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Review

Necrotizing pancreatitis: A review of the interventions

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ABSTRACT

Acute pancreatitis may have a wide range of severity, from a clinically self-limiting to a quickly fatal course. Necrotizing pancreatitis (NP) is the most dreadful evolution associated to a poor prognosis: mortality is approximately 15% and up to 30–39% in case of infected necrosis, which is the major cause of death.

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). Traditionally the most widely used approach to infected necrosis has been open surgical necrosectomy, but it is burdened by high morbidity (34–95%) and mortality (11–39%) rates.

In the last two decades the treatment of NP has significantly evolved from open surgery towards minimally invasive techniques (percutaneous catheter drainage, per-oral endoscopic, laparoscopy and rigid retroperitoneal videoscopy).

The objective of this review is to summarize the current state of the art of the management of NP and to clarify some aspects about its diagnosis and treatment.

1. Introduction

Acute pancreatitis (AP) has increased in incidence reaching up to 0.7 hospitalizations for 1000 inhabitants in the last decade in the US [1]. In about 80% of the patients AP is mild and self-limiting, but in up to 20% it may run a severe course with pancreatic parenchymal and/or peripancreatic tissue necrosis, responsible for substantial morbidity and mortality rate up to 27% [2]. The major cause of death is the infection of the necrotic tissue, which is associated with a poor prognosis: mortality is approximately 15% in patients with necrotizing pancreatitis (NP) and up to 30–39% in those with infected necrosis (which occurs at some point in the clinical course in about a third of patients with necrosis) [3–5].

Intervention is generally required for infected pancreatic necrosis and less commonly in patients with sterile necrosis who are symptomatic (especially in case of gastric or duodenal outlet or biliary obstruction). The traditional treatment so far has been open surgical necrosectomy: it provides a wide access to infected necrosis, but it is highly invasive and associated with reported morbidity rates of 34–95% and mortality rate of 11–39%, due to the physiologic stress of the laparotomic debridment [6–9]. During the last two decades the treatment of NP has evolved towards less invasive techniques: laparoscopy, retroperitoneal and per-oral endoscopic approach and percutaneous image-guided drainage. These minimally invasive techniques may be nowadays either an effective alternative or a complementary approach to open surgery. They may allow to postpone surgery in order to optimize the timing of necrosectomy or even to avoid it.

Abbreviations: AP, Acute Pancreatitis; NP, Necrotizing Pancreatitis; MAP, Mild Acute Pancreatitis; MSAP, Moderately Severe Acute Pancreatitis; SAP, Acute Pancreatitis; IEP, Interstitial Edematous Pancreatitis; APFC, Acute Peripancreatic Fluid Collection; PP, Pancreatic Pseudocyst; ANC, Acute Necrotic Collection; WON, Walled-Off Necrosis; CECT, Contrast-enhanced Computed Tomography; MRI, Magnetic Resonance Imaging; EUS, Endoscopic Ultrasonography; ERCP, Endoscopic Retrograde Cholangiopancreatography; FNA, Fine Needle Aspiration; PCD, Percutaneous Catheter Drainage.

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There is a huge variation in conceptual and technical approaches to interventions for NP, and the evidence of the efficacy of them risks to be unclear. This work aimed at describing all the available treatments for NP by reviewing their last reports.

2. Classification of acute pancreatitis

The Atlanta classification of AP of 1992 was revised in 2012. The AP is distinguished as follows: mild acute pancreatitis (MAP), defined as pancreatitis without organ failure (such as renal or pulmonary failure), or complications (such as necrosis or pseudocysts); moderately severe acute pancreatitis (MSAP), defined by organ failure lasting <48 h, or by local complications; severe acute pancreatitis (SAP), reserved for cases in which organ failure lasts >48 h [10].

Interstitial edematous pancreatitis (IEP) is defined by the lack of pancreatic or peripancreatic necrosis on imaging, and is distinguished from necrotizing pancreatitis (NP), which is subdivided into 3 categories: parenchymal necrosis, peripancreatic necrosis or combined necrosis: all of them may be infected or sterile.

In the setting of AP, typically IEP, a peripancreatic fluid collection occurring within the first 4 weeks is define as an acute peripancreatic fluid collection (APFC) and is characterized by the lack of both a well-defined wall and pancreatic or peripancreatic necrosis on imaging. When an APFC persists over 4 weeks, a well-defined wall will develop and the term pancreatic pseudocyst (PP) is applied.

