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Chapter

Emergency Management of Acute Pancreatitis

Rezan Karaali and Firdes Topal

Abstract

Acute pancreatitis (AP) is the sudden inflammation of the pancreas, and it may be confined to the pancreas, or more life-threatening, affecting all organs and systems. AP is a common gastrointestinal condition Worldwide and is associated with cost to the health care system. It progresses mildly in 80% of patients and resolves with treatment, but in cases of severe AP, with mortality of around 30% recorded. In this section, we will discuss the first management of the AP in the emergency department. Because this is the period when management decisions can change the course of the disease and the length of stay in the hospital. In the management AP, approaches regarding the utility and timing of antibiotics, the timing and type of nutritional support, endoscopic retrograde cholangiopancreatography (ERCP) and cholecystectomy approaches are constantly being updated. Treatment is mainly related to the severity of the disease. With early diagnosis and treatment, most of the patients can be discharged, and the development of complications and mortality can be reduced. Therefore, emergency management is important in acute pancreatitis.

Keywords: acute pancreatitis, complications, diagnosis, emergency, management

1. Introduction

1.1 Definition and incidence

Acute pancreatitis (AP) refers to the sudden inflammation of the pancreas, and it may be confined to the pancreas, or more life-threatening, affecting all organs and systems [1–5]. Recurrence is experienced in 15–30% of patients, and 5–25% can develop chronic pancreatitis. It progresses mildly in 80% of patients and resolves with treatment, but in cases of severe AP, complications such as organ failure and pancreatic necrosis may develop, with mortality of around 30% recorded in this group [2, 4, 5]. AP is an acute gastrointestinal disease that requires hospitalization, and is the most common cause of admission to the emergency room worldwide [1, 6, 7]. Hospital admissions for AP in the United States are in the region of 270,000/year, with a mortality rate of 30% in severe cases. Death is due to systemic inflammatory response syndrome (SIRS) and organ failure in the first two weeks, while death after two weeks can be attributed to sepsis and complications [3, 6, 8, 9].

1.2 Etiology

Gallstones are the most common etiology of AP, being responsible for 40–70% of AP cases [10–12]. The ease at which small gallstones can pass into the bile duct make

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AP more common in this patient group [13]. Although alcohol is commonly blamed as the second most common cause, the link between alcohol and AP is unclear, as AP is seen in only a small number of alcoholics [2, 14, 15]. Recent studies have suggested that alcohol increases the oxidative metabolism in the acinar cells of the pancreas, thereby causing mitochondrial dysfunction and cell death. This increases also the production of acetaldehyde in the pancreatic stellate cells, and increases circulating lipopolysaccharide and tumor necrosis factor alpha (TNFα), leading to fibrosis in the pancreas [16, 17]. Alcohol has also been reported to increase the viscosity of pancreatic juice and to cause ductal obstructions. That said, it has also been suggested that genetic factors play a role in the development of AP,based on the low incidence of AP in people with chronic alcohol consumption [2, 15, 18]. Other causes have been identified as Hypertriglyceridemia (HTR), and diabetes, hypothyroidism, pregnancy and obesity that cause HTR [1]. Patients with a body mass index (BMI) >35 are at risk of both HTR and AP, while those with serum triglyceride levels >1000 mg/dl are at greater risk [19–21]. Following endoscopic retrograde cholangiopancreatography (ERCP) performed by inexperienced practitioners, patients with Sphincter of Oddi dysfunctions may develop AP following ERCP due to difficult cannulation [22].

AP can also occur due to drugs at a rate of 0.1–0.5% [2, 23–25]. Many drugs have been identified that cause acute pancreatitis. Drugs cause AP by different mechanisms. While some drugs cause direct toxicity to the pancreas (eg, diuretics, sulfonamides), some drugs cause acute pancreatitis by causing an immunological reaction (eg, 6-mercaptopurine, amino salicylates, sulfonamides). Diuretics and azothiopurine cause direct ischemia, while hormones such as steroids and estrogen cause vascular thrombosis or ischemic pancreatitis by decreasing the viskosity of the pancreatic juice. Toxic metabolites of drugs such as valproic acid and tetracycline may accumulate in the pancreas and cause pancreatitis [2, 26, 27].

