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Diego Foschi
Giuseppe Navarra *Editors*

Emergency Surgery in Obese Patients

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Diego Foschi • Giuseppe Navarra
Editors

Emergency Surgery in Obese Patients

Foreword by Paolo De Paolis

Springer

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Foreword

The incidence and prevalence of obesity have increased worldwide; in particular, severe obesity is becoming an epidemic, affecting a considerable portion of the population worldwide and leading to health, social, and economic problems. This metabolic disease is associated with a shortened life expectancy, a decreased quality of life, and increased health expenditure for the national health systems.

These considerations clearly demonstrate the necessity for a monograph that takes into consideration all the problems related to emergency surgery in the obese patient; we absolutely need to develop specific appropriate protocols and to strongly promote teamwork strategies.

Therefore, with great satisfaction I introduce this important work by Diego Foschi and Giuseppe Navarra; Diego and Giuseppe, top experts in the field, engaged coworkers whose experience and scientific excellence have produced a high-quality monograph.

This volume highlights all the important aspects of emergency surgery in obese patients, providing updates on hot topics in this area and regarding new techniques, not only related to surgery but also to the perioperative and intensive care management. Considerable attention is also paid to the metabolic and surgical complications of bariatric surgery, without forgetting to discuss the subject of accreditation in emergency bariatric surgery.

The high scientific level makes this volume valuable not only for the young surgeon who wants to understand the issues of bariatric surgery but also for the experienced surgeon who considers the sharing of knowledge and protocols a fundamental aspect of the medical profession.

On behalf of the Italian Society of Surgery, I'd like to thank all the eminent authors who collaborated in producing this very useful monograph.

Turin, Italy
September 2019

Paolo De Paolis
President
Italian Society of Surgery

Preface

We would like to start this preface by thanking the Board of the Italian Society of Surgery for giving us the opportunity to present this volume on "Emergency Surgery in Obese Patients."

In Italy, 10% of the population is obese and this percentage is expected to increase mainly in the younger age groups. Obesity carries a high risk of cardiovascular and respiratory complications and is accompanied by several comorbidities which make the obese a medically challenging patient population. This is especially true in the emergency setting when optimal treatment of obese patients relies on a multidisciplinary approach with close cooperation between many specialists. If the best care is provided, however, the prognosis of obese patients seems to be better than that of normal-weight subjects for many pathologic conditions: this is the obesity paradox. To obtain these results, a profound knowledge of the pathophysiology of morbid obesity and its consequences on different body systems is essential. Resuscitation, anesthesia, and intensive care management of the obese patient raise specific problems and need appropriate solutions. The first part of this volume examines this topic extensively with an easily understandable approach.

The second part of this volume focuses on surgical emergencies in the obese population. Prevention and treatment of the abdominal compartment syndrome, trauma, and burns are also dealt with in this part. Symptoms may be atypical, signs are poor, and the possibility of sudden deterioration of the general condition is very frequent. Patients are often old and affected by several chronic conditions. A rapid diagnosis and prompt treatment improve the results of surgery and lower the incidence of complications in several clinical conditions.

Bariatric surgery has increased dramatically in our country: in the last 5 years, it is estimated that more than 70,000 operations have taken place, 99% of which have been performed laparoscopically, mainly by four procedures: sleeve gastrectomy, Roux-en-Y gastric bypass, adjustable gastric banding, and one-anastomosis-gastric bypass. Other operations are less frequent. Each operation is characterized by specific complications, which can occur early or late after surgery. Although they can be classified as septic, hemorrhagic, and obstructive, the possibility of recognizing their causes after surgery needs a profound knowledge of bariatric surgery. Although some complications are common to all general surgery patients, others are unique to the bariatric patient and a few may follow either general or bariatric surgery but may differ in clinical presentation and management between the two patient populations.

We thank all the contributors to this part of the volume for their effort to present the very difficult aspects of the complications of bariatric surgery. These are very distinguished clinicians and researchers—fellows of the Italian Society of Surgery (SIC), Italian Association of Hospital Surgeons (ACOI), and Italian Society of Obesity Surgery and Metabolic Diseases (SICOb)—who have collaborated together to provide the reader with the in-depth knowledge of emergency surgery in the obese patients after bariatric surgery, under the auspices of the Società Italiana di Chirurgia.

Finally, we would like to acknowledge the fundamental contributions of Juliette Kleemann and Donatella Rizza at Springer and of Marco Martorelli at Scienzaperta in realizing this excellent book.

In conclusion, we hope this volume will offer general and bariatric surgeons as well as emergency medicine professionals a valid tool to help them in the decision-making processes concerning obese patients in the emergency setting.

Milan, Italy — Diego Foschi
Messina, Italy — Giuseppe Navarra
September 2019

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Part I

Anesthesia and Resuscitation in Obese Patients

1 Frailty of the Obese Patient and the Obesity Paradox After Surgical Stress

Diego Foschi, Marcello Lucchese, Giuliano Sarro, and Andrea Rizzi

1.1 Introduction

Obesity is a metabolic disease characterized by abnormal or excessive adipose tissue accumulation and body weight increase. It is recognized on the basis of a number of anthropometric characteristics and can be classified according to the body mass index (BMI = weight [kg]/height [m^2]) into three different classes [1]:

- *class I*: BMI 30–34.9
- *class II*: BMI 35–39.9
- *class III*: BMI ≥40

Obesity reduces life expectancy, especially when BMI > 35 [2, 3], since it is generally related to concomitant chronic metabolic complications (hypertension, insulin resistance, cholesterol and glucose increase), which are prognostic factors for cancer [4], cardiovascular diseases (CVD) and stroke [2, 5].

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1.2 The Obesity Paradox

The obesity paradox has been observed in many cases in the literature; clinically healthy obese patients have an increased overall survival in cases of heart failure, even though their condition reduces life expectancy. Furthermore, under acute stress, such as surgical stress, obesity "protects" against mortality [6–27]. This evidence-based conclusion is supported by the literature, even though a few citations do not confirm these findings [18–22].

In 2003, Dindo et al. [7] studied postoperative complications in 6336 patients undergoing elective general surgery. Patients were classified according to BMI: non-obese (BMI < 30), mildly obese (BMI 30–35) and severely obese (BMI > 35). The incidence of complications was the same in all three groups (16.3% vs. 16% vs. 15.1%) and an additional multivariate regression analysis showed that obesity was not a risk factor. Mortality was not investigated. In a prospective, multi-centric clinical study, Mullen et al. [8] found that in 118,707 patients undergoing non-bariatric surgery, the mortality risk related to BMI showed a reverse J-shaped relationship; highest rates were found in underweight and morbidly obese patients. Overweight and moderately obese patients had the lowest risk of mortality. A prospective analysis of a single center clinical study investigating postoperative complications [9] studied 4293 patients, of whom 743 were obese. Obese patients more frequently reported diabetes (18.1% vs. 4.7%), hypertension (30.3% vs. 14.2%), cardiac (21.3% vs. 16.7%) or pulmonary (18.6% vs. 14.4%) diseases. They used medications more frequently than normal-weight patients, yet they were less frequently smokers (26.9% vs. 35.4%). Obesity resulted in a significantly longer intervention time, higher intraoperative blood loss and rate of surgical site infections (SSI) but not mortality, considered at 30 days. Furthermore, mildly obese or overweight patients had longer overall survival. The above observations were confirmed by several studies. Vargo et al. [10] studied 6,240,995 patients who underwent cardiovascular surgery, of whom about 10% were obese. These patients had lower in-hospital mortality (2% vs. 4.2%, $p < 0.0001$), postoperative stroke (1.3% vs. 2.3%) and incidence of pneumonia (3.6% vs. 5.1%), the most common complication, but a higher incidence of acute renal failure (8.7% vs. 8.2%) and need for blood transfusions (20.9% vs. 19.3%). The risk of wound infection was also higher (1.1% vs. 0.8%). In the vascular surgery setting [11, 12], obese patients had lower cardiac and respiratory morbidity as well as lower mortality in comparison to normal-weight patients. However, a higher rate of wound complications was observed. Obese patients who underwent esophageal [13], gastric [14] and colon surgery [15–17] had prolonged intraoperative intervention time and increased complication rate and SSI but had no difference in perioperative mortality and reoperation rate. Similar results were observed in patients who underwent surgery for Crohn's disease [18]; BMI did not influence cardiac, pulmonary and renal complications or mortality but patients with a BMI > 40 had a higher prevalence of SSI.

In addition, obese patients affected by various organ cancers experienced less serious morbidity and lower risk-adjusted odds of mortality, despite a higher frequency of deep venous thrombosis, renal complications and ventilator dependency (considered as >48 h) [19]. Benjamin et al. [20] retrospectively reviewed the ACS-NSQJP database and extrapolated 101,078 patients who underwent emergency

abdominal surgery between 2005 and 2010; approximately 30% were obese, 32% overweight, 3.5% underweight and the remaining normal weight. A history of diabetes and hypertension was more frequent in the obese group; a higher complication rate was evident in the underweight and morbidly obese patients. Crude mortality was increased in the underweight group alone.

Different results were obtained in obese patients admitted to the intensive care unit (ICU) for blunt trauma [21]. Obese patients had fewer head injuries but more chest and lower-extremity traumas. Nevertheless, they had more complications, longer time of mechanical ventilation and ICU stay. In the above study, obesity was an independent risk factor for mortality. In a prospective study on 1167 patients admitted to the ICU after trauma, Bochicchio et al. [22] examined the outcome of 62 obese (BMI > 30) patients (5.3% of the total). More than a two-fold increase in risk of infection was observed and seven-times higher likelihood of in-hospital death. Ditillo et al. [23] accessed the USA National Trauma Data Base and identified 32,780 morbidly obese patients who had blunt trauma injury. These patients had higher in-hospital complication rate, longer ICU- and in-hospital stay and higher mortality in morbidly obese patients compared with the non-obese population. Furthermore, Diaz et al. [24, 62] measured fasting glucose plasma levels in 1334 blunt trauma patients and found that mortality was related more to hyperglycemia than to morbid obesity (BMI > 40). Observations on adult patients in ICUs, including medical patients, produced contradictory results [25, 26]. Overall an inversely proportional relationship between overweight-mild obesity and mortality was observed, but these patients had an increased risk of infection, multiple organ failure with longer overall stay in the ICU. Most of the studies were heterogeneous and the interpretation of the results should be considered with caution since obesity was defined only on the basis of BMI, an imperfect measure.

1.3 The Obesity Paradox Revised

Several factors must be considered, since it is quite difficult to explain why after surgical stress, overweight and mildly obese patients seem to show a better prognosis in comparison to the normal-weight patients.

- ***BMI*** The definition of obesity using the BMI alone is misleading and incomplete [1]. It does not distinguish if the increase of the fat is peripheral or central, visceral or subcutaneous. We know that adipose tissue is not only an energetic reserve useful during periods of food deprivation, but it forms the diffuse endocrine system. Visceral and subcutaneous adipose tissue possesses different patterns of hormone secretion and regulates specific metabolic pathways. The increase of visceral fat has a higher pathogenic potential than the subcutaneous adipose tissue. Prognosis of obese patients with heart failure had a good linear relationship with overall survival and waist circumference (considered an index of central adiposity) but not BMI [27, 28]. The same observation was true for surgical patients [29].
- ***Inflammatory pattern of obesity*** Obesity is not only a metabolic but also an attenuated inflammatory disease [30]. Adipose tissue secretes TNFα and other

cytokines, which mediate inflammatory cell activation causing endothelial cell dysfunction. The inflammatory pathways elicited by obesity are the same as induced by surgical stress and it may be possible that obese patients have an adaptive immune-protection exerted by an attenuated inflammation against an acute stress. Mullen et al. [8] considered that the nutritional reserve and more efficient metabolic state of obese patients would be able to elicit a more appropriate inflammatory (and immune) response to surgical stress.

- ***Patient selection*** The main results reported in observational studies in the literature [8, 10, 11, 13, 20] showed that obese patients (especially morbidly obese) were younger than the control population. We suspect that obese patients were selected for intervention only when they were young and at low risk of mortality.
- ***Heterogeneity of the obese population*** Obese patients are a heterogeneous population and up to 30% are metabolically healthy (MHO) with normal insulin sensitivity, low visceral fat storage and absence of significant angiopathy [31]. Intra- and postoperative risks are similar to those of normal-weight patients. MHO patients have normal mean arterial blood pressure, C-reactive protein, HDL cholesterol, triglycerides and plasma glucose. The risk of developing type 2 diabetes mellitus (T2DM) and CVD is 1.24 times higher than in the normal-weight population [32], since these patients easily progress to metabolically unhealthy obesity (MUO), in particular metabolic syndrome; gender (female), low HDL-cholesterol levels, greater insulin resistance and more visceral (and abdominal) fat are the prognostic predictors [33]. It is noteworthy that 30-day morbidity following colon resection in colorectal cancer patients can be predicted by visceral fat not BMI [29]. Furthermore, the presence of the metabolic syndrome, including central obesity, entails a higher risk of respiratory events (OR 2.6) and SSI (OR 3.47) following surgery [34]. In the above mentioned study, mortality was not analyzed, and conclusions cannot be made on the influence of central adiposity on the obesity paradox.
- ***Presence of comorbidities*** Table 1.1 illustrates potential risk factors for complications and death after surgery and trauma in obese patients.

Obesity leads to an increase in body mass by augmenting adipose tissue and ectopic fat accumulation in the liver, muscles and other organs. This modification causes morphological, metabolic and functional changes in a unique pattern for each obese patient.

The most frequent complications of postoperative and traumatic stress are repercussions on the cardiovascular and pulmonary system: cardiac failure, pulmonary

Table 1.1 Comorbidities of obesity relevant for surgical risk definition

Blood hypertension
Cardiovascular disease
Restrictive pneumopathy
Obstructive sleep apnea syndrome
Obesity hypoventilation syndrome
Type 2 diabetes mellitus
Nonalcoholic fatty liver disease
Chronic kidney disease
Malnutrition
Sarcopenia

insufficiency, deep venous thrombosis and pulmonary embolism. They are the most frequent causes of death after bariatric surgery [35].

Arterial hypertension, CVD and obesity hypoventilation syndrome should be considered in the assessment of the surgical risk [36–38]. Obstructive apnea syndrome contributes to worsening of cardiovascular and pulmonary function [39]. The use of the STOP-bang questionnaire is a useful prognostic tool to evaluate the risk of ventilation-related complications [40]. T2DM increases the risk of complications after surgery [41, 42]. Obesity and T2DM independently increase the risk of SSI [43–45], but perioperative correction of hyperglycemia is an effective preventive measure [46, 47]. Malnutrition (detected by hypoalbuminemia) and sarcopenia (detected on the basis of functional and morphological changes in muscle mass) also indicate a higher risk of complications after surgery [48–50].

1.4 Risk Prediction in Emergency Surgery of Obese Patients

Elective bariatric surgery is associated with a low risk of complications (2–5%) and mortality (0.18%) [51]. After elective general surgery, the rate of complications in obese patients ranges from 10.8 to 13.8% with a mortality rate of about 1.2%. Following trauma, they are 9.3% and 3%, respectively. Emergency abdominal surgery is characterized by a substantial increase of the complications (17.2–27.7%) and mortality (3.8–4.9%), depending on the obesity class. The excellent results of bariatric surgery depend on a careful selection of the patients, preventive measures for enhanced recovery after surgery and intensive treatment of complications. ASA physical status is the most used system for predicting the risk of surgical patients [52]. Obese patients are classified as class 2 or 3 with substantial underevaluation of the emergency surgery cases. In bariatric surgery, obese patient prognostic factors include male gender, age >45, BMI > 50, hypertension and risk factors for pulmonary embolism [53]. DeMaria elaborated the Obese Surgery Risk Mortality Score (OSRMS) [53] following bariatric surgery by evaluating the prognostic factors stated above scored 1 each. He considered 3 classes: A (score 0–1), 39–65% of the cases, B (2–3), 35–52% of the cases and C (4–5), 2–11% of the cases [52–55], which correlate with progressive mortality rates of 0.31%, 1.90% and 7.56% [52]. The most frequent causes of death were pulmonary failure, pulmonary embolism and cardiac events (60.6%). The OSRMS was validated for mortality in several studies but failed to predict the risk of complications [53–56] and was not validated for general elective or emergency surgery.

A further predictive factor was identified in a multi-centric prospective cohort study by the StarSurg group [57], which found a significant relationship between BMI and major complications in patients affected by gastrointestinal malignancies. All obese patients affected by these cancers are at a high risk of complications and mortality, especially when associated sarcopenia is present.

Finally, we have to consider the role of emergency surgery. In their revision of the ACS-NSQIP, Hyder et al. [58] examined 56,942 emergency and 136,311 elective interventions and found that the mortality rate was 3.97% in the first group and 0.4% in the latter. In a separate paper, Bohen et al. [59] confirmed that major

morbidity and mortality were higher following emergency surgery (16.75% vs. 9.73% and 3.74% vs. 1%, $p < 0.001$). Bohen et al. [59] and Nandan et al. [60] identified 22 risk factors for adverse events after emergency surgery and elaborated the Emergency Surgery Acuity Score. The mortality rate was 22.8% for score 10, 59.1% for score 15 and 100% for score 22. The risk factors were demographic (age > 60 years), clinical or determined by laboratory tests. Most of them are frequently associated with obesity (hypertension, dyspnea, ventilator requirement, congestive heart failure, infection and sepsis), with longer stay in hospital, higher rate of complications, reoperation and death [61]. Under these conditions, optimal resuscitation and perioperative care strategies (see Chaps. 2–5) are essential to achieve the best results we can.

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Emergency Anesthesia and Resuscitation in the Obese Patient

2

Rita Cataldo, Ida Di Giacinto, Massimiliano Sorbello, and Flavia Petrini

2.1 Pathophysiological Peculiarities of Anesthesiological Interest in Obese Patients: Preoperative Assessment in Emergency

Obesity is considered an important risk factor for cardiovascular disease, type 2 diabetes mellitus, dyslipidemia, hypertension and respiratory diseases affecting especially pulmonary function. Body mass index (BMI) by itself, though included in the new ASA classification [1], does not provide information about adipose tissue distribution and function, key factors in the onset of comorbidities.

2.1.1 Respiratory Function

One of the causes of respiratory impairment in obese patients is elevation of the diaphragm by the abdominal fat. This results in increased respiratory work related

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to the decreased compliance and higher resistance of the airways: an obese patient uses about 15% of his oxygen reserve to breathe as compared to 3% in a lean patient. Obese patients have reduced functional residual capacity (FRC) and therefore a tendency to rapidly develop atelectasis with alteration of the ventilation/perfusion (V/Q) ratio, hypercapnia, increased expiratory resistance, wheezing, tachypnea and, most importantly, rapid desaturation.

These problems are often exacerbated in emergency and cause the respiratory system of the obese to work at its maximum even under standard conditions, with no reserve in the event of a critical situation. The reduced reserve compromises the patient's ability to tolerate respiratory insults such as pneumonia. Furthermore, an obese person has increased airway pressure due to increased airway resistance (heavier chest wall and abdomen, lung base atelectasis). All of these will lead to a reduced oxygen supply, increased respiratory work, and a very short desaturation time at the induction of anesthesia with a short safe apnea time.

2.1.2 Cardiovascular Function

Obesity and the related metabolic syndrome also interfere negatively with the patient's cardiovascular function. The metabolic syndrome makes the patient more susceptible to risk factors for ischemic heart disease. Furthermore, obesity is an inflammatory syndrome that also involves the endothelium, placing the arteries at greater risk of atheromas. Finally, the additional work needed to perfuse the increased body surface can result in progressive ventricular dysfunction. The risk of heart failure is elevated in obese patients, and its genesis is complex and often multifactorial. Ventricular hypertrophy and hypertension lead to a higher systolic workload, which is associated with an increase in circulating blood to perfuse the adipose tissue. As soon as the system gets "out of breath", left ventricular dysfunction sets in, associated with cardiovascular decompensation and heart failure. The obese patient is often affected by coronary disorders and arrhythmias such as atrial fibrillation, which worsen cardiac function especially in emergency conditions (altered volemia, pain, respiratory failure and hypoxia) where preoperative optimization is desirable but impossible.

The pulmonary and cardiovascular systems are so closely connected that alterations in their functioning reinforce each other. In the obese patient, the clinical presentation of "obesity cardiomyopathy" can be exacerbated by the pulmonary hypertension commonly associated with obstructive sleep apnea (OSA) and obesity hypoventilation syndrome (OHS) (sometimes combined in an overlap syndrome) and right ventricular dysfunction.

In patients with specific problems (BMI >50, overlap syndrome, obesity cardiomyopathy), rapid echocardiographic assessment is recommended if time permits, even directly on the operating table or in the emergency department.

2.1.3 Risk of Thromboembolism

Because it induces an inflammatory state, obesity is a risk factor for deep venous thrombosis (DVT) and pulmonary embolism (PE). The risk is increased by trauma, emergency abdominal and pelvic surgery, and hospitalization in the intensive care unit (ICU). Prophylaxis against DVT/PE is mandatory and the use of pharmacological strategies must not exclude active mechanical pressure, which can be started in the operating room (OR). Every therapeutic effort must be made towards early postoperative mobilization. A vena cava filter is not routinely recommended for primary prophylaxis.

2.1.4 Risk of Difficult Airway

Obese patients are also at risk of a difficult airway. Although this does not necessarily mean difficult intubation, problems can arise in the perioperative maintenance of adequate oxygenation. Obese patients may not have an increased risk of difficult intubation *per se* but are likely to present difficulties in face mask and supraglottic airway device (SAD) ventilation in linear relation to their BMI. In the case of a "can't intubate, can't oxygenate" (CICO) scenario, the obese patient's anatomy involves an increased risk of difficult cricothyroidotomy because of problems with landmark identification and pretracheal tissue thickness. Therefore, in emergency surgery even more than elective surgery, it is essential to be prepared for a difficult airway and explore and identify predicting indexes.

Standard difficult airway indexes can be easily and quickly assessed even in an emergency setting if the patient is conscious and cooperative (Mallampati score, neck extension, thyromental distance, interincisor distance, presence or absence of teeth or fixed dentures). In addition, there are specific scores such as neck circumference and waist-to-hip ratio (WHR). Neck circumference should be measured (raising an alert above 41 and 43 cm in female and male patients, respectively) or indirectly assessed (shirt collar size), whereas patients with android (apple-shaped) or gynoid (pear-shaped) obesity can be easily identified. Whenever possible, a STOP-Bang questionnaire is recommended: a score ≥ 5 is highly suggestive of severe OSA, resulting in a higher risk of difficult ventilation and intubation. Especially in such cases (obese patient with metabolic syndrome, high OSA risk, android obesity, increased neck circumference), preparing an adequate airway management strategy (including spontaneous breathing techniques) is of paramount importance. Adequate preoxygenation aiming for an EtO_2 >90% with the patient in the ramped position is always imperative; depending on the clinical setting and the time available, positive-pressure ventilation by face mask or a high-flow nasal cannula can be used, taking into account that positive pressure oxygenation is the gold standard in obese patients. Apneic oxygenation during airway instrumentation (the so-called NO-DESAT technique) [2, 3], preferably with a high-flow nasal cannula, should be considered.

2.2 Intraoperative Care

The intraoperative management of the obese patient in an emergency setting presents organizational as well as professional challenges. The skills of the health providers (nurses, anesthesiologists, surgeons) must be complemented by adequate and readily available instrumentation. This means that hospitals must develop appropriate protocols and checklists specifically for the management of obese patients and strongly promote teamwork strategies.

Adequate devices such as surgical tables, stretchers, positioning devices, noninvasive blood pressure (NIBP) cuffs and locoregional anesthesia (LRA) needles are indispensable. Also, compression devices for intraoperative DVT prophylaxis should be adjusted to the size and volume of the obese patient. Computed tomography (CT) and magnetic resonance imaging (MRI) scanners have to be suitable for extra-large patients and an ultrasound (US) scanner must always be available at the bedside.

Venipuncture can be challenging in an emergency; in highly critical situations it may be wise to cannulate a femoral vein with the patient in the "frog-leg position". Access to central veins requires the use of US, as does locating the deep veins of the arm for placement of a peripherally inserted central catheter or midline catheter if easier peripheral approaches are unfeasible.

2.3 Airway Management

2.3.1 Rapid Sequence Induction/Intubation

Rapid sequence induction/intubation (RSII) is not expressly indicated in obese patients but follows the same indications as in the non-obese (full stomach, symptomatic esophageal reflux, pregnancy, diabetes with gastroparesis). Any previous bariatric surgery should be added to the list of indications, as restrictive and malabsorptive techniques favor the reflux or stagnation of fluids in the stomach. The so-called modified RSI (mRSI) includes hemodynamic optimization and abolishes succinylcholine in favor of rocuronium at a dose of 1–1.3 mg/kg calculated on lean body weight (LBW) with neuromuscular block monitoring; Sellick maneuver should be abandoned (if not in presence of clear regurgitation) and ventilating the patient by means of a face mask with low insufflation pressure after adequate preoxygenation up to ETO_2 >90% in the ramped and reverse Trendelenburg position is accepted. Use of an LBW-based propofol bolus in emergency conditions, even if titrated, may result in cardiovascular impairment, which must be prevented by fluid filling (if possible) and/or administration of vasoactive agents. Ketamine, especially in emergencies with cardiovascular impairment, can be a useful clinical alternative.

2.3.2 Strategies

Because the obese patient is at high risk of rapid desaturation, a robust strategy for airway management is essential [4]:

1. use a peri-oxygenation approach, including preoxygenation and apneic oxygenation with a nasal cannula during airway instrumentation (NO-DESAT);
2. decide whether or not to abolish spontaneous breathing and perform awake fiberoptic or videolaryngoscopic intubation;
3. if deciding to abolish spontaneous breathing, consider mRSI;
4. apply "first-pass-success" tracheal intubation, which means that, to maximize the chances of success, the first attempt must be made in the best possible conditions (ramped position; highly experienced anesthetist; use of a videolaryngoscope if present and if the operator is skilled, or a standard laryngoscope with the introducer or stylet premounted on the tracheal tube);
5. in case of intubation failure, switch quickly to SAD or return to mask ventilation;
6. consider cricothyroidotomy regardless of SpO_2 values if it is clear that other strategies are unfeasible. Identify the cricothyroid membrane possibly with the patient awake; a quick US examination before induction might be helpful.

A strategy for safe airway management is incomplete without a plan for safe extubation, whether this is done in the OR or after the patient has been transferred to the post-anesthesia care unit (PACU) or ICU for postoperative care. Extubation, especially in obese patients and even more in the presence of OSA or perioperative respiratory distress, is a high-risk situation because of either the lessened level of attention or unpreparedness. Reversing the neuromuscular block to a TOF ratio $\geq$0.9, preferably 1, is mandatory. The patient has to be extubated in semi-sitting position fully awake, cooperative and preoxygenated with 100% O_2; use of an airway exchange catheter is advisable if difficult extubation is expected. Adequate and tailored post-extubation respiratory monitoring should be considered in selected patients.

2.4 Drugs in the Emergency Setting

Because of the peculiar pharmacodynamics and pharmacokinetics in obese patients, proper drug dosage is challenging and should be adapted as appropriate to total, ideal, adjusted or lean body weight based on the lipophilic and hydrophilic characteristics of the single molecules. Obese patients may experience awareness during intubation due to the rapid redistribution of the hypnotic, and it is necessary to quickly switch to anesthesia maintenance even in conditions of hemodynamic instability; use of vasoactive agents may be required (Table 2.1).

Choosing short-acting drugs and opioid-sparing strategies, and whenever possible a combination of general anesthesia with regional techniques, should be preferred. Even in an emergency, minimally invasive techniques such as laparoscopy or thoracoscopy, although challenging for operators, result in shorter recovery times after surgery and should be preferred over more invasive procedures.

After induction, it is important to monitor blood glucose levels, which can be altered in critical patients with metabolic syndrome or insulin-dependent diabetes mellitus. Care should also be taken to control body temperature, which can easily drop to critical levels in a patient with a large body surface area. This will

Table 2.1 Drug dosage in obese patients

Drugs	Loading dose	Maintenance dose
Neuromuscular blockers and antagonists		
Succinylcholine	TBW	
Vecuronium	IBW	IBW
Atracurium	LBW	LBW
Rocuronium	LBW	LBW
Sugammadex	TBW	
Neostigmine	ABW	
Sedative-hypnotics		
Benzodiazepine	IBW	IBW
Propofol	LBW	ABW
Thiopental	LBW	IBW
Phenobarbital	TBW	IBW
Ketamine	TBW	IBW
Etomidate	TBW	
Dexmedetomidine		LBW
Analgesics		
Morphine	LBW	
Remifentanil	LBW	
Fentanyl	LBW	
Sufentanil	LBW	
Alfentanil	ABW	
Paracetamol	LBW	
Corticosteroids		
Methylprednisolone	IBW	IBW
Anti-epileptics		
Phenytoin	IBW + [1.33 × (TBW − IBW)]	IBW
Valproic Acid		IBW
Carbamazepine		IBW
β-blockers		
Propranolol	IBW	IBW
Labetalol	IBW	IBW
Metoprolol	IBW	IBW
Esmolol	IBW	IBW
Calcium channel blockers		
Verapamil	TBW	IBW
Diltiazem	TBW	titration
Antiarrhythmics		
Lidocaine	ABW	ABW
Procainamide	IBW	IBW
Amiodarone	IBW	IBW
Digoxin	IBW	IBW
Adenosine	IBW	IBW
Catecholamines		
Dobutamine Dopamine Epinephrine Norepinephrine Phenylephrine Vasopressin Milrinone		There are no clinical studies in the obese patient. According to literature, ABW or IBW could be used to avoid overdoses, titrating the dose as a function of the clinical target ABW in patients ≤120 kg

IBW = *male* h(cm) − 100; *female* h(cm) − 110
LBW = *male* 90 kg; *female* 70 kg
ABW = IBW + 40% TBW
TBW total body weight, *IBW* ideal body weight, *LBW* lean body weight, *ABW* adjusted body weight

compromise outcome, as it increases the risk of postoperative residual curarization, coagulation impairment, dehiscence of anastomosis or surgical wounds, infections and decubitus ulcers.

The obese patient, especially in an emergency setting, is at high risk of postoperative pulmonary complications (PPCs). Their prevention starts with a wise and proper intraoperative management of ventilation. Appropriate levels of PEEP and recruitment maneuvers, if needed, can prevent atelectasis and may help to keep the lungs fully aerated [5].

2.5 Level of Care

The predictive factors for the use of care resources and services in obese patients are poorly defined [6]. More than BMI *per se*, the occurrence and treatment of comorbidities together with the degree of surgical invasiveness determine which level of care is needed. In an emergency, the availability of expert staff and dedicated equipment make it possible to regulate the level of care. Not all obese patients require admission to the ICU. A track-and-trigger system defined through proactive strategies including checklists, handovers and the application of dedicated scores such as National Early Warning Scores (NEWS) allows early recognition of the decline of vital functions in the various care settings [7, 8].

2.6 Non-operating Room Anesthesia (NORA)

Obese patients often undergo emergency interventions; because of the high risks associated with obesity, minimally invasive procedures should be used whenever possible. The patient may be given general anesthesia or procedural sedation in unconventional settings that are often suboptimally equipped, both logistically and in terms of operator skills; this is referred to as non-operating room anesthesia (NORA). To ensure adequate safety conditions, interventional departments where regional anesthesia, moderate or deep sedation and even general anesthesia can be provided must have OR safety standards including cardiovascular and respiratory monitoring, equipment to manage a difficult airway, adequate patient handling systems, and beds that can bear the weight of an obese person [6].

In an emergency setting, identifying a procedure as "minimally invasive" should not be taken to mean "minimally dangerous", especially with regard to anesthesiological procedures, which are sometimes riskier and more difficult to manage than general anesthesia with tracheal intubation in the OR. In fact, the patient may be in critical condition with respiratory failure, OSA or OHS, and may not tolerate the fully supine or prone position necessary for certain procedures, especially if sedative drugs are infused; special care is needed during spontaneous breathing sedation, and $EtCO_2$ monitoring is mandatory. Cardiovascular status may be compromised and require vasoactive support in environments where finding a central vein can be complicated and invasive monitoring sometimes impossible. In such scenarios it is safer to proceed with tracheal intubation and invasive monitoring

before starting the procedure than running the risk of performing these difficult maneuvers in an emergency setting.

The message is to not underestimate NORA but rather to consider the high risks it may involve and prevent them by ensuring that the environments where NORA is performed are adequately equipped and managed. Anesthesiological management should tend towards regional anesthesia when possible and towards conscious sedation with drugs of minor impact on the respiratory drive and hemodynamics. The positive role of dexmedetomidine, now also approved in Europe (and Italy) for use in settings other than the ICU, deserves adequate consideration in this context [9].

2.7 Cardiopulmonary Resuscitation

An inevitable consequence of the increased incidence of obesity worldwide is that more obese patients need resuscitation maneuvers for acute illness or trauma.

Resuscitation in the obese poses numerous challenges, including (1) difficulties in airway management, chest compression and venous access; (2) complicated drug management due to obesity-related pharmacokinetic and pharmacodynamic alterations; and (3) obstacles to instrumental diagnosis [10].

In spite of these difficulties a phenomenon known as "obesity paradox" has been described in the literature, characterized by better neurological outcomes after cardiac arrest and better survival in cases of STEMI, NSTEMI, unstable angina and heart failure in obese compared with normal- or low-weight patients [11–14].

Algorithms for cardiopulmonary resuscitation (CPR) in obese adults are the same as in normal-weight adults, with a sequence of 30 compressions at a frequency of 100–120 beats/min and a depth of 5 cm (equal to one-third of the chest depth) alternating with two ventilations. The effectiveness of CPR is determined by early defibrillation and quality chest compressions, which may not be easy to achieve in an obese patient [15].

An obese body type poses problems that are not accounted for in standard situations. For instance, the anterior and posterior thoracic adipose tissue may redistribute the force applied to the chest during cardiac massage, making the compressions more superficial and thus less effective [16]. When lying supine, the patient's abdominal fat may displace the diaphragm cranially, as also happens in pregnant women. In those cases, cardiac massage to the upper half of the sternum is recommended.

In addition, cardiac massage in an obese patient is much more tiring for the operator, with a risk of ineffective chest compressions. Switching operators at less than the standard 2 min recommended by the guidelines can provide a solution. Mechanical chest compression systems (such as LUCAS) cannot be used because they are not designed for obese body types [17].

Although thoracic fat causes high transthoracic impedance, there are no indications regarding the energy to be delivered by the defibrillator in the obese patient,

and no correlation between BMI and the success rate of defibrillation at first shock has been reported in the literature. The usual practice is to start directly with higher energy levels (200 J), to be increased if found ineffective. The higher transthoracic impedance can be counteracted using modern biphasic defibrillators; rectilinear biphasic waveforms seem to be more useful for this purpose [14].

In the case of choking, a variation of the Heimlich maneuver with chest thrusts to the center of the sternum should be applied [17].

Finding a venous access might be challenging, time consuming, and requiring numerous attempts. It is therefore essential to use US guidance, the gold standard in the identification of peripheral and central venous access according to the current guidelines. Despite the difficulties inherent in the use of US in the case of abundant subcutaneous fat, it increases the chances of success and reduces time and complications even in crisis situations [18].

2.8 Other Considerations

2.8.1 Diagnostic Imaging

When standard X-rays are used, the patient's mass may prevent the entire body region (e.g. chest and abdomen) from being captured in a single scan. In addition, certain images such as a lateral view of the cervical spine may be difficult to interpret.

CT and MRI examination may be complicated by problems related to the opening diameter of the machine, the weight limit of the table and the limited field of view. This can result in the need for multiple scans, with increased radiation exposure and a risk of motion artifacts.

US has become the standard of care in emergency diagnostics. However, performing an ultrasound examination in an obese patient is technically complex due to the hypoechogenicity of the adipose tissue and the distance between the skin and the target organ. This makes it necessary to use probes with a frequency of 2 MHz, which allow a greater depth to be reached albeit with lower spatial resolution [19].

2.8.2 Patient Transport

Transporting an obese patient can be extremely difficult. Depending on the patient's build and the environmental circumstances, many people may be needed to lift and transfer the patient to and from the ambulance. There are special stretchers and sheets for the transport of heavy weights on the market. Personnel must be adequately trained, and the hospital must have the means and equipment to accommodate and transport severely obese patients.

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3 Perioperative and Intensive Care Management of the Obese Surgical Patient

Giulia Bonatti, Chiara Robba, Lorenzo Ball, and Paolo Pelosi

3.1 Introduction

The number of overweight patients undergoing major surgical procedures and subsequently admitted to the intensive care unit (ICU) is dramatically increasing [1]. Since these patients are affected by several systemic pathophysiological alterations and comorbidities (Table 3.1), their management in both the operating room and ICU may present several challenges for the clinicians.

Major surgery, general anesthesia and mechanical ventilation (MV) can *per se* contribute to the development of lung and systemic organ failure. However, a reliable tool to assess the perioperative and ICU risk for obese patients has not been yet defined [2].

3.2 Perioperative Management of the Obese Patient

3.2.1 Preoperative Evaluation

Preoperative evaluation of obese patients should take into consideration the assessment of the patient's baseline functional state and comorbidities as well as the complexity of surgery. The vast majority of obese patients undergoing surgery are relatively healthy and their risk is similar to that of normal weight patients [3].

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Table 3.1 More frequent systemic pathophysiological alterations in the obese patient

Respiratory system
↑ Oro-pharyngeal adiposity → upper airway obstruction
↓ Compliance ($C_{chest\ wall} > C_{lung}$) → ↓ compliance during MV
↑ Chest wall elastance
↑ Total respiratory resistance (>during sleep)
↑ Work of breathing
↓ FRC and EELV → atelectasis
Small airway collapse → auto-PEEP, ↑ risk of wheezing
V/Q mismatch → atelectasis
↑ O_2 consumption → ↓ O_2 reserve
↑ Production and ↓ excretion rate of CO_2
↑ Risk of airway hyper-reactivity, OSA, OHS, overlap syndrome
Endocrinological system
↑ Insulin-resistance
↓ Glucose tolerance
Metabolic syndrome
Cardiovascular system
↑ Blood pressure, cardiac output, cardiac workload
Left ventricular hypertrophy → ↑ diastolic and systolic dysfunction
↑ Risk of arrhythmias (> atrial fibrillation)
↑ QT interval
↑ Risk of ischemic heart disease and heart failure
↑ Pulmonary artery pressure → pulmonary hypertension
↓ Right ventricular ejection fraction → cor pulmonale
Prothrombotic state → ↑ risk of myocardial infarction, stroke, VTE
Other
Functional and anatomical hiatus hernia
Altered lipid metabolism
↓ Micronutrients
↓ Gut motility
↑ Risk of visceral fat (>♂)
Drug-specific changes in volume of distribution

$C_{chest\ wall}$ chest wall compliance, C_{lung} lung compliance, *FRC* functional residual capacity, *EELV* end-expiratory lung volume, *PEEP* positive end expiratory pressure, *V/Q* ventilation/perfusion, *OSA* obstructive sleep apnea, *OHS* obesity hypoventilation syndrome

However, several risk factors in obese patients have been correlated to an increased incidence of perioperative complications and mortality, including: central obesity, metabolic syndrome [1], hypertension, BMI >50 kg/m^2, male sex, age >45 years, risk factors for pulmonary embolism, overlap syndrome, obesity hypoventilation syndrome (OHS) and poor compliance to continuous positive airway pressure (CPAP) [4].

Difficult intubation and mask ventilation occur more frequently in obese compared to non-obese patients, and a thorough assessment of these risks should be performed. Risk factors for difficult ventilation include increasing BMI, age above 50 years and history of snoring. The main predictors for difficult intubation are almost the same as for the non-obese patients; other specific factors include neck circumference, the severity of obstructive sleep apnea (OSA) and OHS, pre-tracheal soft tissue thickness and high BMI [5, 6].

