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Chapter

Surgical and Interventional Management of Complications Caused by Pancreatitis

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Abstract

Acute pancreatitis has a broad clinical spectrum: from mild, self-limited disease to fulminant illness resulting in multi-organ failure leading to a prolonged clinical course with up to 30% mortality in case of infected necrosis. Management of local complications such as pseudocysts and walled-off necrosis may vary from clinical observation to interventional treatment procedures. Gram negative bacteria infection may develop in up to one-third of patients with pancreatic necrosis leading to a clinical deterioration with the onset of the systemic inflammatory response syndrome and organ failure. When feasible, an interventional treatment is indicated. Percutaneous or endoscopic drainage approach are the first choices. A combination of minimally invasive techniques (step-up approach) is possible in patients with large or multiple collections. Open surgical treatment has been revised both in the timing and in the operating modalities in the last decades. Since 1990s, the surgical treatment of infected necrosis shifted to a more conservative approach. Disruption of the main pancreatic duct is present in up to 50% of patients with pancreatic fluid collections. According to the location along the Wirsung, treatment may vary from percutaneous drainage, endoscopic retrograde pancreatography with sphincterectomy or stenting to traditional surgical procedures. Patients may suffer from vascular complications in up to 23% of cases. Tissue disruption provoked by lipolytic and proteolytic enzymes, iatrogenic complications during operative procedures, splenic vein thrombosis, and pseudoaneurysms are the pathophysiological determinants of bleeding. Interventional radiology is the first line treatment and when it fails or is not possible, an urgent surgical approach should be adopted. Chylous ascites, biliary strictures and duodenal stenosis are complications that, although uncommon and transient, may have different treatment modalities from non-operative, endoscopic to open surgery.

Keywords: pancreatic pseudocysts, walled-off necrosis, infected pancreatic necrosis, disconnected pancreatic duct syndrome, vascular complications, chylous ascites

1. Introduction

The majority of patients suffering from acute pancreatitis will have a mild, self-limited and uncomplicated course. Pancreatic necrosis may develop in up to

10%-20% of patients, because of insufficient perfusion of pancreatic parenchyma to support metabolic requirements, leading to a prolonged clinical course with up to 30% mortality in case of infected necrosis [1]. Local and systemic complications, mild or life-threatening, such as pancreatic and/or peripancreatic fluid collections, walled-off necrosis, infected pancreatic necrosis, disconnected pancreatic duct syndrome and vascular complications can occur. The successful management of these patients needs a multidisciplinary team composed by gastroenterologists, surgeons, interventional radiologists, and specialists in critical care medicine, infectious disease, and nutrition. Intervention is generally required for infected pancreatic necrosis and less commonly in patients with sterile necrosis who are symptomatic (gastric or duodenal outlet or biliary obstruction) [2]. The surgical odyssey in managing necrotizing pancreatitis is a notable example of how evidence-based knowledge leads to improvement in patient care. Open surgical necrosectomy has been the traditional surgical treatment for years. However, although it provides a wide access but it is associated with high morbidity (34%-95%) and mortality (11-39%). In the last decades treatment has moved towards minimally invasive techniques: laparoscopy, retroperitoneal and endoscopic or percutaneous approaches. These can allow open surgery to be postponed in a sub-acute setting or even to avoid it [3–6].

2. Pancreatic necrosis and pseudocysts

Local complications such as pancreatic and/or peripancreatic fluid collections can occur after an episode of acute pancreatitis or after recrudescence of chronic pancreatitis or a blunt, penetrating, iatrogenic pancreatic trauma. Peripancreatic fluid collections, with or without a necrotic component, are early manifestations of the pancreatic inflammatory process. They are not delimited by a well-defined inflammatory wall and often remain asymptomatic, ending in spontaneous resolution by a gradual reduction in size. After four weeks from the clinical manifestation, persistent collections usually become wall-defined, encapsulated, with (walled-off necrosis) or without (pancreatic pseudocyst) a necrotic component and a varying degree of pancreatic parenchyma involvement [7].

