Fig. 5.10a)—described as the hidden duct syndrome by Strasberg [23]. Fundus first (antegrade) is a useful technique during the open operation in case of a difficult GB. Separation of the fundus from the liver during laparoscopic cholecystectomy, however, loses the retraction of the liver. Strasberg and Gouma [24] have cautioned against the use of fundus down cholecystectomy in presence of a severely inflamed (acute or chronic) GB. Contractive inflammation shortens and thickness the cholecystic plate which results in a right portal pedicle injury within the liver parenchyma when fundus down technique is used. They reported eight such patients in whom fundus down technique was attempted and an extreme vasculo-biliary injury occurred. Four required hepatectomy and one required liver transplant; four patients died, one was still under treatment and only three had a normal outcome.

In a difficult cholecystectomy (fibrosed, frozen, obliterated Calot's triangle), a neck first dissection (Fig. 5.10b) is recommended—a plane is created between the GB neck (at a safe distance from the hepato-duodenal ligament) and the GB bed after which a subtotal (partial) cholecystectomy can be performed.

Fig. 5.9 The Author (VKK) (not scrubbed for the case) providing in vicinity "colleaguography" to his fellows while they are performing laparoscopic cholecystectomy

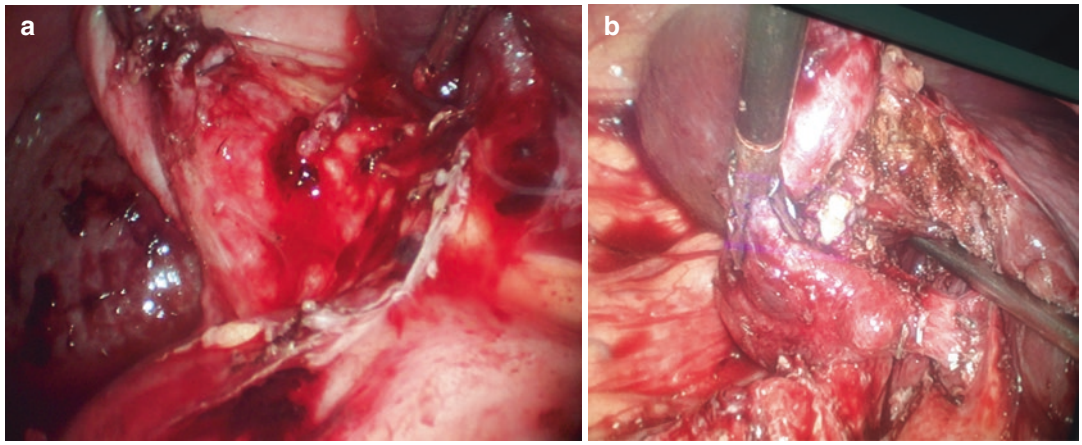

Fig. 5.10 (**a**) No Calot's triangle; (**b**) Neck first dissection

5.14 Subtotal (Partial) Cholecystectomy

The Author prefers the term subtotal (most of the gall bladder removed) to partial (some part of the gall bladder removed) cholecystectomy.

If the critical view of safety (CVS) cannot be demonstrated because the anatomy is not clear or the pathology is difficult, e.g., in case of a difficult/absent Calot's triangle (Fig. 5.11) or when Mirizzi's syndrome is suspected, subtotal cholecystectomy, leaving the cystic duct and even a part of the GB neck behind, is a safe option. GB is opened in the mid-body (Fig. 5.12) and all stones are removed. The residual GB neck may be sutured with a continuous interlocking suture of long-acting absorbable suture or stapled. While taking the suture bites, care should be taken to avoid an inadvertent injury to the adjacent CBD. If a stapler is to be used, adequate space has to be created behind the GB neck; the stapler blades should be applied parallel to the direction of the CBD. The cystic artery (or its two branches—anterior and posterior) embedded in the GB wall may bleed if the GB is divided without using a stapler. If the cystic duct is blocked, which becomes evident when no bile flows back from the CBD into the opened GB, the residual GB neck may even be left as it is (i.e., open) after destroying the mucosa with electrocautery. Strasberg [25] has described these two types of subtotal cholecystectomy as reconstituting (remnant GB neck stump closed) and fenestrating (remnant GB neck stump left open) (Fig. 5.13) types. Subtotal cholecystectomy can be performed laparoscopically also [26]. The

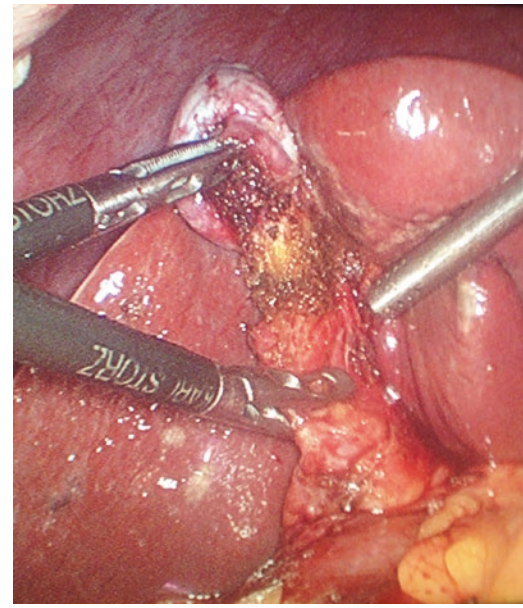

Fig. 5.12 Gallbladder opened in the mid-body for subtotal cholecystectomy

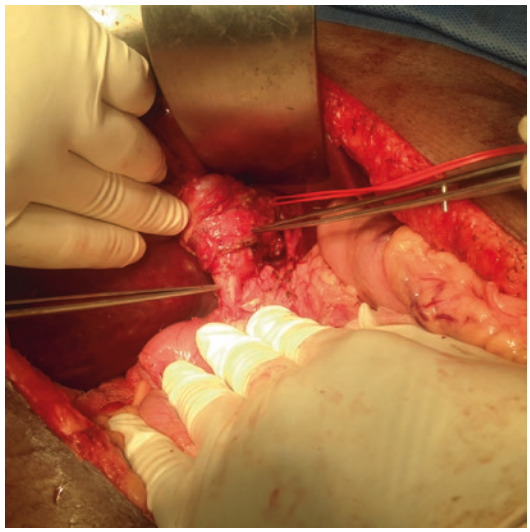

Fig. 5.11 No Calot's triangle

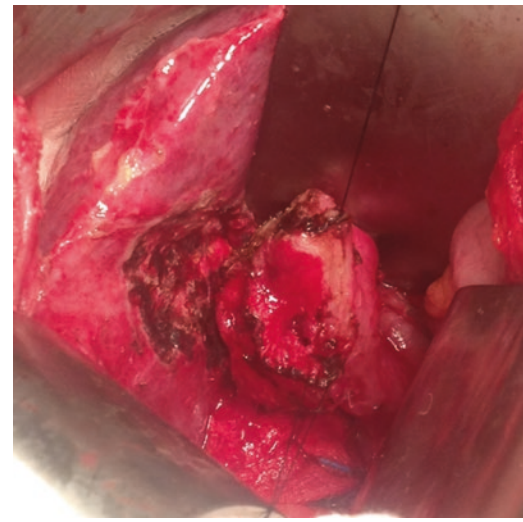

Fig. 5.13 Fenestrating type of subtotal cholecystectomy

residual GB stump, however, may reform stones, become symptomatic, and require completion cholecystectomy at a later date; this can be done laparoscopically in some cases [27]. The fact that a part of the GB (neck) is left behind should be documented in the records and communicated to the patient (for medicolegal) purposes.

> It is easier for the second surgeon to do a completion cholecystectomy after a subtotal cholecystectomy than to repair a benign biliary stricture caused by an inadvertent bile duct injury.

Another form of subtotal cholecystectomy is when the part of the wall of the GB densely adherent to the liver is left behind; the mucosa on the residual GB wall is cauterized and destroyed. This is a useful procedure in presence of cirrhosis.

NOTE: *We had reported "partial" cholecystectomy way back in 1993 [28], but now the Author (VKK) prefers to use the term subtotal cholecystectomy as the attempt should be to remove as much of the GB wall as is safely possible and destroy all GB mucosa.*

5.15 Mirizzi's Syndrome

Mirizzi's syndrome is presence of a large stone in the GB neck causing extrinsic compression on the CBD; in advanced stages, a cholecysto-choledochal fistula may form resulting in the GB stone lying in the CBD. It should be suspected if LFTs (serum bilirubin, alkaline phosphatase ALP and gamma-glutamyl transpeptidase GGTP) are deranged and US shows mid CBD block, with a large stone in the GB neck. Diagnosis can be confirmed by cholangiography—magnetic resonance cholangiography (MRC) or endoscopic retrograde cholangiography (ERC). In presence of Mirizzi's syndrome, the large stone impacted in the GB neck may obscure the Calot's triangle—dissection in the Calot's triangle is hazardous and should be avoided. The GB may be opened on the stone itself and the stone removed; this provides more space for dissection in the Calot's triangle.

Depending on whether a cholecysto-choledochal fistula is present or not and the extent of the fistula (in relation to the diameter of the CBD), treatment options include partial cholecystectomy, cholecysto-choledochoplasty (covering the fistula in the CBD with the remaining part of the GB wall) or biliary-enteric anastomosis. In Mirizzi's syndrome with cholecysto-choledochal fistula, laparoscopic cholecysto-choledochoplasty has been reported [29] but the Author (VKK) recommends conversion to open operation; the extent of the fistula is assessed and either cholecysto-choledochoplasty or hepatico-jejunostomy is performed.

5.16 Bleeding

If bleed occurs during dissection in the Calot's triangle, panic should be avoided; a gauze pack should be introduced and firm pressure applied for at least 5 minutes (*by the clock!*). This will result in most of the (venous) bleeds to stop; in case of a bleed from the cystic artery, the bleeding point (vessel) will now be better seen to be controlled with electrocautery (preferably bipolar), clip or suture. Utmost care should be taken when controlling the bleed in or near the Calot's triangle so as not to cause an inadvertent injury to the CBD. Desperate blind attempts should not be made to control the bleed by applying cautery, clips, or suture in a pool of blood.

Brisk arterial bleeding, e.g., from an avulsed cystic artery which has caused a hole in the right hepatic artery or from the right hepatic artery itself may need conversion from laparoscopic to open operation. While the abdomen is being opened, bleed should be temporarily controlled with a soft, e.g., bowel clamp. Ideally, the divided right hepatic artery should be repaired (by a vascular or transplant surgeon) but if that is not logistically possible, it can be safely ligated/clipped in a patient with normal LFTs with no major adverse effects in the postoperative period (except transient increase in transaminases ALT/AST).

Capillary ooze or venous bleed in the GB bed can almost always be controlled with firm pressure with a gauze for a few (3–5) minutes. The mobilized GB can also be used to apply pressure on the GB bed to control bleeding. Spray mode of cautery at high wattage (and argon beam coagulator ABC, if available) can also be used to control the bleed in the GB bed.

5.17 Conversion

Conversion of laparoscopic to open cholecystectomy should not be considered as a failure or defeat on the part of the surgeon—it is an indicator of sound clinical judgment on the part of the surgeon for the sake of the patient's safety. It is a safety valve in case of a difficult cholecystectomy to avoid the deadly trap of persisting with dissection and ending up with a BDI. Laparoscopic should be converted to open cholecystectomy "by choice" at an early stage in a difficult cholecystectomy, e.g., if the anatomy is not clear or if the pathology is too difficult so that no progress is being made before the surgeon has to convert "per force" due to a complication (e.g., bleeding or BDI) [30]. A low threshold for conversion can keep the incidence of BDI low; conversion rate in the Danish national database of 20,307 cholecystectomies was 7.6% [31] and conversion rate in 348,311 laparoscopic cholecystectomies in the UK (2000–2004) was 5% [32]. It must, however, be remembered that a difficult GB at laparoscopy is difficult at open operation also and a BDI can (and does) occur even after conversion from laparoscopic to open cholecystectomy. Even at open operation, subtotal cholecystectomy is a safe option.

> A score of conversions is any day better than even one bile duct injury.
>
> While several surgeons have been sued by the patients for a bile duct injury, it is very unlikely that a surgeon will be sued by a patient for conversion from laparoscopic to open cholecystectomy.
>
> It is better to be safe than sorry.

5.18 Cholecystostomy

If unexpected empyema is encountered during the operation and the surgeon is not experienced/confident enough to perform a safe cholecystectomy for an acutely inflamed difficult GB, cholecystostomy is an option. GB is opened at the fundus, stones are removed and the cholecystostomy is closed around a Foley catheter placed in the GB through the opening. The catheter may be brought out through a flap of omentum which is wrapped around the GB. This (surgical) cholecystostomy, however, is transperitoneal, i.e., the GB is opened on its peritoneal surface. If logistics permit, an intraoperative laparoscopic guided percutaneous transhepatic cholecystostomy performed by an interventional radiologist in the operation room itself is a better option.

5.19 Retract

If extreme difficulty is encountered during cholecystectomy, e.g., small, fibrotic, contracted thimble thick-walled GB (Fig. 5.14) and the surgeon is not experienced enough to safely handle the situation, it is not a bad idea to abandon the procedure and refer the patient to a more experienced surgeon. In case of difficult to handle acute cholecystitis (edematous inflamed GB), the operation can be abandoned, postoperative US guided percutaneous transhepatic cholecystostomy (Fig. 5.15) performed and an attempt at cholecystectomy can be made again after 4–6 weeks when inflammation will have settled and the operation may become easier.

5.20 Drain

Most surgeons place a subhepatic drain selectively, after a difficult cholecystectomy only; this will ensure an early diagnosis and timely man-

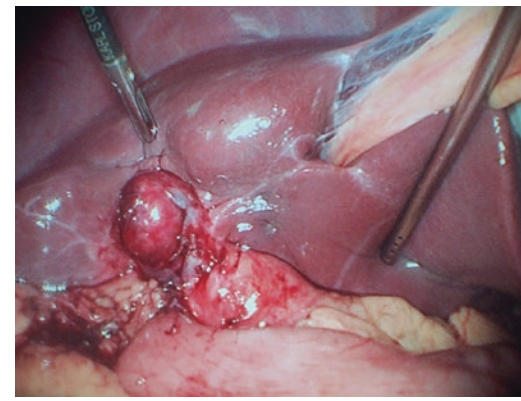

Fig. 5.14 Small fibrotic contracted thimble gallbladder with no Calot's triangle

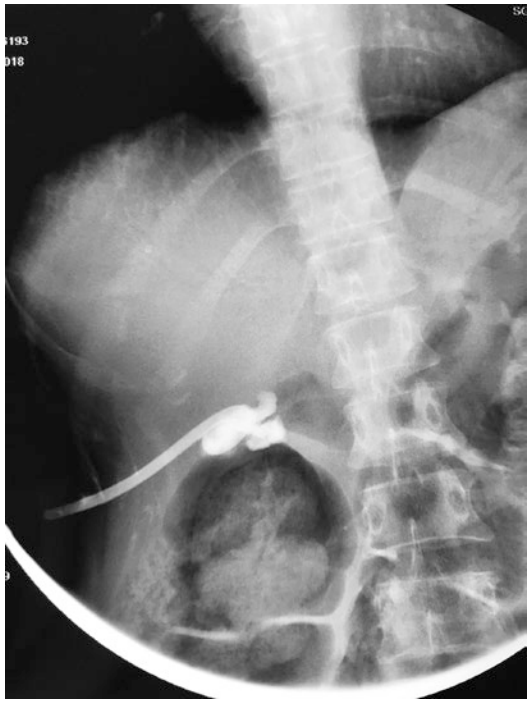

Fig. 5.15 Contrast study performed through a percutaneous cholecystostomy

agement of a bile leak (or bleed). In patients in whom a drain is not placed, a bile leak may not be easy to detect and is suspected only when bile produces symptoms and signs of peritoneal inflammation and sepsis (See Chap. 10). Routine use of a drain after cholecystectomy may result in early postoperative detection of BDI but is not recommended.

5.21 Discharge

A patient who does not look well, has undue pain, is not tolerating orals, has unstable vitals or an unsettled abdomen must not be discharged from the hospital; she needs to be observed and/or investigated.

5.22 CBD Stones

If small stones are detected on IOC/POC in a normal (undilated) CBD, they should probably be left behind for an early postoperative endoscopic removal; surgical attempts to remove multiple stones from such a bile duct may itself cause a BDI.

During (CBD) exploration, CBD should not be mobilized circumferentially as this may strip it of its axial blood supply and the resultant ischemia may cause a delayed (after months or even years) stricture. Only the anterior surface of the CBD should be exposed and choledochotomy made.

If at all, the CBD has to be calibrated or dilated, a soft catheter, e.g., an infant feeding tube or a balloon (Fogarty or Foley) catheter should be used instead of metal instruments, e.g., bougie or Bake's dilator.

5.23 Review

If the operative procedure was (video) recorded, the recording must be reviewed in EVERY case a BDI is suspected or diagnosed—this will not only help identify the type, site and extent of BDI but will also provide tips to prevent a similar BDI in future.

All attempts must be made by the surgeon to make the cholecystectomy safe and prevent a bile duct injury (and other complications of cholecystectomy).

Invited Commentary on Prevention of Bile Duct Injury During Cholecystectomy

L. Michael Brunt

Cholecystectomy is the most common operation performed worldwide by general surgeons. In the early 1990s, laparoscopic cholecystectomy quickly became the gold standard approach to patients with symptomatic gall stone disease. The advantages of this procedure were readily apparent including less pain, shorter hospital stay which is now outpatient in many centers, and overall fewer complications and faster return to full activity. However, despite being first performed almost 30 years ago, serious complications still occur during this procedure, most notably bile duct injury.

In this chapter, Dr. Kapoor has enumerated many of the important principles around safe cholecystectomy. These include recognition of less commonly appreciated anatomic landmarks such as Rouviere's sulcus, the importance of the critical view of safety (CVS) method of ductal identification, and recognition of difficult gallbladder scenarios. Other aspects of safety which are not often discussed that are worth highlighting are the importance of proper selection of cases early in one's career to avoid the most difficult cases and to get help from a more experienced surgeon when possible and use of an angled laparoscope for optimal visualization and flexibility of viewing angles. It is also crucial that any patient who deviates from the expected normal benign postoperative recovery phase should be investigated immediately for possible complications to avoid a missed injury or retained bile duct stone.

In 2014, SAGES launched the Safe Cholecystectomy program with the mission of enhancing a universal culture of safety around cholecystectomy [33]. Using a Delphi consensus method [34], six steps were identified that surgeons can employ to reduce the risk of biliary injury: (1) understand and use the critical view of safety (CVS) in every case (hepatocystic triangle clear of all extraneous tissue, only two structures entering the gallbladder, and the lower 1/3 of the gallbladder separated from the cystic plate of the liver); (2) understand aberrant biliary anatomy; (3) make liberal use of intraoperative cholangiography (IOC) and other means of imaging the biliary tree; (4) perform an intraoperative time-out before clipping and cutting any ductal structures; (5) recognize difficult or dangerous scenarios and alter the approach if the CVS cannot be obtained; and (6) get help in difficult cases whenever feasible.

The recommendations in this chapter are in strong alignment with the principles espoused by SAGES and which are presented in detail in a series of web based didactic modules that are available on the SAGES web site. In particular, for difficult cases of severe acute cholecystitis and Mirizzi's syndrome, it should be emphasized that laparoscopic cholecystectomy is an advanced laparoscopic procedure and surgeons who undertake these cases should be familiar with the alternative bail out options if the CVS cannot be achieved or the dissection is stalled. Intraoperative imaging with cholangiography may be especially important in such cases if available to delineate biliary anatomy and reduce the risk of biliary injury as nicely reviewed by Dr. Kapoor.

As a part of an enhanced culture of safety in cholecystectomy, a number of strategies which are discussed in this chapter should be in one's armamentarium if conditions at operation are unfavorable or dangerous and one cannot achieve the CVS. These include aborting the procedure and placing a cholecystostomy tube and referring the patient to a specialty center, rather than persisting with the dissection and creating a biliary or other injury. Subtotal cholecystectomy, either laparoscopic or open, is also increasingly viewed as a safe alternative in such circumstances. It is important with this approach to remove all stones from the gallbladder and leave a drain because of the risk of bile leakage. At our institution, a fenestrating subtotal resection is preferred over the reconstituting approach, because the latter may increase the risk of recurrent gallstones and symptoms. Recently, a retrospective series was reported of 191 patients who underwent either fenestrating or reconstituting cholecystectomy in Dutch centers [35]. Bile leaks were more common postoperatively in the fenestrating group (18% vs 7%) but recurrent biliary colic was more common in the reconstituting group (18% vs 9%). Finally, the role of conversion to open operation as the reflexive default mode for difficult cases is also being reconsidered for two reasons. First, as noted by Dr. Kapoor, this does not guarantee against potential injury. Secondly, in the US at least, most graduates of surgical training programs have relative little experience with difficult open cholecystectomy, and, therefore, a bail out laparoscopic approach may be safer. The exception, of course, would be bleeding which cannot be controlled laparoscopically in which conversion should not be delayed.

Despite being 30 years into the laparoscopic era, it is clear that much work is yet to be done to

enhance safety of this common operation for our patients. Dr. Kapoor's book is an important contribution to this effort to increase awareness of this problem and will undoubtedly have an impact going forward in educating the surgical community throughout the world for the benefit of our patients.

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6 Pathophysiology of Bile Leak, Bile Loss, and Biliary Obstruction

Vinay K. Kapoor

> Injuries to the bile ducts are unfortunately not rare and often turn out to be tragedies.
>
> Grey-Turner [1]

Bile duct injury (BDI) has been variously described as a catastrophic, dangerous, daunting, devastating, dreaded, lethal, major, most feared, most worrisome, overwhelming, potentially life threatening and serious complication, and even scourge, of a so-called simple, otherwise safe operation viz. cholecystectomy. The pathophysiological effects of a BDI are dependent on the type of the injury (complete or partial), biliary ductal continuity (absent or present), bile leak (present or absent), duration of biliary stricture, and cholangitis (present or absent).

BDI is a complication—definitely for the patient (who may ultimately become a "biliary cripple") and, these days, for the surgeon as well (who may face a medico-legal suit). It is an emotionally and psychologically traumatic experience not only for the patient but for the surgeon too who gets depressed and demoralized because of this disaster. It ends up as a financial and healthcare burden, both for the patient and the society.

6.1 Mortality

Mortality following open cholecystectomy was low (around 0.2%)—mainly in the elderly (>65 years) in whom it was 0.5% vs. young (<65 years) in whom it was <0.05%. Most deaths after open cholecystectomy were unrelated to the cholecystectomy per se and occurred in elderly patients with comorbid medical conditions. After the introduction of laparoscopic cholecystectomy, even young otherwise healthy patients are dying because of iatrogenic bile duct (and vascular and bowel) injuries.

A BDI can cause early death due to bile leak and its complication of bile peritonitis. Biliovascular injuries can cause ischemic necrosis of liver parenchyma which can result in acute fulminant liver failure or can get infected to form an abscess. BDIs are associated with significant (in the range of 4–8%) mortality, much higher than the mortality following most of the major surgical procedures, e.g., esophagectomy, gastrectomy, pancreato-duodenectomy, liver resection, etc. Gouma and Go [2] reported a mortality of 7.8% in 77 patients with BDI. Three out of 200 patients referred to the Johns Hopkins Hospital, Baltimore MD USA for repair of a BDI died of uncontrolled sepsis and multiple organ failure even before any attempt at repair [3]. In a French review, six out of

Also see Invited Commentary on Patho-physiology of Bile Leak, Bile Loss and Biliary Obstruction by John Windsor (pp 69–70)

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640 patients with BDI died before any attempt at repair and four died after repair [4]. Mishra [5] reported 137 patients with BDI—three died at 6, 15, and 24 days; three more died after repair. In one of the largest single institution experiences with 800 BDIs managed between 1992 and 2012, the Academic Medical Center (AMC), Amsterdam, Netherlands reported a BDI related mortality of 28/800 (3.5%) [6]. In a report from Iran, four (4%) of 104 patients who underwent repair of BDI died [7]. Even the so-called minor BDIs can cause death—nine BDI related deaths were reported in 133 BDI related litigation claims in the Netherlands between 1994 and 2006–4 of these patients had a so-called minor (Amsterdam type A, cystic duct leakage) injury but died of peritonitis [8]. Two hundred and sixteen out of 800 BDIs managed at the Academic Medical Centre, Amsterdam were Strasberg type A but mortality in these 216 "minor" BDIs was 9 (4.2%) [9].

BDI also carries long-term risk of death due to its sequelae and related complications such as external biliary fistula (EBF), benign biliary stricture (BBS), recurrent cholangitis, secondary biliary cirrhosis (SBC), portal hypertension and bleeding esophago-gastric varices, liver failure, etc. leading the patient to become a "biliary cripple." The life-time hazard ratio of death, either immediately or later, due to a BDI sustained during cholecystectomy is 2.8 (as compared to patients who had an uneventful cholecystectomy with no BDI [10]. One year mortality in 747 patients who sustained a BDI was 3.9% vs. 1.1% in those who underwent an uneventful cholecystectomy [11]. All-cause mortality in 125 patients with BDI in the New York State (2005–2010) was 20.8% with median time to death 1.6 ± 1.1 years—this was 8.8% more than the expected age adjusted rate of death during follow-up of 4–9 years [12]. El-Dhuwaib [13] reported 500 patients who required bile duct reconstruction for BDI after 572,223 laparoscopic cholecystectomies performed in England (2001–2013)—these patients were ten times (6.0% vs. 0.6%) more likely to die within 1 year as compared to those who had uneventful laparoscopic cholecystectomies and did not require further surgery. In a recent analysis of 3,551 (0.5%) bile leaks in 711,454 cholecystectomies performed in the California state of USA between 2005 and 2014, patients with bile leak were more likely to die at 1 year (2.4% vs. 1.4%); BDI occurred in 1,584 (0.22%) patients—these patients with BDI were even more likely to die at 1 year (7.2% vs. 1.3%) [14].

6.2 Bilo-Peritoneum

BDI during laparoscopic cholecystectomy is more likely to result in a bile leak (wet BDI) than during open cholecystectomy. Bile in the peritoneal cavity (bilo-peritoneum or cholo-peritoneum) following bile leak may present differently in different patients.

1. Bile leak is presence of bile outside the biliary tract, usually in the peritoneal cavity.
2. Biloma (Fig. 6.1) is a localized collection of bile in the peritoneal cavity with no generalized peritonitis; it may be sterile or infected. An infected biloma produces systemic signs of sepsis, e.g., tachycardia, fever, tachypnea, and localized abdominal signs, i.e., guarding and tenderness. An infected biloma may evolve into an intra-abdominal abscess (IAA). A biloma should, therefore, always be drained. This can usually be done by image (US or CT) guided percutaneous catheter drainage (PCD);

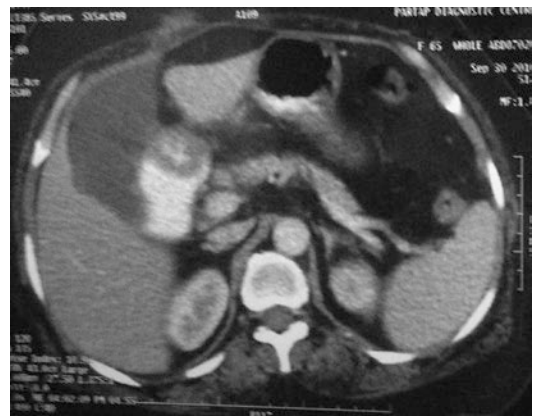

Fig. 6.1 Biloma is a localized collection of bile in the peritoneal cavity

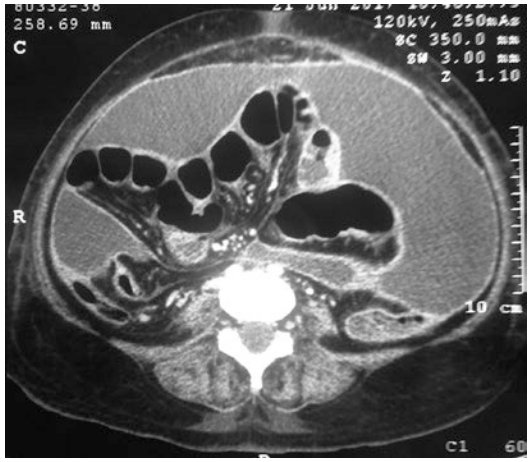

Fig. 6.2 Free bile in the peritoneal cavity; this may or may not be associated with clinical features of peritonitis

surgical intervention, i.e., relaparoscopy or relaparotomy, is rarely required for this.

3. Presence of free bile (Fig. 6.2) in the whole of the peritoneal cavity with clinical features of generalized peritonitis and sepsis is called bile peritonitis. Bile peritonitis is the most common cause of mortality following acute BDI.
4. Presence of free bile in the whole of the peritoneal cavity without clinical features of generalized peritonitis is called as bile ascites; patients with bile ascites have no sepsis.

The clinical presentation in 214 patients with acute BDI managed by us between 1989 and 2009 was as external biliary fistula (n = 99), biloma (n = 85), bile peritonitis (n = 19), and bile ascites (n = 11) [15].

If a sectoral/segmental bile duct is divided without clipping (Strasberg type C injury), it will result in bile leak. Bile leak with an intact common bile duct on cholangiogram or isotope hepato-biliary scan should suggest the diagnosis of an isolated sectoral/segmental BDI. Treatment includes percutaneous catheter drainage (PCD) of the biloma; reconstruction of the divided sectoral/segmental duct may be required later.

Patients, who have had a precholecystectomy intervention, whether endoscopic, e.g., endoscopic retrograde cholangiography (ERC), endoscopic stent or endoscopic naso-biliary drain (ENBD) placement or percutaneous, e.g., percutaneous transhepatic cholangiography (PTC), percutaneous transhepatic biliary drainage (PTBD), are very likely to have infected bile (bactobilia) causing sepsis (peritoneal and systemic) in case bile leak occurs.

Bile leak, e.g., from the cystic duct or a subvesical duct and collection of bile around the hepato-duodenal ligament may cause intense inflammatory reaction and severe fibrosis around the common bile duct resulting in a benign biliary stricture (BBS) later, even in the absence of an injury to the common bile duct.

6.3 Sepsis

Patients with BDI can have sepsis in the peritoneal cavity (due to bile leak), biliary system (cholangitis), and/or in the liver (cholangiolytic abscess).

Lee [16] reported 154 patients with bile collection—21% of them had sepsis and multi-organ failure. Bile in the peritoneal cavity behaves very erratically—while some patients can have liters of bile in the peritoneal cavity with no features of sepsis (bile ascites), others with even a small bile leak (caused by a so-called minor BDI) can result in systemic inflammatory response syndrome (SIRS), uncontrolled severe overwhelming sepsis, and multiple organ dysfunction syndrome (MODS) and can die.

> One of our patients who died after laparoscopic cholecystectomy developed MODS after a minimal bile leak.

Systemic sepsis as a result of bile leak may cause disseminated intravascular coagulation (DIC) resulting in generalized bleeding. Low platelet count, low fibrinogen levels, and increased levels of fibrin degradation products (FDP) are diagnostic. Coagulation profile should be checked and any coagulopathy corrected before any intervention (whether non-surgical viz. endoscopic or percutaneous, or surgical) is planned.

Patients with BDI, especially those with bile leak and sepsis, may require admission into the high dependency unit (HDU) or the intensive

care unit (ICU) because of multiple organ dysfunction syndrome (MODS) requiring close monitoring and organ support. First priority in the management of a patient with acute BDI is to control sepsis. This can be accomplished non-surgically in majority of the cases. The patient requires hospitalization and administration of parenteral antibiotics. Uncontrolled sepsis not responding to parenteral antibiotics may require biliary ductal drainage (endoscopic or percutaneous). Repair of a BDI should not be performed in presence of sepsis; sepsis should be controlled before any repair is attempted.

Majority of patients with biliary obstruction have infected bile and grow bacteria on culture even when clinical cholangitis is not present. Any intervention (endoscopic, percutaneous, or surgical) on an obstructed and infected biliary system can cause bacteremia and, therefore, should be done under adequate antibiotic cover. Most biliary tract infections (cholangitis) are caused by Gram negative bacilli of enteric origin, e.g., *Escherichia coli*, *Klebsiella*, *Proteus*, *Pseudomonas*, etc. Choice of antibiotics, therefore, should be guided accordingly.

6.4 Bile Loss

Bile loss from an external biliary fistula (EBF), even if the fistula is controlled, results in metabolic derangements; the severity of which depends on the completeness and duration of the biliary fistula. A complete EBF results in loss of about 750–1,000 mL of bile every day. Prolonged total (high volume) bile loss may cause chronic dehydration and electrolyte imbalance in the form of hyponatremia and hypokalemia, and hypochloremic metabolic acidosis. The patient may feel weak, tired, and lethargic. Decreased plasma volume (hypovolemia) may lead to low-output acute renal failure and hyperkalemia. Total biliary diversion in a complete EBF may result in disruption of the intestinal mucosal barrier function causing bacterial translocation, peritoneal sepsis, and endotoxemia. Absence of bile from the intestinal tract may result in malabsorption causing protein-calorie malnutrition (PCM) and even weight loss. These patients may be in a catabolic phase because of the combined effect of surgery and sepsis and would require nutritional support before they are operated. They should be given proper advice regarding nutrition—adequate calorie and protein intake with vitamins so that they return to an anabolic phase. Low serum albumin level is associated with poor outcome in patients who undergo repair of a biliary stricture [17]. The ill effects of prolonged bile loss in patients with long-standing high output EBF may be reduced by refeeding the bile [18] but only if it is clear (not muddy) and sterile (on culture). Bile refeed should be encouraged; bile, however, is very sour and bitter in taste and is highly unpalatable; small amount of bile may be taken mixed with food or a sweet syrup, e.g., honey or a fizzy drink. Many patients, however, do not like the idea and are reluctant and hesitant to take bile because of aesthetic reasons. Bile refeeding, even in small amounts, may also restore the intestinal mucosal barrier function [18].

6.5 Vitamin K

Patients with prolonged bile loss due to a persistent EBF and those with surgical obstructive jaundice due to a benign biliary stricture may have deficiency of fat-soluble vitamins, i.e., K, A, D, and E due to absence of bile in the intestines causing fat malabsorption The coagulopathy as a result of vitamin K deficiency manifests as deranged coagulation profile. Complete coagulation profile (i.e., bleeding time, BT, clotting time, CT, prothrombin time, PT, international normalized ratio, INR, and activated partial thromboplastin time, aPTT) should be obtained in all patients with benign biliary stricture and jaundice. Percutaneous interventions, e.g., percutaneous transhepatic cholangiography (PTC), percutaneous transhepatic biliary catheterization (PTBC), percutaneous transhepatic biliary drainage (PTBD), and surgery are contraindicated in presence of uncorrectable coagulopathy. All patients with benign biliary stricture who are

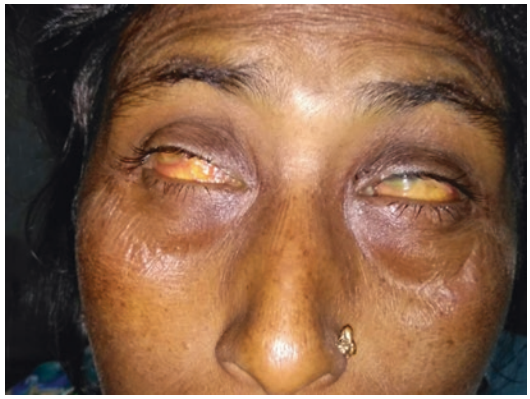

Fig. 6.3 Night blindness due to vitamin A deficiency in a patient with prolonged external biliary fistula

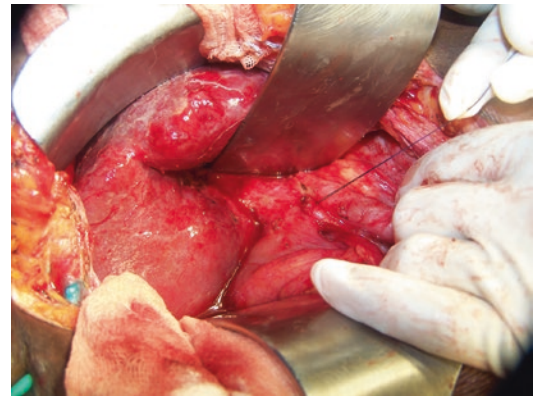

Fig. 6.4 Internal fistula between the proximal bile duct and duodenum

scheduled to undergo operation or even a non-surgical intervention, e.g., endoscopic papillotomy and stenting, PTC, PTBC, PTBD, etc. should receive 3–5 days of vitamin K 10 mg IM daily.

ANECDOTE: *The author remembers at least three patients with bile duct injury (BDI)—external biliary fistula (EBF)—benign biliary stricture (BBS) who had night blindness/complete blindness* (Fig. 6.3) *at the time of their presentation to him.*

ANECDOTE: *One of the deaths in our experience of repairs for benign biliary stricture occurred in a patient with a long-standing external biliary fistula (EBF) who had developed night blindness due to deficiency of vitamin A, which we unfortunately ignored and went ahead with surgical repair—she had persistent uncontrollable diffuse ooze in the intraoperative and postoperative period due to coagulopathy as a result of vitamin K deficiency and died.*

Patients with long-standing biliary obstruction may have coagulopathy due to poor synthetic function of the liver because of secondary biliary cirrhosis (SBC). This coagulopathy will not respond to vitamin K administration and will need correction with fresh frozen plasma (FFP). Fresh frozen plasma should be arranged before any intervention (endoscopic, percutaneous, or surgical) is performed in a patient with benign biliary stricture with surgical obstructive jaundice and coagulopathy even if vitamin K has been administered.

6.6 Internal Fistula

An internal (bilio-enteric) fistula between the bile duct proximal to the benign biliary stricture and the duodenum (Fig. 6.4) may be responsible for the patient being anicteric in spite of a complete transection of the common bile duct as demonstrated on ERC. Patients with BBS and a bilio-enteric fistula may not have jaundice but will have some evidence of biliary obstruction in the form of raised alkaline phosphatase (ALP) or gamma-glutamyl transpeptidase (GGTP), intrahepatic biliary radical dilatation (IHBRD) on ultrasonography (US), and delayed excretion of isotope on hepato-biliary scintigraphy; they may also have recurrent cholangitis which may lead to SBC. The internal fistula does not allow proximal biliary ductal dilatation to occur thus making surgical repair of the BBS technically difficult.

A suprahepatic subphrenic biloma may erode through the diaphragm into the pleural cavity to form a bilio-pleural fistula and then into the lung and a peripheral bronchus to form a bilio-bronchial fistula—patient coughing out bile-stained sputum. Treatment includes intercostal pleural drainage, drainage of the subphrenic biloma, and biliary decompression by endoscopic stenting or PTBD; definitive repair of the BDI in the form of hepatico-jejunostomy can be performed later.

An intrahepatic hematoma, e.g., following a percutaneous transhepatic intervention as a result of a vascular injury or parenchymal necrosis can result in a communication between an intrahepatic

bile duct and a hepatic venous tributary resulting in a bilio-venous fistula with regurgitation of the infected bile into the systemic venous system (bilhemia) causing deep jaundice and severe sepsis.

6.7 Pruritus

Patients with BBS have pruritus due to biliary obstruction. Pruritus due to extrahepatic biliary obstruction does not respond to oral administration of bile acids and bile salts (cf. pruritus due to intrahepatic cholestasis, e.g., in hepatitis, which does). The pruritus can sometimes be severe enough to interfere with sleep and quality of life. Scratch marks of severe pruritus may get infected with Gram positive organisms. Pruritus gets relieved only after the biliary obstruction is taken care of either by endoscopic stenting, percutaneous transhepatic biliary drainage (PTBD), or by hepatico-jejunostomy.

6.8 Cholangitis

Patients with BDI, EBF, or BBS may have recurrent attacks of cholangitis secondary to biliary obstruction. Total leukocyte counts (TLC) and differential leukocyte counts (DLC) must be obtained in all patients with BDI and BBS (even if there is no fever) to diagnose subclinical cholangitis. Cholangitis, if present, needs treatment with appropriate broad spectrum antibiotics; uncontrolled cholangitis (not responding to parenteral antibiotics in 24–48 h) may require biliary drainage—this may be endoscopic (if biliary ductal continuity is present), viz. stent or ENBD, or percutaneous, viz. PTBD. Surgical biliary drainage is difficult but is seldom required.

6.9 Cholangiolytic Abscess

Cholangitis as a result of a BDI/BBS can result in the formation of cholangiolytic liver abscesses (Fig. 6.5), which are usually multiple and small; high grade fever with chills and rigors is characteristic. The abscesses are picked up on ultrasonography (US), computed tomography (CT), or magnetic resonance imaging (MRI). Treatment is largely conservative with broad spectrum intravenous antibiotics. Rarely, a large abscess not responding to conservative management may require percutaneous catheter drainage (PCD).

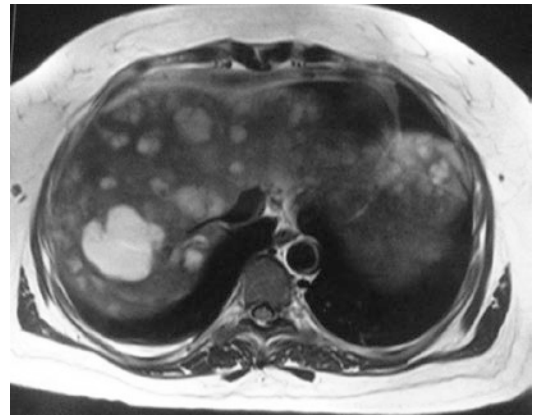

Fig. 6.5 Cholangiolytic abscesses - one large and multiple small

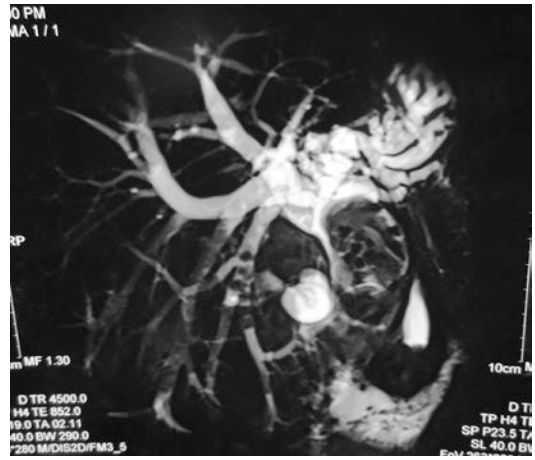

Fig. 6.6 Hepatolithiasis

6.10 Hepatolithiasis

Calculi and sludge may form in the intrahepatic ducts proximal to a BBS and may cause recurrent cholangitis. These intrahepatic ductal calculi can be seen on ultrasonography (US), computed tomography (CT), or magnetic resonance cholangiography (MRC) (Fig. 6.6). Many Japanese surgeons frequently use percutaneous transhepatic

cholangioscopy (PTCS) to remove these intrahepatic calculi and sludge. At the time of repair of a BBS, the intrahepatic ducts should be thoroughly irrigated with copious amounts of warm saline to remove these stones. Intraoperative retrograde cholangioscopy can also be done to locate and remove these stones.

6.11 Isolated Duct Injury

An aberrant subsegmental, segmental (usually segment V), sectoral (usually right posterior), or hepatic (right main) duct in the Calot's triangle may get injured or may be unintentionally ligated/clipped (Strasberg type B injury) and form a stricture. A strictured isolated duct may lead to asymptomatic atrophy of a part of the liver which is detected on US (segmental dilatation), CT, isotope hepato-biliary scan, or MRC; it does not require any intervention. Atrophy may lead to recurrent cholangitis and cholangiolytic abscess necessitating intervention.

6.12 Atrophy

Portal venous blood flow to the liver provides hepatotrophic factors and regulates hepatocyte mass. The commonest cause of liver atrophy is a vascular (portal venous) injury; hepatic artery injury alone does not cause atrophy. The duration after which atrophy occurs is a matter of debate—36 (10%) of 362 patients with biliary stricture operated by us between 1989 and 2005 had atrophy; the median interval after cholecystectomy was 7 months (range 7 weeks to 16 years) [19]. A segment, sector, or lobe of the liver may undergo atrophy as a long-term sequel of biliary obstruction more so if it is associated with recurrent attacks of cholangitis due to BBS. Segmental atrophy may be asymptomatic and is usually a result of occlusion following injury of an uninfected aberrant subsegmental or segmental (commonly segment V) or sectoral (commonly posterior) duct in the Calot's triangle (an infected occluded duct, on the other hand, causes cholangitis). Lobar (commonly on the right side) atrophy is a result of an associated vascular (portal vein or hepatic artery) injury and is associated with compensatory hypertrophy of the left lobe—atrophy–hypertrophy complex (AHC) (Fig. 6.7). On imaging (US, CT, MRI, or isotope hepato-biliary scintigraphy) an atrophic lobe is smaller in size, hypoperfused and the bile ducts are irregular, dilated, and crowded. Atrophy is more commonly associated with high (Bismuth type III or IV) BBS. Twenty six out of 36 patients with atrophy–hypertrophy complex had high (Bismuth type III 20 and type IV 6) BBS [19]. An atrophic lobe/sector/segment is more likely to have recurrent cholangitis.

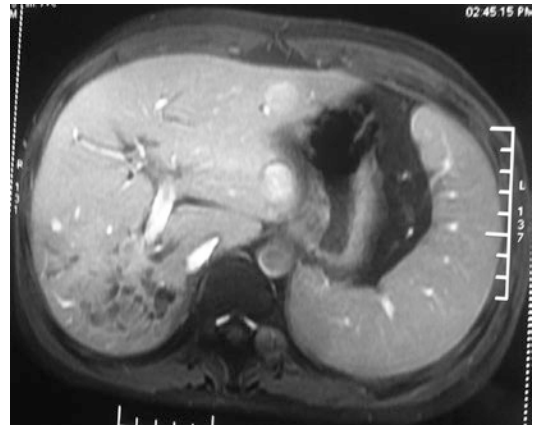

Fig. 6.7 Atrophy–hypertrophy complex (AHC) - atrophy of right lobe and hypertrophy of left lobe (especially segment IV)

Atrophy–hypertrophy complex (AHC) results in rotation of liver with the hepatic hilum and the hepato-duodenal ligament as the axis of rotation. This rotation distorts the normal anatomical relations of the structures in the hepatic hilum and the hepato-duodenal ligament. In the more common right atrophy–left hypertrophy complex, the liver rotates anticlockwise so that the common bile duct (CBD) gets displaced posteriorly and the portal vein gets to lie anteriorly thus making identification and location of the duct difficult. The enlarged hypertrophied segment IV overhangs the hilum and makes access to the hepatic hilum difficult. Preoperative placement of percutaneous transhepatic biliary catheters (PTBC) makes peroperative identification of the hepatic ducts easier. 33 (91%) of 36 patients with atrophy–hypertro-

phy complex had right atrophy; only two had left atrophy and one had segment IV atrophy; in patients with atrophy–hypertrophy complex, operation time, blood loss, and requirement of blood transfusion were more than in patients without atrophy–hypertrophy complex [19].

6.13 Secondary Biliary Cirrhosis

Fibrotic changes are very common in patients with BBS—Negi [20] found grade I fibrosis in 47%, II in 34%, and III in 11% patients. We performed trucut liver biopsy at the time of operation in 71 patients undergoing repair of a BBS. The histological features, i.e., fibrosis, portal inflammation, ductular proliferation, and cholestasis were scored from 0 to 3 as in patients with biliary obstruction due to chronic pancreatitis; a score of 3 was considered to be cirrhosis [21]. All patients with BBS had some degree of fibrosis but severe fibrosis/SBC was seen in 13 (18%) out of 71 patients; these 13 patients had a longer injury to repair interval (270 vs. 90 weeks) [22]. The longer the injury—repair interval, the higher the chances of developing SBC. Patients with long-standing (usually >6 months) biliary obstruction and recurrent cholangitis due to untreated or inadequately treated BBS may develop SBC (Fig. 6.8) resulting in portal hypertension and leading on to end-stage liver disease (ESLD) and chronic liver failure and may require liver transplant. Mishra [5] reported SBC in 46 out of 137 patients with BBS.

While fibrotic changes in the liver which are present in a large majority of patients with biliary stricture may revert after a successful hepatico-jejunostomy, established cirrhosis may persist. Repeat liver biopsy was performed during follow-up in five patients—fibrotic changes regressed in two, remained static in two, and progressed in the remaining one patient [22].

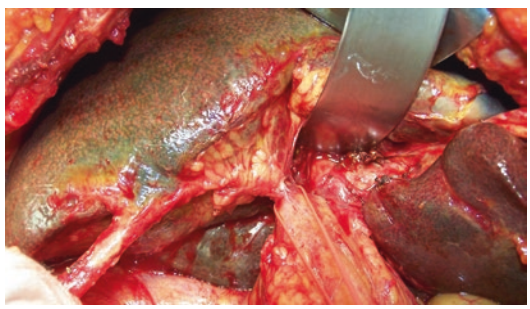

Fig. 6.8 Secondary biliary cirrhosis (SBC)

6.14 Portal Hypertension

Portal hypertension can occur in patients with BBS due to SBC as a result of long-standing biliary obstruction; it may develop as early as within 2 years of biliary obstruction. Mishra [5] reported that six out of 137 patients with BDI had portal hypertension. Six out of 13 patients with cirrhosis on liver biopsy had portal hypertension as opposed to only two out of 58 with no cirrhosis (but moderate fibrosis) [22]. We have reported "latent" portal hypertension, i.e., increased portal venous pressure without clinical, radiological, or endoscopic evidence of portal hypertension in patients with biliary obstruction; this gets reversed (corrected) immediately following surgical relief of biliary obstruction [23]. Nag [24] studied portal pressure in 30 patients with BBS and reported a relationship between portal pressure and injury—repair interval. Portal hypertension may also be caused by associated portal vein injury and resultant thrombosis. Patients with portal vein injury may develop portal vein thrombosis with porto-portal collaterals in the hepato-duodenal ligament. This is akin to portal biliopathy caused by enlarged peri and epicholedochal veins in the hepato-duodenal ligament in extrahepatic portal venous obstruction (EHPVO). Access to the bile ducts in the hepatic hilum is virtually impossible in these patients (due to the risk of excessive bleed from high pressure thin-walled collaterals)—these patients should first undergo a total porta-systemic shunt, which decompresses the collaterals, followed by hepatico-jejunostomy about 3–6 months later. Patients with portal hypertension may present with upper gastro-intestinal bleed from the esophago-gastric varices. An upper gastro-intestinal endoscopy (UGIE) should be performed in all patients with long-standing (>6 months) biliary obstruction to detect the varices.

Presence of portal hypertension may make the repair of BBS difficult and may require a two-stage approach—porta-systemic shunt first followed by bilio-enteric anastomosis later. Some patients with SBC develop end-stage liver disease (ESLD) and liver failure and may even need liver transplant (See Chap. 14).

BDI invariably produces a bile leak which can have serious consequences, i.e., peritonitis and systemic sepsis in the short term. Biliary obstruction due to BBS can cause irreversible changes in the form of SBC, portal hypertension, and liver failure in the long term.

Invited Commentary on Pathophysiology of Bile Leak, Bile Loss and Biliary Obstruction

John A. Windsor

As a life-time student of bile duct injuries and their consequences, Professor Kapoor has written a series of chapters that provide a comprehensive and rigorous text on all the key aspects. This particular chapter on the pathophysiological consequences of bile duct injury is no exception, containing vital information for those managing this iatrogenic and preventable tragedy. All biliary injuries are not the same and the pathophysiological consequences are protean. At one extreme it may be self-limiting problem that requires no more than the retention of a drain for a longer period of time. But it can also represent an abdominal catastrophe, with biliary peritonitis, septic shock, liver failure, and death. The situation is worse when concomitant vascular or duodenal injuries also happen but are overlooked. In the turmoil that is the immediate aftermath of a biliary injury, the surgeon responsible is not always in the best state of mind to make the best decisions. Involving a specialist hepato-biliary surgeon, if only by telephone, is strongly recommended. And there should be a low threshold for early patient transfer to allow early definitive repair, and reduce the risk of the serious systemic consequences.

The chapter rightly starts with the mortality risk of bile duct injury, something that may get glossed over in the patient consenting process for such a common and (usually) safe operation as cholecystectomy. Death from bile duct injury can occur in the short term or long term and for different reasons. The population study by Flum et al. [10] showed that the life-time risk of death from a cholecystectomy with bile duct injury is almost 3× greater than that for an uncomplicated cholecystectomy. An English population study was even more sobering with the risk of death increased 10× in the first year after bile duct injury [25]. These data stress the importance of bile duct injury and the need for a sound approach to prevention which is discussed in other chapters.

Distinguishing biloma from biliary ascites and biliary peritonitis helps direct treatment strategies. Drainage is the mainstay of local control, and patients may require a combination of percutaneous and endoscopic drains to achieve this. Generalized biliary peritonitis will require a laparotomy. When associated with progressive systemic inflammation and evolving multiple organ dysfunction, treatment must cover both the local (e.g., drainage and lavage) and the systemic dimensions (e.g., antibiotics, fluids, and organ support). Damage control surgery and delaying definitive repair of a bile duct injury might be necessary because of the instability of a patient with biliary peritonitis and multiple organ dysfunction.

The metabolic consequences of sustained high-volume bile loss are rightly emphasized and the importance of meticulous fluid replacement is outlined. The concept of bile re-refeeding, preferably through a naso-enteric tube, probably requires more study and wider implementation. The nutritional consequence of biliary "sepsis" makes maintaining or repleting a patient's protein and fat stores difficult, and early control of the sepsis will help prevent malnutrition.

The long term and serious complications of bile duct injury including hepatic atrophy from portal venous or hepatic arterial injury, secondary biliary cirrhosis from chronic biliary obstruction, and portal hypertension are all discussed. The factors that help determine the management

of these late complications also include the type of biliary injury, the presence of intrahepatic strictures or stones, repetitive cholangitis (including cholangitic abscess), and the quality of underlying liver parenchyma. Rarely, liver resection may be required for an extended hilar stricture, multiple stone retention in one sector of the liver, or when the repair is considered technically difficult. Exceptionally, liver transplantation is required when secondary biliary cirrhosis is associated with liver failure and portal hypertension.

This chapter is a sobering reminder of potentially devastating pathophysiological consequences of bile duct injury and provides sound basis for its multi-faceted management.

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7 Non-biliary Injuries During Cholecystectomy

Vinay K. Kapoor

Bile duct injury (BDI) is the commonest iatrogenic injury during laparoscopic cholecystectomy but, less commonly, other (non-biliary) injuries can also occur.

7.1 Trocar Injuries

Creation of pneumoperitoneum with Veress needle and blind insertion of the first trocar may cause injuries to the omentum, bowel, urinary bladder, and great vessels (aorta, inferior vena cava, or iliac vessels). As a routine, the patients should be directed to void and empty their bladder just before they are wheeled into the operation room. Major vessel injuries are more likely to occur in thin built patients in whom the anterior to posterior depth of the abdomen is small. To avoid these injuries, the direction of the Veress needle/umbilical trocar should not be vertical; it should be directed towards the sacral promontory at an angle of 45° to the abdominal wall. (The Author (VKK) does not use Veress needle and uses the open technique for the first trocar insertion as a routine). The first step immediately after insertion of the telescope should be to look for any of these (bowel or great vessel) injuries. Great vessel injury is an indication for immediate conversion to open operation. Bleed from the omentum or the small bowel and a urinary bladder injury may be controlled/repaired laparoscopically by a surgeon with laparoscopic suturing skills. The aforementioned other (non-biliary) injuries should also be kept in mind in addition to BDI in an unsettled patient/abdomen in the postoperative period after laparoscopic cholecystectomy.

Primary access-related complications were reported in 63 (0.4%) out of 15,260 cases in a retrospective study which included major (vascular and visceral) injuries in 11 cases [1]. A meta-analysis revealed 336 (0.044%) major vascular injuries in 760,890 closed laparoscopies vs. no major vascular injury in 22,465 open laparoscopies; visceral injuries were also more common (515 in 760,890) with closed laparoscopies vs. open laparoscopies (11 in 22,465) [2]. Guloglu [3] reported nine major vascular injuries (aorta 3, inferior vena cava 1, and iliac vessels 5) between 1994 and 2002; fortunately, all could be salvaged and no patient died.

Also see Invited Commentary on Non-biliary Injuries During Cholecystectomy by Manuela Cesaretti and Antonio Iannelli (pp 80–81)

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7.2 Vascular Injuries

In an autopsy study, hepatic arterial injuries were found in as many as 7% of patients after open cholecystectomy [4]. Chapman [5] reported associated hepatic arterial injury in 14% of 130 BDIs during open cholecystectomy. BDI during laparoscopic cholecystectomy is more often (10–50% in various reports; Table 7.1) associated with a vascular injury (bilio-vascular injury BVI) because laparoscopic cholecystectomy related BDIs are more proximal (higher) where the right hepatic artery (RHA) is in relation to the common bile duct (Fig. 7.1). The exact incidence of associated vascular injuries is not known because they are not proactively looked for in most cases, i.e., vascular evaluation is not performed before the repair of the BDI; also, it is not easy to detect a vascular injury at operation because of fibrosis present in the hepatoduodenal ligament and at the hepatic hilum (Fig. 7.2). The reported rates of vascular injury in laparoscopic cholecystectomy are usually underestimates of their true incidence [14].

While proper hepatic artery or portal vein may also get injured during cholecystectomy, it is the RHA that is more commonly injured. RHA lies close to the Calot's triangle when it crosses the common bile duct from left to right and may be mistaken for the cystic artery. In the classical laparoscopic BDI, the common bile duct is mistaken for the cystic duct; at the same time, the RHA is mistaken for the cystic artery and is clipped and divided. A tortuous RHA may even come to lie in front of the Calot's triangle or even on the gallbladder neck (Moynihan caterpillar hump) and is liable to get injured during cholecystectomy. An aberrant (accessory or replaced) RHA arising from the superior mesenteric artery, present in about

Table 7.1 Incidence of associated vascular injury in patients with bile duct injury

Author (Ref.)	No. (%) of vascular injuries and BDI	Type of vascular injuries
Deziel et al. [6]	44 (12%) of 365	Hepatic artery
Chapman et al. [5]	18 (14%) of 130	Open cholecystectomy
Wudel Jr et al. [7]	9 (12%) of 74	Hepatic artery
Alves et al. [8]	26 (47%) of 55	Vascular injury
Schmidt et al. [9]	11 (20%) of 54	Right hepatic artery
Stewart [10]	84 (32%) 261	Hepatic artery
Bektas et al. [11]	14 (19%) of 74	Vascular lesions
Li et al. [12]	10 (17%) of 60	Hepatic artery
Sasmal et al. [1]	88 (29%) of 307	Right hepatic artery
Stilling et al. [13]	26 (19%) of 139	Hepatic artery (operative)
SGPGIMS (Prof Anu Behari)	22 (61%) of 36	Right hepatic artery (proactively looked for)

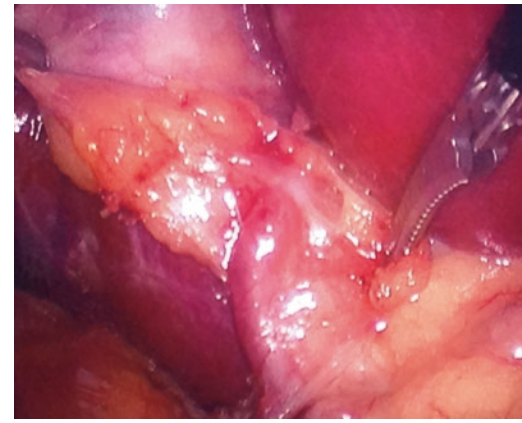

Fig. 7.1 Right hepatic artery, where it crosses on the anterior surface of the common bile duct is likely to be injured during laparoscopic cholecystectomy. Sometimes a Moynihan hump of the right hepatic artery lies in the Calot's triangle or even on the gallbladder neck, making it more liable to injury

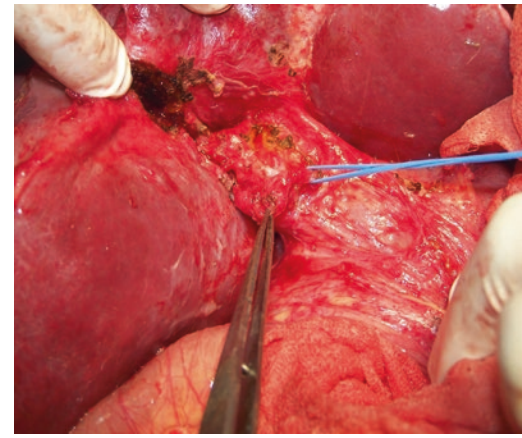

Fig. 7.2 It is difficult to detect the right hepatic artery in the fibrotic scar at the time of repair of BBS; rarely, a stump of the divided right hepatic artery (forceps) can be seen

one in six cases, may travel on the right posterior aspect of the common bile duct behind the cystic duct and may get injured during cholecystectomy.

Vascular injuries are more frequent (present in as many as one-fourth to one-third of cases) in patients with high (Bismuth Type III and IV) biliary strictures. Koffron [15] found associated vascular injury in 71% cases with Bismuth Type IV, 63% cases with Bismuth Type III, and 33% cases with Bismuth Type II benign biliary stricture. Buell [16] found associated vascular (arterial) injury in 26% of 27 Bismuth Type III, IV, or V benign biliary strictures. The incidence of RHA injury was more in higher (proximal) injuries (64% in class IV and 35% in class III) than in lower (distal) injuries (17% in class II and 6% in class I) [10]. Since BDIs during laparoscopic cholecystectomy are higher (proximal) as compared to those during open cholecystectomy, incidence of associated vascular injury is also more during laparoscopic cholecystectomy than during open cholecystectomy. Vascular injuries are more frequent in patients who have had attempts at repair; the artery, embedded in the fibrous scar tissue of the benign biliary stricture, may get injured during the dissection in the hepatic hilum.

Some reports have called bilio-vascular injuries as complex BDIs. Strasberg and Gouma [17] defined extreme vasculo-biliary injury as those involving the bile duct, artery (proper hepatic artery or right hepatic artery), and vein (main portal vein or right portal vein). They described eight such cases (out of a total 400 BDIs)—all following a fundus down (fundus first) approach in presence of severe acute or chronic contractive inflammation of the gallbladder. Four patients required hepatectomy and one required liver transplant; four patients died. The authors advocate subtotal cholecystectomy or even cholecystostomy in such cases.