Table 3.2 Preoperative evaluation in the obese patient

Basic exams	Advanced exams
Respiratory assessment	
Exclusion of OSA and OHS (snoring, apneic episodes, frequent night arousals) Cephalometric measurements SpO_2 Arterial blood gas analysis (lactate, PaO_2, HCO_3^-)	Chest radiography STOP-Bang questionnaire Apnea hypopnea index High-resolution nocturnal oximeter Polysomnography Spirometry PAP therapy titration
Cardiovascular assessment	
Signs of heart failure (ankle edema, high jugular venous pressure) Blood pressure Blood sample (full blood count, hemostasis tests, etc.) Electrocardiogram	Troponins and BNP Echocardiogram Exercise tolerance
Endocrinological assessment	
Strict glycemic control BMI Waist/hip ratio	HbA1c
Renal assessment	
Creatinine, electrolytes, urea	BUN/creatinine ratio, GFR Urine tests

OSA obstructive sleep apnea, *OHS* obesity hypoventilation syndrome, *SpO_2* saturation of peripheral oxygen, *PaO_2* arterial oxygen partial pressure, *HCO_3^-* hydrogencarbonate ion, *PAP* positive airway pressure, *BNP* B-type natriuretic peptide, *HbA1c* glycated hemoglobin, *BMI* body mass index, *BUN* blood urea nitrogen, *GFR* glomerular filtration rate

OSA is frequently undiagnosed (and untreated) until an acute-on-chronic respiratory failure occurs, which during the perioperative period can be exacerbated by the administration of sedatives, opioids and prolonged supine position [7, 8]. Preoperative identification of high-risk patients for respiratory complications is crucial as these patients can benefit from preoperative positive airway pressure (PAP) therapy and eventually ICU admission [6, 8].

The main key points of preoperative evaluations in obese patients are summarized in Table 3.2 [3, 6]. In the case of emergency surgery, the previous described assessments are not always feasible; therefore, the preoperative risk should be evaluated mainly through a quick clinical examination, and arterial blood gas samples to assess blood gas exchanges and serum lactate [3].

3.2.2 Intraoperative Management

3.2.2.1 Regional Anesthesia

Regional anesthesia presents several advantages, especially in obese patients, including minimal airway manipulation, avoidance of cardiopulmonary depression due to anesthetic drugs, reduced opioid requirements and postoperative nausea and vomiting. Unfortunately, loco-regional procedures in obese patients are often

Table 3.3 Intraoperative management of the obese patient: key points

Induction of anesthesia
Consider the use of loco-regional anesthesia
For general anesthesia, better easily reversible and short acting anesthetic drugs
Anesthetic and neuromuscular blocking agents dose-titrated on effect
Prolonged pre-oxygenation (FiO_2 up to 100%, if necessary)
Ramped position or 30° reverse Trendelenburg position
nPAP support, in selected patients
Ready availability of difficult airway management devices
Awake intubation, in selected patients
Supraglottic device as rescue in difficult ventilation or intubation or first line in selected patients
Maintenance of anesthesia
No evidence regarding the best anesthetic strategy to use (propofol versus volatile agents)
Continuous monitoring of sedative and neuromuscular blockade effects
Faster onset and offset of desflurane or sevoflurane compared to isoflurane
Protective ventilation with ↓ Vt (6–8 mL/kg Predicted or Ideal Body Weight), ↓ P_{plat} (<24 cmH_2O), ↓ P_{driv} (<16 cmH_2O)
Use of PEEP and RMs to improve intraoperative oxygenation and compliance
Lowest FiO_2 ensuring satisfactory oxygenation (SpO_2 92–95%)
No data suggesting intraoperative superiority among the different ventilation modes
Emergence from anesthesia
Nerve stimulator to guide neuromuscular blockade reversal
Patient fully awake, with restored airway reflexes
Reverse Trendelenburg position or ramped position, if possible
Immediate post-anesthesia care
Head-up or sitting position
Intensive physiotherapy and incentive spirometry
Early mobilization
Careful fluid management
Opioid-sparing analgesia, if possible
Oxygen therapy to maintain preoperative levels of SpO_2
Early nPAP support, in selected patients (airway pressures <20 cmH_2O)
Extended postoperative prophylaxis for VTE, in selected patients
Frequent glycemic monitoring and delayed reintroduction of diabetic drugs, if necessary
Intensive care support based on comorbidities and surgery

FiO_2 fraction of inspired oxygen, n*PAP* non-invasive positive airway pressure, *Vt* tidal volume, *P_{plat}* plateau pressure, *P_{driv}* driving pressure, *PEEP* positive end expiratory pressure, *RMs* recruitment maneuvers, *SpO_2* saturation of peripheral oxygen, *VTE* venous thromboembolism

technically challenging and may be ineffective and therefore a plan for airway management and intubation is always recommended [6].

3.2.2.2 General Anesthesia

Each anesthesiological step can be potentially more difficult to perform in obese patients compared to the general population (Table 3.3).

Induction of Anesthesia

Ideal or adjusted body weight is used to calculate initial anesthetic drug doses rather than total body weight. Current kinetic models use to titrate anesthetic agents to site

effect that can generate paradoxical concentrations in morbidly obese patients. Therefore, the monitoring of the depth of anesthesia and of the neuromuscular block should be always considered. Caution is required with the use of long-acting drugs, especially opioids [3].

Quick and profound episodes of desaturation are common in obese patients; therefore, prolonged pre-oxygenation in ramped position is suggested to maximize the intrapulmonary oxygen reserve and to increase the safety period of apnea between induction and intubation [2].

In morbidly obese patients or patients with severe OSA, airway obstruction, hypoxemia or acute respiratory failure (ARF), pre-induction with high-flow oxygen with nasal cannula (HFNC) or PAP therapy should be considered [8].

Intubation should always be considered potentially at risk in obese patients and devices for difficult intubation should always be easily available; in particular, videolaryngoscopes have shown to be useful also in obese patients [2].

Supraglottic devices are increasingly used as rescue ventilatory, mainly in difficult ventilation or intubation. The use of supraglottic airway devices as a first line device should be reserved only for highly selected patients undergoing short procedures, when the upper airway is accessible and tracheal intubation quickly feasible [3].

Maintenance of Anesthesia

In obese patients, no evidence is available regarding the best anesthetic strategy to use. Achieving appropriate oxygenation and carbon dioxide levels as well as the choice of the respiratory settings to apply to obese patients during surgery can be challenging because of the previously described respiratory pitfalls [2, 9].

Several ventilatory strategies have been suggested to improve the perioperative outcome of obese patients; lung protective ventilation strategy with low tidal volume (Vt) and the use of positive end expiratory pressure (PEEP) should be considered in the operating room. The use of protective Vt is warranted to avoid high plateau pressure (P_{plat}) and high driving pressure (P_{driv}), even if this can necessitate an increase of the respiratory rate to optimize carbon dioxide levels [10].

The appropriate intraoperative level of PEEP and its effect on postoperative outcome is controversial. Many authors are focusing their research on identifying strategies to set individualized PEEP ($PEEP_{ind}$); however, nowadays the hypothesis that $PEEP_{ind}$ could produce better outcomes has still to be proven. Furthermore, some authors demonstrated that $PEEP_{ind}$ could be significantly higher compared to the routinely used PEEP during anesthesia [11], thus posing the patient at risk for PEEP-related hemodynamic effects [12]. Intraoperative recruitment maneuvers (RMs) can have an important role in atelectasis reduction [13]. It is not clear which is the most efficient RM mode in preventing pulmonary complications [14]; however, it is well known that bag squeezing presents several pitfalls that have to discourage its use in favor of RMs consisting of stepwise transient changes of ventilator settings [15].

In obese patients, there are no data suggesting the superiority in terms of outcome between the different controlled ventilation modes [14]. Pressure support ventilation might be the most beneficial ventilatory mode, as it preserves muscular tone and prevents posterior-basilar atelectasis [2], but it is not often feasible in the intraoperative settings.

Emergence from Anesthesia

During the extubation phase in obese patients, a large number of complications are described. Therefore, an extubation plan should be always put in place (Table 3.3) [16].

3.2.3 Postoperative Management

Obesity predisposes to several postoperative complications, mainly involving the respiratory system [17]. To decrease the risk of complications, there are several postoperative strategies that could be adopted (Table 3.3) [7, 17, 18]. Several studies encourage the use of postoperative PAP therapy in obese patients to improve postoperative outcome [4], mainly in subjects with OHS and/or OSA [2, 8]. To date, there is no evidence supporting the use of a specific patient interface device and ventilation modality in obese patients. Intolerance is reported as a PAP treatment-related complication, while anastomotic leakage does not seem to be connected to the insufflation of PAP [19]. More studies about HFNC are needed before recommending this strategy in the post-extubation phase [2].

Obese patients without major medical comorbidities are managed in the standard post-anesthesia care unit [20]. Indications to ICU admission could be: BMI $\geq$ 50 kg/m^2, long-acting opioid treatment, OSA or OHS and/or PAP therapy requirements, need for respiratory and cardiac monitoring, difficult glycemic control, intraoperative surgical or anesthetic complications and emergency surgery [3].

3.3 ICU Management of the Obese Surgical Patient

Critically ill obese patients may be at higher risk for acute cardiovascular, pulmonary and renal complications in comparison to healthy-weight patients [18]. Furthermore, obesity is associated with an increased risk of morbidity and death in the general population, but a decrease in mortality has been reported by some authors in patients with septic shock and acute respiratory distress syndrome (ARDS) (obesity paradox). The actual existence and basis for this apparent paradox are still debated [21]. In Table 3.4, the complications and corresponding management of obese patients admitted to ICU post-surgery are summarized [18, 20].

Table 3.4 Main ICU complications of the obese surgical patient and suggestions for reducing the risk of complications

Complications	Suggestions
Respiratory system	
Post-extubation stridor	Sitting/ramping position, post-extubation PAP therapy
Extubation failure	Fully awake patients, opioid-sparing analgesia, sitting/ramped position, PAP therapy, tracheotomy
Ventilator-associated pneumonia, ARDS	Individual-tailored ventilator strategies, lung protective ventilation, post-extubation PAP therapy, light sedation, physiotherapy, prone position, nitric oxide, high-frequency percussive ventilation, jet ventilation, and veno-venous extracorporeal membrane ventilation.
Catheter-related pneumothorax	Ultrasound-guided insertion
Cardiovascular system	
Acute congestive heart failure	Careful fluid management and adequate control of hemodynamic parameters
Myocardial infarction	Close monitoring
Atrial fibrillation	Continuous electrocardiogram-monitoring
Acute cor pulmonale	Adequate respiratory treatment
Thromboembolic diseases	Extended postoperative mechanical and pharmacologic prophylaxis for VTE
Endocrinological system and nutritional state	
Protein malnutrition	Isocaloric high protein diet
↓ Gut motility	Early enteral feeding
Hyperglycemia	Glycemic control, adequate insulin therapy, isocaloric high protein diet
Hypoglycemia	Delay in diabetic drugs reintroduction
Others	
CKD and AKI	Monitoring of renal function
Non-alcoholic steatohepatitis	Monitoring of hepatic function
Fluid retention	Isocaloric high protein diet
Acquired infections	Adequate antibiotic therapy
Catheter-related infections	Early change of catheters
Urinary tract infections	Frequent inspection of urinary catheters, routine urinary exams
Wound healing/skin necrosis	Wound management; hypoperfusion, hypoxia and hyperglycemia therapy
Decubitus ulcers	Specific mattresses, adequate protein intake and early mobilization
Neural injuries	Special beds and lifting devices, padding of pressure points
Multiple organ failure	Tailored monitoring and treatment

ICU intensive care unit, *ARDS* acute respiratory distress syndrome, *PAP* positive airway pressure, *VTE* venous thromboembolism, *CKD* chronic kidney disease, *AKI* acute kidney injury

3.4 Conclusions

Obese patients present several challenges in the perioperative period and in the ICU. Obese patients present a bundle of pathophysiologic changes, with consequent pulmonary and cardiovascular issues, which make them susceptible to several

complications. Future studies are warranted to better define the optimal settings of invasive mechanical ventilation, weaning protocols, hemodynamic monitoring and other specific strategies in this cohort of patients.

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Postoperative Complications in the Intensive Care Unit

4

Michele Carron

4.1 Introduction

The incidence and prevalence of obesity have increased worldwide. As a major factor contributing to many chronic diseases and cancer, obesity is a global health concern. At a body mass index (BMI) of 40–45 kg/m^2, life expectancy is reduced by 8–10 years [1].

Obesity increases the risk of perioperative complications [2–4]. With increasing BMI, the likelihood of any complication (odds ratio [OR] = 1.19), and especially postoperative pulmonary complications (PPCs) (OR = 1.35), is increased [2]. Untreated obstructive sleep apnea (OSA) is an independent risk factor for adverse events after bariatric surgery, including desaturation (OR = 2.27), acute respiratory failure (ARF; OR = 2.43), and cardiac events (myocardial infarction, cardiac arrest, arrhythmias) (OR = 2.07) [3]. Compared to normal-weight patients, obese patients with metabolic syndrome (MS) have a 1.6- to 2.3-fold higher odds of central nervous system adverse events, 1.5- to 2.8-fold higher likelihood of pulmonary adverse events, 1.7- to 2.7-fold higher odds of cardiac adverse events, and 3.3- to 7.3-fold higher odds of acute kidney injury (AKI) in perioperative period [4].

Obesity is also a risk factor for postoperative intensive care unit (ICU) admission, especially when accompanied by OSA (OR = 2.81) [3]. ICU admission negatively impacts outcomes [1, 3]. All-cause long-term mortality after bariatric surgery is higher in patients requiring ICU admission than in those who do not (6.2 vs. 0.2 deaths/1000 patient-years, $p < 0.001$) [5]. However, whether obesity increases the risk of death within the ICU is unclear. Bercault et al. [6] and Nasraway et al. [7] reported significant associations between morbid obesity and ICU mortality

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(OR = 2.1 and 7.4, respectively), whereas a recent meta-analysis found no association between obesity and ICU mortality [8]. A U-shaped association between obesity and ICU mortality was observed, with excess mortality in underweight and severely obese patients [8]. This explains the "obesity paradox", an unexpected inverse association between obesity and mortality [1].

Preventing or recognizing postoperative complications is important to minimize negative outcomes in the ICU in obese patients.

4.2 ICU Admissions After Bariatric Surgery

The percentage of ICU admissions after bariatric surgery is quite low: approximately 4.9% of patients in a population-based multicenter study [5]. In this study, ICU admission was more common after open or revision bariatric surgery [5]. Anesthetic (8.8%) and medical (2.2%) complications accounted for small proportions of the primary reasons for ICU admission [5]. Unplanned ICU admissions were less common than elective admissions, occurring in approximately 1.5% of patients [5]. Revision surgery (OR = 13.08), open surgery (OR = 4.18), diabetes mellitus (OR = 2.10), OSA (OR = 4.31), chronic respiratory disease (OR = 6.95), and chronic renal disease (OR = 2.92) were factors most strongly associated with unplanned ICU admission [5]. Gastrointestinal tract surgical leak, abscess, or both were factors strongly associated with multiple ICU re-admissions (OR = 7.46) [5]. Revision (OR = 4.76) and open (OR = 2.78) surgeries were predictors of surgical, but not anesthetic or medical, complications in the ICU post–bariatric surgery [5].

4.3 Surgical Complications and ICU Admission After Bariatric Surgery

Among surgical complications, bleeding, peritonitis, and surgical site infections (SSIs) may occur postoperatively and require ICU admission [9, 10]. The incidence of postoperative bleeding is approximately 0.1% after laparoscopic adjustable gastric banding (LAGB), 1–6% after laparoscopic sleeve gastrectomy (LSG), and 0.4–4% after laparoscopic Roux-en-Y gastric bypass (LRYGB) [10]. Diagnosis is based on physical findings (hematemesis, melena, tachycardia, hypotension, drainage tube output) or a fall in hemoglobin [9, 10]. In any patient with persistent tachycardia (>100 beats/min), low systolic blood pressure (<100 mmHg), transfusion requirement >2 U, melena, or hematemesis, early intervention is important to prevent hemorrhagic shock (endoscopic intervention for intraluminal bleeding; surgery for extraluminal bleeding) [9, 10].

The incidence of peritonitis secondary to anastomotic leak or fistula is 0.5–0.8% after LAGB, 0–7% after LSG, and 0–6.1% after LRYGB [10]. As classic peritonitis signs may be absent in obese patients, clinicians should be aware of non-specific signs, such as fever (>38 °C), dyspnea, and tachycardia, which have been reported in 74%, 98%, and 100% of patients with intra-abdominal sepsis after bariatric

surgery, respectively [11]. Respiratory distress (OR = 23.2) and severe tachycardia (OR = 6.0) are independent predictors of anastomotic leak after bariatric surgery [12]. With sepsis, ARF, hemodynamic instability, and oliguria may occur, further complicating the postoperative course [11, 13]. Open or revision bariatric surgery is recommended when peritonitis is identified [13].

SSIs are associated with a two- to three-fold increased risk of death, and a 60% increased risk of ICU admission [14]. Obesity and morbid obesity are independently associated with SSIs, particularly after clean (obesity OR = 1.757; morbid obesity OR = 2.544]) and clean-contaminated (obesity OR = 1.239; morbid obesity OR = 1.287) procedures [15]. SSIs are more common after open (16%) than after laparoscopic (4%) bariatric surgeries [14]. SSI incidence is 0.1–8.8% after LAGB, 0–8.1% after LSG, and 0–8.7% after LRYGB [10]. Strategies directed toward minimizing infectious complications should be adopted, including using minimally invasive approaches when possible, weight-adjusted antibiotic dosing, and layered closure of incisions [16, 17].

4.4 Medical Complications and ICU Admissions After Bariatric Surgery

Evaluating the Nationwide Inpatient Sample database, Stein et al. found that with routine use of antithrombotic prophylaxis after bariatric surgery, the in-hospital rates of pulmonary embolism (PE), deep venous thrombosis (DVT) without PE, and venous thromboembolism (VTE) (PE or DVT) were 0.9%, 1.3%, and 2.2%, respectively [18]. In-hospital mortality of patients with postoperative PE was 0.03%. Others reported similar rates for fatal PE: 0.03–0.06% [18]. PE risk is increased in patients with a previous DVT, lower limb venous stasis, BMI >55 kg/m^2, and obesity hypoventilation syndrome (OHS) with pulmonary arterial hypertension [9, 10]. DVT risk may differ according to surgical procedure. DVT incidence is 0–0.16% after LAGB, 0.32–1.21% after LSG, and 0–0.64% after LRYGB [10]. It is higher after open surgery [9, 10]. Diagnosing VTE may be difficult in obese patients: tachycardia, tachypnea, or hypoxia (especially without a fever or leukocytosis) suggest the possibility of PE. VTE management is the same as in non-obese patients.

Bariatric patients have an increased risk of PPCs, including ARF, atelectasis, pneumonia, and acute respiratory distress syndrome (ARDS). In the Nationwide Inpatient Sample database, the overall ARF rate was 1.35% among 304,515 patients undergoing bariatric surgery. ARF was more common after open surgery than after laparoscopic surgery (3.87% vs. 0.94%, $p < 0.01$) and was highest after open RYGB (4.10%) [19]. Age, BMI, American Society of Anesthesiologists' physical status, MS, and other comorbidities are associated with an increased risk of PPCs [20]. Despite its relatively low incidence, postoperative ARF is the fourth highest cause of mortality (11.8%) after bariatric surgery [21].

Perioperative atelectasis, which is more common in obese patients, is a main cause of intraoperative and postoperative hypoxemia and pulmonary infections [20]. Eichenberger et al. found that the incidence of atelectasis was significantly

higher in morbidly obese patients than in non-obese patients immediately after tracheal extubation (7.6% vs. 2.8%) and 24 h later (9.7% vs. 1.9%) [22]. Faster mobilization can facilitate faster resolution of atelectasis [20, 22].

Postoperative pneumonia is uncommon nowadays (<1%), as patients are now mobilized early after surgery and laparoscopic techniques produce less respiratory disturbance [23]. When suspected, appropriate antimicrobial therapy should be instituted immediately. Early postoperative pneumonia suggests the presence of a surgical complication [9].

ARDS, a severe acute inflammatory lung injury leading to hypoxemic ARF, is a response to other serious conditions, such as pneumonia, sepsis, or aspiration pneumonitis [24]. Obesity is an important risk factor for ARDS (OR = 1.89); however, obesity has been associated with a lower risk of mortality (OR = 0.63), reflecting the "obesity paradox" in ARDS. Specifically, there is a U-shaped relationship between BMI and ARDS mortality, with an OR of 0.88 in overweight patients, 0.74 in obese patients, and 0.87 in morbidly obese patients [24].

Stroke, myocardial infarction, cardiovascular failure, and AKI, which may require ICU admission, occur in <1% of patients after bariatric surgery [23]. Based on the Metabolic and Bariatric Surgery Accreditation and Quality Improvement Program database, the incidence rates of stroke, pneumonia, and myocardial infarction are 0.01%, 0.04%, and 0.21%, respectively, after this surgery [23].

Rhabdomyolysis can develop after prolonged compression of muscles during surgery, which may lead to AKI and mortality. A systematic review reported an AKI rate of 14% in patients with rhabdomyolysis after bariatric surgery, which was associated with a 25% mortality [25]. Aggressive hydration and diuresis with diuretics should be instituted immediately when serum creatine phosphokinase rises above 5000 IU/L [16].

AKI, usually due to rhabdomyolysis or hypovolemia, has been reported in 17.5% of patients admitted to the ICU after bariatric surgery, with 76.8% of AKI episodes limited to Acute Kidney Injury Network stage 1 [26]. In multivariate analysis of post–bariatric surgery ICU patients, male gender, premorbid hypertension, higher admission APACHE II score, and need for blood transfusion were associated with AKI, whereas pre-existing chronic kidney disease and BMI were not [26]. Prolonged surgery has also been identified as a risk factor for AKI [10].

4.5 Iatrogenic Complications and ICU Admission After Bariatric Surgery

Obesity is associated with an increased risk of ventilator-associated pneumonia, primarily because of an increased duration of mechanical ventilation [1]. Obesity is an independent risk factor for ICU-acquired intravascular catheter infections (obesity OR = 1.9; severe obesity OR = 3.2) and bloodstream infections (severe obesity OR = 2.2) [27]. In obese patients, iatrogenic complications are more common with subclavian access than with internal jugular and femoral venous routes [27]. Timsit et al. found no significant differences in catheter-related bloodstream or major

catheter-related infections between internal jugular and femoral routes in ICU patients with a high BMI [28]. Catheter tunneling, chlorhexidine dressings, and antiseptic-impregnated catheters are recommended for bariatric surgical patients, especially if the femoral vein is used [28].

4.6 Conclusions

Obese patients undergoing bariatric surgery, particularly if afflicted with OSA, MS, and other major comorbidities, should be carefully managed from both surgical and anesthetic perspectives to minimize the risks of postoperative complications and ICU admission, which contribute to morbidity and mortality. Early recognition and treatment of medical and surgical complications require prompt diagnosis and appropriate management to reduce ICU admissions or length of ICU stay, thereby improving outcomes.

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The ERAS Protocol

5

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5.1 The Enhanced Recovery After Surgery (ERAS) Protocols

Surgery is associated with a systemic stress response that can be categorized by duration and severity [1]. In 1995 for the first time a "stress-free" colonic resection based on the laparoscopic technique, epidural analgesia, early oral nutrition and mobilization was proposed [2]. Since 1995 many randomized controlled trials, consensus statements, meta-analyses and reviews have contributed to define safe and effective protocols of Enhanced Recovery After Surgery (ERAS) [3].

As defined by Ljungqvist, the concept of ERAS is based on a multidisciplinary and multimodal approach addressing issues that might delay the recovery and cause complications, changing the management according to a continuous evidence-based analysis and periodical internal audits [4].

In 2014, a meta-analysis of 16 randomized controlled trials (RCTs) performed in patients undergoing colorectal surgery, demonstrated how the application of an ERAS pathway was associated with a better outcome (reduction of respiratory and cardiovascular complications) and length of hospital stay, without affecting the readmission rate [5].

In the following years, ERAS guidelines were developed in different areas of surgery: hepatic and pancreatic, upper gastrointestinal, thoracic, urologic and gynecologic, and orthopedic surgery [4].

All the different ERAS protocols are based on common main surgical and anesthesiological items including strategies for evaluation and stratification of preadmission risk [5], preventing and treating postoperative nausea and vomiting [6], avoiding intraoperative hypothermia [7]. A mini-invasive surgical approach is strongly

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recommended [8, 9]. An adequate pain control with regional anesthesia or multimodal opioid-sparing analgesia [10], a careful perioperative fluid administration, adopting a goal-directed therapy in high-risk patients [11], are recommended to achieve an early mobilization and a quick restart in oral intake of fluids and solids.

Moreover, in the effort to obtain a rapid return to an adequate level of functional activity after a major procedure, ongoing ERAS researches are evaluating the efficacy of multimodal prehabilitation programs [12].

Nowadays, ERAS principles are applied in more than 20 countries and have been associated with an improvement in the quality of recovery after surgery, through a reduction in complications and in the length of hospital stay, with associated economic benefits [4].

Finally, a growing number of data suggest that high-risk patients, such as elderly and frail patients, should markedly benefit from ERAS programs reducing the impact of their comorbidities and the increased risk of developing postoperative complications [13].

5.2 The ERAS Protocol in Bariatric Surgery

The worldwide increase in the prevalence of obesity has led to a rise in the number of bariatric surgical procedures [14, 15]. Obesity leads to numerous changes in individual pathophysiology and requires specific management in order to provide safe bariatric surgery.

The main peculiarites of obese patients include:

1. Different patient shape: visceral vs. peripheral obesity. Compared to those patients with isolated peripheral fat distribution, patients with central obesity are at highest perioperative risk [16].
2. Alterations of the respiratory system: reduced lung functional residual capacity, more atelectasis and shunting in dependent lung regions, and increased work of breathing and oxygen demand.
3. High incidence of obstructive sleep apnea (10–20% in patients with BMI >35 kg/m^2), which is associated with a more than double rate of postoperative respiratory and cardiac failure [17].
4. Increased cardiovascular risk, including atrial fibrillation, arrhythmias and sudden cardiac death, prolonged QT interval, and highest rates of ischemic heart disease and heart failure [18].
5. Prothrombotic state [19].
6. Increased insulin resistance.
7. Different volumes of drug distribution.

5.2.1 ERAS Guideline Recommendations for Bariatric Surgery

Bariatric patients present unique challenges in the management of perioperative risks [20]. The ERAS protocol aims to reduce the body's stress response, reduce

organ dysfunction, and shorten hospital length of stay, and the key endpoint is the quality of the recovery [4]. To date, the literature supporting the use of ERAS in bariatric surgery is limited but it is rapidly growing. In 2016 the ERAS Society published the guidelines for bariatric surgery [21]: the recommendations are summarized below.

5.2.1.1 Preoperative Recommendations

- Provide patient preoperative counseling.
- Prehabilitation may improve the outcome.
- Patients should stop smoking at least 4 weeks before surgery.
- Alcohol abusers should observe at least 2 years of abstinence. The risk of new onset of alcohol abuse or relapse after gastric bypass should be assessed.
- Patients should lose weight before surgery. The risk of hypoglycemia should be acknowledged for patients receiving glucose-lowering drugs.
- Postoperative nausea and vomiting (PONV) and inflammatory response should be prevented with 8 mg of intravenous dexamethasone before anesthesia induction (preferably 90 min earlier).
- Fasting: 6 h for solids and 2 h for clear fluids is required before anesthesia induction. More data are needed for diabetic patients with autonomic neuropathy to assess the risk of aspiration.

5.2.1.2 Intraoperative Recommendations

- Avoid excessive intraoperative fluids and intraoperative hypotension with goal-directed fluid therapy. In the postoperative period fluid infusions should be interrupted as soon as possible.
- Prevent PONV with a multimodal approach.
- Be aware of the possibility of difficult airway management.
- Adopt lung protective ventilation.
- Patients should be positioned in order to improve lung mechanics (anti-Trendelenburg, flexed hip, anti- or beach chair).
- Deep neuromuscular block improves surgical performance.
- Neuromuscular blockade should be completely reversed. The use of qualitative monitoring of neuromuscular blockade is encouraged.
- Adopt BIS monitoring of anesthesia depth if end tidal anesthetic gas is not available.
- Laparoscopic surgery is to be preferred.
- Avoid postoperative nasogastric tube if not necessary.

5.2.1.3 Postoperative Recommendations

- Combine infiltration of local anesthetic and multimodal systemic analgesics.
- Consider thoracic epidural analgesia in laparotomic surgery.
- Adopt mechanical and pharmacological measures for thromboprophylaxis.
- Monitor protein intake. Iron, vitamin B12 and calcium supplementation is mandatory.
- Tight glycemic and lipid control in diabetic patients should be adopted.

- Prophylactic supplemental oxygen should be administered (preferably in head-elevated position or in semi-sitting position for patients suffering from obstructive sleep apnea [OSA]).
- Close monitoring of apneic episodes for patients with OSA.
- Consider CPAP if: BMI > 50 kg/m^2, severe OSA or oxygen saturation ≤90% with oxygen supplementation.
- Use patient's CPAP equipment for patients already with CPAP therapy at home.
- Patients with obesity hypoventilation syndrome (OHS) require intensive care monitoring and non-invasive ventilation

5.2.2 Effectiveness of ERAS Protocol in Bariatric Surgery

One of the main differences with respect to other surgical areas is that bariatric surgery generally involves young and physically fit patients, while in the non-bariatric settings the beneficial data are mainly derived from studies in elderly and frail patients. Furthermore, 30-day morbidity and mortality rates in bariatric surgery are relatively low (0.04% and 3% for severe complications, respectively [22]), and further reductions may be difficult to achieve. However, adherence to the ERAS pathway has shown to be feasible even at a national level, and able to improve outcomes [23]. A recent meta-analysis of ERAS protocols in bariatric surgery [24] demonstrated benefits in morbidity ($p < 0.01$), operative time ($p < 0.01$) and hospital length of stay ($p < 0.01$). Although limited by the methodology and quality of the included studies (only 13 studies retrieved, of which just 2/13 were randomized controlled trials and only 5/13 referred specifically to ERAS protocols), the ERAS protocol proved safe and effective. The whole perioperative care, from preadmission to the postoperative period, by a multidisciplinary team and a comprehensive and meticulous approach to the patient promoted by the ERAS protocols seems to be an effective strategy to improve outcomes also in bariatric surgery.

5.3 The ERAS Protocol for Emergency Surgery in Obese Patients

The ERAS protocol has proved to be feasible and effective in a wide range of surgical fields, allowing for better outcomes, fewer complications and cost savings [4]. The ERAS protocol has been successfully and extensively applied also in bariatric surgery, with a positive impact on many relevant outcomes [24].

Besides bariatric surgery, obese patients can require non-bariatric surgery. As a matter of fact, the global obesity epidemic implies that most (or all) anesthetists and theatre staff will have to deal with surgery in obese patients [25]. The optimal management of the obese surgical patients was addressed in recent authoritative guidelines [17, 26] and expert narrative reviews [27]: experienced anesthetic and surgical staff with good hospital organization and appropriate facilities were reported as crucial.

Emergency non-bariatric surgery in obese patients can be challenging. The few available data suggest that this population requires more resources and is at increased risk of complications compared with non-obese patients [28]. Application (as far as possible) of the guidelines for elective obese surgical patients has been proposed, together with the involvement of senior experienced anesthetists and surgeons [25]. To the best of our knowledge, no study to date has evaluated the feasibility, safety and efficacy of the ERAS protocol in emergency non-bariatric obese patients. Due to the emergency of surgery, preadmission and preoperative optimizations might often be not feasible. On the other hand, considering the benefits that were demonstrated in obese patients it seems reasonable to apply as completely as possible all the intraoperative and postoperative ERAS elements. As for the general management of obese surgical patients, a careful application of the ERAS measures is more likely when the staff is experienced in obese patient management and familiar with the ERAS protocol. This means that hospitals performing a high volume of bariatric interventions might offer the best possibility of an optimal implementation of the ERAS protocol in emergency surgical obese patients. However, before recommending centralization of emergency surgical obese patients in bariatric surgery hospitals further studies are required.

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Part II

Clinical Settings in Obese Patients

Trauma and Burns in Obese Patients

6

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6.1 Trauma in the Obese Patient

6.1.1 Introduction

Many studies in the past evaluated the impact of body habitus using the body mass index (BMI) on trauma patients. In a crash test with dummies, Moran et al. [1] reported that differences in body habitus can impact automotive safety features because of the disparity in the type and severity of injury in obese patients. Choban et al. [2] demonstrated than blunt trauma patients with BMI ≥ 30 had a greater incidence of pulmonary complications and mortality. Boulanger et al. [3] reported that patients with high BMI were more likely to suffer rib fractures, pulmonary contusions and pelvic fractures, while they were less likely to be associated with severe head and abdominal injuries. More recently, Bochicchio et al. [4] found that obese patients after trauma had a twofold increase in the risk of acquiring a bloodstream,

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urinary, or respiratory infection and being admitted to the ICU, with a seven times higher risk of dying in the hospital. Obesity has been considered an independent risk factor for nosocomial infections and multiple organ failure after trauma [5, 6], with the implication of greater resource allocation for this population. A correlation between obesity and mortality has been found in patients included in the German trauma registry [7]. This chapter reviews the anatomic and functional factors associated with trauma in obese patients.

6.1.2 Pathophysiology

Obesity influences the physiologic response to injury, with abnormalities which compromise the cardiovascular, respiratory and metabolic systems. Obese individuals have an increased circulating blood volume and cardiac output, with reduced compliance of the heart and a higher risk of sudden arrhythmias, cardiac failure and death [8]. Obesity leads to an increase in the work of breathing due to increased chest wall resistance and higher diaphragmatic position resulting from increased abdominal pressure. Minute ventilation is higher to compensate for the increased oxygen consumption and carbon dioxide production in the presence of alveolar hypoventilation. The risk of obstructive sleep apnea due to narrow upper airway because of excessive adipose deposition may increase the risk of respiratory failure and prolonged mechanical ventilation. Dyslipidemia, insulin resistance and pro-inflammatory states increase the risk of complications, such as infections and pulmonary thromboembolism. The etiology of increased risk of infections in obesity is a state of chronic low-grade systemic inflammation, due to adipocyte production of inflammatory cytokines with an immunodeficient state after trauma.

6.1.3 Care of the Obese Trauma Patient

Obesity was recognized as an independent risk factor for in-hospital mortality [9]. Obese blunt trauma victims were more likely to suffer pulmonary contusions as well as pelvic, extremity and rib fractures because of higher mechanical energy exchanged during impact and more fragile bones. Therefore, particular attention has to be paid in searching for these injuries in the diagnostic phase. On the contrary, the obese patient less often sustained severe head injuries [3]. Similarly, Arbabi et al. [10] found that obese motor vehicle occupants sustained more severe lower extremity injuries and less abdominal injuries because of mechanical protection of internal organs by fat (“cushion effect”).

In surgical critical care, obese patients represent a particularly challenging population. They often provide difficult airway management to even the more experienced anesthesiologists, and salvage techniques such as laryngeal mask and awake fiberoptic intubation should always be available [11]. Tidal volume in mechanical ventilation should be calculated by using ideal body weight to avoid barotrauma,

and reverse Trendelenburg position may improve pulmonary function. Obesity is associated with an increasing intra-abdominal and intra-thoracic pressure with concomitant stiffness of the heart due to the so-called "obesity cardiomyopathy" [12]. All these factors increase central venous pressure with influence on clinical decision-making regarding circulating volume status. Because of misleading end points, the morbidly obese patient is often under-resuscitated with a slower resolution of base deficit and pH and a higher mortality from persistent hemorrhagic shock due to relative hypovolemia [13]. It has been demonstrated that an underestimation of volume requirements in these patients may be the cause of decreased cardiac output and tissue oxygenation with poor outcome [14]. These data suggest that obese patients may benefit from goal-directed resuscitative therapy which is hopefully the resolution of metabolic acidosis and not only the normalization of standard hemodynamic parameters.

Prevention of deep venous thrombosis can be difficult at best. Mechanical devices often do not fit obese patients and higher doses of low-molecular-weight heparins may be required to achieve therapeutic anti-Xa levels [15].

When considering nutritional needs, the intensivist must be aware that the obese patient will preferentially metabolize proteins in stress states, rather than using fat stores [16], and these metabolic disorders probably represent a concomitant cause of persistent metabolic acidosis after trauma. Finally, obesity is associated with a pro-inflammatory state which affects post-injury inflammatory response with an increased risk of nosocomial infections and organ dysfunction. In a prospective observational study, Ciesla et al. [6] demonstrated that a BMI $\geq$ 30 was independently associated with multi-organ failure. An active surveillance of biologic specimens with early recognition of dangerous micro-organisms and a prudent policy of antibiotic administration are both of paramount importance to prevent the rise of multidrug resistant infections.

6.1.4 Conclusion

The alarming increase of obesity in Western countries causes an increase of patients with abnormally high BMI admitted to Trauma Centers. Further research is needed to investigate the influence of obesity on body response to injury although some conclusions may be drawn from the available literature.

- Obese blunt trauma patients sustain different patterns of injury from lean patients, with fewer and less severe head and abdominal injuries and more thoracic and extremity injuries.
- Resuscitation may be challenging due to difficult airway and ventilator management, impaired vascular access and altered end points for hemodynamic stabilization, leading to under-resuscitation and persistent metabolic acidosis.
- The metabolic syndrome leads to a hypercoagulable state which needs special attention to the prevention of thromboembolic events.

Because of all these considerations, the care of the obese trauma patient presents a distinctive challenge and may require changes to standard protocols with increased resource requirements. These considerations emphasize the need for long-term adjustments of medical and economic concepts for this category of patients, also in trauma settings.

6.2 Burns in the Obese Patient

6.2.1 Introduction

Obesity is a pathological condition that affects a large segment of the world population with enormous social and economic consequences. Burns are one of the most frequent causes of hospitalization after domestic accidents, and in some cases represent a systemic pathology that seriously endangers the survival of the individual. When present simultaneously, these two conditions represent a serious challenge as far as medical care is concerned. The first studies in the literature on the epidemiology of the burned obese patient date back to 1972 [17] and investigate a pediatric population in the USA. In this sample of 265 children, the authors concluded that the obese child was more exposed to the risk of burns because he or she moved less quickly and was therefore less able to avoid the burn agent. In more recent studies, also conducted on pediatric populations, obesity has been defined as a real risk factor in incurring a thermal agent injury [18].

6.2.2 Pathophysiology of the Obese Patient in the Treatment of Burns

Obesity is a systemic disease and as such has repercussions on various organs and systems. From a cardiovascular point of view, excess weight presents a risk factor in itself which, if associated with acute traumatic conditions such as burns, may represent an unfavorable prognosis regarding the treatment outcome [19–21]. It is a well-known fact that the obese patient is more predisposed to ischemic heart disease and heart failure, has a lower capacity for tolerance of physical exertion, and tends to develop arterial hypertension. These conditions can play an important role in the acute phase of the burned patient, in whom hydrating therapy with crystalloids determines high volume flows, and consequently puts the cardiovascular system under stress. Moreover, obesity limits motor rehabilitation activities, leading to an increase in the average length of stay in hospital.

The obese patient often suffers from respiratory disorders of a restrictive nature, resulting from an increase in intra-abdominal pressure, increased fat mass in the chest wall, and increased pulmonary vascular flow [22]; moreover, obstructive sleep apnea is a characteristic of obesity, caused by restriction in the upper respiratory tract. All these elements contribute to a basic respiratory condition that is not conducive to a favorable outcome of the many anesthetic and surgical procedures that a large burn victim undergoes.

In the treatment of the burned patient, drugs such as proton pump inhibitors are administered to prevent the formation of stress-induced gastric ulcer; in this regard it should be noted that the increase in intra-abdominal pressure, which characterizes excess weight, results in a greater tendency to hiatal hernia with less tone of the esophageal sphincter and greater prevalence of reflux disease.