Management of pseudocysts and walled-off pancreatic necrosis (WOPN) rely on patient's symptoms, location and characteristics of pancreatic and/or peripancreatic collections, local complications (such as pseudoaneurysm), expertise and availability of a multidisciplinary group [8].

In asymptomatic patients, clinical observation and periodic imaging follow up (every three-six months) represent the most successful management, due to the frequent reduction in size and spontaneous resolution of non-complicated homogeneous collections and to the morbidity associated to interventional (endoscopic or radiologic) treatment procedures. In these cases, it is possible to associate nutritional and pharmacological support (nasoenteric feeding reduces pain and improves nutritional status; proton pump inhibitors and somatostatin-analogue such as octreotide reduce pancreatic secretion).

Infection will develop in about one third of patients with pancreatic necrosis. It may arise at any time during the clinical course but peak incidence is between the 2nd and the 4th week after presentation [2]. Gram-negative bacteria are the main infectious species isolated, the most common of which are *Escherichia coli* and *Pseudomonas aeruginosa* [9]. Recently, a trend towards increasing incidence of Gram-positive and multi-resistant bacteria has been demonstrated [10, 11].

Prognosis and management are greatly affected by the recognition between sterile and infected pancreatic necrosis. Clues of suspicion should arise in case of clinical signs of systemic inflammatory response syndrome (SIRS) (new-onset

fever, tachycardia, leukocytosis) or organ failure [12]. A blood culture with positive bacterial results and gas in and around the pancreas on a CT scan may give indirect evidence of infection. Prophylactic antibiotic use in patients suffering from acute pancreatitis has not been proven to decrease infection rate and thus, according to the meta-analysis by Wittau et al. [13] it is not recommended a routine prophylaxis. The Cochrane review by Villatoro et al. [14] showed that antibiotic prophylaxis was not associated with a reduced incidence of pancreatic necrosis infection, even though it was associated with significantly decreased mortality. CT- or US-guided fine needle aspiration of pancreatic necrosis for bacteriologic analysis are an accurate, safe and reliable techniques with high accuracy (89.4%-100%) [15, 16].

In symptomatic patients, with rapidly enlarging pseudocysts or systemic manifestations of organ failure sustained by an infectious process, an interventional treatment is indicated. In this case endoscopic drainage approach is the first choice, especially when fluid collection is close to gastroduodenal lumen. A combination of techniques is possible in patients with large collections, extended in pelvis and paracolic gutters, or multiple collections [17].

2.1 Endoscopic drainage

Endoscopic drainage of a walled collection is the preferred method when the drainage criteria are met: mature collections delimited by a well-defined inflammatory capsule and with a mostly liquid content; cystic wall adherent to stomach or duodenum; and collection's size at least 6 cm in size.

This procedure has to be performed by an endoscopist with expertise and when surgical or interventional radiology staffs are available [18]. Contraindications to endoscopic drainage are: presence of pseudoaneurysm due to gastroduodenal or splenic artery erosion, with high risk of bleeding; and collections without a mature wall.

Drainage techniques consist in [19]: *transmural drainage*: creation of a passage through the stomach or duodenum wall into the cyst lumen. This permits cystic drainage after balloon dilatation and placement of one or more stents. This method is preferred to drain WOPN in order to evacuate solid debris. T*ranspapillary drainage*: placement of a ductal pancreatic stent with or without preliminary sphincterotomy to drain cysts in communication with pancreatic duct, especially when endoscopic retrograde pancreatography demonstrates ongoing ductal leak.

Transmural approach is adopted when large and symptomatic walled-off pancreatic fluid collection is close to gastroduodenal structures. Transmural puncture through gastroduodenal wall (where is endoscopically visible a bulge resulting by apposition to the cyst), is nowadays ecoendoscopically guided. This permits to accurately identify puncture site for cystenterostomy, avoiding vessels or other interposed structures and evaluating real distance to pass through [20]. Self-expanding metal stents or plastic double pig-tail stents can be both used. Lumen Apposing Metal Stent (LAMS) are associated with higher bleeding grade but allow immediate procedures such as endoscopic necrosectomy.