In the setting of NP, a collection of not only fluid but also necrosis involving the pancreatic parenchyma or the peripancreatic tissues is defined as acute necrotic collection (ANC) when seen within the first 4 weeks of the disease. Like APFCs, ANCs lack a well-defined wall. When an ANC persists over 4 weeks and becomes encapsulated, it is defined as walled-off necrosis (WON). In summary, APFC contains no necrotic material, whereas ANC contains fluid and necrosis; when these two entities persist over 4 weeks, they become PP and WON, respectively [10].

3. Complications of necrotizing pancreatitis

3.1. Infection

About one third of patients with pancreatic necrosis will develop infection, which is associated with markedly increased morbidity and mortality [3]. Peak occurrence is between 2 and 4 weeks after presentation, but it may occur at any time during the clinical course of NP. Gram-negative bacteria are the usual responsible but a trend towards increasing infections with Gram-positive and multiresistant organisms has been observed [11,12]. The development of infection should be suspected in case of new-onset fever, tachycardia and increasing leukocytosis and it may lead to sepsis, systemic inflammatory response syndrome (SIRS) or organ failure (typically later in the course of disease) [3,13]. The distinction of sterile from infected necrosis is difficult but it is very important as it greatly affects the patient's prognosis and management. The presence of gas (resulting from gas-forming organisms or from a fistula to the stomach, small bowel, or colon) on imaging studies is highly suggestive of infection but it is only present in a minority of cases. However, the gas does not need to be present to state that there is an infection [14,15]. Prophylactic antibiotic use in the presence of AP has not proved to decrease the incidence of infection or mortality and it is not recommended as prophylaxis, as shown in the meta-analysis by Wittau [16]. However, the Cochrane review by Villatoro showed that antibiotic prophylaxis was associated with significantly decreased mortality but not infected pancreatic necrosis in patients with NP: beta lactams were associated with significantly decreased mortality and infected pancreatic necrosis, but quinolone plus imidazole regimens were not [17].

3.2. Bleeding

Hemorrhage can develop in patients with NP especially in the late phase; it is estimated to occur in 1%–6.2% of patients with AP [18,19]. The bleeding may occur within the gastrointestinal tract, the peritoneal cavity, fluid collections or in the pancreatic parenchyma. It usually results from enzymatic degradation of local vessels in the peripancreatic tissues and the development of pseudoaneurysm [20]. Bleeding will often manifest as sudden deterioration in hemodynamics with drop in hemoglobin, the development of a new mass or bloody output from drains placed in the pancreatic bed. Angiography with embolization should be considered as the initial line of therapy and surgery should be reserved for refractory cases [21]. Another cause of gastrointestinal bleeding in pancreatitis is variceal bleeding associated with splenic vein thrombosis, which itself results from pancreatitis and leads to left-sided portal hypertension. Bleeding occurs in 4%–12.6% of patients and splenectomy is rarely recommended [22].

3.3. Abdominal compartment syndrome

The development of abdominal compartment syndrome is associated with a mortality of 49% and a morbidity ranging from 17% to 90% [23]. Surgical decompression has been employed in the form of standard midline laparotomy, bilateral subcostal peritoneum-sparing laparotomy, and subcutaneous, skin-sparing, linea alba fasciotomy [24,25]. In a retrospective study Mentula reported that early abdominal decompression is associated with improved renal and respiratory function and reduced mortality [26]. However, the recent systematic review by van Brunschot suggested that strong data are still lacking regarding the management of abdominal compartment syndrome in the setting of AP [23].

3.4. Pancreatic duct disruption and stricture formation

The necrosis of a long, central part of the pancreas with preservation of viable tissue in the tail may be a consequence of NP. This isolated remnant is in discontinuity with the gastrointestinal tract because the pancreatic duct has been disrupted, resulting in the formation of a pancreatic or peripancreatic collection, pancreatic ascites, pancreatic effusion or pancreatic fistula [27].

Non-operative management of pancreatic duct disruption is often possible and is best performed with a multidisciplinary approach, such as what has recently been termed the SEALANTS multidisciplinary approach (Somatostatin, External drainage, ALternative nutrition, Antacids, Nil-per-os, Total parenteral nutrition, and a Stent in the pancreatic duct) [28]. Surgery with pancreatectomy or internal drainage of the cyst can be reserved for patients who fail non-operative therapy [29,30].

Pancreatic-duct strictures can develop after an episode of NP and may later result in fibrosis and scaring, which is associated with recurrent pancreatitis [31].

4. Diagnosis

4.1. Computed tomography (CT)

Contrast-enhanced Computed Tomography (CECT) is the standard imaging modality in the setting of AP. Because the revision of the Atlanta classification relies so heavily on morphologic criteria to