The high prevalence of arterial hypertension and diabetes mellitus in the obese patient carries a high risk of renal failure with significant alterations also in the pharmacokinetics of different drugs. The doctor must be aware of these risks during the use of resuscitator fluid therapy in the burn patient [23]. Several studies have shown that an increase in BMI corresponds to a high thromboembolic risk [24]. This is above all due to the sedentary nature of the obese, high levels of fibrinogen, elevated platelet activation, and endothelial dysfunctions with the increase of the von Willebrand factor. Anticoagulant and antiplatelet therapies play a significant role in the treatment of the burn victim.

Stress induced by burn damage leads to a hyper-metabolic response of the organism [25] with the release of substances such as catecholamines and cytokines that reach their peak around the third day and lead to an increase in glycemic values; there is a well-known association between obesity and glucose homeostasis disorders [26]. In the treatment of the burn patient, blood glucose control and the early administration of exogenous insulin have proved to be crucial in reducing mortality [27] and improving wound healing. During an acute shock, hormones such as prolactin, growth hormone, thyroid-stimulating hormone, and other hormones are released. Obesity is associated with higher levels of many of these hormones, and some studies have shown that the obese burn patient is prone to developing hypertrophic scarring and galactorrhea due to excessive levels of melanocyte-stimulant resulting from hyperprolactinemia [28, 29].

6.2.3 Treatment of Burns and Obesity

The first action to take when treating a burns victim is to move the patient away from the source of the burn to reduce the contact time with the thermal agent and the extent of the injuries. It is more complicated to get the obese patient away from the burn source, given the body weight of the victim. The assessment of a burn injury is also based on the estimate of body surface area involved; in this regard, specific tables have been drawn up for the estimate as well as the so-called “rule of 9”.

However, this rule is not reliable above 80 kg of body weight as it would underestimate the damage. In fact, in the patient with excessive body weight the estimation values according to the “rule of 9” would be underestimated in body areas such as the lower limbs and the abdomen. According to a recent study, an appropriate estimate of the thermal damage on an obese subject establishes the following percentage distribution: 5% for each upper limb involved, 20% for each lower limb, 48% for the trunk and 2% for the head [28, 29].

The anesthetic procedures which a burn victim must undergo are more complicated if the patient is obese. Obesity is a predisposing factor for pressure sores in hospitalized patients who need continuous mobilization, which becomes problematic

in the case of excessive body weight. Furthermore, it is more complicated to change the position of the obese patient, which is necessary during surgical procedures, leading to longer operating times and greater predisposition to infection. The surgical techniques of escharotomy used in the burn victim become less precise in fat as it is poorly vascularized, and it is difficult to assess the bleeding and tissue viability. It can be complicated to carry out skin grafts, especially concerning immobilization. In fact, excessive weight predisposes to a greater risk of skin grafts detaching and destabilizing, with higher rates of failure. The use of skin grafts directly on fat is associated with lower engraftment rates, greater incidence of bacterial infections, and greater scar instability, all of which are unfavorable to healing in the obese burn patient. In fact, the rich fat component that characterizes the obese patient's body shape predisposes him or her to a greater risk of exposure of the adipose tissue after the surgical procedure of debridement in the treatment of thermal damage of the skin.

Physiotherapy rehabilitation is an essential and integral part of the treatment of the burned patient; the obese patient has serious physical limitations in performing physical rehabilitation and needs more nursing care than do normal-weight patients. He also requires more help regarding personal hygiene and mobilization.

Nutritional support is of fundamental importance in the critically ill patient. Inadequate nutrition reduces the rate of healing of wounds with an excessive imbalance in favor of the anabolic reactions of the body's protein components. Obesity also causes high metabolic demand on the part of the body, and this must be taken into consideration in the estimates of the daily energy and nutritional requirements of the burned patient [30].

There are some studies in the scientific literature that show how obesity is associated with prolonged hospital stay and higher mortality rates. This is certainly due to the greater comorbidity of the obese patient, the greater predisposition to the development of damage in different organs and systems, and the higher risk of postoperative complications [31].

Clearly, obesity has a major impact on all aspects of acute burn treatment and rehabilitation. The use of more accurate methods to calculate the burned body surface, perhaps using modern imaging techniques, will undoubtedly make a great contribution to setting a more precise and calibrated fluid therapy; furthermore, progress in research into optimal resuscitation endpoints may be beneficial to obese patients who are particularly prone to imbalances in water balance.

Obesity, now recognized as a chronic inflammatory condition, is an interesting field of research in pharmacology; studies are being carried out to find ways to counteract the morbidity due to the hypermetabolic response related to burn damage. Studies on the use of anti-inflammatory drugs in acute states of physical stress such as burn damage, associated with immune-stimulating nutritional elements such as glutamine and arginine, still need to be validated in burned obese patients [32].

6.2.4 Conclusion

In conclusion, obesity is a chronic inflammatory disease capable of increasing the systemic inflammatory response of the organism to acute phenomena such as burns

[33]. The excess of adipose tissue determines basic pathophysiological changes with impaired surgical, medical and anesthetic management of the burned patient. The greater rate of comorbidity, and the compromised conditions of various organs and systems in the obese make the treatment of the burned patient a significant social and medical care challenge.

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Perforations of the Upper Gastrointestinal Tract

7

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7.1 Introduction

Obesity is considered an important risk factor for the development of gastrointestinal disorders [1], likely through alterations of gastrointestinal motility. Even though gastroesophageal reflux disease is the condition mainly studied at present, the prevalence of other upper gastrointestinal symptoms has also increased.

Upper gastrointestinal tract perforations occur as a result of various causes. The majority of the perforations that we see today in the esophagus are iatrogenic (about 60%), but they could be spontaneous (Boerhaave's syndrome), traumatic or due to other causes. Perforation of a peptic, gastric or duodenal, ulcer is now less frequent because of the availability of adequate medical therapy. Peptic ulcer disease represented 1% of the discharge diagnosis of patients with a body mass index (BMI) > 25 kg/m^2 admitted to the Surgical Unit of Christchurch Hospital, New Zealand in a 26-month study period [2], and a surgical intervention for perforated viscus accounted for 4.4% of patients with a BMI > 30 kg/m^2 operated on at a US community teaching hospital in 1 year [3].

They represent a surgical emergency and the timing of the intervention is very important. Just one day of delay increases mortality significantly.

Since a detailed discussion of upper gastrointestinal perforations is beyond the scope of this chapter, attention has been directed to examining the peculiar characteristics of this topic in the obese population.

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7.2 Upper Gastrointestinal Complications of Obesity

The increased prevalence of gastrointestinal diseases in the general population may be related to the increased prevalence of obesity, particularly increased abdominal adiposity [1].

Central obesity (apple shape) increases the prevalence of esophageal motility disorders, gastroesophageal reflux disease, erosive esophagitis, Barrett's esophagus and esophageal adenocarcinoma. In contrast, obesity with increased hip circumference (pear-shaped) is related inversely to erosive esophagitis and Barrett's esophagus.

Gastric physiology and its neurohormonal regulation are altered in obesity. Obesity also is associated with symptoms such as upper abdominal pain, nausea, vomiting and gastritis. Obesity is a risk factor for erosive gastritis, hiatal hernia and gastric and duodenal ulcers. Obesity is considered a proinflammatory and procarcinogenic state and is recognized as an important risk factor for gastric cancer.

Obesity has been well recognized for its strong association with gallstone diseases.

7.3 Causes of Upper Gastrointestinal Tract Perforations

Peptic ulcer disease is the most common cause of stomach and duodenal perforation. Paraesophageal hernia [4] and gastric volvulus can all lead to gastric perforation (Fig. 7.1).

Most cases of iatrogenic perforation occur during therapeutic endoscopic procedures [5], particularly during endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD) for treating superficial cancers. The location of a tumor in the upper portion of the stomach is a risk factor for perforation during

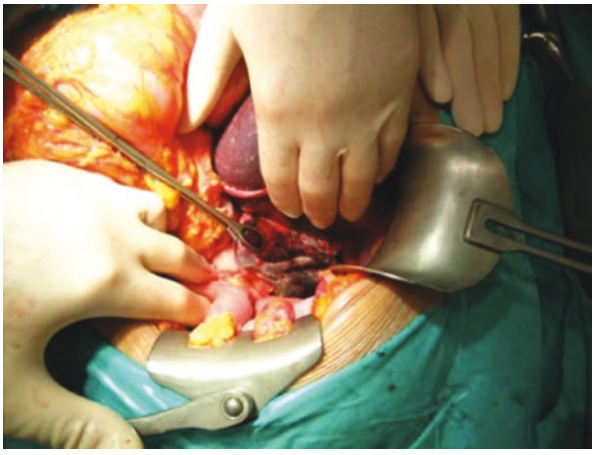

Fig. 7.1 Gastric fundal perforation due to strangulated paraesophageal hernia

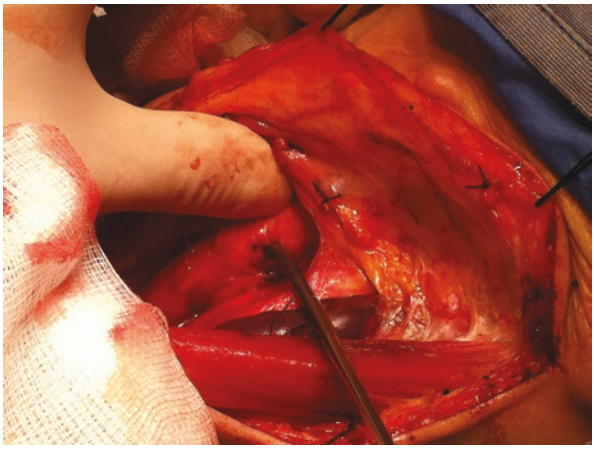

Fig. 7.2 Iatrogenic (after ERCP) perforation of cervical esophagus. *ERCP* endoscopic retrograde cholangiopancreatography

ESD. The location of the esophageal perforation depends also on the cause of the perforation:

- *instrumentation* 20% cervical (Fig. 7.2), 45% thoracic, 25% abdominal;
- *spontaneous* 40% thoracic and 60% abdominal;
- *traumatic* 80% cervical, 15% thoracic, 5% abdominal;
- *foreign body/caustic injury* 85% cervical, 15% thoracic;
- *operative injury* 5% cervical, 20% thoracic, 75% abdominal.

Delayed perforations of the esophagus or duodenum in patients with malignancy can be related to stent placement, following chemotherapy or as a result of radiation treatments.

Perforations have also been described in the context of diagnostic endoscopy. In a multivariate analysis of some 12,000 endoscopic retrograde cholangiopancreatography (ERCP) procedures performed over 12 years, obesity represented a risk factor (OR 5.18) for severe or fatal complications [6]. Obesity is also associated with an increased frequency of sedation-related complications for advanced endoscopic procedures.

7.4 Diagnosis

Patients with upper gastrointestinal perforation can range from completely asymptomatic to very sick. The vast majority of them almost invariably complain of acute neck, chest or abdominal pain to some degree, but a subset of them have little or no pain and will present in a delayed fashion.

If perforation is confined to the retroperitoneum or lesser sac (e.g. duodenal perforation) the presentation may be more subtle.

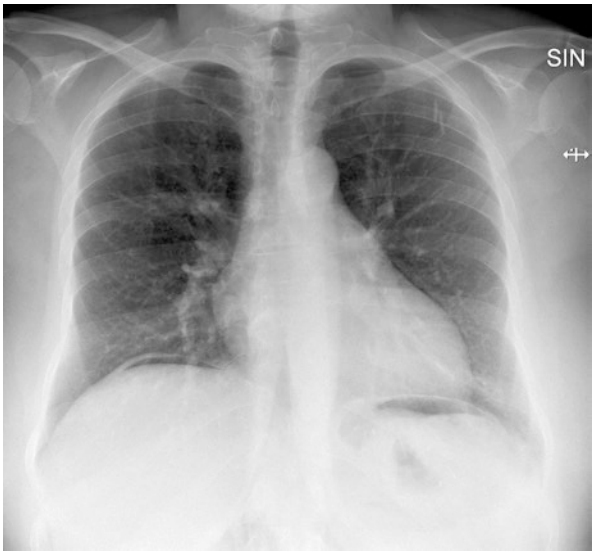

Fig. 7.3 Plain chest X-ray: free air under diaphragm

Sepsis can be the initial presentation. These patients are very ill-appearing, may be febrile (50%), and may be hemodynamically unstable.

Diagnosis relies upon imaging that demonstrates air outside the gastrointestinal tract (Fig. 7.3). Correctly diagnosing acute abdomen in a morbid obese patient is a challenge. The obese patient population can hide intra-abdominal catastrophes: by the time the patient is found to have peritonitis, it is often a late finding with the patient at significant risk for the subsequent development of abdominal sepsis, multisystem organ failure, and death. Diagnosis may be difficult or delayed due to unreliability of the physical examination. Large quantities of subcutaneous fat tissue hinder abdominal examination. Imaging resources are also affected: the quality of ultrasonographic evaluation is directly impaired by obesity, while computed tomography (CT) scan images with oral contrast are limited by patient size, and in extremely obese patients cannot be executed in centers not equipped to routinely image the morbidly obese. In cases of unclear diagnosis, diagnostic laparoscopy is recommended, as it is often a therapeutic procedure [2, 7, 8].

7.5 Treatment

When dealing with upper gastrointestinal perforations in the obese patient, the basic principles of general surgery apply: elimination of the septic process, provision of adequate drainage of any extraluminal collections, augmentation of host defenses by broad spectrum antibiotics and antifungal therapy, maintenance of adequate enteral nutrition [9, 10].

Patients manifesting hemodynamic instability or signs of rapid progression of sepsis or clinical deterioration should be explored without undue delays or extensive workups.

Esophageal perforation still carries significant mortality and morbidity. *Non-operative treatment* can be employed when a small, contained, incomplete tear of the esophagus is recognized right away and only if there is no adverse systemic response, tachycardia, fever or pain.

Surgery for uncontained leak should still be considered the gold standard. Primary repair (debridement, exposure of the mucosal defect, closure in two layers, buttress with local muscles, pleural flaps or gastric fundus) is the mainstay of treatment in the first 12/24 h. Wide drainage of contaminated spaces and jejunostomy for feeding access are mandatory. When diagnosis has been delayed, drainage, creation of an external fistula using a T-tube and diversion of luminal contents (cervical esophagostomy, gastrostomy/jejunostomy) are safer options.

Treatment strategies have been slowly shifting toward less invasive endoscopic approaches [11, 12]. A fully covered stent can be considered either alone or in combination with other techniques (regular clips, over-the-scope clips, endoscopic suturing, endoluminal vacuum therapy) when there has been a delay in diagnosis and surgical intervention is a high-risk strategy, especially if it requires a thoracotomy with one-lung ventilation.

Once a diagnosis of pneumoperitoneum has been made, laparoscopy appears to be the treatment of choice and should be preferred in obese patients. The laparoscopic approach for perforated peptic ulcer is feasible, the procedure is safe, with no increased risk of duodenal fistulae or residual intraperitoneal abscesses [13]. Immune function is better preserved after laparoscopic compared to open repair and may contribute to fewer septic complications in the laparoscopic group. The basis of treatment is closure of the perforation with an omental plug and thorough peritoneal lavage. In the case of gastric ulcer, care must be taken to exclude cancer. In patients with generalized peritonitis due to perforated peptic ulcer, the laparoscopy results are not clinically different from those of open surgery [14].

A large weight capacity table should be used and mechanical self-retaining retractors are needed for adequate exposure during laparotomy. A paraxiphoid extension of the midline incision is particularly important in obese patients, in whom access to the esophageal hiatus may be otherwise poor. Obese patients are considered to be at high risk of developing an incisional hernia after midline laparotomy.

For a laparoscopic approach, the patient must be secured for maneuvers, and longer trocars and instruments should be available. When dealing with “huge fatty liver” cutting the left triangular ligament may be useful to improve exposure.

7.6 Conclusions

No matter the operation performed the surgeon must expect an increased incidence of infectious complications including wound infection, bacteremia, and pneumonia. The widespread application of minimally invasive treatments may mitigate these complications in the obese group, and surgeons with bariatric surgery expertise may have an advantage in having minimally invasive skills compared with acute care surgeons without bariatric training [3]. A strong emphasis should be placed on early

and aggressive mobilization. An organized, multidisciplinary approach in this regard may help decrease the length of stay of obese patients requiring emergency operations.

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Acute Appendicitis in Obese Patients

8

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8.1 Introduction

Acute appendicitis (AA) is definitely one of the most common surgical emergencies, with a worldwide incidence of about 16 million cases in 2013 [1]. The male to female ratio is 1.4 to 1, with an overall lifetime risk estimated at 8.6% for men and 6.7% for women [2]. Appendicitis is most common between the ages of 10 and 30 years, but all age groups may be affected. Different distributions in incidence for AA are also determined by variations in ethnicity, season of the year and nutritional patient-related factors such as obesity [3–5]. Obesity represents a widespread condition in Western countries. In Italy the number of obese people in 2015 was about six million, equivalent to 9.8% of the population, with an incidence that grows with increasing age and is more prevalent among men than women. Another aspect to be considered is the close association between obesity and diabetes, which today in Italy affects 5.3% of the population, with a substantially doubled incidence compared to 30 years ago [6], while the total number of people with diabetes worldwide is projected to rise from 171 million in 2000–366 million in 2030. One of the main pathophysiological features of diabetes is the alteration of the microcirculation, also at the splanchnic level, resulting in an augmented risk of developing AA in this group of patients, together with an increased resistance to antibiotic therapy. A retrospective analysis using the American College of Surgeons-National Surgical Quality Improvement Program (ACS-NSQIP) database from 2004 to 2010 in over 300 US hospitals concluded that patients with diabetes and no other significant comorbidities had a higher risk of developing surgical site infections with longer hospital stay after appendectomy than patients without diabetes [7].

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In cases of AA, all these elements are amplified by the context of surgical emergency. It must then be noted that if these issues are usually well addressed in dedicated bariatric surgery divisions, this is often not so true in common general and emergency surgery units, where most AA cases are actually treated [8].

The current concept is that there are two types of AA, an uncomplicated appendicitis and a complicated appendicitis. An uncomplicated appendicitis is defined as an inflamed appendix without any sign of gangrene, perforation, periappendicular contained phlegmon, free purulent fluid or intra-abdominal abscesses, whereas the presence of one or more of these features distinguishes a complicated appendicitis [9]. Conditions such as obesity increase the risk of evolution from an uncomplicated to a complicated form, thus increasing the incidence of postoperative morbidity.

8.2 Diagnostic Work-Up

The objectives of the diagnostic process in suspected AA are both to provide a correct diagnosis—and thus minimize the risk of negative appendectomies—and to stratify the severity of AA by discriminating between uncomplicated and complicated forms. Despite the efforts, the rate of negative appendectomies is still reported to be up to 27% [10].

Classically, the diagnosis was made by combining clinical and anamnestic data with the biochemical findings of increased blood inflammation markers. However, the clinical presentation of AA may vary from mild symptoms to a pattern of generalized peritonitis and sepsis, making the diagnosis of AA a challenge. Over the years, several score systems have been proposed. The purpose of these classification systems is to guide the clinical decision-making process, in order to optimize the need for diagnostic imaging, reduce admissions, and prevent negative surgical explorations. In high-risk patients such as the obese, the Appendicitis Inflammatory Response (AIR) score showed specificity (97% versus 76%) and positive predictive values (88% versus 65%) statistically better than the Alvarado score [11].

Ultrasound (US) represents a useful, non-invasive and readily available diagnostic tool, with a sensitivity and specificity of 58% and 76%, respectively [12]. However, it must be remembered that US diagnostics is highly operator-dependent and that negativity, especially in the case of an unseen appendix, does not rule out the diagnosis of AA but suggests proceeding with further investigations [13].

Abdominal computed tomography (CT) is superior to US in terms of accuracy, but the radiation exposure of abdominal CT is a concern in children and during pregnancy. If required, a low-dose CT scan is preferred in patients with suspected AA [14]. A CT scan is indicated in elderly patients who may have a neoplasia, in atypical or delayed clinical presentations, in the suspicion of an appendicular mass, and in patients who may underestimate the clinical data such as obese patients.

In obese patients, the diagnostic accuracy of US is diminished due to an increase of the subcutaneous and intra-abdominal fat. Anderson et al. demonstrated that the body mass index (BMI) does not alter the diagnostic accuracy of a CT scan. CT appears therefore more reliable than US in obese patients with the exception of

children and pregnancy [15]. If there are diagnostic doubts in children and pregnant women magnetic resonance imaging (MRI) can be successfully used, having demonstrated both sensitivity and specificity comparable to those of CT, although it represents an expensive examination that is not always available [16].

8.3 Management

Surgery remains the therapy of choice with 95.92% of AA treated by appendectomy, showing a crude rate of mortality and postoperative complications of 0.1% and 3.5%, respectively [17]. Surgery can be performed by open appendectomy (OA), first described by Fitz in 1886 [18] and then codified by McBurney in 1894 [19], or by laparoscopic appendectomy (LA) introduced by Semm in 1983 [20]. However, the paradigm of treatment is increasingly shifting from OA to LA, for both adults and children.

Non-operative management (NOM) for AA in adult patients was recently investigated and represents a topic under debate. Outcomes were discordant, reporting an efficacy between 45% and 81% at 1-year follow-up [21, 22], but globally demonstrated a lower effectiveness of NOM compared to the surgical approach.

Timing of appendectomy after a hospital admission is another topic of discussion. The real question is whether a delay in appendectomy could increase the risk of a progression from uncomplicated to complicated appendicitis and consequently increase the postoperative morbidity rate [23]. A retrospective analysis from ACS-NSQIP on 32,782 patients undergoing appendectomy did not reveal statistically significant differences in terms of morbidity and short-term outcomes between patients operated within 6 h, between 6 and 12 h, and over 12 h from the admission [24]. These results were partially confirmed by the UK study of Banghu et al. according to which the risk of surgical site infections and adverse events would increase only after 48 h [25]. Vice versa, according to Busch et al., an appendectomy delayed more than 12 h in a frail patient should be avoided, as an in-hospital delay represents an independent risk factor for perforation, similar to an age over 65 years old and the presence of significant comorbidity such as obesity, hepatopathy and heart diseases [26]. A recent meta-analysis from Cheng et al. concluded that there are currently no elements of superiority either for early or for delayed appendectomy in AA [27].

To date, LA represents with grade A of recommendation the approach of choice for the treatment of AA, as it demonstrated clear advantages in terms of lower incidence of surgical site infections, less pain, shorter hospital stay and earlier return to daily activities [19, 28]. LA is specifically indicated in obese patients and in frail or high-risk patients and allows better short-term outcomes even in pediatric patients. A recent Cochrane meta-analysis, analyzing 85 studies, 10 of which on children, showed better outcomes of LA over OA in terms of control of pain, wound infection rate, length of hospital stay and return to normal life, while a worse performance was highlighted regarding the incidence of intra-abdominal abscesses (IAAs) [29]. Some other trials tended to reconsider this last conclusion in view of the fact that an

increased IAA rate for LA characterized its early days of diffusion and was therefore related to surgical expertise [30]. A systematic review of nine meta-analyses from Jaschinski et al. confirmed the findings regarding better short-term outcomes following LA versus OA [31]. Data from the analysis of the US Nationwide Inpatient Sample in the period 2003–2011 showed the trend in diffusion of LA, which increased from 41.7% to 80.1%, with greater penetrance to patients over 65 years old (from 9.4 to 11.6%), obese (from 3.8 to 8.9%) and with more comorbidities according to the Elixhauser score (from 4.7 to 9.8%) [17].

These results emerged in both elective [33] and emergency surgery like appendectomy [34], even for increased-risk patients [35]. In patients with BMI over 30 kg/m^2, LA was more effective than OA in terms of overall morbidity and mortality rates [32, 36]. The laparoscopic approach showed reduced operative time and length of hospital stay, as well as a statistically lower incidence of wound infections and IAAs, substantially confirming what has emerged for non-obese patients in recent years [37–39]. Ciarrocchi et al. carried out a meta-analysis of five papers comparing OA versus LA in obese patients, concluding that the laparoscopic approach offered significant advantages in terms of lower intra-abdominal abscesses, wound infection and overall postoperative complication rate, as well as a shorter operative time and hospital stay [40]. Dasari et al. proposed a systematic review of the literature by analyzing eight studies on the role of laparoscopic appendectomy in the obese patient, including one prospective randomized trial and seven retrospective papers, without any intention-to-treat analysis. There were no statistically significant differences in outcomes between obese and non-obese patients undergoing LA, confirming the effectiveness and safety of the minimally invasive technique even in patients with a BMI higher than 30 kg/m^2 [8].

Single-incision laparoscopic surgery (SILS) appendectomy was first proposed by Pelosi in the early 1990s [41] with a view to further minimizing the surgical trauma, and then resumed a few years ago. Randomized clinical trials that compared appendectomy by SILS and conventional laparoscopic approach showed a comparable postoperative morbidity rate, while the outcomes in terms of operative time and conversion rate were detrimental to SILS [42, 43]. SILS was associated with a higher risk of port-site hernias than conventional laparoscopic surgery. There has been evidence that obesity was a risk factor for developing port-site hernias after SILS [28, 44].

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Pancreatic and Biliary Emergencies

9

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9.1 Acute Cholecystitis in Obese Patients

Acute cholecystitis is a high-prevalence disease in Westernized societies: 10–20% of the US population suffers from gallstones and about one-third of them develop acute cholecystitis [1]. Obesity is currently recognized as an important risk factor for the development of gallbladder pathologies: a recent analysis found that 75% of patients who underwent cholecystectomy were overweight or obese, and >20% of all cholecystectomies were performed with a diagnosis of acute cholecystitis [2].

Multiple factors may contribute to the increased risk of cholesterol gallstones in obese patients: increased hepatic secretion of cholesterol in obese patients [3] and, consequently, bile saturated with excess cholesterol [4]; impaired gallbladder motility [5]; enhanced mobilization of cholesterol [6]; reduced hepatic secretion of bile salts due to decreased hepatic bile acid pool [6]; reduced gallbladder contractility [6]; increased secretion of biliary calcium [6].

In obese patients, as in the normal population, cholecystectomy followed by antibiotic therapy is the gold standard treatment for acute cholecystitis. However, some concerns have to be carefully evaluated:

1. Is a laparoscopic approach feasible in obese patients?
2. Is obesity a risk factor for conversion from laparoscopic to open cholecystectomy for acute cholecystitis?
3. Does obesity increase the risk of postoperative complications after laparoscopic cholecystectomy for acute cholecystitis?

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Since the early 1990s, the laparoscopic approach to gallbladder disease has been the procedure of choice. Traditionally, obesity has been considered a relative contraindication to laparoscopic cholecystectomy for symptomatic cholelithiasis [7]. Potential difficulties related to laparoscopic cholecystectomy in obese patients can include excess abdominal wall fat, excess intra-abdominal fat, difficulty accessing the abdomen, and problems with establishing and maintaining pneumoperitoneum [8]. With increasing experience in laparoscopic surgery and the development of better instruments, the routine performance of laparoscopic cholecystectomy in obese patients is rapidly growing and, currently, obesity is not considered an absolute contraindication for a laparoscopic approach. However, a recent study utilizing the National Surgical Quality Improvement Project (NSQIP) database demonstrated that the super-obese (BMI > 50) had a lesser percentage of attempted laparoscopic cholecystectomies (81% in the super-obese vs. 87% in the overall cohort; $p < 0.001$) [9].

Some techniques to combat the potential pitfalls for laparoscopic cholecystectomy in obese patients include: using extended or extra-long trocars [8]; increasing the insufflation pressure to >15 mmHg in order to maintain adequate working room in the peritoneal cavity [10]; using a Veress needle in the left upper quadrant and placing the camera port in a supraumbilical position to decrease the distance between the port site and the operating field [8].

Robotic single-site cholecystectomy is a relatively novel approach and an alternative to the laparoscopic approach for gallbladder removal [11]. Svoboda et al. recently reported their experience with 112 cholecystectomies (of which 23.3% performed for acute cholecystitis) successfully performed with the robotic approach in patients with BMI > 30 without conversion to open, laparoscopic or multiport procedures [12]. Total mean operative time was 69.8 min for obese patients compared to 59.2 min in the non-obese ($p = 0.0012$). There were no major complications recorded, including bile leak, hematoma, or ductal injury [12].

The effect of obesity as an independent risk factor for conversion from laparoscopic to open cholecystectomy has been debated during the last decade and still remains controversial. Several studies found that obesity was a risk factor for conversion [9, 13, 14], whereas other studies did not [15–17]. Despite these controversies, on the other hand, it has been clearly demonstrated that total operative time was significantly longer in obese patients, if compared with the normal population [8, 15].

It is more difficult to evaluate whether obesity must be considered an independent risk factor for the development of complications after cholecystectomy, because conflicting results have been reported. In a large Swiss national study [18], intraoperative complications were significantly higher in those patients with a body weight >90 kg, if compared with those with weights from 60 to 69 kg (OR 1.25), 70 to 90 kg (OR 1.24), and <60 kg (OR 1.34). Moreover, local postoperative complications were also significantly higher in patients with a body weight >90 kg compared with those with a body weight <60 kg (OR 1.53) or 60–69 kg (OR 1.46) [18]. These results were confirmed by another study [19]: obese patients undergoing laparoscopic cholecystectomy for cholecystitis had higher complication rates than non-obese patients (19.2% vs. 15.7%, respectively; $p < 0.0001$), particularly more infectious complications. However, other studies did not confirm these results [8,

14]. Farkas et al. [14], for example, found that the complication rate was not different across the spectrum of BMI values (overall complication rates of obesity class I, II and III patients were 6%, 8.1% and 4.5%, respectively; $p > 0.05$). In confirmation of these data, several studies demonstrated that the postoperative length of stay was not significantly affected by BMI [8, 11, 13]. However, it must be noted that only 15–20% of the patients reported in the previously mentioned studies had acute cholecystitis [12, 15]. A study utilizing the NSQIP database and evaluating outcomes after cholecystectomy for acute cholecystitis in obese patients has been recently published [9]. This study demonstrated that BMI class was not associated with increased risk of death/serious complications and that there was no significant difference in terms of morbidity and mortality between the intended open and laparoscopic converted to open groups, even at the highest BMI classes [9]; on the other hand, open cholecystectomies were associated with an increased risk of mortality and morbidity, independent of BMI class [9]. These results demonstrated that starting with a laparoscopic approach did not seem to put the patient at risk (even if the laparoscopic approach was converted to an open procedure) and that an initial attempt at laparoscopy may benefit patients (even those with very high BMI).

In conclusion, we can summarize that:

- Laparoscopic cholecystectomy is feasible also in obese and super-obese patients.
- It is not clearly demonstrated that obesity is an independent risk for conversion to open surgery. Moreover, studies demonstrated that an initial attempt at laparoscopy should be performed.
- Even though the results of published studies are conflicting, obesity seems to be an important risk factor for developing postoperative complications after cholecystectomy for acute cholecystitis.

9.2 Acute Pancreatitis in Obese Patients

Acute pancreatitis is an acute inflammatory process that involves the pancreas, the peripancreatic tissues and remote organ systems [20]. The incidence of this disease is increasing and ranges from 13 to 45 per 100,000 [21]. The clinical course of acute pancreatitis is extremely variable, ranging from interstitial edematous to severe acute pancreatitis. Approximately 20% of all cases of acute pancreatitis are severe [22], requiring treatment in hospital for several months and with a mortality rate up to 20% [23]. Gallstones are the most common cause of acute pancreatitis, representing close to 60% of all cases [24]; several other risk factors include alcohol, drug consumption, toxins, smoking, and metabolic and/or endocrine disorders [25].

Obesity is recognized as an important risk factor for the occurrence of acute pancreatitis [26]; moreover, not only BMI but also abdominal adiposity and visceral adipose tissue seem to be related to a higher incidence of acute pancreatitis [27]: a nationwide Swedish study, evaluating 68,158 individuals for a period of 12 years, demonstrated that the risk of acute pancreatitis among those with a waist circumference of >105 cm was twofold increased (RR 2.37; 95% CI 1.50–3.74) compared with those with a waist circumference of 75.1–85.0 cm [27].

Multiple local and systemic factors have been implicated in the occurrence and severity of acute pancreatitis in obesity:

- Patients with a high BMI have an increase of pancreatic fat [28], which has a direct toxic effect on the parenchyma of the pancreas [29]. It has also been demonstrated that deposits of intra-abdominal peripancreatic fat in obese patients undergo necrosis during acute pancreatitis, contributing to the developing of necrotizing pancreatitis [30].
- Fat composition in obesity is predominantly unsaturated, and lipolysis, determining an increased release of cytokines, worsens local and systemic injury [31, 32].
- Obesity in itself constitutes a low-grade proinflammatory state: a higher level of proinflammatory cytokines, such as TNF-α, IL-10. IL-6, IL-1β, and plasminogen activator inhibitor-1 have been described [31, 32]. Hence, the inflammatory response is increased and there is an up-regulation of the proinflammatory cytokines that leads to a larger inflammatory response.
- During acute pancreatitis, there is a restriction of chest wall and diaphragmatic movements that leads to a decrease in inspiratory capacity: this determines an increase of physiologic pulmonary arteriovenous shunting, thus leading to hypoxemia. Hypoxemia produces an oxygen deficit and exacerbates the underlying cellular damage from the inflammatory response, which subsequently increases the rate of multiorgan failure and death [32].

The treatment of acute pancreatitis in obese patients follows the same rules as in non-obese patients. Surgery is necessary only in cases of pancreatic infected necrosis and of failure of conservative treatments (antibiotics, endoscopic or radiological procedures). However, in the case of acute pancreatitis in obese patients, two aspects have to be kept in mind:

- these patients have a higher risk of developing a severe form of acute pancreatitis and thus of developing complications;
- consequently, in obese patients a careful attention in the first phase of the inflammatory disease is of great importance.

Several epidemiologic studies suggested that obesity or increased intra-abdominal fat is associated with severe acute pancreatitis, including organ dysfunction, infection, pancreatic necrosis, length of hospital stay, and use of intensive care [33–35]. For example, Krishna et al. in a nationwide inpatient study evaluated the impact of morbid obesity on acute pancreatitis-related clinical outcomes and healthcare utilization [33]. The authors demonstrated that morbid obesity was associated with increased mortality (OR 1.6; 95% CI 1.3–1.9), prolonged hospitalization (0.4 days; $p < 0.001$) and higher hospitalization charges; moreover, performing a propensity score-matched analysis, they demonstrated that acute kidney failure (10.8% vs. 8.2%; $p < 0.001$), respiratory failure (7.9% vs. 6.4%; $p < 0.001$) and mortality (OR 1.6; 95% CI 1.2–2.1) were more frequent in morbid obesity [33]. De Waele et al. investigated the occurrence of complications in different classes of overweight in a group of patients with gallstone pancreatitis [35]. When compared

with normal-weight patients (BMI 18.5–24.9), all categories with BMI ≥ 25 had an increased risk of developing the "severe" form of acute pancreatitis (OR 3.55; 95% CI 1.50–8.40): patients with class I obesity (BMI 30–34.9) developed significantly more organ failure and local complications (OR 3.469; 95% CI 1.15–10.43); patients with class II and III obesity (BMI 35–49.9) had also more metabolic complications (OR 7.33; 95% CI 1.62–33.24), needed more frequently intensive care and had a longer hospital stay than did their normal-weight counterparts [35]. The increasing risk of severe pancreatitis in obese patients has been confirmed by four meta-analyses published within the past 10 years [32, 36–38]. In the most recent of them [32], including 12 clinical studies, obese subjects had an increased risk of developing severe acute pancreatitis (RR 2.20; 95% CI 1.82–2.66), a higher risk of local (RR 2.68; 95% CI 2.09–3.43) and systemic complications (RR 2.14; 95% CI 1.42–3.21) and a higher risk of in-hospital mortality (RR 2.59; 95% CI 1.66–4.03) when compared with non-obese patients [32].

According to these reports, it is clear that prediction of severe acute pancreatitis in obese patients is important for clinical decisions involving triage, admission vs. transfer, and specific treatment of patients. For these reasons, during the last decade several studies have suggested including BMI in scoring systems for prediction of the severity of acute pancreatitis. Among several studies [39–41] evaluating the addition of obesity to the APACHE-II scoring system, only one study successfully demonstrated that APACHE-O was predictably better than APACHE-II [40]. In this study, the authors prospectively evaluated 186 consecutive patients with acute pancreatitis, to allow calculation of the APACHE-II score and BMI (categorized as normal (score = 0), overweight (BMI 26–30, score = 1), or obese (BMI > 30, score = 2)): the addition of the score for obesity to the APACHE-II score gave a composite score (APACHE-O) with greater predictive accuracy. At cut-off >8, APACHE-O had 82% sensitivity, 86% specificity, 74% positive predictive value, 91% negative predictive value and 85% overall accuracy. Although most experts believe that current methods for prediction of severity have limitations for individualized assessments, the consensus is that more studies are needed before including obesity in determining severity [42].

In conclusion:

- Obesity is a well-recognized risk factor for the occurrence of acute pancreatitis.
- In the obese population, it is more frequent to see the development of severe forms of acute pancreatitis, with a significant increase in terms of morbidity and mortality. An early diagnosis and treatment of the disease is therefore mandatory in this subset of patients.

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Bowel Obstruction in Obese Patients

10

Vincenzo Pilone and Mafalda Romano

10.1 Introduction

Bowel obstruction, also known as intestinal obstruction, is a mechanical or functional obstruction of the intestines which prevents the normal movement of the products of digestion [1]. Either the small bowel or the large bowel may be affected. Small bowel obstruction (SBO) is one of the most frequent emergencies in general surgery [2], commonly affecting obese patients. Morbidity and mortality from SBO in the obese are high [3]. Significant progress has been made in the diagnosis and management of bowel obstruction in recent years. However, little is known about whether this progress has benefited outcomes in obese patients, particularly those who have other comorbidities: abnormal cardiorespiratory function, metabolic function and hemostasis, which may predispose to morbidity and mortality after surgery. Bowel obstruction is the cause of about 5–15% of cases of severe abdominal pain of sudden onset requiring admission to hospital [2].

10.2 Signs and Symptoms

The clinical presentation of the patients varies and no one clinical symptom on its own identifies the majority of patients with SBO [4]. Some studies have suggested

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that the absence of passage of flatus and/or feces and vomiting are the most common presenting symptoms [1], with abdominal discomfort/distention the most frequent physical examination findings. Other studies have shown that abdominal pain is present in the majority of patients found to have SBO.

Some signs and symptoms associated with SBO include the following [4]:

- nausea/vomiting 60–80% (vomitus can often be bilious in nature);
- constipation/absence of flatus 80–90% (typically a later finding of SBO);
- abdominal distention 60%;
- fever and tachycardia (late findings and may be associated with strangulation).

In SBO, the pain tends to be colicky (cramping and intermittent) in nature, with spasms lasting a few minutes [5]. The pain tends to be central and mid-abdominal. Vomiting may occur before constipation. In LBO, the pain is felt lower in the abdomen and the spasms last longer [5]. Constipation occurs earlier and vomiting may be less prominent. Proximal obstruction of the large bowel may present as SBO especially in obese patients. Bowel obstruction may be complicated by dehydration and electrolyte abnormalities due to vomiting; respiratory compromise from pressure on the diaphragm by a distended abdomen, or aspiration of vomitus; bowel ischemia or perforation from prolonged distension or pressure from a foreign body. In obese patients, often there are no signs or symptoms and the diagnosis is performed late. In our experience, obese patients with acute abdomen present tachycardia (surgical pulse).

10.3 Etiology

The causes of intestinal obstruction can be broadly divided into mechanical or functional categories, and further subdivided in the following way [4].

Dynamic causes

- Intraluminal
  - impaction
  - foreign bodies
  - bezoars
  - gallstones
- Intramural:
  - stricture
  - malignancy
- Extramural:
  - adhesions
  - hernia
  - volvulus
  - intussusception

Adynamic causes
- Paralytic ileus
- Mesenteric vascular occlusion
- Pseudo-obstruction (Ogilvie's syndrome).

