Acute Pancreatitis

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Gallstones and excessive alcohol intake account for approximately 80% of cases. Other causes have been attributed to viral infections, medications, hypertriglyceridemia and hypercalcaemia.

12.1 Terminology

Severe acute pancreatitis is characterised by the presence of organ failure or local complications. If an uncomplicated course ensues or there is transient dysfunction it is termed mild and moderate, respectively. There are several scoring systems for severity (e.g. Ranson, Imrie, APACHE and CRP). In practical terms, the CRP is very useful for monitoring progress.

Pancreatic necrosis is usually diagnosed by contrast CT. Large areas of swollen and inflamed tissue which are poorly perfused are an indication of likely necrosis. CT can also demonstrate gas formation which is indicative of infected necrosis. CT can also demonstrate peripancreatic fluid. In the first 4 weeks this usually does not have an organising wall. After 4 weeks the fluid is often within a contained capsule. This is the time when pancreatic pseudocyst formation occurs. Fluid collections in necrotising pancreatitis are classified as acute necrotic collection prior to 4 weeks and walled-off necrosis beyond 4 weeks.

Pancreatic pseudocysts appear as cystic structures within and around the pancreas that have formed from inflammatory exudate and fibrosis. It is caused by the disruption of the main duct of the pancreas. The contained fluid has inflammatory material and is enzyme-rich.

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F. Piscioneri et al. (eds.), *Emergency Surgery for Low Resource Regions*,
Hot Topics in Acute Care Surgery and Trauma,
https://doi.org/10.1007/978-3-030-68099-2_12

Necrosoma (organised pancreatic necrosis) results from liquefaction of the pancreatic necrosis. It can take 4–12 weeks to form. It is semi-solid and well demarcated. This makes it more amenable to surgical excision.

12.2 Epidemiology

The incidence varies by region. About 80% have mild disease and make a full recovery. Around half of patients with severe disease will develop infected pancreatic necrosis. These patients have high mortality approaching 50%. Of these, approximately half the deaths occur during the first week as a result of organ failure. Infective complications are responsible for many of the later deaths.

12.3 Pathophysiology

There are two aspects to the disease. There is local inflammation and complications. The other is the Systemic Inflammatory Response Syndrome (SIRS) and Multiple Organ Dysfunction Syndrome (MODS).

The enzyme trypsin is central to the initial insult. It is able to not only activate itself but also activate other enzymes. This leads to autodigestion of the pancreas. The exact mechanism of how this occurs is not fully understood; however, the main risk factors are biliary disease and high alcohol intake. Inflammatory mediators spill out into the systemic circulation leading to SIRS and eventually MODS. The specific inflammatory mediators and their actions will not be discussed here.

12.4 Diagnosis

The majority of patients present with acute pain in the upper abdomen which often radiates to the back. Most patients have associated vomiting. Pain is relieved by sitting forward. Examination reveals marked epigastric tenderness and guarding. There may be a significant history of gallstone disease or recent high alcohol intake. There may also be evidence of retroperitoneal haemorrhage (Cullen's sign and Grey—Turner's sign). These are uncommon and occur usually after 48 h.

The main laboratory tests are serum amylase or serum lipase with levels greater than three times above the upper limit of normal. Patients with chronic pancreatitis who suffer an acute attack may not show significantly elevated levels. As alluded to before, CRP indices are a practical measure of severity and progress.

CT scanning of the abdomen will not only demonstrate acute pancreatitis but can also show areas of necrosis or gas formation. CT is also useful in ruling out other causes of abdominal pain. Ultrasound can demonstrate gallstones in the gallbladder and presence of a dilated common bile duct. It is less useful for diagnosing acute pancreatitis.

12.5 Initial Management

This centres around adequate analgesia, oxygen, aggressive treatment with crystalloids and nasogastric suction for management of gastroparesis and ileus. All patients must have DVT prophylaxis unless otherwise indicated.

Monitoring is by way of laboratory studies for renal function and electrolytes, cardiovascular observations and respiratory tests (blood gas/chest x-ray). Most patients can be managed in a general ward unless there is organ compromise and those patients should then be cared for in an ICU or HDU setting.

There are no specific treatments for acute pancreatitis so close monitoring and effective fluid management are essential. The CRP is a good indicator of patient progress and is readily available in most hospitals. The onset of organ dysfunction indicates a poorer prognosis and the requirements for more aggressive supportive therapy.

Early sphincterotomy (of the sphincterof Oddi) by ERCP is not usually indicated. Where there is common bile duct obstruction leading to acute cholangitis the situation then becomes more urgent for bile duct clearance. Most common duct stones will pass without operative intervention. If the cause is due to excessive alcohol intake, then appropriate preventative measures should be advised.