AP cases have been reported associated with such infectious diseases as Mumps, Coxsackievirus, Hepatitis B, Cytomegalovirus, Varicella-Zoster, herpes simplex and human immunodeficiency virus (HIV) among the viruses; with Mycoplasma, Legionella, Leptospira and Salmonella among the bacteria; with Aspergillus among the fungi; and with Toxoplasma and Cryptosporidium among the parasites [2, 27, 28]. There have been reports of cases of AP with the recent SARS-CoV-2 infection at the heart of the current global pandemic [29, 30]. In a review of current literature, AP was found to be detected in 17% of patients hospitalized due to Covid-19 [29]. Although tests for specific infectious agents are not generally recommended in AP patients, Covid-19 infection should also be kept in mind in AP cases during the pandemic [30].

Concerning other rare causes, pancreatic injury following trauma is an extremely rare condition due to its retroperitoneal nature. Pancreatic duct injuries may occur due to blunt or penetrating traumas [31], while AP may occur due to gallbladder sludge, tumors, autoimmune pancreatitis, hypercalcemia, anatomical and physiological anomalies (pancreatic divisum, biliary cysts, pancreaticobiliary malunion, large juxta-ampullary diverticula, annular pancreas and Sphincter of Oddi dysfunction), and vasculitis [27, 32–36]. Ischemic AP can also be seen after major cardiovascular operations [27, 37, 38]. Patients with an unknown etiology after history-taking, physical examination, laboratory tests, imaging methods and advanced tests are classified as idiopathic. In the event of recurrent AP attacks in this patient group and AP at a young age, genetic factors should be investigated [27, 39].

1.3 Pathogenesis

As its main mechanism, AP blockades the secretion of enzymes while the synthesis of enzymes continues [2, 40]. Under normal conditions, trypsinogen is

produced in the pancreas and secreted into the duodenum where it is converted into protease trypsin, but in cases where secretion is blocked, trypsin continues to be produced in pancreatic acinar cells. While activation continues, elimination is inhibited, and the active trypsin damages the vascular endothelium, interstitium and acinar cells [2, 40, 41]. As a result, autodigestion begins in the pancreas,and ischemia occurs at a tissue level in the pancreas due to the vasoconstriction and stasis of the capillary vessels. The activation of granulocytes and macrophages in response to these events causes a release of proinflammatory cytokines (tumor necrosis factor, interleukins 1, 6 and 8), arachidonic acid metabolites (prostaglandins, platelet activating factor and leukotrienes), proteolytic and lipolytic enzymes, and reactive oxygen metabolites [2, 27, 42, 43]. All of these factors together cause damage to the pancreatic tissue. In general, the inflammation is locally self-limiting, buton occasions, inflammatory agents may cause a systemic response, leading to the damage and failure of distant organs. This, in turn, may result in Acute Respiratory Distress Syndrome (ARDS), pleural effusion, acute renal failure, shock, and even death [2, 27, 44, 45].

2. Clinical features

Patients withAP present to the emergency room with sudden and severe abdominal pain that usually starts in the epigastric region. In patients with gallstones, the pain spreads to the right upper quadrant and is more sharply limited. In 50% of patients, the pain spreads to the back, and is felt around the entire abdomen, like a belt. Nausea and vomiting may accompany,and in rare cases there may be pain on the left side of the abdomen [2, 46–49].

Physical examination findings can vary, depending on the severity of AP and any accompanying diseases. Initial findings typically include mild or generalized tenderness upon abdominal palpation, distension and diminished bowel sounds. In cases of obstruction due to gallstones, jaundice may be observed, while in severe AP, fever, hypotension, tachycardia, tachypnea and hypoxemia may be observed. In cases of pancreatic necrosis, ecchymotic lesions can be seen in the periumbilical region (Cullen's sign) or on the flanks (Gray Turner's sign) [2, 27, 50, 51].

3. Diagnosis

Diagnosis is established based on the presence of two of three criteria: 1) Presence of clinical findings consistent with AP, 2) serum lipase or amylase levels three times greater than normal, and 3) characteristic findings of AP on imaging [2, 27, 47, 48, 52].