10.3.1 Most Common Causes of Bowel Obstruction in Obese Patients

- Mechanical SBO [6]
  - adhesions 80%
  - hernias 8–15%
  - other: Crohn's disease, intussusception, hematoma, bezoar
- Mechanical LBO
  - malignancy (60%, most frequently sigmoid),
  - volvulus (10–15% sigmoid<cecum)
- Functional (also referred to as paralytic) obstruction is relatively rare as a presentation to the emergency department.

In one prospective observational study [4] of 150 consecutive adult obese patients admitted with acute mechanical bowel obstruction, 114 (76%) presented with SBO and 36 (24%) with LBO. Adhesions (64.8%), incarcerated hernias (14.8%) and large bowel cancer (13.4%) were the most frequent causes of obstruction. The most common cause of SBO in obese patients is intra-abdominal adhesions, accounting for approximately 65–75% of cases, followed by hernias, Crohn disease, malignancy, and volvulus.

10.4 Diagnosis

Diagnosis of the type of bowel obstruction is normally conducted through initial plain radiograph of the abdomen, luminal contrast studies, computed tomography (CT) scan [7] or ultrasonography, prior to determining the best type of treatment. Plain radiographs should be obtained first in patients in whom SBO is suspected. Although not sensitive, upright abdominal films may help substantiate the diagnosis if the presence of air-fluid levels or a paucity of gas is observed. Note that supine films may obscure the detection of air-fluid levels. Multislice CT has been shown to be a particularly effective imaging tool for evaluating patients suspected of having SBO, with a sensitivity of over 95% [8]. CT imaging is also capable of detecting complications of SBO not visualized on plain films, including ischemia, perforation, mesenteric edema, and pneumatosis. Ultrasonography is less costly and invasive than CT scanning and may reliably exclude SBO in as many as 89% of patients [9]; specificity is reportedly 100%. It may be a useful alternative imaging modality in children and pregnant women.

Enteroclysis is another valuable diagnostic test in detecting the presence of obstruction and in differentiating partial from complete blockages. This study is useful when plain radiographic findings are normal in the presence of clinical signs of SBO or when plain radiographic findings are nonspecific. However, CT imaging has superseded enteroclysis owing to the increased availability of CT scanners and the increased risk of perforation and aspiration with enteroclysis.

For obese patients, it is sometimes impossible to perform a CT scan due to the small dimensions of the instruments. Many hospitals are not equipped with beds capable of accommodating oversize patients (in general, over 130 kg weight). This kind of patient is transferred to specialist obesity centers.

10.5 Treatment

Some causes of bowel obstruction may resolve spontaneously [10]; many require operative treatment. In obese adults, surgical intervention and treatment of the causative lesion are frequently required. Improvements in radiological imaging of SBO allow for confident distinction between simple obstructions, which can be treated conservatively, and obstructions that are surgical emergencies (volvulus, closed-loop obstructions, ischemic bowel, incarcerated hernias, etc.). Most patients treated with conservative care improve within 2–5 days after initiation of therapy [11]. If the obstruction is complete surgery is usually required.

10.5.1 Conservative Treatment

Conservative treatment of bowel obstruction consists of aggressive fluid resuscitation, bowel decompression, administration of analgesia and antiemetic as indicated clinically, early surgical consultation, and administration of antibiotics.

Initial decompression can be performed by placement of a nasogastric tube for suctioning gastrointestinal contents and preventing aspiration. Airway, breathing, and circulation (ABC) must be monitored [12]. Blood pressure monitoring, as well as cardiac monitoring in selected patients (especially elderly patients or those with comorbid conditions), is also important. Fluid replacement with aggressive intravenous resuscitation using isotonic saline or lactated Ringer solution is indicated. Oxygen and appropriate monitoring are also required. Antibiotics are used to cover gram-negative and anaerobic organisms. In addition, analgesia and antiemetic are administered as indicated clinically. As previously mentioned, a non-operative trial of as many as 3 days is warranted for partial or simple obstruction. Most people with bowel obstruction are initially managed conservatively because in many cases the bowel will open up. Some adhesions loosen up and the obstruction resolves. The patient is examined several times a day, and X-ray images are obtained to ensure there is no clinical worsening. Resolution of the obstruction occurs in virtually all patients with these lesions within 72 h [11].

10.5.2 Operative Treatment

The problem of managing bowel obstruction is still unsolved. In the first instance, surgery is applied to cases of complete bowel obstruction and to cases showing no improvement after 48–72 h [13].

Laparoscopy has taken the place of traditional laparotomy as an elective treatment for obese patients [14]. It is associated with a lower rate of morbidity and shorter hospitalization. As laparoscopic surgery is becoming a treatment option in emergency surgery for acute cholecystitis, acute appendicitis, and peptic ulcer perforation, SBO could be a candidate for adaptation to laparoscopic surgery. Laparoscopic surgery is a non invasive technique to reduce postoperative adhesions [14] and therefore has been widely used in recent years. However, surgeons often tend to hesitate in applying laparoscopic surgery for SBO because of some situational disadvantages such as poor operating space or iatrogenic bowel injury [15]. The safety and effectiveness of laparoscopic exploration depend on the surgeon's experience and the etiology of the obstruction [16]. Steps include examining the abdominal cavity, identifying/alleviating obstruction source(s), running the bowel to assess for viability, confirming resolution of obstruction(s), and identifying/repairing injuries. In surgery for bowel obstruction, the open and laparoscopic approaches each have their own advantages and disadvantages [14]. When deciding on the surgical approach, the condition of the patient and the complexity of the procedure must be considered. Conditions such as age greater than 65, post Roux-en-Y gastric bypass, inflammatory bowel disease, malignancy, virgin abdomen, diabetes, pregnancy, hernia, early postoperative state, and malnutrition deserve special consideration. Patients selected to be operated by the open approach had higher preoperative morbidity than the ones selected for the laparoscopic approach. After matching for this disparity, the laparoscopic approach was associated with a shorter length of hospital stay without differences in complications [17]. The laparoscopic approach may be a preferable approach in selected patients. When operating on obese patients, access to the abdomen can be particularly challenging and performing open surgery in obese patients is fraught with more bleeding, a higher risk of injury to other organs because of difficult exposure, as well as an increased likelihood of postoperative wound infections and incisional hernias.

Moreover, open laparotomy treatment for SBO is associated with postoperative adhesion and the recurrence of SBO. Open laparotomy is widely accepted as the standard approach for SBO in patients who present symptoms that suggest a clinical and physiological emergency such as toxemia or ischemia. Recent reports have shown the rate of conversion to be 10–39%. Two reviews showed that laparoscopic surgery for SBO was completed in 55% and 64% of cases, with conversion rates of 33.5% and 29%, respectively [14]. The common cause for conversion to laparotomy is the inability to maintain a field of vision and to control the operative field to allow for the safe and effective handling of the dilated bowel loops. In the management of SBOs, a commonly quoted surgical aphorism is "never let the sun rise or set on small-bowel obstruction" because about 5.5% of SBOs are ultimately fatal if

treatment is delayed [18]. Morbidity and mortality owing to SBO increase when there is an undue delay in operation and decrease with the institution of appropriately timed surgery [19]. Laparoscopic adhesiolysis for SBO seems to have a clinically proven advantage over the open approach [20]. Laparoscopic adhesiolysis has been shown to be safe and feasible in experienced hands [21]. For selected patients, laparoscopic adhesiolysis offers the advantages of decreased length of stay, faster return to full activity, and decreased morbidity [21]. In the other cases, the treatment is chosen on the type of obstruction. In the case of occlusions of large bowel, resection of bowel is often required. This is the case of malignancy [22], invagination and diverticular disease. Cases of SBO related to cancer are more complicated and require additional intervention to address the malignancy, recurrence, and metastasis, and thus are associated with poorer prognosis.

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Large Bowel Obstruction in Obese Patients

11

Antonio Di Cataldo, Salvatore Perrotti, Carlos Rivera, and Emanuele Lo Menzo

11.1 Introduction

Large bowel obstruction can be mechanical or adynamic. Colorectal cancer is the single most common cause of mechanical obstruction. In the United States approximately 2–5% of patients with colorectal cancer present with complete obstruction. Other causes of colorectal obstruction include diverticulitis, Crohn's disease, schistosomiasis, intussusception, anastomotic stricture, fecal impaction and foreign bodies.

Extraluminal causes include adhesions (more frequent in small bowel obstruction but rare in colonic obstruction), tumors in adjacent organs and volvulus.

Obesity has been recognized as a worldwide problem and it is associated with many co-morbidities such as coronary artery disease, diabetes mellitus, dyslipidemia and sleep apnea. Several large bowel conditions, such as diverticulosis and colorectal cancer, have been reported to have higher incidence in the obese patient. However, some authors concluded that comorbidities such as obesity, hypertension, diabetes and smoking did not correlate with the rate of bowel obstruction [1].

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11.2 Diagnosis

Rapid evaluation beginning with a focused history and physical examination should occur in patients presenting with symptoms of a large bowel obstruction. It is important to elicit a history of chronic constipation or diarrhea, malignancy, and any prior surgery.

Physical examination findings include abdominal distension and tympany. Other findings are palpable abdominal masses, point tenderness, or peritonitis. A digital rectal examination also should be performed to assess for a possible rectal mass or blood.

Basic labs should be obtained including a complete blood cell count for assessment of anemia or elevated white blood cell count, complete chemistry and evaluation of electrolyte imbalances. A lactate also should be included if there is any suspicion of ischemia.

Typical radiographic findings in large bowel obstruction include air/fluid levels within dilated loops of colon, failure of contrast to pass distally, and luminal caliber change and may demonstrate characteristic findings of volvulus [2–4], or pneumoperitoneum. Computed tomography (CT) scan accurately distinguishes between true colonic obstruction and pseudo-obstruction, can accurately diagnose intraluminal, intrinsic, and extrinsic causes [5–7] and sigmoid volvulus. CT is also useful in distinguishing between sigmoid and cecal volvulus, which is important, since the initial treatment may differ because of the different endoscopic success rates [8]. Colonoscopy is not usually needed as an initial diagnostic modality for acute colorectal obstruction, but it can aid in the diagnosis of those patients with chronic symptoms for whom large bowel obstruction cannot be excluded on plain films or abdominal CT.

11.3 Management and Approach

The management is directly correlated to the etiology of the large bowel obstruction.

11.3.1 Preoperative Evaluation. Medical Risk Assessment

Major medical conditions should be identified and optimized before performing colon surgery. Evaluation of cardiac risk, cardiovascular diseases and diabetes mellitus are needed. Obesity has been associated with increased risk, but whether the preoperative approach to obese patients should differ from that in the general population is uncertain [9].

In the presence of large bowel obstruction, any type of bowel preparation is contraindicated but this fact is **not** a contraindication to primary anastomosis [10]. On the other hand, preoperative bowel preparation should be given to patients who have been stented as a bridge to surgery, especially when planning a colocolonic (instead of an ileocolonic) anastomosis [11].

11.3.2 Supportive Care

Initial management of the patient with colorectal obstruction consists of supportive care that includes gastrointestinal decompression and intravenous fluid therapy with correction of electrolyte abnormalities.

Flexible sigmoidoscopy is generally suggested to initially decompress the colon and may be the only treatment in high-risk patients. However elective resection during the same hospital stay is recommended for sigmoid volvulus because of the high rate of recurrence (up to 50%) with endoscopic decompression alone [12].

In patients with acute malignant mechanical obstruction colonic stenting could be used as a bridge to surgery. This provides a window of time prior to surgery to correct fluid depletion and electrolytes, and allow for mechanical bowel preparation, thereby performing surgery under elective, rather than emergent, circumstances [13]. The benefit of this approach is to potentially allow for resection and primary anastomosis (one-stage operation) rather than two-stage operation. Up to 50% of patients with neoplastic large bowel obstruction are not candidates for curative resection [14]. For these patients stenting is a successful alternative in over 90% [15] and palliative chemotherapy can be immediately started [16].

11.3.3 Surgical Management

Acute complete or near complete colorectal obstruction is an urgent surgical problem that accounts for up to 4% of surgical urgent abdominal admissions. The toughest decision facing the surgeon is whether or not to perform a primary reanastomosis at the operation and this decision is determined by weighing the perceived risk of anastomotic leakage against the morbidity of performing a stoma.

Whether to choose a staged procedure depends upon the location of the obstructing lesion, condition of the proximal colon, medical comorbidities of the patient, and presence of proximal perforation [17]. If a stoma is a likely event, an evaluation of its most appropriate location should be done preoperatively by the enterostomal nurse. This is especially true in obese patients, due to the challenges of large pannus.

11.3.4 Technical Considerations in the Obese Patient

The obese patient poses specific considerations and challenges. An adequate operating room table has to be available. Proper position and padding can reduce the incidence of peripheral nerve damage and skin breakdowns. Particular challenges can be faced in positioning the patient in a lithotomy or prone jack-knife position. It is safe to use strapping to secure the patient to the table to avoid shifts and potential falls during surgery. This is particularly important if a laparoscopic procedure is planned, where steep Trendelenburg and reverse Trendelenburg positions with side rotations are necessary to improve exposure. In fact, the frequent presence of heavy omentum, mesentery and thick abdominal wall usually makes surgical exposure

challenging. Trocar placement in laparoscopy has to take into consideration the thickness of the pannus and the subsequent limited mobility. In general ports should be placed with minimal angles and extralong and additional trocars might be necessary. Access to the abdominal cavity can be safely achieved with gasless optical techniques either in the paraumbilical area or at Palmer's point in the left upper quadrant, and care must be taken in the presence of distended bowel. Exposure deep in the pelvis is usually achieved by steep Trendelenburg positions, and patient strapping, body securing and padding are paramount.

For open procedures, generous incisions are usually necessary. The use of lighted retractors and head lamps aid in the visualization of deep and narrow pelvis. Another challenge is the maturation of ostomies through a thick pannus and is achieved by moving the ostomy site more cephalad and medially. Obesity increases exponentially the ostomy complications such as ischemia, retraction in the acute phase, and prolapse and parastomal hernias chronically.

11.3.5 Procedures to Manage Colorectal Obstruction

Whenever possible, a primary anastomosis (one-stage procedure) is the preferred treatment for right- or left-sided colon obstruction [18, 19], but very frequently a diverting ileostomy is advisable. If the local situation requires, the classic Hartmann's operation (two-stage procedure) could be the best solution.

11.3.6 Perioperative Considerations

Besides the usual perioperative care with aspiration prevention and fluid management, the obese patient poses unique challenges. Venous thromboembolic events (VTE) are significantly more prevalent in the obese population and preoperative evaluations for hypercoagulability and presence of acute and chronic thrombosis should be completed. Perioperative deep vein thrombosis (DVT) prophylaxis should include both mechanical and pharmacological methods. Conflicting evidence exists on the best pharmacological agent, dose and duration. In general, the prophylaxis is started prior to induction of anesthesia and maintained until patient discharge. In the presence of particular risk factors (immobility, prior VTE, hypercoagulable states, malignancy) extended use of pharmacologic agents should be considered, although the evidence in the literature is controversial. Early patient mobilization is a key element of VTE prevention with the availability of physical therapist and supporting staff. The use of appropriate beds can reduce the development of pressure ulcers. Fluid resuscitation should be adequate for the body weight and tailored to the fluid losses and urine output. In the immediate postoperative period and for the next 48–72 h respiratory complications related to sleep apnea, atelectasis and pneumonia should be carefully monitored. Continuous pulse oximetry and telemetry are necessary in patients with a history of obstructive sleep apnea. The use of positive pressure machines should be implemented early. Adequate pulmonary toilet and breathing exercises can minimize postoperative atelectasis. Obese patients are at risk for postoperative cardiovascular

events, so close cardiac monitoring, fluid balance and pain control are important. Specific cardiac medications should be restarted as soon as possible, and specific ones, such as beta blockers, should never be discontinued. A solid body of evidence exists on the advantages of continuing certain antiplatelet medications (aspirin) in the perioperative period, with lack of increased bleeding risks. Patients on chronic anticoagulation should be carefully monitored, not only for bleeding complications but also because of the higher risk of perioperative VTE events.

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Abdominal Compartment Syndrome in Obese Patients

12

Jacopo Viganò, Angelo D'Ovidio, Gabriele Bocca, and Paolo Dionigi

12.1 Introduction

The peritoneal cavity can be considered a closed structure characterized by its own compliance and pressure. In obese patients the excess of intra-abdominal adipose tissue can interfere with many internal functions. Abdominal compartment syndrome is a clinical entity recognized in critically ill patients due to an abnormal increase of the intra-abdominal pressure causing severe organ complications and death. This syndrome requires specific medical and surgical treatments that can be effective in many, but not all, patients. Recently, a chronic abdominal compartment syndrome associated with morbid obesity has been identified.

12.2 Intra-Abdominal Pressure, Intra-Abdominal Hypertension, and Abdominal Compartment Syndrome: Background and Definitions

In normal-weight people the physiological value of intra-abdominal pressure (IAP) is 5–7 mmHg. This parameter is strictly related to several internal functions [1, 2].

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In fact, IAP is directly linked to the perfusion pressure of abdominal organs according to the formula:

$$\text{Abdominal perfusion pressure} = \text{Mean arterial pressure} - \text{IAP}$$

There is a close relation between abdominal pressure and renal function as follows [1, 2]:

$$\text{Glomerular filtration rate} = \text{Mean arterial pressure} - (\text{IAP} \times 2)$$

Elevated values of IAP cause a cephalad elevation of the diaphragm, reduce pulmonary gas exchanges, induce a rise in intracranial pressure and a decrease in venous return resulting in lower cardiac output [2]. IAP monitoring is essential in critical patients, since its variations can impair vital functions. However, the increase of IAP is not always connected to organ damage, because physiological mechanisms can balance its effect.

Intra-abdominal hypertension (IAH) is recognized when IAP exceeds 12 mmHg. It is graded as follows: Grade 1 = 12–15 mmHg, Grade 2 = 16–20 mmHg, Grade 3 = 21–25 mmHg and Grade 4 > 25 mmHg.

Abdominal compartment syndrome (ACS) occurs at IAH >20 mmHg when it is associated with one or more organ dysfunction. Usually renal function is compromised first, the kidney being more sensitive to ischemic damage.

Normally a transitional phase without organ impairment between IAH and ACS can be observed. Both IAH and ACS are usually underestimated: their frequency in critical patients varies from 30 to 60% for IAH and from 0.5 to 36% for ACS. The guidelines of the World Society of the Abdominal Compartment Syndrome (WSACS) were updated in 2013 [3, 4].

IAP monitoring is well standardized and provides valuable clinical information [5, 6].

The main risk factors for the development of IAH or ACS can be summarized as follows:

- diminished abdominal wall compliance (i.e., extensive burns, major trauma or prolonged prone position);
- increased intra-luminal content (i.e., mechanical or paralytic ileus);
- increased intra-abdominal content (i.e., hemoperitoneum, pneumoperitoneum or diffuse endoabdominal infections);
- capillary leak with accumulation of liquid in the third space and tissue edema (i.e., during acidosis, hypothermia, septic shock or after massive fluid resuscitation).

According to the classification of the WSACS, IAH and ACS can be divided in three groups:

- ***primary IAH/ACS***, due to diseases of the abdominal/pelvic area that often require emergency surgical/invasive treatments (diffuse peritonitis, hemoperitoneum, retroperitoneal hematomas and ascites);
- ***secondary IAH/ACS***, when the causes are outside the abdominal/pelvic compartment (excessive fluid resuscitation, massive transfusions, acidosis and hypothermia);
- ***recurrent IAH/ACS***, when the increase of intra-abdominal pressure is a direct consequence of a medical or surgical treatment of a primary or secondary ACS. This includes patients undergoing emergency laparotomies for diffuse peritonitis, in which the closure of the abdomen is followed by an ACS due to visceral edema, inflammation, and reduced compliance of the abdominal wall.

As underlined by Reintam et al., it is important to recognize the cause of IAH/ACS because it implies a high risk of mortality. In general, secondary IAH/ACS are more difficult to treat and present a worse prognosis, whereas in primary IAH/ACS a surgical treatment can be rapidly effective [7].

12.3 Principles of Treatment of Patients with IAH and ACS

In cases of IAH/ACS, the WSACS guidelines suggest medical and surgical interventions [4].

12.3.1 Medical Treatment

Since monitoring of the IAP represents the cornerstone for the early diagnosis of ACS, patients with IAH without organ damage must undergo medical treatment to reduce intra-abdominal pressure regardless of the primary or secondary cause (Table 12.1).

Table 12.1 Medical procedures that can counteract the pathophysiological mechanisms of IAH

To increase abdominal wall compliance	Sedation and analgesia Neuromuscular blockade Reverse Trendelenburg position
To reduce endoluminal content	Rectal and nasogastric decompression Prokinetic agents
To reduce endoabdominal fluids	Paracentesis Percutaneous drainage of abscesses
To obtain negative fluid balance	Fluid restriction Diuretics Colloids/hypertonic fluids Hemodialysis/ultrafiltration

IAH intra-abdominal hypertension

12.3.2 Surgical Treatment

The failure of a medical strategy and the appearance of organ damage demonstrate that the patient is developing a true ACS. Patients with established ACS may need either medical or surgical treatments, depending on the underlying pathology.

In primary ACS, the patient undergoes surgery with the dual purpose of decompressing the abdomen through a laparotomic approach and treating the etiological causes. At the end of the surgical procedure, the option to leave an open abdomen should be carefully considered.

In secondary ACS, the initial treatment must be based on the previously described medical strategies. In the case of failure of the medical approach, a laparotomic surgical approach must be considered to decompress the abdomen despite the absence of primary abdominal pathologies.

12.3.3 Open Abdomen

Open abdomen treatment represents a fundamental tool in the presence of secondary ACS refractory to medical approaches. It allows easier removal of primary or secondary causes of ACS up to resolution of the visceral damage.

Final closure of the abdominal wall can be delayed, but it should be done as quickly as possible to avoid the development of further severe complications (infections, enteroatmospheric fistulas, ventral hernias) [8]. When the abdomen is open, a temporary abdominal closure with a passive or active system must be considered [9]. Passive systems are cheap mechanical barriers (like the "Bogota bag" or "Wittmann patch") that protect the abdominal content; they can be used in the case of shortage of resources. An active system associates a suction device with a protective dressing for the so-called "negative pressure wound therapy" (NPWT) [10, 11] (Fig. 12.1). The negative pressure within the system contributes to remove exudates and maintain tension on the abdominal wall. Among the active systems,

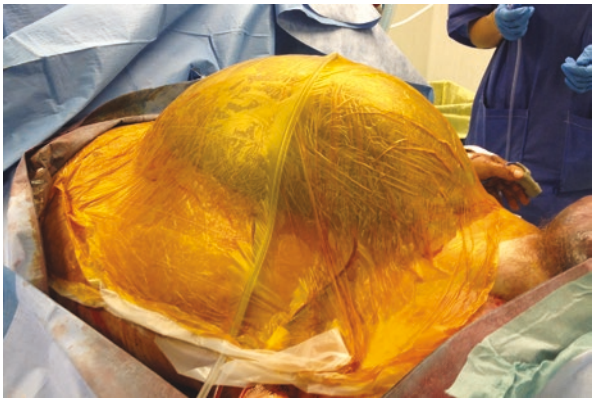

Fig. 12.1 Temporary abdominal closure with NPWT: a perforated barrier covers the viscera and a polyurethane sponge placed between the fascial edges. The wound is covered by an airtight seal and the sponge is connected to a suction drain. *NPWT* negative pressure wound therapy

further progress could be represented by the association of NPTW and a polypropylene mesh, sutured to the fascial margins, progressively cut and sutured to improve final closure of the abdomen because a prolonged delay provokes a severe fascial retraction [11].

12.4 Morbid Obesity and IAP, IAH and ACS

A direct correlation between the degree of obesity and elevated values of IAP is well known. In obese patients (BMI ≥ 30–35) baseline IAP is significantly higher than in the normal-weight population (9–12 mmHg) [11–14]. Central obesity (metabolic syndrome) with an increased sagittal abdominal diameter is associated with an increase in IAP [15]. The elevated chronic value of IAP in morbid obesity could depend on a direct mass effect from the intra-abdominal adipose tissue due to the fact that it is not a fluid and does not equalize pressure inside the abdominal cavity. The mass effect is more clearly detected when the abdominal fascia is intact [5]. In the immediate postoperative period, IAP is increased in obese patients undergoing either open or laparoscopic gastric bypass, most likely in relation to the surgical trauma and inflammatory response or a massive fluid administration [16].

The chronic elevation of IAP may be responsible for the pathogenesis of obesity-related comorbidities such as arterial hypertension, pseudotumor cerebri, pulmonary dysfunction, gastroesophageal reflux and abdominal wall hernias [5], hypertrophic cardiomyopathy, reduction of venous return, reduction of glomerular function: a clinical picture described as “chronic compartment syndrome” [17].

The question of whether the chronically elevated values of IAP observed in morbidly obese patients can promote a faster increase in IAH and a more rapid organ deterioration when ACS occurs has not been answered. The risk of developing an IAH and an ACS in obese patients is higher than in normal-weight subjects due to the fact that physiological compensation mechanisms, normally arising in the case of increased IAP, are already involved in maintaining homeostasis. Therefore, in the critically ill patient with morbid obesity, assessment of the IAP should be a part of common clinical practice in order to establish an early diagnosis of IAH and prevent ACS. Management of IAH is similar in obese and non-obese patients, but the treatment of ACS in the obese is more challenging and may be associated with higher complication rates [4].

12.5 Open Abdomen Treatment in Obese Patients for Abdominal Sepsis

Generalized peritonitis is a major cause of primary ACS especially in obese patients due to the severe peritoneal edema and the presence of intra-abdominal fluids in response to the inflammatory process or perforation of hollow organs. In these patients a surgical decompressive laparotomy represents the first-choice treatment.

The role of surgery is to control the source of the infection and to contribute to treat the cause of ACS. Open abdomen with temporary abdominal closure represents the ideal technique to cure ACS and avoid relapses while putting the surgeon in the condition to easily re-explore the abdomen and to control intra-abdominal infections.

The clinical criteria in favor of an open abdomen are not yet completely defined. Open abdomen treatment is recommended [18, 19] when:

- it is impossible to approach the fascia;
- IAP is above 15–20 mmHg after a partial closure of the abdomen with some stitches;
- after closure of the abdomen hemodynamic changes appear or high ventilation pressures are required;
- it is impossible to perform effective control of ACS with a single laparotomy.

Another indication for open abdomen is represented by obese patients when candidate to a procedure of damage control after trauma.

When an open abdomen treatment is planned in obese patients, it is desirable to use active temporary abdominal closure systems with NPWT. According to the more recent World Emergency Surgery Society guidelines [8], in the management of an acute abdomen due to sepsis, surgeons should follow these recommendations:

- multidisciplinary patient management with close cooperation among all the operators in order to agree on a common strategy; particular attention should be paid to maintain a slightly negative water balance, to correct acidosis, hypothermia and coagulopathy;
- since the patient with open abdomen is hypercatabolic, enteral or parenteral nutritional support becomes mandatory;
- re-explorations of the abdomen should be performed every 24–48 h in the case of clinical worsening of the patient.

Healing may be a very difficult target when enteroatmospheric fistulas are present in the open abdomen. Nutritional and hydroelectrolytic support associated with dynamic wound closure systems are essential. NPWT can be a valuable tool, especially when fistulas arise from the anterior aspect of the exposed viscera [20]. In these cases, through an appropriate use of drains/catheters/nipples or other specific devices, it is possible to exert a negative pressure within the abdomen after isolating the fistula properly. The delay of the final suture of the abdominal wall is mandatory.

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Part III

Clinical Settings After Bariatric Surgery

A Brief History of Bariatric Surgery

13

Giuseppe Navarra, Gianfranco Silecchia, Luigi Piazza, Iman Komaei, and Mauro Toppino

13.1 Introduction

Since its recognition as a disease by the World Health Organization in the late 1940s, an uncontrolled increase in the incidence and prevalence of obesity and its associated debilitating illnesses has led to evolution of this condition into a major public health issue and a global epidemic; worldwide, obesity has nearly tripled since 1975 [1]. Bariatric or weight loss surgery, a relatively young branch of surgery, has been shown to be extremely beneficial, with very low mortality and morbidity, in achieving long-term weight loss results, as well as in prevention, improvement, and often resolution of obesity-related comorbidities.

Although the foundation of bariatric surgery was laid in the early 1950s, the routine application of this practice as a tool in the treatment of obesity did not gain widespread acquisition until the 1990s, when despite all previous non-surgical and

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surgical attempts, the epidemic of obesity remained unchecked [2]. About the same time, the introduction and prompt advancements of minimally invasive and laparoscopic technology revolutionized the bariatric field and led to an increased patient demand and an immense rise in the number of bariatric surgeries performed worldwide. In spite of this rapid transition from open to laparoscopic surgery, mortality in bariatric surgery was lowered and weight loss results remained unchanged. The manifold advantages of laparoscopic over traditional surgery such as quicker recovery, less postoperative pain, better esthetic results, and a huge reduction in wound-related complications soon made it the preferred surgical approach for both patients and surgeons. In the twenty-first century the rate of surgeries performed laparoscopically has risen dramatically, exceeding that of open surgery in 2005. Despite ongoing extraordinary innovations in minimally invasive surgery and the development of novel techniques such as robot-assisted laparoscopic surgery, traditional laparoscopy is still considered the "gold standard" surgical approach in treatment of obesity and its comorbid diseases [3–5].

13.2 Evolution of Bariatric Surgery

The initial idea of utilizing surgery as a tool in the treatment of obesity was pioneered by the observation of significant weight loss in patients who were subject to gastric or small-bowel resection. Hence, the earliest bariatric procedures were designed and aimed to induce chronic malabsorption and weight loss by surgically bypassing a functional segment of the small intestine [2].

13.2.1 Intestinal Bypass Procedures

Viktor Henrikson (1891–1969) is accredited as being the first surgeon to perform weight loss surgery, in Sweden in 1952. His operation involved resection of about 105 cm of an unspecified segment of the small intestine which eventually yielded unsatisfactory results [6]. Inspired by Henrikson's surgical experiment, in 1954 Kremen et al. [7] performed a purely malabsorptive bariatric procedure, named jejunoileal bypass (JIB). In this procedure, the proximal jejunum was anastomosed to the distal ileum, bypassing a long segment of the small intestine. In the early 1960s, Lewis [8] and Payne [9] proposed a jejunocolic bypass as a potential alternative for JIB, with an anastomosis between the proximal jejunum and varying segments of the colon; however, due to severe adverse effects, the procedure was soon abandoned. During the 1960s and 1970s, several modifications were proposed to optimize the intestinal bypass procedure but, due to drastic complications, none of them gained widespread acceptance. The most lethal complication, hepatic failure, was the result of so-called "bypass enteritis", due to overgrowth of anaerobic bacteria in the bypassed intestinal limb. Other frequently seen complications of JIB included severe malnutrition as a result of vitamin, mineral, and protein deficiencies and dumping syndrome, characterized by nausea, bloating, and profuse diarrhea after

ingestion of certain foods, especially sugar [10, 11]. The reported overall mortality rate for JIB was about 4% in the first two postoperative years and was often a sequel of liver failure [12]. By the mid-1970s, JIB became less popular as the field of bariatric surgery was advancing briskly and newer, less morbid procedures were being developed.

13.2.2 Gastric Bypass

In 1966, following previous experiences with intestinal bypass procedures and noticing a remarkable weight loss in patients who had undergone partial gastrectomy for gastric ulcer disease, Mason and Ito [13] conducted the first gastric bypass (GBP) surgery. This procedure can be regarded as the turning point in bariatric surgery, introducing for the first time the concept of gastric restriction as the main contributing factor for weight loss. The surgery originally consisted of a horizontal transection of the stomach with a loop gastrojejunostomy [14]. In 1977, Griffen et al. [15] modified the technique using a Roux-en-Y reconstruction instead of the loop configuration. The addition of a mild malabsorptive component to the technique resulted in greater success in both induction and maintenance of weight loss. Moreover, the loop configuration considerably optimized the former procedure by minimizing the risk of biliary reflux and anastomotic leaks. In the 1980s, the Roux-en-Y gastric bypass (RYGB) gained more popularity and up until today is one of the most frequently performed bariatric procedures worldwide. RYGB has been shown to be extremely effective with both desirable short- and long-term results. Excess weight loss of about 50% is often achieved as early as 6 months after surgery. In contrast to intestinal bypass procedures, RYGB resulted in less diarrhea and protein malnutrition. However, dumping syndrome and marginal ulcers were still problematic. Moreover, calcium, iron, and vitamin malabsorption as a result of bypassing the distal stomach and duodenum required lifelong nutritional support and careful follow-up of the patients undergoing RYGB.

13.2.3 Gastroplasty

In the 1970s, in pursuit of safer alternatives for JIB and RYGB, restrictive procedures were developed. Surgeons believed that elimination of the malabsorptive component minimized the risk of severe malnutrition. The purely restrictive procedures were designed to diminish the volume of food and caloric intake by altering the normal anatomy of the stomach. In 1971, Printen and Mason [16] pioneered horizontal gastroplasty, a technique consisting of transverse partitioning of the stomach using a mechanical stapler, leaving a small orifice between the two gastric pouches. This primary form of gastroplasty revealed undesirable outcomes with very poor long-term weight loss results. Later in the 70s, several modifications to improve the original technique were proposed by Gomez, Pace, Carey, Martin, Long and Collins [17–21]. Nevertheless, results remained disappointing.

In 1980, Mason [22] proposed and performed the first vertical banded gastroplasty (VBG). This procedure involved vertical stapling of the stomach, featuring a small pouch based on the lesser curvature with a small outlet reinforced with a mesh strip or a synthetic ring. VBG became highly popular as the short-term outcomes proved to be promising, with excess weight loss of 60% or more in only 1 year. By 5 years, however, excess weight loss was at a rate of 50% in only half of the patients [23]. Disappointing long-term results were reported to be partially a consequence of staple line dehiscence [24]. Other complications associated with VBG included bezoar formation, vomiting, outlet stenosis and obstruction, perforations, post-surgical leaks, gastroesophageal reflux and megaesophagus [25].

13.2.4 Jaw Wiring

In 1977, Rodgers et al. [26] disclosed their experience with jaw wiring, with early weight loss results corresponding to that of RYGB. Their idea was based on the fact that a forced reduction in food intake would result in sustained weight loss. However, this approach soon proved to be ineffective, as patients still were able to consume high caloric liquids and had weight regain after removal of the wire. Maintaining oral hygiene, frequent emesis and aspiration pneumonia were other concerns with this procedure [27].

13.2.5 Gastric Banding

Gastric banding, a procedure which involved neither resection nor stapling of the stomach was designed with the hope to be the least invasive bariatric procedure. Similar to gastroplasty procedures, the primary objective was a marked reduction of the volume of the stomach and limiting food intake [2]. In 1978, Wilkinson and Peloso [28] placed the first non-adjustable gastric band, a strip of Marlex mesh around the upper stomach. About the same time, similar operations were performed utilizing Dacron grafts and silicone as safer alternatives, but these early bands and grafts did not yield satisfactory results due to complications such as erosions and strictures. In addition, frequent weight regain due to gradual dilatation of the proximal pouch was also reported [29]. Hallberg and Forsell [30] in 1985 and Kuzmak [31] in 1986 reported their experience in the use of adjustable gastric bandings in clinical settings. In this technique the upper stomach was encircled with a silicone ring, which was lined with a balloon on its inner surface, the volume of which could be adjusted by adding or removing saline through an accessible subcutaneous port. The adjustable bands soon became more popular compared to their non-adjustable analogues. The main complications included band slippage and upward gastric prolapse, causing either gastric obstruction or proximal gastric pouch dilatation, band erosion and esophageal dilatation. Although technically safer and less demanding, gastric banding did not achieve the outcomes seen with other bariatric procedures. Excess weight loss averaged around 50%, but in general took at least 2 years to achieve and reoperation rates approached 5% per year [32].

13.2.6 Biliopancreatic Diversion

In the late 1970s, Scopinaro et al. [33] considered the use of combined restrictive and malabsorptive procedures in order to achieve maximum sustainable weight loss and carried out the first biliopancreatic diversion (BPD) surgery, in Genoa, Italy. While the restrictive component of this procedure was thought to be responsible for enhanced initial weight loss, the malabsorptive component was believed to be important in its long-term maintenance. BPD involved a partial distal gastrectomy and closure of the duodenal stump, followed by a gastroenterostomy with an enteric limb (Roux limb) and a biliopancreatic limb (Y limb) anastomosed to the Roux limb proximal to the ileocecal valve [25]. The major advantages included diversion of biliopancreatic juice from the bypassed limb, preventing bypass enteritis, and rapid food transit which greatly reduced caloric and nutrient absorption. BPD proved to be highly effective and the weight loss results have been the best among bariatric procedures, with 70% long-term weight loss in more than 90% of patients [34]. Like any other malabsorptive procedure, flatulence and voluminous foul-smelling stools, deficiencies of minerals, vitamins and proteins were frequently seen. Patients could also suffer from post-gastrectomy syndrome with marginal ulcers and dumping syndrome.

13.2.7 Duodenal Switch

About a decade later, in the late 1980s, Hess and Marceau proposed a variation of BPD, named duodenal switch (DS), aiming to increase the amount of gastric restriction, prevent marginal ulcers, minimize the incidence of dumping syndrome and the severity of protein-caloric malnutrition [35]. The surgery involved resection of the greater curvature of the stomach leaving a hollow gastric remnant with preservation of the pylorus. The enteric (Roux) limb was anastomosed to the post-pyloric duodenum; a long duodenobiliopancreatic limb (Y limb) was anastomosed to the Roux limb proximal to the ileocecal valve [25]. Although DS was a highly effective bariatric surgery, due to the high risk of complications it was reserved predominantly for super obese patients, especially those with a BMI >55 [36]. Despite the lower incidence of post-gastrectomy syndrome, other associated complications were similar to those of BPD. The risk of anatomic complications was higher because of multiple anastomoses and included bowel obstruction, internal hernia, anastomotic leak, fistulas, and abscesses [37].

13.2.8 Sleeve Gastrectomy

The lower rate of malabsorptive complications associated with restrictive bariatric procedures as well as the introduction of novel surgical equipment such as energy-based cutting devices and mechanical staplers aided in the development of one of most popular bariatric surgical procedures: sleeve gastrectomy (SG). This procedure was first performed as a part of the DS procedure in late 1980s. In 1993, Johnston

et al. [38] sought to develop a simple procedure as an alternative to adjustable gastric banding and other stapling procedures, which did not involve the use of implanted foreign materials such as bands and reservoirs. This operation, traditionally known as Magenstrasse ("stomach road") and Mill procedure, consisted of a circularly stapled hole just beyond the incisura angularis with the creation of a hollow tube by stapling just lateral and parallel to the lesser curvature from the circular hole up to the angle of His. In the following years, modifications were applied to the original procedure to further simplify the technique. The step consisting of creation of the circular hole was completely eliminated, by starting the staple line on the greater curvature, 5–6 cm proximal to the pylorus, stapling up to and along a 32–40 French bougie resecting the lateral 80% of the gastric body, leaving a narrow sleeve-like tube.

SG as a standalone procedure has become highly popular in twenty-first century and is today one of the most common bariatric surgeries performed worldwide. The intact gastrointestinal tract, except for reduced gastric volume, significantly reduces the risk of nutritional deficiencies. It is technically less challenging compared to GB or BPD/DS and is associated with minimal morbidity and long-term adverse effects such as dumping syndrome, marginal ulcers, internal hernias, and malabsorptive problems. Most commonly observed complications with this procedure include staple line leaks usually occurring at angle of His and strictures usually occurring at the incisura angularis. Overall excess weight loss is reported to be about 55% in 5 years [39].