Drainage of turbid necrotic fluid suggests debris presence and can be managed with direct endoscopic debridement and/or with the placement of a naso-cystic catheter for post-procedural lavage. Repeated debridement or association with percutaneous drainage or percutaneous endoscopic gastrostomy can be necessary with unresolved fluid collections [21].

For patients with small pseudocysts derived from main pancreatic duct, transpapillary stent placement is indicated as first drainage approach. This provides continuous drainage of pancreatic fluid, leading to resolution of pancreatic ductal disruption that is responsible of pseudocyst. Follow up with CT or EUS is preferred

after four to six weeks if necrotic debridement was not necessary and stents are then removed the fluid cavity is collapsed. More frequent imaging is obtained in patients who underwent necrosectomy, to determine if additional debridement is necessary. When collections are completely evacuated, stents are removed. Long-term stents seem to protect against recurrence allowing ongoing drainage of pancreatic secretions, although cystenterostomy tract matures and persists after eventual stent removal [22].

2.2 Percutaneous drainage

Percutaneous drainage remains an important treatment modality for patients with symptomatic collections. It may be used both as primary therapy or as an adjunct to other techniques. According to the last International [23], American [1] and Japanese [24] guidelines, percutaneous catheter (or endoscopic transmural drainage) should be the first step in the treatment of patients with suspected or confirmed (walled-off) infected necrotizing pancreatitis. This is applied to decompress retroperitoneal fluid collections, to provide a rapid and effective means for source control in patients with infected pancreatic necrosis. It favors clinical stabilization of patients before endoscopic or surgical debridement and is the first choice when endoscopic drainage is unavailable, unsuccessful, or not technically feasible [25].

The positioning can be performed via the transperitoneal or retroperitoneal approaches. It is technically feasible in >95% of patients [26]. Retroperitoneal route is generally preferred because it avoids peritoneal contamination, enteric fistulas and facilitates a possible step-up approach (see "Surgical approach" chapter). Moreover, the catheter tract can act as an entry portal for minimally invasive debridement methods, such as video assisted retroperitoneal or endoscopic debridement [1]. Catheters range from 8 Fr to 30 Fr in diameter; they allow for bedside irrigation and clearance of necrotic material, can be manipulated and replaced according to the evolution of the collections [27].

Percutaneous drainage alone may provide definitive therapy for a subset of patients. The prospective observational multicenter study by Horvath K. et al. in 2010, found that the decrease in the size of the collection of at least 75% after the first 10-14 days predicts successful percutaneous treatment. In 2011, a large prospective multicenter study of treatment outcomes among patients with necrotizing pancreatitis demonstrated that catheter drainage was the first intervention in 63% of cases and did not require additional necrosectomy in 35% of patients [28]. Two prospective randomized trials from the Dutch Pancreatitis Study Group compared various approaches to the management of symptomatic WON. They demonstrated that percutaneous drainage alone was successful in 35%-51% of patients and that a minimally invasive step-up approach was related to a lower rate of pancreatic fistulas, length of hospital stay and death, as compared with open necrosectomy [26, 29].

The risk of pancreatocutaneous fistula formation is the major potential drawback of this technique. The multicentre randomised trial by van Brunschot S. et al. demonstrated that the rate of pancreatic fistula formation was significantly higher in the percutaneous (32%) as compared to the video-assisted retroperitoneal debridement (VARD) group (5%) [29]. The rate is as high as 45% in those with disconnected duct syndrome [30].

