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Management of Acute Pancreatitis in the First 72 hours

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Abstract

Purpose of Review—Acute pancreatitis (AP) is a common condition that affects patients with varying degrees of severity and may lead to significant morbidity and mortality. This article will review the current paradigm in AP management within the first 72 hours of diagnosis.

Recent Findings—Patients presenting with AP should be evaluated clinically for signs and symptoms of organ failure in order appropriately triage. Initial management should focus on fluid resuscitation, with some data to support Ringer's lactate over normal saline. Routine use of prophylactic antibiotics in AP is not recommended, nor is urgent endoscopic retrograde cholangiopancreatography (ERCP) in the absence of concomitant acute cholangitis. Early oral feeding should be encouraged, not avoided, and use of parenteral nutrition is discouraged. Cholecystectomy during the same admission of biliary pancreatitis should be performed in order to prevent future AP episodes. Patients with AP secondary to alcohol should receive alcohol counseling. Finally, there is ongoing interest in the development of prognostic laboratory tests in AP and pharmacological therapies to reduce the inflammation that occurs in AP.

Summary—AP is a common and heterogeneous condition with the potential for significant morbidity. Best practices in AP management focus on triage, hydration and enteral feeding.

Keywords

Acute pancreatitis; ERCP; gallstones; cholecystectomy; resuscitation; SIRS

1.0 Introduction

Acute pancreatitis (AP) is a common condition with an annual cost of 2.6 billion dollars.[1,2] While the overall population mortality rate for AP has remained unchanged, the incidence appears to be increasing.[3,4] No pharmacological therapy currently exists to treat AP, however data from both observational studies and randomized controlled trials have established best practices that lead to reduced morbidity and mortality in AP. This article

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will briefly describe the diagnosis, etiology and prognosis of AP followed by the current paradigms in AP management within the first 72 hours of diagnosis - a period during which appropriate risk stratification and management is critical to optimize patient outcomes for this disease. A number of recent guidelines have addressed this topic, including from the American College of Gastroenterology (ACG),[5] the International Association of Pancreatology (IAP) and the American Pancreatic Association (APA),[6] and, most recently, the American Gastroenterological Association (AGA).[7] Relevant recommendations from these expert groups will be summarized below.

2.0 Main Text

Diagnosis

The diagnosis of AP is established by the presence of at least 2 of the following: stereotypical abdominal pain, serum amylase and/or lipase greater than three times the upper limit of normal, and/or characteristic findings on abdominal imaging[8,9]Patients with AP typically present with mid-epigastric and/or right upper quadrant pain that is constant, stabbing in character with radiation to the back or flank. Contrast-enhanced computed tomography (CT) and/or magnetic resonance imaging (MRI) of the pancreas should be reserved for patients in whom the diagnosis is uncertain from clinical and laboratory evaluation alone or patients who fail to improve clinically within the first 72 hours of hospitalization.[10,11] Figure 1 demonstrates CT imaging of a patient presenting with acute biliary pancreatitis.

Etiology

Gallstones and alcohol are by far the most common causes of AP and thus transabdominal ultrasound and alcohol use history should be obtained in all patients presenting with the condition.[12,13] Alcohol-induced AP is generally considered to require > 50 g of alcohol per day, though less than 5% of chronic alcoholics develop acute pancreatitis for reasons that are uncertain.[14,15,16] In the absence of gallstones or a significant history of alcohol use, a serum triglyceride level should be obtained and considered the cause if > 1,000 mg/dl.[17,18] In medication induced pancreatitis, the most well-established agents are 6-mercaptopurine or azathioprine, isoniazid, loop diuretics and didanosine[19] Though rare, a pancreatic tumor or cystic neoplasm should be considered as a cause of AP in patients older than age 40 without another obvious etiology.[20,21] Post-ERCP pancreatitis is the most common serious adverse event attributed to the procedure, though there is debate over how to define this entity.[22,23] Most studies use the consensus definition which requires the presence of new or worsened abdominal pain, amylase at least three times normal at more than 24 hours after the procedure, and requiring admission or prolongation of planned admission to 2–3 days.[24,25] Idiopathic AP is defined as pancreatitis with no etiology established after initial laboratory tests, transabdominal ultrasound and CT and should be referred to a center of expertise for further evaluation.[5,26]

