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The Mirizzi Syndrome: Multidisciplinary Management Promotes Optimal Outcomes

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Abstract The Mirizzi syndrome (MS) is a rare cause of obstructive jaundice produced by the impaction of a gallstone either in the cystic duct or in the gallbladder, resulting in stenosis of the extrahepatic bile duct and, in severe cases, direct cholecystocholedochal fistula formation. Sixteen patients were treated for MS in our center over the 12-year period 1993–2005 for a prevalence of 0.35% of all cholecystectomies performed. One patient was diagnosed only at the time of cholecystectomy. The other 15 patients presented with laboratory and imaging findings consistent with choledocholithiasis and underwent preoperative endoscopic retrograde cholangiopancreatography, which established the diagnosis in all but one patient. All patients underwent cholecystectomy. An initial laparoscopic approach was attempted in 14 patients, of whom 11 were converted to open procedures. MS was recognized operatively in 15 patients with definitive stone extraction and relief of obstruction in 13 patients. T-tubes were placed in 10 patients and 1 patient required a choledochoduodenostomy. Two patients required postoperative laser lithotripsy via a T-tube tract to clear their stones; and in another patient, MS was detected and treated via postoperative endoscopic retrograde cholangiopancreatography (ERCP). MS remains a serious diagnostic and therapeutic challenge for endoscopists and biliary surgeons.

Keywords Cholelithiasis · Choledocholithiasis · Cholecystectomy · Endoscopic retrograde · Cholangiopancreatography · Mirizzi syndrome

Introduction

In 1948, an Argentinean surgeon Pablo Mirizzi first described an atypical presentation of gallstone disease in which the impaction of a gallstone in either the cystic duct or the gallbladder (GB) caused stenosis of the extrahepatic bile duct by extrinsic compression and/or fibrosis. In some cases, the associated inflammation was noted to progress to cholecystocholedochal fistula formation.[1] Now known as Mirizzi syndrome (MS), this rare cause of obstructive jaundice is reported to occur in 0.7–2.5% of all U.S. patients undergoing cholecystectomy.[2,3] It is of particular importance to surgeons because the diagnosis may not be appreciated preoperatively and because the surgical treatment of this condition is associated with a significantly increased risk of bile duct injury. Furthermore, intraoperative recognition of MS may be difficult, especially if a fistula is present or extensive adhesions complicate the dissection. Accurate definition of the biliary anatomy preoperatively, when possible, is thus critical for optimal surgical planning. To further characterize the contemporary management of this difficult problem, we reviewed our experience with MS over the last 12 years.

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Patients and Methods

We reviewed the medical records of 16 patients who were diagnosed with MS at our institution between August 1993

and July 2005. All patients were identified either from a surgical database maintained prospectively by the Department of Surgery or from an endoscopic retrograde cholangiopancreatography (ERCP) database maintained prospectively by one of the authors (GWWG). Records were reviewed for presenting clinical and laboratory findings, noninvasive imaging and ERCP findings, operative management, and patient outcome. This study was approved by the institutional IRB.

Patients were classified according to the system of Csendes et al.[4] Eight patients were type IA, two were type IB, four were type II, and two were type III. No patients were type IV. All 16 patients underwent cholecystectomy, representing 0.35% of the 4,589 patients who underwent cholecystectomy at our center during the study period. Fourteen patients underwent preoperative ERCP, which established the diagnosis of MS in 13 of the patients. Two additional patients underwent cholecystectomy without preoperative ERCP; the diagnosis of MS was established intraoperatively in one patient and postoperatively in the other.

Clinical Characteristics, Laboratory Findings, and Noninvasive Imaging

Table 1 illustrates the clinical characteristics at presentation. In 14 patients, the chief presenting complaint was acute upper abdominal pain. An additional patient presented with a chief complaint of constipation but upon questioning also admitted to 1 day of mild right upper quadrant pain. The last patient presented with painless jaundice. Table 2 summarizes pertinent laboratory findings upon admission. Of the 16 patients, 15 presented with abnormalities of liver-associated enzymes.