2.3 Surgical approach

The surgical odyssey in managing necrotizing pancreatitis is a notable example of how evidence-based knowledge leads to improvement in patient care. In the

beginning of the 20th century surgeons such as Mayo Robson, Mickulicz, and Moynihan, in the context of the progression of anesthesia, were induced to deploy laparotomy in an effort to treat complications of severe acute pancreatitis [31]. Over the next decades surgical intervention became the therapy of choice despite a mortality rate greater than 50%. Extensive pancreatic resection became the treatment of choice in the 1960s and 1970s. Innovations and increased accuracy in radiological techniques led to new approaches for management. Surgeons were divided between those who reserved the intervention for cases of infected necrosis by proposing delayed exploration, and those who proposed early debridement for all patients with necrotizing pancreatitis. Since 1990s several studies proved that nonoperative management of patients with sterile pancreatic necrosis was superior to surgical intervention, and that delayed intervention provided improved surgical mortality rates. The treatment of infected necrosis shifted to a more conservative approach also thanks to a comprehensive knowledge of the physio-pathological process of the systemic inflammatory response and the adoption of novel antibiotics in curbing systemic toxicity and protecting against organ failure. Recently, endoscopic debridement and minimally invasive techniques has been introduced [31, 32].

The last guidelines of the Working Group of the International Association of Pancreatology (IAP)/American Pancreatic Association (APA) published in 2013 [23] and of the American Gastroenterological Association (AGA) published in 2020 [1] on the management of acute pancreatitis and pancreatic necrosis list the common indications for intervention. A symptomatic sterile pancreatic necrosis is an indication for intervention (either radiological, endoscopical or surgical). Symptoms can be represented by: gastric, intestinal, or biliary obstruction due to the mass effect of walled-off necrosis, pain, persistent unwellness in patients without signs of infection [1]. In case of infected pancreatic necrosis invasive procedures (e.g. percutaneous catheter drainage, endoscopic transluminal drainage/necrosectomy, minimally invasive or open necrosectomy) should be delayed, where possible, until at least 4 weeks after initial presentation to permit the collection to become "walled-off". A randomized clinical trial [33] that compared early surgery (within 72 h) and delayed surgery (11 days after onset) demonstrated mortality rates of 56% and 27%, respectively.

Percutaneous drainage, alone or in combination with other minimally invasive approaches, can be an effective means for source control in patients with infected pancreatic necrosis. A significant number of patients (23%–47%) will resolve their necrosis with percutaneous drainage alone. In those with persistent disease, a step up to operative intervention may be undertaken. The tract of the drain is utilized to access the retroperitoneal space for an intracavitary videoscopic necrosectomy by which drains are left in the cavity for lavage and fistula control [26, 34, 35]. The PANTER Study in 2010, a prospective randomized multicenter trial, compared the step-up approach to open necrosectomy and found a higher rate of new-onset multiple-organ failure in the open necrosectomy group (40% vs. 12%) and an equivalent mortality between the groups [26]. Surgical transgastric debridement is similar to endoscopic transgastric debridement, can be done laparoscopically or open, and is performed by an anterior gastrotomy to access the posterior wall of the stomach for transmural access to the necrosis cavity. Open surgical debridement is still an important resource in the management of these patients for the debridement of necrotic tissue.

Before surgical approach, abdominal imaging is helpful to determine intra-abdominal status. Diagnosis of infected pancreatic necrosis is made by identification of air bubbles in retroperitoneal necrosis (areas with lack of contrast enhancement) on CT scan. Diagnosis can be confirmed by CT-guided fine needle aspiration of necrotic material for culture. CT is also indicated to define extent

and location of necrotic areas, for example into the mesenteric root and down the paracolic gutters; to demonstrate the presence of a disconnected pancreatic segment (a viable pancreatic portion separated by the rest of pancreas by a necrotic segment, that require external drainage to create a controlled external pancreatic fistula); and to evaluate the presence of other local complications, such as gastric outlet obstruction, splenic or portal vein thrombosis and colonic necrosis. Open debridement with external drainage still plays an important, albeit limited, role. After access to retroperitoneum, fluid is evacuated and necrotic dissection and debridement is made. In biliary pancreatitis, cholecystectomy should be practiced but it is associated with increased incidence of postoperative bile leak or biliary injury. Colon resection and colostomy have to be considered if mesocolon is involved in peripancreatic necrosis. A feeding enteral tube and at least two-four drainage tubes should be placed [36].