13.3 Laparoscopic Revolution in Bariatric Surgery

The application of laparoscopy in bariatric surgery occurred for the first time in Australia, in 1992, where Broadbent [40] successfully placed a non-adjustable gastric band in a patient. A year later, in Belgium, Belachew [41] first performed an adjustable gastric banding laparoscopically. Laparoscopic adjustable gastric banding became extremely popular in the following years. However, technical problems with stomach slippage through the band and pouch dilation, as well as reported disappointing long-term results and high reoperation rates, eventually led to abandonment of this technique in many countries. In 1993, the first laparoscopic vertical banded gastroplasty (VBG) was performed by Hess and Hess [42, 43] in Ohio, United States. Patients who underwent laparoscopic VBG, demonstrated a postoperative course associated with less pain, earlier mobilization, and an improved respiratory function compared to patients operated on with an open VBG [44–46]. However, once very popular, VBG is not frequently performed anymore due to a prominent degree of weight regain and high rates of revisional surgery [47, 48].

Wittgrove and Clark [49] performed the first laparoscopic Roux-en-Y gastric bypass (RYGB) in 1994. The initial results of laparoscopic RYGB, in terms of leakage rate and overall complications, were outstanding, suggestive of the safety and feasibility of the laparoscopic approach. Although laparoscopic RYGB soon became the "gold standard" in bariatric surgery, it was still technically challenging. In 1997, Rutledge [50] proposed a simplified version of RYGB; the mini-gastric bypass (MGB), also known as omega-loop or single-anastomosis gastric bypass. This technique involved preparation of a longer gastric pouch, division of jejunum

150–200 cm distal to the ligament of Treitz, which was then used in the creation of an antecolic loop gastrojejunostomy. For several years, MGB was criticized and believed to increase the risk of gastric and esophageal malignancy due to associated biliary reflux and marginal ulcerations [51, 52]. However, increasing experience with this technique has reduced the concerns and in the last decade MGB has gained wide acceptance among many surgeons [53, 54]. The reported results are even superior to RYGB, due to a longer biliary limb and a higher degree of malabsorption [55, 56]. In 1998, Ren et al. [57] performed and published the first series of laparoscopic DS, a technically demanding procedure reserved only for laparoscopic experts and superobese patients. In 2003, to overcome technical difficulties, a two-stage procedure was suggested with the idea to transform first the stomach into a gastric sleeve, inducing an initial 40–50 kg weight loss, thus facilitating the further steps in the second operation [58]. Many patients satisfied by their initial weight loss results never returned for the second stage of the surgery, leading to development of laparoscopic SGs as an independent bariatric procedure [59]. Today, thanks to continuous technological advancements, laparoscopic surgery is the first choice for most patients and surgeons, regardless of the type of procedure.

13.4 Revisional Surgery

Although primary bariatric surgery is safe and effective for weight loss, a small number of patients may require a second operation because of complications or insufficient weight loss or weight recidivism consequent to nutritional, psychological or surgery-related factors. Revisional procedures can be differentiated into three categories:

- *Reversals* Taking down the original operation and restoring the normal gastrointestinal anatomy. These include simpler procedures such as removing a gastric band and more complex ones such as reversing a gastric bypass or a malabsorptive procedure.
- *Conversions* Switching one operation for another, such as removing a gastric band and then performing a gastric bypass or sleeve gastrectomy. Another example would be to convert a sleeve gastrectomy to a gastric bypass.
- *Revisions* Modifying or repairing an operation that has an abnormality such as repositioning a gastric band after a slippage or reshaping a gastric bypass pouch or a sleeve gastrectomy to correct a dilatation.

The most successful conversion strategy relies on selecting the most appropriate revisional procedure and involving a multidisciplinary team approach to the patient.

13.5 Latest Trends in Bariatric Surgery

With the continuous rise in the prevalence of obesity, the field of bariatric surgery is witnessing an ever-increasing demand. Surgeons have been continuously seeking

for an ideal weight loss procedure, with desirable safety, that is efficacious and yields consistent results. Despite the successful revolutionary role of laparoscopy in bariatric surgery, surgeons are still looking for methods to improve patient outcomes with safer surgical techniques with lower rate of complications. Two relatively new approaches, single incision laparoscopic surgery (SILS) and robotic-assisted laparoscopy, have been developed and are still under progress. In SILS, a multi-channel trocar is placed through the abdominal wall to reduce the number of inserted trocars, which yield better cosmetic results and postoperative pain. However, operative time is, so far, similar or even longer compared with traditional laparoscopy [60].

The first case of robot-assisted laparoscopic surgery was reported by Cadiere et al. [61] in 1999. Adjustable gastric banding was the first bariatric procedure performed using a robot. Robotic surgery has provided many advantages to the surgeons such as greater three-dimensional visualization, enhanced dexterity and increased precision [62, 63]. The main limitation of robotic surgery is the perceived higher cost and set-up time compared with conservative laparoscopy. Despite the advent and integration of novel technologies in robotics like fluorescence, integration of images, virtual and augmented reality, single site platforms, and haptic feedback, the utilization of robotics in bariatric surgery is still not widely disseminated.

13.6 New Procedures

- *Laparoscopic gastric greater curvature plication* could be traced back to 1980s, when Tretbar suggested gastric plication as an extension of fundoplication for the treatment of obesity [64]. However, the latest technique of this procedure was proposed by Talebpour et al. [65] and has gained popularity in some centers. The procedure decreases the volume of the stomach as the greater curvature is infolded and fixated with one or more rows of stitches.
- *Single-anastomosis duodeno-ileal bypass with sleeve gastrectomy (SADI-S)/ stomach intestinal pylorus sparing surgery (SIPS) single anastomosis* was introduced in 2007 by Sánchez-Pernaute et al. [66, 67]. SADI-S/SIPS compared with DS eliminates the Roux-en-Y gastric bypass by creating an omega loop by means of a duodeno-ileal anastomosis. According to patient characteristics, it can be performed either with a narrow gastric pouch and a long common channel (300 or 350 cm) or it can simply remain a malabsorptive procedure with a short common channel (200 or 250 cm) and a wider gastric pouch.
- *Sleeve ileal (SASI) bypass* is based on a mini gastric bypass operation and Santoro's operation in which a sleeve gastrectomy is followed by a side-to-side gastro-ileal anastomosis [68].
- *Single anastomosis gastro-ileal bypass (SAGI)* is very closely related to OAGB, with the difference that the gastro-enteric anastomosis is performed at 300 cm proximal to the ileocecal valve and no longer 180–250 cm distal to the angle of Treitz [69].

- *Other procedures*: gastric transit bipartitions, mainly performed in Bolivia and Turkey; SG with jejunal bypass (SG + JJB), mainly performed in China.
- *Endoscopic bariatric therapies* are a new addition to the treatment of obesity. These include devices that are placed and removed via flexible endoscopy, and procedures that utilize instruments that require flexible endoscopy for the indications of weight loss. They include space-occupying devices such as intragastric balloons, devices that allow performing gastric remodeling procedures [70–73] such as endoscopic sleeve gastroplasty or aspiration therapy with the AspireAssist system [74, 75].

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Bariatric Surgery Complications in the Emergency Department

14

Giuseppe Maria Marinari

14.1 Introduction

Obesity is an increasingly widespread pathology worldwide, but despite the pandemic the only effective therapy until now is surgery [1, 2]. Accordingly, each year an increasing percentage of the population becomes former bariatric patients, so that the number of complications in previous bariatric surgery cases is rising [3]. The first bariatric procedures were performed in the 1950s, but until the late 1980s the use of surgery for obesity was confined to a few centers. The great diffusion of bariatric surgery corresponded with the spread of laparoscopic surgery (1990s). The total number of bariatric/metabolic procedures performed in 2016 was 685,874 in the 54 nations participating in the International Federation for Surgery of Obesity [4]. In France the estimate for 2017 was that more than 450,000 patients would undergo bariatric surgery and, given the number of interventions per year, it is expected that in 2021 about 1% of the French population will have a bariatric procedure [5]. In Italy, between 2008 and 2017 about 0.15% of the entire population underwent obesity surgery [6]. Because of its young age, bariatric surgery has long been considered a minor surgery, and therefore many surgeons are not trained in the management of its complications [7].

Surgical complications that require emergency department (ED) visits may be perioperative or long-term. Perioperative surgical complications are in most cases those typical of abdominal surgery, and therefore either bleeding or septic complications. While the former is easy to diagnose, the latter can be particularly insidious in the obese subject, since the clinical signs can be atypical and difficult to interpret, often resulting in delayed management. Respiratory signs can be predominant and lead erroneously to pulmonary or thromboembolic disease [8].

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Along with the perioperative complications there are complications that occur years after surgery, which include internal hernias (perhaps the most insidious and difficult to diagnose), intestinal obstructions, perforated ulcers, slippage or stenosis of the gastric band (a complication with severity ranging from mild to catastrophic). These possible long-term complications usually arise acutely and do not always allow the patient to contact the center where the procedure was performed [3, 9]; also, the risk of these complications persists for a lifetime. The combination of the three factors mentioned above—i.e., the increasing number of people undergoing bariatric surgery, the non-decreasing risk of long-term complications, the not easy access to the hospital where the bariatric procedure was first performed—results in a greater number of potential patients seeking acute assistance from non-bariatric surgeons [3].

14.2 Frequency of Emergency Department Visits

The morbidity of the three most commonly performed procedures is 5.4% for gastric banding, 6.5% for sleeve gastrectomy, and 9.7% for gastric bypass. Considering that the immediate postoperative morbidity for sleeve gastrectomy and gastric bypass is approximately 2–3%, and much less (<1%) for gastric banding, we should expect that at least 5% of bariatric patients will have a complication at home [3]. However, the rate of ED visits for different symptoms is much higher, from 11 to 31.9% [9–12], and 16 to 35% of these ED visits will become readmissions [9–11]. Twenty-five percent of ED visits after bariatric surgery take place in the first 2 years after the surgery [12] and the rate of ED visits that will turn into readmissions is higher in the first 30 postoperative days [11]. The 30-day visits are more frequent after gastric bypass (48.7% of the total), in 39.5% of cases they follow sleeve gastrectomy and in 11.8% of cases they occur after gastric banding [11].

It should be emphasized that about 50% of ED visits occur in hospitals other than the one where the bariatric procedure was performed, and the same applies to the readmissions [9, 11]. This figure is partly justified by health tourism but also by the patient's perception of urgency: in a survey carried out in Michigan on the ED visits in other hospitals after bariatric surgery, the patients stated that they did not call the surgical team that performed the procedure in 36% of cases because "they felt that the situation was urgent", in 17.5% "because it was night", in 15% of cases "because they had not thought" [10].

14.3 Presentation Symptoms

Listed in Table 14.1 we report the most frequent symptoms and causes that lead the operated bariatric patient to seek treatment in the ED [10, 12]; these symptoms may occur alone or more often together. They are mostly non-specific symptoms, and only the combination of two or more of them can help the physician in the process of diagnosis and treatment. As previously reported, attention is drawn to the fact that thoracic symptoms can be the first to lead the obese patient to seek ED care after a bariatric procedure.

Table 14.1 Main symptoms associated with ED visits after bariatric surgery

Symptoms	Rate (%)
Abdominal pain	24–46
Nausea/vomiting	25–38
Dehydration	31
Chest pain	12
Infection	9–11
Nervous system event	10
Respiratory event	6
Cardiac event	6

ED emergency department

Table 14.2 Most common diagnoses observed in ED after bariatric surgery by type of procedure

Type of procedure	Diagnosis	Rate (%)
Gastric banding	Esophageal/gastric perforation	0.3
	Related to access port and connector tubing	4–5
	Pouch dilatation and gastric banding slippage	2
	Gastric banding erosion	0.5–1
Sleeve gastrectomy	Leak	1–2
	Stricture	0.7–4
	Reflux disease	Up to 25
Gastric bypass	Leak	2
	Bowel obstruction and internal hernias	<1–20
	Ulcer	1–7

ED emergency department

14.4 Most Common Diagnoses

The diagnoses that are most frequently observed in the ED as a complication of bariatric surgery vary depending on the operation performed. The next chapters will address the topic in detail, but as an initial guide in Table 14.2 we report the most frequent complications that may prompt a visit [7]. Some of them may indicate life-saving surgery, without granting sufficient time to transfer the patient to a specialized bariatric center. Particularly, a sepsis sustained by perforation or leak, or a possible digestive tract ischemia subsequent to acute pouch dilation or bowel obstruction, usually do not allow much time between the diagnosis and the mandatory urgent operation.

14.5 Conclusions

The spread of bariatric surgery involves a growing number of patients who can present to the ED with a new abdominal anatomy. The complications of bariatric surgery may be both perioperative and long-term, and in cases of emergency the post-bariatric patient is often not directed to the center where the procedure was performed. The severity of possible complications varies depending on the case,

and there are situations in which postponing surgery may have catastrophic consequences.

General surgeons must be familiar with the new post-bariatric anatomy and its possible complications, because some of these require life-saving surgery in emergency conditions and do not allow the patient to be transferred to a bariatric center.

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Metabolic Complications After Bariatric Surgery: The False Acute Abdomen

15

Luca Busetto

15.1 Introduction

Bariatric surgery produces sustained long-term weight loss and reduces comorbidity burden and mortality in patients with severe obesity. Therefore, current international guidelines recommend bariatric surgery, according to body mass index (BMI) levels and associated obesity-related diseases [1, 2]. Bariatric surgery is in general safe and effective, but it can cause new clinical problems and is associated with specific diagnostic, preventive and therapeutic needs [3]. Eating habits need to adapt to the new gastrointestinal physiology and nutritional deficits may arise according to the type of bariatric procedure. Management of obesity-associated diseases needs to be modulated according to weight loss taking into account the possibility of changes in drug pharmacokinetics. Specific problems may arise in women during pregnancy, and the patients may experience some psychological difficulties in adapting to the profound changes in eating behavior and body image. Finally, weight regain can occur and should be prevented and managed [3]. This complex and new clinical scenario should be managed by experienced multidisciplinary teams at least in the first phases of the long-term follow-up [3].

Some of the medical and nutritional problems typical of the post-bariatric phase can give rise to acute manifestations and can be evaluated for the first time by the emergency health services, out of the specialized bariatric centers. In particular, some of them can present with acute abdominal symptoms that can mimic acute surgical problems ("false acute abdomen"). In this brief chapter, we will revise the most frequent nutritional and medical problems giving rise to acute abdominal symptoms in post-bariatric patients. Particular emphasis will be given to the

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elements of clinical history and presenting symptoms that can help in making a correct differential diagnosis, avoiding the need for costly, time-consuming and more invasive diagnostic tests and examinations.

15.2 Acute Abdomen as an Unusual Clinical Presentation of Thiamine Deficiency

The occurrence of vitamin and mineral deficiencies is one of the most common nutritional problems after bariatric surgery. Prevention, detection and treatment of these deficiencies represent cornerstones of long-term follow-up in post-bariatric patients [3]. The frequency and severity of vitamin and mineral deficiencies after bariatric surgery depends on the anatomical characteristics and mechanisms of action of the various bariatric procedures. Therefore, severe nutritional deficiencies are uncommon after purely gastric restrictive procedures not altering intestinal continuity and normal digestive processes, but more common after surgical procedures inducing some degree of malabsorption [4].

Several kinds of mineral and vitamin deficiencies have been described after bariatric surgery, including deficiencies of iron, vitamin B12, folic acid, calcium, fat soluble vitamins (A, E, D, K), zinc, copper, selenium, magnesium, potassium and vitamin B6 [3]. The clinical manifestations of these nutritional deficits tend to have a very gradual presentation, with subtle and mostly chronic symptoms, the only exception being thiamine (vitamin B1) deficiency.

The human body has a low storage capacity for thiamine and can be rapidly (2–3 weeks) depleted without regular and sufficient intake [5]. Typically, thiamine deficiency is encountered in alcoholic patients. However, there are several other clinical settings in which thiamine deficiency may develop and any short period of persistent vomiting impairing regular food intake can precipitate thiamine deficiency [6]. Therefore, symptomatic thiamine deficiency has been described after a few weeks of intractable vomiting after any bariatric procedure, usually as a consequence of mechanical problems such as stoma stenosis after gastric bypass [7], excessive band tightness or band slippage after gastric banding [8] and stomach edema with impaired nutrition after sleeve gastrectomy [9]. Cases of thiamine deficiencies have been reported also after biliopancreatic diversion [10].

The symptoms of acute thiamine deficiency are typically neurologic, with peripheral neuropathy or Wernicke's encephalopathy and Korsakoff's psychoses [6]. These neurologic symptoms are rapidly progressive and may cause permanent neurologic deficits. Neurologic manifestations are linked to anatomical changes in the brain (mostly congestion and hemorrhages) mainly localized in the periaqueductal grey matter, around the third and fourth ventricles, the mammillary bodies and medial thalamus, but extending also to other areas like the superior vermis, the pontine tegmentum, the posterior corpora quadrigemina, the hypothalamus and the cerebral cortex [5].

The diagnosis of Wernicke's encephalopathy is made according to Caine's criteria [11], with two of the following features required for diagnosis:

- history of dietary deficiencies;
- cognitive impairment;
- ocular abnormality;
- cerebellar dysfunctions.

Laboratory determination of thiamine levels is not required for diagnosis.

Gastrointestinal symptoms, such as anorexia, nausea, vomiting and pain in the abdomen, are often described in patients with thiamine deficiency [12] and a few patients may have predominantly gastrointestinal manifestations. Donnino coined the term "gastrointestinal beriberi" for such patients [13]. In some case, the abdominal pain progresses in severity and the clinical picture can raise a suspicion of a surgical acute abdomen, with patients undergoing urgent but unwarranted imaging studies [14]. The clinical history can be confused, and the diagnosis further complicated by the fact that most of the gastrointestinal symptoms, like anorexia, nausea and vomiting, are in these cases both risk factors for and manifestations of thiamine deficiency [14]. Therefore, abdominal symptoms could be both cause and effects in thiamine deficiency, forming a vicious circle where anorexia, nausea and vomiting cause thiamine deficiency and then will be aggravated because of the decreasing thiamine levels [14] (Fig. 15.1). It should be noted that in most cases the acute gastrointestinal symptoms caused by thiamine deficiency are usually accompanied by the typical neurologic manifestations. However, these neurologic symptoms may be very subtle at the beginning and they could be easily missed without a careful and complete neurological examination [14].

In the case of suspected thiamine deficiency, considering the fact that the neurologic symptoms are rapidly progressive and may cause permanent neurologic deficits, the current guidelines strongly suggest considering and starting thiamine supplementation in every bariatric patient suffering from persistent vomiting severe enough to interfere with regular nutrition, even in the absence or before obtaining confirmatory laboratory data [3]. Therefore, parenteral thiamine supplementation (50–100 mg/day) should promptly be considered and started. Oral supplementation

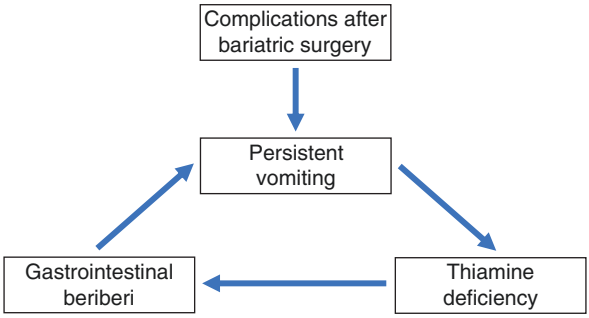

Fig. 15.1 The vicious circle of "gastrointestinal beriberi". Complications after bariatric surgery cause persistent and intractable vomiting precipitating thiamine deficiency; abdominal symptoms of thiamine deficiency (anorexia, nausea and vomiting) can further deteriorate thiamine status

may be used only after 1–2 weeks of parenteral administration (100 mg/day) and continued until the symptoms resolve [4].

In conclusion, a diagnosis of thiamine deficiency should be always considered in a post-bariatric patient presenting with abdominal pain after even a short period of intractable vomiting. A careful neurologic examination should be carried out in order to detect subtle and initial accompanying neurologic manifestations. However, thiamine supplementation should be started as quickly as possible even if only a suspicion of thiamine deficiency is considered.

15.3 Abdominal Pain in the Context of Dumping Syndrome

Dumping syndrome refers to the post-prandial occurrence of a constellation of symptoms elicited by the rapid transit of calorie-dense food to the small bowel. The symptoms may be non-specific, but the most typical manifestations are a syncope or pre-syncopal symptoms accompanied by abdominal discomfort (nausea, abdominal fullness, meteorism, borborygmus). The diagnosis can be facilitated by the application of the Sigstad score, a diagnostic tool based on weighing factors assigned to symptoms of the syndrome: a score index higher than seven points is suggestive of dumping (Table 15.1) [15]. The presence of vomiting does not support a diagnosis of dumping. Dumping syndrome was believed to be typical of gastric bypass (70–75% of patients in the first year after surgery) [4], but it has been described also after sleeve gastrectomy (40% of patients 6 months after surgery) [16].

Dumping was classically attributed to a rapid increase in the osmolality of the intestinal content, with influx of fluid into the intestinal lumen, intestinal distention,

Table 15.1 Dumping symptoms according to the Sigstad scoring system [15]

Symptoms	Score[a]
Shock	+5
Fainting, syncope, unconsciousness	+4
Desire to lie or sit down	+4
Breathlessness, dyspnea	+3
Weakness, exhaustion	+3
Sleepiness, drowsiness, apathy, falling asleep	+3
Palpitation	+3
Restlessness	+2
Dizziness	+2
Headaches	+1
Feeling of warmth, sweating, pallor, clammy skin	+1
Nausea	+1
Abdominal fullness, meteorism	+2
Borborygmus	+1
Eructation	−1
Vomiting	−4

[a]A score ≥7 is suggestive of dumping

fluid sequestration, decreased intravascular volume and hypotension [17], but the increase in the secretion of glucagon-like peptide 1 (GLP-1) described after bariatric surgery probably also plays a role [18]. Dumping syndrome can be classified as early or late, depending on how soon after food ingestion symptoms occur: early symptoms occur about 10–30 min after meal, whereas late symptoms occur 1–3 h after food ingestion [19]. Late dumping is strictly related to the occurrence of reactive hypoglycemia and may be more linked to changes in gastrointestinal hormones and insulin secretion [20].

Dumping syndrome is usually a recurrent phenomenon and the episodes can be usually elicited in the same patient by the ingestion of specific foods. Episodes of dumping can be prevented by appropriate nutritional counselling: eating small but frequent meals, avoiding ingestion of liquids within 30 min of a solid-food meal, avoiding simple sugars, increasing intake of fiber and complex carbohydrates and increasing protein intake [4]. Late symptoms and reactive hypoglycemia may be also prevented by taking a small amount of sugar about 1 h after eating [4]. Pharmacologic management or redo surgery are only required in a very small minority of patients [3].

In summary, at the level of emergency department, dumping syndrome should be suspected in any post-bariatric patient evaluated for syncope or pre-syncope occurring after meals with or without accompanying abdominal symptoms. In cases of late dumping, the documentation of low glucose blood levels is very important for diagnosis.

15.4 Conclusions

Some of the nutritional problems typical of the post-bariatric patients, thiamine deficiency and dumping syndrome in particular, could manifest with acute abdominal symptoms and are first evaluated by the emergency medical services. Knowledge of these problems and attention to the clinical history and presenting symptoms can help in making a correct differential diagnosis, avoiding the need for costly, time-consuming and more invasive diagnostic tests and examinations.

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Complications of Intragastric Balloons

16

Alfredo Genco, Stefano Cariani, and Ilaria Ernesti

16.1 Introduction

The American Society for Gastrointestinal Endoscopy (ASGE) and the American Society for Metabolic and Bariatric Surgery (ASMBS) have defined acceptable thresholds of safety and efficacy for primary endoscopic bariatric therapies (EBTs). Specifically, a given EBT should have an incidence of serious adverse events ≤5% and should result in ≥25% excessive weight loss (EWL) at 12 months, and this EWL should be ≥15% higher than in a control group [1].

In recent decades, several intragastric balloons (IGBs) have demonstrated safety and efficacy, with broad adoption internationally. The U.S. Food and Drug Administration (FDA) has approved the Orbera Intragastric Balloon System (Apollo Endosurgery, Inc., Austin, TX, USA), the ReShape Integrated Dual Balloon System (ReShape Medical, Inc., San Clemente, CA, USA) and, more recently, the Obalon (Obalon Therapeutics, Inc. Carlsbad, CA, USA). The Spatz Adjustable balloon (Spatz Medical, Great Neck, NY, USA) is currently conducting its US pivotal trial. The Elipse Balloon (Allurion Technologies, Natick, MA, USA) has been proven to

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Table 16.1 Features of the FDA-approved IGBs and other balloons [2–5]

IGB	Orbera [2]	ReShape [2]	Obalon [2]	Elipse [3, 4]	Spatz [5]
FDA approval	2015	2015	2016	No	No
Delivery/ insertion	Endoscopy needed	Endoscopy needed	Patient swallows, X-ray	Endoscopy not needed	Endoscopy needed
Removal	Endoscopy needed	Endoscopy needed	Endoscopy needed	Endoscopy not needed	Endoscopy needed
Capacity	500–750 cc (1 balloon)	450 cc/balloon for a total of 900 cc (double balloon system)	Gas-filled balloon with a max volume of 250 cc (up to 3 balloons can be placed: 750 cc)	550 cc (1 balloon)	400–800 cc
Implantation period	Up to 6 months	Up to 6 months	Up to 6 months	16 weeks	Up to 12 months
Weight loss	29% EWL at 12 months	25% EWL at 12 months	25.2% EWL at 12 months	39 and 26% EWL at 16 weeks	34.4% EWL at 6 months

IGB intragastric balloon, *EWL* excess weight loss

be a safe and effective device in European clinical studies and the clinical study for FDA approval is still in process.

EBTs are mostly represented by intragastric balloons and the balloons we are addressing in this chapter have either been approved or are in the process of being approved by the FDA. Table 16.1 summarizes the main characteristics of each balloon [2–5].

The number of adverse events associated with IGB insertion varies across studies [6]. The most commonly reported symptoms after IGBs placement are accommodative in nature, such as abdominal pain, nausea, and vomiting. These generally tend to last only for few days after balloon insertion and are usually self-limiting. Serious adverse events described after IGB placement include dehydration, gastrointestinal ulceration, dislocation of the balloon causing intestinal obstruction, and perforation especially during balloon insertion or removal.

16.2 Orbera

The Orbera Intragastric Balloon System is the most commonly used IGB, approved for use in Europe in 1997. Clinical device surveillance based on reports from European practitioners between 2006 and 2013 revealed 3316 unspecified events/complaints representing 2.1% of 154,955 procedures. The FDA approved the use of Orbera in the USA in August 2015 on the basis of results of the Orbera FDA pivotal clinical trial and two non-US clinical trials in Australia and France [6].

The rates of adverse events after implantation of the Orbera balloon are pooled from a manual review of 67 studies (8500 implantations). Abdominal pain and nausea are frequent side effects after Orbera balloon implantation, occurring in 33.7% and

29.0% of subjects, respectively. Other rates of adverse events observed with the Orbera are: gastroesophageal disease (13.3%), erosion (12%), ulcer (2%), migration (1.4%), small bowel obstruction (0.3%), perforation (0.1%) and death (0.08%) [7]. Medications such as proton pump inhibitors, antispasmodic drugs and antiemetics are usually prescribed few weeks before, during, and after balloon placement to prevent or minimize these expected common side effects. The early removal rate of the Orbera balloon was required in 7.5% subjects. Case reports have been published about asymptomatic microbial colonization of the Orbera, though no clinical significance was noted [8, 9]. Serious side-effects with the Orbera balloon are rare with an incidence of migration and gastric perforation of 1.4% and 0.1%, respectively. Most of the reported perforations with the Orbera were in patients who had undergone previous gastric surgeries. Four deaths associated with the Orbera IGB are reported in the literature, and these were either related to gastric perforation or an aspiration event [10].

16.3 ReShape

The ReShape Duo has a favorable adverse events profile. In the pivotal Reduce US ReShape trial, which evaluated the safety and efficacy of the ReShape Duo IGB in 264 patients, abdominal pain and nausea were common symptoms and were successfully managed medically. Early retrieval was necessary in 15% of patients. Spontaneous balloon deflation occurred in 6% of subjects without balloon migration. Gastric ulcers and erosions were frequent adverse events, initially observed in 39.6% of the study subjects. However, a subsequent device design modification led to decreases in both ulcer frequency (reduced to 10.3%) and in ulcer size (from 1.6 to 0.8 cm). Most of the reported ulcers were not clinically significant, except for one ulcer-related upper gastrointestinal hemorrhage requiring blood transfusion. There were no reported deaths, balloon migrations, or intestinal obstructions. Three serious adverse events were observed with ReShape Duo retrieval, including an esophageal mucosal tear requiring hemoclip application, a contained cervical esophagus perforation managed conservatively with antibiotics, and one post-retrieval aspiration pneumonitis [11].

16.4 Obalon

In a pivotal multi-center randomized blinded clinical trial (SMART study) conducted in the US, 185 patients underwent a combination of lifestyle modifications in addition to the Obalon system, while 181 patients underwent lifestyle modifications with a sham placement procedure. All balloons were removed 24 weeks after insertion. The most common adverse effects reported in patients using the Obalon system were abdominal pain (72.6%), nausea (56.0%), vomiting (17.3%), indigestion or heartburn (16.9%), and bloating (14.6%). Most of these effects were mild in severity and resolved within 14 days. The Obalon system did not report any deflations. Early device removal due to adverse effects occurred in 3.0% of patients. Gastric, esophageal, and esophagogastric bleeding and abrasion, procedure-related

adverse effects identified at balloon removal, occurred in 5.1%, 4.2%, and 3.6% of patients, respectively. One case of bleeding gastric ulcers was seen in 0.3% [12]. Nobili et al. evaluated the effectiveness of the Obalon System as treatment of 17 morbidly obese children. Excess weight was calculated according to Cole's curves for pediatric populations. Fourteen of 15 patients (93.3%) swallowed the first balloon simply and quickly. In two patients endoscopy was planned due to slight mental retardation. In 9 of 17 children enrolled, a second balloon was placed 30–40 days after the first insertion. All devices were endoscopically removed after a mean time of 18 weeks. In the 16 patients who completed the study, the mean weight decreased from 95.8 ± 18.4 to 83.6 ± 27.1 kg ($p < 0.05$), mean BMI decreased from 35.27 ± 5.89 to 32.25 ± 7.1 ($p > 0.05$); with an %EWL of 20.1 ± 9.8 (range 2.3–35.1). As regards side effects, 5 of 18 patients reported mild to moderate epigastric pain/cramping that completely disappeared after few days, using a single dose of oral hyoscine butylbromide. Nausea, recorded in five patients, resolved spontaneously after 1 day (4 cases) to 2 days (1 case) without medication. In the group of nine children who underwent a second balloon positioning, side effects were even less common [13].

16.5 Elipse

The Elipse device (Allurion Technologies, Wellesley, Massachusetts, USA) is a procedureless, swallowable gastric balloon that can be deployed without the use of endoscopy or anesthesia. It is filled with 550 mL water via a catheter, which is then detached, and remains in the stomach for approximately 4 months before it empties and passes through the gastrointestinal tract. The Elipse IGB was approved for the European Union in December 2015. In a systematic review, two studies with Elipse placement reported nausea in 21 out of 42 patients with a meta-analytic rate of 51.42% (95% CI 46.00–57.00%) and vomiting in 23 out of 42 patients with rate of 12.48% (95% CI 8.51–16.44%) [14]. A prospective, observational, open-label, multicenter study demonstrated clinically significant weight loss with the Elipse: the mean percent total body weight loss, BMI point reduction, and waist circumference reduction were 10.0 ± 6.6%, 3.9 kg/m^2 and 8.4 cm respectively, at 16-week follow-up. There were no serious adverse events or serious adverse device effects. Among accommodative symptoms, 18 participants (64%) had vomiting, 15 participants (54%) experienced nausea, and 7 participants (25%) had abdominal pain. In particular, the rate of obstruction incidents ranged from 0.8 to 0.1% after device improvements (new release-valve closure) [14].

16.6 Spatz

The Spatz Adjustable Balloon System (Spatz Medical, Great Neck, NY, USA) is an endoscopically placed IGB that is filled with saline solution. It has an extractable inflation tube that allows for volume adjustment while the IGB remains in the stomach. The balloon volume may be decreased to improve patient tolerance or increased to enhance efficacy. Outside the United States, the Spatz IGB is approved for up to a

12-month implantation. A pivotal multicenter US trial currently is underway. Earlier generations of the Spatz Adjustable Balloon System had a non-collapsible loop with an internal metal chain that maintained a 7-cm balloon diameter within the gastric lumen to prevent or delay a deflated balloon from migrating. This design has been implicated in a higher incidence of migration complicated by balloon impaction, necessitating surgical removal [15]. The Spatz 3 balloon has been modified with removal of the metal chain and stiff catheter, thereby mitigating these unwanted effects. Recently, implantations of Spatz3 in 165 consecutive patients in two centers were retrospectively reviewed. The mean weight loss was 16.3 kg and 67.4% EWL. Down adjustments alleviated early intolerance in 80% of patients. One gastric perforation (0.6%) occurred in a patient who experienced abdominal pain for 2 weeks. Five patients with small ulcers did not require balloon extraction [16].

16.7 Potential Risks with Liquid-Filled Intragastric Balloons

Since 2016, the FDA has received reports of a total of 12 deaths that occurred in patients with liquid-filled intragastric balloon systems worldwide. Seven of these 12 deaths were patients in the U.S. (four with the Orbera Intragastric Balloon System, and three with the ReShape Integrated Dual Balloon System). The FDA, however, has also stated that the "root cause" of these case fatalities is not known, as the evidence only depicts a 1-month or less temporal relationship between balloon placement and death. It was thus uncertain if the cause of death was gastric or esophageal perforation, intestinal obstruction, or through an alternate means. On February 2017 the FDA warned medical providers about the potential risks of fluid-filled balloons after receiving several dozen reports of IGB hyperinflation (reported as "overinflation"), with air or fluid in the stomach, resulting in device removal as early as 9 days following insertion. Symptoms of hyperinflation included intense abdominal pain, abdominal distension with or without discomfort, difficulty breathing, and/or vomiting. The cause of hyperinflation was cited as unknown by the FDA. Due to incidents of hyperinflation of saline-filled silicone breast implants, IGB permeability may have resulted in fluid or air entry by osmosis. Another possibility with regard to air is that anaerobic bacteria, which have been identified in breast implants, may also have been present in IGBs and released gas within the balloon. The FDA also received several reports of acute pancreatitis associated with the Orbera and ReShape, resulting in early device removal as well as hospitalization [17].

16.8 Conclusions

To conclude, the use of intragastric balloon is now a widespread procedure all over the world both as a bridge to any surgery or to control comorbidities in patients with lower BMI no longer able to control the obese-related disease with diet alone. It is very important for any physician to know very well all the possible complications of intragastric balloon treatment in order to manage them properly and above all to prevent the complications.

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Complications of Restrictive Procedures

17

Mirto Foletto, Alice Albanese, and Luca Prevedello

17.1 Vertical Banded Gastroplasty (VBG)

Restriction is achieved by creating a vertical gastric pouch of 20–30 mL either by simply stapling the upper part of the stomach (Mason's technique) or by stapling and dividing the stomach (Mac Lean's technique) along the lesser curvature. A "de novo" pylorus is then fashioned with a synthetic ring. This procedure is nowadays abandoned due to patient intolerance, weight regain and the high rate of complications [1]:

- ***Staple line rupture and gastro-gastric leak*** Frequent vomiting due to overeating or staple line disruption due to uncomplete stomach division (Mason's gastroplasty) can lead to this complication with following loss of restriction;
- ***Dilation of gastric pouch*** Overeating and augmented pressure in the pouch can lead to dilation and impair gastric emptying with vomiting. It is more frequent in Mac Lean's procedure.
- ***Stenosis*** The ring can induce a fibrotic reaction of tissues finally leading to gastric stenosis and vomiting.
- ***Gastric erosion*** The ring can erode through the gastric wall and penetrate into the lumen.
- ***Gastroesophageal reflux*** Symptoms are related with stenosis and dilation of the gastric pouch.

The most suitable option for converting a VBG is to fashion a Roux-en-Y gastric bypass (RYGB), by performing a gastric transection and gastrojejunal anastomosis above the ring.

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17.2 Adjustable Gastric Banding (AGB)

Restriction is achieved by a band placed just below the gastroesophageal junction, thus creating a gastric pouch of approximately 20–50 mL. The band is connected with a reservoir placed in the subcutaneous tissue usually in the upper left quadrant. The injection of saline solution into the port determines the adjustment of the gastric outlet through the band.

Complications after AGB are [2–5]:

- ***Band slippage with outlet obstruction*** This is defined as a downward movement of the band. Its incidence varies among series, ranging from 1 to 22%. The "pars flaccida" technique seems to be related with low rates of slippage as compared to the perigastric approach. Band slippage can create a complete obstruction of the stomach and can be classified in:
  - Anterior slip or Type I prolapse: this is the result of the migration of the anterior wall of the stomach up through the band. Possible determinants are increased pouch pressures due to vomiting, overeating or early insufflation of the band (<4 weeks).
  - Posterior slip or Type II prolapse: this is the result of the herniation of the posterior wall of the stomach through the band. This is more often caused by surgical technique and more frequently associated with perigastric approach.
  - Type IV prolapse: this occurs immediately in the postoperative period and is usually due to a wrong and too low placement of the band around the stomach.
  - Type V prolapse: this is a complicated prolapse with necrosis of the slipped stomach.

    Dysphagia, vomiting, regurgitation and food intolerance are common symptoms of slippage. Diagnosis is made by upper gastrointestinal (GI) series. Upper GI series is the gold standard imaging technique to assess correct positioning of the banding (45° angle toward the spine) and integrity of the port-tube connection. Type I, II, IV and V are acute and always require surgical intervention (band removal or repositioning). Slippage can be complicated with gastric perforation, necrosis of the slipped stomach, upper GI bleeding and aspiration pneumonia.
- ***Pouch enlargement or Type III prolapse*** High intragastric pressure due to overeating or overinflation can enlarge the gastric pouch without changes in band positioning or signs of obstruction. The lower esophagus can be involved in the segmental dilation. Loss of satiety, heartburn, regurgitation and chest pain are common symptoms. The diagnosis is made with upper GI series. Non-operative treatment is the first choice with complete desufflation and liquid/soft diet. Upper GI series have to be repeated after 4–6 weeks. If the pouch size returns to normal and the band is still well positioned, the system can be re-adjusted. If the pouch does not return to normal size within 8–10 weeks after desufflation, surgical treatment is required (band removal or repositioning).
- ***Band erosion*** After a "pars flaccida" approach, the incidence is less than 1%. The band erodes through the gastric wall and penetrates the lumen. Causes are

gastric wall injury during positioning or too tight anterior fixation. Most patients are asymptomatic. Loss of restriction, epigastric pain, GI bleeding, intra-abdominal abscess and recurrent port-site infections are usually associated with erosion. The diagnosis is confirmed by endoscopy. Removal of the banding can be achieved by endoscopy, laparoscopy or a combined endoscopic/laparoscopic approach.

- ***Port-site infections*** Clinical signs are localized skin redness pain and swelling. Early postoperative infections are treated with oral antibiotics and IV antibiotics in non-responders. Surgical port removal is recommended in non-responders to oral and IV antibiotics. A new port with tube connecting can be placed after resolution of the local infection. Late infections manifest several months after surgery and typically do not respond well to antibiotics. They are often caused by delayed band erosion with ascending infection. Loss of restriction can be experienced. Band removal is required. If unrecognized, they can produce intra-abdominal abscesses.
- ***Port breakage*** Leakage can result from damage to the port septum (only non-coring needles should be used) or disconnection with the tube. The leakage can be suspected when injected fluid cannot be aspired and band does not respond to calibration. Leakage from the port, disconnections between the port and tube or rupture of the tube in the abdominal cavity can be documented by contrast injection of diluted nonionic iodinated contrast through the port during under fluoroscopy.