Table 1 Clinical Characteristics at Presentation

	N=16
Sex, M/F	7/9
Mean age, years (range)	48 (22–83)
Presenting symptoms (*n*)	
Acute abdominal pain	15
Location	
RUQ	9
MEP	2
RUQ+MEP	2
RUQ+MEP+LUQ	2
Mean duration, days (range)	2.7 (1–7)
Jaundice	12
Nausea and vomiting	7
Chronic abdominal pain	6
Mean duration, months (range)	83 (2–324)
Fever (>100.5°F)	4
Diarrhea	1
Constipation	1

RUQ: right upper quadrant, *MEP*: midepigastric, *LUQ*: left upper quadrant

Table 2 Laboratory Values at Presentation

Test	Mean (range)
WBC (1,000/μl)	8.9 (3.8–17.3)
Total bilirubin (mg/dl)	5.4 (0.5–12.6)
AST (IU/l)	549 (12–2,176)
ALT (IU/l)	602 (29–2,626)
Alkaline phosphatase (IU/l)	329 (100–692)
GGT (IU/l)	632 (37–1,707)
Amylase (IU/l)	175 (10–1,844)
Lipase (IU/l)	56 (18–131)

WBC: white blood cell, *AST*: aspartate aminotransferase, *ALT*: alanine aminotransferase, *GGT*: gamma-glutamyl transpeptidase

All 16 patients underwent abdominal ultrasonography (US) at presentation. All studies revealed gallbladder stones; and in one patient, a 9-mm cystic duct stone was also described. US showed dilation of the extrahepatic duct (caliber >6.0 mm) in 12 of 16 patients; the mean caliber in all patients was 8.1 mm (range 3.2–12.8). US detected dilation of the intrahepatic biliary tree in seven patients. Six patients additionally underwent computed tomography (CT) of the abdomen, revealing gallbladder stones in the five patients who were studied before cholecystectomy. CT revealed extrahepatic biliary dilation in five of six patients; and in one case, this dilation could be localized to the proximal extrahepatic duct. CT demonstrated intrahepatic biliary dilation in three of six patients. In all cases, the initial presumptive diagnosis after noninvasive evaluation was acute cholecystitis and/or choledocholithiasis. In no case was the diagnosis of MS established by noninvasive imaging.

ERCP Findings

Table 3 summarizes the ERCP findings in 15 patients. One patient underwent cholecystectomy without preoperative ERCP and the diagnosis of MS was not established until postoperative ERCP. Fourteen patients underwent preoperative ERCP with documentation of the diagnosis of MS in 13 cases. Of these patients, 4 had 2 preoperative ERCP procedures each, for a total of 16 preoperative ERCPs, and 1 other patient underwent both preoperative and postoperative ERCPs. All cases identified with MS during ERCP were characterized cholangiographically by the presence of stenosis in the extrahepatic bile duct corresponding to the site of the cystic duct insertion. In some cases, no stone could be visualized at the level of stenosis; in other cases, stones could be clearly demonstrated within the cystic duct

Table 3 ERCP Findings and Interventions in 15 Patients

Finding	Number
Stenosis of the extrahepatic bile duct	14
Cystic duct insertion identified at level of stenosis	14[a]
Intrahepatic biliary dilation	14
Pus present in bile duct	6
Stone(s) within cystic duct adjacent to bile duct	8
Actual or apparent bile duct stone(s)	6
Interventions	
Biliary sphincterotomy	14
Nasobiliary drain or stent placement	11[b]
Extraction of visualized or suspected BDS attempted	14
Successful extraction: complete/partial	2/2[c]
Extraction of cystic duct stone(s) attempted	5
Success	0

[a] In three cases, the cystic duct was shown to be nearly or completely obliterated with fusion of the gallbladder neck to the bile duct.
[b] Drain or stent placement was attempted in 14 patients but was unsuccessful in 3 because of lack of patient cooperation.
[c] See text.

or at the cystic duct–bile duct junction. Rarely, MS was recognized only after multiple attempts to extract stones that appeared to be within the bile duct were unsuccessful and it was realized that the stones were anatomically adjacent to but not within the bile duct (Figs. 1 and 2).