7.3 Diagnosis

Vascular injury should be suspected if significant (excessive) bleed (causing hypotension and/or requiring blood transfusion) is reported to have occurred during the cholecystectomy, if liver enzymes (especially alanine transaminase ALT) are elevated in the early postoperative period, if several clips are seen in the Calot's triangle on imaging or at reoperation, if bile duct injury/benign biliary stricture is high (proximal), if atrophy of liver is seen on imaging or if a previous early repair has been attempted.

An associated arterial injury may not cause any significant clinical problems and may remain silent and go undetected unless looked for, because the predominant blood supply to the liver is portal venous. Associated vascular (especially portal vein) injury may cause devascularization of the liver resulting in ischemic necrosis or infarction in the acute phase and parenchymal atrophy later. The necrotic liver parenchyma may need surgical debridement or even formal liver resection. The necrotic liver parenchyma may get infected and form an abscess which may require drainage (usually image guided percutaneous). Patients who sustain a major combined bilio-vascular injury may develop acute liver failure; there are several anecdotal reports in the literature of such patients even requiring an urgent liver transplant (See Chap. 14).

Combination of biliary obstruction and diminished portal venous blood flow results in atrophy of liver parenchyma in the long term. In the most common situation of right portal vein injury, the right lobe of liver atrophies and the left lobe hypertrophies (atrophy–hypertrophy complex).

Associated portal vein injury at the time of the BDI may result in portal venous thrombosis and extrahepatic portal venous obstruction (EHPVO). The collaterals which then develop in the hepatoduodenal ligament may make surgical access to the hepatic hilum extremely difficult and even impossible due to torrential bleeding. Such patients may require a porta-systemic shunt (to decompress these collaterals) first followed by hepatico-jejunostomy after a few months.

7.4 Pseudoaneurysm

A partial injury (e.g., needle puncture, cautery burn) to an artery, especially the RHA, may lead to the formation of a pseudoaneurysm which

may then erode into the adjacent common bile duct to result in hemobilia presenting as upper gastro-intestinal bleed. The characteristic clinical presentation of hemobilia is with pain (biliary colic due to the blood clot obstructing the common bile duct), jaundice, and melena (Sandblom triad). Upper gastro-intestinal endoscopy (UGIE) may show blood coming out from the duodenal papilla. A pseudoaneurysm can also be suspected on Doppler ultrasonography (US); contrast enhanced computed tomography (CT) (Fig. 7.3), magnetic resonance angiography (MRA)or conventional angiography (Fig. 7.4) are diagnostic. Angioembolization is the treatment of choice for a pseudoaneurysm (Fig. 7.5). Various synthetic materials such as steel coil, gelfoam, cyanoacrylate glue, balloon, etc. are used for embolization of the pseudoaneurysm. Placement of a covered intravascular stent will protect against accidental occlusion of the proper hepatic artery. If the aneurysm is large in size, it may be treated with percutaneous injection of thrombin into the aneurysm [18].

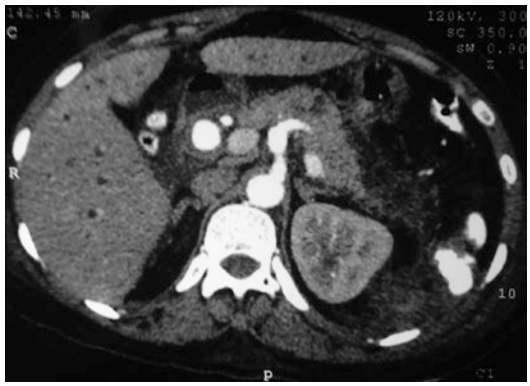

Fig. 7.3 Large pseudoaneurysm of the right hepatic artery seen on CT

Senthilkumar [19] reported symptomatic hepatic artery pseudoaneurysms in eight (3.4%) of 236 patients with BDI managed at the Queen Elizabeth Hospital, Birmingham, UK between 1992 and 2011. Seven had bleeding and one had only pain. Angioembolization was successful in seven patients. One patient developed infarction of liver. One patient died while seven out of eight patients were alive and well at median follow up of 66 months.

Hemobilia can occur after percutaneous transhepatic interventions (e.g., liver biopsy, percutaneous transhepatic cholangiography PTC, percutaneous transhepatic biliary drainage PTBD, etc.) also, which may be required at some stage during the management of patients with BDI or benign biliary stricture.

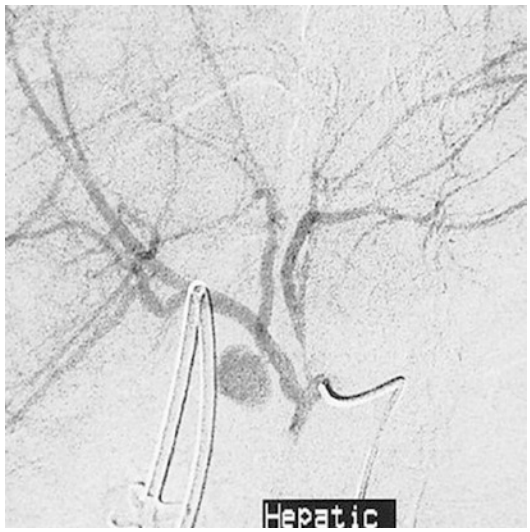

Fig. 7.4 Large pseudoaneurysm of the right hepatic artery seen on conventional angiography

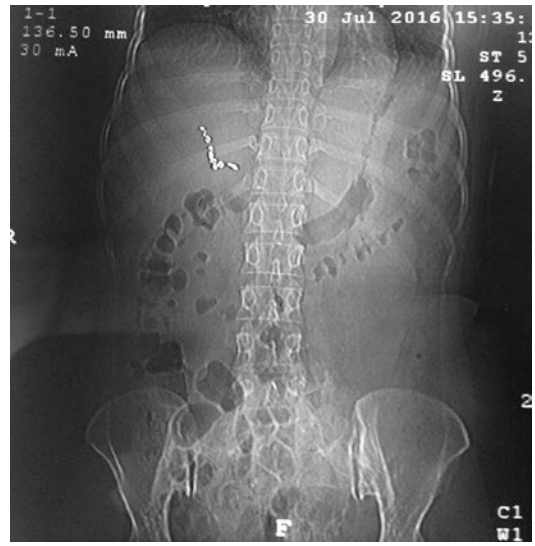

Fig. 7.5 Steel coils used for angioembolization of a pseudoaneurysm

7.5 Investigations

It is recommended that all BDIs should be investigated for an associated vascular injury.

Doppler US is a useful noninvasive investigation for evaluation of associated vascular (hepatic artery and portal vein) injury and for detection of a pseudoaneurysm in a patient with a BDI or benign biliary stricture. It can also show atrophy–hypertrophy complex, secondary biliary cirrhosis (SBC), collaterals of portal hypertension, and ascites, if present.

Contrast enhanced CT done for evaluation of a BDI (to detect a biloma) may reveal an unsuspected asymptomatic ischemic segment/sector/lobe of liver (Fig. 7.6).

Angiography is required to detect the associated vascular injury. This should be a noninvasive CT or MR angiography; conventional invasive angiography should be done only if a therapeutic intervention in the form of angioembolization is required for the pseudoaneurysm detected on Doppler US or CT/MR angiography. Both hepatic artery and portal venous phases must be evaluated on angiography. Both celiac axis and superior mesenteric artery should be evaluated as an aberrant RHA may be originating from the superior mesenteric artery instead of the celiac axis.

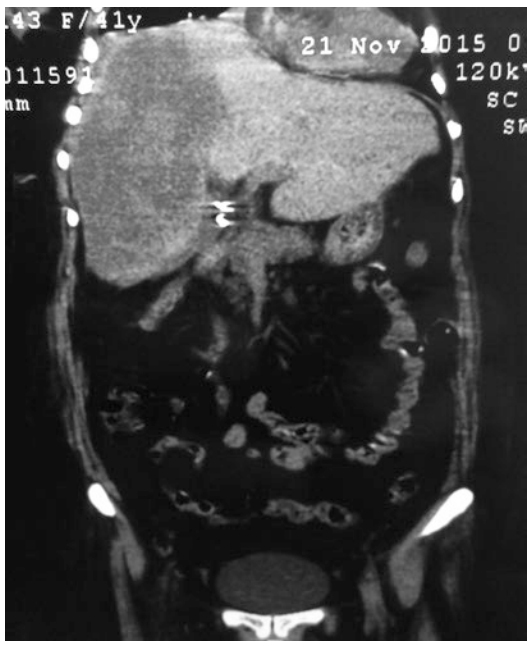

Fig. 7.6 Ischemia of right lobe of liver seen on CT

7.6 Classification

Bismuth's [20] classification for benign biliary stricture and Strasberg's [21] classification for acute BDI does not mention vascular injury. Siewert [22] was the first to propose a classification of acute BDI which mentioned additional vascular injury in Type II and IV; this classification, however, has not been followed. Stewart [10] later mentioned associated RHA injury when they classified BDIs as Class I-IV. Hannover classification [11] included detailed description of vascular lesions viz. d: right hepatic artery, s: left hepatic artery, p: proper hepatic artery, com: common hepatic artery, c: cystic artery, and pv: portal vein. Lau and Lai [23] also included associated vascular injuries as Type 5. The Author (VKK) has proposed new classifications for BDI [24] and benign biliary stricture [25] in which vascular injury was included.

7.7 Management

The arterial injury is usually in the form of transection while it may be just clipping or thrombosis (caused by thermal injury). An injured hepatic artery can be repaired if recognized during the cholecystectomy. This, however, should be attempted only if the assistance of an expert and experienced vascular surgeon can be immediately obtained. Li [12] described rearterialization by direct end-to-end anastomosis (for transection only, without loss of segment) or using autologous (vein) or synthetic vascular graft in five patients referred early (within 4 days). In case of loss of segment, arterial replacement, i.e., anastomosis of divided splenic artery or inferior mesenteric artery to the distal stump of the divided hepatic artery can also be performed. Immediate repair of BDI is contraindicated in presence of an associated vascular injury because of ischemia of the proximal bile ducts. The level of BDI ascends towards the hilum because of this ischemia and a

repair which may be adequate then may become inadequate later. Presence of an associated vascular injury is a contraindication for early repair also for the same reasons.

7.8 Outcome

Vascular injuries increase the mortality of the BDI. Mortality in patients with associated vascular injury was much higher (38%) than in those with no vascular injury (3%) [16]. In a report of 60 BDIs, concomitant hepatic arterial injury was found in as many as 10 (16%) cases—three of these ten patients died [12].

Detection of a vascular injury during angiography does not significantly change the management of an established benign biliary stricture; it, however, documents the preexisting vascular injury for medico-legal purposes (or else the surgeon performing the repair of the stricture may be accused of having caused the vascular injury) and may predict the outcome of the repair. Vascular injury, along with a high benign biliary stricture, is often an indication for hepatectomy. Threshold for hepatectomy for benign biliary stricture may also be low in presence of an associated vascular injury. Atrophy of liver (Fig. 7.7) due to an associated vascular injury may be an indication for hepatectomy in a patient with BDI. Whether vascular injury adversely affects the outcome of a delayed repair is controversial. Some reports [15] observed higher (61%) incidence of associated arterial injury in patients who developed restricture after repair of BDI while others [8, 10] reported that arterial injury did not influence the outcome of repair of the BDI. Sarno [26] reported worse outcome following repair in 18 patients with concomitant vascular injury than in 45 patients with BDI alone. Stilling [13] reported a trend (p = 0.07) towards poor outcome with vascular injury but a review did not find any association between hepatic arterial injury and stricture of hepatico-jejunostomy [27]. An associated vascular injury probably does not increase the risk of anastomotic stricture provided enough time is given for the stricture to mature and the repair is delayed (beyond 4–6 weeks). Also, repair should always be performed at the hepatic hilum, i.e., biliary ductal confluence extending to the left hepatic duct, i.e., hilo-jejunostomy [28].

7.9 Duodenal Injury

Duodenum may get injured during laparoscopic cholecystectomy, especially if it is adherent to the gallbladder (Fig. 7.8) or if there is a cholecysto-duodenal fistula. If recognized during the cholecystectomy, the injury should be repaired (laparoscopically, if suturing skills are available). A naso-jejunal tube may be placed across the duodenal repair for early postoperative enteral

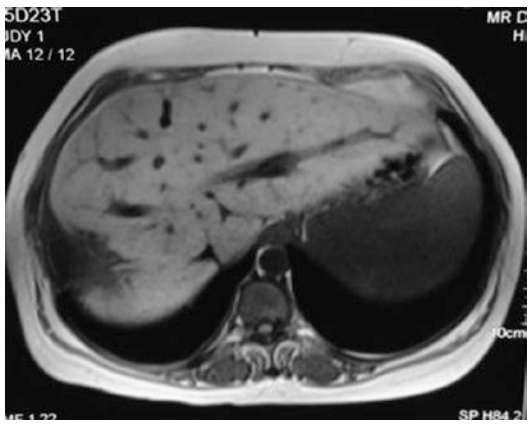

Fig. 7.7 Atrophy of right lobe of liver

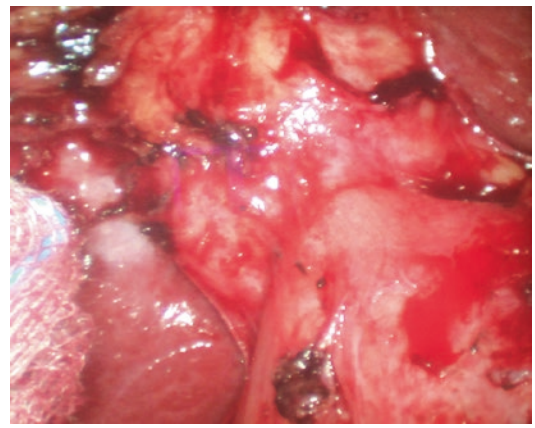

Fig. 7.8 Duodenum adherent to the gallbladder

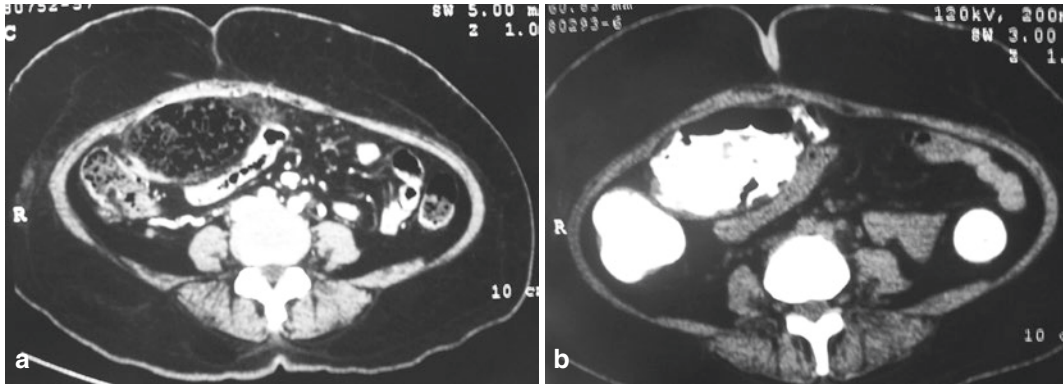

Fig. 7.9 (**a**) Intraabdominal abscess with air due to a missed colonic injury at laparoscopic cholecystectomy. (**b**) Rectal contrast leak into the abscess cavity

feeding; alternatively, a feeding jejunostomy may be performed. An inadvertent thermal injury (caused by electrocautery) to the duodenum may not be appreciated during the cholecystectomy itself—it then presents with a delayed duodenal perforation (due to necrosis) in the early postoperative period. Duodenal injury may then look like a BDI as both cause bile leak. If a duodenal injury is suspected, saline mixed with a color dye (e.g., methylene blue or gentian violet) should be administered orally; if a duodenal injury is present, the dye will appear in the drain. High amylase levels in the drain fluid also suggest a duodenal leak. If a drain is not present, computed tomography (CT) with an oral contrast will be required to detect/rule out duodenal injury.

ANECDOTE: *One of our patients who died after laparoscopic cholecystectomy had a duodenal injury, probably electrocautery related, which was missed during the cholecystectomy*.

ANECDOTE: *One of the patients referred to us with the diagnosis of an external biliary fistula (EBF) had a duodenal injury alone (no bile duct injury).*

7.10 Colon

Rarely, post-cholecystectomy peritonitis may be due to a missed colonic injury rather than a BDI. Right colon may be injured by an improperly placed right paraumbilical trocar. Transverse colon may be injured during cholecystectomy—usually by inadvertent contact with an instrument that had been activated with cautery. If detected during the cholecystectomy, the injury should be repaired—laparoscopically, if suturing skills are available, or by open operation. More often, however, the colonic injuries go unnoticed during the cholecystectomy and the patients present with fecal peritonitis or intraabdominal abscess (Fig. 7.9) in the early postoperative period.

ANECDOTE: *A patient who presented with features of peritonitis after laparoscopic cholecystectomy was found to have a transverse colon injury alone (no bile duct injury) at laparotomy.*

7.11 Small Bowel

An inadvertent electrocautery or instrument injury to a small bowel loop (Fig. 7.10) may go unnoticed and present in the postoperative period as peritonitis.

> US Senator John Murtha, a Democratic Congressman, underwent a scheduled laparoscopic cholecystectomy on 28th January 2010 (after a previous hospitalization in December 2009, probably for an attack of acute cholecystitis) at the National Naval Medical Center in Bethesda, MD. It was described as a "routine minimally invasive surgery," but he required readmission to the Virginia Hospital Center, Arlington, VA three days later—he died on eighth February 2010. It is alleged that the doctors had "hit his intestines."
> http://www.medscape.com/viewarticle/716749
> This could have been an injury to the small bowel (during the insertion of the first trocar), the duodenum

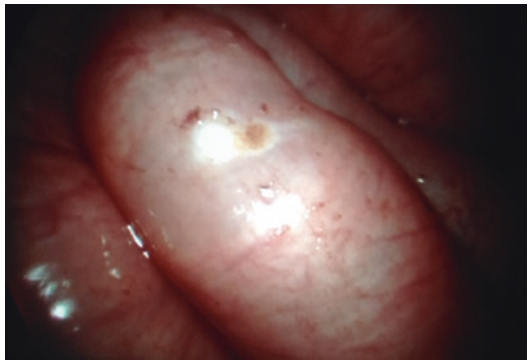

Fig. 7.10 Cautery injury to the small bowel

or the transverse colon (adherent to an inflamed gallbladder).

Every surgeon must keep in mind that non-biliary injuries may also occur during cholecystectomy; these injuries are more likely to be missed and may result in significant morbidity and even mortality.

Invited Commentary on Non-biliary Injuries During Cholecystectomy

Manuela Cesaretti and Antonio Iannelli

After its first description in 1985, laparoscopic cholecystectomy has revolutionized digestive surgery, and it is still considered a part of the basic armamentarium of any digestive surgeon. However, despite improvements in surgical training and the parallel developments in technology, the rate of biliary and non-biliary injuries remains stable over time. This is especially true for the novel techniques of cholecystectomy such as single-incision laparoscopic surgery (SILS), which offers only a better cosmesis as its main advantage but results in an increased rate of biliary and non-biliary injuries as high as 0.72% [29].

Intuitively, it would be expected that the risk of iatrogenic biliary and non-biliary injury declines with increasing surgical experience. Hobbs et al. [30] reported that about half of the total complications (bile duct injury, vascular injury, and intestinal injury) were attributable to relative inexperience as they occurred in the hands of surgeons who had performed between one and 50 procedures. Reduction in the risk of complications by careful mentoring of less experienced surgeons might thus have a measurable impact on overall rates of complications.

A clear understanding of the anatomy of the cystic and hilar plates is mandatory for a surgeon. Right hepatic artery (RHA) injury is the most common iatrogenic vascular complication of cholecystectomy [31]. The close proximity of RHA to the common hepatic duct is probably responsible for the high incidence of this injury. After the first description of normal and aberrant celiac trunk (CT) anatomy in 1756, literature has become abundant of anatomical studies on arterial variants. The "classic" hepatic arterial anatomy is present in approximately 55–80% of the cases [32]; for the remaining, multiple variants have been described. Embryologically, the CT originates from six pairs of ventral splanchnic vessels (subphrenic, upper, middle, lower ventricular, and upper and lower intestinal). During the fetal development, these pairs span and disappear. However, the persistence of longitudinal channels between primitive vessels may lead to anatomical vascular variations. When the RHA does not arise from the proper hepatic artery (PHA) or the common hepatic artery (CHA) i.e., it is aberrant, its origin is shifted to the aorta or any of the arteries whose normal course is towards the right side of the aorta such as superior mesenteric artery (SMA), gastro-duodenal artery (GDA), or right gastric artery [33].

Unlike bile duct injuries, RHA injuries when occuring alone may not lead to significant complications; on the contrary, the association of RHA injury with bile duct injury worsens the latter by inducing biliary ischemia. The true incidence of isolated RHA injury is unknown since they remain asymptomatic in most cases. Moreover, the incidence of combined RHA and biliary injuries is also difficult to estimate. A large study showed that 12% [34] of biliary injuries were accompanied by the RHA injury and other centers have reported up to 41% [8] and 61% [35]. In case of biliary inflammation with Calot's triangle fusion, it may be challenging to identify the cystic duct and artery safely so a prompt change in surgical strategy may result in lowering

the risk of hilar (biliary and vascular) injuries. While in the past, difficult cholecystectomy was strongly associated with conversion to open surgery, more recently, alternative approaches such a partial cholecystectomy are considered over conversion [36].

Another important but usually neglected consideration of these adverse outcomes of laparoscopic cholecystectomy is the financial aspects of the biliary and non-biliary injuries. The costs incurred as a consequence of an injury during laparoscopic cholecystectomy are considerable and are dependent on a variety of factors. Even if it receives little research attention, gallstone disease (without complications) represents the most expensive digestive tract disorder in the USA with an annual cost of more than $6.5 billion dollars. Roy et al. [37] estimated the mean total cost of iatrogenic injury during laparoscopic cholecystectomy (including healthcare service provided, surgery, endoscopy, and radiology) at 1.8 times the cost of an uncomplicated laparoscopic cholecystectomy. Kapoor et al. [38] published a cost analysis on a cohort of 47 patients in India undergoing major bile duct repair after laparoscopic cholecystectomy; the cost of repair was 10 times the cost of an uncomplicated laparoscopic cholecystectomy. However, the published financial analyses are mostly incomplete as economical evaluation is limited to the bile duct injury component of the combined bilio/ luminal-vascular injury alone, without incorporating the major escalation in costs contributed by the associated vascular or digestive injury into the final computation. Even though reported sparsely, neglected or mal-repaired bile duct injuries in the long term lead to chronic liver disease for which liver transplantation represents the ultimate life-saving option [39].

Several factors having a negative impact on the long-term outcome of biliary and non-biliary repair have been identified by previous studies. In particular, high and extended proximal biliary injury, late referral to the tertiary center, multiple previous surgical biliary repairs, and also simultaneous vasculo-biliary injuries represented independent prognostic factors for worse short and long-term outcomes [40, 41].

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8 Nomenclature and Classification of Bile Duct Injury

Vinay K. Kapoor

8.1 Nomenclature

Nomenclature of bile duct injury (BDI) and its consequences is not well defined; different terms are used to describe the same scenario and same term is used to describe different scenarios in different publications. Based on our fairly large experience with management of BDI and benign biliary stricture (BBS), the Author (VKK) would like to propose a standard nomenclature for BDI and its consequences.

8.1.1 Acute Bile Duct Injury

Acute BDI should be differentiated from external biliary fistula (EBF) which may ensue following the BDI and from BBS which may develop later as a result of closure of the EBF.

BDI is an injury to any part of the intrahepatic or extrahepatic biliary system. A bile leak from the surface of the liver following percutaneous intervention, e.g., liver biopsy, percutaneous transhepatic cholangiography (PTC), percutaneous transhepatic biliary catheterization (PTBC), percutaneous transhepatic biliary drainage (PTBD), radio-frequency ablation (RFA), transjugular intrahepatic porta-systemic shunt (TIPSS), etc. is an example of an intrahepatic BDI. The surgical BDI is usually an injury to the extrahepatic bile duct; rarely, an intrahepatic bile duct, i.e., in the gallbladder bed, may be injured during a difficult cholecystectomy or extended (radical) cholecystectomy for gallbladder cancer.

8.1.2 Bile Leak

The commonest manifestation of a BDI is bile leak (extravasation of bile outside the biliary system). Bile leak also occurs as a result of an anastomotic leak after a bilio-biliary anastomosis (BBA) or bilio-enteric anastomosis (BEA). Abdominal pain, distension, tenderness, fever, tachycardia, and leukocytosis are features of bile leak but *their absence does not rule out a bile leak*. It has to be kept in mind that bile in the peritoneal cavity does not get absorbed, is toxic, can get infected, and can cause sepsis (peritoneal or systemic). A ligated or clipped duct may, however, not be associated with bile leak and

With inputs from Rajinder Parshad Department of Surgery, All India Institute of Medical Sciences (AIIMS), New Delhi, India. Also see Invited Commentary on Nomenclature and Classification of Bile Duct Injury by Abe Fingerhut (pp 94–95)

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https://doi.org/10.1007/978-981-15-1236-0_8

may give rise to biliary obstruction (jaundice and cholangitis) and sepsis (cholangitis) directly.

Continuing (ongoing) bile leak needs biliary drainage—endoscopic (if the biliary ductal continuity is present) or percutaneous to decompress the bile duct—to control it.

8.1.3 Bilo-Peritoneum

Presence of bile in the peritoneum because of bile leak may manifest as biloma, bile peritonitis, or bile ascites, all collectively referred to as bilo-peritoneum (chole-peritoneum).

8.1.4 Biloma

A localized collection of bile in the peritoneal cavity with no generalized peritonitis is called biloma—it may get infected to form an intra-abdominal abscess (IAA); a biloma can be detected on ultrasonography (US), computed tomography (CT), or magnetic resonance imaging (MRI) and can be drained by image (US/CT) guided percutaneous catheter drainage (PCD). Surgery (laparoscopy or laparotomy) may rarely be required for multiple or multiloculated bilomas not amenable to PCD.

8.1.5 Bile Peritonitis

Presence of free bile in the whole of the peritoneal cavity with clinical features of generalized peritonitis and sepsis is called bile peritonitis (cf. bile ascites, where there is no infection). Majority of these patients will require laparoscopy or laparotomy for thorough peritoneal toilet and drainage; some patients may be managed by multiple PCDs and peritoneal lavage.

8.1.6 Bile Ascites

Presence of free bile in the peritoneal cavity without clinical features of peritonitis and sepsis is called bile ascites (cf. bile peritonitis, where infection and sepsis are present).

8.1.7 Minor and Major Injuries

BDIs are often classified as minor (bile leak) or major (transection) but the so-called minor injuries can also result in sepsis (peritoneal and biliary), may even cause death, and may eventually evolve into a BBS.

Amsterdam Type A injuries including cystic duct and peripheral duct leaks are classified as minor injuries—it must, however, be kept in mind that biloma forming as a result of these injuries also requires intervention in the form of PCD and endoscopic stenting, may result in major morbidity including peritoneal and systemic sepsis, and rarely, may also result in death due to bile leak, sepsis, systemic inflammatory response syndrome (SIRS), and multiple organ dysfunction syndrome (MODS). One out of 10 patients with Amsterdam Type A injury died [1]. The Amsterdam Medical Center (AMC), Amsterdam, Netherlands group reported four deaths after Amsterdam Type A injuries [2]. Out of a total of 800 BDIs managed at the Academic Medical Center (AMC), Amsterdam, Netherlands, 216 were Strasberg Type A; mortality in these 216 cases was 9 (4.2%) [3].

Several reports have described a lateral common bile duct (CBD) injury as a minor injury failing to appreciate that such an injury may also evolve into a BBS in the long term. The Johns Hopkins Hospital (JHH), Baltimore, MD, USA group correctly classifies partial laceration as a major injury.

> There is nothing like a ‘minor’ bile duct injury; all bile duct injuries are ‘major’.

8.1.8 Incomplete (Lateral/Partial/Tangential) Injury

A BDI involving only part of the circumference of the common bile duct is classified as incomplete (lateral/partial/tangential) injury. EBF in an incomplete BDI is more likely (>90%) to close than in a complete BDI (50–60%) [4]. An incomplete BDI may heal without resulting in any biliary obstruction—no BBS forms and no repair is required.

Terms such as partial transection [5] and incomplete transection [6] have been used to describe an incomplete/lateral/partial BDI though they are inaccurate because transection of a tubular structure literally means its complete division.

8.1.9 Complete (Total/ Circumferential) Injury

Ligation or clipping of the entire circumference and transection, division or excision of a segment of the CBD is classified as complete injury. A complete injury obviously leads to a BBS and needs repair.

8.1.10 High Injury

A BDI involving the biliary ductal confluence or one (usually right) of the hepatic ducts is classified as a high injury. Bismuth Type III and IV BBS involving the biliary ductal confluence are thus classified as high strictures (cf. low—Bismuth Type I and II BBS).

A high BBS may look like a hilar block on cholangiogram and may be difficult to differentiate from a hilar cholangiocarcinoma (cholecystectomy was performed for coincidental gall stones). Percutaneous transhepatic cholangioscopy (PTCS) and biopsy or brush cytology may be helpful to differentiate between the two.

8.1.11 Complex (Complicated) Injury

de Santibanes [7] has defined complex BDI as those involving the biliary ductal confluence (Bismuth Type IV), associated vascular (hepatic artery or portal vein) injury, presence of secondary biliary cirrhosis (SBC) and portal hypertension and after failure (stricture) of a previous hepatico-jejunostomy.

8.1.12 Biliary Drainage

External biliary drainage may be in the form of image (US or CT) guided percutaneous catheter drainage (PCD) of a biloma, percutaneous transhepatic biliary drainage (PTBD), endoscopic naso-biliary drainage (ENBD) or hepaticostomy (placement of a catheter into the lumen of the injured duct). Endoscopic stenting is a form of internal biliary drainage.

8.1.13 Definitive Management

Definitive management of a BDI or BBS may be surgical (suture repair of a lateral injury, bilio-biliary anastomosis (BBA) or bilio-enteric anastomosis (BEA) in the form of a Roux-en-Y hepatico-jejunostomy) or non-surgical (endoscopic or percutaneous balloon dilatation and stenting, if biliary ductal continuity is preserved).

8.2 Classification

8.2.1 Bile Duct Injury

A BDI may range from transient bile leak from a small peripheral intrahepatic bile duct in the gallbladder bed which will stop on its own without any intervention to an excision of a segment of the CBD with major vascular (hepatic artery and/or portal vein) injury resulting in acute liver failure and causing death. An ideal classification of BDI which encompasses all possible BDIs and addresses all issues viz. etiology, mechanism, timing and mode of presentation, associated complications (i.e., bile leak, sepsis, vascular injury), management guidelines, prognosis, and outcome does not exist. The most practical and useful classification, therefore, will be the one which guides management.

The most commonly used classification of acute BDI is the one proposed by Strasberg [8]. Other classifications which have been proposed from time to time by various authors/groups are as follows.

8.2.1.1 Siewert [9]

- Type I: Immediate biliary fistulae (cystic duct insufficiency)
- Type II: Late strictures of main duct without obvious intraoperative trauma to the duct

- Type III: Tangential lesions without structural loss of the duct
  - a: With additional vascular injury
  - b: Without additional vascular injury
- Type IV: Lesion with a structural defect of the hepatic or common bile duct
  - a: With additional vascular injury
  - b: Without additional vascular injury

8.2.1.2 Woods [10]

- Group 1: Cystic duct leak
- Group 2: Major bile duct leak or stricture
- Group 3: Major ductal transection or incision

8.2.1.3 Strasberg [8]

The most commonly used classification of acute BDI is the one proposed by Steven M Strasberg [8] of Washington University at St Louis, MO, USA. Strasberg included Bismuth types (vide infra) of BBS also in his proposed classification of acute BDI:

A. Bile leak from a minor duct (e.g., cystic duct, duct of Luschka, small ducts in the gallbladder bed in liver) still in continuity with the CBD—these are probably the most common injuries following laparoscopic cholecystectomy. Bile leak can stop on its own without any intervention; they also respond to endoscopic management (stenting) which is the treatment of choice.
B. Occlusion of a part of an injured biliary tree—almost always involving an aberrant right (sectoral/segmental) hepatic duct—these injuries are very likely to be missed as there is no bile leak. They usually cause recurrent cholangitis; they may sometimes result in asymptomatic atrophy of the sector/segment of the liver.
C. Bile leak from an injured duct not in continuity with the CBD—transection (without ligation) of usually an aberrant right sectoral/segmental duct—ERC may be deceptively normal as the injured isolated duct is not visualized; a careful review of the cholangiogram, however, will show absence of a part of the (right) biliary system. Hepato-biliary isotope scintigraphy shows bile leak, and MRC or PTC can identify the isolated bile duct. They do not respond to endoscopic intervention; PTBD alone can control bile leak.
D. Lateral injury to the extrahepatic major bile duct (common hepatic duct CHD/CBD).
E. Circumferential injury of major bile ducts (CHD/CBD)—these injuries are further subclassified as E1 to E5 as per Bismuth classification of BBS.

Strasberg A

Strasberg Type A BDI includes cystic duct leak (blow out) (Fig. 8.1) or bile leak from a small (minor) duct in the gallbladder bed in the liver. Treatment includes drainage of bile by percutaneous catheter drainage (PCD) and endoscopic stenting of the CBD. The Author (VKK) prefers an endoscopic naso-biliary drain (ENBD) in such cases as the leak is very likely to stop in a few days' time.

Strasberg B

Strasberg Type B BDI includes injury to an aberrant right (segmental, sectoral, or even main hepatic) duct which is occluded (clipped) so that there is no bile leak. These injuries may remain asymptomatic or present early (within days or weeks) with recurrent cholangitis or late (after

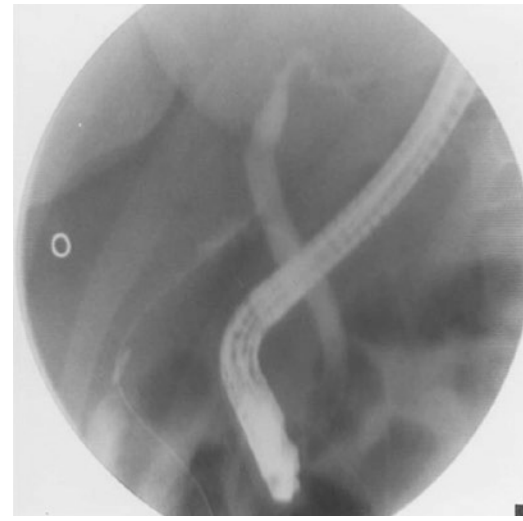

Fig. 8.1 Endoscopic retrograde cholangiography (ERC) shows intact common bile duct (CBD) with bile leak from cystic duct stump area—Strasberg Type A bile duct injury

months or years) with atrophy of a part of the liver. Asymptomatic (atrophy) patients do not require any intervention. Those who are symptomatic (with cholangitis) require PTBD, repair (which may be difficult because of small size of the ducts), or resection of part of the liver.

Strasberg C

Strasberg Type C BDI includes injury to an aberrant right (segmental, sectoral, or even main hepatic) duct which is open so that there is bile leak (Fig. 8.2). ERC does not reveal bile leak and may appear to be normal. Isotope hepato-biliary scintigraphy will show presence of extrabiliary isotope activity. Treatment includes drainage of bile (percutaneous catheter drainage PCD). Endoscopic stenting of the CBD does not help as the injured duct is separated from the CBD; percutaneous transhepatic biliary drainage (PTBD) of the isolated duct may have to be performed (but is technically difficult because of decompressed undilated intrahepatic bile duct) to control the bile leak.

Strasberg D

Strasberg Type D BDI includes a lateral (partial) injury to the common hepatic duct (CHD) or the CBD involving less than half of the circumference (if more than half of the circumference is involved, the injury should be classified as Strasberg Type E). These injuries, if detected intraoperatively, can be repaired. If detected postoperatively, treatment includes percutaneous catheter drainage of the biloma and endoscopic stenting of the CBD.

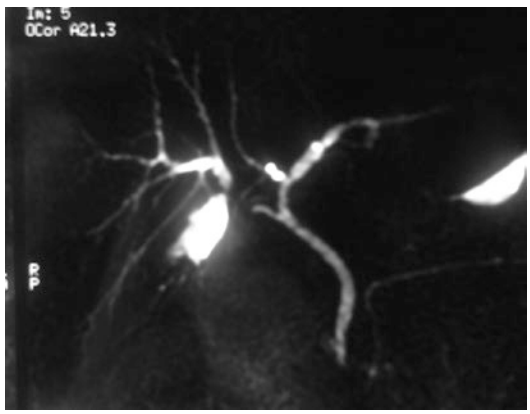

Fig. 8.2 Magnetic resonance cholangiography (MRC) shows isolated right sectoral duct injury with bile leak and biloma—Strasberg Type C bile duct injury

Strasberg E

Strasberg Type E BDI includes complete transection or a lateral (partial) injury involving more than half of the circumference of the CHD or the CBD. They are further subclassified as E1-E5 according to the Bismuth classification of BBS.

Main emphasis in the Strasberg's classification is on the duct involved. Strasberg's classification is very descriptive but does not guide management and prognosticate the outcome of acute BDI.

Connor and Garden [11] added E6 (excision of the extrahepatic biliary confluence) to the Strasberg's classification.

8.2.1.4 McMahon [12]

McMahon [12] classified BDIs as:

1. major BDI—laceration >25% of bile duct diameter, transection of CHD or CBD, development of bile duct stricture
2. minor BDI—laceration <25% of CBD diameter and laceration of cystic duct—CBD junction (buttonhole tear)—can be managed with simple suture repair

8.2.1.5 Amsterdam (1996)

Amsterdam classification of BDI proposed by the Academic Medical Center (AMC), Amsterdam, Netherlands [13] is as follows:

A. Leakage from cystic duct or aberrant or peripheral hepatic (biliary) radicals
B. Major bile duct injury with leakage from the CHD or aberrant segmental extrahepatic branch of the right hepatic duct (RHD)
C. Bile duct stricture without bile leakage
D. Complete transection of the bile duct with or without excision of some part of the biliary tract

The Amsterdam Type A injuries are usually classified as mild or minor; they need intervention in the form of PCD of the biloma and endoscopic

papillotomy and stenting to control ongoing bile leak. A missed/slipped CBD stone may be responsible for the cystic duct leak (blow out) and can be removed endoscopically.

Bile leak from any source, including cystic duct or peripheral hepatic radicals, can also be dangerous and even fatal. Many reports of BDI include anecdotal cases of even mortality in such so-called minor or mild injuries (vide supra).

8.2.1.6 Neuhaus [14]

- Type A: Peripheral bile leak (in communication with the CBD)
  - Type A1: cystic duct leak
  - Type A2: bile leak from the liver bed
- Type B: Occlusion of the CBD (or right or left hepatic duct, i.e., clip, ligation)
  - Type B1: incomplete
  - Type B2: complete
- Type C: Lateral injury of the CBD
  - Type C1: small lesion (<5 mm)
  - Type C2: extended lesion (>5 mm)
- Type D: Transection of the CBD (or hepatic duct not in communication with the CBD)
  - D1: without structural defect
  - D2: with structural defect
- Type E: Stenosis of the CBD
  - E1: CBD with short stenosis (<5 mm)
  - E2: CBD with long stenosis (>5 mm)
  - E3: confluence
  - E4: right hepatic duct or segmental duct

8.2.1.7 Csendes [15]

Csendes [15] proposed a mechanism based classification

- Type I: A small tear of the hepatic duct or right hepatic branch caused by hook or scissors during the dissection of the Calot's triangle
- Type II: Lesions of the cystic-choledochal junction due to excessive traction, clipping of cystic duct close to the CBD, in the use of a Dormia catheter, section of the cystic duct very close or at the junction with the CBD, or to a burning of the cystic-choledochal junction by electrocautery
- Type III: A partial or complete section of the CBD
- Type IV: Resection of more than 10 mm of the CBD

8.2.1.8 Stewart and Way [16]

Stewart and Way [16, 17] classified BDI as follows:

Class I (5%) Associated RHA injury in 0% of cases	Incision in the cystic duct extended unintentionally on to the CBD which is mistaken for the cystic duct, e.g., during intraoperative cholangiography (IOC)/incomplete transection of the CBD with no loss of ductal tissue
Class II (24%) Associated RHA injury in 15% of cases	Severe lateral damage to the CHD that produced stricture (stenosis) and/or fistula (bile leak) formation. This injury can be caused by inadvertent application of a clip on the CBD, e.g., during an attempt to control bleeding in the Calot's triangle, thermal damage, or an attempted exploration of an undilated (normal sized) CBD
Class III (61%) Associated RHA injury in 31% of cases	Transection of the CHD/CBD and excision of a variable portion. This is the classical laparoscopic BDI when there is an error of perception, i.e., the CBD is misidentified as the cystic duct IIIa—remnant of CHD present IIIb—CHD transected at the bifurcation IIIc—bifurcation excised
Class IV (10%) Associated RHA injury in 60% of cases	Transection or injury to an aberrant low lying right main, sectoral, or segmental hepatic duct which is mistaken for the cystic duct with or without injury to the CHD

8.2.1.9 Hannover [18]

- Type A: Peripheral bile leak (with reconnection to the main bile duct system)
  - Type A1: cystic duct leak
  - Type A2: leak in the region of the gallbladder bed
- Type B: Stenosis of the main bile duct (without injury, i.e., caused by a clip)
  - Type B1: incomplete
  - Type B2: complete

- Type C: Tangential (lateral) injury of the common bile duct
  - C1: small punctiform lesion (<5 mm)
  - C2: extensive lesion (>5 mm) below the hepatic bifurcation
  - C3: extensive lesion at the level of the hepatic bifurcation
  - C4: extensive lesion above the hepatic bifurcation

  With vascular lesions (i.e., C1d, C2s, etc.)

- d: right hepatic artery
- s: left hepatic artery
- p: proper hepatic artery
- com: common hepatic artery
- c: cystic artery
- pv: portal vein
- Type D: completely transected bile duct
  - Type D1: without defect below the hepatic bifurcation
  - Type D2: with defect below the hepatic bifurcation
  - Type D3: at hepatic bifurcation level (with or without defect)
  - Type D4: above hepatic bifurcation (with or without defect)

  With vascular lesions (i.e., D1d, D2pv, etc.)

- d: right hepatic artery
- s: left hepatic artery
- p: proper hepatic artery
- com: common hepatic artery
- c: cystic artery
- pv: portal vein
- Type E: strictures of the main bile duct
  - E1: main bile duct short circular (<5 mm)
  - E2: main bile duct longitudinal (>5 mm)
  - E3: hepatic bifurcation
  - E4: right main/segmental bile duct

Hanover is a very detailed and extensive classification with several descriptors for biliary and vascular injuries which can cover most injuries. But this itself becomes its disadvantage—when applied to 74 patients, the Hanover classification produced 21 different injury patterns [18].

8.2.1.10 Lau [19]

- Type 1: leaks from the cystic duct stump or small bile ducts in the liver bed
- Type 2: partial CBD/CHD wall injuries without (2A) or with (2B) tissue loss
- Type 3: CBD/CHD transection without (3A) or with (3B) tissue loss
- Type 4: right/left hepatic duct or sectoral duct injuries without (4A) or with (4B) tissue loss
- Type 5: bile duct injuries associated with vascular injuries

8.2.1.11 Kapoor BCD Classification [20]

The Author (VKK) has proposed a new BCD (bile leak, circumference involved, duct injured) classification of BDI.

Class	Description	Types
B	Bile leak	By—Yes (open duct) Bn—No (ligated or clipped duct)
C	Circumference involved	Cf—full circumference (transection or excision) Cp—partial circumference (clip, cautery, hole or excision)
D	Duct injured	Ds—significant duct (CBD, CHD, RHD, right sectoral or segmental duct) Di—insignificant duct (cystic duct, subsegmental duct, subvesical duct)

A few examples of various injuries are as follows:

1. By Cf Ds—a transected CBD/CHD
2. By Cx Di—cystic duct blowout
3. By Cp Ds—hole in the CBD/CHD
4. By Cx Di—hole in the cystic duct distal to the clips
5. Bn Cf Ds—clipped CBD/CHD
6. Bn Cx Di—clipped subvesical duct
7. Bn Cp Ds—clip on a part of the circumference of the CBD/CHD, thermal injury to the CBD/CHD

A "V" may be suffixed if an associated vascular injury exists.

This classification of BDI is simple and easy to remember, reproduce, and interpret; also, it guides the management and predicts the outcome of a BDI.

8.2.1.12 Cannon [21]

- Grade 1: leaks from the cystic duct stump, duct of Luschka, or accessory right hepatic ducts
- Grade II: all other levels of injury from the common bile duct to the intrahepatic ducts
- Grade III: all combined vascular and biliary injuries

8.2.1.13 EAES ATOM Classification (2013)

European Association for Endoscopic Surgery (EAES) recently proposed an ATOM—anatomic, time of detection (i.e., early or late), and mechanism of injury (i.e., mechanical or thermal) classification of BDI [22]. ATOM is a very exhaustive classification which will possibly cover all injuries but it is too detailed and complicated for use in clinical practice.

8.3 Benign Biliary Stricture

The most commonly used classification of BBS is the one proposed by Bismuth [23]. Bismuth described this classification in the open cholecystectomy era. Bismuth classification is applicable to BBS only and cannot be applied to classify acute BDIs, for which Strasberg's classification (vide supra) is most widely used.

8.3.1 Bismuth [23]

Bismuth classification (Fig. 8.3) was introduced before the laparoscopic era and is not applicable to acute BDI. Bismuth classified post cholecystectomy BBS based on the length of the CHD stump, i.e., lowest level at which normal healthy biliary mucosa is available for the bilio-enteric anastomosis and the patency of biliary ductal confluence. Bismuth classification indicates increasing difficulty of the repair of the BBS and is useful to plan treatment strategy, anticipate technical difficulty during repair, and predict long term outcome in terms of anastomotic stricture of repair.

1. Type I - Stricture >2 cm from the confluence of the right and left hepatic ducts, i.e., CHD stump is >2 cm (low CBD/CHD) (Fig. 8.4)
2. Type II - Stricture <2 cm from the confluence of the right and left hepatic ducts, i.e., CHD stump is <2 cm (high CHD) (Fig. 8.5)
3. Type III - Stricture at the confluence of the hepatic ducts (hilar stricture)—no CHD stump but the confluence is intact (Fig. 8.6)
4. Type IV - Stricture involving the confluence of the hepatic ducts—confluence is not intact and RHD and LHD are separated (Fig. 8.7)
5. Type V - Stricture involving an aberrant (right sectoral or segmental) hepatic duct with a concomitant CHD stricture (Fig. 8.8)

In a later publication, Bismuth [24] mentioned isolated right hepatic duct strictures also.

Bismuth recommended that type I strictures can be repaired without opening the left hepatic duct and without lowering the hilar plate—this would mean anastomosing the jejunum to the CHD. He further recommended that type II strictures can be repaired after opening the left hepatic duct but lowering the hilar plate is not always necessary. The Author (VKK) does not agree with him and recommends that irrespective of the Bismuth type of the BBS all repairs should be done at the biliary ductal confluence including the left hepatic duct after lowering the hilar plate—hilo-jejunostomy [25].

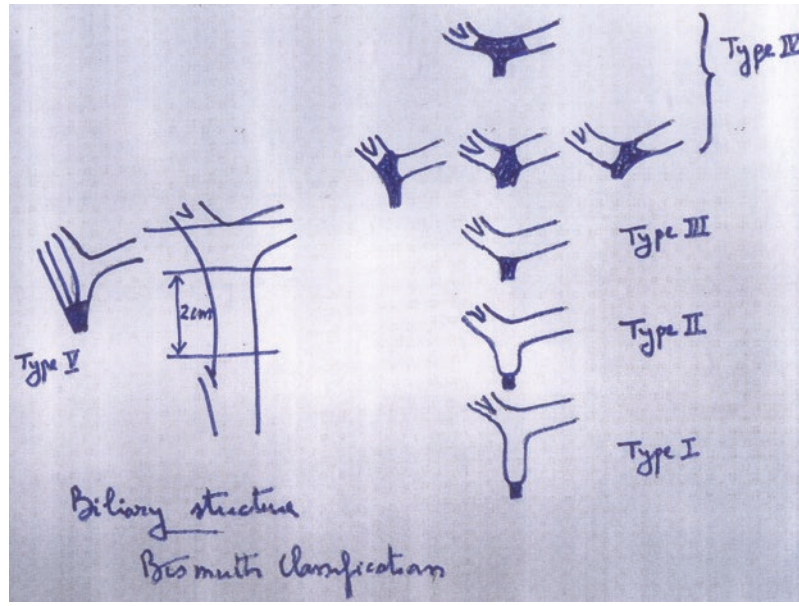

Fig. 8.3 Drawing of Bismuth Types of benign biliary stricture (drawn by the "Master" himself)

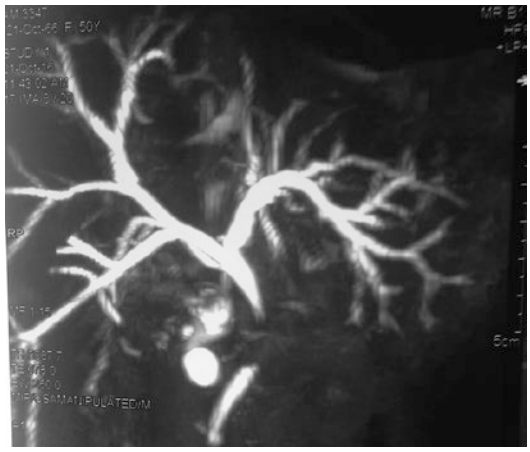

Fig. 8.4 Bismuth Type I benign biliary stricture—common hepatic duct stump >2 cm

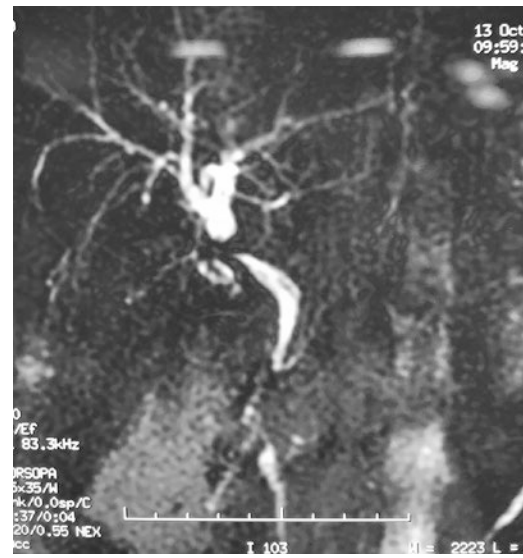

Fig. 8.5 Bismuth Type II benign biliary stricture—common hepatic duct stump <2 cm

We [26] had earlier proposed subclassification of Bismuth Type III BBS (biliary ductal confluence patent but no CHD stump) into subtype IIIa where the floor of the biliary ductal confluence is free, i.e., not involved in the stricturing process and is normal (Fig. 8.9) and IIIb where the floor of the biliary ductal confluence is involved in the stricturing process and is scarred though roof of the biliary ductal confluence is normal and there is communication between right and left hepatic ducts (Fig. 8.10). Type IIIb BBS is difficult to repair and results are worse; we suggested that Type IIIb BBS should be classified and treated as Type IV.

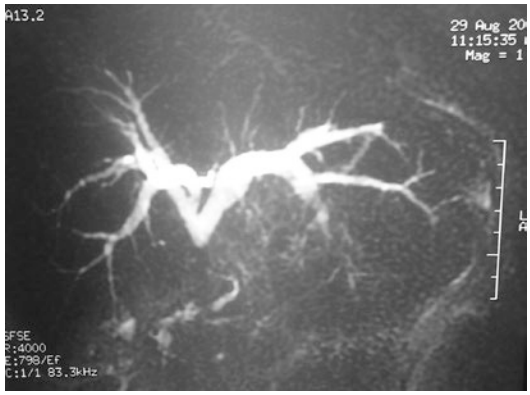

Fig. 8.6 Bismuth Type III benign biliary stricture—no common hepatic duct stump but confluence patent

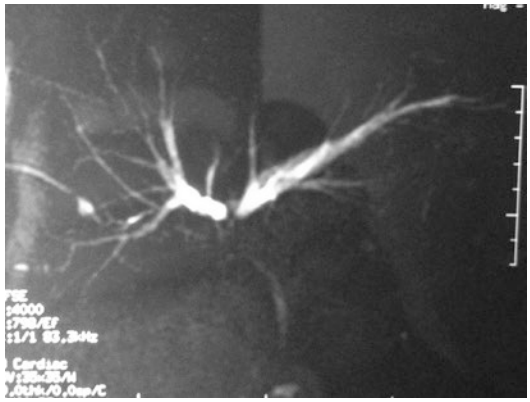

Fig. 8.7 Bismuth Type IV benign biliary stricture—confluence involved (not patent), separation of right and left hepatic ducts

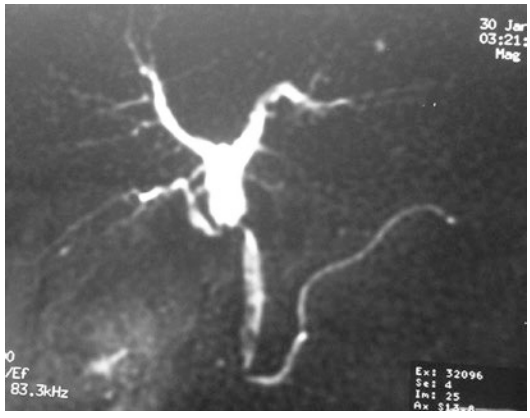

Fig. 8.8 Bismuth Type V benign biliary stricture—involving aberrant right sectoral duct with a concomitant common hepatic duct stricture

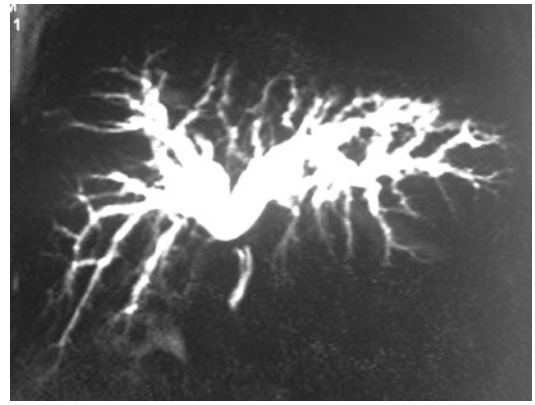

Fig. 8.9 Good Bismuth Type III benign biliary stricture with intact floor of confluence—Sikora Type IIIa

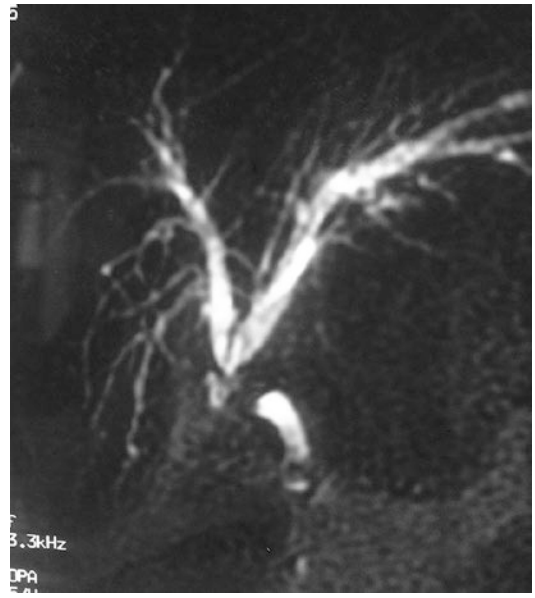

Fig. 8.10 Bad Bismuth Type III benign biliary stricture with floor of confluence involved in stricture—Sikora Type IIIb

8.4 Low, Mid, and High Classification

The author has proposed a new (low, mid, and high) classification of BBS [27].

- L (low)—CHD or CBD stump present (any length) (Fig. 8.11)
- M (mid)—Confluence patent and floor not involved—no CHD stump (Fig. 8.12)
- H (high)—Confluence involved (RHD and LHD separated) or confluence patent but floor involved (Fig. 8.13)
- An "a" may be suffixed to the BBS type if an anomalous duct (usually right sectoral or segmental) is involved in the stricture (Bismuth Type V).
- A "v" may be suffixed to the BBS type if there is an associated vascular (hepatic artery and/or portal vein) injury.
- An "r" may be suffixed to the BBS type if it is a recurrent anastomotic stricture.

A classification should address issues related to mechanism, prevention, treatment, and outcome (prognosis). None of the proposed classifications addresses all issues related to BDI viz. mechanism of injury, mode of presentation, condition of patient including presence of sepsis, associated vascular injury, etc. Classifications of BBS, similarly, do not address its complications, e.g., secondary biliary cirrhosis, portal hypertension, atrophy - hypertrophy of liver, etc. An ideal classification of BDI/BBS still eludes us.

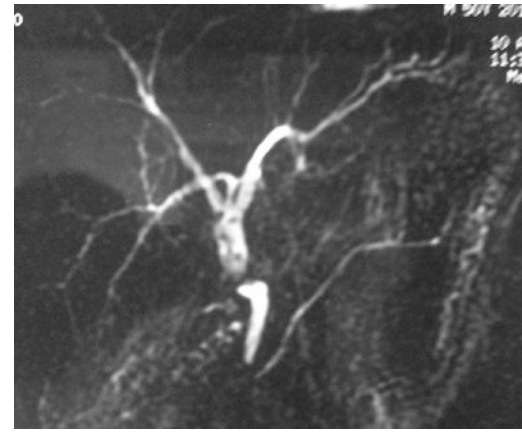

Fig. 8.11 Kapoor classification—low benign biliary stricture (common hepatic duct stump present—any length)

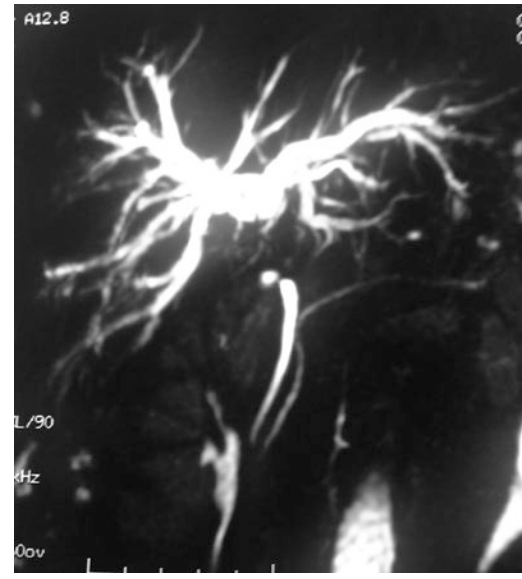

Fig. 8.12 Kapoor classification—mid benign biliary stricture (no common hepatic duct stump; confluence patent and floor not involved)

Invited Commentary on Nomenclature and Classification of Bile Duct Injury

Abe Fingerhut

Definitions in surgery are all too often the product of one person's or team's thoughts, at a specific moment, for a specific reason, sometimes influenced by an event or experience. However, when definitions are not the same and when one wants to compare results between studies, this leads to confusion. Standardization is the only way people can talk about comparing apples with apples and oranges with oranges; otherwise the reader does not know if the defined entity is the same as what he or she believes it is. Kapoor rightly underscores this.

He reviews the major classifications of bile duct injuries and details the one he described in 2008

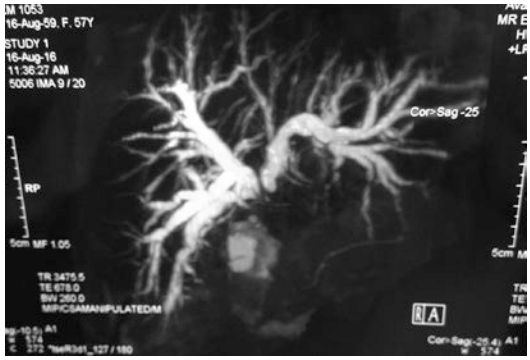

Fig. 8.13 Kapoor classification—high benign biliary stricture (confluence patent but floor involved or confluence involved)

[20]. However, as outlined previously, all these classifications have their strong and also their weak points. It was therefore to try to allow everyone to speak a common language that the European Association of Endoscopic Surgery (EAES) proposed the ATOM classification (ATOM standing for anatomy, time of detection, and mechanism), the goal of which was to unify the language and definitions and be all inclusive, that is any one report from one author using the ATOM classification could compare results with someone using another and different classification once the lesions were transformed into the common language classification, the ATOM classification.

The reasons why previous attempts at uniformization failed were summarized in the EAES publication. One major drawback is that most of these classifications attached specific BDI (occlusion or division, partial or complete) to a specific anatomical level, while in fact these injuries can occur almost anywhere along the biliary tree and in a variety of ways. Moreover, many of these classifications were published before laparoscopic cholecystectomy became the widespread reference technique it is today, and therefore did not take into account the changing pattern of injuries incurred since the introduction of laparoscopic cholecystectomy. Certainly the classical Davidoff injury (mistaking the common bile duct for the cystic duct) is the most common misinterpretation- or misidentification-induced injury, but bile duct injuries created by laparoscopic misadventures are more proximal, more often associated with loss of substance, with concomitant vascular injury, more often detected by bile leak rather than by formation of biliary stricture, and last, more often repaired (or attempted to repair) during the index operation. By combining all the existing items in most of the widely used classifications to date into one all-inclusive universally accepted ATOM classification, the EAES believes this would allow collection of data useful for further epidemiological and comparative studies as a comprehensive classification that collates all types of injury, whether culled independently by endoscopists, radiologists, and/or surgeons, integrated into an user-friendly, anonymous, electronic registry: this may ultimately lead to a more precise determination of the true incidence of BDI incurred during laparoscopic cholecystectomy, and ultimately, to preventive measures.

In this chapter, Kapoor writes, "A classification should address issues related to mechanism, prevention, treatment and outcome (prognosis). None of the proposed classifications addresses all issues related to BDI *viz*. mechanism of injury, mode of presentation, condition of patient including presence of sepsis, associated vascular injury. An ideal classification (of BDI) still eludes us." We do not agree with him—the all-inclusive EAES ATOM classification [22] does all of this.