3.1 Laboratory

In AP, enzymes pass from the basolateral membrane to the interstitial area, and then on to the systemic circulation due to the blockade of the secretion ofpancreatic enzymes, while the synthesis of enzymes continues, resulting in increased levels of pancreatic enzymes in the blood.

At the onset of AP, serum amylase starts to increase within 6–12 hours, peaks at 48 hours, and returns to normal within 3–5 days, although no increase in amylase levels will be observed in alcohol-induced pancreatitis and AP due to hypertriglyceridemia. Sensitivity and specificity in diagnosis are 67–83% and 85–98%,

respectively [2, 27, 48, 53, 54]. Elevated amylase levels may also be seen in non-pancreatic diseases, such as renal failure, salivary gland diseases, acute appendicitis, cholecystitis, perforations, intestinal obstructions or intestinal ischemia, and gynecological diseases. For these reasons, amylase alone is not sufficient for a diagnosis of AP [2, 48, 49]. The increase in serum lipase levels in AP is more specific. Following the onset of symptoms, the levels begin to increase within 8–10 hours, peak at 24 hours, return to normal within 8–14 days, with a sensitivity of 82–100% [2, 48, 53, 55], and may increase in alcohol-induced AP and AP due to hypertriglyceridemia. It is useful in delayed patients who present 24 hours after the onset of pain [48, 55, 56]. Aside from amylase-lipase, liver and kidney tests,a complete blood count should also be made in AP, as this will allow the assessment of the patient's clinical condition, the early identification of complications and the detection of organ failure, and will aid in a therapeutic evaluation. An alanine aminotransferase (ALT) liver function test value in excess of 150 U/L indicates gallstones [2, 47, 52]. There are also specific tests for AP that are not routinely used. Among the enzymes with early elevation are trypsinogen-activating peptide, urinary and serum trypsinogen and trypsin, phospholipase, carboxypeptidase, carboxyl ester lipase, colipase and pancreatic isoamylase [57–59], and an increase is also observed in inflammatory mediators such as C-reactive protein (CRP), interleukin IL-6, IL-8, IL-10, tumor necrosis factor (TNF) and PMN elastase. The elevation of inflammatory mediators is usually proportional to the severity of AP. A CRP level above 150 mg/dl within the first 48 hours has been associated with severe AP [60, 61].

3.2 Imaging

Imaging can aid in determining the etiology of AP, or complications due to AP. Abdominal and chest radiographs may reveal appearances of pleural effusion, atelectasis and ileus accompanying AP. Radiographs should be evaluated to rule out other causes of abdominal pain. Abdominal ultrasound should be performed on every patient with suspected AP, and USG can detect findings that support AP, if present, such as gallstones, obstructions in the common bile duct, intraabdominal free fluid and diffuse enlarged and hypoechoic appearance in the pancreas, as well as peripancreatic fluid, necrosis and abscesses. A normal USG cannot exclude AP [2, 27, 47, 48, 52, 62], while Contrast-Enhanced Computed Tomography (CECT) has a sensitivity of 90% in the diagnosis of AP. However, AP is not routinely recommended for diagnosis, since it is mild and uncomplicated in most patients [2, 47, 48, 52], but may be recommended in cases where other causes of acute abdomen cannot be excluded, or for patients who show no improvement within 48–72 hours [48, 63, 64].

Among the patients considered for CECT, MRI is recommended rather that CECT for those with renal failure, pregnant patients and those with allergies to IV contrast agents [48, 63].

Serum triglyceride levels must be examined in patients with normal test results, but with a strong suspicion of AP, in those with pancreatic tumors aged over 40 years, in the presence of genetic factors in patients under the age of 30 and in recurrent AP cases [39, 48].

3.3 Differential diagnosis

Other diseases that may cause abdominal pain should be excluded in a differential diagnosis. In particular, peptic ulcer disease, choledocholithiasis, cholangitis,

biliary obstruction, cholecystitis, perforated viscus, intestinal obstruction, mesenteric ischemia and hepatitis should be considered in differential diagnosis due to their clinical similarities to AP [2, 27].