In the 14 patients in whom extraction of visualized or suspected bile duct stones was attempted, 1 to 6 passes were made using a 11.5-mm and/or a 15-mm stone extraction balloon. In several cases, both the passage of the guide wire and the balloon catheter beyond the point of obstruction were extremely difficult. Passage of stone extraction baskets above the level of stenosis was attempted in four patients but could be achieved in only three. These

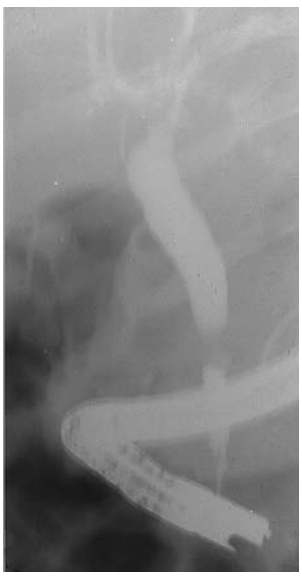

Figure 1 ERCP in Mirizzi syndrome. Stone in the cystic duct overlies the common bile duct and mimics the typical appearance of choledocholithiasis.

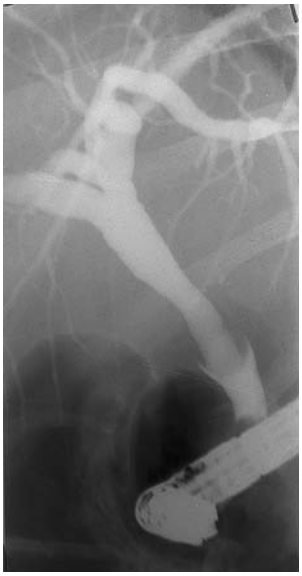

Figure 2 ERCP demonstrates the outline of the obstructing stone in the cystic duct. Contrast meniscus clearly extends outside the wall of the common bile duct.

interventions within the common bile duct yielded recovery of stone material in four patients. In two cases, small stone fragments were delivered to the duodenum but the majority of the stone burden remained in situ. In the third case, a discrete bile duct stone was crushed with a lithotripsy basket and delivered to the duodenum, however, several large stones remained within the adjacent cystic duct with associated compression of the common bile duct. In the fourth case, multiple stones were identified within the bile duct and successfully extracted. However, several stones were clustered adjacent to the bile duct within the gallbladder neck and could not be removed.

Endoscopic extraction of stones in the cystic duct was attempted in five cases. Passage of a guide wire through the cystic duct to the gallbladder past the obstructing cystic duct stones was attempted in four patients with technical success in two. In these two patients, stone balloons were advanced to the gallbladder neck, inflated, and pulled retrograde through the cystic duct to the duodenum, but no stone material could be dislodged. In the fifth patient, a basket was successfully passed deep within the cystic duct, but again, attempted removal of the stones was unsuccessful.

Operative Findings

Cholecystectomy was performed in all 16 patients; Table 4 summarizes the operative findings. The laparoscopic approach was attempted in 14 patients but converted to an open procedure in 11 patients (78.6%) because of indistinct anatomy (6), abnormal cholangiography (3), or technical problems (2). Of the patients, 15 were noted to have extensive adhesions and severe inflammation involving the gallbladder, liver bed, portal area, cystic duct, and/or

Table 4 Operative Characteristics in 16 Patients

	Number
Initial open cholecystectomy	2
Initial laparoscopic cholecystectomy	14
Conversion, laparoscopic to open with reason	11
Inflammation/adhesions/unclear anatomy	5
Abnormal IOC	3
Gallbladder perforation	1
Inadvertent choledochotomy	1
Failure to free cystic duct stone laparoscopically	1
Bile duct entered	11
Deliberate choledochotomy (for CBDE)	6
Defect created by removal of GB and/or stone	3
Inadvertent choledochotomy	1
Puncture for IOC	1
Closure: T-tube	10
Choledochoduodenostomy	1
Bile duct exploration	11
Direct	9
Transcystic	2
Location of stones identified/extracted	15
Cystic duct/bile duct junction	6
Cystic duct	4
Bile duct	3[a]
Gallbladder	2
Intraoperative imaging (IOC/laparoscopic US)	13/1
Stone at cyst duct/bile duct junction	4
Cystic duct stone	2
Inflammatory bile duct stenosis	2
Bile duct stone(s)	2
Normal	4

[a] In one patient, multiple BDS were removed at open CBDE but residual stones remained in the distal BD on the final cholangiogram. In another patient, a large stone adherent to the bile duct wall could not be removed and was left in situ.

common bile duct. Discrete inflammatory masses involving the cystic duct or bile duct were described in three patients.