Conversely, for benign biliary strictures, we agree with Kapoor when he writes that classifications of benign biliary strictures similarly do not address its complications, e.g., secondary biliary cirrhosis, portal hypertension, atrophy hypertrophy, and such. An ideal classification (of BBS) still eludes us.

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9 Management of Bile Duct Injury Detected Intraoperatively

Vinay K. Kapoor

A bile duct injury (BDI) may be detected during the operation (cholecystectomy) or it may present in the early postoperative period or even later (often weeks or months) during the follow-up after cholecystectomy. Intraoperative recognition of the BDI is less frequent during laparoscopic cholecystectomy as compared to during open cholecystectomy. A BDI is detected during laparoscopic cholecystectomy in only one-fourth to one-third of cases. The injury was recognized during the operation (laparoscopic cholecystectomy) in only 31% of 84 patients with BDI referred to the Johns Hopkins Hospital, Baltimore, MD, USA [1]. Less than half (45%) of 65 BDIs in Belgium were recognized during the operation [2]. Less than half (46%) of 235 BDIs which occurred during 56,591 laparoscopic cholecystectomies in 184 hospitals in Italy (1998–2000) were diagnosed intraoperatively [3]. Only 79 (26%) of 307 BDIs reported by Stewart [4] were recognized during the operation. Only 170 (23%) of 741 BDIs which occurred during 51,041 cholecystectomies performed in Sweden were detected intraoperatively [5]. In a report from France, only 193 (36%) of 543 BDIs were detected during cholecystectomy [6]. BDI was recognized in only 22 (16%) of 132 operations completed laparoscopically [7]. Only 23 (19%) of 124 BDIs reported from Iran were recognized intraoperatively [8]. In another one-fourth to one-third of cases, the BDI is detected in the early postoperative period (manifesting as bile leak). In the remaining cases, it manifests later (after weeks or months) during the follow-up as a benign biliary stricture.

> The fact that the patient has gone home after a cholecystectomy does not necessarily mean that the cholecystectomy was "safe."

A well thought of plan of investigations and management should be ready in the mind of every surgeon in case a BDI occurs and is detected during the cholecystectomy.

9.1 Intraoperative Recognition

If bile is seen during cholecystectomy especially during dissection in the Calot's triangle (Fig. 9.1), it should "ring alarm bells." The surgeon should stop and carefully look for the site and source of bile—whether it is coming from the gallbladder or from the common bile duct (CBD). The gallbladder (especially if it is thin-walled) very often gets opened during laparoscopic cholecystectomy and bile leaks out of the gallbladder.

Also see Invited Commentary on Management of Bile Duct Injury Detected Intra-operatively by Keith D Lillemoe (pp 105–106)

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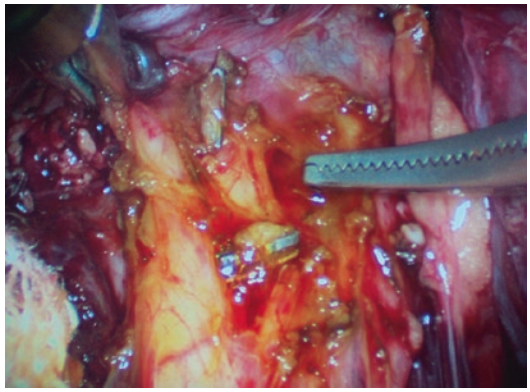

Fig. 9.1 Bile seen in the Calot's triangle should be considered to be a bile duct injury, unless proved otherwise

Gallbladder bile is greenish-yellow, thick, and viscid vs. common bile duct bile which is bright golden yellow, thin, and watery. After division of the cystic duct, the surgeon should carefully observe the direction in which it retracts—divided cystic duct should retract horizontally and medially to the left towards and behind the hepato-duodenal ligament; if the divided "cystic duct" retracts vertically down towards and behind the duodenum, it probably was the common bile duct (the classical laparoscopic cholecystectomy BDI). After division of the "cystic duct" if the gallbladder cannot be easily taken away from the hepato-duodenal ligament and the hepatic hilum, one must suspect whether what has been divided as the "cystic duct" was actually the common bile duct and the gallbladder is still attached to the hepatic hilum by the common hepatic duct (CHD) (Fig. 9.2). A "third" (after the cystic duct and the cystic artery have been divided) structure in the Calot's triangle should raise the suspicion of a BDI. This may indicate that the common bile duct and the right hepatic artery (RHA) have been divided (misidentified as the cystic duct and the cystic artery, respectively) and the "third" stricture encountered now is the common hepatic duct. In these situations, the common bile duct has been divided but recognition of the injury at this stage will avoid division of the common hepatic duct and excision of a segment of common bile duct along with the gallbladder. A divided common bile duct can be recognized during surgery—common bile duct has thinner wall

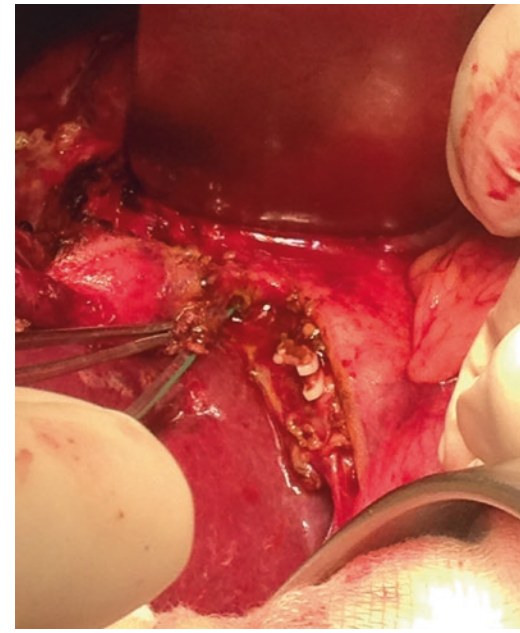

Fig. 9.2 Complete transection of the common bile duct—clip seen on lower common bile duct, infant feeding tube in the proximal common hepatic duct, gallbladder is still attached

and relatively larger lumen (cf. cystic duct which has thicker wall and relatively smaller lumen).

As soon as a BDI is recognized (or even suspected) during the operation, no further dissection should be done so as to minimize/avoid any further damage. The surgeon should spend some time to assess and evaluate the injury viz. site (segmental, sectoral, right hepatic, common hepatic, or common bile duct) and type (hole, lateral tear, or transection) of the injury; whether there is any loss of segment (excision) and associated vascular (usually right hepatic artery) injury. This may necessitate availability of intraoperative cholangiography (IOC). If the gallbladder specimen shows a tube attached to it or has two openings, a segment of the common bile duct probably has been excised with the gallbladder.

9.2 Intraoperative Cholangiography

Intraoperative cholangiography (IOC), also called peroperative cholangiography (POC), cannot be used as an excuse for not delineating and

demonstrating the biliary anatomy during every cholecystectomy because even IOC may not necessarily prevent a BDI but only results in its early detection.

Intraoperative detection of BDI is more common when IOC is performed. Intraoperative detection rate was 68% with the use of IOC vs. 32% without it [2]. Eighty percent of BDIs were detected intraoperatively when IOC was obtained vs. only 45% when it was not [9]. Rystedt [10] reported that 155 (89%) of 174 BDIs were detected intraoperatively because intraoperative cholangiography was performed in 93% patients.

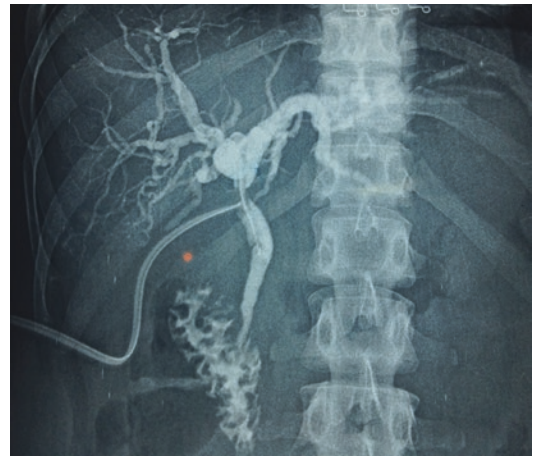

Fig. 9.3 End-to-end repair of a divided duct over a T-tube

9.3 Intraoperative Management

Immediate (intraoperative) repair of a BDI recognized during cholecystectomy is an attractive option as it has been shown to be associated with less inconvenience and morbidity, shorter hospital stay, and reduced treatment costs as compared to later (delayed) repair [11, 12]. The advantage of immediate repair over early repair is absence of sepsis and inflammation. Immediate intraoperative repair results in better physical composite score (PCS) of quality of life (QoL) than referral/delayed repair [13].

These theoretical advantages of immediate repair are, however, offset to a great extent by the poor results of immediate repair as compared to delayed repair. Pekolj [14] reported immediate repair in 17 patients (Type C-3, D-12, and E-2) between 1991 and 2010. Repairs included 10 suture repairs, 4 hepatico-jejunostomies, and 2 end-to-end anastomoses (12 open and 5 laparoscopic). Biliary stricture formed in 2 (12%) patients during early follow-up. In another report, immediate repair was attempted in 140 patients. Majority (102, 59%) of the injuries were minor (C1, <5 mm). Repairs included suture over T-tube (78, 45%) (Fig. 9.3) and hepatico-jejunostomy (30, 17%). 31 (18%) patients developed stricture—19 of these had undergone suture over T-tube [10]. A recent review by the French Surgical Association, however, reported that immediate repair in 194 patients was associated with 39% complication rate and 64% failure rate, much higher than 14% complication rate and 8% failure rate after late (beyond 45 days) repair in 133 patients [6].

Intraoperative management of a BDI depends on the type of the injury and (*more importantly*) the expertise and experience of the surgeon. Complete definition of anatomy, both biliary and vascular (arterial), is a prerequisite for immediate intraoperative repair. Delineation of the biliary anatomy requires IOC or intraoperative ultrasonography (IOUS) whereas vascular evaluation requires intraoperative Doppler—both requiring special equipment and expertise. If this is not possible, attempts at repair can cause further (higher) injury to the bile ducts and vascular (arterial) injury. It may also result in inappropriate/incomplete repair. It must be kept in mind that if facilities and expertise for IOC and IOUS are not available, as is likely to be the case in most situations, it is very difficult, almost impossible, to delineate the nature, type, or extent of injury. In such situations, repair should not be attempted; the easiest, safest, and best option, especially for a non-biliary surgeon, is to drain the subhepatic fossa and refer the patient to a biliary center.

The expertise (and experience) of the surgeon is one of the very important factors which determines the management of a BDI detected during the cholecystectomy itself. It is extremely important for the injuring surgeon to make an honest and realistic self-assessment of his/her expertise and experience to perform a repair in an undilated

duct, more so under the cloud of guilt, anxiety, and stress of having caused the injury. If the injuring surgeon (who performed the cholecystectomy at which the BDI occurred) is not a biliary surgeon and does not have the expertise of reconstructive biliary surgery (as is likely to be the case in most situations), and if help of a biliary surgeon cannot be made available to perform an immediate (intraoperative) repair, no attempt should be made to repair the injury. Attempts at repair by a non-biliary surgeon may cause further injury to the bile ducts (and vessels also). Results of repair by the injuring surgeon are likely to be poor. Xu [15] analyzed 77 intraoperative repairs performed in 15 hospitals in China (1997–2007)—24/35 (69%) repairs performed by a laparoscopic surgeon failed vs. 7/42 (17%) repairs performed by a specialist surgeon. Immediate non-specialist repair is also a risk factor for future litigation [16]. Moreover, the subsequent repair (even if then done by a biliary surgeon) is going to become more difficult and is less likely to be successful.

While conversion (from laparoscopic to open operation) is a safe option for prevention/reduction of risk of BDI in case of a difficult cholecystectomy, conversion is required after a BDI has occurred only if any kind of repair is planned. If immediate repair is not planned, there is no need to convert from laparoscopy to laparotomy. Conversion may be required in case of major bleeding which is not getting controlled laparoscopically. Lavage and drainage can (should) be performed laparoscopically also. This prevents formation of adhesions in the subhepatic area thus making future repair easier. The injuring surgeon, in such a situation, should suck out all the bile, lavage the subhepatic fossa with copious amounts of saline, and provide good subhepatic drainage by placing at least two large bore (24–28 F) drains in the subhepatic fossa so as to convert the acute BDI into a controlled external biliary fistula (EBF). These drains can be placed through the right subcostal and right paraumbilical ports. Omentum may also be placed in the subhepatic fossa to prevent the duodenum and the transverse colon getting adherent to the gallbladder fossa and the hepatic hilum so that dissection during the repair of the benign biliary stricture later is easier. The patient should then be referred to a biliary center, where facilities and expertise for interventional radiology and therapeutic endoscopy are available, for further management. This is in the best interest of the patient (*and the surgeon also*).

> Drain now, fix later.
> Krige [17]

If the injuring surgeon possesses the expertise (and experience) of reconstructive biliary surgery or help of a biliary surgeon is (or can be made) immediately available, an immediate (intraoperative) repair may be performed. An outreach service where a biliary surgeon travels to the hospital where the BDI occurred and performs an immediate (intraoperative) repair has been reported [18]. Even if a biliary surgeon is available to perform an intraoperative repair, the need and type of repair depends on the type and extent of injury which is difficult to assess on operative inspection only; complete and proper assessment of the BDI requires evaluation with IOC/IOUS.

If the surgeon has the expertise and experience to repair the BDI, the laparoscopic operation should be converted to open operation (*the Author (VKK) does not recommend laparoscopic repair of a BDI*). An experienced surgeon with laparoscopic suturing skills may, however, repair a small lateral injury to the common bile duct laparoscopically.

> The best time to fix it is that time—but only provided you know how to fix it.

9.4 Cystic Duct Injury

An injury to the cystic duct, recognized during operation, can be managed by reclipping if an adequate length of the cystic duct stump is still available (Fig 9.4a, b)—another reason to "hug" the gall bladder (and stay away from the CBD) during laparoscopic cholecystectomy. If the injury to the cystic duct occurs close to its junction with the common bile duct, it behaves and

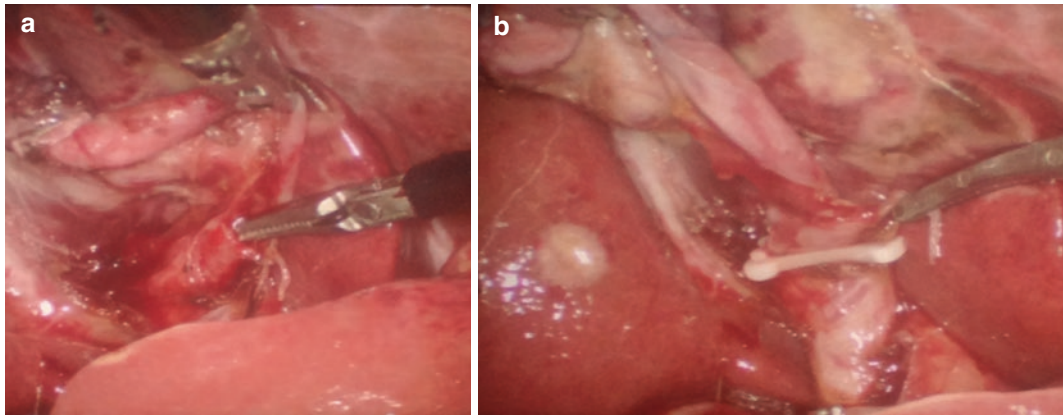

Fig. 9.4 (**a**) Cystic duct hole close to the gallbladder, (**b**) cystic duct clipped beyond the hole

has to be managed as a lateral common bile duct injury (vide infra).

9.5 Subvesical Duct Injury

Small subvesical ducts may be present in the gall-bladder fossa in 20–50% of cases; usually they do not produce intraoperative or postoperative bile leak as they get unintentionally obliterated when electrocautery (or ultrasonic energy) is used to separate the gallbladder from its bed in the liver. If a divided subvesical duct is identified during the operation, i.e., bile seen in the gallbladder bed away from the Calot's triangle (Fig. 9.5), an IOC can be obtained to define the amount of liver parenchyma drained by the divided subvesical duct. If IOC cannot be obtained, the size (diameter) of the duct can be used to guide further management. A small (<3 mm) cholecysto-hepatic, subsegmental or segmental duct in the gallbladder bed can be safely clipped; this will invariably result in asymptomatic atrophy of the segment of liver drained by the duct. If a major (>3 mm) subvesical, segmental, or sectoral duct has been injured, it needs subhepatic drainage or repair (depending upon the expertise available, vide supra) because clipping a significant size duct may result in stricture and recurrent cholangitis.

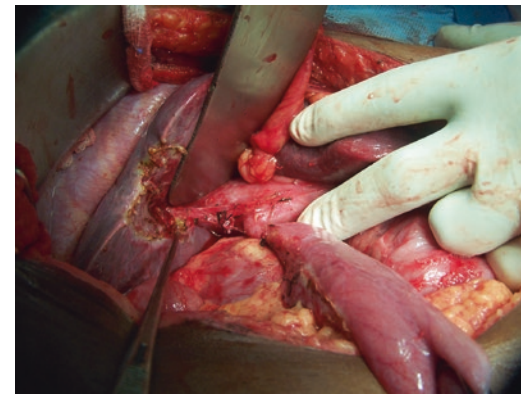

Fig. 9.5 Bile seen in the gallbladder bed indicates an injury to the subvesical duct

9.6 Common Bile Duct Injury

9.6.1 Clipped

If the common bile duct has been clipped mistaking it for the cystic duct (but not yet divided), the clip may be carefully removed (***NOTE*** *Removal of the clip itself may cause more injury to a thin-walled CBD than the application of the clip itself; self-locking clips are almost impossible to remove!*); nothing more needs to be done if the wall of the common bile duct at the clipped area is intact and there is no bile leak. An endoscopic stent may then be placed postoperatively, especially if liver

function tests (LFT), ultrasonography (US), or isotope hepato-biliary scintigraphy reveal evidence of biliary obstruction.

ANECDOTE *On the fourth postoperative day after laparoscopic cholecystectomy, a patient developed deep jaundice—magnetic resonance cholangiography (MRC) revealed clipped common bile duct. How is it that the common bile duct was only clipped and not divided? Probably, bleeding occurred from a short cystic artery stump which was controlled with a clip which went across the common bile duct. Gallbladder was not removed. What to do now? One option is to do relaparoscopy, perform cholecystectomy, and remove the clip on the common bile duct followed by postoperative endoscopic balloon dilatation and stenting.*

9.6.2 Lateral Injury

An incomplete/ partial/ lateral BDI with involvement of less than one-fourth to one-third of the circumference of the common bile duct detected during the cholecystectomy can be repaired with fine (4-0/5-0) long-acting absorbable sutures (e.g., Vicryl^{R}, PDS^{R} Ethicon). A skilled laparoscopic surgeon can perform this repair laparoscopically; or else conversion to open operation is recommended. Some surgeons recommend this repair over a T-tube [19]. *It is, however, important to remember that it is not easy to introduce a T-tube in a normal sized (undilated) common bile duct—repeated unsuccessful attempts to do so may cause even more injury to the CBD.* If used, the T-tube should be introduced through a separate opening in the uninjured part of the CBD (***NOT*** *through the injury itself*). An internal stent, with its lower end across the papilla into the duodenum, can be used in place of a T-tube. This allows the lateral injury to be closed completely but will require endoscopy to remove the stent. Round ligament has been used to reconstruct a bile duct defect [20]. Use of a vein patch for repair of an incomplete/ partial/ lateral injury of the CBD has also been described [21] but is rarely used. A serosal patch of a loop of jejunum has been described but is rarely used for immediate repair of an incomplete/partial/lateral BDI.

Case *A surgeon causes a partial circumference injury to the anterior wall of the CBD while trying to do a laparoscopic cholecystectomy in a patient with Mirizzi's syndrome. He (rightly) placed a subhepatic drain but, at the same time, clipped and closed the lower end of the injured CBD (apparently to avoid pancreatic juice leak). He should not have done that (clipping of the lower end of the CBD) as this resulted in loss of opportunity of endoscopic stenting in the postoperative period. If possible, he could have placed a T-tube in the CBD or just left it open (pancreatic juice does not usually leak through a BDI).*

9.7 Complete Injury

9.7.1 Hepaticostomy

For a complete injury detected during cholecystectomy, the Johns Hopkins Hospital, Baltimore, MD, USA group [22, 23] recommends hepaticostomy (exteriorization of the injured duct) by placing a catheter into the lumen of the injured (divided) proximal duct to convert the acute BDI into a controlled external biliary fistula (EBF) and to prevent intraabdominal bile leak and collection. The Author (VKK), however, differs from them and does not recommend this for various reasons. It is technically difficult to introduce a catheter into the (undilated, normal sized) injured (divided) proximal duct and repeated unsuccessful attempts to do so may itself cause further injury to the proximal bile duct. Even if one is able to place a tube into the proximal duct, it is likely to slip out unless it is secured with a clip around the duct. Placement of a clip around the duct compromises a few more mm of the proximal duct. Moreover, the clip applied to retain the catheter in the proximal duct can cause ischemia of the duct or even a vascular (right hepatic arterial) injury. Also, the catheter may still slip out of the duct and result in intraabdominal bile leak which it was supposed to prevent. A successful hepaticostomy will never allow the external biliary fistula to close and the proximal ducts to dilate. For these reasons, even if hepaticostomy is performed, drain(s) should still be

placed in the subhepatic fossa. The Author (VKK) advocates simple subhepatic drainage in such situations—this will convert the acute BDI into a controlled external biliary fistula.

9.7.2 Clipping of the Divided Duct

If the CBD (or a significant sized segmental or sectoral duct or right hepatic duct) gets divided during cholecystectomy, one option is to clip it with an intent to avoid bile leak and subsequent external biliary fistula; this also allows early dilatation of the proximal ducts making surgical repair easier. But the clipped bile duct invariably undergoes necrosis resulting in bile leak and formation of an external biliary fistula; only 4 out of 45 cases with intentional ligation of the proximal duct developed biliary dilatation; in the remaining 41 cases, the blind end of the proximal duct necrosed resulting in formation of biloperitoneum or biloma [24]. For this reason, even if clipping of the proximal divided bile duct is done, drains must always be placed in the subhepatic fossa to take care of the bile leak in case the clip gives way. In addition, the clip interferes with the axial blood supply of the CBD and causes further ischemic injury to the proximal bile duct. The Author (VKK) does not recommend clipping of a divided major duct.

9.7.3 End-to-End Repair

Various terms, e.g., end-to-end repair, duct-to-duct repair [25], primary ductal repair, primary sutured repair, etc. have been used by various groups to describe a bilio-biliary anastomosis for a BDI detected during cholecystectomy.

An end-to-end repair may be performed if the injury is recognized during the cholecystectomy itself, if it is a transection (division) only with no tissue loss (i.e., no excision of a segment) and if the injuring surgeon is a biliary surgeon or help of a biliary surgeon is (or can be made) immediately available. End-to-end repair is not possible in case a segment of the CBD has been excised (tissue loss)—the classical laparoscopic BDI; in that case a hepatico-jejunostomy will be required (vide infra). Complete kocherization of the duodenum must be done to gain extra length by mobilization of the lower CBD. The end-to-end repair is done using very fine (4-0 or 5-0) interrupted sutures of long-acting absorbable material, e.g., Vicryl^{R} or PDS^{R} Ethicon. This should, however, be done only by an experienced biliary surgeon and NOT by a general surgeon with no or little experience of reconstructive biliary surgery.

Use of T-tube in end-to-end repair is controversial. Placing a T-tube in an undilated duct may in itself be a technically demanding and frustrating exercise and may cause further injury to the bile duct. Liver transplant experience shows that use of a T-tube is not required and may even be associated with increased restricture rate. If at all a T-tube is used, it should be introduced through a separate incision in the CBD (and NOT through the repair itself). If a T-tube has been placed, it can be used to obtain a postoperative cholangiogram. A pure internal stent, e.g., a double pig tailed (DPT) stent or a length of an infant feeding tube can also be placed across the bilio-biliary anastomosis. The lower end should be passed beyond the papilla into the duodenum to facilitate endoscopic removal later.

Unlike subhepatic drainage, end-to-end repair avoids an external biliary fistula and the consequences of bile leak and bile loss as instant bilioenteric drainage is restored. End-to-end repair is a technically simpler procedure than hepatico-jejunostomy; it can be performed by a less experienced surgeon. Unlike hepatico-jejunostomy, it does not extend the injury to the proximal ducts (i.e., biliary ductal confluence and left hepatic duct) and does not carry the risk of injury to the vessels (especially the right hepatic artery).

Chances of stricture following an end-to-end repair are, however, high. Csendes [26] reported that almost half of the intraoperative end-to-end repairs failed (strictured) and required hepatico-jejunostomy later. Stewart [27] reported that primary end-to-end repair over T-tube of a divided duct failed in all 13 patients. 14 (29%) out of 48 end-to-end intraoperative repairs performed in 15 hospitals in China (1997–2007) failed [15].

The largest report in favor of end-to-end repair is of 56 patients referred to the Academic Medical Center (AMC), Amsterdam, Netherlands after an intraoperative end-to-end repair elsewhere (i.e., in a general hospital). T-tube was placed in 49 (88%) patients; it remained in place for mean 52 (range 2–154) days. At the time of referral to the AMC, 38 patients had anastomotic biliary stricture, 10 had anastomotic bile leak, and 8 had both stricture and leak. 47 (84%) of 56 injuries were below the biliary ductal bifurcation, i.e., Bismuth type I–II, only 9 (10%) were high injuries, i.e., Bismuth type III or IV. Three patients were treated with percutaneous balloon dilatation (all 3 successful), 40 were treated endoscopically (32 successful, 8 failures) and 13 underwent surgical repair (11 successful, 2 failures). During a mean follow-up of 7.1 ± 3.3 years, only one patient died due to injury/treatment related complications. Overall 91% patients had 5 year stricture free survival [28]. This actually is the worst case scenario as only cases with complications after an end-to-end repair performed elsewhere were referred to the AMC—successful end-to-end repair cases were, obviously, not referred to them.

The results of a French review are, however, contradictory. Immediate (intraoperative) direct end-to-end repair failed in 101/157 (64%) of cases [6] largely because in more than half of these cases the repair was performed by a non-biliary surgeon.

An anastomotic stricture following end-to-end anastomosis has intact biliary ductal continuity and can be treated easily with endoscopic or percutaneous balloon dilatation. Strictures following a failed end-to-end anastomosis are low (Bismuth Type I or II) and are easy to repair with hepatico-jejunostomy later with good results.

9.7.4 Hepatico-Jejunostomy

If a segment of the CBD has been excised, end-to-end repair is not possible and hepatico-jejunostomy is the only option. Immediate (intraoperative) hepatico-jejunostomy, however, failed in 23 (63%) of 35 cases [6].

Hepatico/choledocho-duodenostomy, even if technically feasible, is not recommended for repair of BDI.

9.8 Arterial Injury

An injured hepatic artery can be repaired if recognized during cholecystectomy. This, however, should be done only if the assistance of an expert and experienced vascular surgeon can be immediately obtained and magnification, e.g., loupe and fine (6-0 to 8-0) monofilament nonabsorbable suture, e.g., polypropylene (Prolene^{R} Ethicon) are available. This can be an end-to-end repair if the artery is divided; if a segment of the artery has been excised, a graft may be required. If the expertise of a vascular surgeon is not available, the injured hepatic artery can be safely ligated (as long as the right portal vein is patent). Elevation of liver enzymes, i.e., alanine transaminase (ALT) and aspartate transaminase (AST) will occur in the postoperative period but no clinical complications will usually ensue.

Mercado [29] performed early (minutes to hours after injury) repair in 32 put of 405 patients managed between 1990 and 2006. Sahajpal [30] defined repair within 72 h of injury also as immediate repair and reported satisfactory results in 13 patients. Arora [31] described an extended (mean 23, range 5–42 h after injury) immediate (prompt) repair in ten patients (all with complete transection, i.e., Strasberg type E injury) referred to them between 2000 and 2009. They could achieve a mean stoma diameter of 10 (range 10–21) mm but had to use transanastomotic stents (thus indicating an unsatisfactory anastomosis) in seven cases. At a mean follow-up of 42 (range 24–110) months, all patients had excellent or good outcome. Theirs, however, is a high volume biliary center in New Delhi, India which otherwise repairs a large number of BDIs and this number is a very small proportion of the cases which were managed by them during this period.

Immediate repairs are more likely to be done in a non-HPB center—117 (60%) of 194 immediate repairs in France were done in a non-HPB

center [6]. More patients who undergo immediate repair are likely to have direct end-to-end repair—157 (81%) out of 194 patients who underwent immediate repair had direct end-to-end repair and only 35 (18%) had hepaticojejunostomy [6]. In Finland, all suspected or diagnosed BDIs are to be referred to a central hospital [32]. In Denmark, a BDI, even if recognized during the cholecystectomy, has to be referred to a biliary center—repair was attempted by the primary surgeon in only five out of 139 BDIs sustained during 1995 and 2010 [7].

The Author does not recommend immediate (intraoperative) repair of a BDI recognized during cholecystectomy because of its practical limitations:

1. majority of the laparoscopic cholecystectomy related BDIs are not even recognized during the operation
2. the injuring surgeon is usually a general or laparoscopic surgeon with no expertise or experience of reconstructive biliary surgery
3. immediate help of a biliary surgeon is usually not available
4. facilities for IOC/IOUS to delineate the biliary and arterial anatomy and the type/extent of BDI may not be available
5. the bile ducts (normal sized) are undilated thus making the repair technically difficult
6. status of an associated vascular i.e. right hepatic artery injury may not be known
7. results of immediate repair are worse than those of delayed repair

The expertise/experience of the surgeon decides the intraoperative management of a BDI detected during cholecystectomy. A general/laparoscopic surgeon should only provide adequate drainage of the subhepatic area (laparoscopically) and refer the patient to a biliary center for further management. A biliary surgeon may undertake an immediate (intraoperative) repair of the BDI after proper evaluation of the site, type, and extent of the BDI; results of immediate repair are, however, inferior to those of delayed (after 4–6 weeks) repair.

Invited Commentary on Management of Bile Duct Injury Detected Intraoperatively

Keith D. Lillemoe

The chapter by Professor Kapoor is a well written summary defining the strategies and outcomes used by surgeons in the management of a bile duct injury (BDI) detected intraoperatively during cholecystectomy. As the Author (VKK) clearly documents, intraoperative detection of BDI during a laparoscopic cholecystectomy still occurs in less than 50% of the cases, therefore eliminating this important option in the management of most patients. Yet, as Professor Kapoor points out, recognition does not necessarily lead to the correct decision-making or best outcomes.

I personally agree with essentially every strategy that Professor Kapoor puts forth in this chapter. The most important message that I might add is that once an injury has occurred during a laparoscopic cholecystectomy, the operating surgeon's goal is to do no further harm. The reasoning behind this important tenet is that in most cases the cholecystectomy resulting in a BDI is not being performed by an experienced biliary surgeon. Thus, not only is emotion and judgment altered by the event, but experience is often lacking. If, however, the situation exists where an experienced biliary surgeon is available, he or she should be brought to the Operating Room immediately to assist in the decision-making, delineation of the anatomy, and the reconstruction.

The best time to repair any BDI injury is at the time that it occurs. Such treatment decreases postoperative complications, stress to the patient (which often leads to lawsuits), and lowers the costs of care associated with subsequent admissions and procedures. Nevertheless, a "botched repair" not only results in an unsuccessful outcome but more often than not it creates a worse situation with respect to the technical repair of the injury. Thus, the messages from this chapter as to optimal management are very important.

I also agree with all the points that Professor Kapoor puts forth concerning end-to-end repair

of a bile duct injury. Although this technique has a high incidence of failure with late stricture, such strictures do allow the biliary endoscopist an opportunity to dilate the stricture without the need for surgery or percutaneous intervention.

I would agree with Professor Kapoor that if a repair is not to be completed, minimizing the complications of the injury by controlling the bile leak is the most important step. The bile duct should not be clipped in order to control the bile leak or to theoretically allow the proximal bile ducts to dilate, in that this action seldom results in the desired outcome in either case and often results in loss of ductal length for ultimate repair.

Finally, the only point that I might add to this excellent review by Professor Kapoor relates to the role of transanastomotic stents to decompress the biliary system after biliary enteric reconstruction. Such stents are placed in a transhepatic fashion in cases when a hepatico-jejunostomy is performed and allows decompression of the biliary tree in cases when an anastomotic leak occurs, allows access for postoperative cholangiography to assess the repair, and allows access for percutaneous intervention such as balloon dilation, if necessary. The technique for placing these stents, as described by the group from the Johns Hopkins Hospital, Baltimore, MD, USA is relatively simple and seldom is associated with additional morbidity or difficulty with reconstruction.

I enjoyed this well written chapter and feel that it is the principles put forth by Prof Kapoor which should be understood by any surgeon performing laparoscopic cholecystectomy. Even with the relative infrequent nature of these injuries, it is important that the surgeon performing laparoscopic cholecystectomy knows what to do in this setting. If, however they do not know anything more, the most important word of advice is to call someone locally or at a nearby academic center for either consultation and/or assistance should a bile duct injury occur.

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10 Management of Bile Duct Injury Detected in the Post-Operative Period

Vinay K. Kapoor

The majority (more so, i.e., about 70–80%, during laparoscopic cholecystectomy cf. during open cholecystectomy) of the bile duct injuries (BDIs) remain unrecognized (are missed) during the cholecystectomy, the surgeon thinking that everything went on well—they manifest in the early postoperative period as bile leak or during the follow-up as benign biliary stricture (BBS) with jaundice and cholangitis. Even in the early postoperative period, many BDIs are missed because a drain is not usually placed after cholecystectomy and the patient is usually discharged early (usually 1–2 days after operation); some reports recommend laparoscopic cholecystectomy even as a day care procedure [1]. A BDI may be missed or diagnosis delayed in such cases. Many patients may then need readmission after a few days. Some patients with BDI may present either with a delayed (after days or weeks) bile leak or BBS (after weeks or months) with no bile leak in the early postoperative period. In the Italian national survey, 123 (54%) of 235 BDIs were diagnosed postoperatively [2]. 36 (63%) of 57 BDIs reported by us were detected in the postoperative period [3].

Also see Invited Commentary on Management of Bile Duct Injury Detected in the Post-operative Period by Dirk J Gouma (pp 121–123)

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10.1 Early Postoperative Period

A BDI may manifest in the early postoperative period as an external biliary fistula (EBF) if a drain was placed (Fig. 10.1) or as bile leak (with its manifestations including biloma, bile peritonitis, or bile ascites) if a drain was not placed.

Earliest symptoms of a BDI are due to bile leak and presence of bile in the peritoneal cavity. Not all patients with BDI have the classical clinical presentation viz. abdominal pain, jaundice, or features of sepsis; many (in fact, most) have nonspecific symptoms such as vague abdominal pain or discomfort, abdominal distension or bloating, anorexia, nausea and vomiting, general malaise and feeling of unwellness, low grade fever, tachycardia, tachypnea, and leukocytosis—absence of even these, however, does not rule out bile leak. Patients with minimal and non-specific symptoms are the very patients in whom the diagnosis is missed or made late and the mortality can be high. The clinical features of an intra-abdominal bile collection were subtle and were not recognized initially in as many as 77% of 154 patients so that the complication remained undiagnosed; delay in diagnosis and drainage of bile beyond 5 days resulted in serious illness [4].

A subhepatic collection of bile can cause inferior vena cava (IVC) compression resulting in diminished venous return and persistent tachycardia and hypotension which does not respond to fluid resuscitation; the patient improves only

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Fig. 10.1 Bile in drain after cholecystectomy

after the collection is drained—usually by image (US or CT) guided percutaneous catheter drainage (PCD)—this is Waltman Walters syndrome.

Most patients who undergo laparoscopic cholecystectomy are discharged within 24–48 h of operation; some centers even perform laparoscopic cholecystectomy as a day care procedure. If a patient with BDI and bile leak but subtle symptoms and signs is discharged prematurely from the hospital without any investigations, her condition may worsen at home in the absence of medical supervision. By the time the patient/relatives suspect that there is something wrong and seek medical attention, systemic sepsis may have set in and it may be too late to salvage the situation. Since most patients who undergo laparoscopic cholecystectomy are discharged from the hospital 1–2 days after surgery, arrangements must be made for at least one hospital visit in the follow-up to look for symptoms/signs suggestive of BDI (and also to check the histology report so as not to miss an incidental gallbladder cancer). It is not a bad idea to counsel the patients to report to the A&E if they are not well (fever, abdominal pain and distension, and jaundice).

A very high index of suspicion is required for early detection of a BDI. Any patient who is not settled (complaining of undue pain with more than the usual requirement of analgesics, not tolerating oral diet and not ambulant and out of the bed) and who has unstable vitals (fever, tachycardia and tachypnea) or unsettled abdomen (distended, tense, tender and silent) on the morning after laparoscopic cholecystectomy should be suspected to have a BDI, unless proved otherwise. Some surgeons obtain liver function tests (LFT) as a routine after laparoscopic cholecystectomy; minor derangements of LFT may be present even after an uneventful laparoscopic cholecystectomy but deranged LFT certainly warrants close monitoring and further investigations to rule out a BDI (or a missed common bile duct CBD stone). A routine US after every cholecystectomy is not required as it will reveal a small collection in a number of patients—this is a seroma which is of no consequence. But US finding of a collection in an unwell patient with unstable vitals and unsettled abdomen should be considered to be a biloma, unless proved otherwise. In case of suspicion, image (US or CT) guided needle aspiration of a fluid collection or ascites (found on US or CT) will confirm a bile leak. An isolated segmental/sectoral duct injury without bile leak may not produce any symptoms in the early postoperative period. The patient may present after few days/weeks with cholangitis (fever and jaundice) or may remain asymptomatic forever if the respective liver segment/sector undergoes atrophy.

Leukocyte counts (total leukocyte count TLC and differential leukocyte count DLC), LFT (especially alkaline phosphatase ALP and gamma glutamyl transpeptidase GGTP), ultrasonography (US), and isotope hepato-biliary scintigraphy should be done to rule out sepsis, biliary obstruction, bile leak, and intra-abdominal collection; all of which indicate BDI.

Cholangiogram is a must but only if an early repair is planned which, however, should not be done if bile leak is present.

ANECDOTE*: One of our patients who died after laparoscopic cholecystectomy came to the A&E 2 days after discharge from the hospital with non-specific symptoms—BDI was not even*

suspected and he was sent home with symptomatic treatment. He returned 1 day later with multiple organ dysfunction syndrome (MODS) and died.

10.2 Investigations

A patient with suspected acute BDI needs investigations to find out

1. Evidence of sepsis (leukocytosis).
2. Evidence (on US, CT, or MRI) of a (bile) collection (biloma) in the peritoneal cavity.
3. Evidence of continuing bile leak (isotope hepato-biliary scintigraphy, cholangiography).
4. Presence or absence of biliary ductal continuity (isotope hepato-biliary scintigraphy, cholangiography).

Evaluation for the extent (type and level) of BDI and assessment of associated vascular injury is not of much importance at this stage, unless an early repair (which the Author (VKK) does not recommend) is planned; this can be done at a later stage when definitive repair is planned.

Cholangiography is usually preferred over isotope hepato-biliary scintigraphy as it shows the site of bile leak also; scintigraphy, though much less expensive, has poor anatomical localization. Magnetic resonance cholangiography (MRC) is usually preferred over endoscopic retrograde cholangiography (ERC) as it is non-invasive but ERC offers opportunity for therapeutic intervention also in the form of papillotomy and stenting to control the ongoing bile leak and removal of residual/retained CBD stones, if present. In patients with a well formed mature (>2 weeks) controlled external biliary fistula (EBF), fistulogram is an easy way to obtain a good cholangiogram.

10.3 Ultrasonography

Ultrasonography (US) is a very useful investigation in the management of BDI and BBS. In acute BDI, US shows presence of fluid (free fluid, i.e., bile ascites or localized collection, i.e., biloma) in the peritoneal cavity; intra-hepatic biliary radical dilatation (IHBRD) in case of a ligated or clipped CBD (***CAUTION****—there may be no IHBRD in patients with ongoing bile leak*) and cholangiolytic abscesses in the liver. IHBRD on US indicates biliary obstruction due to CBD stone or BDI with ligated or clipped CBD as the cause of post-cholecystectomy jaundice (cf. hepato-cellular jaundice due to hepatitis where there is no IHBRD). US, however, is a poor investigation for establishing the presence of biliary ductal continuity. US is also used for image-guided diagnostic needle aspiration or therapeutic percutaneous catheter drainage (PCD) of a bile collection (biloma).

10.4 Computed Tomography

It must, however, be kept in mind that US is a highly subjective operator dependent investigation. Moreover, obesity and gaseous distension (due to paralytic ileus) may render US evaluation of the patient inadequate. In such a situation, if the index of suspicion of BDI and bile leak is high but US is inconclusive or does not reveal on intra-abdominal collection, threshold for cross sectional imaging, e.g., computed tomography (CT), which will again show the biloma (Fig. 10.2), should be low.

A liver perfusion abnormality on the contrast enhanced CT (Fig. 10.3) indicates an associated vascular injury. CT has the advantage of detection of a non-biliary (duodenal or colonic) injury also, provided oral and rectal contrast is administered. Patients with BDI, bile leak, and systemic sepsis may have renal dysfunction as part of multiple organ dysfunction syndrome (MODS); renal functions viz. blood urea and serum creatinine must be checked and patient should be well hydrated before a contrast enhanced CT is done, to avoid further deterioration of renal function.

10.5 Isotope Hepato-Biliary Scintigraphy

Scintigraphy is a non-invasive and inexpensive (though not universally available) investigation to

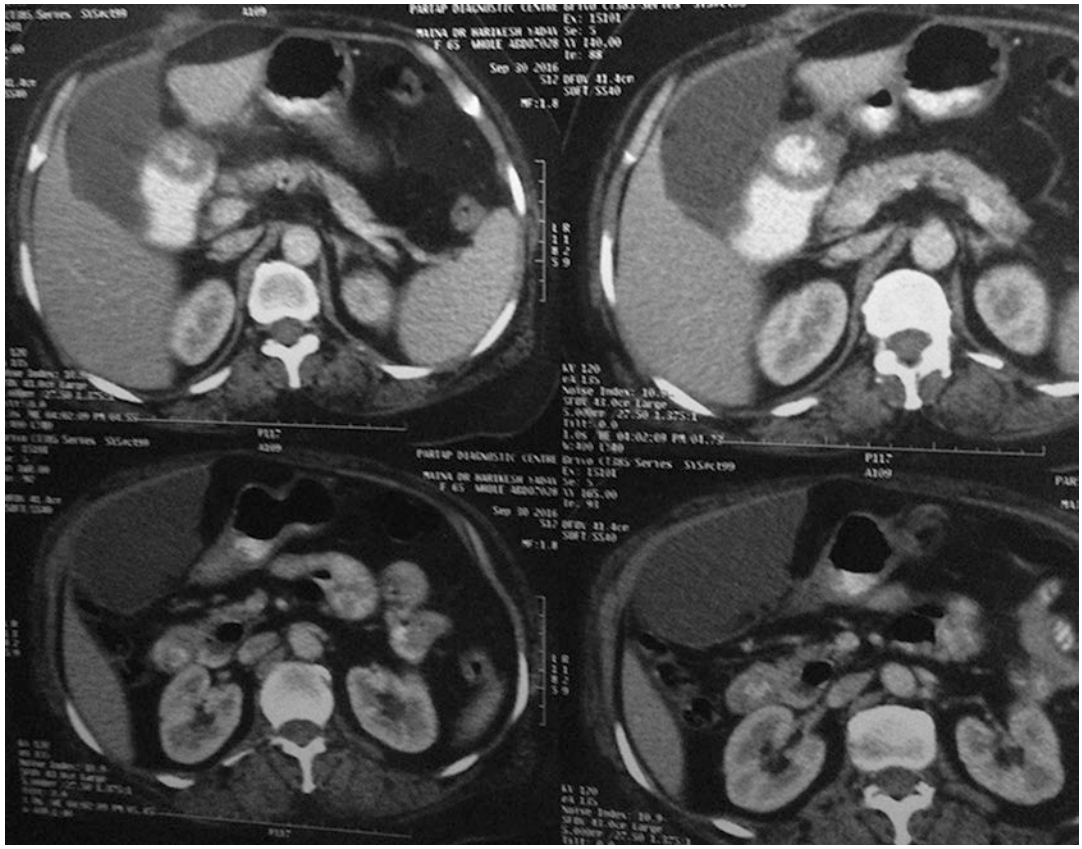

Fig. 10.2 Computed tomography showing biloma

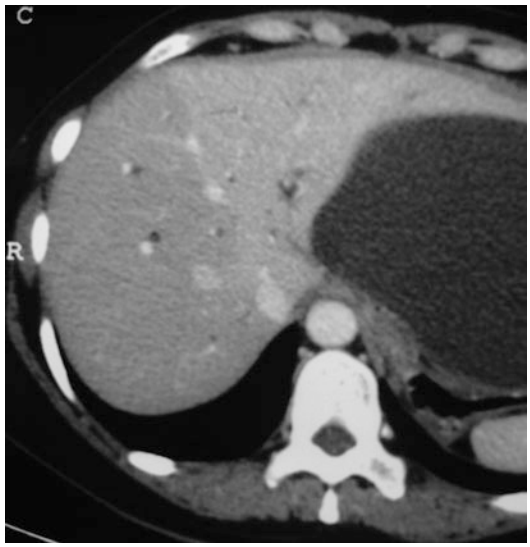

Fig. 10.3 Perfusion abnormality of right lobe of liver on contrast enhanced computed tomography suggests a vascular injury

1. detect or rule out bile leak (Fig. 10.4) in a suspicious case, especially when a drain was not placed during cholecystectomy; if there is no extrabiliary isotope activity on scintigraphy bile leak is virtually ruled out and other causes of an unsettled abdomen, e.g., bowel (duodenum or colon) injury should be suspected and looked for by CT with oral and rectal contrast; if bile leak is found on scintigraphy, CT should be done to locate the biloma,
2. establish the presence of biliary ductal continuity (before endoscopic intervention viz. stenting or endoscopic naso-biliary drainage (ENBD) is attempted to control the ongoing bile leak).

In presence of a major bile leak, however, scintigraphy may be false negative, i.e., no activity seen in the intestine even though biliary ductal continuity is present because of preferential

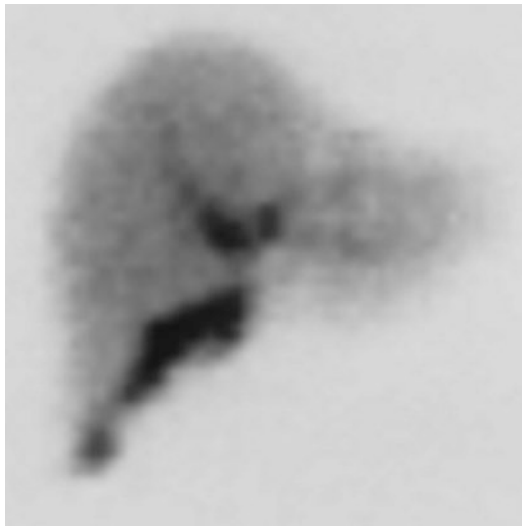

Fig. 10.4 Isotope hepato-biliary scintigraphy shows extrabiliary isotope activity indicating bile leak; in today's clinical practice it is a sensitive investigation to rule out bile leak

excretion of bile (and the isotope) through the low resistance EBF than through the CBD into the duodenum.

Anatomical localization, i.e., site of the bile leak, however, is not very good with scintigraphy. Isotope hepato-biliary scintigraphy is a very useful investigation to rule out bile leak as the cause of an unsettled abdomen—absence of extrabiliary isotope activity rules out a bile leak.

HIDA (hepato-biliary iminodiacetic acid) is the most commonly used isotope for hepato-biliary scintigraphy; BULIDA (butyl iminodiacetic acid) is a variant. DISIDA (Di-isopropyl IDA, Disofenin) and BrIDA (Bromo-triethyl IDA, Mebrofenin) can also be used, especially in patients with high serum bilirubin.

10.6 Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is preferable to CT as most patients with BDI are young females; in addition, it offers the advantage of a cholangiogram also. Cholangiographic information, i.e., whether biliary ductal continuity is present or not is helpful to decide endoscopic intervention viz. stent or

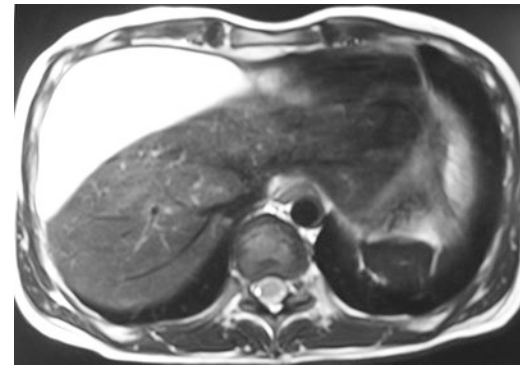

Fig. 10.5 Magnetic resonance imaging shows biloma

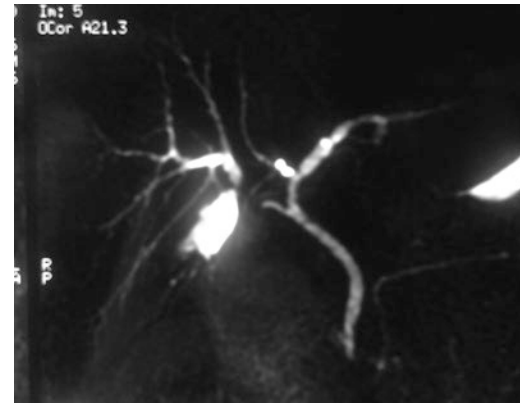

Fig. 10.6 Magnetic resonance cholangiography shows isolated right ductal injury with biloma

endoscopic naso-biliary drain (ENBD) to control ongoing bile leak. MRI detects biloma (Fig. 10.5), ascites, and cholangiolytic liver abscess. MRC also shows an isolated ductal injury (Fig. 10.6) in which case even though biliary ductal continuity is maintained, endoscopic intervention will not control the ongoing bile leak.

MRI is combined with magnetic resonance cholangiography (MRC) which shows bile leak and the status of biliary ductal continuity. MRC, however, may be false negative, i.e., no biliary ductal continuity shown when it is present, especially in patients with major bile leak resulting in collapsed ducts (the principle of visualization of bile ducts on MRC is presence of fluid, i.e., bile, within them). When combined with magnetic resonance angiography (MRA), MRI detects associated vascular injuries also.

Metal clips may interfere with MR evaluation, as does a large biloma or massive ascites which should preferably be drained by percutaneous catheter drainage (PCD) before MRI.

10.7 Endoscopic Retrograde Cholangiography

In acute BDI, endoscopic retrograde cholangiography (ERC) is of paramount value as it confirms or rules out bile leak (Fig. 10.7), detects the site and type of injury, demonstrates biliary ductal continuity, and detects any residual/retained CBD stones. It may, however, fail to detect an isolated segmental or sectoral duct injury (not in continuity with the CBD). ERC appears to be "normal" (no bile leak) in such cases but a careful review of the cholangiogram will show the absence of one of the (right) segmental or sectoral ducts which is injured but isolated from the main ductal system.

ERC is performed in patients with EBF as a part of a therapeutic intervention in the form of endoscopic stenting (ES) or endoscopic naso-biliary drainage (ENBD).

Percutaneous transhepatic cholangiography (PTC) is usually not required in a patient with BDI—MRC (with MRI) is preferred as it is non-invasive and provides more information (fluid collection, cholangiolytic liver abscess, isolated ducts, etc.) than PTC. The only indication for PTC will be as an adjunct to percutaneous transhepatic biliary drainage (PTBD) to control cholangitis or to reduce the ongoing bile leak in a patient with complete BDI or isolated duct BDI where endoscopic stenting is not an option.

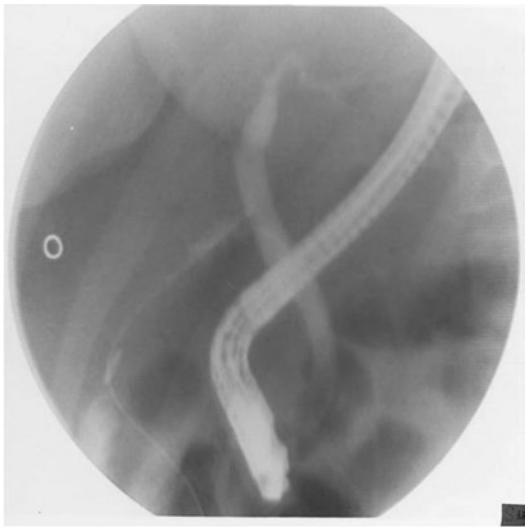

Fig. 10.7 Endoscopic retrograde cholangiography shows bile leak

10.8 Cavitogram

A cavitogram may delineate the size of the cavity after a biloma has been drained by a percutaneously placed catheter. This, however, carries the risk of introducing infection in the cavity and is rarely performed these days because the size of the cavity can be easily assessed by US, CT, or MRI.

10.9 Biliary Ductal Continuity

In a patient with BDI and bile leak, it is crucial to establish whether biliary ductal (enteric) continuity is present or not. This indicates feasibility of endoscopic stenting (or endoscopic naso-biliary drain ENBD placement) and also classifies the BDI into partial or complete, which predicts the chances of closure of the EBF and formation of a BBS in the long term. Normal (yellow) colored stools in a patient with BDI suggest the presence of biliary ductal continuity; stools may, however, be of normal color in a patient with complete ductal transection if an internal (bilio-enteric) fistula is present. Clay-colored stools indicate absence of biliary ductal continuity.

Biliary ductal continuity can be established in a non-invasive manner using radio-isotope hepato-biliary scintigraphy. Presence or otherwise of bilio-enteric continuity can be confirmed by direct cholangiography (T-tube cholangiogram, fistulogram, ERC, PTC, or MRC) also. In patients with intact biliary ductal continuity (partial/lateral/incomplete BDI), endoscopic stenting (or ENBD) will be of use to decrease the bile leak and hasten the closure of the EBF. If biliary duc-

tal continuity is not present, percutaneous transhepatic biliary drainage (PTBD) will be required to control the ongoing bile leak.

10.10 Initial Management

The initial management of a BDI is primarily directed towards control of sepsis and bile leak. Resuscitation is in the form of correction of fluid, electrolyte and metabolic imbalance.

The aims of management are to drain any intra-abdominal bile collection, treat sepsis (peritoneal and biliary), to stop or reduce the ongoing bile leak, and to convert the acute BDI into a controlled EBF. Bile in the peritoneal cavity, even in small amounts, can be very toxic and can cause havoc in the form of systemic sepsis. Any bile in the peritoneal cavity must, therefore, be drained out. This can be achieved in most cases non-surgically by percutaneous radiological and therapeutic endoscopic intervention in the form of image (US or CT) guided percutaneous catheter drainage (PCD) of bile collection (biloma) or intra-abdominal abscess (multiple PCDs may be required in case of multiple/multiseptated collections) (Fig. 10.8) and endoscopic stenting or percutaneous transhepatic biliary drainage (PTBD) of leaking bile ducts; surgery is rarely required, usually for a multiloculated biloma which is not amenable to percutaneous catheter drainage (PCD) or for generalized bile peritonitis. If at all surgical intervention is required, relaparoscopy may be preferred to laparotomy. In patients, who require multiple PCDs, over a period of time, bile drainage gets localized to one (usually subhepatic) drain while other drains become non-bilious (serous) and decrease in amount and can be removed one by one. PCD alone may control the bile leak if the injured duct is small, e.g., cholecysto-hepatic duct or cystic duct which closes on its own without any consequence.

Presence of free (cf. loculated, in biloma) bile in the peritoneal cavity without features of peritonitis or sepsis (cf. bile peritonitis) is called bile ascites. These patients can be managed by percutaneous catheter drainage PCD (in addition, endoscopic stenting or percutaneous transhepatic biliary drainage PTBD may be required to decrease or stop the ongoing bile leak).

Patients with BDI have bile leak, biloma, bile peritonitis, and cholangitis and need cover with appropriate parenteral broad spectrum antibiotics to prevent and treat sepsis. Empirical antibiotic use will be guided by the usual bacteriological flora in the respective practice. Gram-negative organisms, however, need to be covered. Cultures (bile and blood) should be sent at the time of admission and as soon as the sensitivity results are available, antibiotics may have to be changed, if required. Antibiotic cover is also necessary before any invasive cholangiogram (e.g., fistulogram, ERC, PTC) or non-surgical intervention (endoscopic stenting, ENBD, PTBD) is done on the biliary tract.

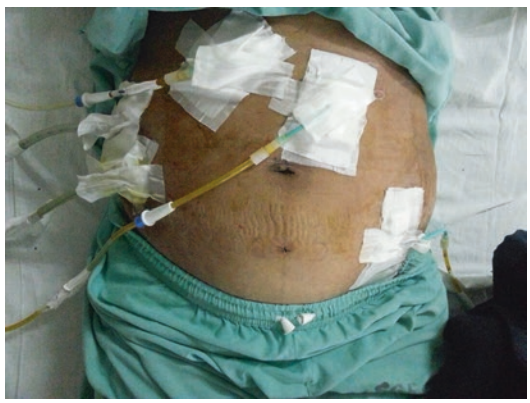

Fig. 10.8 Multiple percutaneous catheters to drain biloma

10.11 Cystic Duct Leak

Cystic duct stump blow out is one of the common causes of bile leak after cholecystectomy. This is more likely in presence of distal CBD obstruction, usually due to a residual/retained CBD stone (which was not suspected preoperatively) and happens around day 3–5. Incomplete clipping (partial circumference or inadequate loose clipping) or a hole in the cystic duct stump (caused by thermal injury or a sharp instrument) may also result in early (day 1 or 2) postoperative bile leak.

Cystic duct leak can be diagnosed by isotope scintigraphy, ERC, or MRC, but ERC is preferred if there is established bile leak because a therapeutic intervention (papillotomy, stent or ENBD placement and stone extraction) can also be performed at the same time. Cystic duct leak may stop on its own but endoscopic intervention viz. stenting or ENBD hastens its stoppage. Endoscopic occlusion of the leaking cystic duct using N-butyl cyanoacrylate has been reported [5].

Cystic duct leak is classified as minor in all classifications but the resultant bile leak can cause severe peritoneal and systemic sepsis and may even result in death of the patient. Peri-choledochal bile collection as a result of a cystic duct leak can cause peri-choledochal inflammation, fibrosis, and scarring and may result in a BBS later.

No BDI is minor.

10.12 Endoscopic Management

Endoscopic intervention plays an important role in management of post-cholecystectomy bile leak [6]. Endoscopic stenting reduces/stops the ongoing bile leak, controls cholangitis, and hastens the closure of the biliary fistula. The timing of endoscopic intervention in a patient with BDI and bile leak is a matter of debate. Different surgeons have different approaches towards a BDI and bile leak. Some groups (surgeons) follow an aggressive endoscopic approach and get an ERC done soon after the bile collection has been drained and as soon as the patient is fit enough to undergo the endoscopic procedure. One of the advantages of an aggressive endoscopic approach is detection and removal of a retained/residual CBD stone responsible for the bile leak as a result of cystic duct blow out. The disadvantage of early and aggressive endoscopic intervention is an unnecessary procedure with its associated morbidity in some patients in whom it may otherwise not be required because many of the minor bile leaks, e.g., from the cholecysto-hepatic duct or the cystic duct stump may stop on their own, i.e., without any endoscopic intervention. The conservatives follow a wait and watch policy in patients with a surgical drain or if the biloma has been drained by a PCD, especially if there is no residual undrained collection (biloma) on repeat imaging (US, CT, and/or MRI) AND if the patient is hemodynamically stable (no tachycardia, no hypotension) and sepsis free (afebrile, no leukocytosis, no abdominal signs). Endoscopic intervention is done in such a patient only if the bile leak does not show a trend towards reduction in amount over the next few days.

Endoscopic papillotomy (EPT) alone may reduce the ongoing bile leak by reducing the pressure in the biliary sphincter but is usually combined with endoscopic stenting (Fig. 10.9) or endoscopic naso-biliary drain (ENBD) (Fig. 10.10) to control the ongoing bile leak by decompressing the biliary tree and reducing the intrabiliary pressure. Some endoscopists do not perform a papillotomy and insert the stent only (without a papillotomy)—this avoids papillotomy related complications, e.g., bleeding. ENBD serves the same purpose as an endoscopic stent, i.e., biliary drainage to control the ongoing bile leak. It is usually not preferred over a stent as it is inconvenient to the patient and also results in

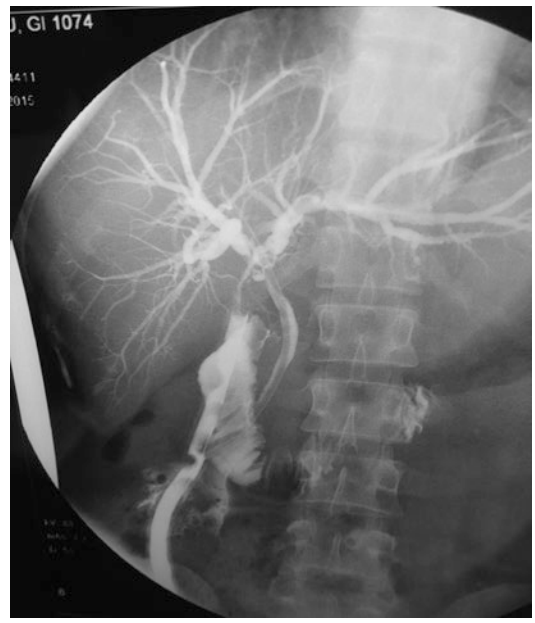

Fig. 10.9 Endoscopic stent placed to decrease/control ongoing bile leak

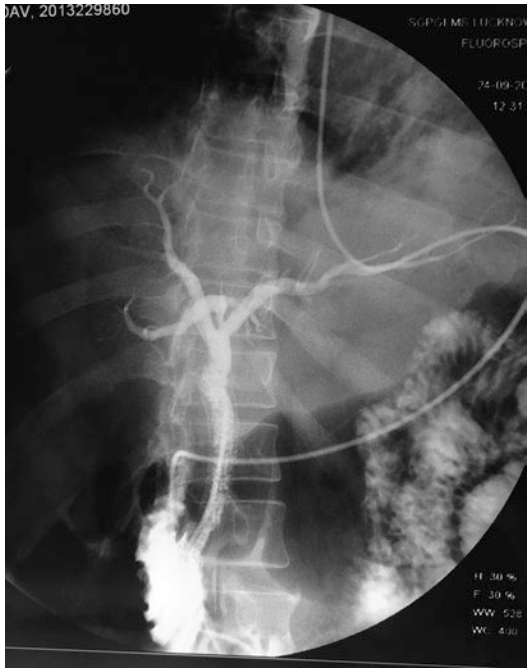

Fig. 10.10 Endoscopic naso-biliary drain (ENBD) in situ

external loss of bile causing fluid and electrolyte imbalance (cf. internal biliary drainage with a stent). The advantage of ENBD (over stent) is the option of flushing/irrigation, the ease of repeated cholangiography, and the ease of removal (without the need of a repeat endoscopy) [7].