17.3 Sleeve Gastrectomy (SG)

SG is one of the most commonly performed bariatric procedures worldwide. Restriction is achieved by cutting out the stomach with the guide of an orogastric bougie (36–40 Fr) along the greater curvature. The most relevant reported complications are [6–9]:

- ***Staple line leak*** Incidence ranges from 0.3 through 7%, mainly depending on the volume of the Centre. It can be a life-threatening complication that requires a multimodal approach and surgical revision. When it occurs earlier (first 48 h after surgery), it may be due to misfiring or technical pitfalls. Otherwise, it can appear from 1 to 4 weeks after surgery due to ischemic conditions of the staple line, especially in the upper third. Surgical drainage is required in septic patients. CT guided drainage, endoscopic stenting or suturing are often adjuvant approaches in stable patients. Leaks can become chronic and require conversion to other procedures.
- ***Staple line bleeding*** Up to 4.9% of patients experience staple line bleeding. Less than 1% require surgical revision. Buttressing materials are safe and effective in high-risk patients (oral anticoagulant therapy, antiplatelet drugs).
- ***Stenosis*** This is a rare complication after SG (incidence less than 15%). Inadequate approximation of the anterior and posterior gastric wall before firing

is one of the major causes. Gastric stenosis is primarily approached through endoscopic dilations, but conversion to another procedure such as RYGB may be required.

- ***Functional disorders*** Twisting of the staple-line due to technical mistakes during firing can lead to dyspepsia, reflux and vomiting. Endoscopic treatment with stenting may be the first approach. For non-responders, conversion to other procedures is to be considered.
- ***Gastroesophageal reflux*** This is a possible underestimated long-term complication. Recent results advocate endoscopic surveillance after laparoscopic SG, due to the potential risk of erosive esophagitis and Barrett's esophagus.

17.4 Gastric Plication (GP)

Laparoscopic gastric plication was first described in 1976 and was later reintroduced and performed laparoscopically by Talebpour and Amoli in 2007 [10, 11]. Restriction is achieved by infolding and suturing (double row) the greater curvature of the stomach. Potential advantages were the avoidance of gastric resection and implantation of a foreign body, a final shape of the stomach apparently similar to SG. In 2011 the American Society for Metabolic and Bariatric Surgery (ASMBS) defined this procedure "investigational" [10–13].

Despite the great weight loss and low rate of complications reported by Talebpour, some authors reported a high rate of surgical revision, even in emergency settings. Gastric wall "pop-ups" in between the stitches maybe related to the atrophy of the infolded stomach with consequent loose suture lines, finally leading to surgical revision. In some patients it showed up as an acute complication with obstruction and ischemia of the gastric wall. In some others it led to reduced satiety and unsatisfactory weight loss.

Surgical revision is always required: fundectomy with the resection of the prolapsed stomach after dismantling the suture line may be mandatory in an acute setting if gastric blood supply is impaired (ischemia). Insufficient weight loss or weight regain due to loss of restriction require conversion to another bariatric procedure electively [14, 15].

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Upper Gastrointestinal Bleeding After Bariatric Surgery

18

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18.1 Introduction

Bariatric surgery is the gold standard for the treatment of morbid obesity and weight-related comorbidities and is far more effective than nonsurgical interventions [1, 2]. In recent years the number of bariatric procedures has constantly increased worldwide [3]. The introduction of laparoscopy, the advances in operative techniques and patient management have significantly improved the safety profile of bariatric surgery despite the high-risk nature of these patients [4, 5]. However, complications may occur, one of them being postoperative bleeding. The incidence of postoperative bleeding ranges from 1 to 4% [6], with an overall mortality rate <1% [5].

Most bleedings occur at the staple line or anastomosis and may be intraluminal or extraluminal (intra-abdominal). Clinical presentation is generally characterized by hypotension, tachycardia, anemia, hematemesis, and melena; however, its severity may range across all grades of the Clavien-Dindo classification of surgical complications [7]. If the bleeding occurs in the early postoperative period and is associated with hemodynamic instability, a laparoscopic or laparotomic reoperation

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is indicated. In hemodynamic stability, a conservative approach (administration of fluids and blood transfusions) and endoscopic treatment can be adopted.

18.2 Bleeding After Laparoscopy in Morbidly Obese Patients

Although laparoscopy is safe and effective in obese patients, intra-abdominal hemorrhages may occur from trocar sites due to the hypertrophy of the subcutaneous venous plexus [8]. Acute intra-abdominal bleeding can be also a consequence of vascular or parenchymal injuries caused by the Veress needle, which is often used to induce pneumoperitoneum in bariatric patients.

18.3 Bleeding After Sleeve Gastrectomy

Sleeve gastrectomy (SG) bleeding occurs in about 2% of cases, frequently within the first 24–48 h. More commonly, it originates from the staple line; other sites of bleeding are the gastroepiploic and the short gastric vessels, which are divided during stomach mobilization, and trocar accesses. Hepatic or splenic injuries may cause severe postoperative bleeding if not recognized and managed intraoperatively [9].

In cases of endoluminal bleeding, the clinical presentation is generally characterized by hematemesis or melena. Endoscopy is mandatory in order to identify the source of bleeding. The most frequent source is the staple-line, so careful inspection is warranted to treat it [10]. In early anastomotic bleeding, adrenaline injection or mechanical hemostasis by clips may be ineffective due to the presence of multiple bleeding foci, while the rate of success with thermal hemostasis (heater probe, argon plasma coagulation) seems to be higher. Nevertheless, some cases of diffuse ischemia or necrosis have been described. Also, the injection of sclerosing agents (such as ethanol or polidocanol) have been associated with a risk of perforation and necrosis, while injection of tissue adhesive (thrombin, fibrin, cyanoacrylate and specific synthetic peptides) proved to be equally effective, but safer [11–14]. The use of the new hemostatic powder has also been proposed, due to the possibility to easily treat a large surface area with a low rate of complications. However, these hemostatic sprays have been recently introduced into clinical practice and their efficacy and safety is still debated [15]. In the case of early postoperative bleeding, endoscopic procedures should be performed in the operating room with endotracheal intubation and CO_2 insufflation since perforation of fresh anastomoses has been described.

Intraperitoneal bleedings are diagnosed by computed tomography scans, which identify the site of bleeding and also estimate the amount of the hemoperitoneum [16] (Fig. 18.1). Patients with arterial hypertension or type 2 diabetes mellitus, or on anticoagulant therapy, showed a high risk of bleeding [7, 9]. On the contrary, routine reinforcement of the staple line regardless of type, has been demonstrated to significantly reduce the incidence of bleeding [17].

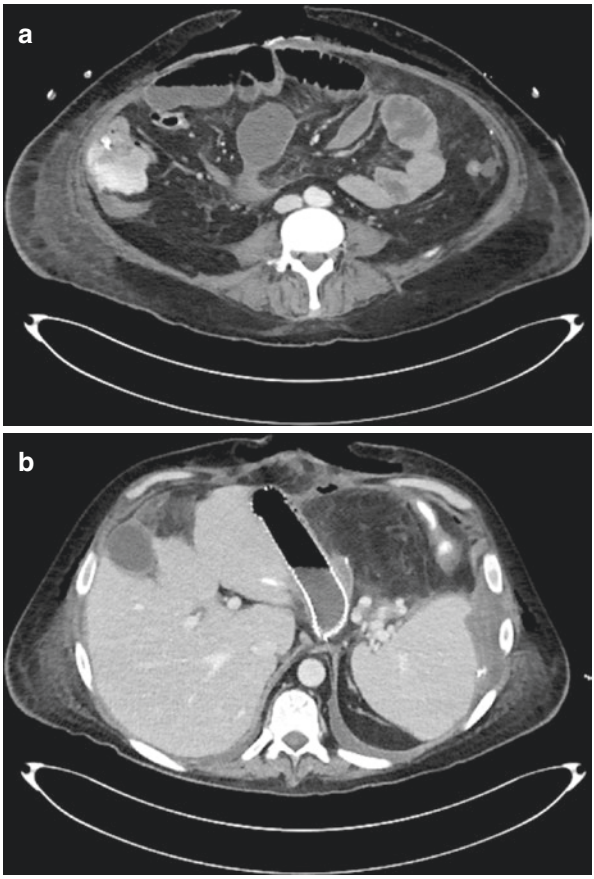

Fig. 18.1 CT scans showing bleeding from an abdominal collection (**a**) in a patient with a leak after sleeve gastrectomy, treated by external drainage and megastent (**b**)

18.4 Bleeding After Roux-en-Y Gastric Bypass

Bleeding rate after Roux-en-Y gastric bypass (RYGB) varied from 1 to 3.8% with an average of 1.93% [6]. The possible bleeding sites after RYGB are several. The most likely sites are the staple lines of the gastrojejunostomy, gastric pouch, excluded stomach, or jejunojejunostomy. The clinical presentation can point to the location of bleeding in some cases. Hematemesis most likely indicates bleeding from the gastric pouch or gastrojejunostomy. Melena most likely indicates more distal bleeding from the jejunojejunostomy or, alternatively, from the excluded stomach staple line [18]. Active hematemesis strongly suggests a proximal source of bleeding and is a reasonable indication for upper endoscopy. This will usually demonstrate the source of gastrointestinal (GI) bleeding if it is located in the esophagus, stomach pouch, or proximal jejunum. Endoscopic treatment for early anastomotic bleeding is similar to that of SG [19].

Several endoscopic techniques may be adopted for treating GI ulcers, and endoscopic treatment mainly depends on the size and location of the ulcer, evidence of active bleeding, device availability and endoscopist experience. Ulcer treatment in post-bariatric patients is not different from that in non-operated subjects, the most effective treatment for active bleeding ulcers (Forrest Ia and Ib) or high-risk ulcers (Forrest IIa) being local injection of diluted adrenaline for temporary vasoconstriction plus a second hemostasis modality (mechanical, thermal or topical). Mechanical hemostasis can be obtained by standard or over-the-scope clips, the goal of this technique being to close the mucosal-submucosal discontinuity by juxtaposing the margins. Although over-the-scope clips allow a deeper closure, they are more expensive and, sometimes, harder to introduce and release than standard clips, and the risk of tissue necrosis is higher (Fig. 18.2). Thermal therapy is performed by using contact and non-contact devices, the former include heater probes (bipolar electrocautery

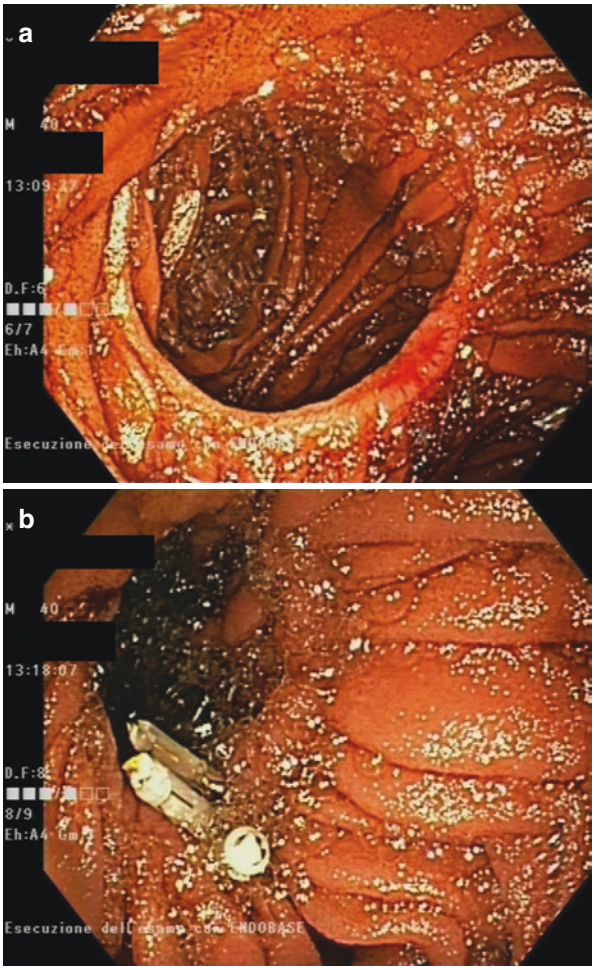

Fig. 18.2 Bleeding from a marginal ulcer in Roux-en-Y gastric bypass (**a**), treated with mechanical hemostasis (**b**)

probes), the latter argon plasma coagulation (APC) tools (high frequency monopolar alternating current conducted to the target tissue by ionized gas). The goal of these devices is to induce the indirect activation of the coagulation, causing edema, protein coagulation and vessel damage. Injection of sclerosing agents is rarely adopted, due to the risk of tissue necrosis, while the use of adhesive glue, like thrombin or synthetic peptides, is increasing due to the low risk of complications and easy application. Hemostatic spray (topical therapy) is actually considered a rescue therapy for treating difficult ulcers, although further studies are needed to clarify the role of these agents as first-line therapy. In the case of failure of endoscopy, transcatheter angiographic embolization (TAE) or surgery should be considered [20]. However, some authors have suggested endoscopic suturing as rescue therapy in non-healing or deep ulcer, and marginal ulcer bleeding not responding to conventional endoscopic therapy [21].

In patients affected by marginal or stomal ulcers as well as by erosive gastritis, *H. pylori* status should be evaluated, and abuse of non-steroidal anti-inflammatory drugs (NSAIDs) should also be investigated. Moreover, a pH test of gastric-pouch liquid may identify patients with poor acid suppression. In cases of intragastric pH >4, increasing proton pump inhibitor (PPI) therapy is unhelpful, while high doses of sucralfate (1 g 4 times daily) seems to improve ulcer healing [22].

In gastric bypass (GB) patients with suspected GI bleeding and negative esophagogastroduodenoscopy (EGD), the examination should be performed by enteroscope or pediatric colonoscope to evaluate jejuno-jejunal anastomosis and, eventually, the gastric remnant. Other imaging modalities such as bleeding scan or capsule endoscopy may be considered, as these will reveal those areas of the GI tract beyond the reach of the flexible endoscope. Balloon-assisted enteroscopy in acute bleeding could also be considered, but the risk of perforation is higher due to the traction on immature anastomoses, so it should be performed only by skilled endoscopists. The gastric remnant can be also achieved by laparoscopic transgastric endoscopy [19].

Delayed bleeding is more frequent in GB and is usually related to marginal ulcers (ulcer on the other site of anastomosis). Sometimes GB patients could have chronic anemia or intermittent melena that may be linked to recurrent ulcer. This clinical entity is often associated with a persistent *noxious stimulus* on the anastomosis, such as excessive acid production, tobacco use, non-steroidal anti-inflammatory usage or the presence of suture materials or staple. When irritation from suture material is present, the extraction of foreign bodies near the anastomosis is suggested also in the absence of an active ulcer. Another etiology of recurrent ulcers is a fistulous connection between the remnant and gastric cavity; hence in these patients a careful search for gastrogastric fistula, by both endoscopy and radiology, is recommended [19, 23–25].

18.5 Bleeding After Duodenal Switch Biliopancreatic Diversion

During the early stage of laparoscopy, postoperative bleeding was higher after laparoscopic duodenal switch than after open surgery (6–10% vs. 1%) [26–31]. However, later

papers have reported a lower hemorrhage rate: in a series of Buchwald et al. [32], the rate of postoperative bleeding that required re-exploration in the operating room was 1.6% (3/190 patients); Biertho et al. reported a rate of bleeding after one thousand cases of around 0.5% [33]. Bleeding can be intraluminal or intrabdominal. As in other bariatric procedures, hematemesis and melena are signs of an intraluminal hemorrhage. Intra-abdominal bleedings usually arise from the staple line or from the duodenoileostomy. Hemorrhages from the duodenal stump should be managed carefully due to the risk of corruption of the staple line and subsequent biliary leak.

18.6 Diagnosis and Treatment

Intraoperative bleedings are usually promptly recognized during the procedure and they may lead to conversion to open surgery. Symptoms of an acute postoperative hemorrhage are tachycardia, hypotension, oliguria and a progressive decrease in hemoglobin levels. Intra-abdominal bleeds could be suspected in the case of a large collection of blood from the drainage. In cases of intraluminal hemorrhages, the patient may experience hematemesis or melena. Intra-abdominal bleedings are diagnosed with laparoscopy, while intraluminal ones are diagnosed and often treated with endoscopy.

The risk of hemorrhages can be reduced by discontinuing anticoagulant therapy preoperatively, when possible, over-sewing the staple lines, using the appropriate cartridges and eventually staple line reinforcements. Intraoperative bleedings can be stopped by using harmonic and bipolar devices or with metallic clips. The vast majority of postoperative bleedings are usually managed conservatively with fluid infusion or blood transfusion. Vital signs should be constantly and regularly monitored together with hemoglobin levels and urine output. In the case of an unstable patient, endoscopic or surgical exploration is mandatory. If the site of hemorrhage is intraluminal, endoscopy allows one to place clips or inject epinephrine on the staple line, the anastomosis or marginal ulcer. Electrosurgery should be avoided due to possibility of thermal injury and delayed perforation. In cases of massive intraperitoneal hemorrhage, conversion to open surgery and oversewing or suturing of the bleeding site may be necessary. In some cases, the cause and site of bleeding are not identified, but peritoneal washout reduces fibrinolysis and is therefore recommendable.

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Peptic Ulcer After Bariatric Surgery

19

Mario Musella and Antonio Vitiello

19.1 Introduction

Peptic ulcers (PUs) are localized mucosal lesions of the gastrointestinal tract with a diameter of 5 mm or larger and extension to the muscularis mucosa. Mucosal defects smaller than 5 mm are usually defined erosions, which can also be a normal finding during a diagnostic endoscopy. PUs are much more likely to cause symptoms or complications such as bleeding or perforation. Both ulcers and erosions may be multiple [1].

Stress and hyperacidity have been considered in the past the main etiological agents of PUs; currently it is clear that *Helicobacter pylori* infection and the abuse of nonsteroidal anti-inflammatory drugs (NSAIDs) play a major role in the pathogenesis of peptic lesions [2].

PUs typically occur on the duodenal bulb or anywhere in the stomach; however, gastric peptic lesions are most frequently localized in the transitional zone on the lesser gastric curve between the body and the antrum. Conversely, ulcers localized in the greater curvature of the stomach are often the expression of a gastric cancer. In these cases, multiple biopsies during esophagogastroduodenoscopy (EGD), followed by pathological assessment, are mandatory.

Some studies have suggested an association between central adiposity and increased risk of gastric and *H. pylori*-negative ulcers. Nevertheless, the relationship between gastric or duodenal ulcers and obesity is still debated.

On the contrary, the etiology of the onset of PU after bariatric surgery is more obvious, but the causes and localization of mucosal defects correlates with the type of surgical procedure. PUs have been detected especially following three bariatric procedures, namely intragastric balloon positioning, the Roux-en-Y bypass (RYGB), and the mini gastric bypass/one anastomosis gastric bypass (MGB/OAGB) [3].

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19.2 Peptic Ulcer After Intragastric Balloon Placement

All intragastric balloons can cause PU due to the mechanical stress on the mucosa.

After placement of the classic BioEnteric Intragastric Balloon, which has been for a long time the most common intragastric device, PU incidence ranges from 1 to 2%. Ulcers appear normally within 6 months from balloon placement (which is also the deadline for balloon removal) [4–6]. The Orbera balloon is a small, flexible balloon introduced in the collapsed state and expanding to a diameter of 11 cm when filled with 500 mL of saline solution; ulcer formation is reported in less than 1% of patients [7]. The ReShape Integrated Dual Balloon System consists of two independent and non-communicating balloons bonded together. Each balloon can be inflated with a maximum of 450 cc of saline, subsequently the stress on the mucosa is higher and the rate of PU can reach 10% of cases [8].

19.3 Peptic Ulcer After Roux-en-Y Gastric Bypass

After RYGB, ulceration of the gastrojejunal anastomosis is one of the most common complications occurring in 0.6–25% of patients at 1–6 postoperative months [9–12]. If the ulceration is located on jejunal side of the anastomosis it is defined a "marginal ulcer", while defects on the gastric side are named "stomal ulcer". The etiology of these two types of ulcers differs. Ischemia is the main cause of stomal ulcers, whereas marginal lesions are due to jejunal exposure to gastric acid. Normally, acid content of the stomach is alkalized by bicarbonate and bile in the duodenum; this physiological buffering does not happen in the Roux limb. Common causes of hyperacidity and PUs are also known as risk factors for marginal ulcers, such as NSAID, alcohol and tobacco use [13]. For these reasons, it is mandatory for patients candidate to bariatric surgery to stop smoking and cease alcohol consumption. Non-absorbable sutures have been advocated to induce inflammation as foreign bodies increasing the incidence of marginal ulceration when compared to absorbable stitches [14, 15]. The role of *H. pylori* in the pathogenesis of postoperative marginal ulcers is still controversial; some studies have stated that even after eradication, the pre-existing mucosal damage could increase the risk of marginal ulcers; however, the vast majority of other evidence does not support this theory [16, 17].

Some technical factors have also been considered possible causes of ulcers after RYGB. A gastric pouch greater than 50 mL, and an anastomosis greater than 20–30 mm, seem to be related to a higher rate of marginal ulcers.

19.4 Peptic Ulcer After Mini Gastric Bypass/One Anastomosis Gastric Bypass

Unlike RYGB, the MGB/OAGB procedure is not burdened by a high risk of postoperative PU. Despite a longer gastric pouch, in which acid output may be present, the loop reconstruction brings bile from the afferent bowel to the anastomosis, reducing

the hyperacidity in both the anastomosis area and the efferent limb. In the Italian experience on 974 consecutive cases, the rate of marginal ulcer was 1.7% [18]. Carbajo et al. in their experience on the first 1200 OAGBs reported only 6 cases of PUs (0.5%) [19]. Similarly, a multi-institutional survey on 2678 patients with a mid-term (5 years) follow-up found a marginal ulcer only in 1.1% of patients [20].

Moreover, Chevallier et al. reported 20 cases (2%) of anastomotic ulcer following 1000 MGB/OAGB procedures with 2 cases of gastric perforation. However, especially heavy smokers remain candidate to develop PUs following MGB/OAGB [21].

19.5 Diagnosis of Peptic Ulcer After Bariatric Surgery

Diagnosis of PU in bariatric patients can be a challenging task. Even if localized, nocturnal epigastric pain is predictive of marginal ulcer, but diagnosis is not always easy due to the fact that the classic symptoms of PU are also present in bariatric patients without PUs. In the vast majority of cases, clinical manifestation resembles the presentation of PUs in non-operated patients (heartburn, epigastric pain, nausea, vomiting and dysphagia). However, complications such as perforations or bleeding have been reported without presenting previous significant clinical manifestations.

Upper endoscopy (EGD) remains the gold standard in bariatric patients with suspected ulceration both in the elective or in the emergency setting, when an upper gastrointestinal bleeding occurs. Conversely, if a perforation is the suspected diagnosis, a plain abdominal X-ray in the upright position, is the radiological tool of choice. In uncertain cases, abdominal CT scan with the administration of oral water-soluble contrast must be performed.

19.6 Treatment of Peptic Ulcer After Bariatric Surgery

Regardless of etiology of the ulcer, antisecretory agents represent the first-line therapy both for the relief of symptoms and tissue healing. In the 1970s and 1980s, histamine type 2 receptor antagonists (H2RAs) were introduced for the treatment of peptic disease. Many different drugs (cimetidine, ranitidine, etc.) have been developed with different pharmacokinetic properties and side effect profiles. In the 1990s, H2RAs was replaced by proton pump inhibitors (PPIs), which proved to be more effective.

The common therapeutic approach lasts from 4 to 8 weeks; in some cases, larger ulcers may require 3–4 months of oral medical treatment to heal. Resolution should be confirmed by upper endoscopy.

As stated above, reduction of alcohol consumption, cessation of smoking and eradication of *H. pylori* should also be considered to prevent and/treat postoperative PUs.

In the case of PUs due to a foreign body, like a non-absorbable suture, endoscopic removal should be performed [22]. Endoscopy could also be effective in the case of a bleeding lesion.

Table 19.1 Rate of perforated and bleeding ulcer after bariatric surgery

	RYGBP	MGB/OAGB	Balloon
Bleeding	5.0% (Carr et al.) 3.0% (Pyke et al.) 0.1% (Moon et al.)	0% (Musella et al.) 0.5% (Carbajo et al.) 0.1% (Chevallier et al.)	0.63% (Genco et al.) 2.1% (De Castro et al.) 2.3% (Mohammed et al.)
Perforation	1.4% (Carr et al.) 0.8% (Pyke et al.) 0.5% (Moon et al.)	1.1% (Musella et al.) 0.5% (Carbajo et al.) 0.1% (Chevallier et al.)	1.27% (Genco et a.l) 0% (De Castro et al.) 2.3% (Mohammed et al.)

Sources: Carr et al. [10]; Pyke et al. [12]; Moon et al. [23]; Musella et al. [20]; Carbajo et al. [19]; Chevallier et al. [21]; Genco et al. [4]; De Castro et al. [6]; Mohammed et al. [5]
RYGBP Roux-en-Y bypass, *MGB/OAGB* mini gastric bypass/one anastomosis gastric bypass

In the past, the surgical approach to peptic disease represented the gold standard. Antrum resection with loop (Billroth II) or Roux-en-Y reconstructions was widely performed in all general surgery departments. Currently, the spread and efficacy of H2RAs and PPIs have drastically reduced the need for surgery. After bariatric procedures, surgical revision of the anastomosis may be necessary for recurrent or refractory ulcers; the aim of the intervention is to incorporate healthier jejunum into the new gastrojejunostomy [23].

19.7 Management of Complications of Peptic Ulcers

The rates of perforated and bleeding ulcers after bariatric surgery are reported in Table 19.1. In stable patients, bleedings can be managed conservatively with fluid infusion or blood transfusion. If the vital signs are unstable, endoscopic or surgical exploration is mandatory. Endoscopy is diagnostic for hemorrhages from marginal ulcers and hemostasis can be attempted through clipping or injecting of epinephrine. Cautery should be avoided due to the possibility of delayed perforation. In cases of massive intraperitoneal hemorrhages or perforation, laparoscopy is usually the initial approach for bariatric patients. Conversion to open surgery may be necessary for complicated cases. The bleeding site is not always identified, but peritoneal washout is mandatory to remove blood clots and reduce fibrinolysis.

Surgical treatment of perforated ulcers should be the same as in common perforations with a surgical omental patch repair [24]. Simple oversewing of ulcers is not recommended due to the risk of a recurrence of the perforation. In severe interruption of the gastrojejunostomy, a refashioning of the anastomosis is necessary.

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Bowel Obstruction After Bariatric Surgery

20

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20.1 Etiology

Small bowel obstruction (SBO), although infrequent, is a possible scenario after bariatric surgery associated with considerable morbidity and mortality especially when not recognized early [1, 2]. The etiology is very varied, including causes specifically related to the surgical intervention and sometimes to the surgical technique [3, 4].

Intragastric balloon (IGB), adjustable gastric banding (AGB) and sleeve gastrectomy (SG) are exceptionally related to SBO. Some cases of intestinal migration of a self-deflated intragastric balloon have been reported [5, 6], most of them with a silent course ending with the patient's unconscious expulsion of the balloon. Few cases develop an SBO necessitating urgent laparoscopy and sometimes laparotomic conversion for its retrieval.

AGB and SG are more rarely associated with SBO [7, 8]. The intestinal transparietal migration of the gastric band connecting tube or more simply intestinal kinking around it may be at the origin of a subocclusive/occlusive syndrome. Conversely, only postoperative adhesive syndromes were found at the basis of the rare cases of SBO reported after SG [9].

The use of a ring applied to primary Roux-en-Y gastric bypass (RYGB) or SG to prevent gastric dilatation, such as a Fobi ring, can sometimes lead to peri- or postanastomotic slippage with high gastrointestinal obstruction, similarly to what happens for gastric band slippage.

RYGB and biliopancreatic diversion (BPD), and less frequently one-anastomosis gastric bypass (OAGB) are undoubtedly the procedures at higher risk of SBO, determining alone almost all the global incidence of SBO after bariatric surgery,

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Table 20.1 Etiology of SBO after bariatric surgery

IGB-, AGB-, SG-related	IGB migration Transparietal migration/volvulus with AGB connecting tube Adhesive syndrome
RYGB-, BPD-, OAGB-related	Internal hernia (mesocolic window, Petersen's hernia, meso-mesenteric hernia) Anastomotic stenosis/angulation (gastrojejunostomy, jejunojejunostomy) External hernia (trocar site hernia) Mesocolic window stenosis Intraluminal factors (blood clots)

SBO small bowel obstruction, *IGB* intragastric balloon, *AGB* adjustable gastric band, *SG* sleeve gastrectomy, *RYGB* Roux-en-Y gastric bypass, *BPD* biliopancreatic diversion, *OAGB* one anastomosis gastric bypass

reported from 1.9 to 7.3% [10]. With the advent of the laparoscopic approach, an important reduction in postoperative SBO due to adhesive disease and incisional hernias has been documented [11–13]. At the same time, there has been a higher incidence of SBO due to postoperative internal hernias and obstruction at the jejunojejunostomy (JJ) [14–16] (Table 20.1).

- ***Internal hernia (IH)*** represents the first cause of SBO after bariatric surgery [17]. Laparoscopic RYGB and BPD are associated with a higher incidence of IH compared with open surgery [18, 19], mainly related to reduced postoperative adhesions, which may help to fix the small bowel, technical difficulty in accurately closing all the mesenteric defects and excessive mobility of the bowel after massive mesenteric thinning. The classical presentation of IH is within 2 years from the first operation. Typically, it may occur at three potential sites: at Petersen's space (between the mesentery of the Roux limb and the transverse mesocolon), at the common channel (below the latero-lateral JJ) or at the transverse mesocolic defect after a retrocolic approach. Herniation of the Roux limb at the mesocolic window and at Petersen's space is the most common type of IH. It is difficult to estimate the real incidence of IH after RYGB, as it depends on the surgical technique, the need for large series and long and complete follow-up periods. More than the laparoscopic approach, other predisposing factors to IH are the Roux limb configuration (antecolic vs. retrocolic), the closure/non-closure of the mesenteric spaces, and the speed of weight loss: the incidence of IH is reported to be lower after RYGB with antecolic Roux limb, with closure of both the mesenteric spaces in patients [17, 20] with a gradual weight loss (the fast reduction of the mesenteric fat produces an enlargement of the mesenteric defects promoting the herniation of the small bowel).
- ***Obstruction at the JJ*** is reported in between 0.5 and 0.8% of patients after RYGB [21]. It includes any proven partial or complete obstruction at the JJ usually occurring within the first 30 postoperative days. It may be caused by technical problems such as stenosis or acute angulation of the anastomosis; other minor causes are postoperative anastomotic edema and intraluminal blood clot, which

tend to be partial and transitory and to spontaneously resolve with conservative measures. A primarily stenotic anastomosis has been correlated to the mechanical closure of the common enterotomy. Koppman et al. [17] in a review based on 8912 RYGB operations found an 18-fold increase in JJ stenosis with stapled closure compared with hand-sewn closure.

- ***Incisional hernia*** may be a not infrequent cause of SBO after bariatric surgery [22]. The clinical onset of a trocar site hernia often occurs within the 30th postoperative day. The trocar incision is the specific hernia site after the laparoscopic approach, even though the widespread use of dilating trocars has largely reduced its occurrence. Most complications occur with 10/12/15-mm trocars, but some have occurred at 5-mm and 3-mm trocar sites. Predisposing surgical factors are widening fascial defects, manipulation of trocar sites, undetected omentum or bowel entrapment after the trocar removal, perpendicular insertion of trocars rather than oblique. Interestingly, many authors suggest that most hernias occur at the site of midline trocars, probably due to the weakness of the paraumbilical region. So, the oblique introduction of the trocar into the abdominal wall and the choice to avoid the midline position for trocar placement may be instrumental in reducing trocar site hernias. In our experience, the few cases of trocar site hernia causing SBO occurred after SG at the left hypochondrium wound, widened for the specimen extraction and unsatisfactorily closed. Conversely, ventral hernias after the open approach occur in approximately 5% of patients.
- ***Adhesive syndrome*** in parallel with incisional hernia has been drastically reduced with the advent of laparoscopy. Its incidence is reported under 1% in several experiences [16, 17]. However, adhesive syndrome after laparoscopy has usually a different anatomic connotation than after traditional open surgery: it often presents with single-band adhesions rather than a diffuse and broad-based one, being more likely associated with SBO, due to kinking or volvulus of the bowel.
- ***Intestinal intussusception*** is an extremely rare cause of SBO (incidence <0.5%) [4, 23]. Although the exact cause is unknown, the most accredited theories propose the possibility of bowel dysmotility and lead points, in particular from the suture line and adhesions. Thin bowel mesenteries found in patients with successful weight loss may be an important predisposing factor to late intussusceptions. Regardless of the causes, most intussusceptions are typically retrograde after RYGB, at or distal to the JJ.

20.2 Clinical Presentation

The clinical presentation is very heterogeneous varying on the basis of the underlying etiology. In general, it may be acute—early (within 30 days after surgery) or late (usually between 30 days and 24 months)—with the common symptoms and signs of acute SBO (abdominal pain and tenderness, nausea, vomiting, tachycardia, constipation) or more often chronic—usually late—with subtle and recurrent vague intestinal symptoms (abdominal discomfort or recurrent cramping pain associated with occasional nausea/vomiting).

The timing of onset may suggest the specific obstructive cause: early SBO occurs most frequently at the JJ, due to technical problems such as kinking, narrowing or acute angulation of the JJ or to other causes including edema, ischemia or intraluminal blood clots. In the remaining cases, early obstruction can be less frequently caused by incisional hernias or intestinal intussusception. Conversely, late SBO is most often from IH, especially where closure of the mesenteric defects was not performed or was partial because of technical difficulties. Instead, the incidence of late SBO due to adhesions is negligible after the advent of the laparoscopic approach. In patients being treated with intragastric balloon, the occurrence of SBO should be always related to the possibility of migration of the balloon.

The level of obstruction after RYGB may be at the origin of different clinical manifestations: if proximal to the JJ, the patient will usually present with nausea, alimentary vomiting and abdominal cramps; bilious vomiting indicates obstruction at or beyond the level of the JJ; if the obstruction is distal to the JJ, the patient will develop symptoms indicative of gastric remnant and biliopancreatic limb distension, such as fullness, tachycardia, hiccoughs and shoulder/back pain.

20.3 Diagnosis

An accurate knowledge of the bariatric procedures, in particular of RYGB and its technical variants which represent the most frequent cause of SBO, is crucial for understanding the normal postsurgical anatomy and for diagnosing complications [11, 24]. The diagnosis always begins from the correlation of the patient's clinical signs and symptoms with his post-surgical history and his radiologic assessment.

Although many patients have nonspecific upper gastrointestinal symptoms and the physical examination is often poorly expressive in obese patients, meticulous history may direct the clinical suspicion towards the etiopathogenesis of the SBO and the level of obstruction. Laboratory testing is unhelpful, even though elevated transaminases and pancreatic amylases can be often found in biliopancreatic limb occlusions.

The patient's bariatric history is fundamental to guide the diagnostic suspicion, in particular knowledge of the RYGB technical variant performed, the closure or non-closure of the mesenteric defects, the timing from surgery, the speed and the amount of weight loss. Several authors have compared the different techniques performed for RYGB in terms of IH incidence, demonstrating lower rates in antecolic Roux limb configuration with closure of Petersen's and meso-mesenteric spaces. Quick and consistent weight loss is the condition at higher risk of IH development, caused by the enlargement of the mesenteric defects due to the rapid reduction of the intra-abdominal fat mass, even if intra-operatively sutured.

Radiologic assessment is mandatory before deciding the correct—surgical or conservative—management of SBO after bariatric surgery. Computed tomography (CT) with possible oral contrast is the most accurate radiologic examination to diagnose a SBO and hypothesize the underlying causes [25]. Whereas it is relatively simple to identify the point of arrest of a migrated balloon and the level of occlusion

in the case of adhesive syndrome and incisional hernia, some difficulties may arise when facing a suspicion of IH. Apart from the classical radiologic images of SBO, other six signs have been reported in association with IH after RYGB [26]:

(a) swirled appearance of the mesenteric fat and vessels at the mesenteric root (mesenteric swirl sign);
(b) mushroom-shaped herniated mesenteric root with crowding and stretching of the mesenteric vessels (mushroom sign);
(c) clustered loops of small bowel, usually in left hypochondrium (clustered loops sign);
(d) tubular distal mesenteric fat surrounded by bowel (hurricane eye sign);
(e) bowel other than duodenum posterior to the superior mesenteric artery (SMA) (small bowel behind SMA sign);
(f) right-sided location of the distal JJ.

Among all of the above, a mesenteric swirl is the best indicator of IH after laparoscopic RYGB, with a sensitivity and specificity reported from 61% to 83% and from 67% to 94%, respectively. The combination of swirled mesentery and mushroom shape of the mesentery increases the diagnostic sensitivity compared to swirled mesentery alone gaining in sensitivity from 78 to 83%, but unchanged specificity (from 67 to 89%).

A crucial aspect for radiologists is to familiarize themselves with the post-bariatric surgery CT appearance and with the signs of complications. It is important for the bariatric surgeon to realize that the clinical presentation has a central role in making decisions regarding the SBO, and where it suggests an IH, it may lead the surgeon to directly opt for laparoscopic exploration.

20.4 Management

In the approach to patients with symptoms predictive of subocclusive/occlusive syndrome following bariatric surgery, two aspects should be always taken into account: timeline after surgery, because early SBOs are usually attributable to technical problems at the JJ and less frequently to incisional hernias while late SBOs are nearly always due to IH and rarely to adhesions after laparoscopic surgery.

Despite the lack of clear evidence, some technical choices while performing a RYGB have been suggested to reduce the SBO rate [14, 16, 17]:

(a) the antecolic configuration should be preferred to the retrocolic one because it avoids the creation of the mesocolic defect, which is reported to be the most common site of intestinal herniation;
(b) a hand-sewn closure of the common enterotomy during the creation of side-to-side JJ should be performed in place of a stapled closure, most frequently associated with JJ strictures;
(c) mesenteric defects should be closed where technically possible, by means of non-absorbable sutures or clips.

The goal of treatment in patients with SBO, even after bariatric surgery, is to promptly identify those who need urgent surgery because at risk of bowel ischemia, due to extreme dilatation or strangulation, and to ensure adequate resuscitation by stabilizing the vital signs and volume status and starting antibiotics where necessary.

Conservative management may be offered, as a first approach, in patients with mild and intermittent symptoms occurring early after bariatric surgery, suggestive of edema or stenosis of the JJ. In these cases, bowel rest followed by a more gradual weaning and the administration of short-term corticosteroid therapy can be sometimes a non-surgical solution.

Surgical management is mandatory when faced with acute SBO and should not be delayed because of potential life-threating complications [14, 15, 27]. Nasogastric tube has limited to no efficacy, including the risk of a blind perforation. Laparoscopy should be the approach of choice in expert hands. A specialized knowledge of post-surgical anatomy and of the technical variants and recognition of the potential causes of SBO are crucial. Treatment varies on the basis of the etiology. In cases of IH, it consists of laparoscopic reduction of the intestinal herniation with subsequent closure of the mesenteric defect through a running non-absorbable suture or clips. Two are the potential sites of IH after RYGB performed according to the antecolic Roux limb configuration: Petersen's space (most commonly) and the meso-mesenteric defect below the JJ. In the absence of suffering and excessively dilated bowel which makes its laparoscopic manipulation hazardous, the repair is easy, safe and nearly always feasible in laparoscopy. Instead, the intra-operative finding of a technical problem at the JJ, such as kinking, narrowing or acute angulation of the anastomosis may require the creation of a new JJ between the distal Roux limb and the proximal common channel to bypass the obstruction at level of the original JJ, which is not resected. Treatment of the adhesive syndrome and incisional hernias does not present any peculiar surgical detail. The removal of a migrated balloon is comparable to that of any endoluminal foreign body. In all circumstances, conversion to laparotomy can be helpful to facilitate the management of the more critical situations.

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Acute Peritonitis and Abscess After Bariatric Surgery

21

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21.1 Definition

Acute peritonitis and peritoneal abscess are severe abdominal complications that may occur after bariatric surgery procedures for morbid obesity. Acute peritonitis can be defined as inflammation of the serosal layer resulting from diffusion of contaminated fluid to the whole abdominal cavity. Abscesses are the consequence of a localized infected fluid collection, usually adjacent to the surgical site.