4. Initial management

AP can be classified into two groups as mild AP, in which patients have no accompanying organ failure, and recover and can be orally fed within 48 hours; and severe AP, which is accompanied by organ failure and a lack of response to treatment. Most patients with severe AP have not suffered organ failure at the time of admission to emergency room, and so may be evaluated as mild AP,but deteriorate rapidly due to inadequate hydration and inadequate treatment. As such, the severity of the disease should be determined along at the time of diagnosis in the emergency room, and treatment should be planned accordingly [47, 48, 52, 65].

According to the Atlanta classification, severe AP is characterized by resistant/persistent organ failure with no improvement within 48 hours, although in the absence of organ failure, the presence of local complications alone is an indicator of severe AP [66]. Patients who develop transient organ failure alongside local complications are classified as moderately severe AP (**Table 1**). The Atlanta classification evaluates the presence of organ failure based on Marshall's organ failure criteria. Accordingly, the presence of shock (systolic BP <90 mmHg), pulmonary failure (PaO2 < 60 mmHg), renal failure (creatinine >2 despite adequate hydration), and/or the presence of gastrointestinal bleeding (>500 ml blood loss within 24 hours) should be evaluated as organ failure [48, 52, 67].

Besides the Atlanta classification, several scoring systems have been proposed for the determination of the severity in AP. These include Ranson's criteria,Acute Physiology and Chronic Health Examination-II, modified Glasgow score, Bedside Index for Severity in Acute Pancreatitis and the Balthazar CT Severity Index,none of which has been shown to be superior to any other, and they have only limited use in the emergency room, as they rely on too many parameters, and some give results only after 48 hours [68, 69]. The assessment of the patient in the emergency department is of utmost importance, with patient-related risk factors such as age, weight, comorbidities and vital signs as well as laboratory findings all being evaluated together (**Table 2**) [47, 52, 56, 65].

Mild AP	**Moderately AP**	**Severe AP**
Absence of local complications	**Local complications** Peripancreatic fluid collection Pancreatic or peripancreatic necrosis(sterile or infected) Gastric outlet disfunction Splenic or portal vein thrombosis Colonic necrosis AND/OR	**Persistent organ failure > 48 h** GI bleeding (>500 cc/24 hr) Shock – SBP < 90 mmHg PaO 2 < 60% Creatinine >2 mg/d
Absence of organ failure	**Transient organ failure < 48 h**	
	GI bleeding (>500 cc/24 hr) Shock – SBP < 90 mmHg PaO 2 < 60% Creatinine >2 mg/d	

Table 1.
Atlanta classification 2015.

Patient characteristics	The systemic inflammatory response syndrome (SIRS)	Laboratory findings	Radiology findings
Age > 55 years Obesity (BMI >30 kg/m2) Altered mental status Comorbid disease	• pulse >90 beats/min • respirations >20/min or PaCO2 > 32 mmHg • temperature > 38°C or < 36°C • WBC count >12,000 or < 4,000 cells/mm3 or > 10% immature neutrophils (bands)	BUN >20 mg/dl Rising BUN HCT >44% Rising HCT Elevated creatinine	Pleural effusions Pulmonary infiltrates Multiple or extensive extrapancreatic collections

Table 2.
Initial assessment for risk of severe AP.