> The Author (VKK) prefers an ENBD over a stent if the bile leak is suspected to be from a minor duct, e.g., subvesical duct or cystic duct which is likely to close in a few days' time.

Techni cal success rates for endoscopic stenting are high in case of minor injuries, e.g., cystic duct leak or cholecysto-hepatic duct leak, where the CBD is intact; rates may be lower in case of CBD injury. In case of a large lateral CBD injury, endoscopic intervention may be technically difficult as the guidewire may go out of the lateral hole in the CBD into the peritoneal cavity.

Endoscopic intervention is, however, possible only in presence of biliary ductal continuity which can be demonstrated by isotope hepato-biliary scintigraphy, fistulography, or MRC. In patients with a complete injury, i.e., ligation/clipping or transection/excision (Strasberg Type E BDI), endoscopic intervention is not possible. The only way that the bile leak can be stopped/reduced is by a percutaneous transhepatic biliary drainage (PTBD) which may be technically difficult because of absence of IHBRD due to the ongoing bile leak causing biliary decompression. PTBD may also be required to control uncontrolled cholangitis (not responding to parenteral antibiotics within 24–48 h) in such a case. A rendezvous (percutaneous transhepatic + endoscopic) approach has been described to stent the bile duct even if ductal continuity is not present but requires a lot of expertise and experience.

Endoscopic management reduces bile leak in patients with Strasberg Type A and D injuries where the injured duct is in continuity with the main ductal system. A "normal" (no contrast leak) ERC in presence of a clinical bile leak suggests an aberrant isolated segmental/ sectoral duct (Strasberg Type C) injury—the injured duct is disconnected from the main biliary tree and is not opacified in the cholangiogram and contrast leak is not seen. A careful review of the cholangiogram will, however, show a missing segmental/sectoral duct. An isotope hepato-biliary scintigraphy will demonstrate the isolated injured duct and the bile leak in such cases. Ongoing bile leak in such cases will not respond to endoscopic stenting but will require PTBD of the isolated duct.

For some BDIs, e.g., cystic duct leak, cholecysto-hepatic duct injury, small lateral CBD injury, PCD and endoscopic stenting alone will control the bile leak and the patient will recover without any sequel such as BBS. Some patients with lateral injury may develop BBS and require repair. In patients with complete injury, the EBF closes in about half of the patients who develop BBS and require repair. In the remaining half of the patients with complete injury, the EBF may not close at all and the repair may have to be performed with ongoing EBF and no proximal dilatation [8].

Patients undergoing endoscopic intervention should be closely watched and monitored for post-ERC complications, e.g., bleeding, perforation, cholangitis, pancreatitis, etc.

10.13 Cholangiolytic Abscess

Most cholangiolytic abscesses are small (and multiple). They usually respond to a course of parenteral antibiotics and biliary drainage (endoscopic or percutaneous). A large cholangiolytic abscess not responding to parenteral antibiotics within 48–72 h may need image (US or CT) guided percutaneous needle aspiration or catheter drainage.

10.14 Reoperation

If a BDI is diagnosed in the postoperative period after cholecystectomy, the temptation to reoperate upon the patient should be avoided by the injuring surgeon. The immediate management of a BDI is directed towards drainage of bile from the peritoneal cavity, control of sepsis (peritoneal and biliary), and control of bile leak—this can be accomplished in majority of the cases by nonsurgical (radiological and endoscopic) intervention. If facilities for radiological and endoscopic intervention are not available or if generalized peritonitis is present, relaparoscopy (after laparoscopic cholecystectomy) or relaparotomy may be required to perform peritoneal lavage and drainage. Reoperation may also be required if PCD of a biloma fails, e.g., in a multiseptate or multiloculated collection or one with much solid necrotic debris.

10.15 Early Repair

Early repair has been variously defined as repair within 2 weeks [9, 10] to 6 weeks; it has also been called intermediate repair [11].

Some groups [12] recommend early repair as it reduces the hospital stay and causes less inconvenience to the patient. Argument in favor of early repair is that it avoids the morbidity of percutaneous and radiological interventions which are often required if delayed repair is planned. Presence of biloma, peritonitis, or systemic sepsis are, however, contraindications for early repair. Before an early repair is planned, a complete cholangiogram (preferably MRC) must be obtained to delineate the extent (partial or complete) of the BDI—early repair should be done for a complete injury only. No attempt should be made for early repair in patients with partial BDI because not all patients with partial/lateral/incomplete BDI evolve into a BBS; some patients with partial injury of the CBD may settle with PCD and endoscopic stenting alone and heal without forming a BBS and may not need any further intervention. Associated vascular injury should be ruled out by Doppler, MRA, or CTA, as an associated vascular injury is a contraindication for early repair [13]. The ischemic damage to the bile ducts caused by the associated vascular injury evolves over a period of time to result in fibrosis of the proximal bile ducts thus making the level of the biliary injury/stricture higher (closer to the hilum). In presence of an associated vascular injury, the biliary stricture is likely to ascend and should be given time to do so before definitive repair is done. Bismuth [14] observed that the established biliary stricture is generally one level higher than the level of the acute BDI; this would mean that an early (as also an immediate i.e., intraoperative) repair will be done at a level lower than that of the final biliary stricture.

The Author (VKK) does not believe in, practice or recommend early repair of a BDI. Most patients with BDI have bile leak and biloma. Bile ducts are inflamed, edematous, and friable. No attempt must be made at this time to repair the injury—it is difficult to identify the ducts and even if the anatomy is clear, sutures may not hold in the inflamed edematous vascular friable tissues (Fig. 10.11) because of peritoneal sepsis caused by the bile leak. Sutures cutting through the bile duct result in higher risk of anastomotic leak which is the most important risk factor for anastomotic (recurrent) stricture. Repair in presence of an ongoing external biliary fistula (EBF) is also technically difficult as the ducts are collapsed (undilated).

Early repair may be performed, ***as an exception***, in a rare patient who has undergone laparoscopic cholecystectomy (when adhesions are less

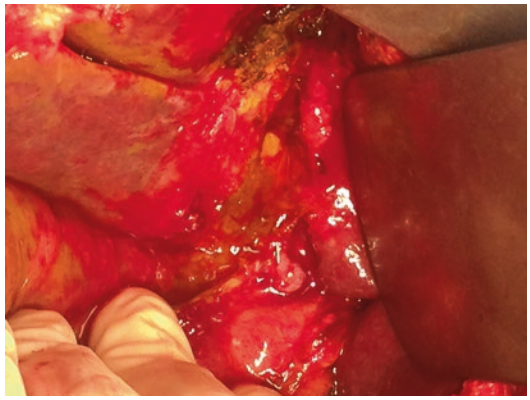

Fig. 10.11 Unhealthy (inflamed friable vascular) tissues in the hilum in a patient who was taken for early repair; the repair was abandoned

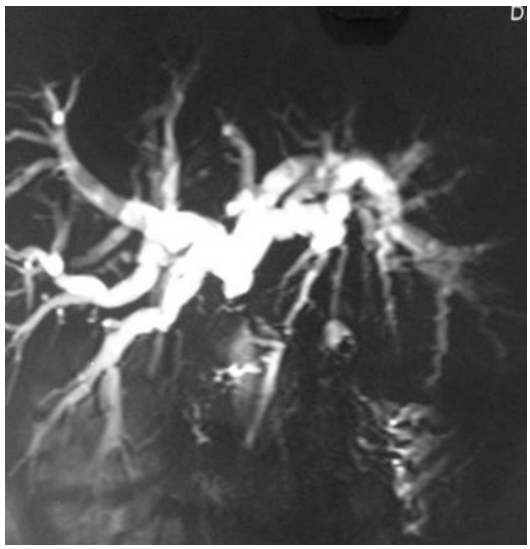

Fig. 10.12 Clipped/divided common bile duct with no bile leak and favorable anatomy—a suitable exception for early repair

likely to be present) with a ligated/clipped bile duct with no bile leak or biloma (and hence no peritoneal sepsis), no cholangitis, rapidly rising bilirubin, adequate proximal dilatation and favorable biliary anatomy (low, i.e., Bismuth Type I or II stricture with a common hepatic duct stump) (Fig. 10.12).

De Reuver [15] reported only 15 early (within 6 weeks) repairs—11 of these were performed between 1991 and 1999 and only 4 between 2000 and 2005; this change occurred because of "bad experience in the earlier years after semi-acute repair." The authors observed that "patients generally prefer early repair, we had to convince them for the potential benefit of the delay." Mercado [16] performed only 32 early (minutes to hours after the injury) repairs in 405 patients managed by them between 1990 and 2006.

There are few reports of successful early repair but in small number of cases. Wudel [17] performed early (median 2 days) repair in 74 patients with only (11%) failures but cautioned that this approach should be used only in stable patients with no ongoing sepsis or hemodynamic instability; follow-up was, however, very short (mean 24 months) in this report. Thomson [10] reported early (within 2 weeks) repair in injuries recognized during operation and referred to them immediately or in injuries recognized in the early postoperative period but in selected patients with minimal signs of peritonitis or sepsis; patients with peritonitis, sepsis, or organ failure were managed initially by drainage of the abdominal collections, biliary drainage, and treatment of sepsis, and repair was delayed beyond 6 weeks. Early repair was performed in only 22 out of 47 patients who were referred within the first 2 weeks of injury between 1988 and 2003. Holte [9] reported early (within 2 weeks) repair in 32 cases; during a follow-up of 9.2 years as many as 10 (24%) patients developed anastomotic stricture. Perera [18] reported that results of early (within 3 weeks) repair in 43 cases were similar to those of delayed repair in 41 cases. Felekouros [19] reported similar results of early (within 2 weeks) repair in 34 patients and late (after 12 weeks) repair in 22 patients. Arora [20] reported 10 prompt (within 72 h) repairs in 10 patients referred to a tertiary referral teaching hepato-biliary center between 2000 and 2009. Non-viable tissue at the end of the injured proximal duct was excised to get a healthy vascularized viable duct. The ducts were narrow—mean diameter was 7.5 (range 5–10 mm) and the size of the anastomotic stoma was 16.3 (range 10–21) mm. This necessitated use of anastomotic stents

in 7 cases. Over a mean follow-up of 42 (range 24–110) months, 7 patients were grade A and 3 Grade B—none required intervention.

Contrary to the results of these small reports, a large French Association of Surgery (AFC) review of 543 BDIs [21] reported higher (29% vs. 14%) rate of complications and higher (43% vs. 8%) failure rates after 216 early repairs than after 133 late repairs. Early repair is usually not advised as results of repair in presence of sepsis (biliary and/or peritoneal) and inflammation are poor. 7 out of 13 patients who had peritonitis at the time of early repair had failure [22]. Acute (within 6 weeks of injury) repair was associated with increased risk of complications both on univariate (OR = 5.7, CI 1.6–19.5) and multivariate (OR = 5.4, CI 1.2–24.4) analysis in 151 patients who underwent reconstructive surgery for BDI at the Academic Medical Center (AMC), Amsterdam, Netherlands; restricture occurred more frequently (5/15, 33%) after early vs. delayed (5/96, 5%) repair [15]. Recurrent stricture occurred more frequently after early (within 7 days) repair (8/44, 19%) than after delayed (median 79 days) repair (3/40, 8%) [23]. Stilling [24] reported results of a large prospective national database (1995–2010) of early (median 5 days) repair of BDI performed at 5 hepato-pancreato-biliary (HPB) centers in Denmark in 139 patients. Complications were more common (46%) after early repair—15 (11%) of 139 consecutive patients who underwent early repair required reoperation for anastomotic dehiscence or bleeding. During a median follow-up of 102 months, the hepatico-jejunostomy strictured in 42 (30%) patients at a median of 12 months—23 were managed with dilatation while 19 underwent repeat hepatico-jejunostomy. Finally, 5 patients required hepatectomy and 1 needed liver transplant; biliary specific mortality was 6 (4%). Analysis of American College of Surgeons (ACS) National Surgical Quality Improvement Program (NSQIP) database of 293 hepatico-jejunostomies done from 2005 to 2012 revealed an overall mortality of 2%—it was 5% for early repair vs. 0% for late repair; the authors recommended control of infection and improvement of functional status of the patient before repair [25].

10.16 Intermediate Repair

Sahajpal [11] defined intermediate repair as repair done after 72 h but before 6 weeks. In a report of 69 repairs, those done in the intermediate period (72 h—6 weeks) were significantly associated with biliary stricture 9/34 (26%); repair was recommended either in the immediate (0–72 h) or delayed (<6 weeks) periods [11]. They attribute this high recurrence to the presence of acute inflammation due to bile leak. Results of immediate repairs (2/13 recurrence) were better due to absence of acute inflammation; delayed repair also produced good results (0/22 recurrence) as inflammation had settled by the time the repair was done.

While early repair is debatable, repair in the intermediate period, i.e., 2–6 weeks should certainly not be done as it is invariably associated with poor results [11, 19].

10.17 CBD Stone

It should be kept in mind that the commonest cause of post-cholecystectomy jaundice is not a BDI or BBS but residual or retained CBD stone(s) which was not suspected preoperatively—these patients usually have pain and jaundice (cf. BBS where the jaundice is usually painless). If a CBD stone is suspected as the cause of post-cholecystectomy jaundice, ERC is the investigation of choice as it offers therapeutic opportunity also in the form of endoscopic papillotomy and stone extraction; a pre-ERC MRC will confirm the diagnosis of residual CBD stone (Fig. 10.13).

10.18 Other Causes

An unsettled abdomen after cholecystectomy (almost) always means a BDI and bile leak. If a BDI and bile leak have been ruled out (by isotope hepato-biliary scintigraphy and/or MRC/ERC), non-biliary i.e., bowel injuries should be strongly suspected and looked for by CT with oral and rectal contrast (see Chap. 6). Rarely, some other (unrelated) cause may be responsible.

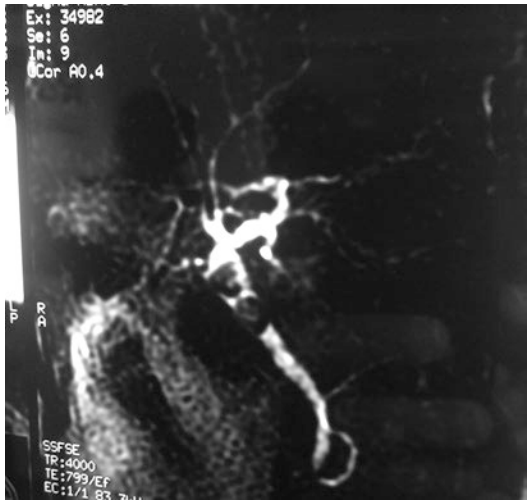

Fig. 10.13 Magnetic resonance cholangiography shows residual common bile duct stone after cholecystectomy

Not all jaundice after cholecystectomy is surgical, a patient who has had cholecystectomy can also, like anyone else, have hepatitis. Very high levels of liver enzymes (AST/ALT) with only mild elevation of alkaline phosphatase (ALP) and gamma glutamyl transpeptidase (GGTP) and absence of IHBRD on US suggests viral hepatitis.

ANECDOTE: *The Author (VKK) performed laparoscopic cholecystectomy in a young man with acute lymphatic leukemia (ALL) who had received chemotherapy, including steroids. Tissues were very soft and friable but the Calot's triangle anatomy was normal and the surgical procedure went on well. Next morning, the patient was febrile and the abdomen was distended and tender. There was no doubt in our mind that the soft and friable cystic duct stump had given way and there was a bile leak. US guided aspiration of the peritoneal fluid, however, revealed serous fluid only (no bile) and isotope hepato-biliary scintigraphy was normal (no extrabiliary isotope activity). Hemogram and peripheral smear then confirmed that it was a blast crisis.*

10.19 Late Presentation

An ischemic BDI caused by either thermal (electro-cautery) injury to the CBD or excessive circumferential mobilization of the CBD may result in a biliary stricture weeks, months, or sometimes even years after the cholecystectomy with no bile leak in the postoperative period.

Many BDIs manifest in the early postoperative period as an unwell patient, unstable vitals, and unsettled abdomen. The aim of investigations is to find a bile collection which should then be drained (percutaneously). Ongoing bile leak can be controlled/stopped with endoscopic stenting in patients with intact biliary ductal continuity. Early repair, though advocated by several groups, is not recommended by the Author (VKK).

Invited Commentary on Management of Bile Duct Injury Detected in the Post-Operative Period

Dirk J. Gouma

A bile duct injury (BDI) is one of the most dramatic complications during cholecystectomy and adequate management is crucial to limit the enormous extra burden on the patient. The present chapter on management of BDI by Dr. Kapoor is an excellent overview of the risk factors, diagnostic workup, and treatment options and clearly composed by a surgeon with an enormous experience in this subject. There are in fact very limited controversies to discuss here. Therefore I will try to make a few comments based on the experience and outcome of studies we performed in more than 800 patients referred for treatment of BDI to the Academic Medical Center (AMC), Amsterdam [26]. We should realize that there is still ongoing controversy concerning some aspects of diagnostic workup, timing and treatment of BDI. This is partly due to the selection of patients with BDI in those studies; ranging from a cohort study at a primary institute or a selected group of patients from a referral center or a nationwide survey about BDI. Subsequently, many different classification systems have been used. A potential associated vascular injury is also not well defined. Finally the (long term) outcome of treatment of BDI is reported with different endpoints ranging from complications, reoperation, or mortality after surgery to normal

liver function tests during follow-up or stricture formation and quality of life.

After all, most patients undergoing cholecystectomy worldwide will depend on their "index" general surgeon. The most important message might be to help them know how to succeed before, during, and after surgery. This chapter is not summarizing the procedure itself, but I would start mentioning that during a difficult procedure intraoperative cholangiography (IOC) under any uncertainty might be helpful. Visualization of the common bile duct does not only prevent an injury, but also might improve the repair strategy of small lesions, and prevents the index surgeon dealing later with a more severe BDI. This is helpful in understanding the local anatomy and not making the bad worse.

It was mentioned that after the laparoscopic cholecystectomy (LC) a drain might be helpful, but a systematic review and meta-analysis on intra-abdominal drainage showed that there is no significant advantage of drain placement and routine use seems to have unfavorable outcome. We do not use it. There was also a suggestion that after a LC as day care procedure, many patients may need readmission, but in our RCT on day care LC, we did not see any readmission and overall costs for the day care patients were substantially lower [27].

I fully agree, and one of the most important message is, that any patient who is not returning to normal activity within 24–48 h after LC should be suspected to have a BDI, unless proven otherwise. Laboratory examination, leukocyte counts and liver function tests (LFT), should be performed together with ultrasound and if a fluid collection is found, guided needle aspiration of the fluid collection should be performed. Please do realize that within LFT after a few days a normal alkaline phosphatase and gamma glutamyl transpeptidase and high bilirubin (resorption) is an indication for bile leakage, but increased alkaline phosphatase and gamma glutamyl transpeptidase and high bilirubin suggest biliary obstruction. In the past, scintigraphy was frequently used, but because there is not anatomical location of the bile leakage, cholangiography by MRC or ERC is now preferred. At a later stage, fistulography can be performed.

After visualization, classification should be the next principal step in patients suffering from a BDI. I was personally involved in the development of two different classification systems, the Amsterdam classification (too limited) and the recent published extended ATOM classification, which might be difficult for routine use [28]. Currently, the Strasberg classification is still being used most frequently.

The presence of biliary ductal continuity is mentioned as classification factor of BDI into partial or complete; but it is also useful in relation with the feasibility of using endoscopic stenting. Even if biliary ductal continuity is not present we start with ERC and in the absence of crossing of the lesion, perform a percutaneous transhepatic biliary drainage (PTBD), and continue with a percutaneous-endoscopic rendezvous procedure to restore continuity. In a recent study, this rendezvous approach was analyzed in 47 patients of whom 31 (66%) were diagnosed with complete transection of the duct (Strasberg Type E injury). The primary success rate of rendezvous approach was 94% (44/47 patients). We concluded that in experienced hands, the rendezvous procedure is a safe procedure, with a long term success rate of 55% and the rendezvous approach should be considered, either as definitive treatment or as a bridge to elective surgery [29].

Concerning treatment, it is indeed remarkable that a cystic duct leakage, Type A lesion, is not only classified as minor injury, but also considered as being a minor problem! The biliary leak will indeed frequently lead to severe peritoneal and systemic sepsis and may result in death of the patient. In our series, a Type A injury was diagnosed in 216 patients [30]. Treatment after referral was endoscopic in most patients ($n = 192$) and complications related to ERC procedure were limited, but BDI-related mortality was relatively high, 4.2% (9/216). The high in-hospital mortality for a relative "minor injury" was not due to failure of the endoscopic procedure, but mainly due to the patient condition at referral such as infection and sepsis, in particular in older patients with ASA 3 or 4. So this subgroup of patients with "minor" BDI lesions will have substantial risk of mortality [30]. Another important aspect is that

biliary strictures after a long term stenting or rendezvous procedure are in fact the optimal condition for an elective hepatico-jejunostomy (HJ) with a low complication rate [29–31].

Regarding reconstruction by HJ there is still controversy about the timing of surgery; in the discussion on early versus delayed repair, Dr. Kapoor mentioned that he does not recommend early repair of a BDI. Our previous study, showing that restricture occurred more frequently (5/15, 33%) after early vs. delayed (5/96, 5%) was also quoted, and indeed we showed 13 years ago that "acute repair" was an independent negative predictor of outcome after reconstructive surgery [31].

However, there is a recent study from Rystedt et al. showing that quality of life (QoL) is comparable to uneventful cholecystectomy, as long as the injury is diagnosed intraoperatively and immediate repair is also performed by the index surgeon [32]. In our comment, it was mentioned that only five patients underwent a direct HJ and only one was performed by the index surgeon; so again there was bias in selection [33]. Therefore the discussion should not be early versus delayed but the circumstances/ patient's condition/ preoperative criteria to select for an early or delayed procedure [33]. As mentioned in the Chapter by Dr. Kapoor already, Perera [34] also showed that when experienced HPB surgeons intervened early, even by traveling during the index procedure towards that hospital, long term outcomes were excellent.

We also realized that in our previous [31] study "acute repair" was defined within 6 weeks after the injury and delayed after 6 weeks; which seems to be a wrong definition, considering the clinical course in general. Acute repair should not be considered up to 6 weeks. Kapoor also mentioned that the 2–6 weeks interval is an "intermediate period" and reconstruction should not be done in this period. In a recent analysis, we changed the definition for early repair to "within 2 weeks after the injury" which seems more appropriate [35]. Using this new definition in our series of 265 patients with HJ we found the clinical pattern at referral, mostly delayed presentation with ongoing biliary peritonitis and sepsis, by far the most important factor for the choice of early (within 2 weeks) versus delayed surgery strategy. These factors of the patient population should also be included in outcome studies addressing this ongoing topic about timing of intervention.

Finally considering the remark about an ischemic BDI caused by either thermal injury to the CBD or excessive circumferential mobilization of the CBD, this might indeed result in a biliary stricture later on. Therefore, it might also be important that if a reconstruction is performed in patients with associated vascular (arterial) injury, the biliary anastomosis should be made higher up at the level of the biliary ductal confluence to prevent recurrent stricture formation.

This chapter of Dr. Kapoor has summarized nicely that BDI after cholecystectomy is a dramatic complication and adequate diagnostic workup, classification and selection of type, and timing of intervention, preferably in earlier stage by a multidisciplinary HPB team, will be crucial to improve the outcome.

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Consequences of Bile Duct Injury: External Biliary Fistula

11

Vinay K. Kapoor

Biliary fistula is an abnormal communication between the biliary tree and skin (external biliary fistula) or the gastro-intestinal tract (internal biliary fistula). External biliary fistula (EBF) is an intermediate stage between the acute bile duct injury (BDI) and an established benign biliary stricture (BBS). While much has been written about the long term sequel of BDI, i.e., BBS, not much information is available in the published literature about the intermediate event, i.e., EBF.

Like BDI, almost all EBF are traumatic—most are iatrogenic, following operations or interventions on the biliary tract or liver.

Leak from a bilio-enteric anastomosis, e.g., hepatico-jejunostomy, also results in an EBF. The Academic Medical Center (AMC), Amsterdam, Netherlands reported anastomotic leak in 24 (2.3%) out of 1033 patients who underwent hepatico-jejunostomy [1]. In a leak from a choledocho-duodenostomy, the biliary fistula is complicated by a duodenal fistula also; following pancreato-duodenectomy, it is a combined biliary-pancreatic fistula. Liver resection and drainage of a liver cyst (especially a hydatid cyst with biliary communication) or liver abscess may also cause an EBF. Rarely, a bile duct injury may occur during gastrectomy and may cause a bile leak and an EBF. Percutaneous interventions, e.g., liver biopsy, percutaneous transhepatic cholangiography (PTC), percutaneous transhepatic biliary drainage (PTBD), radio-frequency ablation (RFA) may also result in bile leak and EBF. Spontaneous EBF through a previous surgical scar as a result of a complication of acute cholecystitis and gall bladder perforation is rare. Similarly, traumatic EBF caused by abdominal trauma (more frequently penetrating) is also uncommon; most traumatic biliary injuries are associated with liver, pancreas, or duodenal injuries. A pure biliary fistula does not cause much damage to the skin but a mixed biliary-intestinal/biliary-pancreatic fistula can cause much damage to the skin which needs to be protected.

Cholecystostomy, surgical or percutaneous, most commonly performed for the management of complicated acute cholecystitis, e.g., empyema, is a surgically (or radiologically) created controlled EBF. This fistula will persist if the cystic duct is blocked or if there is common bile duct (CBD) obstruction, e.g., by a stone. If there is no distal obstruction, the fistula should close spontaneously after the tube is removed. Cholecystectomy can be performed later, after 6–8 weeks.

T-tube choledochostomy after a CBD exploration, e.g., for CBD stones is also a surgically created controlled EBF. The T-tube may be clamped

Also see Invited Commentary on Consequences of Bile Duct Injury—External Biliary Fistula by Irving S Benjamin (pp 132–133)

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after a T-tube cholangiogram shows no contrast leak, no obstruction at the lower end of the CBD and free passage of contrast into the duodenum. The T-tube should be removed after 2–3 weeks of placement once a firm tract has formed. Using isotope hepato-biliary scintigraphy, we have shown that earlier (<2 weeks) removal of the T-tube is associated with a higher incidence of extrabiliary isotope leak, indicating subclinical bile leak [2]. A transient EBF may occur after removal of the T-tube—it closes spontaneously in 2–3 days. If the fistula persists after removal of the T-tube, distal obstruction, most commonly due to a residual CBD stone, should be suspected. Endoscopic naso-biliary drainage (ENBD) and PTBD are also controlled EBF.

11.1 Post-Cholecystectomy EBF

Post-cholecystectomy EBF is bile leaking through the surgically placed drain (Fig. 11.1) or the abdominal wound following a BDI at cholecystectomy.

11.2 Controlled EBF

A controlled EBF is one with bile drainage directly from the biliary tract to the exterior (abdominal wall) without any intra-abdominal bile collection and no sepsis (peritonitis or cholangitis). Cholecystostomy, T-tube choledochostomy, and percutaneous transhepatic biliary drainage (PTBD) are examples of surgically or radiologically created controlled EBF. Bile in drain but no intra-peritoneal collection of bile following cholecystectomy is a controlled EBF. A controlled EBF can be managed expectantly. Patients with controlled EBF do not have peritoneal sepsis but they may have recurrent attacks of cholangitis. The Academic Medical Center (AMC), Amsterdam, Netherlands group recommends a naso-gastric tube or percutaneous gastric catheter to refeed bile. Bile refeeding reduces the chances of metabolic and nutritional consequences of prolonged bile loss; it also reduces endotoxemia and improves renal function. Bile refeeding also decreases postoperative complications specific to obstructive jaundice [3].

Fig. 11.1 Bile in drain after cholecystectomy—external biliary fistula

11.3 Uncontrolled EBF

EBF with intraperitoneal bile leak and sepsis in the form of intra-abdominal collection (localized or generalized) in the form of biloma (Fig. 11.2), bile peritonitis or bile ascites is defined as uncontrolled EBF. Lee et al. [4] reported 154 patients with undrained bile collections managed between 1990 and 1999—as many as 21% had serious complications including systemic sepsis and multiple organ failure. Symptoms and signs were subtle in a large majority of patients resulting in delay in diagnosis. An uncontrolled EBF may cause sepsis and needs intervention. An uncontrolled EBF will not close on its own. Every attempt must be made to convert an uncontrolled EBF into a controlled EBF by

1. Draining the intra-abdominal collection—this can be done by image (US or CT) guided percutaneous catheter drainage (PCD), even multiple (Fig. 11.3), in most cases; if facilities/expertise for percutaneous intervention are not available, a relaparoscopy may be performed to drain the intra-abdominal collections (bilomas) in order to control an uncontrolled

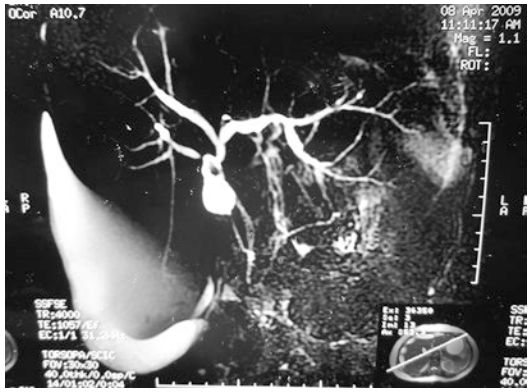

Fig. 11.2 Bile leak and biloma – uncontrolled biliary fistula

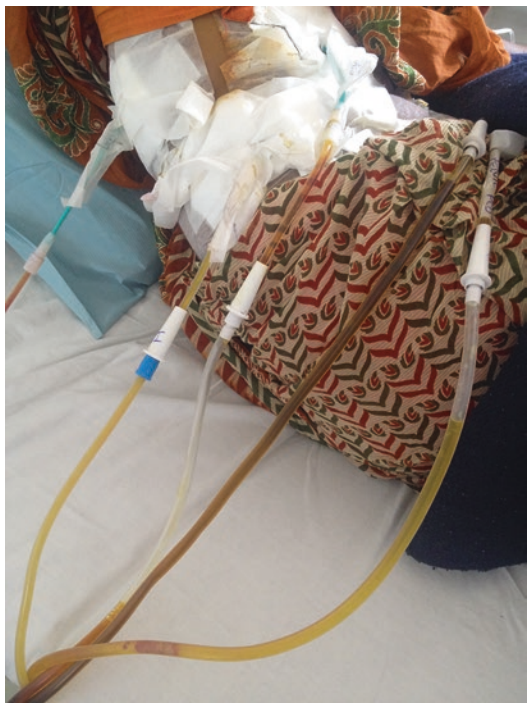

Fig. 11.3 Multiple percutaneous catheters (and one intercostal chest tube to drain pleural effusion) required to drain bilomas

EBF. Relaparoscopy is technically easy in the early postoperative period than after a few days when adhesions would have formed. At relaparoscopy, an obvious cystic duct blow out may be treated by reclipping the cystic duct but only if a good (long) clippable cystic duct stump is available. Patients with multiple, multi-septate or multi-loculated collections and those with solid necrotic debris in the collection may also require surgical intervention, i.e., laparoscopy or laparotomy.

2. Draining the bile duct to decrease/control the ongoing bile leak—usually by performing endoscopic papillotomy and placing an endoscopic stent (Fig. 11.4) /ENBD or by PTBD (Fig. 11.5). Surgical drainage of the bile duct, i.e., T-tube placement is very rarely required.

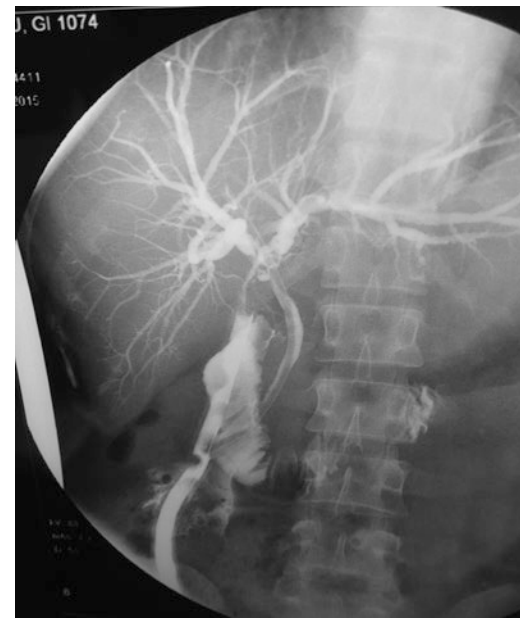

Fig. 11.4 Endoscopic retrograde cholangiography (ERC) shows bile leak—stent has been placed to decrease/control the ongoing bile leak

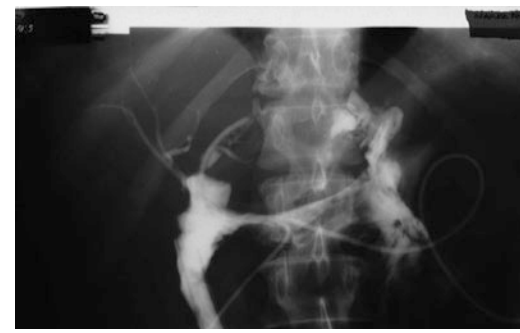

Fig. 11.5 Percutaneous transhepatic biliary drainage (PTBD) to decrease/control the ongoing bile leak in the absence of biliary ductal continuity

Repair of any kind should not be attempted in presence of an uncontrolled EBF because inflamed tissues are likely to be edematous and friable and sutures are likely to cut through resulting in anastomotic leak which is the most important risk factor for recurrent stricture.

11.4 Consequences of EBF

Patients with prolonged high volume EBF, even if controlled, may suffer from consequences of bile loss which result in chronic dehydration, decreased plasma volume, and low output renal failure. Chloride loss causes metabolic acidosis. Protein and calorie malnutrition and (fat-soluble) vitamin deficiency (especially that of vitamin K, causing coagulopathy) may supervene. Recurrent attacks of cholangitis may also occur while waiting for the fistula to close.

In patients with EBF following cholecystectomy, it is important to demonstrate whether the fistulous output is pure bile or bile mixed with intestinal contents which indicates a duodenal injury. Bile alone does not usually cause skin excoriation. Bile causes skin excoriation only when it is mixed with enteric (duodenal) contents. Presence of skin excoriation in a patient with EBF should, therefore, raise the suspicion of a duodenal injury. This can very easily be confirmed or ruled out by oral administration of saline mixed with a color dye (methylene blue or gentian violet) which shows in the drain or the wound after a few minutes or hours if a duodenal fistula is present.

11.5 Fistulogram (Also Called Tubogram)

In patients with a controlled and mature (>2 weeks) EBF, fistulogram can be performed through the drain or through a tube introduced in the fistulous tract—this may delineate the proximal biliary tract and provide a cholangiogram. It may also demonstrate the presence of biliary ductal continuity (Fig. 11.6) leading to endoscopic intervention (stent or ENBD) to decrease bile loss and hasten fistula closure. Fistulogram should be performed after about 2–3 weeks of BDI when a firm fistulous tract has formed and matured; if performed earlier, it may result in free leak of the contrast into the peritoneal cavity.

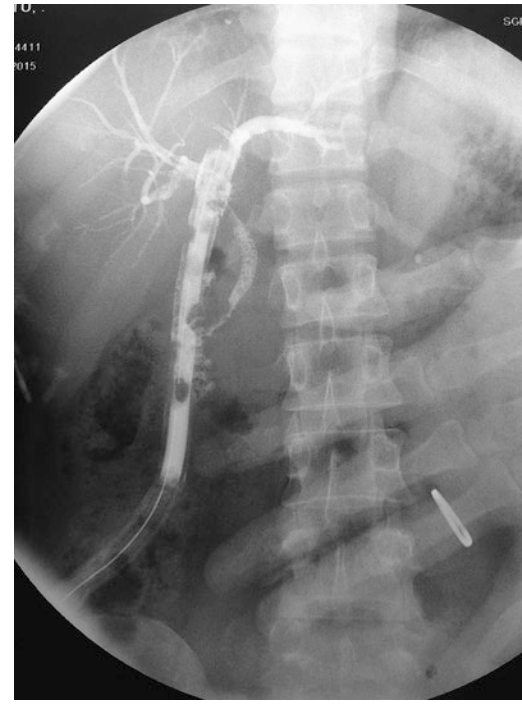

Fig. 11.6 Fistulogram through a mature fistulous track may delineate the proximal bile ducts and show biliary ductal continuity

11.6 Closure of EBF

In our experience, 74% of EBFs following cholecystectomy closed and 79% of patients with EBF developed a BBS. As many as 84% of EBF associated with an incomplete/partial/lateral BDI will close; some of these patients will develop a BBS and require repair while others remain asymptomatic with normal LFTs and no proximal intrahepatic biliary radicle dilatation (IHBRD) on follow-up. However, only 57% of EBFs associated with a complete BDI will close. Obviously, all patients with a complete BDI will require repair [5]. Mishra et al. [6] reported 46 patients with EBF—fistula closed in 31 patients after 60 (range 5–240) days. EBF from an injured isolated duct (not in continuity with the main bile duct) will not respond to endoscopic stenting and will require PTBD for its control.

EBF following minor injuries, e.g., cystic duct leak, duct of Luschka injury, and partial injury close early; those following injury to an isolated duct or complete injury take longer to close or may not close at all.

Prof Henri Bismuth (personal communication) says that all EBF, including those with complete BDI, will close and form a BBS, if one is willing to wait long enough. The Author (VKK) feels that a period of waiting of 3–6 months is good enough to show whether a fistula is likely to close or not. If an EBF does not close or does not show a trend towards closing in 3–6 months, repair of the BDI may have to be done with an ongoing EBF even though a BBS has not formed. Prolonged waiting carries the risk of recurrent cholangitis and secondary biliary cirrhosis (SBC).

Bansal et al. [7] operated upon 138 patients with BBS between 2005 and 2011; 26 (18%) had an ongoing EBF at the time of repair.

BBS with an ongoing EBF is sometimes called a wet stricture (cf. dry stricture, without an EBF). In patients with a persistent EBF, the bile ducts remain undilated (Fig. 11.7). Repair in presence of undilated ducts is technically difficult and chances of failure are higher. The only advantage is that the fistulous tract leads to the proximal bile ducts resulting in their easier identification (Fig. 11.8).

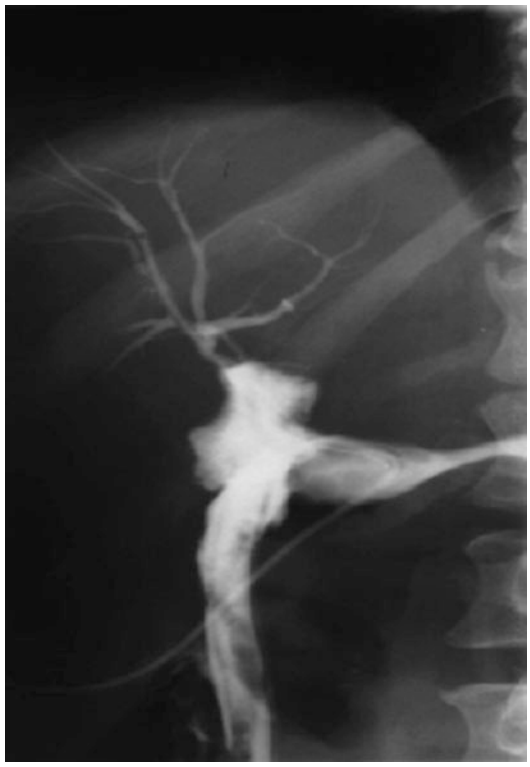

Fig. 11.7 An ongoing external biliary fistula (EBF) does not allow proximal bile ducts to dilate and makes repair technically difficult

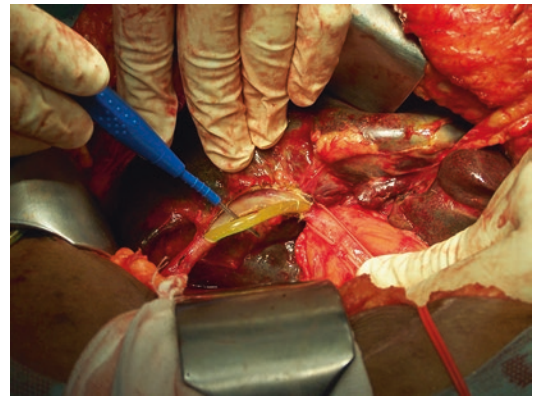

Fig. 11.8 The fistulous tract usually leads to the proximal bile ducts

11.7 Internal Fistula

Some patients with BBS may be anicteric in spite of a complete transection of the CBD as demonstrated on cholangiogram because of formation of an internal (biliary—enteric) fistula between the CBD proximal to the BBS and the duodenum (Fig. 11.9). The fistula is formed by erosion of a biloma, which is communicating with the CBD, into the duodenum. Rarely, an internal fistula may occur into the colon or stomach. The internal fistula predisposes the patient to recurrent attacks of cholangitis. Also, it does not allow the proximal bile ducts to dilate.

Rarely, a suprahepatic/subphrenic biloma or abscess may erode through the diaphragm into the pleural cavity and then into the lung/bronchial tree resulting in bilio-pleural or bilio-bronchial fistula [8] presenting as bilious pleural effusion or bilioptysis (bile stained sputum).

A bilio-venous fistula between an obstructed intrahepatic bile duct and a tributary of a hepatic vein following a percutaneous transhepatic intervention, e.g., livery biopsy, PTC, PTBD, TIPSS, etc. causes bilhemia—liver function tests (LFT), reveal very high serum direct bilirubin with

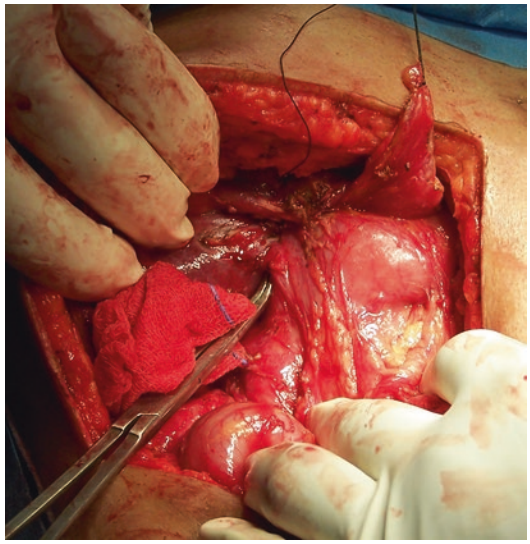

Fig. 11.9 Internal (bilio-duodenal) fistula—the patient may be anicteric even in presence of complete transection of the common bile duct

normal liver enzymes (ALT/AST). Relief of biliary obstruction usually resolves the condition because normal pressure in the bile ducts is less than the systemic venous pressure. If it persists, the internal fistula can be obliterated percutaneously using glue, sclerosants, or plug.

Bile leak following BDI leads to an EBF. The principle aim of management of EBF is to convert an uncontrolled EBF to a controlled EBF by draining all bile collections. In patients with intact biliary ductal continuity, endoscopic stenting decreases/controls ongoing bile leak. In some patients with an incomplete/partial BDI, the fistula closes and biliary ductal system remains normal; others may form a BBS. EBF is less likely to close in presence of a complete BDI; all these patients will require a repair in the form of a Roux-en-Y hepatico-jejunostomy.

Invited Commentary on Consequences of Bile Duct Injury—External Biliary Fistula

Irving Benjamin

Cholecystectomy remains the commonest elective abdominal operation, and the vast majority are now performed laparoscopically. Injury to the biliary tree remains the most feared complication, and as hospital stay has progressively reduced, with many patients now discharged on the day of surgery, such cases often go unrecognized at the time of the procedure. Most will present 1–5 days after discharge with signs of complete or incomplete biliary obstruction, or with an intra-abdominal bile collection. Spontaneous drainage of a biliary leak through the access ports of a laparoscopic operation is relatively uncommon, and an external biliary fistula more frequently follows radiological drainage of a bile collection. This occurrence will of course immediately raise alarm bells for the unfortunate surgeon, who will fear a significant ductal injury until proven otherwise. Fortunately, the majority prove to be due to a leak from the cystic duct stump, due to a technically inadequate closure or to severe inflammation at the point of application of the clip, resulting in subsequent necrosis or slippage [9].

The management objectives in the case of a cystic duct leak are straightforward: firstly to remove any collection of (almost always infected) bile from the peritoneal cavity, and secondly to prevent further biliary leakage from the open cystic duct. “Turning off the tap” can be achieved in several ways. In a series of 58 cases reported by Wise et al. [10], management included ERCP and ductal decompression in 27 patients, percutaneous drainage in 13, open laparotomy in 14, and laparoscopy in three. Sharma and Bird [11] reported the results of 46 patients who underwent ERCP for a biliary leak, and 32 of those had simple cystic duct stump leaks. Endoscopic treatment, the least invasive option, was successful, with stenting in the majority.

A more serious situation arises when the fistula does originate from damage to the extrahepatic biliary tree. The dilemma for the surgeon is now to determine when the issue is a simple leak and when it will prove to be a problem fistula. The key to this distinction lies in precise anatomical definition, either by invasive means or, as the technology has progressively improved, increasingly by high definition computed tomographic scanning and magnetic resonance cholangiog-

raphy (MRC). The principles of management remain fundamentally simple: ensuring drainage of any collection which could be a source of systemic sepsis, converting an uncontrolled into a controlled fistula, and detection of any ongoing outflow obstruction. In the absence of distal obstruction, the majority of controlled external biliary fistulae will close, as reported by Prof Kapoor in this chapter.

The issue of timing of intervention requires experienced judgment. We reported 12 patients who presented with an external biliary fistula in a series of 123 patients with post-cholecystectomy strictures managed at the Hammersmith Hospital, London [12]. Distal obstruction was a feature in five patients and required formal repair, since there was little prospect of spontaneous closure, as Prof Kapoor has also noted in his report [5]. In 3 out of 7 patients in our series, the fistula closed without complication at 5–9 weeks and no further surgery was required. The remaining four patients required delayed operation, having subsequently developed cholangitis or jaundice, underlining the importance of continued surveillance in the most difficult cases. As long as the general condition of the patient remains good, and there is no distal obstruction and no ongoing sepsis, prolonged observation of a controlled external biliary fistula will commonly pay dividends.

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Consequences of Bile Duct Injury: Benign Biliary Stricture

12

Vinay K. Kapoor

NOTE *While iatrogenic (usually post-cholecystectomy) biliary stricture is the commonest benign biliary stricture, there are other causes also of a benign biliary stricture. In this monograph, however, the Author (VKK) has used the term benign biliary stricture (BBS) as synonymous with iatrogenic post-cholecystectomy benign biliary stricture.*

12.1 Etiology

A biliary stricture may be malignant or benign.

Malignant biliary strictures are caused by cholangiocarcinoma, gallbladder cancer, and pancreatic and periampullary cancers. On cholangiography, malignant biliary strictures are irregular and asymmetric with shouldering (cf. benign biliary strictures which are usually smooth, symmetric, and tapering). Sometimes, it may be difficult to differentiate between a post-cholecystectomy benign biliary stricture (BBS) and a malignant biliary stricture, especially hilar cholangiocarcinoma and gallbladder cancer.

Even a post-cholecystectomy biliary stricture may not always be benign—it may be a malignant biliary stricture due to a missed gallbladder cancer or cholangiocarcinoma, the gallstones for which cholecystectomy was done were probably incidental and either the gallbladder was not subjected to histopathological examination because it looked grossly normal or an early gallbladder cancer was missed even on routine histopathological examination which includes only few (usually three) sections from the fundus, body, and neck of the gallbladder. Pre-cholecystectomy evidence of biliary obstruction (raised serum bilirubin, elevated alkaline phosphatase, ALP, and gamma-glutamyl transpeptidase, GGTP, and intrahepatic biliary radical dilatation, IHBRD, on ultrasonography, US), uneventful (with no bile leak) post-cholecystectomy course, very high serum bilirubin level, and high (proximal) biliary obstruction should raise the suspicion of a missed gallbladder cancer (Fig. 12.1) as the cause of a post-cholecystectomy biliary stricture [1].

Biliary strictures can be iatrogenic. Almost all iatrogenic biliary strictures are caused by surgical trauma. Rarely, a biliary stricture may follow percutaneous or radiological intervention, e.g., liver biopsy, percutaneous transhepatic cholangiography (PTC), percutaneous transhepatic biliary catheterization (PTBC), percutaneous transhepatic biliary drainage (PTBD), percutaneous transhepatic radio-frequency ablation (RFA) for liver tumor, transjugular intrahepatic porta-systemic

Also see Invited Commentary on Consequences of Bile Duct Injury—Benign Biliary Stricture by TM van Gulik (pp 144–146)

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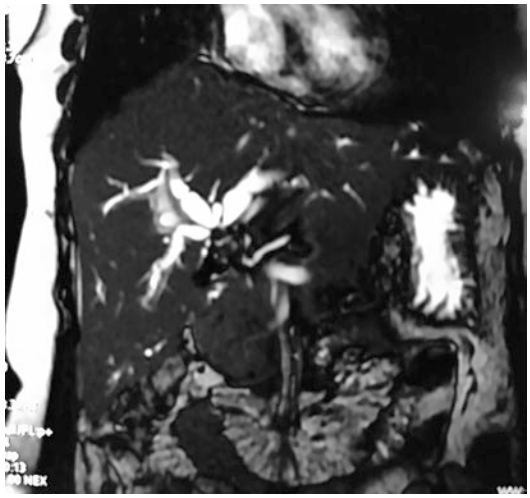

Fig. 12.1 Missed gallbladder cancer with local recurrence may sometimes look like a post-cholecystectomy high (proximal) benign biliary stricture

shunt (TIPSS), etc.; these biliary strictures are usually intrahepatic. Endoscopic interventions on the common bile duct (CBD), e.g., papillotomy, stone extraction, etc., and on the duodenum, e.g., sclerosant injection for bleeding peptic ulcer, may cause a low biliary stricture. Biliary stricture caused by accidental trauma is extremely rare.

Most surgical iatrogenic biliary strictures are a result of a bile duct injury (BDI) caused during cholecystectomy. The BDI and its consequent bile leak cause intense inflammatory reaction in and around the common bile duct; healing leads to formation of a fibrotic scar resulting in narrowing of the common bile duct—benign biliary stricture (BBS). During a median follow-up of 15 (range 6–98) months, 153 (71%) of 214 patients with BDI developed BBS and 61 (29%) had normal biliary system (normal LFT, no IHBRD on US, normal hepato-biliary isotope scintigraphy and/or normal cholangiogram); complete BDI, high fistula output, and persistent (>4 weeks) fistula were predictors for formation of a BBS [2]. All complete BDIs and some partial BDIs (both with and without external biliary fistula) lead to the formation of a BBS because of healing by fibrosis. This takes about 4–6 weeks of time but may occur earlier in a clipped or ligated duct. BBS may not form at all in a patient with persistent external biliary fistula after a complete BDI. An ischemic BBS can form even without a BDI during cholecystectomy due to damage to the blood supply of the common bile duct caused by circumferential mobilization of the common bile duct over a length (the common bile duct was misidentified as the cystic duct and mobilized; the mistake was then identified and further dissection abandoned). An ischemic injury to the common bile duct may result in a stricture after a long time (months or even years) without bile leak in the postoperative period. A thermal (cautery) injury to the common bile duct may also result in an ischemic stricture without a bile leak. BDI and BBS are more common after laparoscopy than after open cholecystectomy. Common bile duct exploration (choledochotomy) can also result in a biliary stricture. Biliary stricture following common bile duct exploration is a low stricture and is easy to repair. Other operations during which a BDI can occur are hepatectomy, gastrectomy, operations for bleeding duodenal ulcer, and head coring (Frey's procedure) for chronic pancreatitis. Bilio-enteric anastomosis performed during other procedures, e.g., pancreato-duodenectomy, choledochal cyst excision, excision of cholangiocarcinoma, liver transplant, etc., may also stricture and result in a benign biliary stricture.

Other causes of benign biliary stricture are inflammatory (recurrent cholangitis due to common bile duct stones), Mirizzi's syndrome, chronic pancreatitis, primary sclerosing cholangitis (PSC), recurrent pyogenic cholangitis (RPC), oriental (Asiatic) cholangio-hepatitis (OCH), penetrating chronic duodenal ulcer, etc. Recurrent cholangitis caused by common bile duct stones may lead to inflammatory ulceration and healing by fibrosis leading on to formation of a stricture—usually in the lower end of the common bile duct. They can be treated by endoscopic removal of stones and balloon dilatation and prolonged stenting of the stricture. Mirizzi's syndrome is biliary obstruction caused by a large stone impacted in the gallbladder neck or Hartman's pouch. At a later stage, a fistula may form between the gallbladder and the common bile duct (cholecysto-choledochal fistula) and the stone may then actually come to lie within the common bile duct. The classical US findings show IHBRD and dilated proximal common bile duct but normal (undilated) distal com-

mon bile duct with a large stone in the gallbladder neck. The stricture in chronic pancreatitis involves lower (distal) intrapancreatic common bile duct—the stricture is usually long, smooth, and concentric. Primary sclerosing cholangitis (PSC), a chronic progressive cholestatic disease, is an uncommon cause of benign biliary stricture; it is more common in men (2:1). Clinical presentation is with recurrent cholangitis (fever and jaundice). Cholangiogram shows multiple short strictures with areas of dilatation in between (beaded appearance) in the intrahepatic and extrahepatic bile ducts. Diagnosis can be confirmed by a liver biopsy. Half to two-thirds of patients with primary sclerosing cholangitis also have ulcerative colitis (but only 5% of patients with ulcerative colitis develop primary sclerosing cholangitis). Primary sclerosing cholangitis usually leads to liver failure and may cause cholangiocarcinoma in 10–15% of cases. Ursodeoxycholic acid (UDCA) may help to relieve symptoms. Percutaneous dilatation of strictures may be required. Liver transplant is the only curative treatment. Tuberculous involvement of peri-choledochal (e.g., hilar, hepato-duodenal ligament, retro-duodenal, and retro-pancreatic) lymph nodes may cause extrinsic benign biliary obstruction; rarely, tuberculosis may involve the bile duct itself and cause a benign biliary stricture. Radiotherapy to the right upper abdomen for a cancer may cause a benign biliary stricture; endoscopic dilation and stenting is the treatment of choice.

12.2 Clinical Presentation

A post-cholecystectomy BBS invariably presents as jaundice. The commonest cause of post-cholecystectomy jaundice, however, is not a BDI or BBS but residual or retained common bile duct stone(s)—patients with CBD stones usually have pain and jaundice (cf. BBS where jaundice is usually painless). If a common bile duct stone is suspected as the cause of post-cholecystectomy bile leak (due to cystic duct blow out) or jaundice, endoscopic retrograde cholangiography (ERC) is the investigation of choice as it offers therapeutic opportunity also in the form of endoscopic papillotomy and stone extraction.

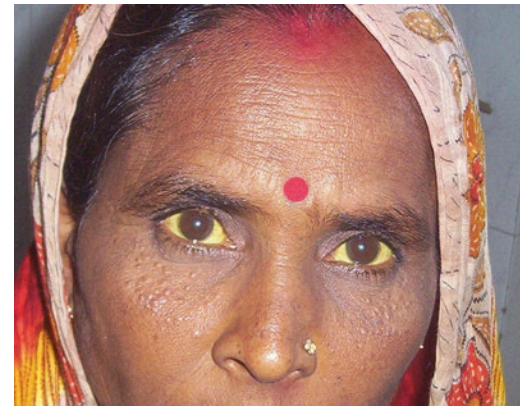

Fig. 12.2 The clinical presentation of post-cholecystectomy benign biliary stricture is as surgical obstructive jaundice with recurrent attacks of cholangitis

Most patients with BBS present with jaundice (Fig. 12.2) and recurrent episodes of cholangitis (high grade fever with chills and rigors); pruritus is also commonly present. Patients may have systemic sepsis as a result of multiple cholangiolytic abscesses in the liver. An internal fistula may form between the proximal bile duct and a viscus (most commonly duodenum, rarely colon). Jaundice may not be present in patients with an internal bilio-enteric (choledocho-duodenal or colonic) fistula or those with an isolated segmental/sectoral duct injury/stricture. Some of these patients (even in presence of a complete BDI) may be largely asymptomatic. Occlusion of an isolated subsegmental, segmental, or even sectoral duct may not necessarily result in jaundice or biliary sepsis in the form of cholangitis but may instead lead to an asymptomatic atrophy of a part of the liver, detected only on imaging (US, CT, MRI, or isotope hepato-biliary scintigraphy).

12.3 Investigations

The aims of investigations before repair of a BBS are

1. To exclude sepsis (biliary or peritoneal)—repair should be deferred if sepsis is present; repair should be attempted at least 2 weeks after the control of sepsis

2. To evaluate liver functions (or dysfunction) including coagulation profile which, if deranged, needs to be corrected (with vitamin K and/or fresh frozen plasma)
3. To delineate the Bismuth type of the BBS
4. To evaluate liver anatomy (atrophy—hypertrophy complex)
5. To detect associated vascular injury, if any

A complete hemogram including hemoglobin (Hb), total leucocyte count (TLC) and differential leucocyte count (DLC), and platelet count should be obtained. Patients with recurrent/chronic sepsis may be anemic; leucocytosis (especially polymorphonuclear) indicates sepsis.

Complete liver function tests (LFT) viz. serum bilirubin (total/direct), liver enzymes, i.e., aspartate transaminase/ alanine transaminase (AST/ALT), alkaline phosphatase (ALP), gamma-glutamyl transpeptidase (GGTP) and synthetic functions of liver (serum protein/albumin) and prothrombin time/international normalized ratio (PT/INR) should be obtained.

Elevated counts (TLC, DLC) and liver enzymes (AST/ALT) suggest cholangitis.

Patients with surgical obstructive jaundice due to BBS may have renal dysfunction. Renal function tests (RFTs)—blood urea, blood urea nitrogen (BUN), serum creatinine, and serum electrolytes—should be obtained in all patients with BBS.

12.4 US (Ultrasonography)

US is a very useful investigation in the management of BBS (as also acute BDI).

In patients with BBS, US demonstrates IHBRD, intrahepatic stones and sludge, delineates the level of biliary obstruction and the type of the stricture i.e. patency of the biliary ductal confluence and the length of the common hepatic duct stump (US alone, however, is not reliable to type the stricture; cholangiogram is a must before repair). US also evaluates liver parenchyma (echotexture), liver volume (for atrophy - hypertrophy complex), cholangiolytic abscess, splenomegaly and collaterals (due to portal hypertension).

US demonstrates the echotexture of the liver parenchyma but this evaluation is very subjective; elastography can evaluate the liver echotexture more objectively.

Doppler US is a useful noninvasive investigation for evaluation of associated vascular (hepatic artery and portal vein) injury and detection of a pseudoaneurysm in a patient with a BDI and BBS. It should be obtained in all cases. Doppler, however, may be falsely negative (arterial flow seen) even in presence of an arterial injury because of presence of collaterals.

CT scan, of use in patients with acute BDI to detect biloma, is usually not required in patients with BBS unless there is suspicion of malignancy e.g., cholangiocarcinoma or gall bladder cancer (vide supra). If done, CT may show IHBRD, evaluates liver parenchyma for volume (atrophy—hypertrophy complex) and cholangiolytic abscess.

12.5 Cholangiogram

A road map in the form of a good and complete cholangiogram visualizing all intrahepatic segmental ducts is a *MUST* in every patient before attempting the repair of a BBS. A cholangiogram is a must before repair of the BBS, irrespective of the timing of repair. In case of an immediate (intraoperative) repair, it has to be intraoperative, i.e., peroperative cholangiography (POC); for later (postoperative) repair, the preferred cholangiogram is magnetic resonance cholangiography (MRC). As many as 27 (96%) of 28 repairs performed without preoperative cholangiogram were unsuccessful; repair was unsuccessful in 69% cases in which the cholangiogram was done but it was incomplete; complete cholangiogram resulted in a successful repair in 16 (84%) of 19 patients [3].

Cholangiogram is required to delineate the biliary ductal anatomy and to detect the site (in relation to the biliary ductal confluence), nature (partial or complete), and extent (loss of segment) of the BDI/BBS. This is important to ensure that all ducts are drained by the bilio-enteric anastomosis and to avoid missing an isolated segmental or sectoral duct.

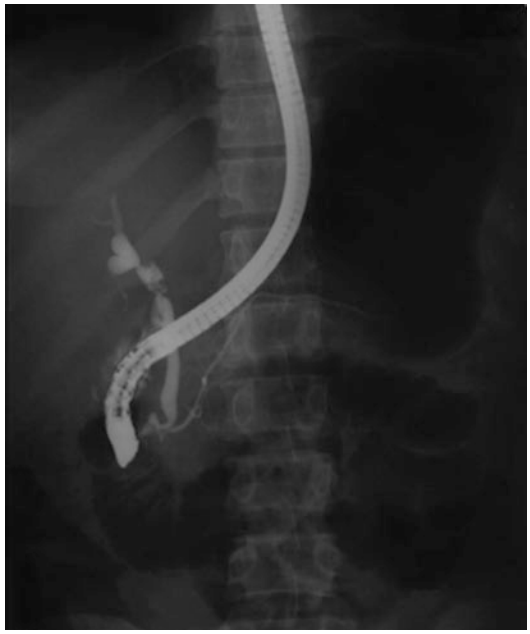

Fig. 12.3 Endoscopic retrograde cholangiogram (ERC) does not provide good delineation of proximal bile ducts and is not preferred in the work up of post-cholecystectomy benign biliary stricture

ERC which is of great value in the management of acute BDI is not of much use in BBS as it may not delineate the proximal bile ducts well (Fig. 12.3) or at all (in case of ligation or transection of the common bile duct). Moreover, injection of contrast into the obstructed undrained proximal biliary system beyond the stricture may introduce infection and precipitate cholangitis. ERC does not delineate an isolated ductal stricture as it is disconnected from the common bile duct. ERC and therapeutic endoscopic intervention are associated with complications, e.g., bleeding, cholangitis, pancreatitis, perforation.