Prognosis

Initial evaluation of a patient with AP should include assessment of systemic inflammatory response criteria and resuscitative measures should be initiated if needed. Numerous AP

severity scoring systems exist to assist in predicting a patient's clinical course, however many are cumbersome to calculate and often take >72 hours to become positive, at which point the patient's clinical course is likely obvious. These scores, including APACHE II, Ranson and Glasgow demonstrate relatively poor positive predictive value.[27] Even new scoring systems, including the Bedside Index of Severity in Acute Pancreatitis (BISAP), have not been shown to be more accurate than the previous scoring systems.[28,29]

In general, the current practice in determining AP severity is based upon the revised Atlanta criteria (Table 1).[9] Mild AP is defined by absence of organ failure or local complications; moderately severe AP is defined by transient organ failure (less than 48 hours) and/or local complications; severe AP is defined by persistent organ failure (greater than 48 hours). Organ failure is typically defined using the Modified Marshall scoring system for organ dysfunction (Table 2).[30] Patients with severe AP should be admitted to an intensive care unit or intermediary care setting whenever possible.[5] Factors associated with worse outcomes in AP include advanced age, multiple comorbid health problems, elevated body mass index, presence of systemic inflammatory response syndrome, elevated BUN and /or hematocrit, pleural effusions and/or infiltrates and altered mental status.[31,32,33,34,]

Management - Fluid resuscitation

Fluid therapy to prevent hypovolemia and organ hypoperfusion is a well-established cornerstone of sepsis care, which has numerous physiological similarities to AP.[35] Typical goal-directed therapy focuses on heart rate, mean arterial pressure, central venous pressure, urine output, blood urea nitrogen concentration, and hematocrit. These goals are outlined in Table 3. Recent AGA, ACG, and IAP/APA guidelines recommend goal directed therapy for fluid resuscitation of AP patients utilizing various parameters.[5,6,7] However, it should be acknowledged that the evidence supporting these recommendations is relatively weak, and compared to non-targeted therapy, there is not clear evidence that goal-directed therapy results in significant improvement in important outcomes such as mortality, prevention of pancreatic necrosis, or decrease in the rate of persistent multiple organ failure.[7] Nevertheless, using physiologic metrics to guide initial fluid resuscitation is important both to ensure adequate organ perfusion and to avoid overly aggressive fluid therapy, which can be associated with harms in AP, including respiratory complications and abdominal compartment syndrome.[36,37] It should be noted that aggressive fluid resuscitation is most useful in the first 12–24 hours of admission, and should generally be curtailed after this point to ovoid fluid overload.[5,38,39,]

Two randomized controlled trials evaluating Ringer's lactate vs normal saline as the optimal fluid solution for resuscitation demonstrated a reduction in systemic inflammation.[40,41] These studies may have limited application, however, as they used surrogate markers of severity and did not focus on important clinical outcomes, such as organ failure, necrosis, or mortality. Despite these limitations, Ringer's lactate is recommended by ACG and IAP/APA guidelines when possible because of the theoretical benefits over normal saline.[5,6] The recommended infusion rate is 250–500 ml per hour, unless there are cardiovascular, renal, or other related comorbid conditions present.[5]

Hydroxyethyl starch (HES) has also been used as a resuscitative fluid in AP[42]. However, the studies to date failed to demonstrate improved mortality compared to fluid resuscitation without HES and in one trial multiple organ failure was significantly increased in patients receiving HES fluids.[43, 44] For these reasons, HES-containing fluid is not recommended as a resuscitative agent in AP.

Management – Prophylactic Antibiotics

Mortality doubles when pancreatic or peripancreatic necrosis becomes infected in patients with organ failure.[45] Previous rationale for administering prophylactic antibiotics was in hopes of reducing the risk for infected necrosis and thereby improving morbidity and mortality. In patients with predicted severe AP and necrotizing pancreatitis older trial data suggest that prophylactic antibiotics were associated with a reduction in the risk of infected pancreatic and peripancreatic necrosis. Recent trial data (since 2002), however, demonstrate no differences in risks of infected pancreatic and peripancreatic necrosis or mortality.[46] In patients with mild AP there appears to be no role for prophylactic antibiotics in the absence of cholangitis or other extrapancreatic infections. Further, prophylactic antibiotics have not shown an impact on the rates of persistent single organ failure, multiple organ failure, and hospital length of stay.[47] Accordingly, recent ACG and AGA guidelines have recommended against the routine use of prophylactic antibiotics in AP patients.[5,7]

