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Acute Pancreatitis – Be Quick or Be Sorry

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Acute pancreatitis (AP) is an acute inflammatory disease of the pancreas with high morbidity and mortality if not managed properly. Reported mortality rates from the USA of this condition range from 3% for patients with interstitial oedematous pancreatitis[1] up to 20–40% for those with severe pancreatitis.[2]

The most common causes of AP are gallstone disease (40-70%) and alcohol (25-35%)[3]. Other less common causes are iatrogenic (thiopurines, valproate, post-endoscopic retrograde cholangiopancreatography), metabolic disorders (hypertriglyceridaemia, hypercalcaemia), congenital abnormalities (pancreas divisum, annular pancreas, choledochal cysts), tumours (pancreatic tumours, periampullary tumours), pancreatic trauma, autoimmunity, genetic, toxic (venom), viral infections and obstruction by parasites (ascariasis).

This review will mainly focus on the initial management steps in acute pancreatitis since management decisions in this period can alter the course of the disease and decide whether the patient will develop multi-organ failure within the next few days.

Diagnosis

The diagnosis of acute pancreatitis requires the presence of ≥2 of the following criteria:[4]

- Abdominal pain consistent with pancreatitis – sudden onset severe epigastric pain that may radiate to the back and is usually relieved by bending forward
- Serum amylase or lipase >3 x upper limit of normal
- Characteristic findings from abdominal imaging - ultrasound (US) / contrast-enhanced computer tomography (CECT) / Magnetic resonance cholangiopancreatography (MRCP)

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In most instances, US abdomen is adequate for the diagnosis. CECT and MRCP are generally reserved for patients in whom the diagnosis is unclear even after US imaging. Further imaging by CECT / MRCP may be warranted later on in the disease process for evaluation of local complications if the patient does not clinically improve. Since the extent of pancreatic necrosis may not be clearly defined during the initial few days of the disease, imaging at 3-4 days after the onset of acute pancreatitis is more reliable.[5]

For identification of the aetiology of acute pancreatitis, liver biochemistry within 48 hours after the onset of symptoms may be important. An alanine aminotransferase (ALT) level >150 U/L discriminates biliary pancreatitis with a positive predictive value of >85%. Alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT) and bilirubin levels also may indicate the possibility of gallstone pancreatitis. US abdomen may show a dilated biliary tree from an obstructed gallstone.[5]

Further testing may be warranted once the patient has recovered from the acute illness when a clear aetiology is not revealed by the history and basic investigations. CECT abdomen may identify gallstones not detected by US and may reveal pancreatic or ampullary masses. Depending on availability, MRCP or endoscopic ultrasound should be performed when conventional imaging is negative as they are the best investigations for microlithiasis (tiny biliary calculi), pancreatic duct abnormalities and small pancreatic masses and periampullary masses.[5]

Serum triglyceride and serum calcium should also be part of the routine screening process for an aetiology.

Classification

The Revised Atlanta Classification system (2012) is an internationally accepted system that categorizes acute pancreatitis according to severity into mild acute pancreatitis, moderately severe acute pancreatitis and severe acute pancreatitis depending on the presence or absence of local complications (peripancreatic fluid collections, gastric outlet ob-

struction, splenic or portal vein thrombosis, colonic necrosis) and systemic complications (respiratory, cardiovascular or renal organ failure or exacerbation of pre-existing co-morbidity precipitated by acute pancreatitis).[4]

Mild acute pancreatitis is where there are no local or systemic complications of acute pancreatitis. These patients typically improve and are able to start feeding by 48 hours. The diagnosis of moderately severe acute pancreatitis requires fulfilment of one or more of the following criteria: local complications, transient organ failure lasting <48 hours, and exacerbation of any co-morbid diseases due to acute pancreatitis. Severe acute pancreatitis is defined by the presence of persistent organ failure lasting >48 hours.[4]

An issue with categorizing patients according to this system into moderately severe acute pancreatitis and severe acute pancreatitis is that the final categorisation is only possible retrospectively after 48 hours have elapsed. Because of this, all patients with organ failure will have to be managed initially as potentially having severe acute pancreatitis.