Cholangiogram can be obtained through the fistulous tract also i.e., fistulogram but only after it has matured.

12.6 Liver Biopsy

Preoperative liver biopsy is not indicated in patients with BBS except to document secondary biliary cirrhosis (SBC) when there is gross liver dysfunction/failure and/or portal hypertension and liver transplant is being considered as an option.

12.7 PTC (Percutaneous Transhepatic Cholangiography)

Percutaneous transhepatic cholangiography (PTC) used to be the gold standard for delineation of proximal biliary ductal anatomy before repair of BBS (Fig. 12.4). Double (bilateral) punctures may be required to opacify both right and left hepatic ducts in patients with Bismuth type IV BBS where the primary biliary ductal confluence is involved. Multiple punctures may be required in patients where the Bismuth type IV BBS involves the right secondary biliary ductal confluence (of right anterior and posterior sectoral ducts) also. An isolated strictured bile duct, e.g., segmental or sectoral, may still be missed. If PTC does not show all the segmental or sectoral ducts on the right side, a separate puncture of the isolated segmental/sectoral duct in a Bismuth type V BBS may have to be done so that it shows all the intrahepatic bile ducts. Multiple punctures were required in about one-third of cases with BBS

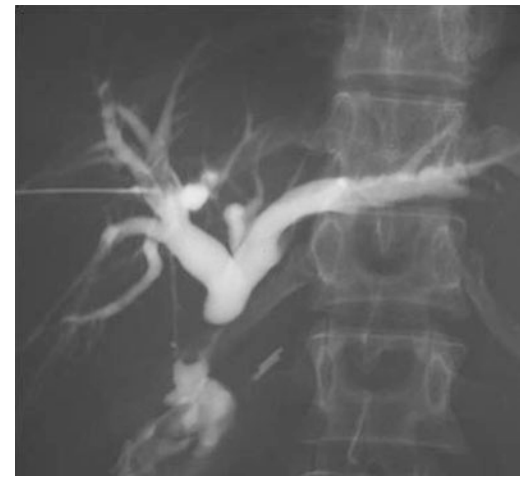

Fig. 12.4 Percutaneous transhepatic cholangiography (PTC) is invasive and involves radiation and is not preferred in the work up of post-cholecystectomy benign biliary stricture

[4]. PTC is preferably done a day before or on the morning of surgery, after correction of coagulopathy and under antibiotic cover. PTC is invasive, involves radiation, and is associated with complications, e.g., bleeding (intraperitoneal, intrahepatic parenchymal, and intrabiliary—hemobilia) and bile leak (and peritonitis).

Because of availability of MRC (vide infra) today, PTC is not preferred. An immediate preoperative PTC may, however, be obtained (under antibiotic cover) in a patient who has already had PTBD done for uncontrolled cholangitis or a PTBC placed for intraoperative identification of bile ducts (vide infra). In an occasional case, a direct cholangiogram, i.e., PTC may throw a pleasant surprise by showing a patent biliary ductal confluence (Bismuth type III BBS) where MRC had previously shown absence of biliary ductal confluence (Bismuth type IV BBS).

12.8 CT Cholangiography

Thin-section spiral multi-detector CT after administration of intravenous cholangiographic agents can also produce a good cholangiogram.

12.9 MRC

The cholangiogram of choice today is magnetic resonance cholangiography (MRC) (Fig. 12.5). MRC has replaced PTC as the investigation of choice for the delineation of the biliary ductal anatomy before repair of a BBS as it is noninvasive and involves no radiation. MRC has the advantage over PTC for Bismuth type IV and V BBS because all the intrahepatic ducts (including isolated ducts) can be visualized (whereas double, or even multiple, punctures will be required in case of PTC).

MRC images are produced by the fluid (bile) in the bile ducts. MRC images may be shadowed by a large biloma (Fig. 12.6) which should first be drained (usually by a percutaneous catheter) before MR imaging is done. In presence of an ongoing external or internal biliary fistula, the bile ducts are collapsed and may not be well visualized on MRC. MRC may be "false-high"

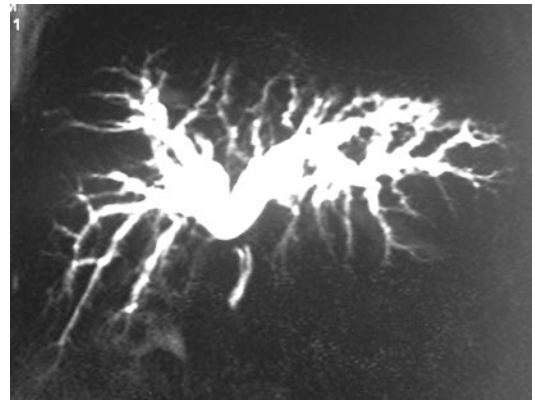

Fig. 12.5 Magnetic resonance cholangiogram (MRC) is the cholangiogram of choice for the work up of post-cholecystectomy benign biliary stricture

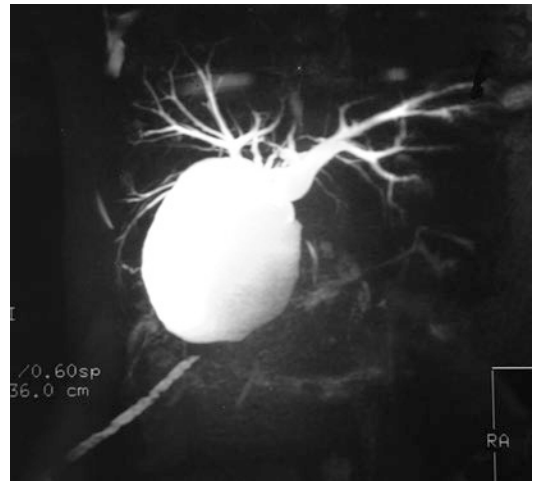

Fig. 12.6 A large biloma may obscure the delineation of biliary ductal anatomy on magnetic resonance cholangiogram (MRC); it should be drained before MRC is done

for the Bismuth type of the BBS, e.g., a sludge ball in the common hepatic duct stump may result in a type I or II BBS look like type III (no common hepatic duct stump) and a sludge ball in the biliary ductal confluence may result in a type III (biliary ductal confluence patent) BBS look like type IV (biliary ductal confluence not patent, right and left hepatic ducts separated) (Fig. 12.7). MRC may be "false-low" for the type of the BBS, e.g., overlap of separated right and left hepatic ducts (Bismuth type IV BBS) may give a false impression of them joining (Bismuth type III BBS) or even presence of a

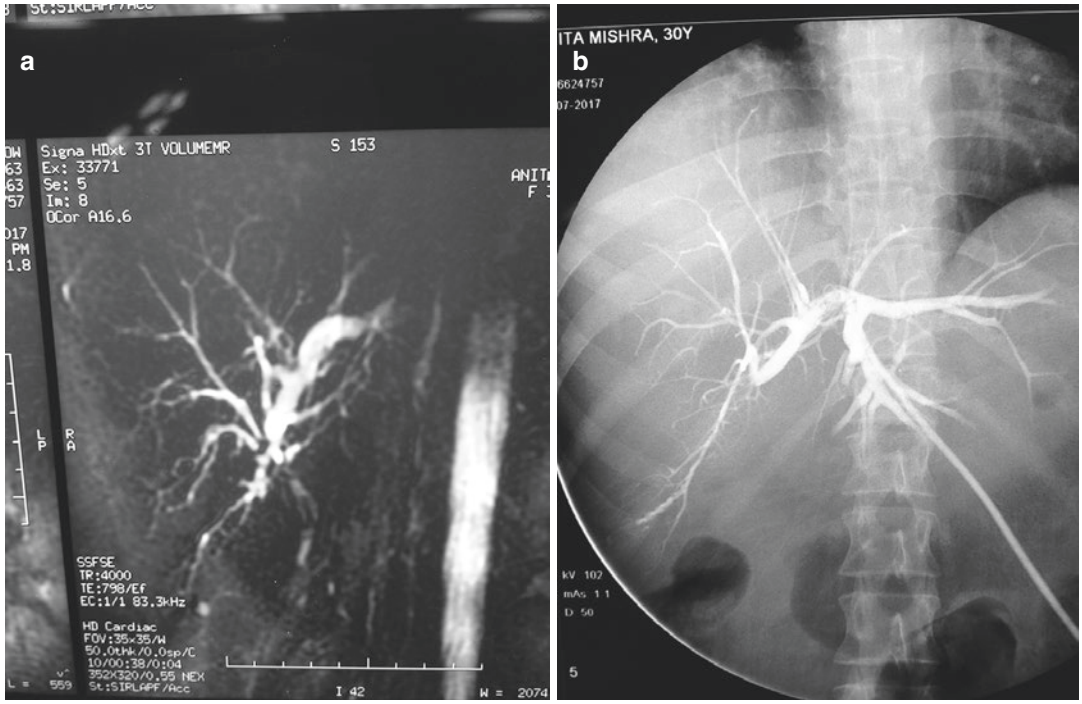

Fig. 12.7 False high level of biliary stricture on magnetic resonance cholangiogram (MRC) (**a**). MRC image shows an apparent high biliary stricture with four separate ducts; (**b**). Percutaneous transhepatic cholangiogram (PTC) through a left sided puncture in the same patient shows that the biliary ductal confluence is patent and the right biliary system is delineated

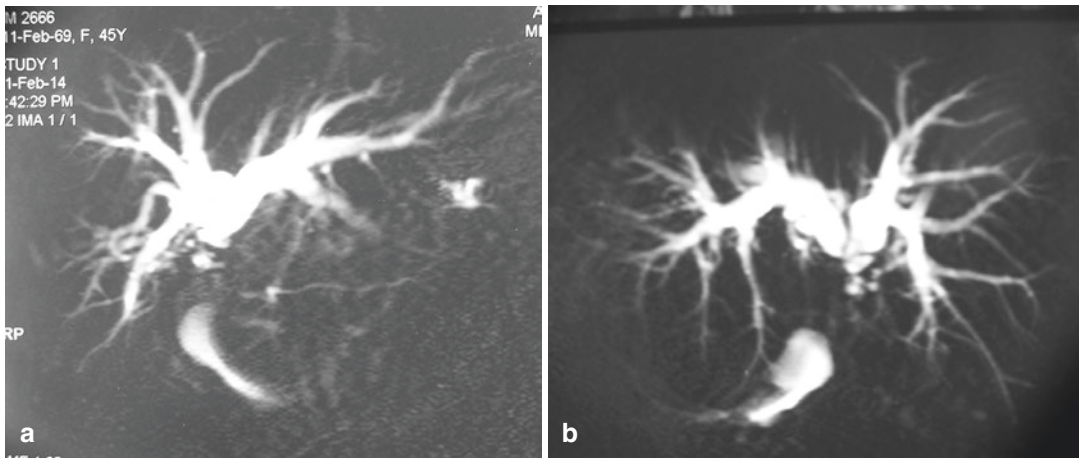

Fig. 12.8 False low level of biliary stricture on magnetic resonance cholangiogram (MRC) (**a**). MRC image shows an apparent Bismuth Type II benign biliary stricture due to overlap of right and left hepatic ducts; (**b**). rotation of MRC image shows that the right and left hepatic ducts are separate and the biliary stricture is actually Bismuth Type III

common hepatic duct stump (Bismuth type II or I BBS); this overlap can be removed by rotating the image to a different angle (Fig. 12.8). MRC can be combined with MR arteriography (MRA) (Fig. 12.9) to evaluate the hepatic artery and with MR porto-venography (MRPV) to evaluate the portal vein for any associated vascular injury.

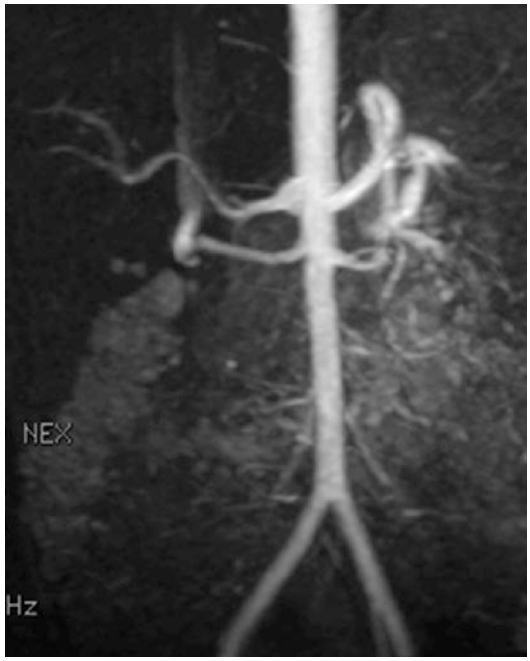

Fig. 12.9 Magnetic resonance angiography (MRA) can be combined with magnetic resonance cholangiogram (MRC) to delineate vascular anatomy and detect any associated vascular injury

A conciliation of the CT (liver volume and shape) with the cholangiogram (biliary ductal anatomy) can be done to ensure that all intrahepatic bile ducts have been visualized and are anastomosed during the repair and no intrahepatic bile duct is missed during repair.

12.10 Fistulography

In patients with persistent external biliary fistula, contrast may be injected through a tube (drain) placed in the fistulous tract to obtain a fistulocholangiogram. This, however, should be done after 2–3 weeks of BDI so that the fistulous tract has matured and the contrast does not spill into the free peritoneal cavity.

12.11 UGIE

Upper gastro-intestinal endoscopy (UGIE) should be performed in patients with long (>6 months) BDI—BBS interval to look for esophago-gastric varices and portal hypertensive gastropathy (PHG) because of portal hypertension as a result of secondary biliary cirrhosis (SBC) caused by prolonged biliary obstruction.

12.12 Angiography

Most groups now perform (and recommend) some angiography (usually MR angiography Fig. 12.9 along with MRC) to look for an associated vascular injury. It will also reveal a pseudoaneurysm (usually of the right hepatic artery) which, if present, should be angioembolized before repair of the BBS.

12.13 Preparation

Patients with surgical obstructive jaundice need correction of malnutrition (high calorie, high protein diet), anemia (packed red blood cells), dehydration (intravenous fluids to ensure good urine output and to prevent postoperative hepato-renal syndrome and renal failure), electrolyte imbalance, coagulopathy, and prophylactic antibiotics.

Vitamin K has to be administered and fresh frozen plasma (FFP) may have to be transfused before any intervention (endoscopic, percutaneous, or surgical) is performed in a patient with surgical obstructive jaundice and coagulopathy due to a BBS.

12.14 PTBD (Percutaneous Transhepatic Biliary Drainage)

NOTE *Percutaneous transhepatic biliary drainage (PTBD) is to be differentiated from percutaneous transhepatic biliary catheterization (PTBC) (vide infra).*

Jaundice, even when high, in a patient with BBS does not require PTBD before the stricture is repaired by a hepatico-jejunostomy (cf. jaundice in patients undergoing major liver resection or pancreato-duodenectomy where preoperative biliary drainage in the form of PTBD or endoscopic stenting may be done to bring the serum bilirubin down).

The Author (VKK) does not advocate routine preoperative PTBD before repair of a BBS. Preoperative PTBD may be required in some cases. PTBD (Fig. 12.10) is indicated in a patient with complete BDI (because in a patient with an incomplete BDI, biliary ductal continuity is present and endoscopic biliary drainage with a stent is preferable) for uncontrolled cholangitis not responding to parenteral antibiotics, severe coagulopathy, renal dysfunction, very high (say >20 mg) bilirubin, social reasons (patient not ready for surgery) and to buy time, e.g., to improve poor nutritional status. Another place for PTBD is in a patient with complete BDI to decrease the fistula output/hasten the closure of the external biliary fistula (EBF).

Preoperative biliary drainage results in the dilated proximal bile ducts to collapse, thus making the performance of the hepatico-jejunostomy technically difficult. It also increases the risk of introducing infection (cholangitis) in the obstructed biliary system and increases the risk of septic postoperative complications.

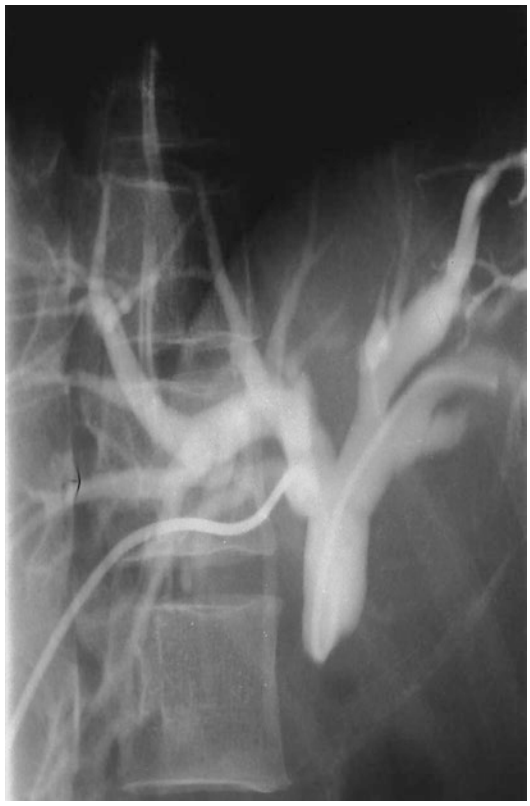

Fig. 12.10 Percutaneous transhepatic biliary drainage (PTBD) is not required before repair of a benign biliary stricture unless indicated for control of cholangitis

In case a PTBD has been performed, the same catheters can be used to obtain a direct percutaneous transhepatic cholangiogram (PTC) the day before/on the morning of operation. The catheter can also be advanced to the hilum for helping intraoperative identification of the bile ducts in case of a high/difficult BBS. It can also be used as transanastomotic stent after the anastomosis has been completed and to obtain a postoperative cholangiogram to evaluate the anastomosis.

12.15 PTBC (Percutaneous Transhepatic Biliary Catheterization)

Percutaneous transhepatic biliary catheterization (PTBC) is introduction of catheters into the intrahepatic bile ducts (usually right) under image (US or CT) guidance just before operation for easier intraoperative identification of the bile ducts in a high/difficult BBS. PTBC (placed immediately before operation for biliary ductal identification) should be differentiated from PTBD (placed few weeks before operation) to control cholangitis (vide supra).

The Johns Hopkins Hospital, Baltimore MD USA group places a preoperative transhepatic catheter into the biliary system in all cases before repair of a BBS. The catheter is advanced to the hilum of the liver (biliary ductal confluence) and then pushed out of the biliary system into the peritoneal cavity in the subhepatic space [5]. Strasberg et al. [6] described preoperative intubation in 23 patients with isolated right sided BBS. Buell et al. [7] also described placement of preoperative PTBC in all cases. The Academic Medical Center (AMC), Amsterdam Netherlands group reported placement of percutaneous transhepatic catheter for management of persistent bile leak or to drain bile in case of complete occlusion of the common bile duct in 73 (48%) out of 151 patients with BDI who underwent reconstructive surgery; they, however, did not differentiate between PTBD and PTBC [8]. The Author (VKK), however, differs from this approach and places a PTBC not in all

cases but in only high (Bismuth type IV) or difficult (Bismuth type V) BBS and does not push them out of the biliary system into the peritoneal cavity; the catheter is advanced only to the hilum of the liver (Fig. 12.11). In a very high BBS, if even right anterior and posterior sectoral ducts are separated from each other, two separate catheters may have to be placed—one in the right anterior and the other in the right posterior sectoral duct. These catheters help in intraoperative identification of the intrahepatic ducts, especially on the right side (the left hepatic duct, in most cases, has an extrahepatic course for some length and is easy to identify during surgery). During surgery, saline stained with a color dye, e.g., methylene blue or gentian violet, may be injected into the catheter and needle aspiration is done to identify the bile duct at the hepatic hilum. Intraoperative cholangiography may also be performed after injecting radiological contrast through the catheter.

The PTBC can also be used to perform a leak test during the operation after the anastomosis has been completed. This can be done in two ways—subhepatic area is filled with saline and air is injected through the PTBC to look for air bubbles; jejunal Roux limb can be soft clamped to further increase the intra-jejunal air pressure. Alternatively, dye stained saline can be injected through the PTBC and a fresh gauze placed around the anastomosis. The PTBC catheter may be left behind as a biliary drainage proximal to the biliary-enteric anastomosis or it can be used for guiding and railroading a transhepatic anastomotic stent across the biliary-enteric anastomosis.

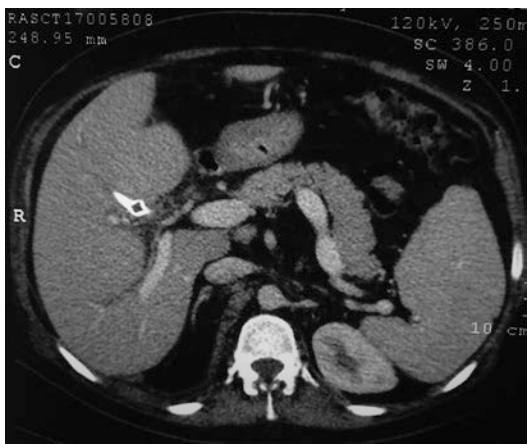

Fig. 12.11 Percutaneous transhepatic biliary catheter (PTBC) advanced up to the liver hilum – this will help in intraoperative identification of (right sided) bile ducts in a high/difficult biliary stricture

12.16 PTCS (Percutaneous Transhepatic Cholangioscopy)

Percutaneous transhepatic cholangioscopy (PTCS) is used, more frequently by the Japanese surgeons, for removal of calculi and sludge in the intrahepatic bile ducts in patients with high BBS. It also helps to obtain biopsy or brush cytology to rule out an intrahepatic or hilar cholangiocarcinoma, a differential diagnosis of a high BBS.

Percutaneous interventions, e.g., PTC, PTBD, PTBC, and PTCS are invasive and may result in complications such as bleed (intraperitoneal, intraparenchymal, and intrabiliary—hemobilia), bile leak into the peritoneal cavity, cholangitis, pneumothorax, and bilio-plural fistula (if a transpleural approach is used). Patients should be investigated for coagulopathy which, if present, should be corrected and antibiotic cover should be provided.

One of the consequences of a bile duct injury and external biliary fistula is a benign biliary stricture. Patients with benign biliary stricture present with jaundice and cholangitis. Benign biliary stricture requires repair in the form of a biliary-enteric anastomosis after preoperative cholangiography.

Invited Commentary on Consequences of Bile Duct Injury—Benign Biliary Stricture

Thomas M. van Gulik

Bile duct injury (BDI) remains a disastrous complication following cholecystectomy which seems to have increased in the era of laparoscopic

cholecystectomy. Initial management should be undertaken in a specialized center by a multidisciplinary team consisting of hepato-biliary surgeons, gastroenterological endoscopists, and interventional radiologists. Formation of a biliary stricture is a difficult complication with great impact on the quality of life requiring prompt treatment. Whereas early strictures usually result from direct surgical trauma to the bile duct(s), late strictures may be caused by associated vascular injuries predominantly to the hepatic artery and its branches.

Management plan is based on mapping of the biliary tree by (duplex) ultrasound and MRC. ERC is indicated only when a therapeutic measure is foreseen such as sphincterotomy or insertion of plastic stents, which in most cases efficiently takes care of cystic duct leakage or simple (partial) injuries of the bile duct wall. Percutaneous transhepatic cannulation and biliary drainage (PTBD) is used when continuity of the proximal and distal biliary ducts has been lost as with complete transection of the bile duct or when a disconnected part (often B6/7 i.e., right posterior sector) of the ductal system had been demonstrated. Usually, subsequent drainage is required to prevent cholangitis and/or biliary fistula. In case of major injuries such as with inadvertent resection of part of the bile duct, ERC and PTBD may fail to overcome the defect in the bile duct. Percutaneous-endoscopic rendezvous procedure is then an option before moving on to surgical repair.

Surgical reconstruction is undertaken when endoscopic and/or percutaneous methods have failed. These procedures may be complex and should be carried out by specialized hepato-biliary surgeons. The Roux-en-Y hepatico-jejunostomy (HJ) is the optimal technique whereas an end-to-end bile duct anastomosis may be considered as primary repair at the initial operation (provided the surgeon has sufficient experience). The timing of surgical reconstruction and its influence on long-term outcomes still is a matter of debate. Delay in surgical repair has been associated with a lower risk of postoperative complications compared to early repair. Delayed surgical repair allows for adequate sepsis control, restoration of vascular damage, and clinical improvement of the patient. Early repair, however, leads to shorter hospital stay and probably lower costs. We recommend an individualized approach taking into account the type of injury, biliary leakage, septic complications, and condition of the patient in the decision for early or delayed treatment.

A high, proximal anastomosis is recommended in surgical repair to prevent ischemia of the HJ anastomosis and the risk of anastomotic leakage and restricture formation. For intrahepatic BDI or complex vasculo-biliary injuries, a partial liver resection in conjunction with biliary reconstruction may be carried out. Although rarely necessary, postoperative morbidity of partial liver resection is considerable with mortality reported up to 18%.

Secondary, anastomotic stricture formation comprises a second order complication after surgical repair and has been reported in 10–20% of cases. The median time to stricture formation has been reported between 11 and 30 months, implying that patients require a long follow-up period after surgical repair with assessment of cholestatic parameters every 6 months for at least 3–5 years. Several factors have been reported to influence outcome after surgical repair. Vascular injury, level of injury, sepsis or peritonitis, postoperative bile leakage, and overall postoperative complications have been identified as risk factors for stricture formation. Long-term complications of anastomotic strictures are secondary biliary cirrhosis, portal hypertension, and end-stage liver disease (ESLD) ultimately requiring liver transplantation.

Due to the altered anatomy after HJ, the percutaneous transhepatic approach (PTBD) with balloon dilatation of the stricture at the HJ and internal drainage is the treatment of choice for recurrent (anastomotic) stricture. Overall success rates of 66–76% have been reported with low procedural morbidity. PTBD with balloon dilatation is, therefore, advised as the first step in the treatment of anastomotic strictures before reverting to surgical revision.

In summary, clinical outcomes of endoscopic, radiologic, and surgical treatment of BDI are

favorable with success rates reported around 90%. A step-up approach starting with initial endoscopic and/or percutaneous management [9], moving on to surgical treatment is recommended as decided by a multidisciplinary team in a referral center with expertise in BDI [10].

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13 Surgical Management of Benign Biliary Stricture: Hepatico-Jejunostomy

Vinay K. Kapoor

The first sentence of the chapter on Benign Biliary Strictures in Blumgart's Surgery of Liver, Biliary Tract, and Pancreas reads

> Benign biliary strictures are difficult management problems.

In various texts, benign biliary strictures have correctly been described as a surgical challenge and hepatico-jejunostomy as a technically challenging procedure.

NOTE: *The term benign biliary stricture (BBS) has been used in this monograph to mean iatrogenic post-cholecystectomy biliary stricture.*

13.1 Indications for Surgery

Surgical repair is the treatment of choice for benign biliary stricture (BBS). A BBS needs a durable (surgical) repair as most patients are young and have long years to live (cf. in malignant biliary strictures, where patients are usually older and the life expectancy is short. Non-surgical intervention is, therefore, an acceptable and even a preferable option).

The Author (VKK) strongly recommends against non-surgical definitive treatment (e.g., endoscopic or percutaneous dilatation and stenting) of BBS, except in patients who are at very high risk for surgery.

All BBS, including those which are asymptomatic (anicteric) due to the presence of an internal (biliary—enteric) fistula, should be repaired. An asymptomatic (no jaundice, no fever, and no cholangitis) stricture of an isolated subsegmental or segmental (and may be even a sectoral duct) may, however, be left alone for observation and may be followed up as it may result in asymptomatic atrophy of the corresponding liver parenchyma and may not require any intervention.

13.2 Bilio-Enteric Anastomosis (BEA)

An extrahepatic bilio-enteric anastomosis (BEA), also called bilio-digestive anastomosis (BDA), may be

(a) Choledocho-duodenostomy (CDD), performed sometimes for common bile duct (CBD) stone disease, is not recommended for the repair of BBS

With inputs from my former fellows
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Also see Invited Commentary on Surgical Management of Benign Biliary Stricture - Hepatico-jejunostomy by Henry A Pitt (pp 173–174)

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(b) Hepatico-jejunostomy performed to the common hepatic duct (CHD) or the left hepatic duct (LHD).

NOTE: *Transduodenal sphincteroplasty (TDS) is also a side-to-side bilio-enteric anastomosis.*

Surgical repair of a BBS is a bilio-enteric anastomosis, invariably in the form of a Roux-en-Y hepatico-jejunostomy. One hundred and five out of 130 patients reported from the Hammersmith Hospital, London, UK underwent hepatico-jejunostomy; only 3 patients had choledocho-duodenostomy [1]. Out of 175 surgical repairs performed in the Johns Hopkins Hospital, Baltimore, MD, USA only 3 were end-to-end repairs—2 performed at the time of cholecystectomy and 1 performed after 7 days; the remaining 172 repairs were hepatico-jejunostomy. Other than for repair of a BBS, Roux-en-Y hepatico-jejunostomy is also performed after choledochal cyst excision, as a part of pancreatoduodenectomy (PD) and following resection for cholangiocarcinoma. In order to differentiate the bilio-enteric anastomosis performed for BBS from that performed for these other indications, the Author (VKK) has proposed that the bilio-enteric anastomosis performed for BBS should be called hilo-jejunostomy (as it should ideally be performed at the hilum—the biliary ductal confluence), instead of hepatico-jejunostomy [2]. Jarnagin and Blumgart [3] have described the technique of operative repair of BDI involving the hepatic ductal confluence.

13.3 Contraindications

Patients with BBS who are not fit for general anesthesia or surgery due to poor performance status or uncontrollable comorbid conditions may not be operated and may be treated non-surgically by endoscopic or percutaneous balloon dilatation and stenting (See Chap. 15).

Severe coagulopathy because of obstructive jaundice is a relative and temporary contraindication for surgical repair of BBS; it should be corrected with vitamin K and/or fresh frozen plasma (FFP) before a surgical repair is undertaken.

Multiple previous unsuccessful surgical repairs and a high (Bismuth Type IV) BBS combined with a vascular injury may also be a relative contraindication for surgical repair in the form of hepatico-jejunostomy and such patients may require liver resection (See Chap. 14).

Patients with poor liver function due to secondary biliary cirrhosis (SBC) and those with severe portal hypertension and end stage liver disease (ESLD) may be candidates for a liver transplant (See Chap. 14) instead of hepatico-jejunostomy.

13.4 Timing of Repair

Hepatico-jejunostomy is a technically challenging operation—to obtain the best results, it should be performed at an appropriate time (delayed, i.e., at least 6 weeks after the bile duct injury), in an appropriate patient (no sepsis) and by an appropriate surgeon (a biliary surgeon). Prerequisites for selecting the time of repair are no bile collection, no bile fistula, and no (recent) cholangitis.

There is lot of confusion regarding the terminology of the timing of repair of a bile duct injury (BDI). The Author (VKK) proposes that the timing of repair of a BDI/BBS should be classified as:

1. *Immediate*—intraoperative repair of a BDI detected during the cholecystectomy itself (should be done only if the injuring surgeon himself is a biliary surgeon or if the help of a biliary surgeon can be obtained); the philosophy of immediate repair can be extended to 48–72 h in the postoperative period (prompt repair [4]) (See Chap. 9)
2. *Early*—repair within 6 weeks of the injury.
3. *Delayed*—repair at least 6 weeks after but within 6 months of the injury. In most cases, the author recommends delayed repair of a BDI/BBS.
4. *Late*—repair more than 6 months after the injury which carries a risk of development of secondary biliary cirrhosis (SBC).

   Sahajpal [5] defined repair done after 72 h but before 6 weeks as intermediate repair.

The Author (VKK) recommends delayed repair of a BBS in most of the cases.

Dominguez-Rosado [6] reported their experience with 614 cases and recommended delayed (>6 weeks) repair after adequate sepsis control.

13.5 Delayed Repair

Different groups have defined delayed repair of the BDI in varied ways. Bismuth [7] recommended waiting for at least a month and a dilatation of biliary confluence to at least 10 mm; this takes about 2–3 months. Thomson [8] defined delayed repair as that done between 2 weeks and 6 months after the BDI. The Author (VKK), however, defines delayed repair as that done between 6 weeks and 6 months after the BDI —the ideal time to repair it. Iannelli [9] also defined delayed repair as that done 45 days after injury. Sicklick [10] extended the definition of delayed repair to 12 months. Risk rates for failure were 16% for repair performed within 14 days, 19% for repair between 14 and 90 days, and 10% for repair done after 90 days [11].

The best time to repair a BDI (with bile leak) is when all sepsis (peritoneal and biliary) has been controlled and the external biliary fistula (EBF) has either closed (and proximal biliary ductal dilatation has occurred) or is controlled. This usually takes a few weeks' time. This delay allows sepsis, inflammation, and edema to settle. The extent (level) of the BDI may progress (ascend) as a result of infection, inflammation, and ischemia, more so if an associated vascular injury is also present. Even if an associated vascular injury is not present, the stricture takes time to establish and mature to its highest level and the proximal bile ducts to dilate. Dilatation of the proximal bile ducts is desirable before a surgical repair is attempted for a BBS. This can occur only when the external biliary fistula (EBF) closes. It is technically easier to perform hepatico-jejunostomy in the presence of proximal ductal dilatation. Also, a larger stoma diameter can be obtained in the presence of proximal ductal dilatation. Degree of dilatation correlated with results of repair in 134 patients [12]. Another point in favor of a delayed repair is that laparoscopic BDIs are very often associated with a thermal injury to the bile duct which takes time (4–6 weeks) to show its maximum effect.

In the French review of 543 BDIs [9], best results were obtained after late (>45 days) repair—only 10/133 (7.5%) failed. The possible reasons for these good results were

1. Local inflammation caused by peritoneal and biliary sepsis had settled
2. The effect of vascular injury in the biliary stricture had stabilized
3. All late repairs were done in tertiary hepatobiliary centers
4. 122 out of 133 patients had hepatico-jejunostomy

Delayed repair (n = 133) was associated with fewer complications (14% vs. 39% and 29%), lower mortality (0.8% vs. 2.8% and 2.2%), and lower failure rates (7.5%. vs. 64% and 43%) as compared to immediate (n = 194) and early (n = 216) repair [9].

13.6 Late Repair

The Author (VKK) defines late repair as that performed after 6 months of the injury. Late repair carries the risk of development of secondary biliary cirrhosis (SBC) and its consequences.

13.7 Primary Repair

Primary repair should be defined as the first surgical repair of a BDI/BBS with no prior non-surgical or surgical attempt at repair (excluding, however, percutaneous catheter drainage, laparoscopy, or laparotomy, which may have been performed for drainage of biloma and endoscopic stent, which may have been placed for controlling ongoing bile leak).

13.8 Secondary Repair

Secondary repair should be defined as surgical repair of a BDI/BBS after a failed previous therapeutic non-surgical (i.e., endoscopic or percutaneous) intervention, e.g., balloon dilatation and/or stenting.

Secondary repair should be differentiated from primary repair (*vide supra*) with no previous therapeutic non-surgical or surgical intervention and from revision repair (*vide infra*) after a failed previous surgical repair.

In a report from the Academic Medical Center (AMC), Amsterdam, Netherlands, failure after secondary referral (after prior intervention) was 12/87 (14%) vs. 2/63 (3%) after primary referral (no prior intervention) [13].

13.9 Revision Repair

Revision repair should be defined as surgical repair after a failed previous surgical repair. Revision repair of a failed hepatico-jejunostomy may be required if the failure occurs early due to technical failure following an inappropriate first repair or if the failure occurs later during follow-up due to anastomotic stricture.

Revision repair should be differentiated from secondary repair (*vide supra*) which is surgical repair after a failed previous therapeutic non-surgical, i.e., endoscopic or percutaneous intervention and completion repair (*vide infra*) for a missed duct after a surgical repair.

13.10 Completion Repair

If an isolated duct (usually on the right side) is missed during the hepatico-jejunostomy, as can happen in Bismuth type IV or V BBS, reoperation may be required to drain the undrained duct into the same Roux limb.

13.11 Difficult Stricture

High (Bismuth Types III and IV) strictures, Bismuth Type V strictures and strictures in patients with atrophy-hypertrophy complex (AHC), secondary biliary cirrhosis (SBC), and portal hypertension are difficult to handle, even by an experienced biliary surgeon. In most patients with BBS, the left hepatic duct can be easily identified and dissected, but in a very high (involving the right anterior and posterior sectoral ducts) and long-standing BBS with repeated attacks of cholangitis, there may be extensive and dense fibrotic scarring in the hilum; associated atrophy-hypertrophy complex may result in rotation of the hilum and may cause the left portal vein to lie in front of the left hepatic duct, identification of even left hepatic duct, which is usually easy, may be difficult in such cases and it may be better to place a preoperative percutaneous transhepatic biliary catheter (PTBC) in even the left hepatic duct (in addition to placing similar catheters in the right anterior and posterior sectoral ducts which are mandatory) for easier intraoperative identification of these ducts.

ANECDOTE: *In one case, the Author (VKK) found it very difficult to locate the lumen of the left hepatic duct because of a very thick wall (due to recurrent cholangitis) and because the ducts were collapsed (due to PTBD).*

In another case, the Author (VKK) was about to mistake the segment IV duct as the left hepatic duct. If anastomosis were done to this (segment IV) duct, the left hepatic duct could have been missed and left undrained. Eventually, intraoperative cholangiography (IOC) revealed the correct biliary ductal anatomy and the left hepatic duct was identified and drained.

Box 13.1 Principles of a Successful Bilio-Enteric Anastomosis (BEA)

Good (complete) cholangiogram, i.e., MRC (Fig. 13.1) to delineate all intrahepatic ducts; Good vascularity (of both the bile duct and the Roux limb of jejunum); No tension (between the bile duct and the Roux limb of jejunum); Mucosa-to-mucosa approximation; Adequate stoma size; Interrupted sutures of fine delayed absorption suture material

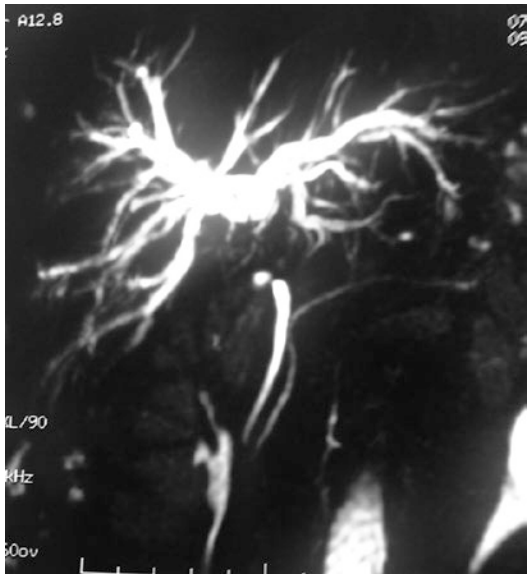

Fig. 13.1 A complete cholangiogram, in the form of a magnetic resonance cholangiogram (MRC), is essential before the repair of a benign biliary stricture

13.12 Anesthesia

General anesthesia is required; epidural analgesia may be added for better postoperative pain control.

13.13 Incision

The incision should provide adequate exposure of the hepatic hilum to repair a BBS. A liberal generously long right subcostal incision should be used for performing a hepatico-jejunostomy for BBS. Chevron (bilateral "right longer than left" subcostal, inverted V) incision is also used.

NOTE: *Chevron's logo, though, is a double V, not inverted V.*

In thin built patients with a narrow costal margin, a long upper midline incision may be used. Extreme caution should be exercised when opening the abdomen.

ANECDOTES: *In one case, the abdominal wall incision went into the edge of the liver which was densely adherent to the scar of previous laparotomy in the parietes. In another case, a jejunal loop densely adherent to the fistulous tract, got injured when opening the abdomen.*

Some groups, with expertise in laparoscopic pancreato-duodenectomy, have reported laparoscopic hepatico-jejunostomy in a small number of select cases (with an intact biliary ductal confluence [14]. Robotic hepatico-jejunostomy has also been reported [15]. The Author (VKK), however, recommends laparotomy for performing hepatico-jejunostomy for BBS.

13.14 Mobilization of Liver

Liver should be completely mobilized by dividing adhesions between the liver and the diaphragm and the parietes (Fig. 13.2) which are invariably present due to the bile leak following the BDI. The falciform ligament should be divided and taken off from the diaphragm and the left triangular ligament divided in order to further mobilize the liver. This enables the liver to be retracted well for proper exposure of the hepatic hilum for performing the hepatico-jejunostomy. This (mobilization of the liver) should be done

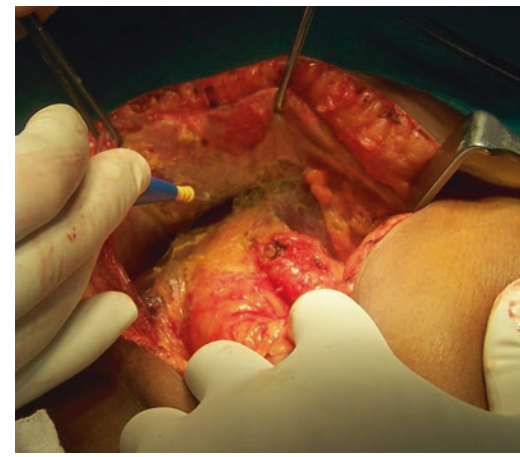

Fig. 13.2 Adhesions between the liver and the parietes need to be taken down so that liver can be retracted

before a self-retaining retractor is applied to the right costal margin otherwise liver capsule may tear where it is adherent to the parietes or where it joins the falciform ligament.

Some surgeons will later use the mobilized falciform ligament to provide a serosal cover for the hepatico-jejunostomy after it has been completed.

13.15 Retraction

A self-retaining table-mounted retractor, e.g., Omnitract®, is a valuable asset for retracting the right costal margin for better exposure of the hepatic hilum during the repair of a BBS. One designed by Dr SP Haribhakti (one of our former fellows) of Kaizen Hospital, Ahmedabad India (available from Keyog Surgicals, Ahmedabad India) is an inexpensive alternative option.

13.16 Pack

A folded sponge may be placed between the diaphragm and the superior surface of the liver to push the liver down for better exposure of the hepatic helium.

13.17 Illumination

A head mounted light provides better illumination at the hepatic hilum.

13.18 Lateral to Medial

The initial dissection on the undersurface of the liver starts at its lateral (right) edge and proceeds medially (towards left) mobilizing the hepatic flexure and the transverse colon.

13.19 Colon

Patients, who have had a bile leak and collection following the BDI, usually have adhesions in the right upper quadrant. These could be between the parietes and the liver and between the undersurface of the liver and colon (hepatic flexure and proximal transverse colon) (Fig. 13.3), duodenum, and jejunum. These adhesions have to be taken down (separated) taking care not to enter the liver capsule on one side and the bowel on the other side in order to expose the hepatic hilum. This may result in an inadvertent injury to the liver capsule or parenchyma (causing bleeding) or to the colon, duodenum, or jejunum. Sharp (scissors) dissection is preferred.

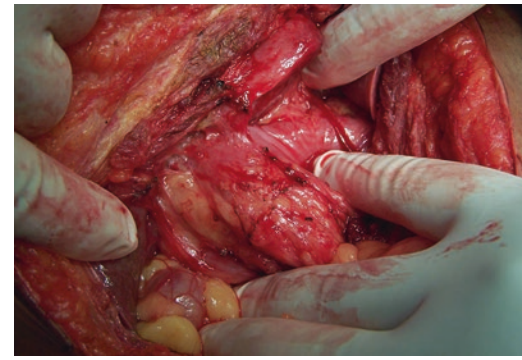

Fig. 13.3 Adhesions between the liver and the colon need to be taken down so that the colon can be retracted

13.20 Duodenum

After the transverse colon has been dissected and retracted down, the duodenum (first part) (Fig. 13.4) is dissected off the hepatic hilum and retracted down. Sharp dissection with scissors is preferred. An internal fistula (Fig. 13.5) may be present between the proximal bile duct and the duodenum; it may get opened as the duodenum is dissected down from the hepatic hilum. The opening in the duodenum should be repaired at this stage only (*lest it is forgotten later during the operation, if and when it runs into bad weather!*). The proximal opening of the fistula may then lead to the proximal bile duct (Fig. 13.6).

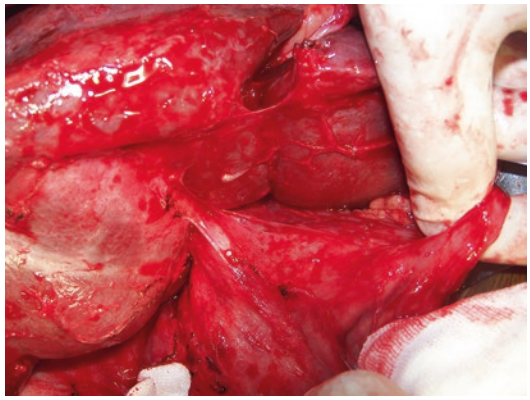

Fig. 13.4 Adhesions between the liver and the duodenum need to be taken down so that the duodenum can be retracted

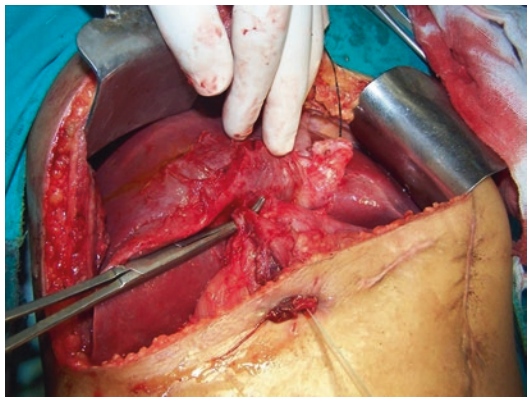

Fig. 13.5 Internal fistula between the proximal bile duct and the duodenum

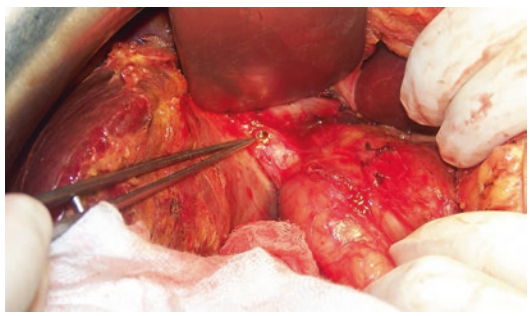

Fig. 13.6 Proximal opening of the fistulous tract leads to the proximal bile duct

13.21 Hepato-Duodenal Ligament

The hepato-duodenal ligament gets exposed once transverse colon and duodenum have been dissected down from the hepatic helium; it is usually fibrosed, scarred, shortened, and distorted. The pulsations of the proper hepatic artery should be palpated in the hepato-duodenal ligament from time to time; they should remain as a guide, dissection proceeding from lateral to medial (right to left), stopping short of them and not going to the left of these pulsations.

13.22 Bile Duct

The bile duct proximal to the biliary stricture needs to be located and identified. The T-tube, drain, or the fistulous tract may lead to it; if a hepatico-duodenal fistula is present, it may also lead to the duct as the fistula gets opened during the dissection.

The Johns Hopkins Hospital, Baltimore, MD, USA group mobilizes the proximal hepatic duct and resects the injured strictured ductal tissue back to the normal duct [16] resulting in an end-to-side anastomosis. The Author (VKK) does not agree with them; circumferential mobilization of the bile duct may result in an injury to the portal vein behind. The Author (VKK) always performs a side-to-side biliary-enteric anastomosis; resection of the stricture is done only if malignancy (cholangiocarcinoma) is suspected.

13.23 Fibrosis

Previous endoscopic biliary stenting leads to inflammatory fibrosis around the extrahepatic bile duct [17] making its dissection during a later surgical repair difficult.

13.24 Hilum

Hilum of the liver is approached from anterior to posterior (to avoid injury to the portal vein which lies posterior to the common bile duct).

In ALL cases (including Bismuth type I or II BBS, where a good common hepatic duct stump is available), bilio-enteric anastomosis should be performed at the biliary ductal confluence (as the blood supply is richest here) and should be extended to the left hepatic duct [18]—the Hepp-Couinaud [19, 20] approach. The extrahepatic horizontal part of the left hepatic duct MUST be exposed by lowering the hilar plate so that the incision in the common hepatic duct (CHD) and at the biliary ductal confluence is extended into the extrahepatic horizontal part of the left hepatic duct (Hepp—Couinaud approach) this provides a wide stoma even in Bismuth Type III BBS where there is no common hepatic duct stump.

13.25 Hilar Plate

Hilar plate (Fig. 13.7) is the Glisson's capsule (visceral peritoneum on the surface of the liver) as it reflects from the undersurface of the liver at the base of the segment IV (quadrate lobe and the hepatic hilum) on to the peritoneum of the lesser omentum and the hepato-duodenal ligament. Inflammation, because of bile leak and cholangitis, results in the base of the quadrate lobe getting adherent to the hepato-duodenal ligament thus giving an impression that the left bilio-vascular pedicle is intrahepatic. The hilar plate is incised at the base of the segment IV (quadrate lobe); hilum is lowered and liver parenchyma dissected superiorly—this exposes the left hepatic duct at the base of the segment IV. This is a key step in performing the hepatico-jejunostomy using the Hepp—Couinaud approach. A bridge of liver tissue is often present between segments IV and III of the liver at the base of the umbilical fissure—it does not contain any significant ducts or vessels and can be divided safely; this division provides better exposure of the left hepatic duct for hepatico-jejunostomy.

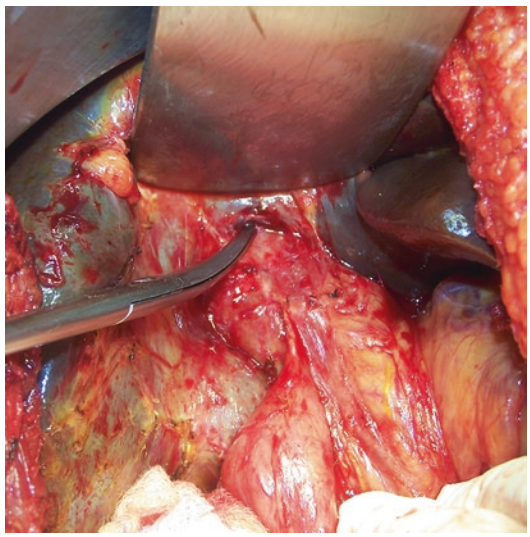

Fig. 13.7 Hilar plate at the base of the quadrate lobe (segment IV) needs to be lowered to expose the left hepatic duct

13.26 Identification

Intraoperative identification of the proximal bile ducts (which will be used for the biliary-enteric anastomosis) may be difficult in high strictures and those with persistent external biliary fistula (which leads to decompression of proximal bile ducts).

The fistulous tract should be followed towards the hilum as it may lead to the proximal bile ducts. When an internal fistula is divided, the proximal opening of the fistula will lead to the bile ducts. Clips identified during the operation may also point towards the location of the proximal dilated bile duct.

Preoperatively placed percutaneous transhepatic biliary catheters (PTBC) are difficult to palpate peroperatively because of fibrotic thickening in the duct wall. Saline stained with a color dye, e.g., gentian violet or methylene blue, is injected into the catheter and aspiration is done at the hilum with a fine (23G) needle to locate the proximal bile duct. Contrast can be injected through the catheter and intraoperative cholangiogram (IOC) done. Intraoperative US (IOUS) may also help to locate dilated intrahepatic ducts at the hilum. A fine (23G) needle should be used to aspirate bile to identify the proximal bile ducts at the hepatic hilum before it is opened for the bilio-enteric anastomosis. This becomes more important in presence of atrophy-hypertrophy complex when there is rotation of the hilum and the portal vein comes to lie in front of the common bile duct and may get accidentally incised if it is mistaken for the bile duct.

Before making an incision, the bile duct must be confirmed by needle aspiration to avoid making an inadvertent incision in the left portal vein (Fig. 13.8). All ducts (mainly left hepatic duct and right anterior and posterior sectoral ducts (Fig. 13.9) must be identified, opened, and included in the anastomosis. An incision in the common hepatic duct extended into the left hepatic duct is good enough for Bismuth Types I, II and III BBS. For Bismuth Type IV BBS, this is not enough as there is no common hepatic duct and the right hepatic duct and the left hepatic duct are not communicating with each other. Right hepatic duct, therefore, also needs to be drained separately. In Bismuth Type V BBS, the isolated strictured segmental/sectoral duct also needs to be drained separately.

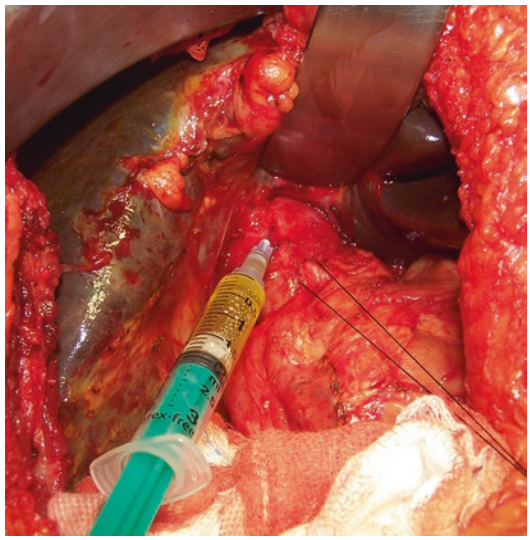

Fig. 13.8 Needle aspiration of the left hepatic duct to confirm that it is the bile duct and not the left portal vein

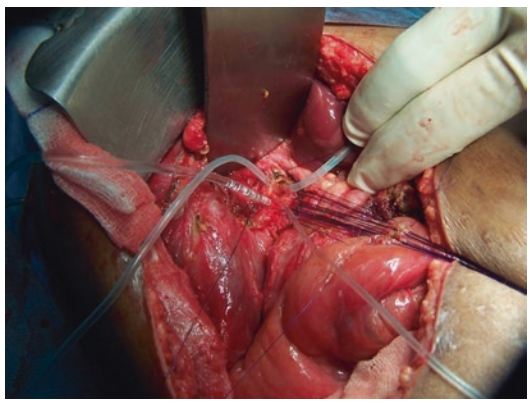

Fig. 13.9 All proximal bile ducts should be identified and accounted for

13.27 Circumferential

Stewart [21] reported incomplete excision of the scarred duct to be a risk factor for failure of hepatico-jejunostomy. Complete circumferential mobilization of the common hepatic duct and resection of the stricture with end-to-end anastomosis is practiced by the Johns Hopkins Hospital, Baltimore, MD, USA group [16]. Circumferential mobilization of the bile duct is, however, risky as it may result in an injury to the portal vein lying behind the common hepatic duct. The Author (VKK) prefers a side-to-side hepatico-jejunostomy which does not require complete circumferential mobilization of the common hepatic duct. Only the anterior surface of the duct should be exposed and a side-to-side biliary-enteric anastomosis should be performed. Complete circumferential mobilization of the common hepatic duct and resection of the stricture is required only if there is suspicion of malignancy, i.e., cholangiocarcinoma.

Patients who have had recent cholangitis may have very vascular bile ducts which bleed when they are opened. Bleeding vessels on/in the wall of the bile duct should be carefully controlled by cautery, preferably bipolar, to avoid thermal damage and ischemia to the wall of the bile duct. A small vessel (artery) is usually encountered and bleeds in the anterior wall of the left hepatic duct as a ductotomy is made in its anterior wall; it should be controlled with bipolar cautery. If these bleeding vessels are not controlled, they may continue to bleed into the anastomosis (resulting in hemobilia) and the resultant clot may cause anastomotic obstruction in the early postoperative period. The proximal intrahepatic ducts should be irrigated with saline using a catheter before the anastomosis is started in order to flush out stones and sludge (and any blood/clots).

13.28 Scar

While performing hepatico-jejunostomy for a BBS, the fibrotic scar in the bile duct should be avoided—anastomosis should be performed proximal to the fibrotic scar in healthy well-vascularized proximal bile duct with normal mucosa.

Every attempt must be made to avoid injury to the hepatic artery (especially right which lies in close relation with the common hepatic duct) which may be embedded in the fibrotic scar so as to avoid ischemia of the proximal duct which will be used for anastomosis.

13.29 Hemostasis

Hemostasis in the operative area should be secured before the bilio-enteric anastomosis is started; attempts to do so after the anastomosis has been completed may put undesirable traction on the anastomosis.

13.30 Drain

A subhepatic drain will always be placed after a hepatico-jejunostomy. The subhepatic drain should be placed *BEFORE* the anastomosis is started (Fig. 13.10); trying to place or position the drain after the anastomosis has been completed may put undesirable traction on the anastomosis.

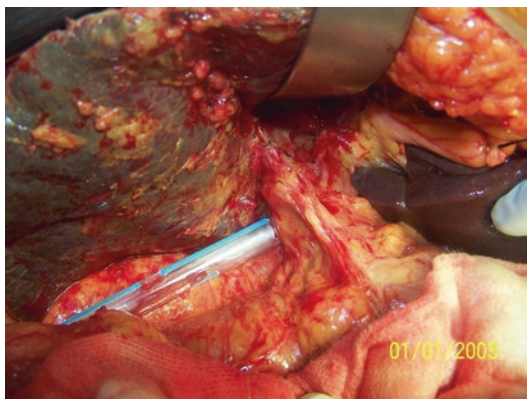

Fig. 13.10 Drain should be placed before the anastomosis is begun

Minimum manipulation should be done around the anastomosis after it has been completed. We use a single large (24–28 F) tube drain (and an extra anteriorly placed closed suction drain in case of a difficult precarious anastomosis) but the Johns Hopkins Hospital, Baltimore, MD, USA group uses multiple (median 3, range 0–4) closed suction drains after hepatico-jejunostomy.

13.31 Roux-en-Y Limb

The Author (VKK) prefers to call Roux a 'limb' (cf. Braun, which is a loop).

A Roux-en-Y limb of the jejunum is prepared. To create a Roux-en-Y limb, the jejunum should be divided about 30 cm from the duodeno-jejunal flexure in order to preserve the first foot of the proximal jejunum, which has important absorptive functions, in the "food" limb. Branches of the superior mesenteric vessels are avoided while dividing the mesentery for creation of a Roux-en-Y limb of jejunum by identifying them with the help of transillumination. No major vessels should be ligated. The mesentery of the Roux limb of the jejunum should be well mobilized so that the limb reaches the hepatic hilum comfortably for a tension-free anastomosis with the bile ducts. The distal end of the jejunum is closed and the Roux limb of the jejunum is brought from the infracolic compartment to the supracolic compartment (subhepatic fossa) in a retrocolic fashion through a small (so that the jejunal limb fits into it snugly) avascular mesocolic window to the right of the middle colic artery. In some patients, especially those who have had bile leak and sepsis, this may be difficult as the mesocolon may be edematous, thickened and inflamed and the colon may be adherent to the stomach and duodenum. In such cases, attempts to create a mesocolic window may cause inadvertent injury to the colon or duodenum or the middle colic vessels; the Roux limb may have to be taken up in an antecolic fashion.

A vascularized (pedicled) interposition jejunal loop between the proximal bile duct and duodenum with a proximal hepatico-jejunostomy and distal jejuno-duodenostomy has also been described for repair of BBS but is seldom used.

After creation of a Roux-en-Y limb, the proximal jejunum is anastomosed to the distal jejunum about 45–60 cm distal to the hepatico-jejunostomy. A 45–60 cm long (defunctioned) Roux limb is essential to prevent reflux of intestinal contents into the bile ducts and subsequent cholangitis. Bismuth [7] recommends 70 cm. This (the jejuno-jejunal anastomosis) can be an end-to-side or a side-to-side sutured or stapled anastomosis. The mesenteric gap between the proximal jejunum and the Roux limb should be closed after performing the jejuno-jejunostomy to avoid internal herniation.

13.32 Mesentery

A Roux-en-Y limb of jejunum is always preferred for hepatico-jejunostomy. While creating a Roux limb of the jejunum, a thick fat-laden small bowel mesentery or a mesentery with collaterals in patients with secondary biliary cirrhosis (SBC) due to long-standing biliary obstruction and portal hypertension and in patients with previous acute pancreatitis due to gall stones which necessitated the index cholecystectomy at which the BDI occurred may have resulted in inflammatory thickening of the small bowel mesentery may make division of the mesentery difficult and bloody. A simple loop of the jejunum with a distal side-to-side jejuno-jejunostomy between the two limbs of the jejunum (Braun loop) may be used in such patients.

13.33 Bilio-Enteric Anastomosis

13.33.1 Stoma

The diameter of the hepatico-jejunostomy anastomotic stoma should be as large as possible, preferably 2–3 cm. This is important as the surgical stoma is expected to contract in the long term to about 1/3rd of its original diameter. This is easily possible if the ducts are dilated but can be achieved even in the presence of undilated ducts by extending the incision in the common hepatic duct across the biliary ductal confluence into the left hepatic duct which almost always has an extrahepatic horizontal course at the base of the segment IV (quadrate lobe) between the hepatic hilum and the base of the round ligament. A side-to-side bilio-enteric anastomosis also helps to achieve a wide stoma even if the ducts are not dilated. The ideal stoma size of 2–3 cm may be difficult to achieve if the ducts are not dilated, e.g., in presence of an external or internal fistula and if the left hepatic duct has a vertical course which results in a short extrahepatic length. In such situations, as large a stoma as is possible should be created.

13.33.2 Jejunotomy

A jejunotomy is made on the antimesenteric border of the jejunal Roux limb, a few cm away from its closed (blind) end. The length of the jejunotomy should be smaller (about two-thirds; even one-half may be enough—Dr Ramesh Ardhanari of Meenakshi Mission Hospital Madurai; personal communication) than the length of the opening in the bile ducts as the jejunal opening always tends to expand. This is more likely to happen if the jejunum is opened when it is in the phase of contraction; jejunum should, therefore, be opened when it is in the phase of relaxation.