Management – Enteral Feeding

Historical management of AP allowed the patient to take little by mouth in order to avoid a theoretical risk of further stimulating an inflamed pancreas. This paradigm has shifted dramatically over the last decade as further evidence has accrued to suggest early feeding does not exacerbate pancreatic parenchymal inflammation, and is actually beneficial in AP. This rationale stems from the understanding that enteral nutrition likely serves to protect the mucosal barrier of the gut and reduce bacterial translocation. This in turn may reduce the risk of developing infected pancreatic and peripancreatic necrosis.[48]

Delayed feeding (generally defined as >24 hours) is associated with higher rates of infected peripancreatic necrosis, multiple organ failure, and total necrotizing pancreatitis.[49,50,51] Success of early feeding has been demonstrated with low-fat, normal fat, and soft or solid consistency and thus it is not necessary to start AP patients on a clear liquid diet before advancement to a solid diet.[52,53] Patients who cannot tolerate an oral diet may require enteral tube placement for nutritional support, however the risk of aspiration should be considered in patients with severe AP. There does not appear to be an advantage to post-pyloric tube placement over gastric tube placement.[54] There is, however, clear evidence demonstrating enteral nutrition over total parenteral nutrition (TPN) is associated with reduced risk of infected peripancreatic necrosis and multiple organ failure.[55,56] For this reason, TPN use in AP is discouraged.

Management – Urgent ERCP

Urgent ERCP (typically defined as occurring within the first 72 hours) is indicated in patients with acute cholangitis regardless of the presence of acute biliary (i.e. gallstone) pancreatitis.[57,58] However, in patients with gallstone pancreatitis who do not have

cholangitis, the role of ERCP has been debated. Data from randomized controlled trials on this topic show that compared to conservative management, urgent ERCP in gallstone pancreatitis has not been demonstrated to improve critical outcomes, such as mortality and multiple organ failure, infected pancreatic and peripancreatic necrosis, or total rates of necrotizing pancreatitis.[59,60,61,62] Thus, urgent ERCP is typically not recommended in AP in the absence of acute cholangitis. There may be a reduction in hospital length of stay for patients with AP undergoing ERCP, though this was only demonstrated in a single study.[63]

Management – Cholecystectomy

In cases of AP secondary to gallstone disease, cholecystectomy should be performed to prevent recurrent episodes of AP.[64] The optimal timing of surgery weighs the challenges of operating on a patient with an acutely inflamed pancreas against the risk for recurrent gallstone-related complications. Patients discharged without a cholecystectomy have a significant risk of recurrent AP and/or cholangitis.[65] Data from a randomized controlled clinical trial found that cholecystectomy during the initial admission for patients with gallstone pancreatitis was associated with reduction in mortality and gallstone-related complications, readmission for recurrent pancreatitis, and pancreaticobiliary complications.[66] The current recommendations favor cholecystectomy during the same admission when the etiology of AP is determined to be gallstones.[43]

Management – Alcohol Counseling

In patients with a first attack of AP with a clear history of alcohol use, counseling has demonstrated a reduction in total hospital admission rates and can reduce the risk of AP recurrence.[67,68]The SBIRT (Screening, Brief Intervention, and Referral to Treatment) approach to alcohol counseling is effective in reducing alcohol consumption.[69] Therefore, because reduction or cessation of alcohol abuse clearly reduces the risk of recurrent alcoholic AP,[70] and given the negligible harms and relatively low cost of the intervention, it is generally recommended that patients presenting with AP secondary to alcohol abuse receive at least brief alcohol counselling during the index admission.

3.0 Conclusion

Current evidence supports the benefit of goal-directed fluid resuscitation and early oral feeding in all patients with AP. Patients with biliary pancreatitis should preferentially undergo same-admission cholecystectomy. Alcohol counseling for patients with alcohol-induced pancreatitis should be provided. Current data do not support the routine use of prophylactic antibiotics in AP or routine ERCP in patients with AP without accompanying cholangitis.

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4.0

Key Points

- Acute pancreatitis is defined by two of the following: stereotypical abdominal pain, amylase and/or lipase > 3 times the upper limit of normal and imaging findings consistent with the condition.
- Patients presenting with AP should undergo transabdominal ultrasound to evaluate for gallstone disease and a thorough alcohol use history should be obtained.
- Fluid resuscitation in acute pancreatitis should focus on improving heart rate, mean arterial pressure, central venous pressure, urine output, blood urea nitrogen concentration, and hematocrit. Ringer's lactate is generally favored over normal saline as the resuscitative fluid of choice.
- Early oral feeding should be encouraged in AP and if a patient cannot tolerate oral intake, placement of an enteral feeding tube should be considered.
- Cholecystectomy during the same admission of biliary pancreatitis should be pursued and patient with AP secondary to alcohol should receive counselling on alcohol abuse.