Among all the complications of bariatric surgery, peritonitis and abscesses are the most common causes of early postoperative morbidity; a missed diagnosis may result in sepsis, which itself can cause acute renal and respiratory failure [1].

21.2 Etiology

Although diffuse peritonitis or peritoneal abscesses can rarely result from a contamination of undrained fluid collections in the abdominal cavity, these complications are usually the consequence of the loss of integrity of the gastrointestinal (GI) tract. Peritonitis and abscesses have been observed as adverse events in all bariatric surgical procedures; while leak rates are close to zero after adjustable gastric banding, higher rates have been observed after sleeve gastrectomy (SG), single anastomosis gastric bypass and Roux-en-Y gastric bypass (RYGB) [2].

Peritonitis and abscess typically occur within the first 10 days after surgery, with an incidence of about 1% after one-anastomosis gastric bypass, 1–6% after RYGB and 3–7% after SG [3].

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The literature has identified male gender, open surgery, revisional surgery, hypertension and hypoalbuminemia as independent risk factors for complications after bariatric surgery [4].

Laparoscopic SG is nowadays considered a safe and effective procedure [5], but many studies report an incidence of staple line leaks ranging from 0.5 to 7% [6]; risk factors can be identified in visceral ischemia, defects in surgical technique, stapler failure, diathermy-related gastric wall damage, thinner thickness of gastric fundus wall, redo surgery (previous removal of adjustable gastric banding) and the increase of intragastric pressure (mainly related to the patient's weak compliance with the diet) [7]. Our recent study demonstrated that the incidence of staple line leaks after SG is inversely proportional to the surgeon's experience; moreover, the use of cartridges with buttress in the upper part of the stomach seems to be associated with a lower risk of fistula [8].

The incidence of anastomotic leakage after RYGB varies from 0.1 to 5.6% according to the various definitions given; leaks are likely to be responsible for up to 30% of the overall mortality following this procedure [9]; gastrojejunal anastomosis leakage, the most frequent adverse event after gastric bypass, may occur in the early postoperative period; however, the literature reports several cases of late peritonitis due to the perforation of chronic gastrojejunal anastomotic marginal ulcers [10].

Peritonitis after one-anastomosis gastric bypass is rarer if compared to RYGB (<1%); the most frequent cause is a gastrojejunal anastomosis leak, a gastric staple line leak being rarer [11].

Revision surgery and open surgery are the most important risk factors for gastrojejunal anastomosis leakage [12].

Some studies demonstrate that acute peritonitis and peritoneal abscess after laparoscopic SG and laparoscopic RYGB, due to GI leaks have statistically the same incidence (2.3% vs. 1.9%) [13].

Adjustable gastric banding, currently one of the most minimally invasive surgical techniques for the treatment of morbid obesity can be indirectly responsible for peritonitis and abdominal abscess, in cases in which surgical removal of an eroded and/or dislocated band is necessary [14]; nevertheless, the presence of a surgical prosthesis can predispose to the onset of rare forms of peritonitis (Mycobacteria contamination) as observed in some cases reported in the literature [15].

Cultures performed on the peritoneal fluid of patients treated with bariatric surgery have detected the presence of Gram-positive strains in 63% of cases, and less frequently mixed infections with Gram-positive/Gram-negative bacteria; the presence of multidrug-resistant strains is a rare condition after bariatric surgery; fungi contamination, mainly caused by *Candida albicans*, occur in 11% of cases [2].

21.3 Diagnosis

Acute diffuse peritonitis and peritoneal abscesses are one of the most common complications following bariatric surgery; however, the underlying condition, especially

a GI leak, could be difficult to diagnose because of the onset of non-specific symptoms; some studies report a delayed diagnosis ranging from 1 to 18 days because of lack of symptoms or limited clinical signs [16].

Acute upper abdominal pain, often associated with fever and interscapular pain, is highly suggestive of a leak; blood tests can detect leukocytosis and elevated C-reactive protein and procalcitonin.

Classical signs of peritoneal irritation in the obese patients are not usually present, as there is no abdominal wall guarding and rigidity; it is crucial to give importance to non-specific signs that could nevertheless raise alert: a spike of fever, abdominal heaviness, hiccups, tachycardia and acute urinary retention [1].

In cases of a clinical suspicion of GI leak following a bariatric procedure, abdominal computed tomography with oral contrast administration (Fig. 21.1) seems to be the diagnostic gold standard, in order to provide evidence of peritoneal diffuse or localized collection and to plan surgical or percutaneous radiological procedures, conventional upper GI radiological study being useful only to reveal the presence of a fistula but obviously not to detect a peritoneal collection.

The routine use of a postoperative upper GI radiological study after bariatric surgery remains common in accredited centers, but this practice seems to be associated with a prolonged hospital stay with no significant effect on the diagnosis of leak rate [17].

Intraoperative leak testing during SG is performed by many bariatric surgeons, but it has demonstrated a very low sensitivity and no advantages in terms of the incidence of postoperative leaks [18].

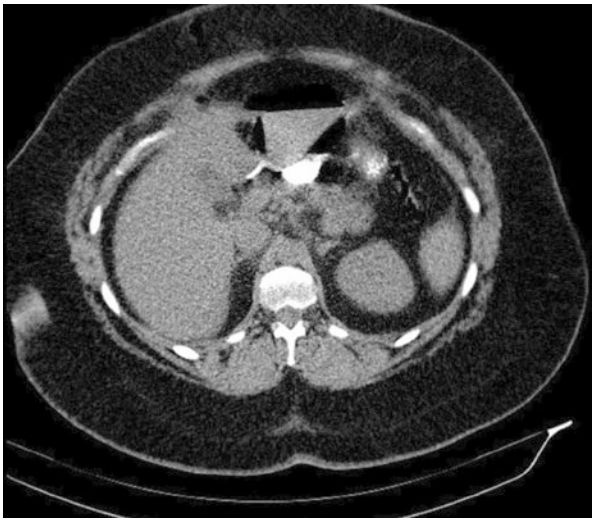

Fig. 21.1 Computed tomography scan showing perigastric fluid collection after sleeve gastrectomy with evidence of intraperitoneal gastrografin effusion

21.4 Treatment

Diffuse peritonitis and peritoneal abscess after bariatric surgery are severe and potentially life-threatening clinical conditions, requiring always an immediate hospitalization and starting of adequate treatment as soon as the diagnosis is achieved; mortality rates can reach 24%.

International guidelines recommend an antibiotic prophylaxis with cefazoline for surgical procedures not involving the small bowel (i.e., SG) and with an association of cefazolin and metronidazole for RYGB or single-anastomosis gastric bypass.

Gram-positive cocci are the most frequently isolated bacteria in the cases of peritonitis and infected abdominal collections after bariatric surgery; the incidence of Gram-negative involvement is significantly lower; multidrug resistance is a rare condition, a previous prolonged antibiotic treatment being the most important risk factor; antibiotic monotherapy with broad spectrum drugs is usually the best choice [19].

Treatment of peritonitis and peritoneal abscesses has to be performed with adequate drainage of contaminated fluid (Fig. 21.2); drainage can be achieved surgically (laparoscopic or open) or with a percutaneous approach.

Patients should be re-treated in highly specialized centers, preferably by the same team who performed the surgical bariatric procedure, in order to provide the best treatment (usually endoscopic or minimally invasive).

The adequate and definitive therapy of post bariatric surgery peritonitis and abscesses can be achieved only if the underlying leak is recognized and treated.

A staple line leak after SG is usually managed by endoscopic placement of a self-expandable stent [20]. In our experience, we use a specifically designed self-expanding metallic stent (Niti-S Beta stent by Taewoong Medical, Gyeinggido, South Korea). This over-the-wire nitinol stent has a small-cell mesh, a specific design with antimigration features (outer double layers coated with silicone) and a length between 18 and 20 cm to allow extension from the esophagus to the antrum. The stent diameter is 24 mm (32 mm proximal flared end) in order to achieve optimal adherence to the esophagus. The stent introducer is 22 Fr and accepts a 0.038-inch guidewire; we obtained a success rate close to 90%, with a stent migration rate lower than 10%.

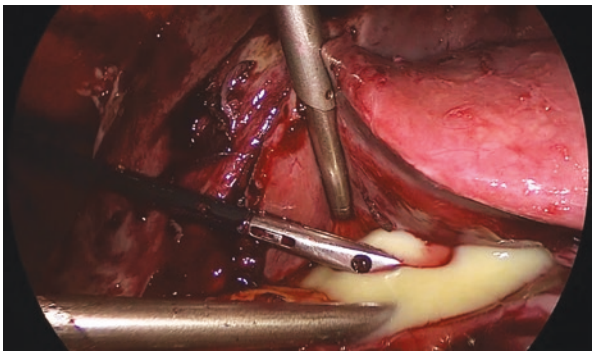

Fig. 21.2 Laparoscopic drainage of a peritoneal abscess after sleeve gastrectomy

Laparoscopic peritoneal cavity lavage is necessary in patients presenting peritonitis or abscess not suitable for conservative treatment or radiologic percutaneous drainage.

Another option is the rendez-vous technique of inserting a pigtail catheter or a nasobiliary tube into the abscess cavity through the fistula, draining the abdominal cavity as well as the stomach [21].

Acute abdominal sepsis after one-anastomosis gastric bypass caused by a gastrojejunal anastomosis leak can be successfully managed with emergency surgical conversion to RYGB with adequate peritoneal drainage; the conversion procedure seems to be related to lower morbidity and mortality compared to simple anastomosis revision [15].

Minimal gastrojejunal anastomosis leaks after RYGB can be treated with endoscopic wallstents or with endoscopic suture followed by nasogastric tube placement; in cases of jejunojejunal anastomosis leak, redo surgery with anastomosis reinforcement or remake should be performed.

21.5 Outcome

Acute peritonitis and peritoneal abscess can be life-threatening condition if not immediately diagnosed.

Despite an average young age and a low incidence of underlying diseases, peritonitis after bariatric surgery seems to be related to a higher mortality rate if compared to peritoneal contamination after other surgical procedures, with also higher rates of initial renal failure [2].

An adequate surgical laparoscopic revision with fluid drainage, associated with endoscopic fistula repair, is to be considered the gold standard in order to achieve the complete resolution of this postoperative complication and to prevent the evolution towards sepsis.

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Anastomotic Leak After Bariatric Surgery: Prevention and Treatment

22

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22.1 Introduction

Anastomotic leak is one of the most feared postoperative complications after bariatric surgery. The origin of this fear finds its roots in the obese patient itself, because of the changes induced in the inflammatory response caused by obesity and the comorbidities associated with morbid obesity, which may transform a small problem into a huge disaster [1–3].

The bariatric patient must be considered a high-risk patient, and every effort must be spent to prevent any complication. Even though an apparent decreased incidence is described in the literature [4], gastrointestinal (GI) leak remains an important cause of morbidity and mortality. Sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB) are the two main bariatric procedures performed worldwide, with a reported incidence of leak between 0–7% after SG and 0.1–8.3% after RYGB [5].

Every bariatric technique should consider the possibility of a leak. The etiologies are mechanical causes, tissue causes and ischemic causes. These factors must be always considered when manipulating small bowel, identifying the thickness of tissues, or when choosing cartridges, in order to avoid narrowing, excessive tension or twisting/kinking of mesentery [6].

When a GI leak develops in bariatric patients, the clinical presentation is usually more subtle or delayed compared to normal patients, and could lead to a delayed diagnosis and treatment. Another important aspect that must be taken into account is the absence of a standardized treatment for anastomotic leak, although many different approaches and techniques are described in the literature.

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These aspects underline once again the importance of prevention when operating on obese patients.

The aim of this chapter is to analyze the main factors involved in leak development for each surgical intervention for morbid obesity, and the most useful strategies to prevent and treat this complication.

22.2 Laparoscopic Sleeve Gastrectomy

22.2.1 Pathophysiology

Leaks after SG, though occurring with a lower incidence compared to postoperative bleeding (0–7% in different series), represent the most feared complication because of difficulties in treatment and healing. SG itself is at the origin of this kind of complication: the presence of a long suture line in an organ with a high blood supply can result in the creation of ischemic tracts. Furthermore, the pathophysiology of the leak is well described by the Laplace law: the reduced stomach creates a high-pressure organ, and the thickness of the gastric wall changes along the suture line. The vast majority of leaks after SG occur in the upper third of the gastric remnant, and this can be caused by an ischemic event combined with the Laplace effect.

The most used classification of leaks is based on their timing of occurrence and divides them into three groups: early (within fourth postoperative day), intermediate (between the fifth and the ninth postoperative day) and late (from the tenth postoperative day) [7]. Several series report leak occurrence in the intermediate period in 50–80% of patients.

22.2.2 Prevention

The first prevention strategy that must be applied is the strict adherence to a well-standardized technique [7, 8]: the use of an adequate bougie size (>40 Fr) [9, 10], the section of the great gastric wall starting from 5 cm from the pylorus, remaining 1–2 cm lateral to the angle of His and maintenance of a systolic blood pressure >100 mmHg while stapling [11, 12].

Staple-line reinforcements have been suggested to prevent leaks after SG, but their role is still controversial. Moreover, many different studies have shown conflicting results in terms of prevention of bleeding and leakage, no matter what kind of reinforcement was used (including oversewing the staple line, buttressing the staple line or applying fibrin glue or biological glue to the staple line) [6, 10, 13–15], and this unclear role is confirmed in two different meta-analyses [16, 17].

In conclusion, it is not possible to recommend the use of any kind of staple-line reinforcement, oversewing and/or glue for leak prevention during SG. Furthermore, the routine use of upper GI contrast radiograms is not recommended for the early detection of leaks, as most leaks occur after discharge from hospital [5], so they should be performed in selected patients.

New technologies have been suggested for the prevention of leaks, such as the use of indocyanine green fluorescence angiography during SG, in order to determine the vascular patterns to the stomach and the gastroesophageal junction and to prevent ischemia-related leaks, with promising results [18].

22.2.3 Treatment

Leak after SG is a real challenge, both for the clinical aspects (i.e. delayed presentation in frail patients) and for the anatomical position (gastroesophageal junction) of the majority of cases.

In the acute setting, patients discharged from hospital with regular postoperative course and normal upper GI radiograms come back with acute abdominal pain, fever and tachycardia. In these patients, when possible, the best assessment is computed tomography (CT) scan with oral and IV contrast [19, 20].

When the leak is confirmed, or when patients are septic and other causes are excluded, the first step must be drainage of the abscess or collection in order to clean out the abdominal cavity. Depending on the clinical presentation, technical feasibility and clinician expertise, this can be done with radiological percutaneous drainage or with laparoscopic revision of the abdomen [6]. If possible, during surgical re-operation, primary repair of the leak may be effective, especially in the very early leak. Otherwise, after draining out the origin of sepsis, the leak can be successfully treated with multiple approaches such as covering the hole with endoscopic stents [21] or placing large endoscopic clips to close the defect of the staple line [22]. All these options demonstrate the necessity to have several specialists available and trained in bariatric surgery in order to address this complication and to treat it in the best possible way.

In conclusion, although not possible to standardize an appropriate treatment for leaks after SG, clinical evidence shows that early leaks (within 72 h postoperatively) may be treated with laparoscopic drainage and primary suture and repair of the defect with good results, while delayed leaks may be treated with a multidisciplinary approach.

22.3 Laparoscopic Roux-en-Y Gastric Bypass

22.3.1 Pathophysiology

This operation has four staple lines that may develop leaks: the gastrojejunal anastomosis, the jejunojejunostomy, the gastric pouch staple line and the gastric remnant staple line. The mean incidence is 0.1–8.3% [6].

The most frequent location of the leak, as shown in the literature, is the gastrojejunal anastomosis. Integrity of the gastrojejunostomy depends on the sutures or staples used until tissue healing acquires sufficient strength to offset the increased loads placed across the anastomosis [23].

This greater incidence is probably related to the creation of an anastomosis that may suffer from an excessive tension of the alimentary limb, which is not present in the other staple lines. Furthermore, suture lines on the stomach do not suffer from increased intraluminal pressure after food ingestion as occurs in SG [24].

Leaks after RYGB mainly occur during recovery after the primary operation, unlike those after SG that tend mostly to occur after discharge from hospital. Recent studies have shown that RYGB has a higher mortality and morbidity rate when compared to SG, especially in elderly patients, although this is not necessarily related to leaks [25, 26]. All these aspects can be explained with the different mechanical aspects involved in the two different techniques.

22.3.2 Prevention

Several strategies have been suggested to prevent leak formation after RYGB, and many different tricks have been described to avoid anastomotic leak. First of all, to avoid excessive tension of the alimentary limb, this should be placed in a retrocolic position. Suture line reinforcements like oversewing, application of fibrin glue or tissue sealants have been used to prevent leaks [27, 28]. Particularly, two recent meta-analysis showed superior results in patients in whom suture line reinforcements were used, compared to RYGB with no reinforcements [29, 30], but prospective randomized studies are needed to prove their efficacy. Another important aspect is intraoperative leak assessment. It is no doubt useful to detect intraoperative leak (both for gastrojejunostomy and jejunojenunostomy if RYGB is performed with a "double-loop" technique), by distending the anastomosis with endoscopy or with methylene blue [31].

Routine placement of a drainage tube near the gastrojejunal anastomosis has an unclear role, with some authors always placing it to have an "external eye" on the anastomosis and others stating that it may cause decubitus on the anastomosis itself.

Since the leak after RYGB develops mostly in the early postoperative days, most surgeons prefer to perform upper GI radiograms routinely to assess the gastrojejunostomy, whereas others suggest obtaining them only if there is a clinical suspicion of leakage [32, 33].

22.3.3 Treatment

The first aspect that must be taken into account is the clinical presentation of the patient with a suspected leak. If her/his conditions are unstable and symptoms are highly suspicious, there is no time to perform any radiological assessment and surgical exploration is mandatory.

It is well known that an early aggressive treatment of leak—i.e. re-exploration with laparoscopy or laparotomy—is the best available tool to address this complication, even if the leak is only suspected [34, 35]. This is also explained by the fact that sometimes even the CT scan of the abdomen with double contrast may be

interpreted as normal or unclear despite the presence of a leak [36], leading to delayed treatment with an increased risk of the patients developing sepsis and multiorgan failure.

Depending on the severity of the intra-abdominal situation, surgical treatment may vary from a simple lavage and drainage of the abdominal cavity, to placing some stitches where the leak is documented or, in the worst cases, to complete reconstruction of the anastomosis. Finally, in some cases many authors perform a jejunostomy to feed the patient. Given this, stable patients or patients with small blind-ended leaks may be treated conservatively with prolonged parenteral nutrition or with endoscopic stent placement [37], or with percutaneous radiological drainage. In these cases, adequate management is targeted on the single patient, since few data are yet available.

22.4 Laparoscopic Mini Gastric Bypass/One-Anastomosis Gastric Bypass

22.4.1 Pathophysiology

For this emerging technique, which is spreading worldwide, few data on leak development are available. Although the reported incidence of leaks varies from 0.8 to 2.6%, these still represent the most severe and worrisome complication. The low rate of leaks can be partially explained by the specific aspects of mini gastric bypass/one-anastomosis gastric bypass (MGB/OAGB). The gastric pouch is long but not under pressure, and normally no tension is applied to the gastrojejunal anastomosis [38–40]. Despite this, more than half of the leaks arise from the gastrojejunal anastomosis, and few from the gastric tube.

Since this technique is largely used as a revisional procedure, some studies report an increased leak rate when MGB/OAGB is performed in this fashion [41]. These data are not confirmed in other series in which redo surgery does not represent an independent risk factor for leak development [42].

Biliary reflux may be advocated as a cause of delayed leak, which may occur even several months later and, in some cases, may require immediate re-operation in patients presenting with acute abdomen, but in the vast majority of the series no significant nor symptomatic bile reflux is described, and further data are required to understand its real impact.

In conclusion, what emerges from the literature, is that the leak rate after MGB/OAGB is not related to any particular identified technical factor [43].

22.4.2 Prevention

The first consensus conference on MGB/OAGB described some important steps to be followed in order to perform a standardized procedure and to reduce the complication rate: construction of the MGB/OAGB pouch should start in the horizontal

portion of the lesser curvature of the stomach to make it as long as possible (this aspect may reduce tension on the gastrojejunal anastomosis); routine use of staple-line reinforcement is unnecessary; routine use of antireflux sutures is unnecessary; an intraoperative leak test is recommended; routine placement of surgical drains is unnecessary [40]. Close adherence to these recommendations is the most useful tool for preventing complications.

22.4.3 Treatment

When leakage occurs, the surgeon must treat it rapidly. This, because patients present with acute intra-abdominal sepsis and require immediate re-operation in the vast majority of cases.

Data are discordant on the mean time of leak presentation, but more than half of the patients are usually discharged before onset of the leak, so they come back to the hospital with a systemic inflammatory response syndrome (SIRS). As for all leaks, radiological assessment with double contrast CT scan is useful in stable patients, but in some cases it may be unhelpful or may even delay the treatment [42].

Surgical exploration, by laparoscopy or laparotomy, is mandatory both in confirmed or highly suspected leaks. It is not possible to standardize the surgical management of leaks, because each patient has a different presentation and a different intra-abdominal situation: some of them may be treated with laparoscopic lavage and drainage, or with prolonged parenteral nutrition, while some others may require a redo operation with stitches applied on the leak site or conversion to a RYGB, sometimes with the creation of a feeding jejunostomy [44]. What must be kept in mind is that surgical exploration should never be delayed.

22.5 Biliopancreatic Diversion-Duodenal Switch

22.5.1 Pathophysiology

Despite biliopancreatic diversion-duodenal switch (BPD-DS) has excellent metabolic long-term results, this operation represents only a very small percentage of the bariatric operations performed worldwide [45]. This is related to the high technical complexity, high complication and mortality rates reported in the literature [46].

The reported incidence of leaks is between 0 and 4.6% and the leaks may involve the gastric suture line after SG, the duodenoileal anastomosis, the ileoileal anastomosis and the duodenal stump.

One of the main reasons that can explain the high morbidity afflicting this procedure lies in the patients themselves. BPD-DS is mostly performed to treat super-obesity, that is to say, subjects at high risk due to their initial condition.

Other aspects strongly related to leak development are the number of anastomoses and suture lines and the fact that the SG is performed with all the intrinsic risks described above.

22.5.2 Prevention

Given its complexity and its targeted super-obese patients, BPD-DS must be performed in high-volume centers, to decrease morbidity and mortality [47].

All the general precautions listed in the introduction have to be followed to reduce the risk of leak development and, since a SG is performed as well, meticulous adherence to the "SG dogmas" must be present. Routine usage of staple line reinforcements is not recommended, while an intraoperative leak assessment may be useful to detect immediate staple line defects on the gastric tube or in the duodenoileal anastomosis, but it does not affect postoperative leak development.

Drain placement—as well as upper GI radiogram performance—basically depends on the surgeon's experience and habits, since no clear role is demonstrated in early leakage detection.

22.5.3 Treatment

Management of leaks mostly depends on their site associated with the clinical presentation.

When possible, a CT scan with oral and IV contrast may be useful to detect the origin of the fistula and to plan a strategy. In stable patients with duodenal stump leak or with low-output anastomotic fistulas, percutaneous drainage plus total parenteral nutrition may be the treatment of choice [48]. In unstable patients, with anastomotic leakage (duodenoileal anastomosis is still demanding [49]) a prompt and aggressive treatment must be undertaken, by laparoscopy or laparotomy, including direct suture, anastomotic reconstruction and/or jejunostomy of the biliopancreatic limb for decompression [48].

22.6 Conclusions

Leaks after bariatric surgery are still one of the most feared complications since they are associated with high morbidity and mortality rates in high-risk patients. The most important prevention strategy is close adherence to standardized techniques. Clinical symptoms must never be underestimated and must guide the surgeon's strategy. Several minimally invasive treatments (radiologic and/or endoscopic) are available for leaks but, when necessary, prompt and aggressive surgical treatment, by laparoscopy or laparotomy, must be the final choice.

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Gallstones and Related Complications, Cholecystitis and Cholangitis After Bariatric Surgery

23

Marco Antonio Zappa and Elisa Galfrascoli

23.1 Introduction

In Europe, the overall prevalence for cholelithiasis is 19% in women and 9% in men and it varies between different ethnicities, whereas higher rates have been observed in Caucasians, Hispanics, and Native Americans [1]. The most important risk factors for gallstone formation are age >40 years, female gender and obesity [1]. Weight loss is a further major contributor to the development of lithiasis [2].

Despite the beneficial effects of bariatric surgery, bariatric patients are prone to the formation of gallstones with a postoperative cumulative risk of 30–53% [3]. This occurs as early at 3 months following surgery and peaks at 16 months. However, after 24 months, when the weight stabilizes, this risk may decrease [4]. Several studies have demonstrated that the prevalence of cholelithiasis, symptomatic or non-symptomatic, is directly correlated to the body mass index (BMI) [5]. The risk of gallstone formation is proportional to the degree of overweight, and its incidence is eight times greater in patients with BMI >40 kg/m^2 [6, 7].

In addition to that, the rapid loss of weight induced by diet or surgery in obese individuals is frequently associated with the formation of gallstones. Approximately 11–28% of obese subjects undergoing severe dietary restrictions and 27–43% of patients who have undergone bariatric surgery develop gallstones within a period of 1–5 months after their treatment [7–10]. Specific risk factors for gallstones identified during weight loss are:

- relative loss of weight greater than 24% of initial body weight;
- rate of weight loss greater than 1.5 kg per week;
- very-low-calorie diet with no fat;
- long overnight fast period;
- high serum triglyceride level [11].

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23.2 Pathophysiology

In obese patients most gallstones are cholesterol gallstones; the remaining are pigmented gallstones, which are chemically different and have a different set of risk factors, not common among obese patients. At least three physical conditions appear necessary for the formation of cholesterol gallstones.

1. Cholesterol supersaturation of bile, based on the relative concentrations of cholesterol, bile salts, and phospholipids, is a precondition of cholesterol crystallization. A cholesterol saturation index greater than 1 is considered supersaturated [12].
2. Gallbladder hypomotility, which produces stasis of bile in the gallbladder that accelerates cholesterol crystal nucleation and growth, increased cholesterol supersaturation, and opportunities for cholesterol crystallization as well as gallstone formation and growth.
3. The presence of a kinetic defect due to antinucleating and pronucleating proteins, perhaps derivatives of gallbladder mucin [5].

Lithogenesis is modified following bariatric surgery; the hypersecretion of cholesterol from the liver is considered to be the main cause of an increased risk of gallstone disease in obese patients [3]. Other directly correlated factors are a decreased secretion of biliary acids due to caloric restriction, an increased mucin production enhancing crystallization and gallbladder hypomotility [13–15]. This hypomotility is secondary to decreased cholecystokinin secretion related to the hypocaloric diet or obesity-related resistance to cholecystokinin, to gastroduodenal exclusion, or intraoperative injury to the hepatic branches of the vagus nerve [16].

Genetic factors related to lipid metabolism, including apolipoprotein-E (Apo-E) and cholesteryl ester transfer protein (CETP) gene polymorphisms, are shown to play a role in the gallstone formation after bariatric surgery. These data suggest that Apo-E genotyping may be useful in selecting patients for gallstone prevention [17].

23.3 Different Bariatric Procedures

The incidence of gallstone formation differs between the various types of bariatric procedures [18].

The incidence of cholelithiasis is so high in biliopancreatic diversion that cholecystectomy is part of the procedure. In the other operations—such as Roux-en-Y gastric bypass (RYGB), gastric banding and laparoscopic sleeve gastrectomy (LSG)—routine cholecystectomy is still controversial [6].

Restrictive procedures such as gastric banding and LSG should have a comparatively lower risk of gallstone development, as gallbladder contraction mechanisms and enterohepatic circulation are not directly disturbed. However, factors that promote cholelithiasis such as reduction in gallbladder emptying, increased gallbladder residual volume and decreased refilling have been demonstrated after gastric

banding [19]. In addition to that, studies on hormonal effects after gastric bypass have shown no significant change in cholecystokinin level before or after meals [20, 21]. After gastric banding, patients developed gallstones at a rate of 27%, with 6.8% of them symptomatic [22, 23]. In published series using regular ultrasound surveillance after gastric bypass, the rate of asymptomatic gallstone formation ranged from 30 to 53% within 6–12 months after the operation and 7–16% of the patients experienced symptoms [10, 24].

The published data are not illuminating in LSG. To the best of our knowledge, only few case series exist in the literature. Moreover, there is lack of protocols concerning the management of gallstones.

23.4 Time for Cholecystectomy

Gallbladder management during bariatric surgery is controversial. Three different approaches have been proposed:

- prophylactic concomitant cholecystectomy in all obese patients undergoing surgery;
- a selective approach, based on which cholecystectomy is only performed in the presence of gallstones or biliary symptoms;
- a wait-and-see approach, based on which no concomitant cholecystectomy is performed, and patients could receive prophylactic medication against biliary disease (ursodeoxycholic acid).

Several factors need to be considered before deciding whether or not to perform concomitant cholecystectomy:

- Technical difficulties in diagnosing biliary lithiasis in patients with morbid obesity.
- Concurrent cholecystectomy during laparoscopic bariatric surgery is technically difficult because of suboptimal port placement, visceral obesity, and prolonged surgical time, and the gallbladder is often engulfed by the large liver [25].
- After bariatric surgery only 6.5–30% of gallstones become symptomatic, requiring cholecystectomy [6, 26].
- The main cause for the subsequent cholecystectomy is uncomplicated biliary disease while choledocholithiasis and biliary pancreatitis occur very rarely and are usually preceded by at least one previous episode of biliary colic.
- About 95% of subsequent cholecystectomies are performed laparoscopically with a very low conversion rate and low risk of complication (0.1%) [27].

Therefore, a routine concomitant cholecystectomy cannot be recommended when weighing the observed low long-term morbidity against the potential detrimental effect on the short-term outcome. Considering that concomitant cholecystectomy significantly increases the overall perioperative complication rate by nearly

1% and that also the mortality rate is higher, we recommend against the routine association of this procedure in patients without gallstones [28].

The question that remains to be answered is how to proceed with patients with cholelithiasis. Considering the aforementioned factors, it is reasonable to adhere to the recommendations for the general population and suggest a concomitant cholecystectomy only in patients at greater risk of developing complications:

- in the presence of symptoms;
- if the calculi are smaller than 3 mm or larger than 2 cm;
- life expectancy is over 20 years [29].

In RYGB, concomitant cholecystectomy is often suggested in the presence of any type of lithiasis since biliary complications such as choledocholithiasis or biliary pancreatitis are very difficult if not impossible to treat with endoscopic retrograde cholangiopancreatography (ERCP).

Some techniques have been described in order to overcome this limitation such as single-/double-balloon enteroscopy and laparo-endoscopic transgastric approaches. However, these techniques are expensive, time consuming, not easily performed and often not successful [30].

Some authors proposed a gastric bypass with fundectomy and gastric remnant exploration [31]. This technique guarantees easy access to the biliary tract and in this case the concomitant cholecystectomy can follow the same indications of other restrictive procedures.

The use of gallstone-lowering prophylaxis in bariatric surgery has been a topic of debate for a while. Ursodeoxycholic acid use seems to reduce the gallstone formation rate and consequently biliary complications [32]. There is, however, other evidence that in terms of cost, the routine use of these drugs may not be an effective strategy and the side effects experienced by some patients can lead to very low compliance [33]. Therefore, these medications should be prescribed only in selected high-risk and compliant patients.

23.5 Gallstone Complications

The incidence rate of biliary complications after bariatric surgery is relatively low (0.55% patients/year) and most of these are minor complications such as biliary colic.

Acute cholecystitis, acute pancreatitis and common bile duct migration are rare conditions with an incidence rate of 0.14%, 0.01%, 0.03% every year, respectively [25]. The complication rate tends to be even lower among patients with lower weight loss, such as those subjected to adjustable gastric banding. Other complications—such as hydropic cholecystitis, fistula formation, and Mirizzi syndrome—are only anecdotal events.

In the case a gallstone complication occurring after bariatric surgery, the treatment of choice should be the same as that adopted in the general population. We

strongly recommend directing bariatric patients to high-volume centers with specific bariatric expertise to ensure the correct choice of treatment with better outcome for the patients.

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Emergencies After Bariatric Surgery: The Role of Flexible Endoscopy and Interventional Radiology

24

Antonio Granata, Michele Amata, Valeria Provenzano, and Mario Traina

24.1 Introduction

With the increasing number of bariatric surgery procedures performed, it has been noted that the incidence of post-bariatric-surgery complications is rising. In general, between 4 and 10% of patients who undergo bariatric surgery develop complications within 30 days after surgery, while between 9 and 25% develop complications after 30 days [1, 2]. The postoperative mortality rate at 30 days has been reported as 0.2% after laparoscopic Roux-en-Y gastric bypass (RYGB), 0.5% after open RYGB, and ~0.2% after laparoscopic sleeve gastrectomy (LSG) [3]. In RYGB, complications are frequently at the gastrojejunal junction, with different incidences: bleeding in 1–4%; stenosis in 3–28%; leak in 0.1–5.5% for laparoscopic RYGB and 1.6–2.6% for open RYGB. The most frequently reported complications post-LSG are staple-line complications, which occur in 5% of cases [4].

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24.2 Bleeding

Early gastrointestinal (GI) bleeding occurs after RYGB more than after other bariatric procedures. It usually involves the gastrojejunostomy anastomosis and may be due to an early stoma ulcer at the staple line, which usually heals in 2–3 months if the cause is removed, or to a late marginal ulcer located at the gastrojejunal or jejunojejunal anastomosis, which may be single or multiple and needs a longer healing time (2–6 months) [5]. Early postoperative bleeding occurs in 1–5% of cases after RYGB, and is lower after LSG [6]. Around 50% of early postoperative bleeds are extraluminal while the majority of late bleeds are intraluminal and usually originate from marginal ulcers [7].

24.2.1 Etiology

The etiology of marginal ulceration is multifactorial and depends on surgical technique (reduced perfusion and increased acidity at the anastomosis, use of linear versus circular staplers versus non-absorbable sutures), ischemia, and *Helicobacter pylori* infection [8]. Furthermore, smoking or poorly controlled diabetes may lead to microvascular ischemia, and occult use of medications such as non-steroidal anti-inflammatory drugs (NSAIDs) can contribute to ulcer development. Drugs, such as heparin and clopidogrel are used to prevent thromboembolic events, but the risk of postoperative bleeding using these agents must be balanced against the risk of post-surgical thromboembolic events. Numerous studies have compared the use of different types of heparin associated with lower or higher incidences of acute postoperative bleeding, without a clear indication [9].

24.2.2 Diagnosis and Management

The majority of GI bleeding occurs during or soon after surgery. Up to 70% of bleeds can be diagnosed within 4 h of surgery [7]. Early GI bleeding can be intraluminal, and usually secondary to marginal ulcers, or extraluminal. When extraluminal, abdominal pain and distension associated with a sanguineous abdominal drain output, if present, are indicators of active bleeding. A diagnosis of intraluminal hemorrhage can be made in the presence of hematemesis, melena, or hematochezia. In the case of marginal ulcers, abdominal pain after food intake is the first symptom. Associated signs of bleeding can include tachycardia, hypotension, and a drop in hemoglobin. Endoscopic management of early postoperative intraluminal bleeding is challenging and controversial due to the risk of dehiscence and perforation at the anastomotic site [9]. If indicated, only a skilled endoscopist should perform the procedures with caution, preferably using CO_2 insufflation or using only saline infusion. If such an endoscopist is not available, reoperation is

preferred [10]. In the case of mild and self-limited hemorrhage, endoscopy is usually not necessary but should be considered in severe bleeding (hemodynamic instability and/or loss of about 2 g of hemoglobin) or when rebleeding occurs after conservative management (proton-pump inhibitor and sucralfate infusion, discontinuation of antithrombotic prophylaxis). Gastrojejunostomy bleeds after RYGB are approached using a standard upper endoscope. Jejunojejunostomy or excluded stomach bleeds require an enteroscopic approach. Signs of bleeding at the gastrojejunostomy include active oozing (48%), visible vessel (26%) and adherent clot (26%), which must be aspirated in order to evaluate an ongoing hemorrhage [7]. Standard endoscopic therapy includes epinephrine injection, heater probe coagulation such as argon plasma coagulation, mechanical therapy such as metallic endoclip placement, or the combination of these techniques [11]. The use of endoclips is favored when technically possible because they do not produce additional tissue injury and, at the same time, they can be used to manage concomitant anastomotic leaks and iatrogenic perforations. The use of a hemostatic mineral powder is useful in the case of diffuse bleeding from a large area [12] but it has to be considered with caution in cases of suspected perforation. Interventional radiology, if locally available, is considered an option when standard endoscopy fails to detect or stop the bleeding or in the case of hemodynamic instability. Angiographic embolization, however, can potentially reduce blood supply to fresh staple lines leading to an anastomotic leak. In this scenario, we suggest a schematic flowchart to use in emergencies (Fig. 24.1).

24.3 Leak

Leaks are defined as the exit of luminal contents due to a defect of the intestinal wall at the anastomotic site, while fistulas are abnormal connections, usually between two hollow viscera or communicating with the skin, which result from chronic healing of local inflammation caused by leaks [13]. Depending on the time of occurrence, leaks are classified as early (<14 days postoperative), intermediate (2–6 weeks), and late or delayed (>6 weeks).

24.3.1 Etiology

Leaks are usually caused by ischemia at the staple line, with a progressively decreasing frequency at the gastrojejunostomy, gastric pouch and jejunojejunal anastomosis. In the case of LSG, an increase in intraluminal pressure, pyloric dysfunction and twisted or atonic sleeve are the principal causes of leaks, but ischemia can also be involved. Indeed, ligation of the short gastric arteries and cardio-tuberosity branch can contribute to local ischemia and promote chronic inflammation [14].

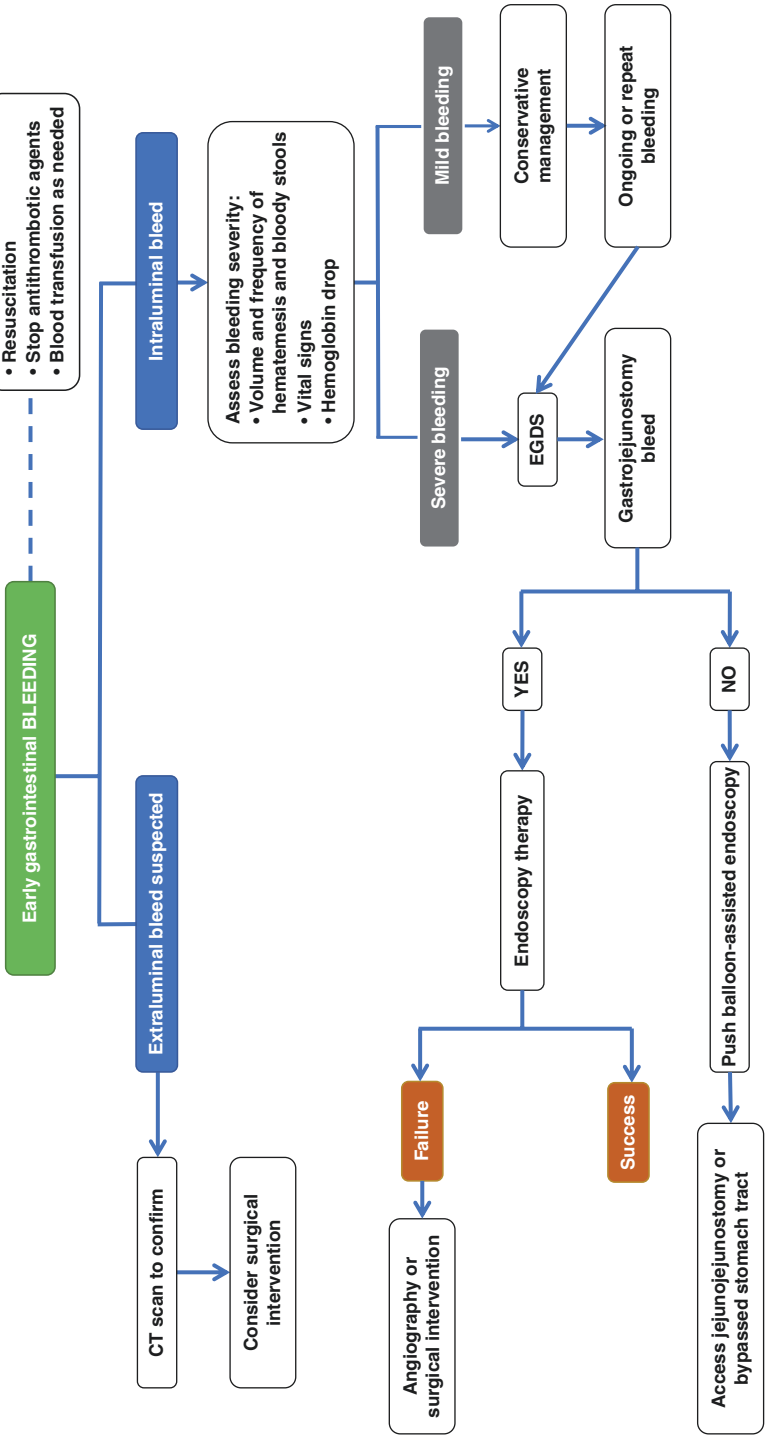

Fig. 24.1 Proposed therapeutic algorithm for early gastrointestinal bleeding. *CT* computed tomography, *EGDS* esophagogastroduodenoscopy

24.3.2 Diagnosis and Management

The clinical presentation of leaks is variable, ranging from minor symptoms to multiorgan failure. Early suspicion of a leak must be raised in cases of any deviation from a standard postoperative course.