13.33.3 Single Layer

The bilio-enteric anastomosis should be performed in a single layer (*NOT two layers, as described in some surgical texts*) with interrupted sutures (*NOT continuous suture which causes ischemia*).

13.33.4 Mucosa-to-Mucosa

In a bilio-enteric anastomosis, mucosa-to-mucosa approximation is of utmost importance to prevent an anastomotic stricture. To achieve this, healthy bile duct proximal to the fibrotic scarred stricture

has to be dissected, opened, and anastomosed to the Roux limb of the jejunum. While taking bites in the jejunum, it is important to make sure that the mucosa does not retract and is included in each bite.

13.33.5 Suture

Fine (3-0/4-0/5-0, depending on the thickness and friability of the bile duct wall) monofilament long-acting absorbable suture, e.g., PDS® Ethicon (polydioxanone), on a small (13 mm) ½ circle round-bodied needle should preferably be used for the bilio-enteric anastomosis; poly-filament braided long-acting absorbable suture, e.g., Vicryl® Ethicon may also be used. Monofilament PDS® has better tissue passage than braided Vicryl® but has poor knotting properties (5–6 throws are, therefore, required); it is, however, more expensive than Vicryl®. Non-absorbable suture, e.g., silk, polypropylene, nylon, should not be used as it is a risk factor for unsuccessful outcome of hepatico-jejunostomy [21].

13.33.6 Staggered

If the bile duct wall is very thin and friable, fine (5-0) monofilament (e.g. PDS®) suture should be used for bilio-enteric anastomosis and the bites in the duct wall should be staggered to avoid a tearing effect; horizontal mattress sutures with knots on the jejunal wall may also be used to avoid cutting of sutures through the thin friable bile duct wall.

13.33.7 Crowding

Bites should be 1–2 mm apart and 1–2-mm deep during a bilio-enteric anastomosis. Adequate number of sutures should be placed so as to prevent bile leak from in between two far placed interrupted sutures but at the same time crowding of sutures should be avoided so that duct wall does not suffer ischemic damage.

13.33.8 Blumgart Kelly Technique

The two corner sutures—right and left—are placed first. The Author (VKK) prefers to make a box (U) stitch, outside-in and then inside-out, so that the knot lies outside, at the two corners in order to ensure that the corner is well covered.

13.33.9 Preplaced Anterior Sutures

All sutures on the anterior wall of the bile duct should be preplaced (outside-in) (Fig. 13.11) and held seriatim (to avoid their criss-crossing) in mosquito forceps before the posterior layer of the bilio-enteric anastomosis is started because it will be difficult to take bites on the anterior wall of bile duct after the posterior layer of sutures has been completed (tied). One could start from one end (i.e., right) and then go towards the other (i.e., left) but the Author (VKK) always takes the two corners (i.e., right and left) first, then divides the length of the anterior wall of the bile duct into two equal halves by taking the middle suture; each half is then divided into two quadrants by taking the middle suture in each half; adequate number (usually 2 or 3) of sutures is then taken in each quadrant. These preplaced sutures (with needles intact) will be used later to take bites in the anterior wall (inside-out) of the opening in the Roux limb of the jejunum.

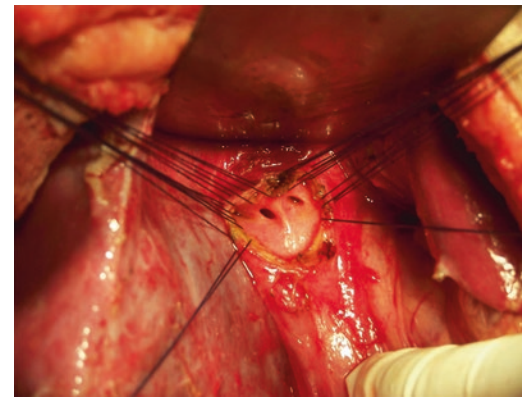

Fig. 13.11 Anterior preplaced sutures in the bile duct

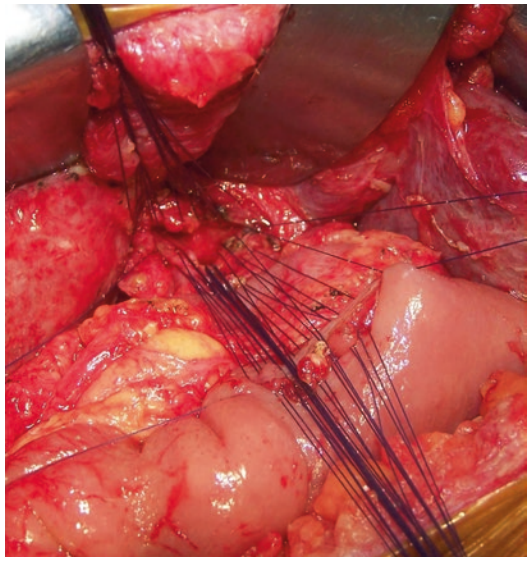

Fig. 13.12 Posterior row of sutures

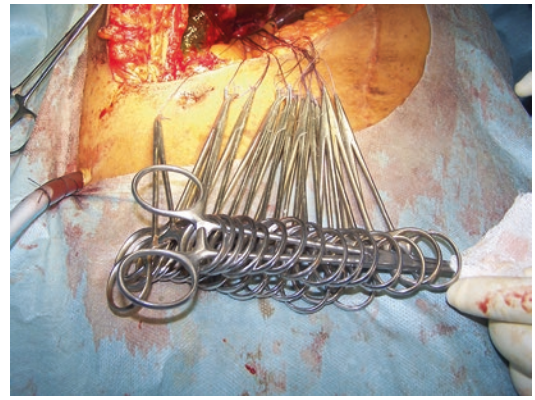

Fig. 13.13 The sutures are held seriatim in mosquito forceps

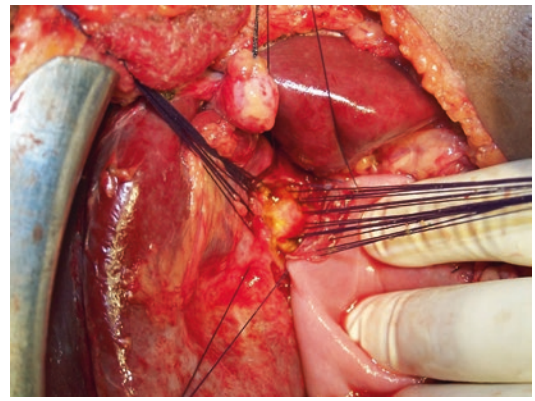

Fig. 13.14 Jejunal limb railroaded up to the bile duct

13.33.10 Posterior Sutures

After the anterior layer of sutures has been preplaced in the bile duct wall, the posterior layer is taken (Fig. 13.12). Once again, the Author (VKK) divides the length of the posterior walls of the jejunum and the bile duct into two equal halves by taking the middle suture; each half is then divided into two quadrants by taking the middle suture in each half; adequate number (usually 2 or 3) of sutures is then taken in each quadrant. All posterior sutures are once again held seriatim (to avoid their criss-crossing) in mosquito forceps (Fig. 13.13).

13.33.11 Railroad

After all posterior sutures have been taken, the jejunum is railroaded down (Fig. 13.14) over the posterior row of sutures to the hepatic hilum (lubrication of sutures with sterile jelly helps here). It must be ensured that the two walls (jejunum and bile duct) get approximated with no gap in between. Posterior sutures are now tied one by one (knots inside) starting on the right and going towards the left. As one suture is being tied by the surgeon, the immediate next suture (to the left of it) is held up taut by the assistant—this takes away tension, if any, from the suture being tied and avoids its cutting through the bile duct wall. The sutures are cut by the surgeon (to avoid inadvertent cutting of the immediate next untied suture) (Fig. 13.15).

The preplaced sutures in the anterior wall of the bile duct are now taken inside-out through the anterior wall of the jejunum (middle first, middle of two halves next, and then 2–3 sutures in each quadrant) (Fig. 13.16) and are tied.

Inversion of jejunal mucosa should be ensured by taking a smaller mucosal bite and bigger seromuscular bite in the jejunal wall.

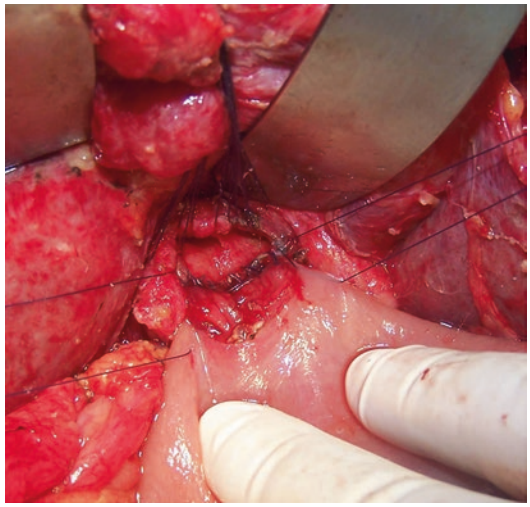

Fig. 13.15 Posterior layer of the anastomosis completed

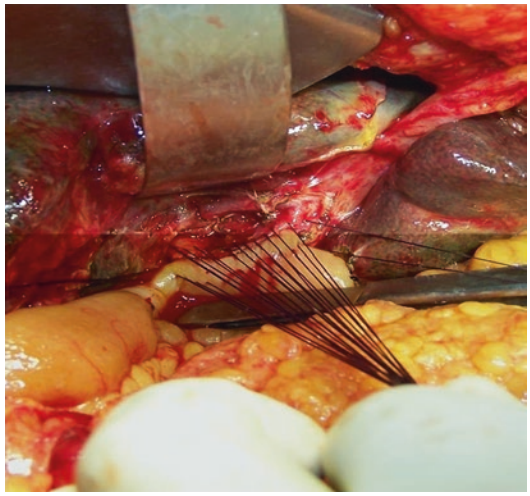

Fig. 13.16 Anterior sutures taken

The completed biliary-enteric anastomosis is thus at the hilum of the liver (Fig. 13.17)—hilo-jejunostomy [2].

13.33.12 Tension-Free

After the hepatico-jejunostomy is completed, the Roux limb of the jejunum should be fixed (anchored) to the undersurface of the liver with 2–3 sutures on either side of the anastomosis (Fig. 13.18) to take away the tension on the anastomosis due to gravity and peristalsis.

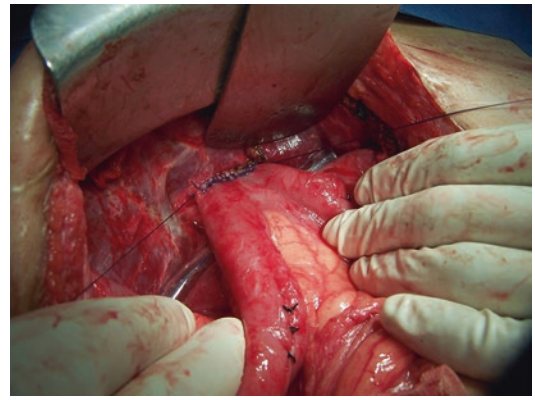

Fig. 13.17 Hilo-jejunostomy completed

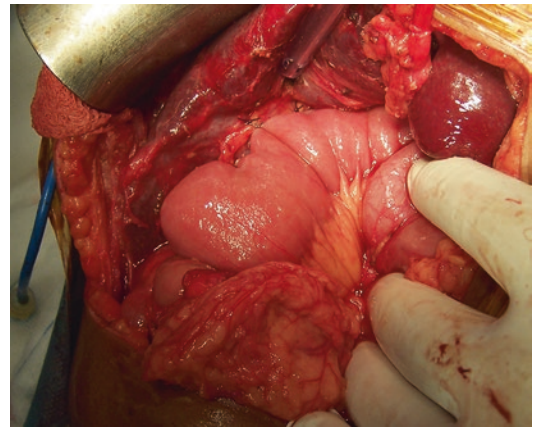

Fig. 13.18 Roux limb fixed to the undersurface of the liver

13.33.13 Omental Flap

A vascularized omental flap based on an epiploic vessel, which the Author (VKK) uses as a routine to cover the gastro-duodenal artery stump during pancreato-duodenectomy [22], may be used to provide cover to a precarious or unsatisfactory bilio-enteric anastomosis to decrease the risk of postoperative anastomotic leak. Mobilized falciform ligament may also be used.

13.33.14 Leak Test

Area around the hepatico-jejunostomy is washed, cleaned, and dried and a fresh dry opened gauze piece is wrapped around the anastomosis and left

in place for a few minutes. It is then removed and examined carefully for any bile staining. A gap between two anterior sutures can still be closed with an additional suture but a bile leak from the posterior layer, cannot be repaired at this stage. In case of a bile leak from the posterior layer, the Author (VKK) places an extra suction drain anterior to the anastomosis. In presence of a percutaneous transhepatic biliary catheter (PTBC), the area around the anastomosis is filled with saline, jejunal Roux limb is soft clamped distal to the biliary-enteric anastomosis and air is injected into the PTBC—absence of bubbling even when the jejunal Roux limb gets tensely distended with air confirms no leak.

13.33.15 Liver Biopsy

Biliary obstruction, especially if longstanding, leads to fibrosis and scarring around the intrahepatic bile ducts. Liver biopsy (wedge as well as needle) may be performed at the time of the repair of a BBS in patients with long-standing biliary obstruction to document fibrosis/cirrhosis. Fibrosis may regress or even reverse to normal after repair of the BBS; established cirrhosis, on the other hand, is irreversible and persists [23].

13.34 Special Considerations

13.34.1 Anastomotic Stent (Also Called Trans-Anastomotic Splinting)

Anastomotic stents may be required in patients with undilated ducts (patients with ongoing external biliary fistula), in patients with Bismuth Types IV and V BBS where a separate hepatico-jejunostomy is done to the right ducts in which (unlike the left hepatic duct) the stoma size cannot be increased.

The Author (VKK) suggests that the term proximal biliary drainage (catheter placed in the bile duct proximal to the anastomosis and retained for short term, i.e., 3–4 weeks) should be differentiated from the term anastomotic stenting (catheter placed across the biliary-enteric anastomosis and retained for long, i.e., 6–12 months).

Proximal biliary drainage decompresses the biliary system and reduces the intrabiliary pressure in order to protect a precarious bilio-enteric anastomosis; it also reduces the risk and consequences of a postoperative anastomotic leak. The Author (VKK) uses proximal biliary drainage in patients with thin friable inflamed bile ducts (sutures cutting through) where the anastomosis is precarious and unsatisfactory and chances of postoperative anastomotic leak are high. This is more likely if an immediate or early repair (which, in any case, the Author VKK does NOT recommend) is performed or in the presence of a recent episode of acute cholangitis. The proximal biliary catheter, when placed, can be used to obtain a postoperative cholangiogram at about 7–14 days to document that all the intrahepatic ducts have been drained and there are no missed isolated ducts, and that the anastomosis is secure and there is no anastomotic leak. The catheter can then be removed at about 3–4 weeks once the track is mature. Documentation of a complete and adequate anastomosis is useful for medico-legal purpose also if the patient develops recurrent problems, e.g., anastomotic stricture, cholangitis during the follow-up.

An anastomotic stent, on the other hand, is a tube placed across the biliary-enteric anastomosis and retained for long (6–12 months) to prevent or lessen the risk of the recurrent anastomotic stricture due to fibrosis of healing. Like any other anastomosis, a bilio-enteric anastomosis, i.e., hepatico-jejunostomy, is prone to anastomotic narrowing due to fibrosis as a part of healing. It is presumed that a hepatico-jejunostomy stoma will eventually narrow to about one-third of its original size. Placement of a transanastomotic stent limits this fibrotic narrowing to the size (diameter) of the stent. For this reason, the stent should be as large as possible but as the dilated ducts collapse on a large diameter stent the openings of the side ducts may get occluded by the wall of the stent. For this reason, the anastomotic stents should be of small diameter so as to provide drainage of bile across the anastomosis around the stent. Silastic® (Dow Corning) (silicone and plastic) is the preferred material for anastomotic stenting as it is inert and flexible.

An anastomotic stent may be placed transhepatic (preferred) or trans-jejunal (not preferred as it is likely to slip down into the jejunum) or in the form of a U-tube coming out through both liver and jejunum (not commonly used now). After the posterior sutures have been tied and cut, the preoperatively placed percutaneous transhepatic biliary catheter (PTBC) can be used to guide and railroad the anastomotic stent across the hepatico-jejunostomy. The stent should be positioned into the jejunum after the posterior layer of the anastomosis has been completed before the anterior layer of the anastomosis is started (Fig. 13.19). A preoperatively placed percutaneous transhepatic biliary catheter (PTBC) can be used to railroad an anastomotic stent—this should be done before the anastomosis is started so as to avoid any disruption of the anastomosis. Intraoperative placement of a transhepatic stent through the thick liver parenchyma is difficult. The trans-jejunal anastomotic stent comes out of the jejunum a few cm distal to the hepatico-jejunostomy where the jejunum is fixed to the parietes in the right flank. Trans-jejunal stents should be fixed to the posterior wall (using one of the posterior wall sutures) of the biliary-enteric anastomosis to prevent their distal migration into the jejunal loop with peristalsis. U-tube is a transhepatic tube across the bilio-enteric anastomosis which also comes out of the jejunal Roux limb in the flank.

Use of anastomotic stents is very controversial. Cameron [24] described use of long-term (6–12 months) transhepatic silastic stent in hepatico-jejunostomy for hilar hepatic duct strictures in 10 patients. Mayo clinic used transanastomotic stents in 43 out of 59 patients; stents were retained for 45 ± 3 days [25]. The Johns Hopkins Hospital, Baltimore, MD, USA group places preoperative percutaneous transhepatic biliary catheters (PTBC) in all patients and then replaces them with larger transanastomotic soft silastic biliary stents; a median of 2 (range 0–4) stents were placed. The stent is initially placed on external drainage. It is used to perform a postoperative cholangiogram after a median of 5 days. If the cholangiogram shows no anastomotic leak, the stent is internalized (capped). The stents are exchanged on a routine basis every 2–3 months. The duration (6–12 months) of stenting depends upon the type of injury, the clinical status and cholangiography findings. A biliary manometric perfusion study is performed and the stent is repositioned above the anastomosis for 2 weeks before its removal [26]. Mercado [27] recommends use of an anastomotic stent when the bile ducts are unhealthy (ischemic, scarred) or undilated (<4 mm). The Academic Medical Center (AMC), Amsterdam, Netherlands group uses transhepatic anastomotic stents for only 2–6 weeks [13] (*in the Author's VKK opinion, such short-term catheters should actually be called proximal biliary drains*). In the report of 139 early repairs from Denmark, transhepatic or trans-jejunal stents were used selectively—in presence of peritonitis or in case of a narrow anastomosis [28]. Dominguez-Rosado [6] in a large experience with 614 repairs, however, reported that use of anastomotic stents is associated with anastomotic failure (stricture). The Author VKK does not place anastomotic stents as a routine in all cases but uses them selectively in some cases only if the anastomosis is not satisfactory (undilated ducts, resulting in inadequate stoma size or anastomosis to unhealthy scarred bile ducts). Thus an anastomotic stent may be required when an anastomosis has been done to an undilated right hepatic duct in a Bismuth Type III/IV BBS or a segmental/sectoral duct in a Bismuth Type V BBS. Left hepatic duct stoma is usually wide and does not usually require an anasto-

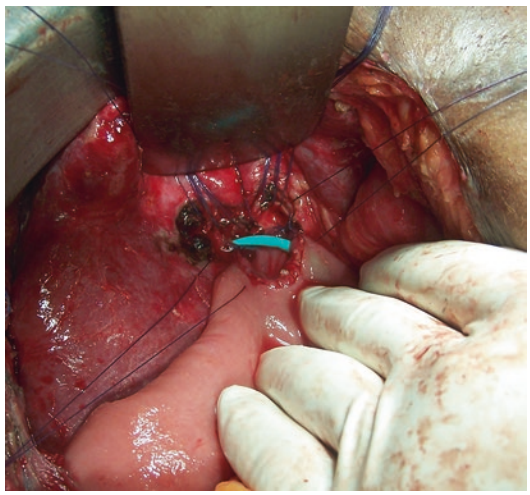

Fig. 13.19 Transanastomotic stent—posterior layer completed and anterior preplaced sutures in situ

motic stent. The Author (VKK) retains the anastomotic stents for at least 6 months; may be for 12 months.

> "Some people swear by stents, others swear at them".
> SM Vickers, in Sicklick [10]

Anastomotic stents can be used for postoperative percutaneous transhepatic balloon dilatation of an unsatisfactory anastomosis or an anastomotic stricture.

13.34.2 Septoplasty

In Bismuth Type IV BBS where the biliary ductal confluence is not patent and the right hepatic duct and the left hepatic duct are separated, a septoplasty between the two ducts may be required to achieve a single stoma hepatico-jejunostomy. If the separation of the right hepatic duct and the left hepatic duct is too much so that there is no septum to be divided, two separate stomas may have to be created. Three stomas may be required in a Bismuth Types IV BBS when even right anterior and posterior sectoral ducts are separate or in a Bismuth Type V BBS.

To perform a septoplasty, two fine (4-0/5-0) sutures are taken and tied at two corners of the septum (Fig. 13.20); the septum is then divided (with knife, scissors, or energy) between the sutures (Fig. 13.21). This process may be repeated 2–3 times to ensure complete division of the septum. It must, however, be kept in mind that as one goes deeper into the septum, it gets thicker and can bleed.

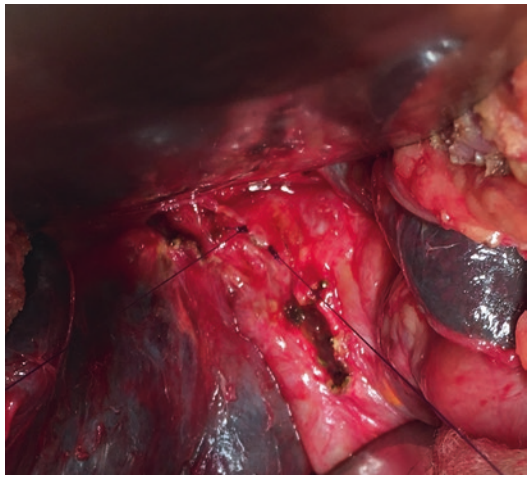

Fig. 13.20 Septum between right hepatic duct and left hepatic duct—two sutures taken

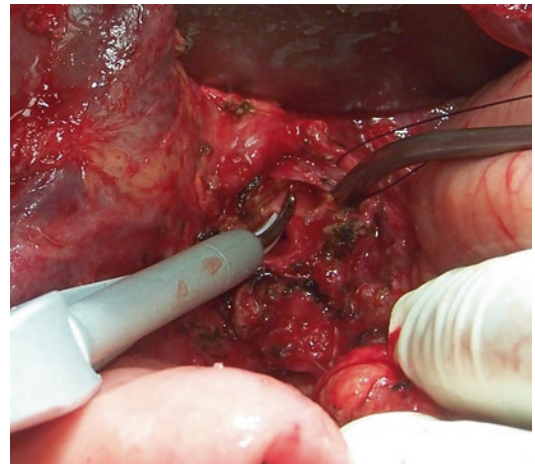

Fig. 13.21 Septum being divided with Harmonic scalpel

ANECDOTE: *In one case of obvious type III (confluence patent) BBS, the Author (VKK) made a ductotomy in the left hepatic duct but the right-angled Lahey's clamp would not go into the right hepatic duct. It was after spending a few minutes trying to manipulate the clamp from the left hepatic duct to the right hepatic duct that the Author (VKK) realized that it was a large septum which was preventing the right-angled clamp from entering the right hepatic duct—rotating and twisting the clamp little lower into the confluence allowed it to sweep around the overhanging septum and enter the right hepatic duct. A "septectomy" (excision of the septum) was done before bilio-enteric anastomosis was performed.*

13.34.3 Right Hepatic Duct

In Bismuth Types I, II and III BBS, the hepatico-jejunostomy is done to the biliary ductal confluence and the left hepatic duct; since the biliary ductal confluence is patent, the right hepatic duct also gets drained by this bilio-enteric anastomosis.

In Bismuth Type IV BBS, there is no common hepatic duct, the biliary ductal confluence is not patent and the right and left hepatic ducts are separated; a left hepatico-jejunostomy alone

will not drain the right liver and a separate bilioenteric anastomosis will have to be done to the right hepatic duct (in addition to the left hepatic duct). Access should be obtained to the non-scarred, non-ischemic, intrahepatic right hepatic duct. Preoperative percutaneous transhepatic biliary catheterization (PTBC) (Fig. 13.22) must be done for easier intraoperative identification of the intrahepatic right hepatic duct in such cases. Methylene blue injected into the PTBC will show at the hilum (Fig. 13.23). Intraoperative ultrasonography (IOUS) may also help to identify a dilated intrahepatic duct of the right system.

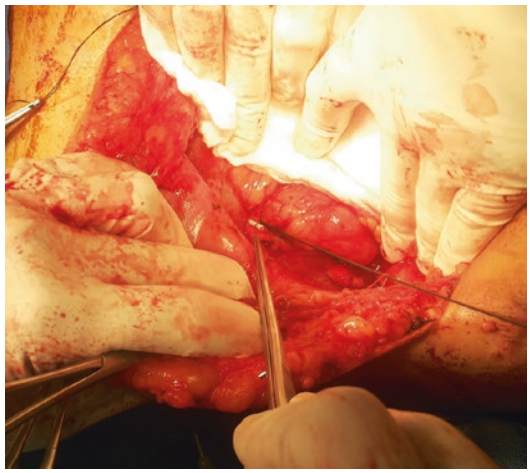

Fig. 13.22 Percutaneous transhepatic biliary catheter (PTBC) punctured through the bile duct into the peritoneal cavity

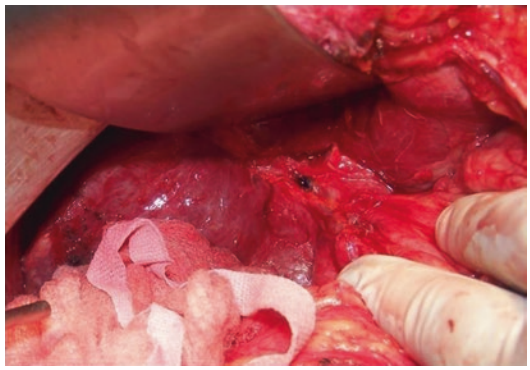

Fig. 13.23 Methylene blue injected into the percutaneous transhepatic biliary catheter (PTBC) shows at the liver hilum

In a very high (Bismuth Type IV) BBS there may be extensive scarring at the hilum involving not only the primary biliary ductal confluence but also the secondary right biliary ductal confluence so that even the right anterior and posterior sectoral ducts are separated. When there is no extrahepatic right hepatic duct, the intrahepatic right anterior sectoral duct in the portal pedicle may be exposed by hepatotomy or coring of liver parenchyma (Fig. 13.24) or even partial resection of a wedge of liver in the gallbladder bed at the junction of the undersurface of segments IVb and V. The right anterior sectoral duct lies in the gallbladder bed at a depth of 2–5 mm only. Sutherland [29] described a posterior approach to the right hepatic duct by incising the parenchyma in the caudate process but it has not become popular. Strasberg [30] described resection of hepatic parenchyma around the right hepatic duct (identified by preoperatively placed percutaneous biliary catheters). Mercado [31] described partial (wedge) resection of liver (anterior part of the inferior half of segment IV, i.e., quadrate lobe and segment V over the hilar plate) in the gallbladder bed to gain access

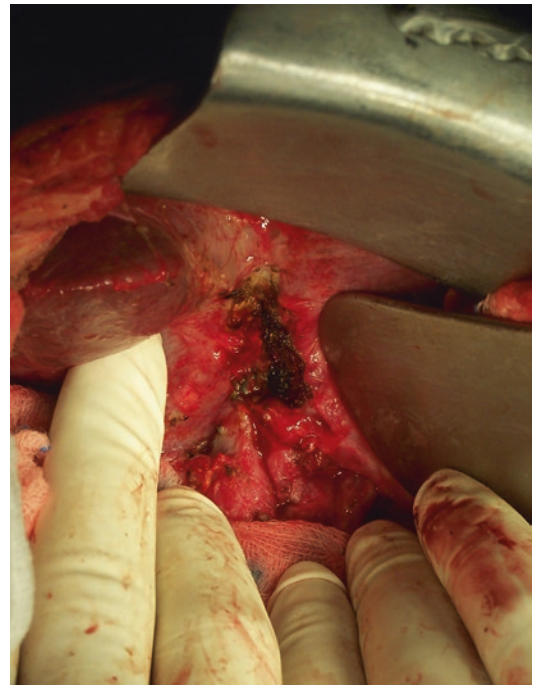

Fig. 13.24 Coring of liver parenchyma in the gallbladder bed to expose the right anterior sectoral pedicle

to the non-inflamed, non-ischemic, non-scared intrahepatic right ducts in case of a difficult BBS; they used this technique in 136 patients. In a later report [32], an intrahepatic repair was required in 198 (following partial resection of segments IV and V in 136 cases) out of 405 patients with BDI. Miyazaki [33] described a transhepatic approach—after division of the liver parenchyma to the left or right of the middle hepatic vein (MHV) (depending on the pattern of its tributaries)—to the intrahepatic right sectoral or segmental ducts for repair of a very high BBS. Cavitron ultrasonic surgical aspirator (CUSA), used for dissection within the liver parenchyma during a formal hepatectomy, may be used for hepatotomy or coring of liver parenchyma in the gallbladder bed during repair of a high BBS also.

Right hepatectomy may be required if the right hepatic duct is not reparable, especially if associated with a vascular injury (See Chap. 14).

13.34.4 Multiple Stomas

In patients with Bismuth Types IV and V BBS, multiple stomas may have to be created during a bilio-enteric anastomosis if the separated ducts cannot be converted into one stoma by septoplasty (*vide supra*). All biliary openings may be anastomosed to a single jejunal opening or separate jejunal openings may be made for each bile duct. Anterior sutures in all biliary ducts should be preplaced first before the anastomosis is started.

13.34.5 Portal Hypertension

Long-standing BBS gets complicated by secondary biliary cirrhosis (SBC) and portal hypertension and collaterals in the hepato-duodenal ligament which may prevent surgical access to the bile duct for a bilio-enteric anastomosis. Injudicious dissection in the hepato-duodenal ligament or at the hepatic hilum may cause torrential massive uncontrollable and even fatal bleeding from the high-pressure thin-walled venous collaterals. In such a situation, the surgical plan should be changed and a porta-systemic shunt should be performed first to decompress the collaterals before a bilio-enteric anastomosis is attempted at another operation (two stage repair) after some time (3–6 months) when the collaterals get decompressed by a patent porta-systemic shunt. In an older report, Chapman [1] reported portal hypertension in 23 (18%) out of 130 patients with biliary stricture; this high incidence was because majority (80, 62%) of these patients had undergone multiple operative procedures before referral; 4 of these patients required a porta-systemic shunt before repair. Mortality in these 23 patients with portal hypertension was much higher (n = 5, 23%) as compared to those without portal hypertension (2%). In our experience [34], only 11 (3.7%) of 300 patients had portal hypertension; all underwent repair without a prior porta-systemic shunt but mortality was 9% (cf. mortality of 1% in patients without portal hypertension). Agarwal [35] reported 13 patients with BBS and portal hypertension managed between 2000 and 2006—hepatico-jejunostomy could be performed in 11 patients with no mortality or major morbidity; operation time was 3.5 h and blood loss was 300 mL; follow-up was, however, short (median 17 months). Perakath et al. [36] reported 14 consecutive patients with BBS and portal hypertension managed between 1989 and 2001—13 patients were operated but only one underwent a porta-systemic shunt (shunt was attempted in one more patient but could not be performed). Hepatico-jejunostomy was possible in the remaining 11 patients. No patient died. Nine patients were available for follow-up—one had cholangitis and another had jaundice (failures); 7 were asymptomatic (though ALP levels were elevated in 5). Mishra [37] reported 6 patients with portal hypertension; two were deemed to be unfit for operation (one was lost to follow-up and the other was managed with PTBD alone), in one patient, the hepatico-jejunostomy was abandoned due to hypotension during operation, 3 patients underwent repair but two died and only one was well at 2.5 years; they recommended liver transplant for these patients. In case the biliary ductal continuity is preserved, these patients may be candidates for endoscopic or percutaneous balloon dilatation and stenting as the definitive non-surgical management of BBS.

13.34.6 Atrophy-Hypertrophy Complex (AHC)

A bilio-vascular injury may lead to atrophy (of the right lobe)—hypertrophy (of the left lobe) complex (AHC). The atrophy-hypertrophy complex results in rotation of the liver and the hepatic hilum and, as a consequence, of the hepato-duodenal ligament. In the more common right atrophy and left hypertrophy complex, this rotation is in an anti-clockwise direction so that the common bile duct, normally anterior in position, comes to lie on the right lateral or even posterior aspect of the hepato-duodenal ligament making it difficult to access surgically. The rotation also brings the portal vein to lie anterior to the common bile duct and makes it (and the hepatic artery) liable to injury during dissection in the hepato-duodenal ligament or the hepatic hilum. Common hepatic duct should, therefore, be looked for on the right lateral or posterior aspect of the hepato-duodenal ligament in such cases. Moreover, the hypertrophic segment IV may overhang the hepatic hilum and make the hilar dissection, e.g., lowering of the hilar plate, difficult [38].

In patients with atrophy-hypertrophy complex, hepatico-jejunostomy can be performed if a bile duct is available for anastomosis; hepatectomy may, however, be required if an adequate healthy bile duct is not available at the hepatic hilum, e.g., in a difficult Bismuth Type IV biliary stricture (See Chap. 14).

13.34.7 Isolated Duct

Isolated segmental or sectoral duct injuries are not uncommon. An isolated injury to the right hepatic duct or one of the right sectoral or segmental ducts may occur in some cases. These injuries are not mentioned in the Bismuth classification.

A good MRC should reveal such an injury (Fig. 13.25a, b, c). PTBC must be placed in all isolated ducts for their identification during operation. Lillemoe [39] reported access to an isolated right duct stricture after resection of the base of the gallbladder fossa in 9 cases. Strasberg [30] reported repair of isolated strictures in 22 cases—no recurrent problems occurred over median follow-up of 3 years. Colovic [40] reported 19 isolated segmental, sectoral, or right hepatic duct injuries over a 26 year period—only 7 patients had closure of the fistula not requiring repair. Results of repair of an aberrant duct are poorer than those of the main duct because of shorter extrahepatic length and smaller diameter of these ducts.

13.34.8 Cholangio-Jejunostomy

An intrahepatic cholangio-jejunostomy (to the segment V duct in the gallbladder bed or to the segment VI duct after removing a wedge of the liver in segment VI on the right side and to the segment III duct on the left side) may rarely be required in a high (Bismuth Type IV) BBS when the biliary ductal confluence is involved. The segment III duct is exposed at the base of the round ligament (ligamentum teres) in the umbilical fissure on the left side after ligation and division of the radicals of the left portal vein [41], or by performing a vertical hepatotomy in segment III to the left of the falciform ligament, or by removing a wedge of the liver in segment III.

13.34.9 Longmire Procedure

Longmire [42] procedure, i.e., hepato-jejunostomy, is resection of a part of the left lateral segment of the liver thus exposing multiple intrahepatic bile ducts and anastomosis of the capsule of the cut surface of the liver to a Roux-en-Y loop of jejunum for difficult biliary strictures; it is rarely performed as the left hepatic duct should be available for an adequate bilio-enteric anastomosis in almost all cases. Hepato-jejunostomy is not a mucosa-to-mucosa anastomosis and should be differentiated from an intrahepatic cholangio-jejunostomy (*vide supra*) which is a mucosa-to-

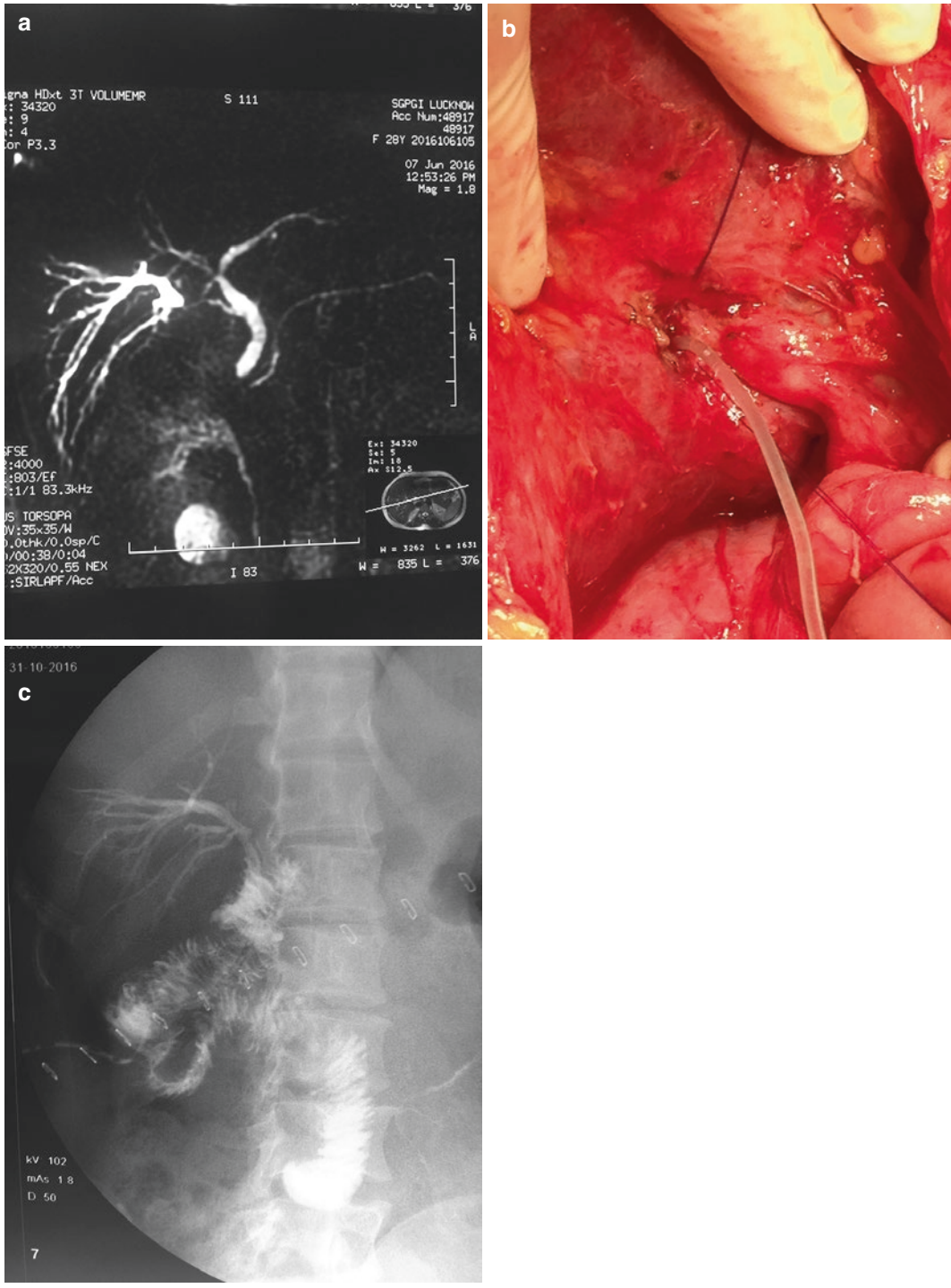

Fig. 13.25 (**a**) Right posterior sectoral duct stricture; (**b**) Percutaneous transhepatic biliary catheter (PTBC); (**c**) Postoperative stentogram—right anterior sectoral duct and left hepatic duct are not seen

mucosa anastomosis. Longmire procedure cannot be performed on the right side.

13.34.10 Smith's Mucosal Graft

In a very high BBS, the fibrotic scarring extends into the intrahepatic right hepatic duct; it is not possible to obtain normal mucosa in the right hepatic duct and a mucosa-to-mucosa anastomosis may be technically difficult. Smith's mucosal graft [43, 44] is indicated in such cases. An elevated dome (cone) of jejunal mucosa (after excision of a seromuscular disc) is pulled into the intrahepatic duct by a transhepatic tube to achieve a suture-less mucosa-to-mucosa approximation (NOT anastomosis). It is rarely performed now-a-day for BBS; parenchymal coring or hepatotomy in the gallbladder bed usually exposes the intrahepatic right hepatic duct for anastomosis or else a right hepatectomy may be performed.

ANECDOTE: *The only Smith's mucosal graft performed by the Author (VKK) in a patient with a difficult high Bismuth Type IV BBS (fortunately) lasted (functioned) for almost a decade when it strictured resulting in a cholangiolytic abscess and atrophy of the right lobe later.*

13.34.11 Porto-Enterostomy

In a very high (usually recurrent) stricture, when the proximal ducts cannot be found and a mucosa-to-mucosa anastomosis is not possible, the fibrotic scar at the hilum is excised to expose multiple bile ducts and a porto-enterostomy to the capsule or parenchyma of the liver at the hilum (as is done for extrahepatic biliary atresia EHBA) may be an option. It is, however, very likely to restricture and is not recommended. In such circumstances, it is better to perform a right hepatectomy, after which the left hepatic duct is better exposed for an adequate bilio-enteric anastomosis.

Gao et al. [45] reported porto-enterostomy combined with use of biliary stents in 10 patients (6 benign and 4 malignant) with hilar strictures. Ha [46] performed porto-enterostomy with multiple internal biliary stents in one patient with BDI and called it as cluster hepatico-jejunotomy—no biliary complications occurred during 5 years follow-up. Mercado [47] defined porto-enterostomy as less than 50% of the circumference of the anastomosis including biliary epithelium and reported its need in 26 out of 53 patients with loss of biliary ductal confluence (Bismuth Type IV stricture).

Intrahepatic cholangio-jejunostomy, Longmire procedure, Smith's mucosal graft, and porto-enterostomy are not recommended for BBS because of a high risk of restricture. In cases requiring one of these procedures, a liver resection should be considered (See Chap. 14).

13.34.12 Access Loop

The Roux-en-Y limb of jejunum used for hepatico-jejunostomy may be brought to the surface for easy percutaneous radiological access for any intervention during the follow-up—this is called access loop. It may be the closed end or the side of the jejunal limb. The access loops may be brought to the surface either in the midline (end of the limb) or in the flank (side of the limb)—the latter is preferred by the interventional radiologists as it avoids their hands coming in the radiation field during fluoroscopy. The loop may be hitched to the parietal peritoneum or brought out even further into the subcutaneous tissue. The site of the access loop may be marked with radio-opaque markers, e.g., metal clips, steel wire, etc., for easier location under fluoroscopy. The access loop can be accessed radiologically for percutaneous intervention. A formal end muco-cutaneous stoma of the access loop which can be accessed endoscopically is rarely performed.

An internal access loop, i.e., a formal anastomosis of the end of the jejunal Roux limb to the stomach [48] or to the duodenum has also been described. The distance between the jejuno-gastrostomy (or jejuno-duodenostomy) and the hepatico-jejunostomy should be about 10–15 cm to prevent reflux and cholangitis. The internal access loop can be accessed endoscopically for intervention. Selvakumar [49] reported creation of a gastric access loop in the form of an end-to-side jejuno-gastrostomy in 13 patients between 1999 and 2003. No patient had bile gastritis. During a mean follow-

up of 51 (range 20–81) months, the jejuno-gastrostomy, however, strictured in 3 patients.

Access loop is not performed as a routine in all cases but should certainly be performed in case of a difficult anastomosis which is more likely to stricture and require reintervention (dilatation). Though some authors recommend creation of an access loop in all patients with a high biliary stricture [50], the Author (VKK) does not recommend them. This is because of the philosophy of our interventional radiologists; even in cases where an access loop was made, they preferred to use the transhepatic route of intervention for recurrent problems. No access loop was reported in 139 early hepatico-jejunostomies reported from Denmark (1995–2010) [28].

13.34.13 Choledocho-Duodenostomy

Choledocho-duodenostomy, though technically easier than hepatico-jejunostomy and recommended by some groups [51], is not a preferred procedure and should not be performed for a bile duct injury or BBS; the procedure of choice for repair of BDI or BBS is a Roux-en-Y hepatico-jejunostomy. Only for a low benign biliary stricture, e.g., in chronic pancreatitis or one caused during distal gastrectomy, choledocho-duodenostomy may be performed. Cheng [52] reported use of tubular gastric wall with vascularized pedicle for repair of such low biliary strictures.

13.35 Complications

13.35.1 Intraoperative Complications

1. Injury to the colon and duodenum (and any other adherent loop of bowel, e.g., jejunum) during separation of subhepatic adhesions.
2. Bleeding may occur during a bilio-enteric anastomosis
   (a) from the surface of liver if the liver capsule gets torn during separation of adhesions
   (b) an aberrant right hepatic artery originating from the superior mesenteric artery as it ascends to the right of the hepato-duodenal ligament and lies behind and to the right of the common bile duct; it may get injured as dissection is done in the hepato-duodenal ligament
   (c) from the right hepatic artery lying on the anterior wall of the common hepatic duct during dissection of the duct
   (d) from the proper hepatic artery if dissection is taken too much to the left in the hepato-duodenal ligament
   (e) from the segment IV branch of the left hepatic artery when lowering the hilar plate
   (f) from an artery in the wall (vasa ductorum) of the left hepatic duct when it is opened—it should be controlled (on both ends), preferably with bipolar cautery to minimize thermal damage to the duct wall
   (g) as a result of puncture of an artery (usually right hepatic) or vein (left portal vein) lying behind the bile duct if deep bites are taken in the lower (inferior) lip of the opened left hepatic duct during the anastomosis—remove the suture and apply firm pressure with a gauze piece for adequate time (do NOT tie the suture as this will convert a small needle puncture hole in the vessel into a large laceration)
   (h) bleeding from the bile duct or the jejunal wall may result in clot obstruction of the bilio-enteric anastomosis in the early postoperative period. Adequate hemostasis of the cut ends of the bile duct and the jejunal wall is, therefore, of utmost importance before the hepatico-jejunostomy anastomosis is started. This is best done with bipolar cautery using a fine forceps
   (i) from the vessels in the jejunal mesentery and the transverse mesocolon especially if they are fat laden
   (j) from the site of liver biopsy at the end of the operation

TIP: *A recent attack of cholangitis results in increased vascularity in and around the common*

bile duct causing more than usual intraoperative bleeding—repair should be attempted about 2 weeks after the last attack of cholangitis has subsided to allow the inflammation to settle.

TIP: *If the repair is performed late (beyond 6 months), secondary biliary cirrhosis (SBC) and portal hypertension may result in collaterals in the parietes, hepato-duodenal ligament, liver hilum and jejunal mesentery. If excessive bleeding is encountered, attempts at bilio-enteric anastomosis should be abandoned and a porta-systemic shunt should be performed instead. This will reduce the portal pressure and decompress the collaterals; bilio-enteric anastomosis should be attempted after 3–6 months.*

13.35.2 Postoperative Care

If a proximal biliary drain or an anastomotic stent has been placed at the time of the hepatico-jejunostomy, a contrast study (cholangiogram) (Fig. 13.26) can be done through it in the post-operative period (around 7–10 days) to show the adequacy of the bilio-enteric anastomosis, i.e., no anastomotic leak and to demonstrate that all the intrahepatic bile ducts have been drained and that no isolated bile duct has been missed.

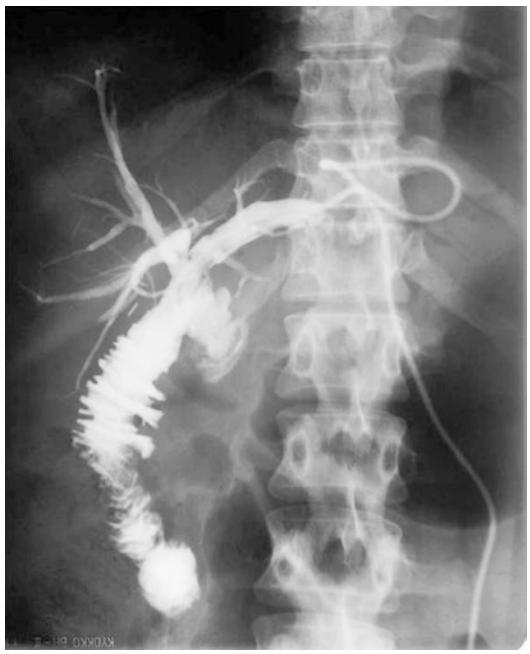

Fig. 13.26 Postoperative cholangiogram through percutaneous transhepatic biliary catheter (PTBC)

The Johns Hopkins Hospital, Baltimore, MD, USA group performs a manometric perfusion study of the biliary system before removal of an anastomotic stent. The Author (VKK), however, relies mainly on a cholangiogram.

13.35.3 Postoperative Complications

Hepatico-jejunostomy being a major operation may be associated with general postoperative complications, e.g., venous thrombo-embolism (VTE), cardio-pulmonary complications, wound complications, adhesive intestinal obstruction.

The Academic Medical Center (AMC), Amsterdam, Netherlands group has classified postoperative specific complications of hepatico-jejunostomy as minor (abscess, cholangitis, and wound infection) and major (anastomotic leak, bleed, relaparotomy, and anastomotic stricture) [13]. Schmidt [53] repaired 54 strictures between 1990 and 2002. Surgical repair in presence of peritonitis, high (at or above the bifurcation) injury, concomitant right hepatic artery injury, and previous surgical repair were factors associated with major biliary complications.

Mortality of biliary reconstruction can be high. Hypoalbuminemia, elevated serum bilirubin, and presence of liver cirrhosis and portal hypertension were factors for mortality after repair [1]. Johns Hopkins Hospital, Baltimore, MD, USA group reported a mortality of 3/175 (1.7%) [39]. Walsh [54] reported mortality of 4% in 84 patients after biliary-enteric reconstruction. Overall biliary specific mortality in the report from Denmark was 6 (4%) out of 139 cases [28]. In-hospital mortality in patients with major BDI who needed operative reconstruction in USA (2001–2011) was 4.4% [55]. Mortality after hepatico-jejunostomy in 293 patients in the American College of Surgeons (ACS) National Surgical Quality Improvement Program (NSQIP) database (2005–2012) in USA was 2% (5% for early repair and 0% for delayed repair) [56]. The Academic Medical Center (AMC),

Amsterdam, Netherlands reported 281 hepatico-jejunostomies (1991–2016)—90 day mortality was 0.7% [9]. Our mortality in 534 patients has been 6 (1%) (unpublished data).

13.35.4 Bleeding

Drainage is usually bilious (because of the high serum bilirubin) or sero-sanguinous fluid; excess of blood or bile in the drain should be a cause for concern. Early intra-abdominal bleed may be a surgical bleed but is more commonly due to coagulopathy. Correction of coagulopathy with vitamin K and fresh frozen plasma (FFP) will control the bleed in most cases; surgical bleed, if large, will require reoperation.

Intraluminal bleed (melena) is caused by hemobilia due to bleeding from the cut edge of the bile duct or jejunum or as a result of the liver biopsy. Large or persisting bleed will require investigation with angiography. Treatment is with radiological angioembolization.

13.35.5 Anastomotic Leak

One of the significant early complications of a bilio-enteric anastomosis, e.g., hepatico-jejunostomy, is anastomotic leak. This is more likely if repair is performed in the presence of sepsis (peritoneal or biliary)—especially soon after the bile duct injury (early repair)—when the ducts are inflamed, edematous, and friable and sutures may cut through. Chances of anastomotic leak are reduced if the hepatico-jejunostomy is performed as a delayed repair (at least 6 weeks after the BDI) when the bile leak has stopped, external biliary fistula (EBF) has closed and fibrosis has occurred to form a BBS with proximal ductal dilatation. Proximal biliary drainage in the form of a catheter placed in the biliary system proximal to the hepatico-jejunostomy may decrease/prevent anastomotic leak by reducing the intraductal pressure and decrease the ill effects of a leak, if it still occurs, by reducing the amount of bile leaking out.

The anastomotic leak manifests as bile leak (bile in the drain). Adequately placed drains in the subhepatic fossa (behind the hepatico-jejunostomy) detect the bile leak which, if required, can be confirmed by isotope hepato-biliary scintigraphy and a contrast study through the preoperatively placed percutaneous transhepatic biliary catheter (PTBC) or the anastomotic stent if it was placed. Imaging (US or CT) should be done if a leak is detected, to rule out any bile collection which, if present, should be drained by image (US or CT) guided percutaneous catheter drainage (PCD). However, most anastomotic leaks after hepatico-jejunostomy are small and minor; they do not usually require any intervention and settle with conservative management. If the leak continues for more than say a week, a proximal percutaneous transhepatic biliary drainage (PTBD) may be considered to reduce the amount of bile leak; reoperation is required only rarely, in case of a large anastomotic leak and/or generalized bile peritonitis.

An anastomotic leak and the resultant bile contamination cause inflammation and fibrosis around the hepatico-jejunostomy stoma and may result in anastomotic stricture in the long term. Anastomotic failures are more likely to occur in the long term in patients who had an anastomotic leak in the early postoperative period than in those who had an uneventful (no anastomotic leak) postoperative outcome.

13.35.6 Stent-Related Complications

Transhepatic placement of stents may cause hemobilia (*vide supra*). Bile leak at the exit site of the anastomotic stent in the dome of the liver was seen on postoperative cholangiography in 10.3% of patients [8]. Use of anastomotic stents may also increase infectious complications, e.g., cholangitis. The intrahepatic part of the anastomotic stent may sometimes cause obstruction to the openings of small intrahepatic ducts and cause stent-induced cholangitis. For this reason, a tube with multiple side holes should be used. Stent-induced cholangitis may necessitate removal of the stent earlier than planned.

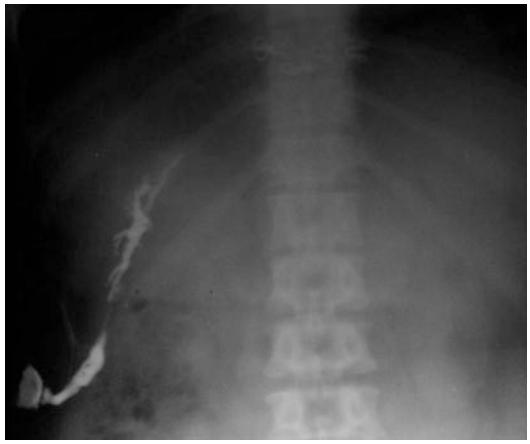

Fig. 13.27 Missed right sectoral duct presenting as an external biliary fistula after repair of a benign biliary stricture, seen here on fistulogram

13.35.7 Missed Duct

In patients with Bismuth Type IV BBS and wide separation of the right hepatic duct and the left hepatic duct—only the left hepatic duct may be repaired and the right hepatic duct may be missed. In case the stricture has separated the right anterior and right posterior sectoral ducts also from each other, one of these may be presumed to be the complete right hepatic duct and the other may be missed (Fig. 13.27). An isolated segmental or sectoral duct in a Bismuth Type V BBS may also be missed. These patients may continue to have jaundice and cholangitis or have recurrent cholangitis and cholangiolytic abscess and usually have an early failure. The part of liver drained by the missed undrained duct may sometimes fortunately undergo spontaneous atrophy but completion repair in the form of drainage of the isolated obstructed duct into the already made Roux-en-Y jejunal limb may be required.

13.35.8 Reoperation

Reoperation is not uncommonly required for bleeding or sepsis. Seven out of 151 patients who underwent reconstructive surgery for BDI required reoperation [13]. Fifteen (11%) of 139 patients required reoperation within 30 days of hepatico-jejunostomy [28].

13.35.9 Roux Limb Complications

Stasis in the jejunal Roux limb may cause recurrent cholangitis even in the presence of a patent biliary-enteric anastomosis. It can be diagnosed by following the movement of the normally excreted isotope in the Roux limb on isotope hepato-biliary scintigraphy. Inadvertent torsion of the Roux limb can occur. Jejuno-jejunostomy, like any other anastomosis, can leak.

13.35.10 Duodenal Ulcer

Dyspeptic symptoms are very common in patients who have had a bilio-enteric anastomosis using a Roux-en-Y jejunal limb. A duodenal ulcer has been reported in 10% of patients after a Roux-en-Y bilio-enteric anastomosis. H_2 receptor antagonists (H_2RA) or proton pump inhibitors (PPI) should be prescribed.

13.35.11 Anastomotic Stricture

Risk factors for anastomotic failure (stricture) include bile leak after cholecystectomy, ongoing EBF at the time of repair, proximal (high) BBS, previous attempts at repair, undilated ducts, and postoperative anastomotic leak. Ortiz-Brizuela [57] reported 117 episodes of acute cholangitis suggesting anastomotic stricture in 70 (13%) of 524 patients who underwent a biliary-enteric anastomosis between 2000 and 2014.

13.35.12 Malignancy

Tochhi [58] followed, 1,003 patients who underwent a biliary-enteric anastomosis (trans-duodenal sphincteroplasty TDS, choledocho-duodenostomy CDD, or hepatico-jejunostomy) between 1967 and 1997 for mean

130 months—55 (5.5%) bile duct cancers were found—the incidence was maximum (7.2%) after choledocho-duodenostomy and lowest (1.9%) after hepatico-jejunostomy.

Roux-en-Y hepatico-jejunostomy (the Author VKK prefers to call it "hilo-jejunostomy" [2]) is the surgical procedure of choice for BBS. It should, however, be performed by a biliary surgeon in order to obtain good short-term and long-term results. Bleeding and leak are the two most common specific complications of hepatico-jejunostomy.

Invited Commentary on Surgical Management of Benign Biliary Stricture: Hepatico-Jejunostomy

Henry A. Pitt

The chapter on surgical management of benign biliary strictures is comprehensive and contains many surgical pearls. The Author's (VKK) preferences for a surgical repair and a Roux-en-Y hepatico-jejunostomy are shared by the vast majority of hepato-biliary surgeons. While more controversial, his preference for a delayed repair is carefully justified and is further supported by an analysis from the Indiana University [59]. Delay also is advised when a vasculo-biliary injury has occurred as progression of the ischemic damage is time dependent [60]. Management of a strictured hepatico-jejunal anastomosis is more controversial. An attempt at percutaneous balloon dilation is usually justified, and ultimate results will be predicted by (a) the initial ability to pass a guidewire across the stricture and (b) the ease of dilation as judged by an experienced Interventional Radiologist.

The principles of achieving a tension-free side-to-side hepatico-jejunostomy which extends onto the left hepatic duct are well accepted. Pearls regarding hepatic mobilization, retraction, temporary packing above the liver, illumination, lysis of adhesions and dissection of the hepatic flexure, and the duodenum are very important. Similarly, palpation and ultrasound to avoid injury to the hepatic arteries and the portal vein during dissection of the scarred hilar strictures are key maneuvers. Following a drain track, careful incision of the hilar plate, and/or dissection of the adhered liver to the left hepatic duct also are important steps in identifying the injured proximal duct(s). The Author's (VKK) recommendation to perform bilateral liver biopsies to establish the degree of liver injury also should be followed.

For more proximal injuries, preoperative placement of percutaneous transhepatic catheters may assist in duct identification as well as placement of large bore silastic transhepatic stents. Care in creation of the Roux-en-Y limb, as outlined in the chapter, is very important. Bringing the jejunal limb to the hepatic hilum in a retrocolic fashion is key even when the mesocolon is very scarred. Having the Roux limb reach the hepatic hilum when the right lobe is atrophied can be challenging but is worth the extra effort to avoid tension. Creating percutaneous access to the Roux limb should be avoided because these maneuvers may compromise the anastomosis. If necessary, expert interventional radiologists will be able to access the anastomosis from above through the liver. Also, access via the Roux limb frequently leads to leakage of enteric contents and painful skin excoriation.

Another advantage of transhepatic stents is that intraoperative cholangiography can be performed to assess that all ducts are drained and that no anastomotic leakage is apparent. Careful drain placement adjacent to the hepatico-jejunostomy or cholangio-jejunostomies as well as the exit site(s) of transhepatic stent(s), if utilized, is important. Creation of an omental flap to prevent a bile leak should not be necessary. If a bile leak occurs, discontinuation of suction, gradual drain retraction, and/or replacement will prevent the need for reoperation. Another advantage of transhepatic stent placement is that external drainage will facilitate healing of a bile leak and help to manage postoperative cholangitis.

The Author's (VKK) advice to avoid choledocho-duodenal anastomoses, Longmire procedures, Smith's mucosal grafts, and porto-

enterostomies also should be followed. Another pearl, not mentioned in the chapter, is the utilization of intravenous octreotide when portal hypertension is encountered. The octreotide infusion can be tapered over 24–48 h when the risk of excessive hemorrhage has passed. One aspect of the chapter that is not addressed is the expected short- and long-term outcomes. If the author's (VKK) recommendations are followed, more than 90% of patients should remain symptom free over 5–10 years. As the Author (VKK) suggests, the outcomes of surgical repair are better than those for the endoscopic or percutaneous approach [59]. A recent report suggests that surgical repair of a benign bile duct stricture also provides improved survival compared to endoscopic therapy [61]This ref no. will change.

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14 Surgical Management of Benign Biliary Stricture: Hepatectomy

Vinay K. Kapoor

Roux-en-Y hepatico-jejunostomy (See Chap. 13) is the procedure of choice for the repair of a benign biliary stricture (BBS). In a high (Bismuth Type IV) BBS, the left and right hepatic ducts are separated; in severe cases, the fibrotic scarring may be extending into the sectoral ducts also and even the right anterior and posterior sectoral ducts may be separated. While the left hepatic duct is usually available for anastomosis, the right hepatic duct may not be accessible or even if accessible, it may be scarred (fibrotic) and mucosa-to-mucosa bilio-enteric anastomosis may not be possible. A bilio-enteric anastomosis may fail in a small number of cases resulting in an anastomotic (recurrent) stricture (and consequent recurrent cholangitis) during the follow-up. Treatment of choice for an anastomotic stricture is percutaneous balloon dilatation and long-term stenting (See Chap. 15) but the stricture may recur yet again.