Video-assisted retroperitoneal debridement approach requires preoperative percutaneous retroperitoneal access. Radiological catheter insertion is a route to guide the subsequent procedure directly down into necrotic cavity and postoperative lavage. The advantage is minimizing the risk of peritoneal contamination, but the access is limited and precludes other procedures over debridement [34]. Postoperative complications are: intra-abdominal residual fluid collections, derived from pancreatic leak not well controlled by drains; bleeding, due to vascular lesion during debridement maneuvers or rupture of pseudoaneurysm, related to vascular erosion caused by mechanical drain damage or infection associated with uncontrolled pancreatic fistula; pancreatic fistulas: amylase-rich (concentration greater than three times the upper limit of normal serum amylase) fluid coming from drains; biliary injury; and pancreatic endocrine and exocrine insufficiency, that may requires supplemental insulin and oral pancreatic enzyme replacement.

Each approach has distinct peculiarities with pros and cons that must be weighted in each case planning: pattern of disease, physiology of the patient, expertise of the multidisciplinary team, and the resources of the center [1].

3. Disconnected pancreatic duct syndrome

The term disconnected pancreatic duct syndrome (DPDS) refers to a subset of patients suffering from a disruption of the main pancreatic duct leading to a normal upstream pancreatic gland having no communication with the gastrointestinal tract [1, 37]. Up to 50% of patients with pancreatic fluid collections might have an underlying disconnected duct. It is best recognized using secretin-stimulated magnetic resonance cholangiopancreatography [38]. DPDS can be the result of acute necrotizing pancreatitis, chronic pancreatitis, and pancreatic trauma. Pancreatic juice is still secreted from the disconnected gland resulting in different resolutions that are a continuum of the same pathophysiologic process: recurrent acute pancreatitis, internal persistent pancreatic fistula (most often presenting as a peripancreatic fluid collection), external fistula, pancreatic pleural effusion, pancreatic ascites, or disconnected pancreatic tail syndrome [39, 40].

Internal fistulae are the result of ductal disruptions that are not contained by the inflammatory response. Anterior ductal disruptions result in pancreatic ascites, posterior ones result in pancreatic pleural effusions. Positive testing for a collection rich in pancreatic enzyme gives the secure diagnosis. A percutaneous drainage is the initial treatment to obtain a controlled fistula that in 70-82% of cases results in a spontaneous closure.

External fistulae may develop after pseudocyst percutaneous drainage. The stricture or the obstruction of the Wirsung result in ductal hypertension thus

increasing the chance of developing this complication. Endoscopic retrograde pancreatography (ERP) with sphincterotomy or transpapillary stenting should be then performed, both in internal and in external fistulae, to reduce resistance of pancreatic juice flow to the duodenum [41].

If the disruption is in the body or the tail (disconnected pancreatic tail syndrome), open distal pancreatectomy and debridement associated with drainage are the traditional surgical procedures. These are characterized by a high periprocedural morbidity that is counterweighted by the single procedure and a concise overall course. Distal pancreatectomy can be undertaken during the first 30–60 days of illness, in the subacute setting [1].

The high morbidity and mortality associated with open surgical procedures, especially for poor surgical candidates, recommend a minimally invasive endoscopic [42]. Partial duct disruption can be treated with endoscopic transpapillary stent bridging with a fistula resolution rate of 56%, according to Varadarajulu et al. [43]. One possible endoscopic approach in case of complete duct disruption is the use of permanent indwelling transmural stents that allow the creation and maintenance of a fistulous tract into the gastrointestinal lumen [42].

Correct choice of procedure, as well as correct choice of timing of intervention, are mandatory for success.

4. Vascular complications (haemorrhage, pseudoaneurysm and thrombosis)

Haemorrhage, pseudoaneurysm and thrombosis are the main vascular complications with an incidence ranging from 1% to 23% in patients with acute pancreatitis. Arterial complications are less frequent than venous complications (1.3-10% vs. 22%) [44].