In the past, much emphasis was placed on scoring systems (e.g. APACHE II, Ranson, modified Glasgow) in predicting severe acute pancreatitis. However, these scores are cumbersome to calculate and typically require 48 hours to become accurate, by which time the disease severity is obvious regardless of the score. Currently, the best marker for predicting acute severe pancreatitis is considered as fulfilment of systemic inflammatory response syndrome (SIRS) criteria on admission (≥2 of the following: PR >90/min, RR >20/min or PaCO2 <32 mmHg, temperature >38 °C (100.4 °F) or <36 °C (96.8 °F), WBC >12,000 or <4,000 cells/mm 3 or >10% immature neutrophils). The best strategy to predict the outcome of acute pancreatitis is considering a combination of 3 factors: host risk factors e.g. age (>55 years indicating poor prognosis), co-morbidity, body mass index (obesity indicating poor prognosis); clinical risk stratification e.g. SIRS; monitoring response to initial therapy e.g. persistent SIRS, non-response of hypovolemia (rising haematocrit, blood urea, creatinine).[5]

Management

During the evolution of acute pancreatitis, two peaks of mortality have been identified. The first is during the first week of the disease when there is sterile inflammation of the pancreas. This inflammation can subsequently progress to a systemic level (SIRS) and result in organ failure. The second peak occurs after the first week and is due to infection of the necrotic pancreatic tissue[6]. To prevent mortality these 2 phases of disease must be managed properly.

Fluid management

Hypovolemia may occur from multiple factors affecting patients with AP, including vomiting, reduced oral intake, third spacing of fluids, increased respiratory losses and diaphoresis. Pancreatic hypoperfusion leads to increased pancreatic necrosis and ongoing release of pancreatic enzymes activating numerous cascades. Correct fluid management is the most important aspect of management during the early phase of the disease.

To prevent future complications, all patients should receive aggressive hydration during the first 24 hours of the disease. The recommended rate of fluid administration is 5-10ml/kg/h (250-500 ml/h in a 50kg patient)6. A patient in shock may need more rapid repletion as boluses, while the rate of administration may have to be reduced in those with coexisting cardiovascular or renal disease.

Replacement should be with an isotonic crystalloid fluid but there is still no consensus as to which fluid is best, with some guidelines recommending Lactated Ringer's solution5 while others see no difference between normal saline and Ringer's lactate.[7]

However, overly aggressive fluid therapy also increases morbidity and mortality due to pulmonary oedema and abdominal compartment syndrome. Therefore, the patient should be closely monitored and the fluid administration reassessed at frequent intervals to achieve goals of heart rate <120, mean arterial pressure (MAP) >65 mmHg, urinary output >0.5 ml/kg/h and haematocrit 35–44%.

After the resuscitation goals are met, the rate of fluid administration can usually be reduced to 2–3 ml/kg/h.[6]

During the early critical phase, vasopressors might be administered as an adjunct to fluid administration to temporarily increase a low MAP.[6]

Patients undergoing volume resuscitation should have the head of the bed elevated, undergo continuous pulse oximetry, and receive supplemental oxygen.