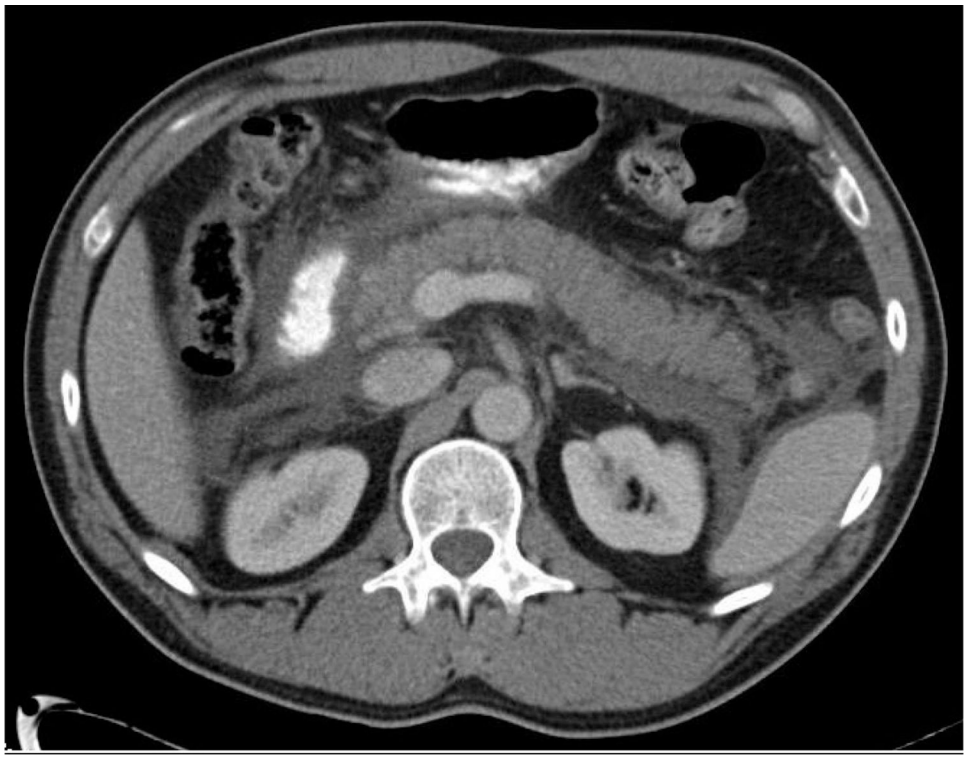

Figure 1:
Contrast enhanced computed tomography of a patient presenting with acute pancreatitis from gallstone disease. CT demonstrating characteristic imaging findings of interstitial edematous pancreatitis, with a heterogeneously enhancing, edematous pancreas.

Table 1:

The Revised Atlanta Classification of Acute Pancreatitis (2013)

Mild acute pancreatitis
Absence of organ failure
Absence of local complications
Moderately severe acute pancreatitis
1. Local complications **AND/OR**
2. Transient organ failure (<48 hours)
Severe acute pancreatitis
Persistent organ failure >48 hours

Table 2:

Modified Marshall scoring system for organ dysfunction.

	Score				
Organ system	**0**	**1**	**2**	**3**	**4**
Respiratory (PaO_2/FiO_2 ratio)	> 400	301 – 400	201 – 300	101 – 200	≤ 101
Renal (serum creatinine)	< 1.4 mg/dL	1.4 – 1.8 mg/dL	1.9 – 3.6 mg/dL	3.6 – 4.9 mg/dL	> 4.9 mg/dL
Cardiovascular (systolic blood pressure, pH)	> 90 mm Hg	< 90 mm Hg, fluid responsive	< 90 mm Hg, not fluid responsive	< 90 mm Hg, pH < 7.3	< 90 mm Hg, pH < 7.2

A score of ≥ 2 in any system defines the presence of organ failure.

Transient organ failure: organ failure resolving after ≤ 48 hours; Persistent organ failure: organ failure persisting > 48 hours.

Table 3:

Fluid therapy goals to prevent hypovolemia and organ hypoperfusion in acute pancreatitis[23]

Central venous pressure
8 – 12 mm Hg
Mean arterial pressure
65 – 90 mm Hg
Central venous oxygen saturation
>=70%