The diagnosis of MS was established or confirmed intraoperatively in 15 of 16 patients, including the patient who was admitted with normal liver tests. In one patient, the presence of MS was not appreciated at the time of the initial operation. Because of nausea, vomiting, abdominal pain, and fever on the third postoperative day, an ERCP was attempted but failed to achieve biliary access despite needle–knife sphincterotomy. Subsequent percutaneous transhepatic cholangiography (PTC) revealed a high-grade distal bile duct stenosis without evidence of intraductal stones. A repeat ERCP on postoperative day 10 confirmed a tight stenosis in the distal bile duct, which was by-passed with extreme difficulty. No intraductal stone was noted, and multiple passes with a stone extraction balloon failed to yield stone material. The cystic duct stump was shown to insert low on the common bile duct, corresponding to the level of obstruction, suggesting distal bile duct compression from a stone in the adjacent cystic duct stump. A plastic endoprosthesis was placed and a repeat ERCP performed after a 3-month delay. After the extension of the biliary sphincterotomy, a large stone was successfully extracted from the junction of the cystic duct stump and the common bile duct with complete relief of biliary obstruction.

Postoperative Course and Management

The mean length of hospital stay in this series was 13.5 days (range 3–59). Initial postoperative recovery was uneventful in all patients except for a single case of *Clostridium difficile* diarrhea, which resolved with standard medical therapy. Postoperative T-tube cholangiography (TTC) was available in 11 patients and found to be normal in 5. In one patient, a 1.5-cm long inflammatory stricture was found in the middle aspect of the common bile duct. The patient required a brief interval of antibiotics but a repeat TTC 1 month later showed complete resolution of the stricture. Residual common bile duct stones were observed in another five patients. In three patients, stones passed spontaneously through a preexisting endoscopic biliary sphincterotomy and did not require further intervention. In the other two patients, ductal stones persisted and required endoscopic ablation and removal with the use of a Holmium laser using a previously described technique. Overall, MS was definitively treated during the index operation in 13 of 16 patients (81.3%).

Discussion

Born in Cordoba, Argentina, Dr. Pablo L Mirizzi (1893–1964) was recognized during his lifetime as a leading figure in surgery and is still recognized today for his enduring impact on current medical practice. Mirizzi's contributions to the surgical management of biliary tract disease occurred in the first half of the twentieth century at a time when the field was still in its infancy. Open cholecystectomy had only recently been introduced by Langenbuch in 1882 and surgeons were just beginning to confront the challenge of concomitant bile duct stones (BDS).[5] Courvoisier reported the first operative bile duct exploration in 1890,[6] but it was not until 1931 that Mirizzi first introduced intraoperative cholangiography (IOC).[7] This technique, in which a lipid contrast agent is injected into the cystic duct to achieve a radiographic image of the biliary tree, was initially termed *mirizzigrafia* and represented a major diagnostic advance. Such studies allowed for the intraoperative visualization of the biliary system and remain today as a vital element of the diagnostic algorithm used to identify biliary obstruction, BDS, biliary tract injuries, congenital ductal anomalies, and other biliary conditions.

In his original publication, Mirizzi described in detail several cases in which the intraoperative cholangiographic images were consistent with the syndrome that now bears his name. In each case, the common hepatic duct was obstructed by a gallstone located in the gallbladder. Subsequently, other investigators have demonstrated that MS commonly develops as a result of inflammatory changes involving the infundibulum of the gallbladder or the cystic duct in response to the impaction of a stone. The presence of a long intramural cystic duct or a low insertion of the cystic duct into the common bile duct appears to predispose to the syndrome, but these anatomic variants are not always present. With progressive inflammation, the gallbladder may contract and become fused to the common duct. This may produce secondary stenosis of the common duct and, in the severest cases, can promote fistula formation from direct pressure necrosis of the adjacent duct walls by the impacted stone.[2] These varying degrees of pathologic involvement are well-represented in our patients.