The etiopathology of bleeding in patients with severe pancreatitis can be summarized in four main causes. The first one is due to the local spreading of lipolytic and proteolytic enzymes during a severe pancreatitis or necrosis that leads to the disruption of the tissue and the release of pancreatic fluids thus resulting in the arterial wall damage [45]. The second cause is related to a iatrogenic damage: improper surgical management of acute pancreatitis with an early operation for non-infected necrosis has been reported in Literature as a possible cause of wall arterial weakening thus leading to bleeding due to the activated enzymes [46]. Another iatrogenic source of damage is associated to the radiological positioning of drains that could give a direct trauma to the vessels and a continuous local inflammation that can diminish arterial wall integrity [47]. A third pathogenic mechanism is splenic vein thrombosis due to the necrotizing process, pseudocyst and severe inflammation that could lead to portal hypertension and, as a late sequelae, to esophageal varices formation [45]. The last remarkable pathogenic mechanism is the formation of a pseudoaneurysm that derived from the rupture of a vessels into a long-standing pseudocyst [48]. Symptoms are gastrointestinal bleeding, abdominal pain and splenomegaly and they depend on the localization of pseudoaneurysm. The most common vessels are splenic (35-50%), gastroduodenal (20%), and pancreaticoduodenal (20%) artery. Other vessels involved are tributaries of the gastric, colic and hepatic bloodstream [40, 49].

Ultrasound (US) and Computed Tomography (CT) are the gold standard to diagnose a vascular complication. Specially, CT imaging showed a higher sensibility in the diagnosis of pseudo-aneurysm, and US has an important role in identifying thrombosis or in patients with iodine allergy or renal insufficiency [50]. Enhanced-contrast CT locates necrotic areas, abscess cavity, pseudocysts, and bleeding site.

Angiography is the gold standard technique for the location and the control of the bleeding [45]. Interventional radiology is the first line treatment in both elective and emergency management of vascular complications. Angiography followed by trans-arterial embolization (TAE) is the gold standard management [51]. Different techniques can be used: the one preferred is the sandwich technique with coil located proximally and distally to the pseudoaneurysm to minimize the risk of potential rebleeding [52]. Haemostasis can be implemented with glue, N-butyl cyanoacrylate (NBCA), thrombin, ethiodised oil or gelfoam. Patients with unsuccessful TAE or in which is technically impossible, an emergency haemostatic surgery should be performed. Ligation of bleeding arteries is the technique of choice although related to a high rate of rebleeding. In extreme cases, open packing or salvage emergency pancreatectomy may represent the only chances for survival [45].

Vascular complications are rare but potentially fatal with a difficult management that is why they should be treated in a tertiary centre.

5. Chylous ascites

Pancreatitis is a rare cause of chylous ascites (CA) and in Literature, only few cases about acute pancreatitis are reported since its discovered in 1984 [53, 54]. Other causes related to CA are abdominal trauma, malignancies, sarcoidosis, lymphangiomatosis, yellow nail syndrome, cirrhosis, and mycobacterial infections [55]. CA diagnosis is based on the presence of a milky triglyceride- rich fluid collection in the peritoneal cavity. Patients complain about abdominal pain, distension, weight loss, oedema, anorexia, and weakness.

Diagnosis requires peritoneal fluid sampling with documentation of a lipid rich fluid, triglyceride concentration > 1.2 mM (110 mg/dl), peritoneal-to-plasma protein concentration ratio of >0.5 and presence of microscopic fat. The minimum daily volume of CA considered significant ranges between 100 ml to 600 ml [56, 57].

The pathogenesis is not completely clarified especially when CA is due to acute pancreatitis. The main possible reason is the spreading of proteolytic and lipolytic enzymes associated to necrosis of pancreatic tissue that damage the lymphatic vessels thus provoking a lymph leakage. Other possible reasons are AP related and include: splenic vein thrombosis leading to portal vein hypertension thus causing the rupture of lymphatic vessels; and the severe inflammation that could cause lymphatic vessels obstruction and lymphatic exudation [58, 59].