There is a small group of patients where no obvious cause is found and MRCP or ERCP in these patients can be performed in order to determine if there is a structural cause leading to pancreatitis.

The more severe cases are complicated by SIRS followed by multiple organ dysfunction syndrome (MODS). The transition usually occurs towards the end of the second week. There is some overlap of the conditions.

12.6 Role of Antibiotics

There is no indication for the routine use of antibiotics in acute pancreatitis. If infection is suspected by increased inflammatory markers, worsening observations or by CT evidence of infection, then directed antibiotics should be used. It is preferable that a fine needle aspiration is performed to identify the organism. Up to 30% of infected pancreatitis can contain candida so an antifungal therapy should be considered. Appropriate antibiotics are guided by local antibiotic stewardship practices. The length of course of antibiotics is controversial but any decision to prolong antibiotics greater 14 days should be taken on a case-by-case basis and in consultation with local infectious diseases experts. If the unit is receiving a transferred patient who has already been commenced on antibiotics then the 14-day course should be completed.

12.7 Nutrition

In mild acute pancreatitis, there is no need to fast the patient unless they are vomiting. If the patients are unable to tolerate oral intake, other methods need to be contemplated as these patients are in a state of catabolism. Most patients tolerate a

nasogastric tube and can be fed peptide-based feeds. For those that do not tolerate this then nasojejunal tubes can be inserted endoscopically or under radiological guidance. If either of these approaches are not successful then total parenteral nutrition should be considered.

12.8 Clinical Management

The management is influenced by whether the disease is mild or severe.

Those with mild disease usually resolve in 4–5 days. When the cause is gallstones, cholecystectomy should be undertaken during the index admission following duct clearance or resolution of the acute episode. Otherwise, the recurrence rate is in the order of 30% over the next few months.

Severe pancreatitis can be divided into early and late phases. The acute phase is related to the SIRS factors with features of hypovolaemia, inflammation and fluid exudation. The later phase is more closely related to MODS and usually manifests itself during the second week of disease. Death due to the complications of organ dysfunction are unusual in the first week.

Mild cases can be treated in a general surgical ward with supportive therapy and progress to discharge as the patient improves over the first few days.

Severe pancreatitis is quite resource-intensive. These patients are best treated in a HDU setting. An early contrast-enhanced CT scan will help determine the initial extent of tissue damage.

Management is related very much to how the patient is progressing. If the signs are that the patient is improving then repeat CT after 7–10 days is appropriate. If the patient deteriorates then a CT can help guide management. The main worrying concern at this stage is the infection of necrotic pancreatic tissue. If the inflammatory markers are rising and there are signs of gas bubbles on the CT then it has to be assumed that there is infection. Tissue sampling is required and this can be done either with a fine needle aspiration or the insertion of the pigtail catheter into the retroperitoneal space if there is a collection of fluid. Antibiotics should be started immediately and should be broad-spectrum and based on local antibiotic guidelines including consideration of antifungal therapy.

Early debridement of tissue should be avoided as surgical intervention in the first few weeks has a high mortality. It is best to wait until the necrotic tissue has organised and is better demarcated. This usually occurs by the third–fourth week. The traditional approach has been through open repeated laparostomies. However, there is now a trend to percutaneous drainage and repeated use of a resectoscope under direct vision.

If the facilities or skills do not exist for safe surgical intervention then patients should be transferred to a centre that can provide this level of care. In low resource regions where transfer is not an option, operative intervention should be avoided and supportive therapy is maximised.

A special mention should be made about haemorrhage. It can either be as a result of necrotising pancreatitis or iatrogenic due to intervention. The latter can be

associated with early surgical intervention of the necrotic tissue. This reinforces the need to delay surgery until at least 3or 4 weeks as stated above.

The inflammatory effect of pancreatitis can lead to pseudoaneurysm formation typically in the splenic, left gastric or gastroduodenal arteries. The haemorrhage can be massive and often rapidly fatal. If the bleeding is slow, then there is time for the patient to be adequately resuscitated and angioembolization to be organised and performed. This approach gives the best overall chance of survival.

Venous bleeding is less common and more difficult to diagnose. It requires carefully controlled resuscitation. If bleeding continues the only surgical option would be surgical removal of the distal pancreatic tissue.

12.9 Summary

Acute pancreatitis can be managed non-operatively in most situations. Operative intervention should be left to those with appropriate experience. Where resources are very limited it is best to transfer those with severe pancreatitis to a major centre when it is safe to do so.