If a surgical drain is in place, leaks can be diagnosed by the characteristics of the fluid collection in the drain reservoir [15]. Generally, patients present nausea, vomiting, epigastric pain and abdominal distension. There can also be hypotension, tachycardia (the most sensitive indicator of a leak), fever, leukocytosis, and an increase in inflammation markers. Later in the postoperative period, especially after complete removal of the postsurgical abdominal drains, the diagnosis of leakage is much more challenging. Usually, if the patient develops tachycardia, hypotension, and fever that is unresponsive to empirical antibiotics, this can be the clinical manifestation of a walled-off extra-digestive collection (Fig. 24.2). However, if a leak is suspected the patient should undergo radiologic (barium swallow, computed tomography) or endoscopic evaluation.

Preoperative resuscitation and management in the ICU should be reserved for physiologically compromised patients. Supportive therapy based on fasting, parenteral nutrition, and an infusion of broad-spectrum antibiotics is implemented. According to the recommendations suggested by an expert panel during a 2011 consensus conference [15], only unstable patients with evidence of leak should immediately undergo (48–72 h postoperatively) surgical exploration for peritoneal irrigation, abdominal cleansing, and identification and repair of the defect. Reinterventions such as gastrectomy or gastric bypass after a complicated LSG are associated with less morbidity than salvage surgery on acute leakages, when performed for a more chronic defect [16]. Radiology with percutaneous drainage is an option if an undrained abdominal cavity is detected [17]. Based on recent evidence, endoscopy is becoming the first-line treatment, and is preferred due to its non-invasiveness [18]. Endoscopic management can cover or exclude, close or drain the defect.

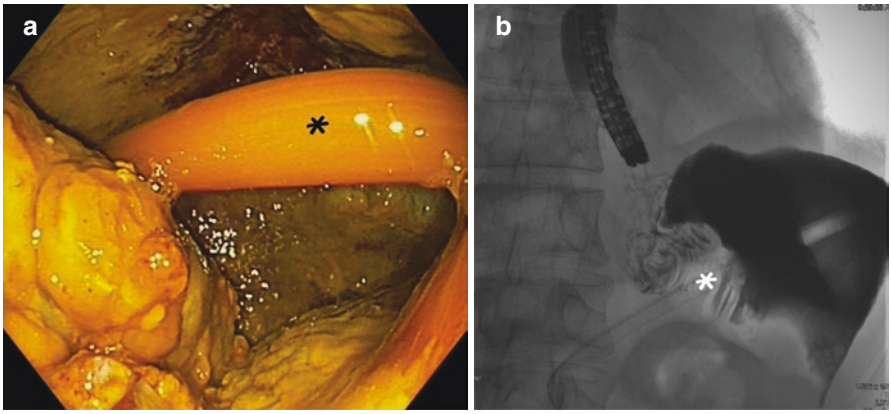

Fig. 24.2 Endoscopic (**a**) and fluoroscopic (**b**) visualization of a large infected extra-digestive collection in a post-RYGB leak with a percutaneous drainage tube (*asterisk*) in place. *RYGB* Roux-en-Y gastric bypass

24.3.2.1 Internal Drainage

Drainage and cleansing of the extra-digestive space is part of the initial treatment strategy for every leak. If still in place, surgical drains are maintained or repositioned in order to remove mucous fluids from the leak margins and promote healing. If an undrained collection is present, external drainage must be established surgically or radiologically.

In patients with delayed leak, endoscopic internal drainage (EID) of a paragastric collection in the digestive lumen can be a good alternative. This approach consists of endoscopic insertion of a pigtail nasocystic catheter or, preferably, one or two double pigtail stents in the collection (Video 24.1 shows the "drain" technique in a leak following a mini-gastric bypass: through placement of pigtail plastic stents, endoscopic internal drainage allows direct reabsorption of the wall and adjacent collection). After clinical and radiological confirmation of proper evolution, oral diet can be rapidly resumed [19]. This kind of drainage has often been described as a bridge or a complement to other endoscopic techniques, but it can also be used as an isolated modality. Donatelli et al. found a high-resolution rate (91%) with EID in a group of 33 patients who underwent RYGB, with a mean removal time of 61 days and at a follow-up of 10 months [20]. In another retrospective monocentric study, Lorenzo et al. analyzed a group of 100 patients with a leak after LSG to compare EID versus a stenting and clipping technique. The group treated with EID had higher resolution rates. An interval of >21 days between fistula diagnosis and the first endoscopy, large (>5 cm) and gastrobronchial fistula, sepsis, and previous surgical revision were associated with a failure of endoscopic treatment [19].

Recently introduced for the treatment of walled-off pancreatic necrosis, the novel lumen-apposing metal stents (LAMS) are an alternative solution for endoscopic ultrasound-guided drainage of well-circumscribed extra-digestive collections within 10 mm from the GI wall. This technique avoids the risk of external fistula formation and the secondary infection that can appear in the case of percutaneous drainage. However, experience is still scarce [21].

Another technique for internal drainage of infected cavities is based on the endoscopic insertion into the cavity of a sponge connected transnasally to an external vacuum system. The open-cell sponge stimulates tissue granulation, and the vacuum pressure optimizes cleansing of the cavity. Endoscopic replacement of sponges is necessary every 2–4 days until complete reabsorption of the collection, but requires a long therapeutic period and increases costs for the health system. The results are equivalent to the other techniques, especially in abscesses and leaks after major gastroesophageal surgery [22]. Leeds et al. found a 100% rate of resolution (9/9 patients) in the management of post-LSG leak, but the mean number of procedures was 10.3 for regular replacement of the sponge [23].

24.3.2.2 Covering

According to evidence in the literature, endoscopic stent placement has a high success rate: 76% in post-RYGB, and 73% in LSG [24]. Different types of stents can be used to cover the leak: fully or partially covered self-expandable metal stent

(SEMS) with different release modalities (through-the-scope or usually over-the-wire), with or without anti-migration system.

Stent placement reduces intraluminal pressure, which is one of the causes of leakage. By excluding the fistula, the covered stent prevents any fluid leak, supports cell growth and, consequently, promotes the closure. In addition, SEMS are easier to insert because of the small caliber and the higher flexibility of the delivery system and, when in place, they permit resumption of oral nutrition intake in approximately 3–7 days.

In the case of over-the-wire SEMS release, the endoscope is advanced well into the Roux limb (in post-RYGB patients) or to the third portion of the duodenum (in post-SG patients), and a guidewire is introduced. The leak site and squamocolumnar junction are marked with placement of an external marker under fluoroscopy, and the endoscope is removed. The stent delivery system is inserted under fluoroscopy (by using a stiff guidewire if acute angulation must be traversed). The stent should be deployed distally enough from the upper esophageal sphincter to avoid globus syndrome. The distal end of the stent should not impact the enteral wall because bleeding, ulceration, or perforation can result. Adjustment of the stent position can then be performed endoscopically using forceps. Common complications include transient nausea, vomiting, chest pain that irradiates to the back caused by stent expansion, and stent migration. Stents are usually left in place for 2–8 weeks because a longer stenting period can lead to extraction difficulties or complications [17].

One of the most important complications of this procedure is migration, which can require emergency surgery if the device migrates into the small intestine, causing obstruction. A higher migration rate is observed in the fully-covered SEMS (FCSEMS) compared with partially covered (PCSEMS). These induce tissue hyperplasia, which occurs in the uncovered meshes, promoting water tightness, with diminished risk of migration. However, tissue ingrowth makes stent removal difficult and increases the risk of bleeding, mucosal stripping, and perforation. To solve these problems, the stent-in-stent technique has been introduced [25], which uses the argon plasma coagulation (APC) action to ablate the hyperplastic tissue, with limited results. More recently, a large-bore over-the-wire fully-covered metal stent with a double antimigration system (Beta stent; TaeWoong Medical Co, Gyeonggi-do, South Korea) ensures optimal adherence to the luminal wall and, according to the specific architecture designed, prevents migration (Fig. 24.3). This stent has been used in several studies, but the available data are not conclusive. Tringali et al. had good results, with complete healing of leaks after bariatric surgery in 100% (10/10 patients) of cases after the second endoscopic treatment [26]. Other groups have introduced a novel technique by attaching, with clips or an endoluminal suturing system (Fig. 24.4), the proximal flanges of FCSEMSs to the esophageal wall, with a stent migration rate of less than 10% [27], and a low suture-related rate of adverse events (3.7% of cases) [28].

24.3.2.3 Closing

Instead of covering the leak and leaving the defect to close on its own, several authors have reported techniques for directly closing the wall discontinuity. The

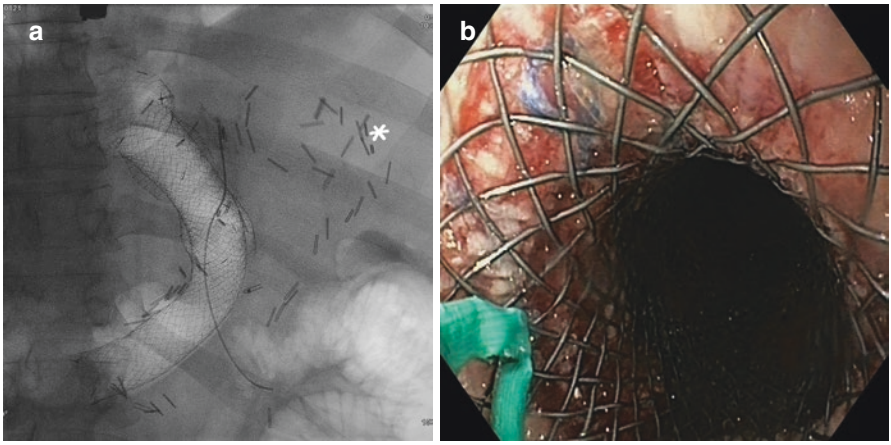

Fig. 24.3 (**a**) Radiographic evaluation of a large-bore over-the-wire fully covered metal stent with a double antimigration system (Beta stent) for the treatment of a post-LSG leak: multiple surgical clips on the resection area (asterisk). (**b**) Endoscopic view of the proximal flange of the stent. *LSG* laparoscopic sleeve gastrectomy

OverStitch Endoscopic Suturing System (Apollo Endosurgery Inc., Austin, TX, USA) has been shown to be effective in leak management by placement of full-thickness sutures through a flexible dual channel endoscope (Fig. 24.5) [29]. In the largest multicenter retrospective study, of eight American centers, Sharaiha et al. found high technical and clinical success (97.5%) for the closure and stent anchoring in a total of 122 patients with postsurgical GI defects [30]. There are also many case reports and series reporting safe resolution of leakage in bariatric patients with endoluminal suture alone [31, 32] or in combination with covering techniques [33].

Use of regular endoscopic clips is another way of closing the GI wall defect, though, as reported in several case reports, the use of this device is limited in post-bariatric leak. Clips are used to reach the tissue surrounding the defect to close the leak. The clip should be deployed perpendicular to the long axis of the defect. If necessary, multiple clips can be used. Thermal ablation or mechanical scraping of the tissue before releasing the clips results in a more resistant seal [17].

Another approach to close the leak is the Over-The-Scope Clip (OTSC—Ovesco Endoscopy AG, Tubingen, Germany), an elastic, nitinol clip that is biocompatible. It facilitates a full-thickness tissue closure through teeth arranged in a "bear-trap" fashion. The actual application and deployment of the clip is similar to that of a band ligature device, with which most endoscopists are familiar, with the addition of a grasping device (used primarily for larger defects), which can be used to pull the defect and/or mucosa into the cap before clip deployment. This feature, together with suction, maximizes the potential for effective closure [34]. Keren et al. found resolution of leakage in 21/26 patients with sleeve gastrectomy, with an overall success rate of 80% [35].

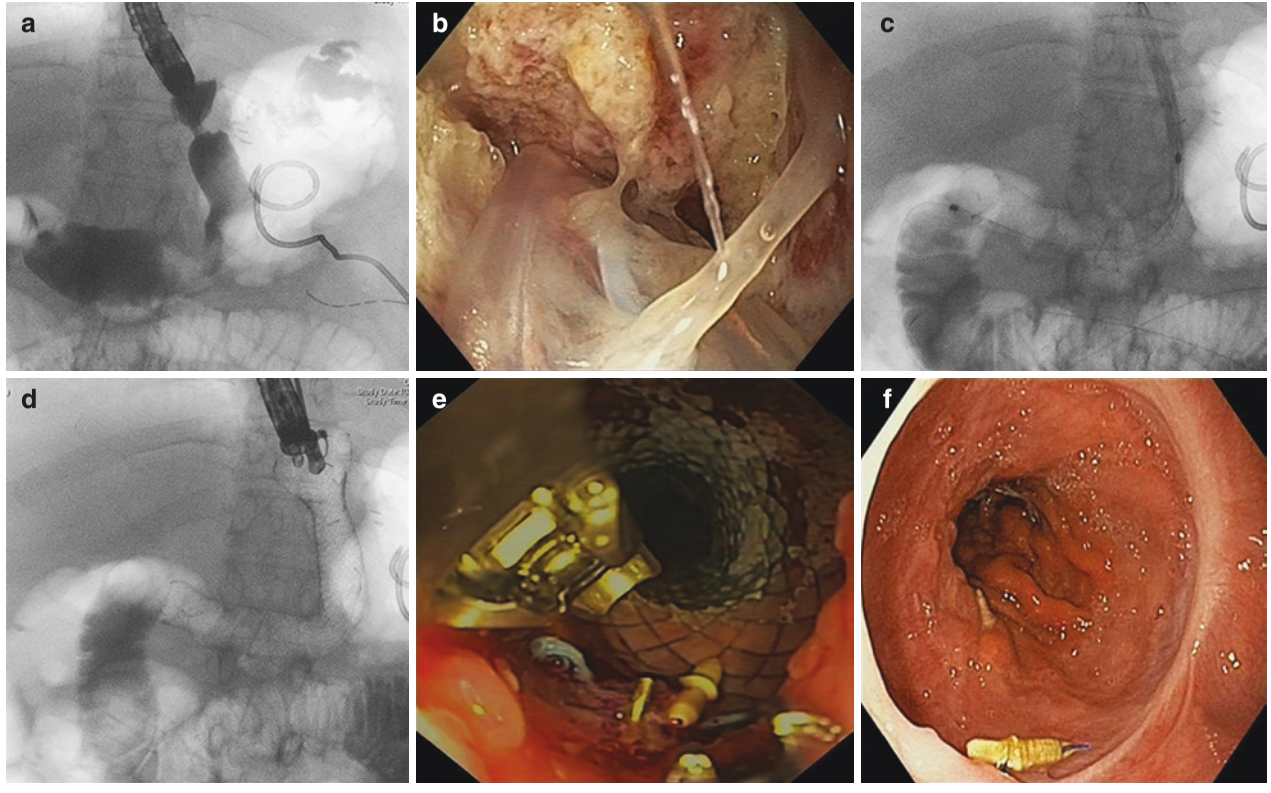

Fig. 24.4 Post-LSG leak with an infected collection drained by a percutaneous drainage. (**a**) Fluoroscopic view. (**b**) Endoscopic view. (**c**) Over-the-wire release of FCSEMS in order to "cover" the wall defect. (**d**, **e**) Anchoring of the proximal flange of the stent with an endoluminal suturing system. (**f**) Endoscopic view at 2-month follow-up. *LSG* laparoscopic sleeve gastrectomy, *FCSEMS* fully covered self-expandable metal stent

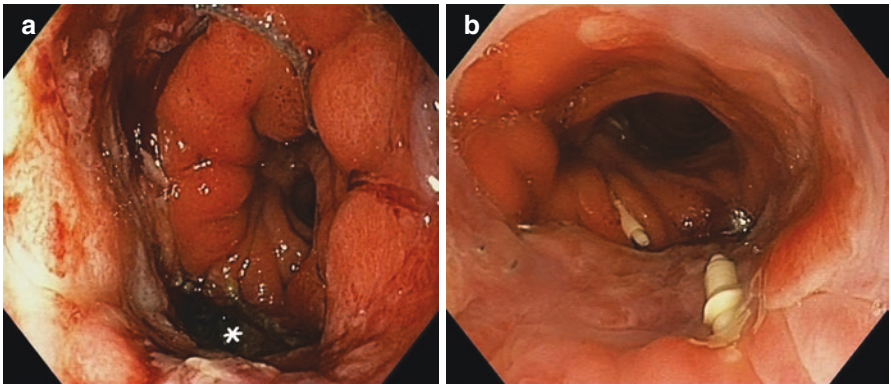

Fig. 24.5 The "closing" technique: post-RYGB leak treated with the OverStitch Endoscopic Suturing System. (**a**) Time zero. (**b**) 2-month follow-up. *RYGB* Roux-en-Y gastric bypass

Table 24.1 Overview of endoscopic techniques used in the treatment of leak after bariatric surgery

Technique	Pros	Cons
EID (to drain)	Technically easier Low cost of device Only two procedures required High success rate	Small effect if there is large or infected collection Many negative predictive factors Possible collateral effect
Endosponge (to drain)	Success rate (80%) Validated clinical studies	Sponge substitution every 2–4 days Longer hospitalization Increased cost for health system
Stent (to cover)	Easy technique Variable efficacy	Migration (20–40%) Post-procedural complications (bleeding <5%, obstruction or kinking <30%, incarceration <8%, low tolerability <10%)
Endoluminal suture (to close)	Full-thickness closure for large wall defects (>10 mm)	Requires skilled endoscopist Limited data
OTCS (to close)	Full-thickness wall closure (<10 mm) Good outcomes Few hospitalization days	Increased failure in chronic leak Conflicting data

EID endoscopic internal drainage, *OTSC* Over-The-Scope Clip

24.4 Conclusion

Therapeutic options and measures for the treatment of leak are summarized in Table 24.1 and Fig. 24.6.

A combination therapy might be the best approach, in our opinion, if a drain is in place allowing for a faster stent removal (2 weeks). Video 24.2 shows a post-LSG leak

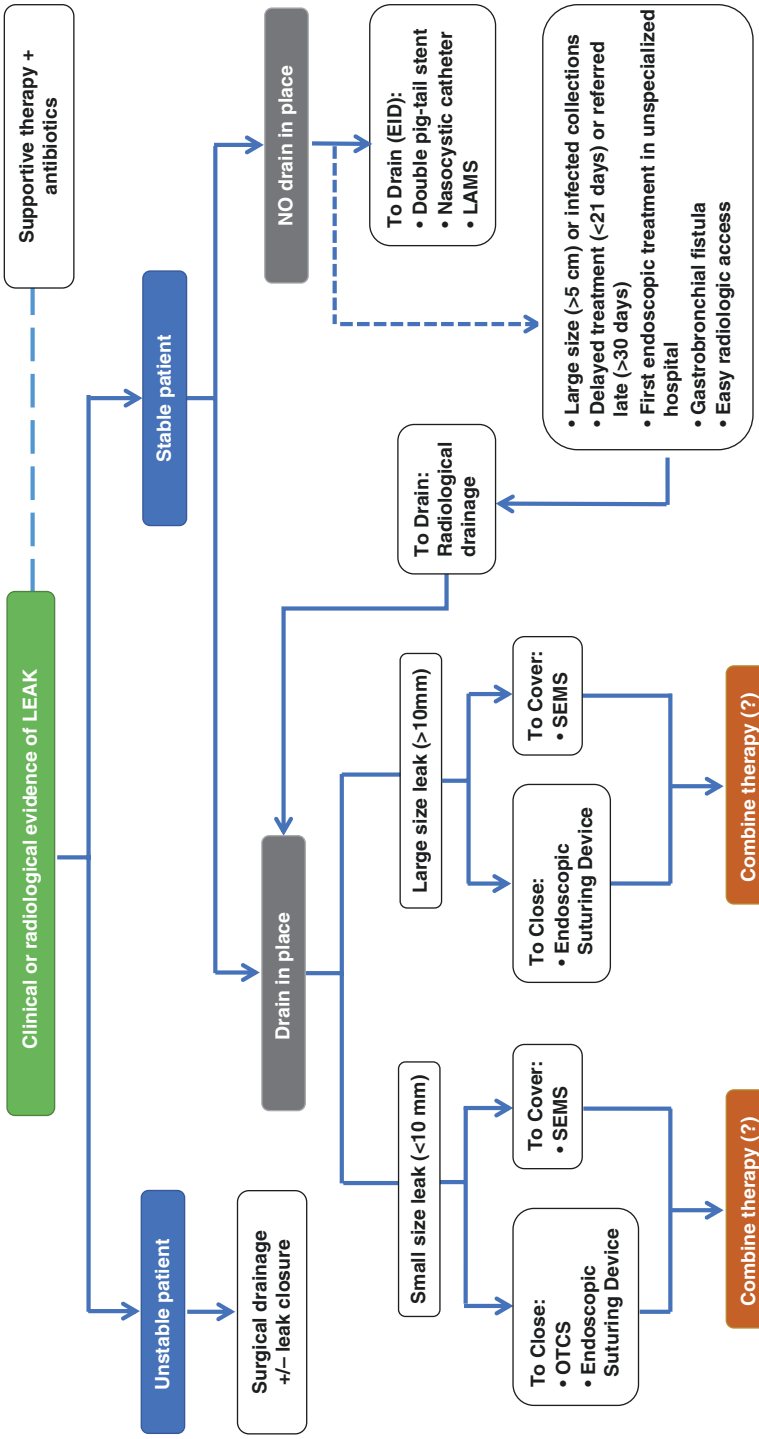

Fig. 24.6 Proposed therapeutic algorithm for leakage. *SEMS* self-expandable metal stent, *OTSC* Over-The-Scope Clip, *LAMS* lumen-apposing metal stent

successfully treated with a combination therapy. After visualization of the wall defect, the endoluminal suturing system permits "closing" and the FCSEMS avoids the recurrent contact damage, providing better local conditions for a faster and solid healing.

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Accreditation of the Surgeon in Emergency Bariatric Surgery

25

Valerio Ceriani, Ferdinando Pinna, and Marta Tagliabue

In a constant effort to enhance patients' clinical results and their overall safety, the American Society for Metabolic and the Bariatric Surgery (ASMBS) in 2004 and the American College of Surgeons (ACS) in 2005 independently prompted in the USA the identification and accreditation of bariatric surgery Centers of Excellence (COE) and in 2013 both programs merged into the Metabolic Bariatric Surgery Quality Improvement Program. In Europe, a similar policy was implemented by the International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO) in 2008 [1].

Despite a few differences between North America and Europe, the accreditation of bariatric centers focuses on the presence of surgeons with previous formal training in bariatric surgery, adequate facilities and infrastructural resources, organizational processes and an adequate annual case volume.

Today, in spite of some initial controversies [2], a vast amount of scientific data supports the accreditation of bariatric centers and the identification of COEs [3–5].

By systematically reviewing the relation between case-volume and surgical outcomes in bariatric surgery—with a total of 458,032 patients—Zevin et al. found evidence of improved patient outcomes for increasing surgeon volumes and, even though with lower strength, a negative relation between increasing hospital case-volume and patient postoperative complications [3].

Markar et al. provided a pooled-analysis of 15 studies about the relation between hospital and surgeon case-volume and patient outcomes [4]. The analysis showed a significant volume-outcome relation for both hospital and surgeon, though a very high level of statistical heterogeneity was observed. It was not possible to identify a specific volume threshold for outcome improvement.

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More recently, Azagury et al. conducted a systematic review of the effects of the accreditation process in the USA on patient outcomes [5]. Of the 13 studies included in the review, 10 showed a significant benefit of bariatric COE accreditation on the patients' postoperative mortality and 8 out of 11 studies showed a significant reduction in postoperative morbidity related with COE accreditation.

Although the overwhelming majority of scientific data on the subject comes from the USA, some experiences from Italy, Germany and England seem to confirm the positive effects of the accreditation of bariatric centers on early postoperative results [6–8].

All these observations ultimately pertain to the context of elective bariatric surgery. Obese patients, however—in consideration of their anatomical, pathophysiological and semiological features—pose specific types of problems even in the context of emergency surgery. This raises the question whether dedicated centers may offer improved outcomes to obese patients even in the context of emergency surgery. The answer could appear obvious for obese patients who reach the emergency department (ED) with acute complications of a bariatric procedure, although the issue has not been formally addressed in the literature. A typical case could be that of a subcardial fistula after a sleeve gastrectomy, where, besides surgical expertise, a multidisciplinary approach with specialized radiologic and endoscopic skills is of paramount importance to achieve the best results [9–11].

In effect, some indirect evidence to support these considerations can be found in some large-scale studies. Gebhart et al. conducted a retrospective cohort analysis of surgical outcomes of bariatric patients registered in the Nationwide Inpatient Sample (NIS) database, electively operated in accredited (ACs) versus non-accredited centers (NACs) between 2008 and 2010 [12]. The findings showed that, in spite of a similar incidence of serious postoperative complications (5.3% and 4.5%, respectively), in-hospital mortality was significantly reduced in ACs (0.08%) compared to NACs (0.19%; OR 3.1). Patients with moderate-to-severe illness at hospital admission had a significantly lower in-hospital mortality in ACs versus NACs (0.17% and 0.45%, respectively). Very interestingly, somewhat similar results were observed by Jafary et al. in comparing high-volume bariatric NACs and ACs [13]. By studying data of the same NIS database related to the period 2006–2010, the authors observed a significantly higher in-hospital mortality in high-volume NACs versus high-volume ACs (OR 3.57), in spite of a reduced incidence of postoperative complications in NACs (OR 0.84). These observations suggest that dedicated centers, in which specialized expertise has been achieved, may offer better results in the management of postoperative bariatric complications.

Nevertheless, the proper management of some clinical conditions needs to be arranged in a very limited time lapse. For example, the management of occlusive complications after a gastric bypass or acute obstruction of the alimentary limb in a biliopancreatic diversion both need a very rapid surgical approach to prevent ischemic injury to the occluded segment [14, 15] and a level of awareness that only a dedicated bariatric surgeon can offer, once such an injury has already occurred. In these cases, referral to a dedicated center may not be possible or may lead to a significant delay in therapy.

In a recent observational study from Wisconsin, approximately one-third of the post-bariatric patients presented to an ED during the first postoperative year, and only one-third of the ED visits were at the same hospital where the bariatric procedure had been performed [16]. Thus, it becomes clear that a significant number of patients might be evaluated and managed for post-bariatric emergency conditions outside a dedicated bariatric center. The literature lacks a formal evaluation of surgical outcomes in this subset of patients, but we think that there could be no reason to hypothesize *a priori* significant differences from what has been considered above.

Other considerations pertain to emergency non-bariatric surgery in obese patients. In this context, it could be hypothesized that the availability of specific skills and expertise from a dedicated bariatric team may translate into better postoperative outcomes even in the context of general emergency surgery.

In effect, very few studies have been published on the topic. Only marginally pertinent to the issue, but containing some useful clues, the same paper by Gebhart et al. cited above reported a subgroup analysis of the postoperative results of morbidly obese patients operated on for elective general surgical procedures [12]. The authors reported an increased risk of serious postoperative complications for morbidly obese patients operated on in NACs versus ACs with respect to laparoscopic cholecystectomy (OR 2.36) and antireflux surgery (OR 2.03), while the postoperative outcomes related to colectomy were not different between groups (OR 1.11, *p* not significant).

Only one paper directly addresses the issue, by retrospectively comparing the postoperative outcomes of emergency general operations on obese patients performed by bariatric and non-bariatric surgeons [17]. The results were similar between groups, with only a slightly more frequent laparoscopic approach in non-routine cases and shorter hospital stays in bariatric surgeons' patients.

In conclusion, we think that the available scientific evidence is too limited to allow any conclusion regarding the need to specifically accredit surgeons and facilities for emergency surgery on obese patients. Nevertheless, we recognize that the topic has practical interest and warrants further investigation.

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Litigation After Bariatric Surgery

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Ludovico Docimo and Salvatore Tolone

Severe obesity is becoming an epidemic that affects a considerable portion of the population worldwide [1]. This is leading to health, social, and economic problems, with a shortened life expectancy, a decreased quality of life, and increased health expenditure for the national health systems [2]. However, despite incontrovertible evidence documenting not only the genetic component of obesity but also the link between obesity and metabolic, cardiovascular and general comorbidities, there is a popular belief (sometimes also supported by physicians) that obesity can be considered a simple weight gain rather than a true disease, and that if the patient had more willpower, he or she could control the process. Traditional treatments, such as diet and behavior modification, are often frustrating because of a small weight loss in most patients and a high rate of recidivism [3]. In this context, bariatric surgery has been demonstrated to achieve an optimal weight loss that is stable in time, while reducing associated comorbidities and the related health expenditure. Over the last 10 years there has been a increasing trend in the utilization of bariatric surgery worldwide, with more than 60,000 procedures performed yearly [4], with optimal results and low postoperative complication and mortality rates, especially when the surgery is performed in centers with high volume and expertise in the field [4]. However, even if low complication and mortality rates are the goal of all surgical specialties, these improvements have led to increased expectations and disappointment, as patients and relatives felt that the unavoidable complications or consequences of surgery might have been caused by not receiving an adequate diagnosis and/or treatment. As a result, the number of negligence claims after surgical procedures has increased over the last 30 years. The reasons for this include a cultural shift in attitudes toward the medical profession and the growth of the legal services

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industry, with a significant financial impact for both healthcare workers and facilities and with a significant psychological impact on the physicians.

There are many published papers regarding litigation claims in different specialties [5, 6]. In order for a claim to succeed the patient must prove that four consequential situations have occurred: he/she was owed a duty of care, the duty was breeched, the breech caused or contributed to damage, there was loss or damage as a consequence. An interesting paper from the English National Health Service (NHS) [7] investigated claims over a 10-year period among ten surgical specialties in England (cardiothoracic, general, neurosurgery, obstetric, oral and maxillofacial, orthopedic, otorhinolaryngology, pediatric, plastic, urology and vascular surgery). Over this period, a total of 31,697 claims were made, of which 13,661 (43.1%) were successful; 8433 (26.6%) unsuccessful and 9603 (30.3%) were open at time of data request. The number of claims continued increasing until 2011, whereas they started to decrease in 2013. Likewise, the proportion of successful claims to the claimant decreased over time. Orthopedic, obstetric and general surgery were the three surgical specialties that consistently received the largest number of claims, with a total expense of £1.5 billion paid by the NHS in damages and legal fees combined.

Given the specific difficulties of bariatric surgery, especially as regards the peculiarity of the obese patient (with the increased risk for comorbidities), one could speculate that this subspecialty of general surgery is exposed to many claims. However, few data exist about litigation in bariatric surgery.

As done for all surgical specialties, the UK NHS investigated the claims filed after bariatric procedures [8]. Collecting data from their National Bariatric Surgical Registry, the authors found a very low complication and mortality rate, with an estimated survival of 99.9%. Then, looking at litigations from January 2003 to December 2013, they found a total of 7 claims, of which 4 were successful, with a total payout amount of £210,000. Among the four successful claims, two were caused by inappropriate timing/delay of treatment, one for retained instrument and the last for inappropriate informed consent; surgical complications were among the unsuccessful claims. A further comparison between bariatric surgery claims and claims relating to other surgical specialties showed that bariatric surgery had the lowest incidence of litigation.

Similar data were reported in the first survey on medical malpractice in bariatric surgery in the US in the late 90s [9]. The authors sent surveys to all surgeons affiliated to the American Society for Bariatric Surgery; a total of 93 claims were recorded, 54 following Roux-en-Y gastric bypass (RYGB) and 32 following vertical banded gastroplasty (VBG). Eight additional lawsuits were filed following some types of malabsorptive procedure and six after revisions. The prevalence rate was very low (0.00126 for RYGB and 0.00163 for VBG). Interviewed surgeons also listed the patients' reasons for the claims: "pain and suffering" was the most common reason followed by death, an unsatisfactory result, or infection. Another 18 reasons were reported, such as "lack of informed consent," "malignant hyperthermia," and a variety of other accusations ranging from "mercury poisoning" to "seduction". The most common surgical complications that led to the lawsuit was leakage at the intestinal anastomosis (gastrojejunal or jejunoileal), followed by

death caused by sepsis or embolism. Finally, at the time of publishing the study, among the initial 93 claims, 10 (10.7%) suits were dismissed as frivolous, 1 (1%) additional suit was dismissed with summary judgment for the defendant, 21 (22.6%) suits were abandoned, 36 (38.7%) were still open, with only 19 (20%) settled for the plaintiffs.

Subsequently, in an analysis of litigation trends in bariatric surgery in the US carried out to prevent further lawsuits and improve patient care, a total of 100 consecutive bariatric lawsuits (from 1997 to 2007) were reviewed by a consortium of experienced bariatric surgeons and an attorney specializing in medical malpractice [10]. Of the 100 lawsuits, 45% were reviewed for defense attorneys. In 42% of cases, the surgeons had less than 1 year of experience, whereas only a small difference was observed for the volume of procedures (26% with less than 100 procedures performed vs. 38% with more than 300 procedures). The surgical procedures were performed between 1997 and 2005 and included RYGB (78% total, 33% open, and 45% laparoscopic), VBG (3%), mini gastric bypass (6%), biliopancreatic diversion/duodenal switch (4%), and revision (9%). Of the 100 cases, 32% involved an intraoperative complication and 72% required additional surgery. The most common adverse events initiating litigation were leaks (53%), intra-abdominal abscess (33%), bowel obstruction (18%), major airway events (10%), organ injury (10%), and pulmonary embolism (8%). From these injuries, 53 patients died, 28% had a full recovery, 12% had a minor disability, and 7% had major disabilities. Evidence of potential negligence was found in 28% of cases. Of these cases, 82% resulted from a delay in diagnosis and 64% from misinterpreted vital signs.

Even though the analysis of 10-year trends from the late 90s seemed encouraging, after 2007 the use of bariatric surgery increased resulting in a parallel increase in overall claims. Weber et al. [11] analyzed the claims trend from 1990 to 2009, comparing the first and second decade. They found that from 1990 to 1999, 198 (95.5%) claims were recorded, of which 189 were associated with the ICD-9 code for obesity and 9 (4.5%) with the ICD-9 code for morbid obesity. On the contrary, from 2000 to 2009, 377 claims were recorded; among them 128 (33.9%) were associated with the ICD-9 code for obesity and 249 (66.1%) with the ICD-9 code for morbid obesity. RYGB was the most involved procedure, but there was no difference in incidence in the two periods. Notably, even though the total number of claims increased, the total number of successful claims diminished from 56 to 38%.

These data were recently confirmed by Choudry et al. [12], who studied the 140 recorded cases of medical malpractice in the US ranging from 1986 to 2015. The majority of reports belonged after 2004 (77%) and concerned RYGB (75%); the claims were fully successful for plaintiffs in only 21% of cases, with another 18% of cases in which a settlement was reached. Notably, the most common alleged reason for a malpractice claim was delay in the diagnosis or management of a complication in the postoperative period ($n = 66$, 47%), the most common of which was an anastomotic leak (45%, $n = 34$). Death was reported in 74 (52%) cases.

In Europe, similar trends of growing numbers of claims being filed have also been recorded, even though only few data are present in International Indexed database. In France, bariatric surgery has grown spectacularly in recent years, with a

number of procedures that has tripled from 15,000 to 43,000 between 2002 and 2013; on the other hand, this phenomenon has left litigation in its wake, the cost of which has tripled over 10 years [13]. It has been reported that bariatric surgery caused 129 deaths in 2013 and currently represents approximately 25% of all complaints from visceral surgery [14]. Furthermore, the majority of cases ended with the judge statement that no medical error had taken place, whereas 30% of complaints were based on negligence errors. Notably, most of these were secondary to diagnostic delay, usually because physicians and particularly the surgeon had underestimated potentially present clinical signs (e.g. advising that postoperative abdominal pain was "normal", delaying further examinations).

In Italy, there is a similar perception of litigation in bariatric surgery. However, official scientific data are not currently available, and we must rely on data provided by the local Health Agencies, which report similar trends of a massive increase in claims after surgical procedures (estimated 34,000 cases every year for all medical specialties, for a total of 2.5 billion of Euros claimed), an increase in bariatric surgery utilization and a parallel increase in claims. On the other hand, and similarly to other countries, the rate of successful claims should not exceed 20% of cases. Nevertheless, we should consider that in Italy claims for death and the consequences of surgery (considered sometimes as personal injuries) can first be filed under the penal code and then under the civil code, subjecting the surgeon to frustrating trials (up to 6!), with huge personal legal costs; in other countries medical claims are only filed as civil cases and there is an increasing use of amicable, non-legal settlements of medical complaints.

These data lead to several considerations. Worldwide there has been an increasing trend in claims, probably due to the patients' changed perception of physicians, who are perceived as acting against, rather than for, the patients' wellbeing. Also, common unavoidable complications are no longer tolerated, as if taking on the care of a patient were equal to guaranteeing a "perfect outcome", which is not possible for a science like medicine. These hypotheses are supported by the large number of claims and the low rate of successful claims (usually around 20%). However, exposing surgeons to such a large amount of lawsuits leads to an attitude known as "defensive medicine", in which physicians, for fear of possible claims, prescribe additional unnecessary testing (e.g. labs, imaging, and so on), and treat patients with the procedure exhibiting lower complication rates. This results in a greater expense for the health system as well as in possibly less effective treatments for the patients.

It should also be remembered that although claims are increasing in bariatric surgery, this increase is occurring in parallel with the rise in the utilization of such procedures. However, even though the successful claims are few, most of them relate to non-specialized centers or low-volume centers, and most of them concern a delay in the appropriate treatment. Thus, the emerging role of specialized training in high-volume hospitals is crucial. Many countries worldwide have created an accreditation program for high-volume and quality centers, as well as fellowship programs, in order to reduce the rate of complications due to inexperience.

Another crucial factor is the high rate of successful claims related to delays in treatment/diagnosis. Although appropriate treatment or timing is somewhat arguable and should be considered on a single-patient basis, we believe that proper guidelines developed by scientific societies could be helpful in providing a clinical pathway. While the existing guidelines provide information about indications or contraindications in particular patient settings, we believe they should be improved by providing information about clinical pathways in difficult cases or after specific complications. One example could be suture line leakage after sleeve gastrectomy, for which there are no features or indications about treatment with a conservative approach, or by placing a stent or by endoscopic drainage.

In conclusion, litigation after bariatric surgery is increasing due by rise in its utilization, but with a low rate of successful claims. These are usually related to delay in treatment/diagnosis. In this setting, specific training and high-volume accredited centers, as well as an effort to develop new guidelines for particular clinical pathways, are warranted in order to reduce medical lawsuits.

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