A complex BBS i.e. a combination of a high (difficult), i.e., Strasberg E4/E5 (Fig. 14.1) or Bismuth type IV or recurrent (anastomotic) (Fig. 14.2.) BBS, associated vascular injury and atrophy (of a segment, sector, or even lobe of liver) (Fig. 14.3), or ongoing sepsis (recurrent cholangitis and cholangiolytic abscesses in an atrophic segment, sector, or lobe of liver) may direct the management away from a bilio-enteric anastomosis towards a possible liver resection, i.e., hepatectomy. Associated vascular (usually right hepatic artery) injury is present in a large number of patients with laparoscopic bile duct injury (BDI)—more so in patients with a high (proximal) BDI and in patients who have had previous attempts at repair of the BDI. Vascular injury alone is NOT an indication for hepatectomy; it is the combination of bilio-vascular

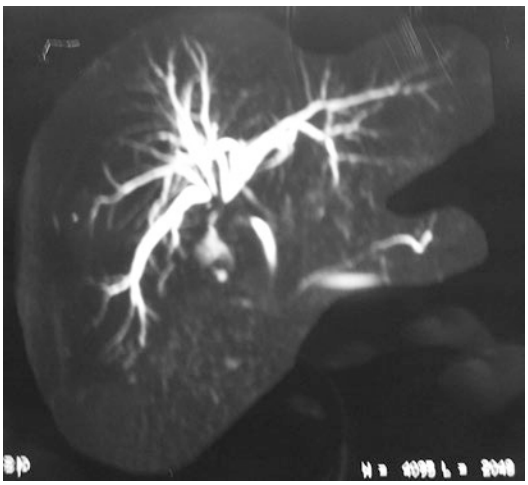

Fig. 14.1 High bile duct injury which may require (right) hepatectomy

Also see Invited Commentary on Surgical Management of Benign Biliary Stricture: Hepatectomy by Marcos V Perini (pp 181–182)

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Fig. 14.2 Recurrent stricture after hepatico-jejunostomy which may require (right) hepatectomy

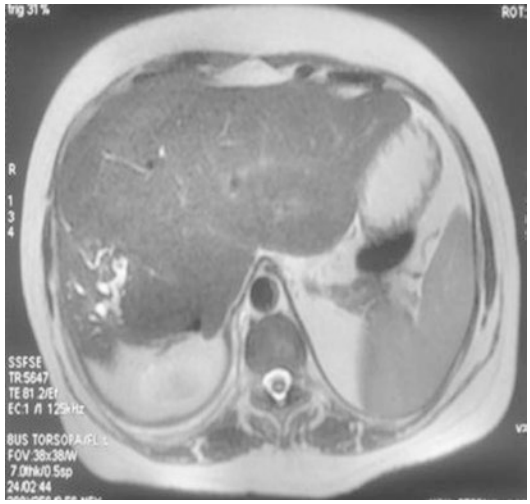

Fig. 14.3 Atrophy (crowding of bile ducts) of right lobe of liver—an indication for (right) hepatectomy

injury which may require hepatectomy. Patients with combined high biliary and hepatic arterial injury were 43 times more likely to need hepatectomy [1]. In patients with a high (proximal) biliary stricture and associated vascular injury, primary liver resection should be considered as an alternative option to hepatico-jejunostomy. In some patients with high (Bismuth Type IV) stricture, where fibrotic scarring extends into the intrahepatic right sectoral or even segmental ducts, a right hepatectomy may be a better option than an inadequate anastomosis to fibrosed scarred ducts. Stricture of isolated right sided segmental (Fig. 14.4)/sectoral (Fig. 14.5)/main hepatic (Fig. 14.6) duct (Bismuth Type V BBS) with recurrent attacks of cholangitis may be better managed with right segmental/sectoral/lobar hepatectomy rather than an unsatisfactory bilioenteric anastomosis to a scarred (fibrotic) bile duct. Recurrent stricture (on the right side) may also indicate a (right) hepatectomy.

In a report from France, vascular injury was present in 26/55 (47%) patients with BDI—12/55 underwent right hepatectomy because of right lobe atrophy [2]. In another report, four out of 84 patients with BDI and associated right hepatic artery injury required hepatectomy [3]. Thomson [4] reported that 14 out of 119 patients with Strasberg Type E BDI managed between 1984 and 2003 required either liver resection ($n = 9$) or transplantation ($n = 5$). Laurent [5] reported 18 out of 120 patients with BDI who required hepatectomy between 1987 and 2002—all had high injury involving the biliary ductal confluence; 13 had associated vascular (hepatic artery in 11) injuries and 15 had atrophy of the liver. Out of 41 patients with BDI who underwent HJ, three required segmental hepatectomy and one required liver transplant over a median follow-up of 9.3 years [6]. Ten (13%) of 76 patients with BDI managed in Tubingen Germany between 1998 and 2007 required hepatectomy [7]. Some of these reports, however, suffer from a referral bias to a transplant center. Hepatectomy was required in 31 out of 125 BDIs in New York State (2005–2010) [8]—this high rate of hepatectomy is probably because only major BDIs requiring surgical reconstruction were included. The true need for hepatectomy in patients with BDI is probably reflected in publications from large volume biliary centers. None of the 200 patients with BDI reported by the Johns Hopkins Hospital, Baltimore, MD, USA, however, required hepatectomy [9]. Mercado [10] reported ten (3%) hepatectomies in 355 patients with BDI. The Academic Medical Center (AMC), Amsterdam, Netherlands reported only 11 (1.4%) hepatectomies in 800 patients managed between 1990 and 2012 [11]. Only five out of 139 patients with BDI in Denmark required hepatectomy and one required liver transplant [12]. Only nine (6%)

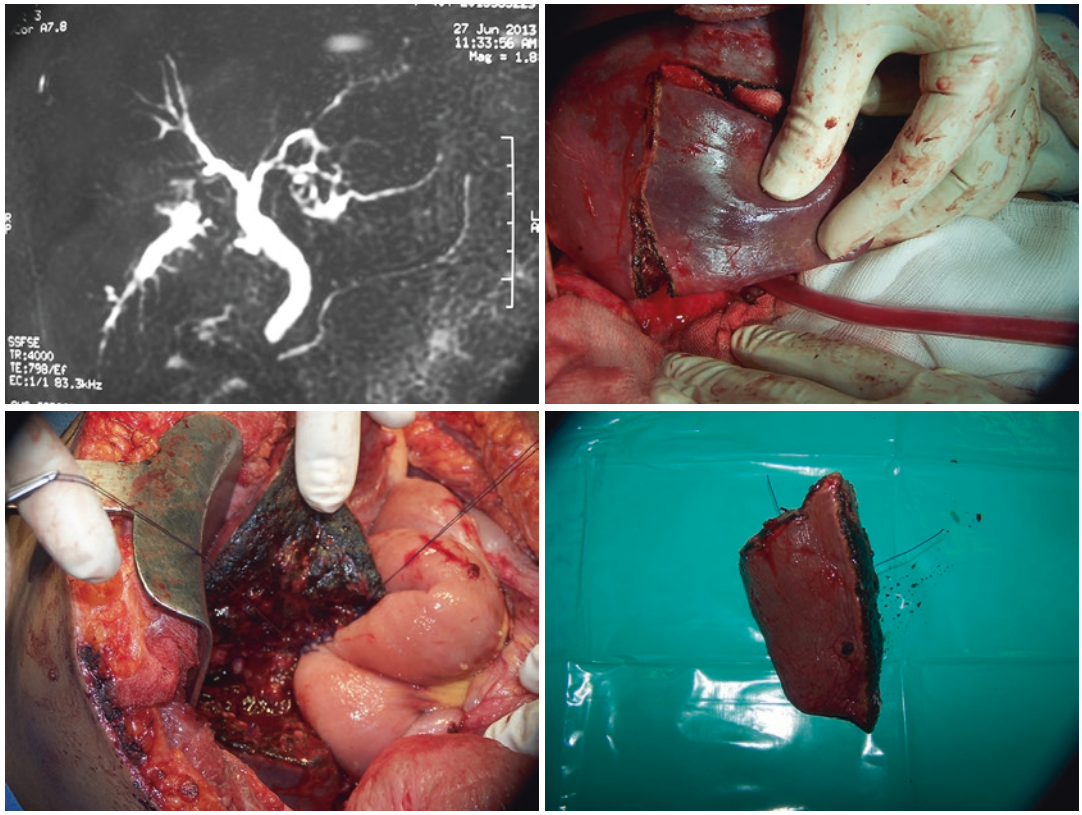

Fig. 14.4 (**a**). Isolated right segmental (VI) duct stricture (**b**). Marking of segment VI on surface of liver (**c**). Hepatico-jejunostomy to an intrahepatic duct (**d**). Specimen of segment VI hepatectomy

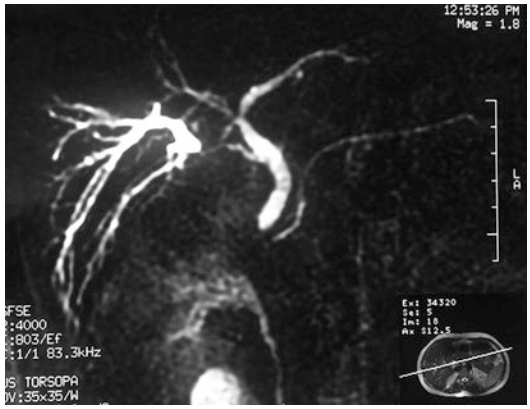

Fig. 14.5 Isolated right sectoral duct stricture which may require (sectoral) hepatectomy

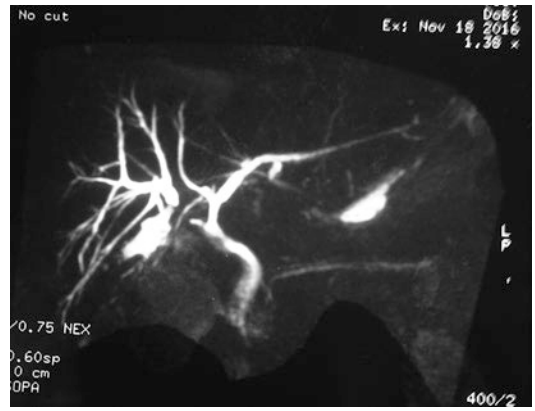

Fig. 14.6 Isolated right hepatic duct stricture which may require (right) hepatectomy

of 148 patients with BDI required hepatectomy [13]. We have performed 15 hepatectomies (eight as primary procedure and seven for failed repairs) in our experience of 782 cases of BBS repair (unpublished data). A review reported need for hepatectomy in 99 (5.6%) of 1756 patients with BDI reported in 31 publications [1].

Hepatectomy is usually required as a secondary procedure during the long-term follow-up—it was done at a median of 58 months [11] and

71 months [14]; usually after several (mean 2.4) attempts at repair [13]. Sixteen of 18 patients who required hepatectomy had already undergone an average of two repairs [5].

Most of the times when hepatectomy is required, it is right hepatectomy (Fig. 14.7) because the right hepatic duct is more difficult to access and repair, right hepatic artery injury is the most frequent vascular injury and failure (restricture) of the hepaticojejunostomy is seen more frequently on the right side [15]. After right hepatectomy, the left hepatic duct is better exposed and offers a technically easy and wide bilio-enteric anastomosis to a normal non-fibrotic non-strictured soft supple duct. Rarely, left hepatectomy may be required because of recurrent cholangitis and cholangiolytic abscess—this was the case in two out of ten cases in one series [10] and one out of nine cases in another [13]. Two out of 15 patients in our experience required left hepatectomy; one each due to cholangiolytic abscess and AHC.

Hepatectomy in BDI/BBS is technically more difficult and demanding than in hepato-cellular carcinoma (HCC) or in colo-rectal liver metastases (CRLM) for various reasons. Anatomy in the hepato-duodenal ligament may be distorted because of rotation caused by the atrophy-hypertrophy complex. Tissues in the hepato-duodenal ligament and liver hilum are scarred and shrunken because of inflammatory fibrosis—this makes dissection of the portal pedicle difficult. Bile is invariably infected because of biliary obstruction and previous non-surgical (endoscopic or percutaneous radiological) interventions. For these reasons, hepatectomy for BDI/BBS is associated with higher morbidity and mortality than when done for other indications, e.g., HCC or CRLM. Reported mortality of hepatectomy for BDI is 0/18 [5], 1/10 [10], 1/10 [7], and 3 (2 in hospital + 1 long term)/11 [11]. In our experience, 2 out of 15 patients, who underwent hepatectomy, died. In the long-term follow-up, patients can still require liver transplant [13] or die because of biliary complications [11]. Variable long-term results have been reported after hepatectomy for BDI/BBS. In one report, 17 out of 18 patients had excellent or good outcome at a median follow up of 8 years [5]. Six out of nine patients in another report had good/fair outcome at a median follow-up of 34 months [7]; eight out of nine patients had good result (one required transplant) at a mean follow-up of 69 months [13].

Liver parenchymal necrosis due to a vascular injury and consequent sepsis (liver abscess) may require emergency/urgent (within few weeks of BDI) hepatectomy if a significant volume of liver parenchyma is affected. Li et al. [7] reported ten hepatectomies for BDI—five were urgent; Booij [11] reported 11 hepatectomies for BDI—two were urgent. Emergency hepatectomy is associated with high mortality—4/9 in a review [16].

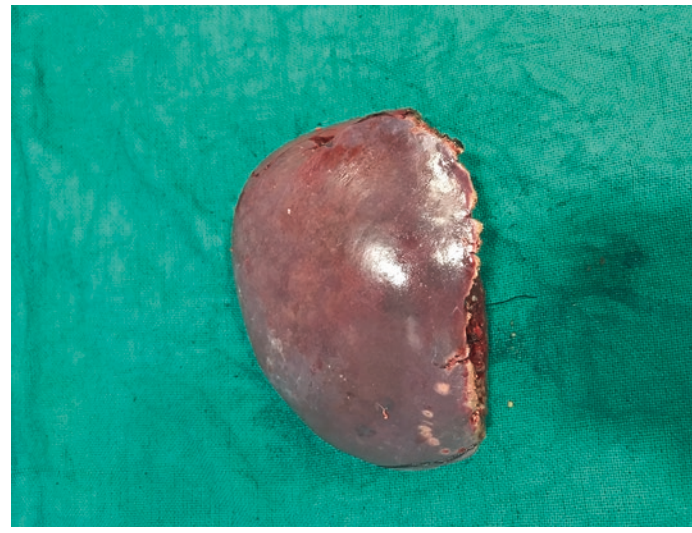

Fig. 14.7 Right hepatectomy for benign biliary stricture—unfortunately, the normal liver parenchyma has to be removed because no adequate normal hepatic duct is available for anastomosis

14.1 Liver Transplant

Patients with BBS who are not treated timely and properly may go on to develop secondary biliary cirrhosis (SBC) due to prolonged biliary obstruction, portal hypertension with recurrent variceal bleeding, and chronic liver failure (end stage liver disease ESLD), eventually requiring liver transplant. Liver transplant may also be required in a patient with recurrent (anastomotic) stricture after failure of a hepatico-jejunostomy. Repeated attacks of cholangitis, progressive jaundice, intractable pruritus, intractable ascites, repeated episodes of variceal bleeding, and poor quality of life are indications for transplant.

Thomson [4] reported five patients out of 119 with BDI (1984–2003) who were considered for liver transplantation for liver failure—two died while waiting for liver transplant and one died after liver transplant. de Santibañes et al. [17] of Argentina reported 20 patients with end stage liver disease (ESLD) secondary to BDI who were listed for liver transplant—four died while waiting for liver transplant and 16 received liver transplant—five died after liver transplant. Five out of 300 patients who received liver transplant at a center in Poland between 2002 and 2011 required it for SBC due to BDI [18]. In 18 centers in Argentina, 19 patients received liver transplant for BDI with 5 year and 10 year survival of 68% and 45%, respectively [14]. Addeo [19] reported six patients with BDI who underwent liver transplant between 1990 and 2012 at a median interval of 206 (range 96–384) months after the injury; all six patients had Strasberg type E injury and had multiple previous attempts at repair. They also reviewed the literature and identified 56 patients with BDI who were either listed for or underwent transplant —22% for acute liver failure. Out of these, 72% had had previous attempts at repair and 41% had associated vascular injury. Mortality of transplant for BDI was 34% and 5 year survival was 75%. Parrilla et al. [20] reported that as many as 27 patients with BDI were listed for liver transplant in 24 units of the Spanish Liver Transplantation Study Group between 1987 and 2010; 5 year survival in 20 patients who received an elective liver transplant for BDI was 68%.

There are several reports of need for emergency liver transplant in patients with complex biliary and vascular injuries resulting in massive hepatic necrosis and acute fulminant liver failure; seven such patients who needed an emergency liver transplant for BDI were reported from Spain [20]. Two of these seven patients died before transplant could be done. Results of emergency transplant were very poor—four out of five transplanted patients died within 30 days and only one survived beyond 30 days.

Sotiropoulos [21] reported a 36-year-old woman who died 7 days after a liver transplant was done for secondary biliary cirrhosis (SBC) as a consequence of failed repair of a BDI sustained during laparoscopic cholecystectomy; they also reviewed the published literature and found nine more deaths after liver transplant for BDI but observed that fatal post transplant outcome is infrequently reported.

Every surgeon who performs cholecystectomy should always keep in mind that if the patient sustains an inadvertent BDI, she may eventually end up having a hepatectomy or even liver transplant.

Invited Commentary on Surgical Management of Benign Biliary Stricture: Hepatectomy

Marcos V. Perini

The management of benign biliary stricture has evolved over the last few decades mainly due to the advances in interventional radiology and therapeutic endoscopy. It has moved from major operations, i.e., hepatico-jejunostomy, to a minimal non-surgical approach, in which endoscopic and percutaneous radiological interventions are used. However, in a small subset of patients in which the minimally invasive approach and the classical hepatico-jejunostomy have failed, liver resection can be used as a salvage procedure to avoid deleterious effects of recurrent episodes of cholangitis on liver function.

In this chapter, Prof Kapoor has outlined the role of hepatectomy in selected cases of benign biliary stricture in which the classical Roux-

en-Y hepatico-jejunostomy approach has failed. Liver resection is a salvage procedure in cases where the vascular supply to one side of the liver is inadequate for healthy healing biliary tissue, leading to poor drainage of bile from the contralateral liver, recurrent attacks of cholangitis, and the occurrence of liver abscess. This ultimately leads to atrophy of the ipsilateral hemi-liver and the development of secondary biliary cirrhosis in the contralateral side due to cholangitis. Therefore, rather than an alternative to hepatico-jejunostomy, liver transplant can be a requirement to avoid long-term liver failure, and end stage liver disease.

Arterial vascular inflow damage (associated with or without portal vein injury) and high bile duct injury are the main findings in this subset of patients. Due to anatomical and physiological communication in the arterial inflow between the right and left side of the liver and due to the lack of reliable tests to assess micro arterial circulation of the biliary system, we cannot predict in which patients a liver resection will be suitable apart from giving them the proof of time. The ones capable of supplying the contralateral liver will probably heal and the ones not capable will develop recurrent strictures. Following this rationale, hepatectomy should be performed in cases in which liver atrophy/abscess has developed over a period of time with the aim to remove the unhealthy tissue to avoid remnant parenchymal damage to the contralateral side.

Apart from being a challenging procedure itself, hemi-liver resection in the setting of previous hilar surgery can have major complications, like inadvertent arterial injury to the remaining liver and bleeding from the parenchymal transection. However, in well selected cases and in experienced hands, better exposure of the left biliary tree can be achieved and a safer and better hepatico-jejunostomy can be performed in healthy and well vascularized biliary tissue. Thus, the likelihood of a successful hepatico-jejunostomy to the remaining left hepatic duct will be higher.

To summarize, this chapter of Prof Kapoor reviews the evolving challenges of proposing a hemihepatectomy in patients with high bile duct injury associated with vascular injury. The treatment of this devastating disease with such a huge socio-economic impact is a remarkable achievement for hepato-biliary surgeons dealing with the complex (previous operations, malnourished, infected) young patient. It is never late to say that the first shot is the one with the highest likelihood to achieve success: therefore, experience and multi-disciplinary team work do matter in the management of such a complicated problem.

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15 Non-surgical Management of Benign Biliary Stricture

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All bile duct injuries (BDI) and benign biliary strictures (BBS) should preferably be managed at a biliary center which is equipped with the facilities and expertise for diagnostic and therapeutic endoscopy, diagnostic and interventional radiology, and reconstructive biliary surgery. Percutaneous radiological intervention, i.e., catheter drainage of biloma and percutaneous transhepatic biliary drainage, and endoscopic intervention, i.e., biliary stenting are invaluable in the management of an acute BDI and bile leak.

Endoscopic and percutaneous management has been successfully used in other benign biliary strictures, e.g., chronic pancreatitis, primary sclerosing cholangitis (PSC), post-transplant choledocho-choledochal anastomotic stricture. The author, however, does not believe in, practice or recommend non-surgical management of post-cholecystectomy BBS. Pitt [1] reported best overall success rates with surgery (88%) vs. endoscopy (76%) or radiology (50%) in multidisciplinary management of 528 patients with BDI over 18 years. Booij [2] also reported better long-term survival in surgically treated vs. endoscopically treated patients with BBS. Fong [3] also reported better survival with operative vs. endoscopic management in patients with BDI. Cumulative costs at 1 year were also less with operative vs. endoscopic approach (US$ 60,539 vs. 118,245). Non-surgical management is an option in patients with long-standing BBS resulting in secondary biliary cirrhosis (SBC) and portal hypertension who are poor candidates for surgical management; it can also be used as a bridge to liver transplant in these cases. Coagulation profile should be checked and coagulopathy, if present, corrected (with vitamin K and/ or fresh frozen plasma FFP) before any intervention is done.

15.1 Diagnostic Endoscopy

Endoscopic retrograde cholangiography (ERC), though very useful in the assessment of an acute BDI, is not of much help in patients with an established BBS as it does not provide good delineation of the proximal bile ducts which is of importance for the surgical repair of the BBS (Fig. 15.1). Delineation of biliary ductal anatomy, viz. site of the stricture (in relation to the biliary ductal confluence) is essential before its repair, i.e., hepatico-jejunostomy; this is best done with magnetic resonance cholangiography (MRC). The only indication of ERC in post-cholecystectomy jaundice is when a residual

Also see Invited Commentary on Non-Surgical Management of Benign Biliary Stricture by Guido C Costamagna (pp 190–191)

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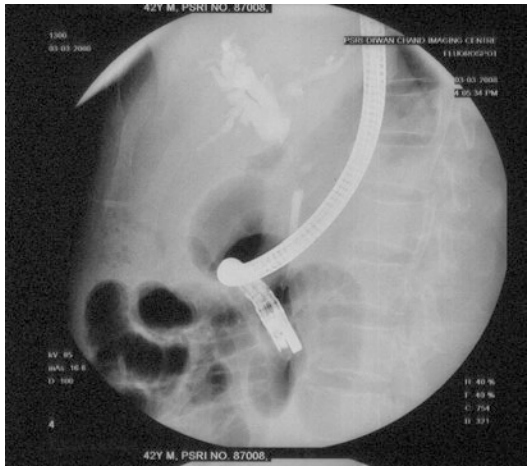

Fig. 15.1 Endoscopic retrograde cholangiography (ERC) has virtually no role in diagnosis of a benign biliary stricture as it does not well delineate the proximal bile ducts

common bile duct stone is suspected or cannot be ruled out—even then, an endoscopic ultrasonography (EUS) or MRC may precede the endoscopic intervention.

15.2 Therapeutic Endoscopy

Therapeutic endoscopic intervention plays an important role in the management of acute BDI to decompress the biliary system and reduce/stop the ongoing bile leak [4]. Endoscopic management is the treatment of choice for Strasberg Type A (e.g., cystic duct leak) and Type D (i.e., lateral CBD injury) BDI. It is not required in Strasberg Type B (occlusion of a segmental/sectoral duct) as there is no bile leak. It does not help in Strasberg Type C (bile leak from an isolated duct) and is not technically feasible in Strasberg Type E (circumferential) injury.

Some endoscopists advocate endoscopic management of BBS because it is easy, less invasive, safe, and repeatable. Endoscopic management is, however, possible in BBS with intact biliary ductal continuity (following incomplete/partial/lateral BDI) only and is not possible in patients with BBS following a complete BDI (transection or excision) with no ductal continuity (Fig. 15.2). Endoscopic management may be recommended for delayed (ischemic), low (Bismuth Type I or II),

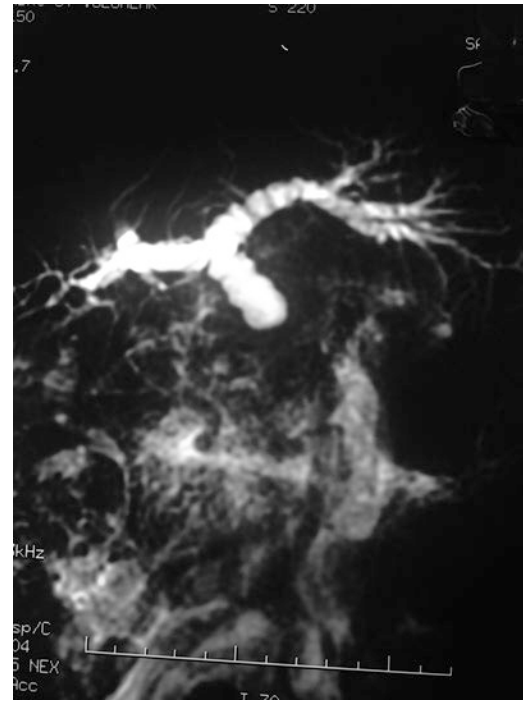

Fig. 15.2 A complete bile duct injury with no ductal continuity is not suitable for non-surgical intervention; a rendezvous approach (percutaneous + endoscopic) may work in some cases as an exception

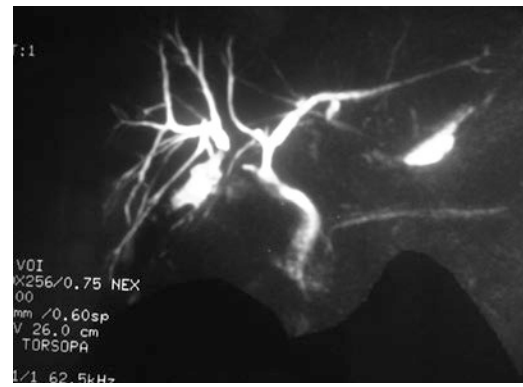

Fig. 15.3 Isolated (right) duct stricture which is not in continuity with the main biliary ductal system is not amenable for non-surgical intervention

and short segment BBS or in patients who are at high risk for surgery because of comorbidities and those with secondary biliary cirrhosis (SBC) and portal hypertension. Endoscopic management is not suitable for Bismuth Types IV and V BBS; it is also not applicable in isolated stricture of aberrant right ducts (Fig. 15.3).

Therapeutic endoscopic intervention may be used for BBS if one believes in endoscopic management. Endoscopic management of BBS involves papillotomy and passage of a fine (0.018–0.035 inch diameter) flexible malleable hydrophilic guide wire with straight or curved (J-shaped) tip across the stricture (this may, however, be difficult because of fibrosis and angulation), pneumatic balloon (4–8 mm, inflated to 4–20 atmospheric pressure) dilatation, stenting and frequent (every 3 months) exchange of stents for a period of 1 year. A single large (10–12 F) stent is placed in the first go; in further interventions, multiple stents can be placed (Fig. 15.4).

In the Amsterdam protocol [5], multiple (1–3, usually 2) 10 F polyethylene stents are placed after endoscopic balloon dilatation of a BBS. Repeat ERC is required every 3–4 months to evaluate the stricture. Stents are changed every 3 months as they tend to get clogged over time and are kept for 12 months. In the Rome protocol [6], increasing number of stents are placed at repeat interventions every 3–4 months.

Multiple interventions increase the morbidity and costs of treatment and decrease patient compliance. Stent block due to clogging and recurrent cholangitis, necessitating an earlier than scheduled reintervention, is the commonest complication; stents may also displace (migrate). Other complications of endoscopic intervention include creation of a false passage, bleeding, acute pancreatitis, acute cholangitis, and duodenal perforation. Cholangitis is more common if the guide wire has been passed beyond the stricture but dilatation can not be achieved and stent can not be placed; in such a situation, an endoscopic naso-biliary drain (ENBD) (6F) should be placed to prevent cholangitis. Non-compliant patients suffer from the risk of complications related to a forgotten stent. Non-response after 3–4 attempts at dilatation and recurrence after removal of stents is an indication for surgical repair. Stents in the common bile duct cause inflammatory fibrosis and thickening of the bile duct [7] making subsequent surgical repair more vulnerable to failure.

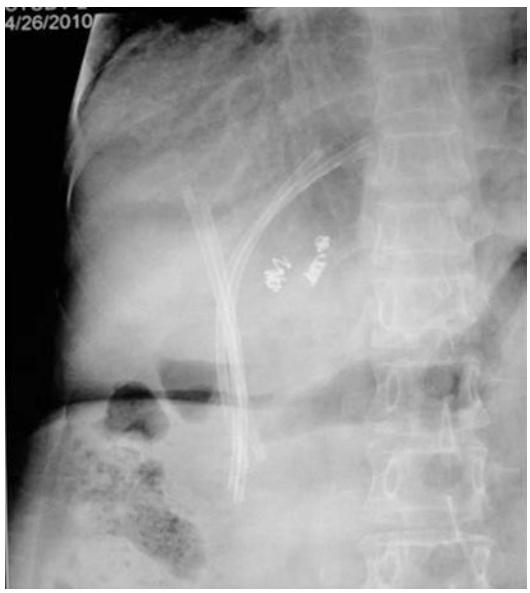

Fig. 15.4 Multiple stents placed as a part of endoscopic management of a benign biliary stricture

Patients should be followed up with LFT, US and isotope hepato-biliary scintigraphy every 6–12 months after stent removal. A large number of such-treated strictures, however, restricture and need further dilatation and stenting or surgical repair. Dilatation is, therefore, inferior to a surgical repair in the form of a hepaticojejunostomy and should only be performed in patients who are poor risk for surgery.

Stewart [8] reported 29 patients who underwent endoscopic management of biliary stricture—it was successful in only seven patients; the duration of illness in patients who underwent endoscopic treatment was longer than those who underwent surgical treatment (584 vs. 177 days). Csendes [9] reported 94 patients managed with multiple endoscopic stents (5–10 F) over 8 months—excellent/good outcome was achieved in 76% of 49 cases who had 3 years follow-up. In a report from the Academic Medical Center (AMC), Amsterdam, Netherlands, a total of 96 eligible patients were treated endoscopically with a mean of 2 (range 1–4) stents. Stent-related morbidity was seen in 23%. The median duration of stenting was 12 (range 2–96) months. After a mean follow-up of 6 (range 0–20) years the overall success rate was 67% after stenting and 82% after additional treatments. After stent removal, as many as 20% of patients developed recurrent stricture within 2 years of stent removal [5].

Vitale [10] reported 48 patients with Amsterdam Type B or C BDI treated with balloon dilation plus 3 monthly endoscopic stenting for 12 ± 10 months. One patient with a large lateral injury developed bile peritonitis after stent placement and required laparotomy. Another patient required surgical intervention for severe cholangitis during follow-up. The endoscopy protocol could be completed in 46 patients. Pancreatitis occurred in 8% of patients. At a follow-up of 31 ± 24 (range 2–96) months after stent removal, 10 out of 46 (22%) had recurrent stricture—6 were treated with further endoscopic stenting and 4 required hepatico-jejunostomy. The authors concluded that endoscopic stenting is an effective treatment for post-cholecystectomy BBS and is an alternative to hepatico-jejunostomy in selected patients.

Fatima [11] reported 13 out of 159 BDIs managed with sustained endoscopic therapy (median stent time of 7 months) at the Mayo Clinic from 1998 to 2007; it was successful in 10 (77%). Kuroda [12] reported 121 + 64 (range 31–254, median 120) months follow-up in 21 patients with postoperative bile duct strictures treated endoscopically—overall long-term success was 20/21 (95%). Tuvignon [13] reported 96 patients; mean 1.9 ± 0.9 (2–4) stents were placed for 12 (2–96) months. During a follow-up of 6.4 ± 3.8 (0–20) years, success was achieved in 64 (67%) after the period of stenting and in 79 (82%) patients after additional treatment.

A self-expanding metal stent (SEMS), made of Nitinol (nickel titanium alloy), used very frequently for palliation in unresectable malignant biliary strictures, may be used for definitive management of a BBS also but only when surgery is definitely and permanently contraindicated, e.g., in a poor risk elderly patient with severe uncontrollable comorbidities (Fig. 15.5). Bonnel [14] used metal stents in 25 patients with recurrent (anastomotic) strictures; more than half developed recurrent problems during follow-up. A review of 400 patients with various types (including post-cholecystectomy) of benign biliary strictures in 37 studies showed that only 25% of such stents remained patent at 3 years; in 123 post-cholecystectomy strictures, patency was only 38% at 2 years [15].

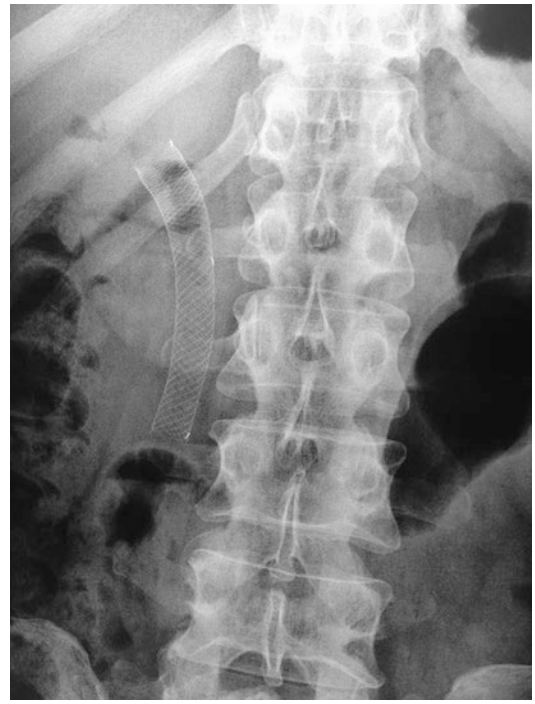

Fig. 15.5 Self expanding metal stent (SEMS) is not recommended for management of a benign biliary stricture except in a patient who is not fit for surgical repair for some reasons

Costamagna [6] strongly advises against placement of uncovered metal stents in benign biliary strictures. Removable fully covered SEMS are being increasingly used in benign biliary strictures, e.g., post-cholecystectomy, chronic pancreatitis, post liver transplant. Covered stents are, however, more prone to migration as compared to uncovered stents; anchoring flaps and flared ends reduce the risk of migration. A large multinational study involving 13 centers reported use of fully covered SEMS, which could be removed, in 187 patients with various types of benign biliary strictures. Removal success in post-cholecystectomy patients, however, was low (61%). Even after a short median follow-up of 20 months, recurrence of the stricture occurred in 27% patients. Moreover, stent insertion or removal related serious complications (most common being cholangitis) occurred in 27% of patients [16]. A recent prospective multicenter European study reported use of fully covered SEMS in 43 patients with benign biliary strictures but only 4 were post-

cholecystectomy [17]. Hu [18] reported use of short fully covered SEMS (FCSEMS) with a retrieval lasso in 45 patients with BBS; the stent could be removed in 33 patients - stricture resolved in 30 out of these 33 patients. Gimenez [19] reported use of biodegradable stents which dissolve on their own over 1–2 years in 13 patients with recurrent biliary strictures.

15.3 Rendezvous

In difficult cases, a combined (rendezvous) percutaneous plus endoscopic approach may be required. In case endoscopic cannulation is not possible, e.g., in a tight stricture, the interventional radiologist can pass a guide wire via percutaneous transhepatic route through the stricture in the duodenum where it is retrieved by the endoscopist. Shin [20] reported a reverse rendezvous procedure in a patient with complete transection of the common bile duct—a guide wire was placed by the endoscopist through the distal common bile duct into the biloma—it was then retrieved by the interventional radiologist through percutaneous transhepatic route.

The Academic Medical Center (AMC), Amsterdam, Netherlands reported use of rendezvous approach in 47 (including 31 with complete transection, i.e., Amsterdam Type D/Strasberg Type E) out of 812 BDIs seen between 1995 and 2016; primary success was 94% (44/47). There was no 90-day mortality; after a median follow-up of 40 (IQR 23–54) months, final success rate was 55% (26/47) [21].

15.4 PTBSD (Percutaneous Transhepatic Balloon Stricture Dilatation)

A high (intrahepatic) stricture in the right hepatic duct or one of the right sectoral ducts may be difficult to access surgically and is better treated by percutaneous transhepatic balloon stricture dilatation (PTBSD). PTBSD is the initial treatment of choice for recurrent (anastomotic) stricture after hepatico-jejunostomy also (Fig. 15.6).

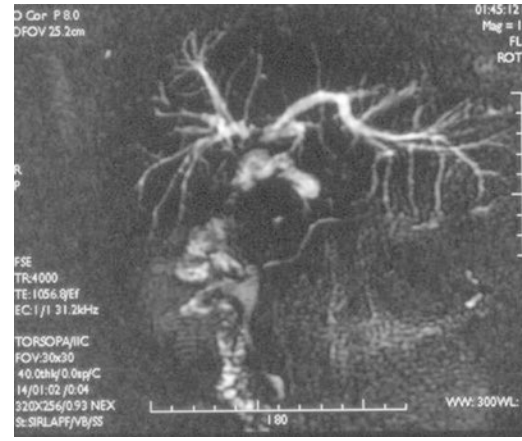

Fig. 15.6 Recurrent (anastomotic) stricture after hepatico-jejunostomy is best treated with percutaneous transhepatic balloon stricture dilatation (PTBSD)

PTBSD involves an initial transhepatic catheter access to the dilated intrahepatic ducts, passage of a guide wire through the catheter across the stricture, and passage of an inflatable balloon over the guidewire.

The Johns Hopkins Hospital, Baltimore, MD, USA group managed 51 (out of 148) post laparoscopic cholecystectomy BDIs with percutaneous dilatation and stenting—30 out of 51 patients had a successful outcome over a median follow-up of 77 months [22]. Complications of percutaneous intervention include bleeding (intraperitoneal, intraparenchymal, and intrabiliary—hemobilia) and bile leak.

Park [23] reported sclerotherapy with acetic acid for an isolated right posterior sectoral duct injury followed by coil embolization of the fistulous tract.

15.5 Anastomotic Stricture

An anastomotic stricture after an end-to-end repair, i.e., bilio-biliary anastomosis (BBA) of a transected or divided common bile duct is an ideal indication for endoscopic balloon dilatation and stenting. An anastomotic stricture following a bilio-enteric anastomosis (BEA) in the form of a hepatico-jejunostomy (HJ) responds very well to percutaneous transhepatic balloon stricture dil-

atation (PTBSD) which is the preferred treatment of choice for an HJ anastomotic stricture. Kim [24] treated 21 bilio-enteric anastomosis strictures with percutaneous transhepatic cholangioscopic dilatation. Strictures recurred in 8 (38%) patients at a mean of 17 (2–38) months; final success rate after repeat interventions was 81% but follow-up (mean 33, range 12–19 months) was short. The Academic Medical Center (AMC), Amsterdam, Netherlands reported 15 anastomotic strictures in 151 patients over a median follow-up of 4.5 years—12 were treated by percutaneous transhepatic dilation and only 3 required reoperation [25]. In a later report from the same group, 33/37 recurrent strictures were treated with percutaneous dilatation and only 4 were reoperated [26]. Most interventional radiologists prefer placement of multiple (usually three—one each in right anterior and posterior sectoral and left hepatic ducts) catheters (Fig. 15.7); multiple sessions of dilatations are invariably required. The duration of stenting in 40 patients who were treated endoscopically at the Academic Medical Center (AMC), Amsterdam, Netherlands for failure of an intraoperative end-to-end anastomosis (EEA) done elsewhere was 359 (39–1355) days; the median number of stent replacements was 5 (range 1–15) [27]. Benkabbou [28] managed 44 failures of hepatico-jejunostomy in Hospital Paul Brousse, Villejuif France between 1996 and 2006—26 patients were reoperated (re hepatico-jejunostomy 22, hepatectomy 4) and 18 were treated percutaneously. Overall success of 89% was achieved over a follow-up of 49 ± 40 months. Parlak [29] reported endoscopic management in 156 patients with a median follow-up of 6.5 years; recurrence occurred in 18 (11%) patients at a short median follow-up 9 months.

Fig. 15.7 Multiple percutaneous transhepatic biliary catheters placed as a part of percutaneous transhepatic balloon stricture dilatation (PTBSD)

Non-surgical (percutaneous and endoscopic) management plays an important role in the management of acute BDI. Established BBS should, however, be treated with surgical hepatico-jejunostomy. Treatment of choice for anastomotic stricture after end-to-end repair is endoscopic balloon dilatation and after hepatico-jejunostomy is percutaneous transhepatic balloon stricture dilatation.

Invited Commentary on Non-Surgical Management of Benign Biliary Strictures

Guido Costamagna

Benign biliary strictures (BBS) may occur as a result of bile duct injury (BDI) during cholecystectomy or other biliary tract surgery, after liver transplantation with choledocho-choledochal anastomosis, or in the course of other benign diseases involving the biliary tract such as chronic pancreatitis and primary sclerosing cholangitis (PSC). More rarely, benign strictures of the biliary tract may reflect IGG4 related cholangitis or long-standing inflammation resulting from common bile duct stones. Due to their different etiology, leading to various pathophysiology patterns, it appears evident that benign biliary strictures cannot be considered as a single entity, but deserve separate evaluation. This commentary will be focused only on benign biliary strictures occurring as a result of biliary injury during cholecystectomy or other biliary tract surgery.

Schematically, three main clinical pictures are typical of a bile duct injury: two of them, i.e., bile leak (choleperitoneum) and external biliary fistula, appear in the immediate postoperative phase, while the third one, i.e., cholesta-

sis and jaundice, with or without the features of acute cholangitis, may present either early in the postoperative period or at distance from the operation. The clinical picture may also combine these main symptoms in various ways. When the diagnosis is made in the immediate postoperative period or in the early days after surgery, the main objective of treatment should be to stop/control the bile leak and to avoid any septic complications at the peritoneal level or within the biliary system. Non-operative treatments (percutaneous catheter drainage of choleperitoneum or localized bile collections and endoscopic or, less frequently, percutaneous transhepatic drainage of the biliary tree) are commonly accepted as the first line management strategy in this setting, the only partial limitation being a complete transection of the common bile duct, which may require early surgical repair. When a benign biliary stricture has established, as a consequence of an early recognized and treated bile duct injury or as an evolution of an undiagnosed damage at a distance from surgery, various treatment options are available. Historically, surgical reconstruction of the biliary continuity with hepatico-jejunostomy has been the only available option able to provide good long-term results. With the development of non-operative interventional techniques, more options are available today and must be taken into consideration, if possible in the setting of a multi-disciplinary evaluation with surgeons, therapeutic endoscopists, and interventional radiologists. If the stricture does not completely interrupt the continuity of the common bile duct and a guidewire can be passed through it, an endoscopic treatment entailing progressive dilation of the stenosis with plastic stents can almost always be undertaken: in our experience, the level of the stricture and its complexity (involvement of the main biliary ductal confluence) does not influence the early and late results of treatment. If well conducted, the treatment strategy of placing an increasing number of large bore plastic stents until complete disappearance of the stricture over an appropriate time frame (usually 1 year) is able to heal more than 95% of the patients without procedure related mortality [30] and to maintain the results after more than 10 years of follow-up in 90% of the successfully treated patients (unpublished data on 151 patients with a mean follow-up of more than 10 years) [31]. Furthermore, the majority of patients with relapsing symptoms due to recurrent stricture may be very easily retreated endoscopically with excellent results. The same strategy, when applicable, may be used also in strictures involving only an aberrant right duct (Type V of the Bismuth classification) [32]. The main drawback of such a strategy is that an effective recall (follow-up) system must be put in place to avoid delayed stent exchanges and patients need to be very compliant: this is not always easy to obtain, usually when the patient lives far away from the tertiary biliary center performing the endoscopic procedures. Another limitation is the experience of the endoscopic team: accurate interpretation of the cholangiographic findings, insertion of the guidewire into the selected intrahepatic biliary segmental ducts, and effective insertion of the required number of stents, are the keystones of a successful treatment. In our experience, surgical repair with hepatico-jejunostomy and endoscopic treatment with multiple plastic stents are not competing strategies, but complementary to each other; careful evaluation of every single case by a multi-disciplinary team with experience in both techniques is of paramount importance for the good outcome of the treatment. Biliary injuries may occur everywhere: in big academic hospitals as well as in peripheral medical institutions, but management of these difficult patients should be centralized in specialized referral centers where all the diagnostic and therapeutic options are available.

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16 Follow-Up After Repair of Bile Duct Injury

Vinay K. Kapoor

ALL patients who are suspected to have or who have sustained a bile duct injury (BDI) during cholecystectomy and *ALL* patients who have bile leak (even if it is shown to be a minor leak from the cystic duct stump or a cholecysto-hepatic or subvesical duct in the gall bladder bed) following cholecystectomy need to be followed up. Even the so-called minor (including Strasberg Type A, managed conservatively or non-surgically with endoscopic/percutaneous intervention and Type D, repaired over a T-tube) injuries should be followed up with liver function tests (LFT), ultrasonography (US), and isotope hepato-biliary scintigraphy to ensure that a benign biliary stricture (BBS) is not forming.

Every bilio-biliary anastomosis (BBA), i.e., end-to-end repair or bilio-enteric anastomosis (BEA), i.e., hepatico-jejunostomy (HJ) is at risk to restricture, hence the need for follow-up (Fig. 16.1). Majority of anastomotic strictures developed within 2 years but they may occur up to 10 years [1]. 25 out of 43 end-to-end repairs required further surgery over 10 years [2]. Anastomotic stricture occurred at a median of 12 (2–14) months after repair—71% of 42 strictures occurred within 2 years [3]. Ahrendt [4] reported that two-thirds of restrictures occurred within 3 years and 80% within 5 years. The longer the follow-up, more patients develop problems; when 33 patients who underwent repair were followed for a minimum of 3 (mean 6.5) years, 12 had further episodes of cholangitis [5]. Restricture developed at a median of 13 months [6]. 42 (30%) of 139 patients who underwent HJ developed an anastomotic stricture at a medium of 12 (2–141) months; two-thirds of the anastomotic strictures developed within 2 years [7]. AbdelRafee [8] reported 120 patients who underwent HJ between 1992 and 2007 and had a median follow-up of 149 (range 20–246) months; they observed that patients could develop restricture even after 17 years; hence, the need for long-term (preferably 10–20 years; ideally life-long) follow-up. At the Academic Medical Center (AMC), Amsterdam, Netherlands, clinically relevant restrictures were seen in 37 (13%) patients who underwent HJ at a median follow-up of 10.5 years [9]. Table 16.1 shows results of HJ for BDI/ BBS in various reports.

ANECDOTE: *The Author (VKK) has launched an online education portal called Prashna India http://prashna-india.weebly.com/ (Sanskrit: prashna = question) wherein experts conduct live online interactive sessions called Ru-Ba-Ru (Urdu: ru-ba-ru = face-to-face) with students. In one of the sessions,*

Also see Invited Commentary on Follow-Up after Repair of Bile Duct Injury by Graeme J Poston (pp 202–203)

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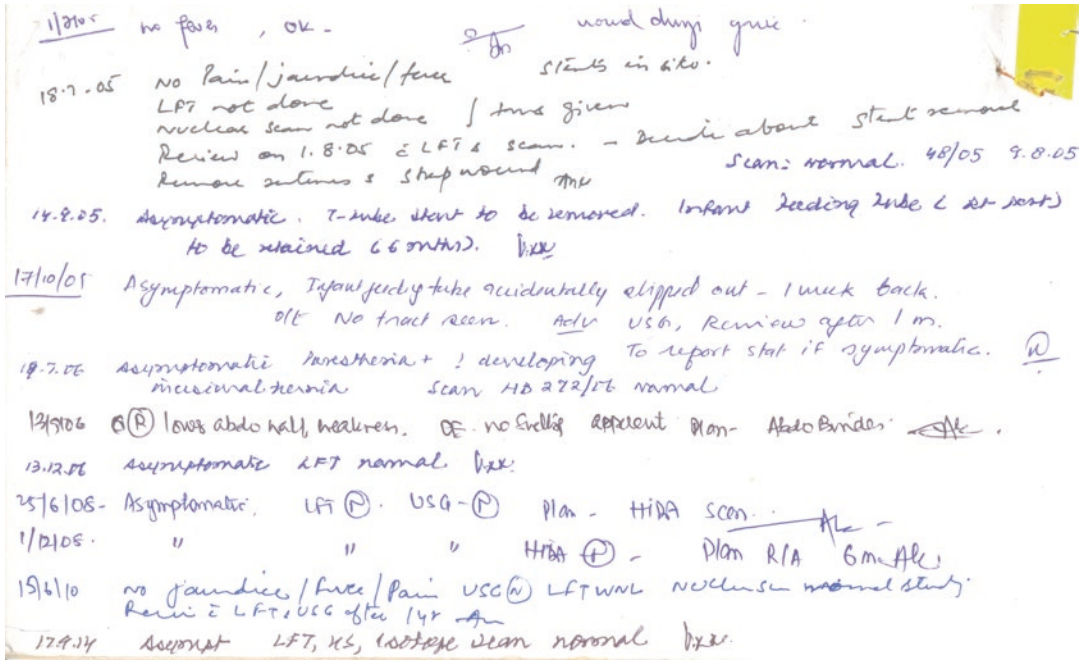

Fig. 16.1 Follow-up card of a patient who underwent hepatico-jejunostomy. All patients with bile duct injury/benign biliary stricture who undergo repair need long-term, preferably life-long, follow-up

Table 16.1 Results of hepatico-jejunostomy for bile duct injury/benign biliary stricture

Series	Duration	No.	Mortality	Follow-up	Failure	Final success
Chapman [10]	12 years	108	2%	Mean 7.2 (1–13) years	25 (23%)	90 (87%)
Stewart and Way [11]		84			54 (63%)	
Murr [3]	1990–1997	59		3.7 ± 0.3 years	5 (9%)	91%
Lillemoe [12]	1990–1999	142	0.6%	58 months	13 (9%)	91%
Schmidt [13]	1990–2002	54		Median 45 months	19%	42/46 (92%)
Bektas [14]	1990–2005	74	2 (3%)	1.5–15 years		53%
De Reuver [15]	1991–2005	151		Mean 5.3, median 4.5 years	15 (10%)	
Pottakkat [16]	1989–2007	364	1.5%	Median 61 (6–212) months	30 (8%)	92%
Winslow [17]	1992–2006	113		4.9 years	5%	
Holte [18]	1994–2008	41		Median 9.2 years	10 (24%)	
Gupta [19]	2001–2010			2.6 (0.16–6) years		62/75 (83%)
Pitt [20]	18 years	104			18%	99%
Bansal [21]	2005–2011	138	1%	54 (6–83) months		92%
Ibrarullah [22]	2004–2012	59		40 months	1 death, 1 failure	
Mishra [23]	2005–2011	107		30 (18–80) months	4	92%
Booij [9]	1991–2016	281	2 (0.7%) 90 day	10.5 years	37 (13%)	

the students presented the case of a 47-year-old lady who had undergone HJ for post-cholecystectomy BDI and EBF 16 years ago and now presented with recurrent cholangitis. MRC showed a high stricture with involvement of the right sectoral ducts (Fig. 16.2).

Patients with BBS can develop complications of biliary obstruction and die of biliary causes in

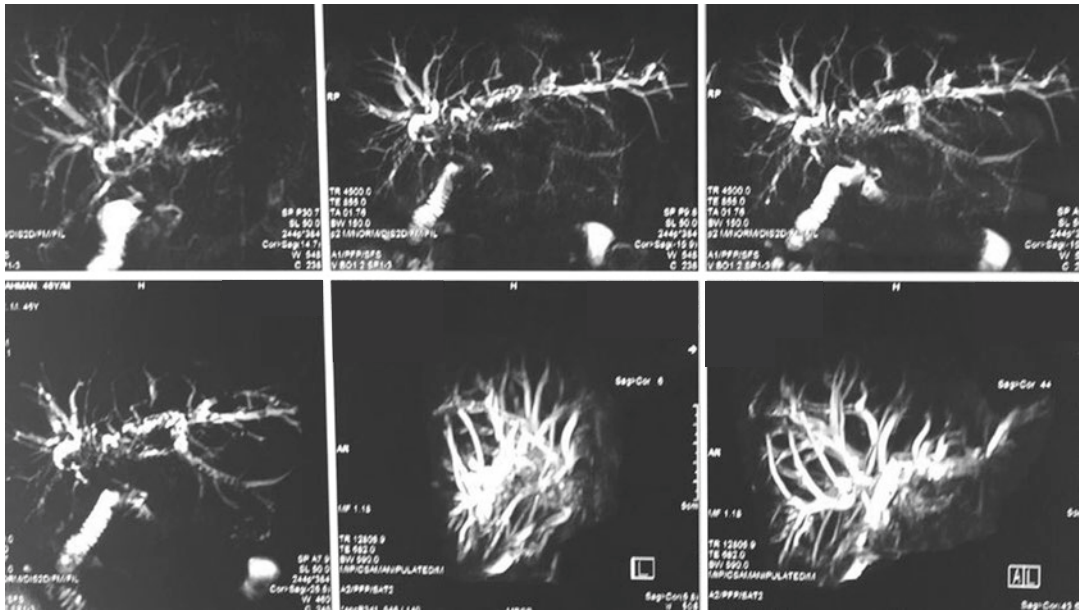

Fig. 16.2 Recurrent (anastomotic) stricture 16 years after hepatico-jejunostomy

the long-term follow-up. Chapman [10] operated on 122 patients with BDI at the Hammersmith Hospital, London, UK, over 12-year period—7 died in hospital and 7 more died of related causes during mean follow-up of 7.2 years. In another report, long-term biliary complications developed in 10 (19%) out of 54 patients, 5 developed secondary biliary cirrhosis (SBC) and 3 patients died of biliary complications during median follow-up of 62 months [13]. 5 out of 47 patients died during follow-up [24]. Walsh [6] reported 84 patients who underwent HJ between 1990 and 2005; over a mean follow-up of 67 months, 5 developed chronic liver disease (including 1 on wait list for liver transplant) and 3 patients died. Mortality at 1 year in 747 patients who sustained a BDI during cholecystectomy was 3.9% vs. 1.1% in those who did not [25]. 6 out of 139 patients who underwent HJ died of biliary causes during long-term follow-up [7]. Patients (n = 500) who required bile duct reconstruction because of a BDI sustained during cholecystectomy (n = 572,223) performed in England between 2001 and 2013 were 10 times (6% vs. 0.6%) more likely to die within a year [26]. Booij [27] reported BDI related mortality in 28 (3.5%) of 800 cases.

Follow-up after repair of a BDI/ BBS can be

1. Clinical—for features of cholangitis such as jaundice, fever, and pruritus.
2. Biochemical—liver function tests (LFT), especially serum alkaline phosphatase (ALP) and gamma glutamyl transpeptidase (GGTP).
3. Ultrasonography (US) to detect any evidence of biliary obstruction, e.g., intrahepatic biliary radical dilatation (IHBRD) or cholangiolytic abscess; sludge and stones may also be seen in the intrahepatic ducts.
4. Isotope hepato-biliary scintigraphy using hepato-imino diacetic acid (HIDA), butyl iminodiacetic acid (BULIDA), or mebrofenin to evaluate hepatic uptake and biliary excretion.
5. Cholangiography (preferably magnetic resonance cholangiography, MRC) is indicated if there is any suspicion of biliary obstruction on above parameters. MRC will show the anastomotic stricture (Fig. 16.3); it may also show cholangiolytic abscess (Fig. 16.4) and stones and sludge in the dilated proximal intrahepatic bile ducts (Fig. 16.5). Percutaneous transhepatic cholangiography (PTC) is indicated only if therapeutic intervention, viz. balloon dilatation and stenting is planned.

Fig. 16.3 Magnetic resonance cholangiography (MRC) showing recurrent (anastomotic) stricture after hepaticojejunostomy (See Fig. 16.9 also)

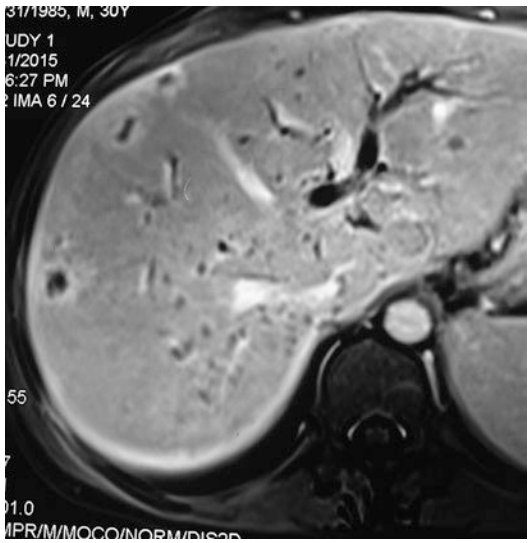

Fig. 16.4 Magnetic resonance cholangiography (MRC) showing cholangiolytic abscess secondary to recurrent (anastomotic) stricture

Mayo Clinic [28] criteria are most commonly used to assess the outcome of repair of a BBS. They are as follows:

(a) Excellent—asymptomatic (no symptoms of biliary obstruction), normal liver function tests (LFT)
(b) Good—largely asymptomatic but occasional fever or pain, transient mild derangement of liver function tests (LFT)

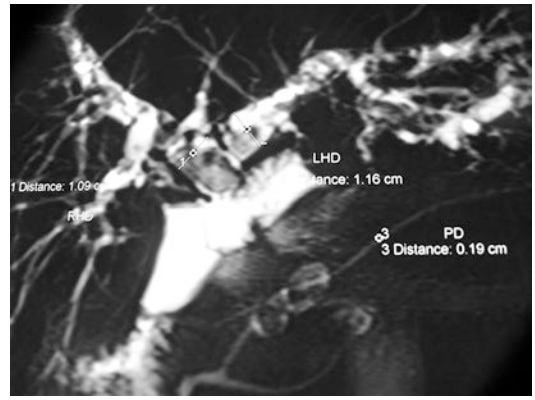

Fig. 16.5 Magnetic resonance cholangiography (MRC) showing stones and sludge in the intrahepatic ducts proximal to recurrent (anastomotic) stricture (See Fig. 16.10 also)

(c) Fair—symptomatic (cholangitis, i.e., fever and jaundice) with persistently abnormal liver function tests (LFT)
(d) Poor—need for further intervention (endoscopic, percutaneous radiological or surgical)

Excellent and good outcomes are classified as success of repair; fair and poor outcomes are classified as failures of repair.

Patients with secondary biliary cirrhosis (SBC) on liver biopsy at the time of repair of the biliary stricture may continue to have deranged LFTs in spite of a patent anastomosis. We had earlier suggested sub-classification of Grade B into B2, i.e., derangement of LFTs but no symptoms of cholangitis due to persistent preexisting secondary biliary cirrhosis (SBC) in presence of a patent biliary-enteric anastomosis [29]. The author now suggests to add E (death during follow-up because of a biliary complication) to this classification.

Chapman [10] reported results of biliary stricture repair as good (no further intervention) or poor/unsatisfactory (requiring further intervention).

Tocchi [30] defined outcome as:

1. excellent (no symptoms)
2. good (transient symptoms)
3. fair (medical therapy required)
4. poor (recurrent stricture)

The Johns Hopkins Hospital, Baltimore, MD, USA group [4] classified results of biliary stricture repair as:

1. Excellent (no biliary symptoms)
2. Good (mild biliary symptoms not requiring invasive investigations)
3. Poor (requiring therapeutic intervention)

Iannelli [31] defined success as no need for further intervention either non-surgical, i.e., endoscopic or percutaneous, or surgical.

Pitt [20] defined successful outcome as no need for further intervention for 12 months.

In the absence of a standard or widely accepted way of reporting outcomes of treatment of biliary injuries, an international group of surgeons, biliary endoscopists, and interventional radiologists have recently reported a proposal to standardize terminology and reporting of results of treating biliary injuries. This includes definition of patency, definition of index treatment periods, grading of severity of biliary injury, grading of patency, metrics, comparison of surgical to non-surgical treatments, and presentation of case series [32].

16.1 Factors for Failure

Several factors have been found to predict recurrence of a stricture after previous repair. The level of stricture is the most important predictor of outcome—low (Bismuth Types I and II) strictures have better results (low failure rates) than high (Bismuth Types III and IV) strictures (Fig. 16.6) which have higher failure rates. Bismuth Type IV stricture and 3 or more previous attempts at repair were risk factors for failure of repair [10]. Use of anastomotic stents is controversial but if they are used, the stents should be left in situ for long, with regular frequent (every 3 months) exchanges; HJ was more likely to fail if stents were left for less than 4 months [33]. Presence of peritonitis at the time of repair is also a factor for failure [13]. Walsh [6] reported 35% restricture rate in Strasberg E4 injury vs. 9% in E3 and 0% in E1 + E2 injury. Proximal (high) BDI, associated vascular injury, delayed referral to a biliary center and early repair were predictors of worse outcome [15]. Repair in presence of a fistula, whether external or internal, also predicted failure [34]. Other factors for failure are presence of cirrhosis, portal hypertension, atrophy hypertrophy complex (AHC), and previous attempts at repair, especially in the form of HJ. Associated vascular (especially hepatic artery) injury increases the risk of anastomotic stricture due to ischemia of the proximal ducts. Repairs in the hands of a general surgeon have higher failure rates. Postoperative anastomotic leak also predisposes to an anastomotic stricture. Ortiz-Brizuela [35] identified 117 episodes of acute cholangitis (suggestive of anastomotic failure) in 70 out of 524 patients who underwent a biliary-enteric anastomosis for BDI between 2000 and 2014; patients with preserved biliary ductal confluence were less likely (OR 0.5 CI 0.3–0.9) while those who had postoperative biliary complications were more likely (OR 2.6 CI 1.4–4.7) to develop acute cholangitis during the follow-up.