CA treatment is multimodal. Conservative treatment is based on total parenteral nutrition (TPN) or medium chain triglyceride (MCT)-high protein enteral feeding with or without addition of octreotide and reaches the resolution in two to six weeks in 60-100% of cases [60, 61]. Interventional and surgical approaches should be reserved for cases in which conservative treatment has failed. A second line therapy is bipedal lymphangiography (BPLAG) with lipiodol. This technique permits to identify the normal lymphatic stream and locate the leakage site or the obstruction site. The accumulation of injected lipiodol determines an inflammatory response that acts as an embolic agent and determines leakage resolution in up to 70% of cases [62].

Van der Gaag and colleagues has considered any duration of chylous ascites, longer than 14 days despite therapy, a requirement for surgical intervention [63]. Surgical treatment may vary from a peritoneovenous shunt to open surgical ligation of the leaking lymphatics [64]. Surgical approach should be chosen only in case of persistent CA despite treatment, symptomatic patients, or impossibility to perform interventional radiology.

6. Biliary and duodenal complications

Biliary stricture (BS) and duodenal stenosis (DS) are uncommon complication of AP. Pathogenesis of these events is strictly related to the anatomical position between the pancreatic head, the common bile duct and the duodenum. BS and DS are, in most cases, early and transient conditions associated to severe inflammation [65]. The main causes for temporary BS are inflammatory oedema and pseudocyst formation and enlargement in the area proximal to the pancreatic head that create a compression of the common bile duct, thus causing jaundice, nausea, vomit, abdominal pain, pruritus, and fatigue to the patient [66].

A duodenal early complication is gastric outlet obstruction related to the abnormal peristaltic wave and following ileus caused by the severe inflammation and the possible compression of the duodenal loop by the enlarged neck of the pancreas that cause a lumen obstruction [67].

BS ad DS usually solve with a conservative treatment intended to overcome the acute inflammatory phase. Pseudocyst management is resumed in previous chapters.

In many studies, late BS is associated to pancreatic duct disruption (PDD) with pancreatic juice leakage when duct of the head/neck of pancreas is involved in pancreatic necrosis [68]. When PDD is suspected, contrast-enhanced CT should be performed to confirm it and after that an endoscopic retrograde cholangiopancreatography (ERCP) to localize the leakage and positioning a stent [69]. If this procedure failed, and a progression of the common duct stricture has developed, surgical procedure is indicated [53].

The process that leads a transient DS to an irreversible one is still unclear. Literature suggests that the underlaying cause is a possible ischemic and thrombotic event. Indeed, inflammation may induce arterial narrowing and/or thrombosis of the pancreaticoduodenal circulation producing local ischemia and resulting in chronic fibrosis [70]. Patients who present intermittent symptomatic episodes of upper gastrointestinal tract obstruction should undergo surgical bypass, chosen considering the pathophysiology (gastrojejunostomy or gastroenterostomy with vagotomy to prevent marginal ulcer) [71].

7. Conclusion

The majority of patients suffering from acute pancreatitis will have a mild, self-limited and uncomplicated course. Local and systemic complications, mild or life-threatening, such as pancreatic and/or peripancreatic fluid collections, walled-off necrosis, infected pancreatic necrosis, disconnected pancreatic duct syndrome and vascular complications can occur.

The successful management of these patients needs a multidisciplinary team composed by gastroenterologists, surgeons, interventional radiologists, and specialists in critical care medicine, infectious disease, and nutrition. However, it must be considered that the requisite technical expertise and judgment for many of these procedures is not widely available in all centres. Intervention is generally required for infected pancreatic necrosis and less commonly in patients with sterile necrosis who are symptomatic. The surgical odyssey in managing necrotizing pancreatitis has been described. Operative approaches to the treatment of acute pancreatitis complications have undergone a dramatic transformation over the past few decades. Prospective, randomized trials have further clarified the value of the latest minimally invasive approaches to the treatment of this disease. This is the notable example of how evidence-based knowledge leads to improvement in patient care.

Conflict of interest

The authors declare no conflict of interest.

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