As also documented in the current series, Mirizzi's syndrome is not easily diagnosed in the preoperative period. In large part, this reflects the fact that the syndrome is not associated with a well-defined set of demographics or unique clinical features. The patients in our series ranged in age from 22 to 83 years. In addition, most presented with clinical and laboratory findings generally consistent with obstructive choledocholithiasis in which the degree of obstruction, as measured by serum enzyme and bilirubin levels, ranged from mild to severe. This absence of reliable clinical indicators presents a major challenge for primary care physicians and their consultants.

Because of these limitations, the preoperative diagnosis of Mirizzi's syndrome depends heavily on appropriate imaging studies. Plain abdominal films alone have not proven useful. Similarly, US or CT scans are not often definitive although both studies may demonstrate findings that strongly suggest the diagnosis, such as: (1) dilatation of the biliary tree above the level of the gallbladder neck, (2) impaction of a stone in the gallbladder neck, and (3) a normal caliber CBD below the level of impaction.[8] Recent case reports suggest that magnetic resonance cholangiography can also be an effective method of diagnosing MS using the same criteria.[9] In addition, Wehrmann, et al. reported using intraductal ultrasonography to achieve a diagnosis of MS in 30 patients with a sensitivity of 97% and a specificity of 100%.[10] Of note, the imaging findings associated with MS may be indistinguishable from those of a ductal malignancy. An appropriate clinical history and the presence of portal or hepatic mass lesions or adenopathy may help to differentiate these two different pathologies.

ERCP remains the most effective preoperative test for Mirizzi's syndrome and can provide a relatively precise localization and characterization of the source of the biliary obstruction. Typical findings of Mirizzi's syndrome at ERCP include (1) midbile duct obstruction with dilated proximal CHD and intrahepatic ducts combined with normal duct caliber distal to the obstruction, (2) insertion of the cystic duct at the point of obstruction and/or complete obliteration of the cystic duct, and (3) a stone visualized at the point of obstruction either within the cystic duct or the common duct. Nonetheless, even ERCP is not always successful; as evidenced by the one patient in our series in which the ERCP failed to establish the correct diagnosis.[11]

Therapy for Mirizzi's syndrome continues to be problematic and must be individualized depending on the stage of the disease and the expertise of the responsible consultants. The obstructing stone, whatever its precise anatomic location, is commonly refractory to endoscopic extraction; and thus in most reports, the definitive treatment of MS remains surgical. In our series, endoscopic stone extraction was attempted in 14 of 16 patients and resulted in the recovery of a single stone and additional stone fragments in only 4 patients. However, the biliary obstruction persisted in all cases. Cystic duct cannulation was performed in five patients without the successful removal of the stones. Nonetheless, temporary relief of biliary obstruction was provided in the majority of our patients by the endoscopic insertion of a plastic stent or drain. Biliary stents subsequently proved to be a valuable guide during operative dissections.

A greater role for endoscopic therapy has been recommended by several investigators. Seitz et al. achieved successful fragmentation and clearance of stones in 38 cases utilizing electrohydraulic lithotripsy delivered via cholangioscopy with a mother–daughter endoscope system.[12] Tsuyuguchi et al. used a similar approach in 25 patients diagnosed with Mirizzi syndrome.[13] They were able to clear the obstructing stone in all 23 type II patients. Nonetheless, four of the patients required rehospitalization for acute cholangitis when residual gallbladder stones migrated into the common bile duct. Cholangioscopy was unsuccessful in two type I patients. Whereas these advanced technologies are not typically available in most practice settings, they may represent the next logical step in the evolution of endoscopic methods available for the management of Mirizzi's syndrome.