Antibiotics

Routine administration of antibiotics in acute pancreatitis is discouraged in international guidelines[5-7]. This is because prophylactic antibiot-

ics have not been found to have any impact on the rates of organ failure and length of hospital stay.[7]

Antibiotics are indicated if infected pancreatic necrosis is suspected based on failure to clinically improve after 7–10 days of hospitalization, imaging signs of infection (i.e. gas in peripancreatic collections) and if organisms are found in percutaneous fine needle inspiration (FNA) of peripancreatic collections. When infected necrosis is suspected, antibiotics are started empirically after obtaining blood cultures and discontinued if cultures are found to be negative. The choice of empirical antibiotics should be based on local sensitivity patterns as well as the antibiotic's ability to penetrate pancreatic necrosis. Carbapenems, quinolones, cephalosporins in high doses and metronidazole have good pancreatic tissue penetration.[8]

Analgesia

Opiates are often necessary to achieve effective analgesia in these patients. There is no evidence from human studies to indicate which specific opiates are best.[9]

Nutrition

There is a longstanding misconception that the inflamed pancreas requires prolonged rest by fasting. Bowel rest is associated with disturbed intestinal motility, bacterial overgrowth and intestinal mucosal atrophy, which leads to bacterial translocation from the gut that can result in infection of the necrotic pancreatic tissue. Therefore, early oral feeding is recommended if the patient is clinically improving with a reduction in nausea & vomiting and abdominal pain. If the patient cannot take orally, enteral feeding by nasogastric tube is recommended. If the patient does not tolerate nasogastric feeding due to delayed gastric emptying, naso-jejunal feeding is an option. Parenteral nutrition should be the last option if the patient does not tolerate any of the enteral feeding methods even by 5th day after admission.[5,9]

Invasive interventions in acute pancreatitis
Interventions for local complications

The most common local complication that can occur with acute pancreatitis is peripancreatic fluid collections. The vast majority of patients with fluid collections can be managed without interventions and unnecessary invasive procedures can increase morbidity and mortality.

Indications for drainage of collections are suspicion of infection, obstruction of surrounding structures by the collection (e.g. biliary obstruction, gastric outlet obstruction, intestinal obstruction), persistent symptoms such as pain, loss of appetite and loss of weight (persisting >8 weeks after the onset of acute pancreatitis)[5]. In the absence of these indications, collections do not warrant intervention regardless of their size or location.

Interventions should preferably be delayed for >4 weeks from the onset to allow for the development of walling-off of the collection but in unstable patients, interventions may have to be performed earlier.[5]

It is preferable to use the least invasive means to drain the collection as this reduces pro-inflammatory activity and reduces mortality and hospital stay. Endoscopic drainage or percutaneous image-guided drainage are therefore the preferred methods while surgical drainage is restricted to patients in whom the less invasive methods are not possible[5].

Gallstone pancreatitis

Endoscopic retrograde cholangiopancreatography (ERCP) should not be used routinely for patients with gallstone pancreatitis because it can increase complications. There are only two instances when urgent ERCP (within 24 hours) is warranted in gallstone pancreatitis – concurrent acute cholangitis and the presence of ongoing biliary obstruction.[5]

To prevent the recurrence of gallstone pancreatitis, cholecystectomy is recommended during the index admission rather than a more delayed approach[7].

Summary

Acute pancreatitis is a condition that can lead to much morbidity and mortality if there is mismanagement in the initial 1-2 days of the disease. The best marker for predicting acute severe pancreatitis is considered as the fulfilment of SIRS criteria on admission. The most important aspect of the initial management is adequate fluid resuscitation. The recommended rate of fluid administration is 5-10ml/kg/h. Routine administration of antibiotics is not recommended and antibiotics are indicated only if infected pancreatic necrosis is suspected. Opiates are often necessary to achieve effective analgesia. Prolonged bowel rest by fasting is not recommended and enteral feeding is recommended if the patient is clinically improving. The vast majority of patients with peripancreatic fluid collections can be managed without interventions. Indications for drainage of collections are suspicion of infection, obstruction of surrounding structures by the collection and persistent symptoms. Interventions should

preferably be delayed for >4 weeks from the onset of the disease. ERCP should not be used routinely for patients with gallstone pancreatitis and is warranted only if there is concurrent acute cholangitis or ongoing biliary obstruction. To prevent the recurrence of gallstone pancreatitis, cholecystectomy is recommended during the index admission rather than a more delayed approach.

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