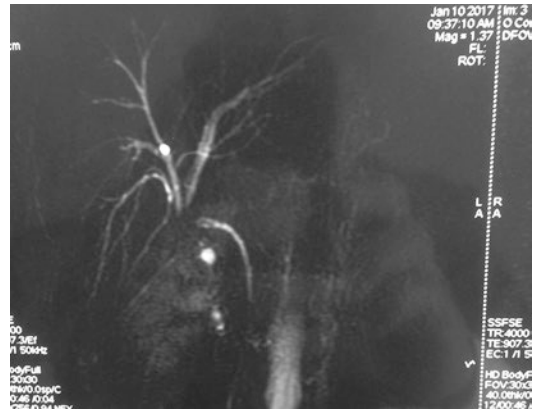

Fig. 16.6 Hepatico-jejunostomy for a high (Bismuth Type III or IV) benign biliary stricture is more likely to restricture

Luo [36] placed a 10 mm diameter transanastomotic balloon across the HJ and the anastomosis was dilated for 2–4 hours each time 4 times a day starting on day 7 for 3 months; after discharge the dilatation was done by the patients themselves. Restricture occurred in only 1 out of 54 patients who had anastomotic dilatation vs. in 7 out of 58 patients who did not have anastomotic dilatation.

16.2 Repair—Recurrence Interval

Recurrence of symptoms (jaundice, fever, and cholangitis) after repair of a BBS is early (within weeks or months) in case of a technical failure, e.g., faulty surgical technique, and a missed isolated duct (usually on the right side); it is late (after months or years) in case of a true failure (anastomotic stricture due to fibrosis). An early recurrence due to technical failure usually requires repeat surgical intervention in the form of a proper repeat HJ. The repair–recurrence interval in 55 patients referred to us after failure of a repair done elsewhere was much shorter (median 1.6 months), thus indicating technical failure, than in patients in whom the stricture was first repaired by us (median 35.0 months) indicating a true anastomotic failure [16].

16.3 Recurrent (Anastomotic) Strictures

An anastomotic stricture may be pre-empted by persistently high or progressively increasing alkaline phosphatase (ALP) or gamma glutamyl transpeptidase (GGTP) or delayed excretion of isotope on hepato-biliary scintigraphy. US may show intrahepatic biliary radical dilatation (IHBRD) and intrahepatic calculi and sludge proximal to the anastomotic stricture. The anastomotic stricture can be documented by cholangiography—preferably MRC; percutaneous transhepatic cholangiogram (PTC) should be done only if a therapeutic intervention, e.g., percutaneous transhepatic biliary drainage (PTBD) for cholangitis or balloon dilatation of the anastomotic stricture as definitive treatment is anticipated.

Treatment of choice for anastomotic stricture is percutaneous radiological intervention which involves a PTC, balloon dilatation (of the stricture), and stenting (across the stricture). Multiple sessions are usually required [33]. Complications include bleeding and bile leak. Anastomotic stricture of an end-to-end repair can be balloon dilated and stented endoscopically.

11 of 22 failures in 110 patients who underwent repair of a BBS could be salvaged by radiological or surgical intervention [10]. 19 out of 26 failures reported by Stewart [11] were managed successfully by dilatation and stenting. In a report from the Johns Hopkins Hospital, Baltimore, MD, USA, 13 out of 156 surgical repairs failed—12 of these were treated with percutaneous balloon dilatation; only one required surgical revision [12]. Misra [33] reported percutaneous management including cholangiography, biliary catheter placement, balloon dilatation, and stenting in 51 patients; success was more frequent when previous operative repair was done at the Johns Hopkins Hospital itself. Bektas [14] reported 74 repairs—2 patients (3%) died. 16 required surgical intervention for early complications and 16 required further operations (including re-HJ in 4, liver segementectomy in 1, liver transplant in 1, adhesiolysis, incisional hernia repair, and revision of Roux-en-Y jejunal loop in the remaining) for late complications during follow-up of 1.5–15 years; long-term success was 53%. In a recent report, 23 out of 42 strictured HJs were treated by percutaneous dilatation while 19 underwent re-HJ [7]. At the Academic Medical Center (AMC), Amsterdam, Netherlands, 33 out of 37 recurrent strictures were treated percutaneously—only 4 required repeat surgical repair [9].

Previous repair makes a subsequent repair difficult and challenging. A low repair, i.e., end-to-end repair or choledocho-duodenostomy, however, leaves an untouched proximal duct at the hilum for a subsequent repair. A previous HJ, on the other hand, produces fibrosis and scarring at the hilum rendering the reoperation technically difficult. Recurrent strictures after failure of a previous repair in the form of HJ are difficult to manage—they are higher and more frequently associated with vascular injuries; outcome of repair of a recurrent stricture is poorer than that of a primary stricture. The author's approach to an anastomotic (recurrent) stricture of HJ is based on where (i.e., by whom) the previous repair was done (viz. whether general surgeon or biliary surgeon) and the current anatomy of

the stricture (whether the common hepatic duct/left hepatic duct is still available). If the previous repair was done by a general surgeon (who probably did the anastomosis to the common hepatic duct only and did not extend the incision to the left hepatic duct) and if cholangiography shows intact left hepatic duct (Fig. 16.7), we tend to prefer reoperation for a repeat (proper) HJ. If, however, the previous repair was done by a biliary surgeon and if the left hepatic duct is not available for anastomosis (Fig. 16.8), we prefer percutaneous radiological intervention in the form of balloon dilatation (vide supra). Re-repair of an anastomotic stricture after previous HJ is associated with poorer results. In a very high (Bismuth Type IV) recurrent biliary stricture with associated vascular injury and atrophy/hypertrophy, we now have a low threshold for right hepatectomy (See Chap. 14).

Chaudhary [37] reported an experience with 41 patients who were referred to a tertiary care center for management of recurrent strictures following surgical repair of a BDI elsewhere. Cholangitis after BDI, no cholangiogram before repair and repair within 3 weeks of injury were factors associated with failure of initial repair. At the time of the index repair, only 12 (29%) patients had stricture at or above the biliary ductal confluence but as many as 37 (90%) patients had a hilar stricture at the time of referral. Over a mean follow-up of 4.2 years, 90% patients had satisfactory outcome after the repeat repair.

36 (12%) out of 300 BBS repaired by us between 1989 and 2003 [16] were recurrent strictures—only 6 (16%) of these 36 patients had undergone a proper Roux-en Y HJ at the time of the primary repair; the remaining 30 patients had undergone direct (end-to-end) repair or unconventional procedures, e.g., choledocho-duodenostomy, fistulo-duodenostomy, or a loop HJ. All 36 patients with a recurrent stricture underwent re-HJ (Fig. 16.9). 35 of these 36 patients were followed for a median of 37 months;

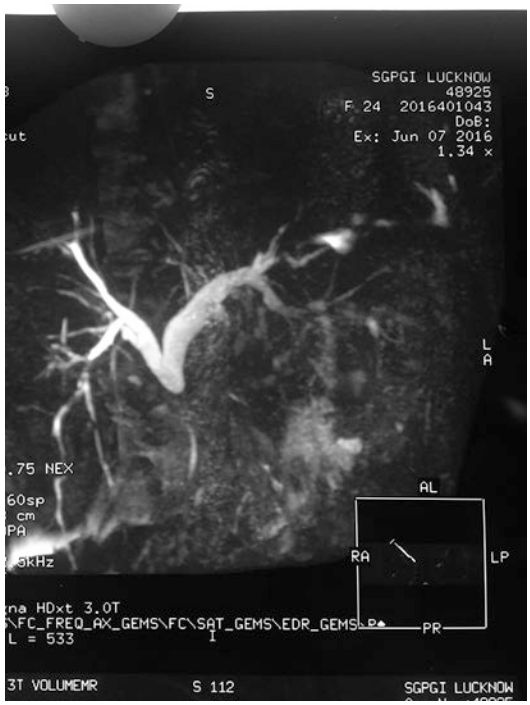

Fig. 16.7 Recurrent stricture following an inadequate hepatico-jejunostomy—anastomosis was done to the common hepatic duct; left hepatic duct is untouched. This is a suitable case for a repeat hepatico-jejunostomy

Fig. 16.8 Recurrent stricture after a proper hepatico-jejunostomy—anastomosis was done at the hilum. This is a suitable case for percutaneous radiological balloon dilatation

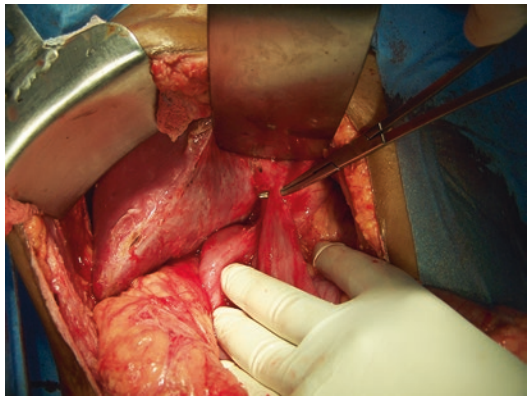

Fig. 16.9 Strictured hepatico-jejunostomy at reoperation (See Fig. 16.3 also)

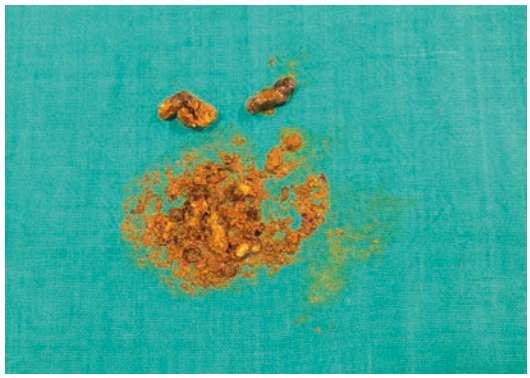

Fig. 16.10 Stones and sludge removed from the intrahepatic ducts proximal to recurrent (anastomotic) stricture (See Fig. 16.5 also)

2 of these 35 patients had poor (McDonald grade D) outcome—one of these 2 patients underwent a third repair which was successful. On the other hand, only 10 (4.7%) of 242 primary (non-recurrent) biliary strictures repaired by us had a poor outcome—4 of these could be salvaged by percutaneous balloon dilation and 4 by surgical intervention, 1 died.

Patients who have undergone surgical repair, i.e., HJ, for a BDI/ BBS need long-term, preferably life-long, follow-up. Recurrent stricture is not uncommon. Most recurrent strictures can be managed non-surgically with percutaneous radiological balloon dilatation.

Invited Commentary on Follow-Up after Repair of Bile Duct Injury

Graeme J Poston

Iatrogenic bile duct injury remains the most feared complication of cholecystectomy, whether laparoscopic or open, and in many countries is an increasing source of medico-legal litigation. Following apparently successful bile duct reconstruction, patients face loss of life expectancy [38, 25] and frequently never recover the quality of life they enjoyed prior to the index cholecystectomy [39–42]. The fundamental issue, as this chapter by Prof Kapoor demonstrates, is that subsequent complications, the most important being the consequences of stricture at the site of the bile duct repair (by whatever method is employed for biliary reconstruction) can occur many years, and even decades later [6, 43–44]. As such, these patients need to be offered long-term follow-up, if necessary for life. The problem is that the symptoms of early stricture can be very subtle, especially if the initial injury is Strasberg E4 necessitating a double repair to separate right and left hepatic duct systems, when if one side strictures and the other hepatico-jejunostomy anastomosis remain patent, then jaundice does not occur, and the presentation is not classical for cholangitis. Therefore, ideally such follow-up should be within a tertiary hepato-biliary center, which will also have access to all the necessary skills and disciplines (interventional radiology, biliary endoscopy, hepato-biliary surgery, including possible liver transplantation) required to manage this complication when it occurs.

If a patient remains asymptomatic and well, then they only need to be reviewed six monthly during the first year after bile duct repair surgery, and then annually thereafter. Follow-up of well patients requires no more than clinical review and routine blood liver function tests. It is this author's (GP) experience that alkaline phosphatase levels not infrequently never recover to within the normal range in these circumstances, and so persistent modestly elevated alkaline phosphatase

levels, in the absence of any other abnormality (clinical or biochemical) per se, do not require further investigation. However, recurrent bouts of low grade sepsis, in particular with deteriorating blood liver function tests, jaundice, and/or itching require immediate investigation. Biliary ultrasound scanning may detect intrahepatic biliary radical dilatation (IHBRD), but this may be subtle, possibly unilateral or may be only sectoral or segmental (and therefore difficult to interpret by an inexperienced sonographer). Magnetic resonance cholangiography (MRC) with liver specific contrast is the ideal early investigation in these circumstances, but is best interpreted by a radiologist who specializes in this field. It is this author's (GP) experience [43] that percutaneous dilatation of such strictures (with or without self-expanding metal stent SEMS) is effective in the majority of cases, and revision hepatico-jejunostomy only infrequently required. If percutaneous transhepatic cholangiography (PTC) is attempted then it is crucial to remember that if an obstructed bile duct system is entered but the anastomotic stricture cannot be crossed then do not simply withdraw the cholangiogram needle as bile will now leak from the liver capsule and cause biliary peritonitis. It is imperative, therefore, that in the investigation and management of this late complication that there is extremely close collaboration at all stages between the interventional radiologist and the hepato-biliary surgeon.

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17 Healthcare Issues Related to Bile Duct Injury

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Optimum management of bile duct injury (BDI) and benign biliary stricture (BBS) requires a multi-disciplinary team including a therapeutic endoscopist, an interventional radiologist and, foremost, a biliary surgeon. Management of a BDI and repair of a BBS is one of the most difficult challenges faced by a surgeon, even a biliary surgeon. The most important factor which determines the outcome of repair of a BDI/ BBS is the expertise and experience of the repairing surgeon.

17.1 Injuring Surgeon

Most BDIs will occur in the hands of non-specialist general or laparoscopic surgeons who, in most cases, are not biliary surgeons (*vide infra*) and who do not have the expertise/experience for reconstructive biliary surgery. The injuring surgeon has the natural temptation to '*fix the leak then and there*' and to perform an immediate (intraoperative) or early repair (in order to hide or cover up the injury); as many as 50% of the injuring surgeons in UK [1] and 60–75% in USA and Canada [2, 3] attempted to repair the BDI caused by them. An Italian survey found that out of 100 injuries recognized during operation as many as 93% were repaired by the injuring surgeon who performed suture or reconstruction of the common bile duct with T-tube in 65 patients and (proper) hepaticojejunostomy in only 27 patients [4]. In the USA, the first operative repair was performed by the primary surgeon in 163 out of 300 cases [5].

Immediate repair in the hands of a non-biliary surgeon is most likely to fail. Chances of success following repair by the injuring surgeon (vs. biliary surgeon) were low—21% vs. 95% for a biliary surgeon [5]. Desperate attempts at identification of the site of the BDI and unsuccessful attempts at its repair may make the injury worse (higher ductal injury, additional vascular injury). An attempted repair necessarily means dissection in the hilum and loss of length of the proximal bile duct; the stricture which forms following an unsuccessful attempt is higher and more complex than the initial injury. A previous attempt at repair is also more likely to be associated with a vascular injury; it is also going to make the second repair more difficult. An (unsuccessful) attempt at repair of BDI/ BBS decreases the chances of success of the repeat repair and is one of the most important risk factors for poor long-term outcome of a subsequent repair, even if done by an expert and experienced specialist biliary surgeon. Moreover, repair by the injuring surgeon is a risk factor for litigation

Also see Invited Commentary on Healthcare Issues Related to Bile Duct Injury by Philip R de Reuver (pp 209–211)

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also. Repair by the primary surgeon is more expensive ($120,000/QALY) than that by a biliary surgeon ($48,000/QALY for early repair and $74,000/QALY for delayed repair) [6]. The average length of illness was longer (222 days) in patients first treated (repaired) by the primary (injuring) surgeon than in those in whom the first repair was performed by a tertiary care biliary surgeon (78 days) [7]. If immediate repair is not to be done (as is recommended for most injuries), there is no need to try to find out the site or the type of injury—just *lavage and drain*. The BDI MUST be repaired by a biliary surgeon only as the '*first attempt at repair is the best chance for repair*'. The first attempt to repair a BDI or BBS should, therefore, be a serious attempt made by an expert and experienced biliary surgeon (and not by the injuring non-specialist general surgeon), at an appropriate time (i.e. delayed repair). For this, either the patient should be referred (after initial management i.e. resuscitation and stabilization) to a biliary center or if the injury is detected during the cholecystectomy itself, a biliary surgeon should be called to the operation room to repair the injury. Silva [8] has described an outreach service in the UK where a biliary surgeon travels to the site where the BDI has occurred to repair the injury on the operation table itself—22 such repairs were done.

The injuring surgeon or center with no facilities and expertise for therapeutic endoscopy, interventional radiology and biliary reconstructive surgery should refer a patent with BDI/ BBS to a biliary center which has these facilities and expertise. Repeated unsuccessful interventions, whether surgical or non-surgical, make the BDI or BBS worse and decrease the chances of successful results of future interventions. The mean time from injury to referral (to the Academic Medical Center AMC, Amsterdam Netherlands) in 151 patients was 25 days. Patients who underwent any (non-surgical i.e. endoscopic or percutaneous, or surgical) therapeutic interventions elsewhere had a much higher risk of having major postoperative complications viz. anastomotic leak, bleed, anastomotic stricture and need for relaparotomy than those who were referred primarily (21% vs. 6%). Secondary referral was also associated with higher reoperation rate (8% vs. 0.7%). Restricture occurred in more (13.8% vs. 3.1%) patients with secondary referral than those who were primarily referred [9].

> To injure is human, to refer divine.

17.2 Biliary Center and Surgeon

Biliary center is a center where facilities and expertise for diagnostic (i.e. magnetic resonance cholangiography MRC, percutaneous transhepatic cholangiography PTC) and therapeutic interventional (percutaneous catheter drainage PCD, percutaneous transhepatic biliary drainage PTBD, percutaneous transhepatic biliary catheterization PTBC, etc.) radiology, and diagnostic (endoscopic retrograde cholangiography ERC) and therapeutic (endoscopic papillotomy EPT, endoscopic stenting, endoscopic naso-biliary drainage ENBD, etc.) endoscopy and reconstructive biliary surgery are available (Fig 17.1).

Biliary surgeon is a surgeon who has the expertise and reasonable amount of experience to perform reconstructive biliary surgery i.e. hepatico-jejunostomy for BBS, after excision of choledochal cyst and cholangiocarcinoma and as a part of pancreato-duodenectomy.

> All bile duct injuries should be managed at a biliary center. All benign biliary strictures should be repaired by a biliary surgeon.

17.3 Volume

Like other difficult and complex surgical procedures e.g. coronary artery bypass, liver resection, pancreato-duodenectomy, management of BDI/ BBS should include a clinical pathway (sequence of procedures for investigations and management to reduce variations in practice) in order to improve the quality of care, achieve better outcomes, optimize resource utilization and reduce costs [10]. Results of repair of BBS are better from high-volume centers. Patients with BDI/ BBS should, therefore, be referred to a biliary center for management. There is a need to regionalize the care and centralize the referral and management of patients with BDI/ BBS to preidentified biliary centers. In Finland, diagnosis

Fig 17.1 Sanjay Gandhi Post-Graduate Institute of Medical Sciences (SGPGIMS) at Lucknow in India is a 1000+ bed tertiary level university teaching hospital with facilities and expertise for diagnostic and therapeutic interventional radiology, diagnostic and therapeutic endoscopy and reconstructive biliary surgery—a biliary center. All BDIs should be managed and all BBS should preferably be repaired at a biliary center

and treatment of all suspected or confirmed BDIs is regionalized in central hospitals [11]. In Denmark, all major BDIs which require surgical intervention i.e. hepatico-jejunostomy are referred to HPB centers; only 5 out of 139 injuries reported between 1995 and 2010 were repaired by the primary non-HPB surgeon [12].

Bile duct injuries and benign biliary strictures are difficult problems which can cause major morbidity and even mortality; in order to obtain the best results, they should preferably be managed by biliary surgeons at biliary centers. The injuring general/laparoscopic surgeon should NOT attempt to repair the BDI; he/she should place drains in the subhepatic fossa and refer the patient to a biliary center after resuscitation and stabilization.

Invited Commentary on Healthcare Related Issues to Bile Duct Injury

P. R. de Reuver

Bile duct injury (BDI) is a feared surgical complication of laparoscopic cholecystectomy with an estimated incidence of 0.5%. BDI is associated with increased morbidity, mortality, high rates of litigation claims, and poor long-term quality of life. In this chapter, Professor Kapoor summarizes the persistent difficulties of primary repair, adequate referral and definitive multidisciplinary treatment in these complex cases. Despite the good functional outcome in BDI patients reported by several tertiary centres, the patient-reported outcome remains unsatisfactory. Healthcare related issues to bile duct injury in terms of quality of life, medical litigation, costs and work related limitations are a significant burden to the health care system and society.

Quality of Life

Quality of life assessment in BDI patients was initiated by our group as patients who had been treated for BDI reported many undefined abdominal complaints, whereas objective symptoms of recurrent jaundice or cholangitis could be demonstrated in only a few patients [13]. Patients told us they were still preoccupied with the unexpected course of events after the removal of the gall bladder. They remained disappointed by the prolonged hospital stay, the occasionally delayed diagnosis, the additional invasive interventions or even relaparotomy in the worst cases.

Data from the Academic Medical Center (AMC), Amsterdam group assessed by a survey in a large number of BDI patients showed that

after a mean follow-up of 5.5 years generic quality of life (SF36) in injured patients was significantly lower in 3 of the 8 domains compared to patients who underwent cholecystectomy without an injury [14]. In 7 of the 8 QoL domains, injured patients scored significantly worse than the healthy population norms ($p < 0.05$). No improvement was found in a longitudinal study after 5.5 and 11 years of follow-up. Clinical characteristics such as the type of injury and type of treatment did not affect outcome. In patients who filed a malpractice claim after BDI QoL was worse. However, these patients reported better QoL if the claim was resolved in their favour compared to patients whose claim was rejected.

Claims

Malpractice litigation among BDI patients is common with a variation in incidence from 19% in the Dutch series up to >80% in studies from the USA [15]. Clinical factors associated with initiation of a litigation claim are young age, the severity of the injury and definitive surgical treatment. Socio-economic factors as employment during the initial cholecystectomy and postoperative use of social securities were also associated with litigation. Data analysis from the largest Dutch hospital insurer for medical liability showed that a complete transection of the common bile duct is an independent predictive factor for starting a claim procedure (OR 7.53, CI 1.85–30.63). In this Dutch series the median compensation was € 9826 (range € 1588 – € 55,301), which is in sharp contrast with the average payment in the United States. In 2006, McLean [16] reported an average payment of US$ 508,341 in 104 patients who underwent a complicated laparoscopic cholecystectomy, but compensations up to US$ 800,000 are reported [17]. Of interest is a Dutch survey which demonstrated the frail agreement among surgical experts in malpractice litigation. In one of the ten BDI cases, unanimous agreement among the experts was obtained. In the majority of cases, half of the reviewers judged that negligence had occurred while the others judged the opposite, or could not determine whether negligence of care had occurred based on the presented medical histories [18]. Therefore it was concluded that defendants, plaintiffs, experts, and lawyers should be aware of the drawbacks of expert witness testimonies.

Costs

The large socio-economic impact of BDI is illustrated by the significant increase in hospital costs but also costs associated with the absence from work, and the use of disability benefits. An analysis from 24 BDI patients in Sweden estimated that the overall costs for the society for the management of both mild and severe bile duct injuries would be between € 473,690 and € 608,789 annually per million inhabitants. These estimations were based on calculations on the total costs based on information on cholecystectomy, incidence, complications and costs in Scandinavia [19]. Previous studies for the United States estimated BDI related overall hospital costs ranging from US$ 100,000 to US$ 30,000, strongly depending on inclusion of costs associated with loss of work, caretaker costs, loss of eventual productivity, pain and suffering, and court claims [20].

Conclusion

BDI results in clinical and socio-economic long term consequences in terms of quality of life, claims and costs. Immediate honest postoperative communication to patients and relatives about diagnosis, treatment and prognosis are of great importance. The unexpected course after the cholecystectomy, the prolonged hospital stay, and the occasionally delayed diagnosis will probably remain, and form a patient's physical and mental burden. Although an association between malpractice litigation and quality of life in BDI patients was shown in previous studies, the causality dilemma remains unanswered: what came first? Is it the poor quality of life that causes the patient to file a claim, or does the litigation process have a detrimental influence on a patient's mental and physical wellbeing? Surgeons should

be aware of the possibility of being sued after the occurrence of BDI. Honest and open communication with the patient and adequate documentation of clinical findings and therapeutical considerations might prevent a long and distressing litigation process [14, 21].

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18 Socio-Economic and Medico-Legal Issues Related to Bile Duct Injury

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The advantages of laparoscopic cholecystectomy for an individual patient, viz. less postoperative pain, early recovery from the operation, early discharge from the hospital, early return to work, and better cosmesis are offset, to a great extent, by the increased risk of bile duct injury (BDI) in the entire cohort of patients who undergo laparoscopic cholecystectomy as compared to those who used to undergo open cholecystectomy earlier. A BDI has been described as a "devastating" complication of cholecystectomy—not only for the patient who may become a "biliary cripple" but for the surgeon also who may face and then lose a medico-legal suit.

Majority of patients who sustain a BDI during cholecystectomy and subsequently develop a benign biliary stricture (BBS) are young, otherwise healthy, and in the productive years of their life, as gall stone disease for which the cholecystectomy is done is a disease of the young; majority of them are women—a wife, a mother, a homemaker, a teacher, or even a professional.

Also see Invited Commentary on Socio-economic and Medico-legal Issues Related to Bile Duct Injury by JEJ Krige, Eduard Jonas and Jessica Lindemann (pp 220–222).

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18.1 Economic Aspects

A BDI has significant financial implications for the patient in terms of the costs of treatment and loss of work and wages; it also has a financial impact for the surgeon in the form of an increase in malpractice premium/indemnity, costs of litigation, and payment of compensation.

For an uncomplicated laparoscopic cholecystectomy, a patient will usually need one preoperative outpatient visit, one hospitalization for 1–2 days, and one follow-up visit after the operation. On the other hand, a patient who sustains a BDI during the cholecystectomy will require multiple outpatient visits, at least one extra hospitalization for the repair of the BDI and multiple follow-up visits to the hospital. A BDI results in a significant increase in healthcare expenses as compared to an uncomplicated cholecystectomy—it has been described as a "financial disaster" as the costs of management of a BDI are 5–26 times the costs of a cholecystectomy [1].

The mean cost of management of common bile duct transection/excision increased to US$ 9061 from the mean cost of US$ 2681 for an uncomplicated cholecystectomy [2]. In 49 patients with BDI managed at the Johns Hopkins Hospital, Baltimore, MD, USA, healthcare costs included 10 outpatient days, 32 inpatient days, biliary intubation for >12 months, and mean total hospital charges of US$ 51,411 per patient [1]. These costs are even higher if the

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recognition of the BDI is delayed. The charges for BDI which occurred at the Johns Hopkins Hospital itself, was recognized during the cholecystectomy and repaired immediately were the least (US$ 22,565); while those for a patient who sustained a BDI elsewhere, had a major bile leak for which an operation was required at the injuring hospital and then a delayed repair was done at the Johns Hopkins Hospital were the highest (US$ 130,345) [1]. In Belgium, the cost of an uneventful laparoscopic cholecystectomy was BF 1721; it increased fourfold to BF 7250 when a BDI occurred and was repaired immediately during cholecystectomy itself and fivefold to BF 9258 when delayed repair was performed [3]. Cannon et al. [4] suggested a novel classification system for BDI to address the financial impact—grade I (minor injuries), II (major injuries), and III (vasculo-biliary injuries). The costs of management were US$ 12,457 in grade I (n = 14), US$ 46,481 in grade II (n = 74), and US$ 69,368 in grade III (n = 20) BDI. It must, however, be kept in mind that if the immediate repair is performed by a non-biliary surgeon, it is more likely to fail. Costs of a failed repair will be more than that of a successful repair because of need for further investigations and interventions. Costs were highest (US$ 120,000/QALY) when repair was done by the primary surgeon; costs were lower (US$ 48,000/QALY for early repair and US$ 74,000/QALY for delayed repair) when repair was done by a biliary surgeon [5]. Hospital (admission to discharge) costs of repair of a major BDI in 44 patients in a South African hospital were ZAR 215,711 (range 68,764-980,830); operation theater expenses (22%), intensive care expenses (21%), radiological investigations (17%), and specialist fees (12%) accounted for major expenses [6]. Mean total costs in 49 patients with BDI treated in Turkey were TRY 8924 (US$ 4022) during a mean hospital stay of 36 (range 4–111) days [7]. The cumulative costs at 1 year of management of BDI were US$ 60,539 for operative repair and US$ 118,245 for endoscopic management [8].

We interviewed 47 patients with BDI who were managed by us. These patients had to make a median of 8 (range 1–50) outpatient visits to a hospital and required a median of 2 (range 1–4) hospitalizations for a median of 10 (range 15–51) days elsewhere before being referred to us. They had to make a median of 1 (range 1–8) outpatient visits to our hospital, required a median of 1 (range 1–3) hospitalizations for a median of 12 (range 7–25) days with us before admission for the operation for definitive repair of the BDI. Even after the repair of the BDI, these patients had to make a median of 5 (range 1–11) outpatient visits and required a median of 1 (range 1–3) hospitalizations for a median of 10 (range 7–30) days. The median total cost of the management of BDI was INR 93,046 (range 22,204-562,790), equivalent to US$ 2045 (range 488–12,369)—this was more than 8 times the median monthly income of these patients. These costs were about 15–20 times the cost of an uncomplicated cholecystectomy at another university hospital in India. None of these 47 patients was insured for medical treatment; only 7 out of 47 patients received reimbursement of their expenses from their employer. Ours is a state government funded university teaching hospital where treatment is highly subsidized, i.e., the patients pay for consumables and drugs only, services are not charged for. Costs of treatment at a private corporate hospital where services are also charged will be much higher. Moreover, we could not calculate some indirect costs, viz. loss of wages of the patient, spouse, or attendants. In addition to the definable, measurable, and calculable direct monetary costs, there are many non-measurable non-monetary costs, e.g., loss of school for children, missed examinations, non-celebration of festivals, etc. which increase the socio-economic burden on the family. We proposed an umbrella insurance against BDI for every patient undergoing cholecystectomy. Premium is to be paid by the patient as well as by the surgeon. Patient premium is decided by the predicted difficulty of cholecystectomy; surgeon premium is decided by the qualifications, training, accreditation, certification, experience, volume, and results of cholecystectomy of the surgeon [9].

Many hospitals offer packages for cholecystectomy; the costs of management of BDI in one patient have to be distributed to all patients

undergoing cholecystectomy. The extra costs of management of a BDI have to be kept in mind by the insurance companies when calculating the premium for a policy covering cholecystectomy. These costs can also guide the amount of compensation paid to a patient who wins a medico-legal suit.

18.2 Quality of Life

According to the WHO, health is not only absence of disease but also physical, mental, and social well-being. Health-related quality of life (QoL) is the extent to which a disease or a procedure impacts the physical, mental (psychological), and social aspects of a patient's life. It is, therefore, not only important but imperative also that in addition to clinical outcomes, QoL scores should also be measured.

Laparoscopic cholecystectomy is usually offered to the patient as a "small" operation—admission in the evening before or even on the morning of operation and discharge the following morning or even on the evening of operation. The patients are assured of quick recovery and an early return to work. The patient is not mentally (and even financially) prepared for a complication such as BDI requiring prolonged hospitalization and multiple invasive interventions. The unexpected BDI, and its complicated and prolonged treatment, comes as a rude shock to the patient.

QoL was studied in 82 patients, 70 months after BDI—QoL was impaired in all 8 subscales (physical functioning, role functioning, bodily pain, general health, vitality, social functioning, emotional, and mental health) of SF-36 as compared to those who had undergone uncomplicated laparoscopic cholecystectomy 2 years ago [10]. Fifty patients who were treated for laparoscopic cholecystectomy related BDI between 1991 and 2003 and who were 62 months after injury (48 of these had no biliary stricture and normal LFT at the time of assessment) were compared with 74 patients who underwent uncomplicated laparoscopic cholecystectomy for QoL using Karnofsky Performance Scale (KPS), SF-36, and physiological adjustment to illness scale (PAIS). The mean KPS of BDI patients was significantly lower (77 vs. 93) than that of controls. BDI patients had significantly lower scores in all 8 subscales of SF-36. The SF-36 component summary scales scores, viz. physical components scale (PCS) and mental components scale (MCS), were also lower in patients with BDI. BDI patients had higher PAIS global scores (45 vs. 33) than those who underwent uncomplicated laparoscopic cholecystectomy, indicating poorer psycho-social adjustment. Eleven BDI patients who filed a legal suit had a poorer SF-36 component score than those who did not. Patients with BDI could return to work but almost 3 months later than those who underwent uncomplicated laparoscopic cholecystectomy. BDI has been called a negative life altering event [11]. The Academic Medical Center (AMC), Amsterdam, [12] also found that patients with BDI had poorer QoL than those who did not have a BDI; the impaired QoL did not improve with time. Ejaz et al. [13] surveyed 62 patients for health-related QoL at a median follow-up of 169 (range 125–222) months; patients reported depressed mood (49%) or low energy level (40%) at the time of BDI but these symptoms improved (18% and 18%, respectively) after repair. Limitations in physical activity and general health remained unchanged before and after surgical repair of the BDI—27 (44%) of 62 patients reported having substantial financial hardship and 16 (27%) believed that their job was in jeopardy at some point during the course of their treatment [13]. Rystedt and Montgomery [14] compared QoL of 107 patients who sustained a BDI with 205 controls who underwent laparoscopic cholecystectomy without BDI. Most (n = 98, 92%) of the BDIs were recognized intraoperatively on peroperative cholangiography and majority (n = 59, 60%) were minor (<5 mm) injuries and most (88/98) were repaired immediately. QoL (physical composite score, PCS and mental composite score, MCS) of BDI patients was comparable to that of controls. Patients who underwent immediate repair had better physical component score than those subjected to referral/delayed repair. Booij et al. [15] compared 800 patients who sus-

tained a BDI during cholecystectomy with 175 patients who underwent uneventful cholecystectomy. Patients with BDI had significantly worse physical QoL, worse disease-specific QoL, higher loss of productivity of work, and significant hindrance even in unpaid work; more patients with BDI (35% vs. 20%) were receiving disability benefits. QoL scores were better in patients whose claims were accepted than in those whose claims were rejected. Hariharan et al. [16] reported that BDI not only affected QoL (physical and mental) but also impaired long-term survival. In a recent study from Mexico, 46 patients who had a BDI which was repaired with bilio-enteric diversion had significant reduction in 4 (general health, physical functioning, physical role, and social functioning) of 8 evaluated parameters of SF-12 questionnaire as compared to 51 patients who had an uncomplicated cholecystectomy [17].

A recent meta-analysis of 6 publications including 831 patients concluded that BDI has a long-term detrimental effect on mental health, but not physical health, related QoL as compared to an uneventful cholecystectomy [18].

In addition to the patient, a BDI can seriously and adversely affect the QoL of the surgeon also. The surgeon who causes a BDI during cholecystectomy suffers from a mix of remorse, guilt, low self-esteem, depression, and fear of a possible medico-legal suit. In addition to the financial burden of increased liability premiums and payment of compensation in case of an accepted claim, the surgeon faces immense psychological stress for a long time as the process of settlement of the claim is usually prolonged, ranging from 3 to 6 years [19]. The median duration of closure of 88 claims was 2 years (range 5 months—6.5 years) in the Netherlands [20].

18.3 Litigation

In surgical texts, we describe BDI as a complication (mishap by chance or adverse event related to the procedure) of cholecystectomy but the lay public (patients/relatives) may consider it to be an error (mistake) or even negligence on our part and take course to legal action. Every surgeon, who performs cholecystectomy, must remain prepared for such an eventuality. Litigation is not uncommon after a complicated cholecystectomy; a BDI is quite likely to result in a medico-legal suit. Litigation used to be uncommon after open cholecystectomy—only 68 cases of litigation after open cholecystectomy were found over 20 years period in USA [19] but has increased after the introduction of laparoscopic cholecystectomy in the late 1980s. 300 claims following cholecystectomy were filed in the UK between 1995 and 2008—about 4 per 10,000 cholecystectomies [21]. In another report from the UK, this rate was 6 per 10,000 cholecystectomies—418 claims were filed between 1995 and 2009 [22]. The litigation rate further increased to 7 per 10,000 cholecystectomies between 2000 and 2005 when 208 claims were filed [23]. About 0.08% of laparoscopic cholecystectomies resulted in a BDI related litigation claim in the Netherlands [20]. In one report from the Academic Medical Center (AMC), Amsterdam, 11 out of 50 patients with bile duct injury (BDI) filed a law suit against the surgeon [24]. In another report from the UK, 22 (33%) out of 67 patients with BDI had resorted to litigation [25]. In the USA, as many as 43 (71%) of 62 patients who responded to a survey had sought litigation for their BDI [13]. Among the members of the Association of Upper GI Surgeons (AUGIS) of Great Britain and Ireland, 22% of 117 respondents to a questionnaire reported a medico-legal experience following laparoscopic cholecystectomy [26].

McLean [27] goes on to say that "if a surgeon's sole goal is to minimize the risk of litigation after cholecystectomy, open cholecystectomy should be preferred over laparoscopic cholecystectomy". (*Admittedly, such a consideration may be incompatible with market realities and patients' desires today*).

BDI is one of the common causes of a medico-legal suit filed by a patient against the surgeon; it is the commonest cause of litigation after gastrointestinal surgery. Of the 44 legal cases in USA, 27 (61%) were triggered by a BDI; other causes included bowel injury in 7 (16%), vascular injury

in 4 (9%), and other complications in 6 (14%) cases [28]; the injury was missed during the operation in 83% cases. In another report from the USA, 81 (78%) out of 104 litigations following laparoscopic cholecystectomy were related to BDI, the remaining were related to vascular injury (7%) a, bowel injury (2%), and other injuries (13%) [27]. In one report from UK, 27 (65%) out of 44 litigations after cholecystectomy were because of a BDI [22]. In the National Health Service (NHS), UK, BDI was the commonest 41% (others being bile leak 12%, bowel injury 9%, bleeding 9%, and death 9%) cause of 300 malpractice litigations filed after cholecystectomy between 1995 and 2008 [21].

Young (<52 years) patients and those with an associated vascular injury are more likely to resort to litigation after BDI during cholecystectomy [25]. More severe BDIs are more likely to result in litigation; one-third of patients with major (transectional) BDI resorted to litigation [29]. Another reason for a medico-legal suit is delay in diagnosis of the post-cholecystectomy complications. In a report of malpractice litigation after 46 BDIs, the average delay in diagnosis was 10 days [30]. In a report of 104 BDIs involving litigation, 86% of the injuries were missed during the operation [27]. Late recognition of BDI and late transfer of the patient to a higher center were found in 20 out of 23 legal cases in Turkey [31]. A medico-legal suit is more likely to be lodged against the surgeon if repair of the BDI is attempted by the injuring surgeon or in the injuring hospital than if the patient is referred to a biliary surgeon or a biliary center. Immediate (intraoperative) repair by a non-specialist surgeon is an independent predictor for litigation [25].

18.4 Negligence

Strasberg [32] described negligence as carelessness but standards and quality of care are very subjective and vary from one level of healthcare to another.

A BDI is a complication which can (and will continue to) occur in the hands of almost every surgeon who performs cholecystectomy; the following may, however, be considered as negligence:

- When adequate steps do not seem to have been taken to prevent a BDI during cholecystectomy.
- When postoperative symptoms and signs of BDI are not recognized in time.
- When there is delay on the part of the injuring surgeon in suspecting BDI.
- When investigations, i.e., diagnostic imaging to detect a BDI, are not asked for in time.
- When there is delay in the diagnosis of a BDI.
- When the management of a BDI is inappropriate (which includes not referring or delayed referral of the patient to an appropriate biliary center in case expertise and facilities for management are not available at the injuring hospital).
- When BDI is managed in the injuring center which is not a biliary center.
- When relaparotomy and repair are done by the injuring surgeon who is not a biliary surgeon.

The case is more likely to be decided against the surgeon if repair of BDI is attempted by the injuring surgeon or in the injuring hospital than if the patient is referred to a biliary surgeon or a biliary center [21].

- When medical file documentation and communication with the patient/relatives are poor.

Complete transection of the CBD was the only independent predictor for settling a claim procedure [33].

> To injure is a complication,
> to miss is a mistake,
> to mismanage is negligence.

Example: *Open cholecystectomy—drain showed 200–300 ml of bile—drain got accidentally removed—patient not well, abdomen not settled—yet no investigations done and patient discharged home—this is mismanagement and will be judged as negligence!*

18.5 Compensation

Medical (surgical) experts are often called to assist the jury which is composed of lay (non-medical) persons. Strasberg [32] observed that experts could be grouped into three categories—those who considered misidentification (of the common bile duct as the cystic duct) always negligent, sometimes negligent and never negligent. There is, however, very little agreement among the experts—when experts were asked to opine on 10 cases of litigation for BDI during cholecystectomy all of them agreed in only one case; only half of them agreed in seven cases [34]. More and more experts now opine that BDI is an inherent risk of laparoscopic cholecystectomy—the percentage of accepted/settled claims in the Netherlands decreased from 50% in 1994–1998 to 18% in 2003–2005 [20]. Fellmer et al. [35] analyzed 13 legal verdicts (7 upheld and 6 rejected) related to BDI in Germany (1996–2009) and observed that the BDI was considered as evidence of below the expected standards of care during 1996–2002 but as something inherent to the treatment recently, i.e., 2004–2009.

The chances that the case will be decided in favor of the patient (i.e., against the surgeon) are high in case of a BDI—25 (52%) out of 44 cases in a report from Germany [36], 40 (86%) out of 46 case in a report from USA [30] and 66 out of 83 cases [23], 198 (65%) out of 303 cases [21] and 19 out of 22 cases [25] in reports from the UK. The acceptance rates are much higher in the USA—25 (57%) of 44 claims [28] were decided in the plaintiff's favor. 29 (71%) of 43 patients in the USA who had sought litigation reported that they had won the law suit [13]. The rate was, however, much lower in Netherlands. Between 1994 and 2006, 133 claims were filed for laparoscopic cholecystectomy related BDI in the Netherlands; 88 cases were closed—61 rejected, only 16 accepted in favor of the patient, and 11 settled out of the court [20].

Successful cases result in high amounts of compensation paid to the patient. The mean compensation amount in 4 cases in USA was US$ 188,772; mean payment in 21 cases settled out of court was US$ 469,711 [28]. The amount of compensation paid in Netherlands is much lower; median compensation in the 16 accepted cases in which liability was acknowledged was € 9826 (range € 1588-55,301) [20]. BDI related death is very likely to result in settlement of claim—in 4 out of 11 settled and 1 out of 16 accepted claims the patient had died, cf. only 1 out of 61 rejected claims [20]. Compensation in the UK ranged from £ 40,000 to 100,000 [21, 23, 25]. It was £102,829 in another report from the UK [22]. A total of £4.3 million were paid as compensation in BDI related claims in the UK [23]. Compensation in the USA ranged from US$ 214,000 [30] through US$ 438,000 [28] to as high as US$ 508,341 [27].

Delay in diagnostic imaging, delay in diagnosis, relaparotomy, and repair in the injuring center, involvement of a plaintiff's attorney, and death of the patient were factors significantly related to the amount of financial compensation. Higher amounts were awarded in cases of delay in diagnosis and reoperation in the injuring hospital [20]. Delayed recognition of the BDI correlated with more costly litigation [23]. Mean compensation for BDI was £53,900 but it increased to £89,930 if the patient had died [23]. Claim process is shorter and financial compensation is smaller in the Dutch arbitration system than in the US liability system.

Though the premium may appear to be an unnecessary and wasteful expenditure to begin with, every surgeon performing cholecystectomy must take an indemnity insurance for an adequate amount to pay the compensation if and when a case is filed against him (which is very likely) and is lost.

18.6 Defense

A BDI often results in a litigation (medico-legal suit) by the patient against the surgeon. Following steps can strengthen the surgeon's defense against the litigation:

1. proper (structured) training in the techniques of laparoscopic surgery,

2. proper detailed written informed consent from all patients undergoing laparoscopic cholecystectomy. This should include a mention of the chances of conversion in every case but higher chances of conversion in case of an anticipated difficult cholecystectomy and all possible complications, including a small but significant risk of BDI, of cholecystectomy. In the USA, 45 (73%) out of 62 patients who responded to a survey did not believe that they were adequately informed of a possibility of a BDI before cholecystectomy [13]. Use of procedure-specific consent forms (PSCFs) containing nationally standardized list of common and major risks/complications is recommended [37],
3. low threshold for early and timely *conversion by choice* from laparoscopic to open operation to prevent a BDI (not *conversion per force* after an injury has occurred to repair the BDI) in case a difficulty is encountered [38],
4. careful postoperative monitoring of the patient and high index of suspicion for a BDI,
5. low threshold for prompt investigation for a BDI in case the postoperative course of the patient is not uneventful,
6. timely and appropriate management of the BDI (if facilities and expertise for proper management of the BDI, i.e., interventional radiology and therapeutic endoscopy, are available at the injuring hospital) or timely referral of the patient to a biliary center (if facilities and expertise for proper management of the BDI, i.e., interventional radiology and therapeutic endoscopy, are not available at the injuring hospital),
7. detailed referral note including complete information about the operative findings and the operative procedure while referring the patient to a biliary center (Fig. 18.1),
8. proper documentation of the case records including the operative findings (especially difficult pathology) and the operative procedure (operative pictures and preferably video recording of the operation). If the cause of BDI was aberrant anatomy or difficult pathology, these documents may offer some defense to the surgeon who faces a medico-legal suit.

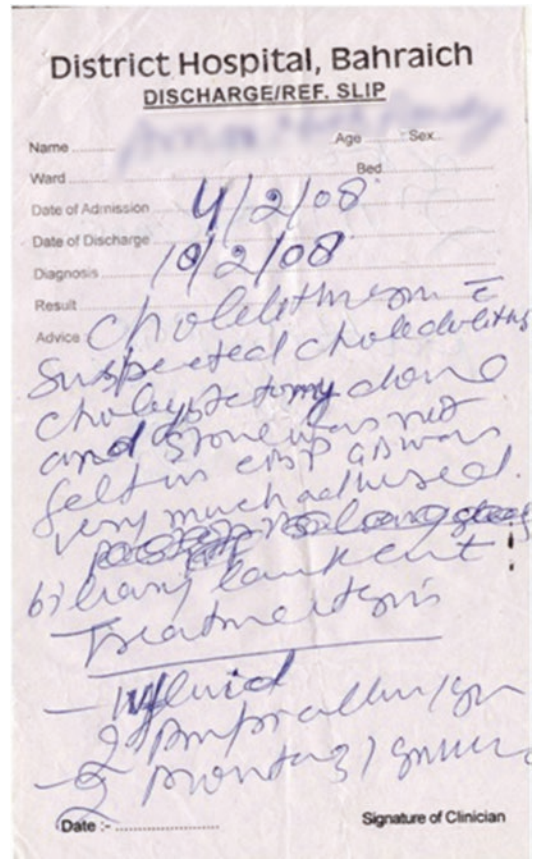

Fig. 18.1 Example of an incomplete and inadequate referral note. A detailed referral note including complete information about the operative findings and the operative procedure should be sent while referring the patient with bile duct injury to a biliary center

Most litigations are not because of the medical error itself but because of poor or no communication between the surgeon and the patient. Frank, truthful, and honest communication with the patient and the relatives about the fact that a BDI has happened goes a long way in preventing their filing a medico-legal suit against the surgeon. Documentation of this communication in the patient's case records offers defense to the surgeon even if a case is filed against him/her [39]. Communicating a BDI to the patient/relatives, however, is not an easy task. Barrios et al. [40] conducted a very interesting study; general surgery residents were asked to convey two hypothetical situations to a mock family member—a BDI during cholecystectomy or an

incidental finding of cancer in the gall bladder after cholecystectomy. They found the disclosure of the BDI more difficult than the disclosure of the incidental finding of cancer. Poor communication of the BDI to the patient/relatives was a cause of litigation in a German report; providing insufficient information to the patient/relatives was considered malpractice [36]. Even in the UK, a large majority of the patients reported that they were not adequately informed, either before (47/67, 70%) or after (50/67, 75%) surgery when a BDI occurred [25].

Document;
communicate;
communicate what you document;
document what you communicate;
preserve all documents and communication.

A BDI may not be considered to be negligence on the part of the surgeon when adequate steps are taken to prevent it, if postoperative symptoms and signs of bile leak are recognized in time and investigations are ordered without delay and if the injury is appropriately managed [41].

Invited Commentary on Socio-Economic and Medico-Legal Issues Related to Bile Duct Injury

J. E. J. Krige, Eduard Jonas, and Jessica Lindemann

In this chapter, Professor Kapoor comprehensively addresses two very important aspects regarding iatrogenic bile duct injury (BDI). While the medico-legal issues are often reported and frequently sensationalized, there has been less focus on the socio-economic impact, in particular from the patients' perspective. In the worst case scenario, a patient can face financial ruin with severe repercussions, especially where he or she is the (sole) breadwinner for an extended family. The implications of a BDI can thus be profound for both the patient and the surgeon. The looming specter of protracted hospitalization and investigations, anxiety of further major reconstructive surgery, a lengthy rehabilitation period, subsequent decreased quality of life (QoL), loss of income, and in some cases prolonged and unpleasant litigation aggravate this distressing complication [42]. In addition, the associated mea culpa concerns and moral liability as well as the consequences of litigation may have a detrimental effect on the injuring surgeon's life and psyche. Possible reputational damage to the surgeon's practice may be further compounded by angst, loss of confidence, uncomplimentary media coverage, and protracted litigation. Ultimately, both parties, the patient and the surgeon, may become victims as two opposing legal teams quibble, spar, and joust for pecuniary advantage and a favorable verdict.

The financial consequences implicit in the treatment of a major BDI can be substantial for the health care provider, the injuring surgeon, malpractice insurance companies, and not least the patient and, therefore, warrant a critical assessment [42]. Whereas there are data on the costs incurred for the health care provider and the magnitude of financial settlements by the injuring surgeon and/ or insurance companies, there is little in-depth information from the patients' perspective. Despite the limited detailed data on the topic, Professor Kapoor has succinctly summarized the available information on costs in the USA, Belgium, Netherlands, and India, emphasizing that the total cost of BDI repair is considerably greater than that of an uncomplicated laparoscopic cholecystectomy (LC) and that the quantum increases with delayed recognition of the injury and with an inadequate repair by the injuring surgeon. Savader [1] reported that the mean cost of a definitive bile duct reconstruction was $51,411and ranged from 4.5 to 26 times the cost of an uncomplicated LC. In the analysis of financial liability in our study from Cape Town South Africa, all costs ($1 = ZAR12) plus adjustment for inflation in the cohort of 44 patients with major BDI repaired by Roux-en-Y hepatico-jejunostomy were considered, including a comprehensive calculation of costs from the time of the hospital admission for the definitive repair to discharge. The mean cost of ZAR 215,711 was substantial and 6.4 times the cost of an uncomplicated LC with the most expensive repair amounting to ZAR 980,830 which involved 86 days in hospital [6]. For methodological rea-

sons, it was not possible to accurately calculate the costs incurred at the referring hospital in our study. Accumulated costs before referral can be substantial, including imaging, intervention for complications, and attempted repair of the injury. Data on correlation of the cost of the repair with possible modifiable factors responsible for increasing the cost of repair are lacking. Intuitively, delayed recognition, sepsis, and complications ought to have an adverse effect on the eventual outcome and cost of repairing a BDI due to increased length of ICU and hospital stay, increased imaging investigations and interventions to address intra-abdominal sepsis. However, these factors did not have a statistically significant effect on the costs in our study. Early recognition of a BDI and referral to a hepato-biliary surgeon/center are essential to reduce the morbidity and ensure a satisfactory surgical outcome. Yet in our study, more than half (57%) of the injuries were only recognized after 5 days, with 19 of 25 patients presenting with sepsis due to cholangitis or biliary peritonitis, a finding similar to the previously reported data [6]. These findings reinforce the maxim that all LC patients in the event of any unexpected postoperative symptoms or signs require careful assessment and thorough investigation to exclude an iatrogenic BDI. The actual BDI is a single event at a point in time where surgical misjudgment is the compelling *malfaisant*, and mitigating circumstances such as advanced pathology, although not an absolutely defensible justification, could be argued. On the contrary, delays in diagnosis of the injury and not taking appropriate action as the result of misinterpreting clinical signs and failure to act are, from a medico-legal perspective, regarded in a more serious light [42].

The threat of litigation looms large in complications following laparoscopic surgery. In no other surgical field has the growth of litigation been as rapid as in LC and one-third of general surgery indemnity (money paid out by insurers) arises from laparoscopic procedures. The vulnerability of BDIs to civil litigation is well known to the plaintiff and in medical malpractice litigation, biliary injury ranks first on the list of negligence claims worthy of pursuance. Four studies from the USA by Kern [28], Chandler [43], McLean [27], and Carroll [30] have examined litigation following LC. Among these, McLean [27] reported an average payment of US $508,341 in 104 patients where complications followed LC. In England, data from the National Health Service Litigation Authority on clinical negligence claims following LC showed that 418 claims were made in 2006, of which 303 were settled, two-thirds in the claimant's favor. The average payout for a successful claim was $168,337 [22]. However, the application and outcome of medical malpractice litigation vary considerably world-wide. For example, the system in the Netherlands differs from the litigation and tort system used in the US. Dutch malpractice litigation is conducted through an arbitration system, as is common in several European countries. There are no jury trials, there is no contingency system, and large settlements are less common with a mean financial settlement of €12,795 for BDI [20].

For patients, the impact of iatrogenic BDI is well documented in terms of morbidity, mortality, length of hospital stay, and long-term outcomes. Detailed QoL assessment, by contrast, has received considerably less attention. Assessing health-related QoL (HRQoL) is important and relevant as patient-reported outcomes provide a method of quantifying the impact and consequences of a BDI from the patient's perspective. However, previous studies on QoL outcome after BDI have provided discordant results due to underpowered designs, sample selection, selection bias, and inclusion of heterogeneous populations treated with either endoscopy, interventional radiology, or surgery [44].Some previous studies found no difference in HRQoL between patients who had a BDI during a LC and those who had an uneventful LC, while other studies have demonstrated a significant long-term reduction in both physical and mental HRQoL [18].

In the first detailed analysis of the impact of BDI during a LC on HRQoL, the Academic Medical Center (AMC), Amsterdam, Netherlands group used the Short Form 36 HealthSurvey® (SF-36) and reported worse physical and mental HRQoL after BDI, compared to an uncomplicated LC cohort and average general population values [12]. These findings were later replicated in an

expanded sample by the same group and were similar to Moore's [11] report of worse SF-36 physical and mental HRQoL in BDI patients compared with LC patients. In a study that used a modified City of Hope HRQoL assessment tool, Melton [45], however, reported worse mental HRQoL in BDI patients but no effect of BDI on physical and social HRQoL, compared with a healthy cohort. This is in contrast to the reports by Sarmiento [46] and Hogan [47] who found no differences in HRQoL between BDI and comparison cohorts. Sarmiento [46] reported no difference in HRQoL after a minimum of 5 years between patients who had surgical reconstruction after BDI and those undergoing a LC while Hogan [47] concluded that the QoL of surviving patients after BDI compares favorably with that of an uncomplicated LC.

In the largest study to date from Academic Medical Center (AMC), Amsterdam, Netherlands, 800 BDI patients were evaluated after multimodality treatment. Besides the anticipated clinical outcomes, which included significantly worse general physical and disease-specific QoL, several other highly significant social determinants such as loss of productivity in paid and unpaid work and high rates of disability benefits usage occurred in BDI patients [15]. A binary regression analysis showed a significant reduction in QoL in patients involved in malpractice litigation. In this subgroup, disease-specific QoL was significantly better when the verdict of the malpractice claim was in the patient's favor than when it was not [20].

As mentioned earlier, there is a paucity of data on the indirect and personal costs of a BDI incurred by the patient. Analyses should incorporate diverse costs such as loss of income due to time off work, travel expenses, medical fees, rehabilitation and litigation costs to accurately quantify the overall financial implications of a BDI. Loss of income encompasses not only time away from work while in hospital, but also during the convalescent period and follow-up visits. This may also extend to a partner or spouse involved in a supportive capacity who may need to travel long distances between home and the hospital where the repair is done [6]. In a worst case scenario, inability to return to his or her profession may result in permanent loss of income.

Professor Kapoor has elegantly outlined the essential socio-economic and litigation implications of a major BDI. He has emphasized that a BDI incurred during LC remains a serious concern for patients, healthcare providers, and employers and is associated with long-term QoL impairment as well as increased costs that may extend years after surgery. The ensuing consequences may lead to litigation which results in a substantial financial drain on the health care and insurance systems.

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Institutional Experiences with Bile Duct Injury

19

Vinay K. Kapoor

Some institutions and several individuals with large experiences have made significant contributions to our knowledge about bile duct injury during cholecystectomy. The following is a select list of these contributions:

19.1 Hôpital Paul-Brousse, Villejuif South Paris France

Henri Bismuth

Corlette MB, Bismuth H. Biliobiliary fistula. A trap in the surgery of cholelithiasis. Arch Surg. 1975 Apr;110(4):377–83. PMID:1147754

Bismuth H, Franco D, Corlette MB, Hepp J. Long term results of Roux-en-Y hepaticojejunostomy. Surg Gynecol Obstet. 1978 Feb;146(2):161–7. PMID:622659

Hepatico-jejunostomy in 123 patients – no mortality – 1 restricture and 6 intrahepatic lithiasis during 5.5 years follow-up, 2 peptic ulcers

Bismuth H. Postoperative strictures of the biliary tract. In Blumgart LH, editor. The Biliary Tract. Clinical Surgery International vol. 5. Edinburgh: Churchill Livingstone 1982: 209–218.

Gugenheim J, Ciardullo M, Traynor O, Bismuth H. Bronchobiliary fistulas in adults. Ann Surg. 1988 Jan;207(1):90–4. PMID:3337567

Bismuth H, Majno PE. Biliary strictures: classification based on the principles of surgical treatment. World J Surg. 2001 Oct;25(10):1241–4.

Bismuth H. Surgical management of bile duct stricture following laparoscopic cholecystectomy. Acta Chir Belg. 2003 Apr;103(2):140–2. No abstract available. PMID:12768854

19.2 Hammersmith Hospital and King's College Hospital, London UK

Leslie H. Blumgart and Irving S. Benjamin

Blumgart LH, Kelly CJ. Hepaticojejunostomy in benign and malignant high bile duct stricture: approaches to the left ducts. Br J Surg. 1984 Apr;71(4):257–61. PMID:6367885.

Blumgart LH, Kelley CJ, Benjamin IS. Benign bile duct stricture following cholecystectomy: critical factors in management. Br J Surg. 1984 Nov;71(11):836–43. PMID:6326926

63 patients with benign biliary stricture (BBS) underwent hepatico-jejunostomy over an 8-year period—90% had a satisfactory outcome at a median follow-up of 3.3 years

Czerniak A, Thompson JN, Soreide O, Benjamin IS, Blumgart LH. The management

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of fistulas of the biliary tract after injury to the bile duct during cholecystectomy. Surg Gynecol Obstet. 1988 Jul;167(1):33–8. PMID:3381182.

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Repair of 110 benign biliary strictures (BBS) with 1.8% mortality. At a follow-up of 7.2 years, 76% patients had good result. 11 out of 22 failures had a good result after reintervention (radiological or surgical)—final 87% good result.

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19.3 Johns Hopkins Hospital, Baltimore MD USA

John L. Cameron, Henry A. Pitt, and Keith D. Lillemoe

200 major bile duct injuries (including 175 surgical repairs) between 1990 and 2003

Cameron JL, Skinner DB, Zuidema GD. Long term transhepatic intubation for hilar hepatic duct strictures. Ann Surg. 1976 May;183(5):488–95. PMID:1275590

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19.4 Academic Medical Center (AMC), Amsterdam Netherlands

Dirk J. Gouma and Thomas M. van Gulik

More than 800 bile duct injuries managed between 1991 and 2005.

Gouma DJ, Go PM. Bile duct injury during laparoscopic and conventional cholecystectomy. J Am Coll Surg. 1994 Mar;178(3):229–33. PMID:8149013

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19.5 National Institute of Medical Science and Nutrition, Tlalpan Mexico City Mexico

Miguel Angel Mercado

National referral center for bile duct injuries in Mexico has performed 614 bile duct reconstructions between 1989 and 2014.

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Bansal VK, et al, Kapoor VK. SELSI Consensus Statement for Safe Cholecystectomy — Prevention and Management of Bile Duct Injury — Part A. Indian J Surg 2019 https://doi.org/10.1007/s12262-019-01993-2

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Bile Duct Injury Stories

20

Vinay K. Kapoor

20.1 My Tryst with Destiny: Cholecystectomy and Bile Duct Injury

Manali Arora

All readers are urged to go through the story of Manali Arora who had to undergo multiple operations and interventions for a bile duct injury sustained at a laparoscopic cholecystectomy performed in 2006.

Arora M, Kapoor VK. My tryst with destiny – cholecystectomy and bile duct injury. National Medical Journal of India 2010; 23: 32-33.

The article is available at http://archive.nmji.in/archives/Volume-23/Issue-1/PDF-volume-23-issue-1/Speaking-For-Myself.pdf

Manali had to undergo a right hepatectomy in 2008. After finishing her MBBS from Gauhati Medical College, Guwahati Assam in 2011, Manali obtained MD (Radio-Diagnosis) in 2015 from Assam Medical College, Dibrugarh Assam and then passed DNB (Radio-Diagnosis) in 2017. Manali married Vishal Thakker in 2015. After working as Assistant Professor of Radiology at Pramukhswami Medical College, Anand Gujarat, she is currently pursuing a Post Doctoral Certificate Course (PDCC) in Breast Radiology at the All India Institute of Medical Sciences (AIIMS), Rishikesh Uttarakhand.

20.2 Doctor, Is this My Last Surgery?

Shweta Amrita Lakra

Lakra SA, Kapoor VK. Doctor, is this my last surgery? Indian Journal of Surgery 2018; 80: 377-381.

The article is available at https://link.springer.com/article/10.1007/s12262-017-1675-2

Shweta was operated for her bile duct injury in 2014. She obtained her PhD (Environmental Sciences) from the Indian Agricultural Research Institute (IARI), New Delhi in February 2017. Shweta married Pankaj Abhishek Toppo in January 2017. After working as Assistant Professor (Environmental Sciences) at St Xaviers College, Ranchi Jharkhand, she is currently a Food Safety Officer with Government of Jharkhand.

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