Surgical intervention remains the definitive treatment for the majority of patients and should satisfy three goals: extraction of the obstructing stone, removal of the gallbladder, and restoration of normal biliary drainage. In general, the choice of the specific operative technique depends on a relatively precise definition of the biliary anatomy. With this in mind, McSherry et al. in 1982 classified MS into two anatomic groups, based on the results of ERCP and PTC: type I (external compression of

the CHD without fistula) and type II (erosion of the stone into the CHD with cholecystocholedochal fistula formation).[14] Csendes et al. further modified this classification to better guide surgical management and demonstrated in 219 patients that the more severe grades were associated with significantly higher rates of both postoperative morbidity and mortality.[4] In our series, there were no deaths, but 11 of 14 laparoscopic cases required conversion to an open procedure and 2 patients required additional postoperative procedures for the complete clearance of intraductal stones.

Like others, we found conventional retrograde dissection inadvisable in most cases because of the risk of injuring critical structures within the triangle of Calot. Our preferred surgical approach is now a fundus-down dissection with early incision of the gallbladder fundus or body to remove the impacted stone. The latter step can aid in the identification of the subtype of the disease as a sudden gush of bile into the open gallbladder strongly suggests the presence of a fistula. A cholangiogram should be obtained for clarification and confirmation.[2,15] Depending on the patient's age and the operative findings, frozen section pathologic analysis may also be indicated to assess the specimen for malignancy as the associated incidence of carcinoma with Mirizzi syndrome has been noted to be as high as 27.8%, presumably secondary to chronic inflammation.[16] Partial cholecystectomy is recommended in selected type I patients who have severe inflammation, as it minimizes the risk of CBD injury by limiting dissection in the inflamed tissues of the triangle of Calot. A subsequent CBDE is generally not necessary as the neck of the gallbladder and the cystic duct are left in situ.[15] This approach appears to be particularly useful when primary laparoscopic procedures are attempted as previously noted by Binnie et al.[17] and Rohatgi and Singh.[18] Similarly, Chowbey, et al. were able to complete a laparoscopic cholecystectomy in 78% of 27 patients with Mirizzi syndrome when they used an endoscopic stapler to divide the gallbladder infundibulum.[19] In 2003, Yeh et al. reviewed all available English language reports of laparoscopic treatment of MS and found a total of 82 cases, including 11 of their own.[20] An overall conversion rate of 31.7% was reported. In contrast, an audit of 39 patients with Mirizzi syndrome conducted by the Swiss Association of Laparoscopic and Thoracoscopic Surgery revealed a conversion rate of 74% for type 1 lesions and 100% for type 2 lesions; an experience similar to ours.[21] Our current approach is to begin with an exploratory laparoscopy but to convert to an open operation if inflammation is severe and anatomy is obscure.

Type II defects with small fistulae can be usually treated with either complete or partial cholecystectomy followed by closure of the fistula around a common bile duct T-tube.[2] A completion cholangiogram should be obtained to clear the duct. Direct suture of the ductal defect is generally inadvisable as such closure of the thickened and friable duct can be technically difficult and strictures or leaks may result.

For repair of larger defects (types III and IV), a longitudinal choledochotomy may be performed directly over the gallstone followed by a near-total cholecystectomy. A small pedicle of mobilized gallbladder wall can be sutured around a T-tube to assure patency of the common bile duct.[13,22,23] Larger fistulas may require a more generous choledochoplasty using a vascularized and bile-tolerant gallbladder flap.[4,24] However, in both cases, strictures can develop because of continued inflammation of the flap. For this reason, we and others generally prefer a biliary-enteric bypass with a Roux-en-Y choledochojejunostomy or a choledochoduodenostomy for large fistulas or badly damaged bile ducts.[2,15,25]

In summary, the Mirizzi syndrome remains a rare but important cause of obstructive jaundice and poses major challenges for both the endoscopist and the surgeon. Noninvasive laboratory investigations and specialized radiological imaging provide only supportive diagnostic information. A preoperative ERCP or PTC is usually necessary to establish the diagnosis and define the anatomy and can also be used to achieve preoperative decompression of the obstructed ductal system. Definitive therapy usually requires an operation that is tailored to the specific clinical and anatomic findings. Available surgical options range from simple cholecystectomy to more complex choledochoplasties and biliary-enteric anastomoses. Achieving optimal outcomes depends on a skilled, multidisciplinary team that is experienced with the management of advanced biliary disease.

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