5. Treatment

5.1 Fluid replacement

The initial approach to AP involves aggressive fluid therapy, pain management and nutritional support. In AP, there is a large amount of fluid deficit due to losses from vomiting, reduced oral intake, passage of fluid into the third space, respiration and sweating. If the patient has no additional cardiovascular or renal disease, fluid replacement should be initiated at 5–10 ml/kg/hour. For patients presenting with evidence of hypovolemia and shock, 3 ml/kg of fluid should be given for 8–12 hours following a fluid bolus of 20 ml/kg in 30 minutes, with isotonic normal saline preferred as the fluid [47, 48, 52, 70–72]. A prospective study found hydration with Ringer's lactate solution to be more beneficial, although Ringer's lactate solution has been shown to activate trypsin in acinar cells, thereby making the patient more susceptible to injury due to its low pH. With normal saline, there is a risk of developing non-anion gap metabolic acidosis, and patients should be monitored accordingly during fluid replacement [2, 72]. An assessment should be made after 6, 24 and 48 hours to as certain whether the fluid administered is sufficient. With adequate hydration, the heart rate should drop below 120/min, mean arterial pressure (MAP) should be maintained between 65 and 85, and hematocrit (HCT) should be 35–44%. If the BUN value is initially high, a decrease upon hydration is an indicator of adequate hydration. Changes in blood urea nitrogen (BUN) values within the first 24 hours are particularly important [27, 47, 48, 73, 74]. If the BUN values continue to be high, or increase even further, acute tubular necrosis or resistant volume deficit should be suspected [27, 47, 52, 65, 75]. Another parameter that should be monitored during hydration is hematocrit. Continued hemoconcentration for more than 24 hours suggests the development of necrotizing pancreatitis, and so the patient's urine output, BUN and HCT values should be closely monitored. The development of severe pancreatitis should be considered in patients who do not respond to aggressive hydration for 6–12 hours [47, 48, 52].

5.2 Pain management

Adequate hydration and the resolution of hypovolemia relieve ischemic pain secondary to hemoconcentration. Nevertheless, opioid analgesics are recommended for rapid pain management. Fentanyl can be used safely, especially in patients with kidney failure, in which intravenous (IV) fentanyl of 20–50 microgram is administered slowly over 10 minutes. Meperidine can be used as an alternative to morphine due to the spasm effect of morphine on the Sphincter of Oddi [2, 27, 76, 77].

Vital signs	Laboratuary findings	Patient condition
pulse <40 or > 150 beats/min; systolic arterial pressure < 80 mmHg (<10.7 kPa) or mean arterial pressure < 60 or diastolic arterial pressure > 120 mmHg respiratory rate > 35 breaths/ min;	serum sodium <110 mmol/l or > 170 mmol/l; serum potassium<2.0 mmol/l or > 7.0 mmol/l; paO2 < 50 mmHg pH < 7.1 or > 7.7; serum glucose >800 mg/dl (>44.4 mmol/L); mmol/L); serum calcium >15 mg/dl (>3.75	coma. Furthermore, a patient with severe acute pancreatitis as defined by the revised Atlanta Classification (i.e. persistent organ failure)

Table 3.
Assessment for intensive care.

5.3 Monitoring

AP patients should be followed closely for 24 hours, with continued monitoring of blood pressure, temperature, pulse, oxygen saturation and urine output. Blood tests should be monitored for hematocrit, BUN and electrolytes (calcium, magnesium), and blood glucose should be maintained between 180 and 200 mg/dl [2, 27, 52]. Intensive care follow-up is required for patients whose vital signs and laboratory values are unstable and / or continue (**Table 3**) [52].

5.4 Nutrition

It is no longer recommended to stop oral intake until the AP has fully resolved and the enzymes have returned to normal limits in order to put the pancreas at rest. Patients ceasing oral intake may develop atrophy in the mucosa of gastrointestinal tract [27, 47, 48, 52, 78, 79], and so oral feeding should be initiated in patients without nausea, vomiting or ileus and with relieved pain, as soon as they can tolerate [47, 48, 52, 79–81]. Liquid, light and low-fat foods should be given at first [82]. In cases of severe AP, enteral feeding may be initiated in patients who are still unable to tolerate oral feeding after 5 days, and in those with complications. For enteral nutrition, a nasojejunal or nasogastric tube should be used for feeding. A nasogastric tube insertion may be easy, but there is a risk of aspiration, while a nasojejunal tube requires an operation. Depending on the conditions, both methods can help provide effective nutrition [47, 48, 82]. If the goal of enteral nutrition is not achieved within 48–72 hours, or if the patient cannot tolerate, parenteral nutrition should be initiated [80, 81, 83].

5.5 Antibiotics

20% of patients develop extrapancreatic infections that may be cholangitis, catheter infection, urinary tract infection or pneumonia. Prophylactic ABs, even if severe, are not routinely recommended in AP without an unidentified focus of infection or presence of infection. ABs for infective necrosis prophylaxis are not recommended, even for patients with sterile necrosis [2, 27, 47, 48, 52, 65, 84, 85].

6. Management of complicatıons

If, during the follow-up of moderately severe or severe AP patients, signs of sepsis appear, no improvement occurs within 72 hours or the condition deteriorates gradually, then complications should be suspected and a CECT should be performed.

6.1 Local complications

6.1.1 Acute peripancreatic fluid collection

Acute peripancreatic fluid collection occurs early, and has no specific wall. It resorbs spontaneously [27, 48].

6.1.2 Necrotizing pancreatitis

Necrotizing pancreatitis can involve both the pancreas and peripancreatic tissues. A variable amount of fluid and necrotic tissue may develop within the necrosis,and is known as Acute Necrotic Collection (ANC) when a clear wall cannot be defined, and as Wall-off Necrosis (WON) when there is a mature, encapsulated and well-defined wall. WON is a pancreatic pseudocyst that occurs around 4 weeks after an AP attack, and that has a noticeable wall, for which drainage may be required. In either case, the necrotic area may be sterile or infected, and the type of treatment is determined based on the presence or absence of infection [84, 86–88].

6.1.2.1 Infected necrosis

Infection should be suspected in patients with pancreatic or extrapancreatic necrosis upon clinical deterioration or a lack of improvement within 7–10 days of hospitalization. Infectious agents are usually of intestinal origin (such as Escherichia coli, Pseudomonas, Klebsiella and Enterococcus), and may be suspected with the emergence of clinical signs of infection in patients and the presence of gas around the pancreas on imaging [89, 90]. Empirical AB may be initiated in these patients, with ABs that can penetrate the pancreas well (carbapenem alone; or quinolone, ceftazidime, or cefepime combined with an anaerobic agent such as metronidazole)being recommended [27, 47, 48]. Fine needle aspiration (FNA) or sampling is not recommended in such patients. Necrosectomy may be scheduled for patients who show no improvement, but should be delayed as much as possible, since many patients respond well to AB therapy [48, 90–92]. Antibiotic therapy should have been completed 4 weeks prior to a decision of necrosectomy. For the necrestomy, endoscopic or invasive percutaneous procedures should be tried first, and if these fail, surgery should be scheduled [47, 48, 52, 91–93].

6.1.2.2 Sterile necrosis

In patients with necrotizing pancreatitis, sterile necrotizing pancreatitis should be suspected when there is no improvement despite treatment, and no clear clinical or imaging findings of infection. In such cases, FNA sampling is indicated, and if the collected material is sterile, there is no need to continue the ABs. Even ABs cannot prevent sterile necrosis from turning into infected necrosis [47, 52, 94]. In sterile necrosis in the absence of any sign of infection, interventions will be required in the following cases:

- Continued obstruction of the gastric outlet, intestine or bile ducts, caused by mass effects after 4–8 weeks following the onset of acute pancreatitis.

- Persistant symptoms (e.g. abdominal pain, nausea, vomiting, anorexia or weight loss) identified more than eight weeks following the onset of acute pancreatitis.

- Disconnected duct syndrome (full transection of the pancreatic duct) with persistent symptomatic collections with necrosis (e.g., pain, obstruction) more than 8 weeks following the onset of acute pancreatitis.

Aside from these, CT and FNA should be repeated 5–7 days later in patients with sterile necrosis detected by CECT and FNA, but with signs of systemic toxicity [48, 52].

The much rarer complications include peripancreatic vascular complications, splanchnic vein thrombosis, abdominal compartment syndrome and pseudoaneurysm. Furthermore, patients may risk developing diabetes in the following periods [27, 52, 95].

6.2 Systemic complications

Respiratuar insufficiency includes pneumonia, atelectasis, and ARDS. Renal complications are prerenal azotemia, hypotansion and acute tubuler necrosis. Shock is caused by third space losses, vomiting and interstitial edema. Hypo-hyperglicemia, coagulation disorders, fat necrosis and pancreatic encphalophaty are other rare systemic complications of AP [27].

7. Management of predisposing underlying conditions

7.1 Nonsurgical management

The detection and treatment of the underlying diseases that cause AP are as important as AP itself. Most gallstones that pass into the common bile duct advance to the intestines, and are excreted with feces. However, stones that cause obstructions to the pancreatic duct and/or biliary ducts may result in severe AP and/or cholangitis. ERCP is recommended within the first 24 hours for AP patients with stones detected as causing an obstruction. The removal of stones by via a sphincterotomy with ERCP prevents both severe AP and the cholangitis and future development of biliary AP. ERCP should be performed within the first 24 hours in AP patients due to gallstones accompanied by acute cholangitis. A papillotomy, or the surgical removal of stones, with ERCP reduces the severity of AP [48, 52, 96–98]. It has been reported that mortality decreases with early ERCP in patients with no cholangitis, with biliary duct obstructions, and with elevated liver function test scores. That said, it is unnecessary to perform ERCP within the first 24 hours on patients with no increase in liver function tests, with therapeutic ERCP recommended for such patients before or during the cholecystectomy. It is recommended that EUS and MRCP be performed prior to ERCP in patients without cholangitis or jaundice, but with suspected choledocholithiasis, pregnant women and patients on whom ERCP cannot be performed anatomically [47, 48, 52, 65, 99].

7.2 Surgical managment

The removal of stones through the use of ERCP in patients without cholangitis can prevent the development of AP in the future, but it cannot prevent the development of biliary colic or cholecystitis. Accordingly, cholecystectomy is recommended prior to discharge in patients with mild AP and with gallstones [47, 48, 52, 65, 100–103]. Preoperative MRCP or EUS, or intraoperative cholangiography may be carried out for the selection of patients with common bile duct stones who need to be treated

through an operative bile duct exploration or endoscopic sphincterotomy during a cholecystectomy [48, 52, 99]. A cholecystectomy may be avoided in ineligible elderly patients (>80 years of age), particularly if a sphincterotomy has already been performed [48, 52, 96, 97]. A cholecystectomy should be performed in patients with gallbladder sludge and AP. In patients with necrotizing biliary AP, cholecystectomies should be delayed until the active inflammation subsides and fluid collections have resolved or stabilized. If collection takes longer than 6 weeks to resolve, the cholecystectomy should be delayed until it can be performed safely [47, 48, 52, 65]. Asymptomatic pseudocysts and pancreatic and/or extrapancreatic necrosis require no surgical intervention, regardless of the size, location and/or extension. In asymptomatic patients with infected necrosis, surgical, radiological and/or endoscopic drainage should be delayed for more than 4 weeks to allow for the liquefaction of the content and the development of a fibrous wall around the necrosis (WON). Minimally invasive necrosectomy methods are preferred in symptomatic patients with infected necrosis [47, 48, 52, 84, 87]. Percutaneous drainage and/or endoscopic drainage/debridement are minimally invasive alternatives to open surgery [104].

Percutaneous CT-guided catheter drainage: The procedure is performed under local anesthesia. Depending on the size and location of the necrosis, the catheter is placed under CT guidance. Irrigation with saline every several days after insertion [105, 106]. Although percutaneous catheter drainage was used for patients who are too unstable to undergo surgical debridement, approximately one third to one half of patients can be managed with this method alone [106, 107]. The only disadvantage of this method is the risk of persistent pancreatico-cutaneous fistula [108].

Endoscopic debridement: It is performed via transgastric or transduedonal [104, 105, 109]. Cystenterostomy is created using wire-guided balloon dilators. Mechanical debridement is performed using snares, baskets, and stone retrieval balloons. Following this, a stent is placed in the cavity. The flow of necrotic contents into the stomach or duodenum is provided [109]. Minimally invasive operative approaches are preferred to open surgical necrosectomy and given lower morbidity [110].

8. Conclusion

Although new guidelines have been published, there are several knowledge gaps identified in the initial management of the AP. Risk stratification of patients with AP is important to ensure the appropriate level of care. Therefore, there is a need to develop fast, easy and practical systems that can be used in the emergency room. There is also a need to define targeted therapies in AP. Future research will enable prevention of relapse, chronicity, and cancer development, improvement of quality of life and reduction of mortality.

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Conflict of interest

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