Chapter 27
Acute Pancreatitis

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Key Points

- Early diagnosis and treatment are crucial in the management of acute pancreatitis to prevent complications and to reduce morbidity and mortality.
- Other life-threatening conditions which mimic acute pancreatitis should also be considered and ruled out simultaneously while managing the patient.
- Prophylactic antibiotics are not indicated in sterile pancreatic necrosis.
- Consider early admission in intensive care unit after initial resuscitation in the emergency department.

Introduction

- Acute pancreatitis (AP) is an acute inflammatory process in which there is autodigestion of pancreas by its own enzyme.
- Annual incidence of AP varies between 4.9 and 73.4 cases per 100,000 worldwide with an increasing trend in the annual incidence [1, 2]. Even though the case fatality rate for AP has decreased over time, the overall population mortality rate for AP has remained unchanged [3].
- Aetiological variation has been seen depending upon the lifestyle in different population.

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- Generally, acute pancreatitis is more common in males than females. In males, the aetiology is more often related to alcohol; in females, it is more often related to biliary tract disease.
- The overall mortality in patients with acute pancreatitis is 10–15 %. Mortality due to biliary pancreatitis is high as compared to alcoholic pancreatitis. Twenty percent of patients present with severe disease (organ failure) in whom, mortality is approximately 30 % [4].

Pathophysiology (Fig. 27.1)

Aetiology

- The causes of acute pancreatitis have been listed in Table 27.1 [5–8].
- Cholelithiasis is the most common cause of acute pancreatitis (40–70 %), whereas alcohol is the second most common cause (25–35 %) [9–11].

Classification

- Revised Atlanta criteria 2013 (Table 27.2) defines severity of acute pancreatitis into three categories – mild acute pancreatitis, moderately severe acute pancreatitis and severe acute pancreatitis [12].
- Local complications include peripancreatic fluid collections and pancreatic/peripancreatic necrosis (sterile or infected).
- Organ failure is defined as a score of 2 or more using the modified Marshall scoring system (Table 27.3) [12, 13].
- Phases of severe pancreatitis [14, 15]:
  - Early – usually last for the first week in which patient may present with systemic inflammatory response syndrome (SIRS).
  - Late – follows the early phase and lasts from weeks to months, usually characterised by local complications and/or persistent organ failure.
- Most patients with severe pancreatitis present to emergency department during the early phase without any signs of organ failure and local complications, thus leading to errors in clinical management of this disease [16].

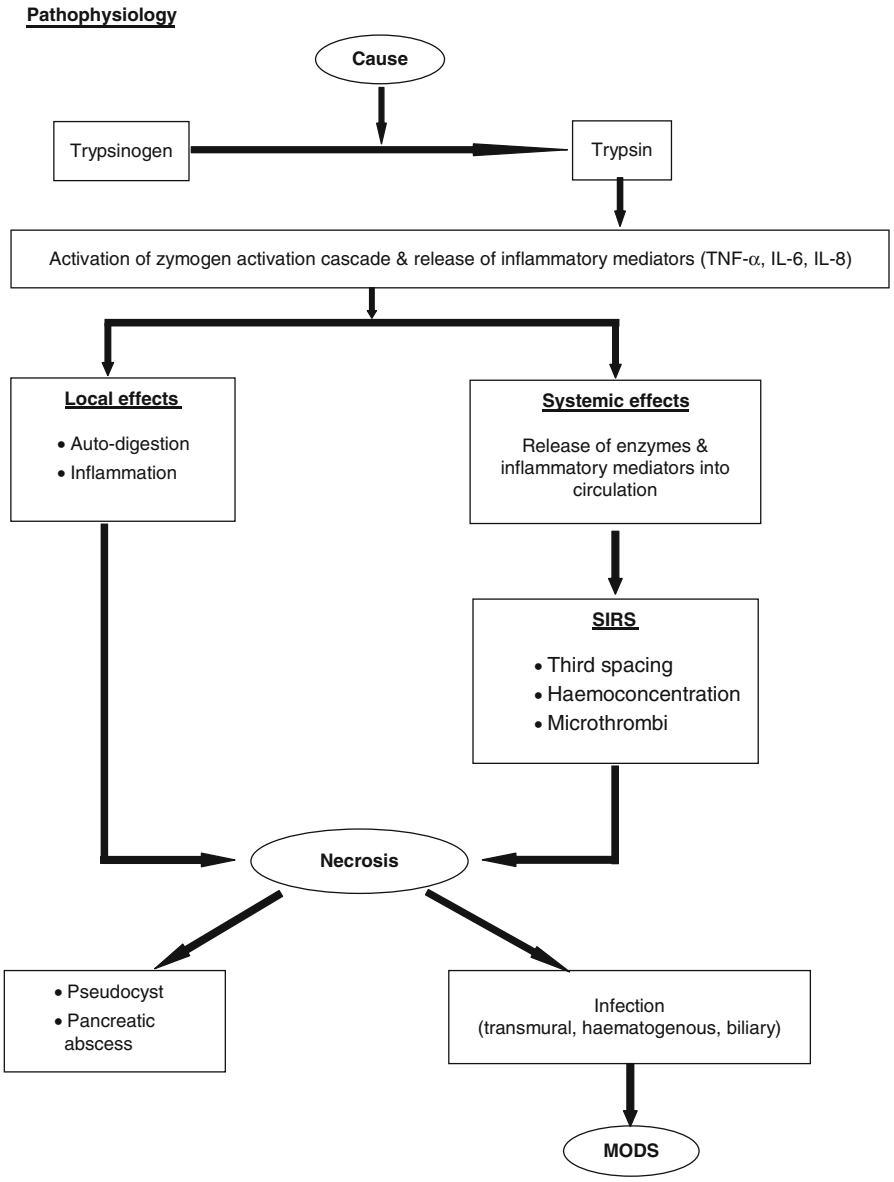

Fig. 27.1 Pathophysiology of acute pancreatitis

Table 27.1 Causes of acute pancreatitis

Cholelithiasis
Ethanol abuse
Idiopathic
Infections *Mumps*, *Coxsackie B*, *Mycoplasma*, *ascariasis*, *viral hepatitis (A, B, C)*, *HIV*, *Cytomegalovirus*, *varicella*, *Epstein-Barr virus*, *echovirus*, *adenovirus*, *legionella, leptospirosis*, *Campylobacter jejuni*, *tuberculosis*, *Mycobacterium avium*
Metabolic *Hypercalcaemia*, *hyperchylomicronaemia*, *diabetic ketoacidosis*, *uraemia*, *hypothermia*, *pregnancy (third trimester)*
Trauma *Postoperative trauma*, *blunt abdominal trauma*, *postrenal or cardiac transplant*, *ERCP*
Penetrating duodenal ulcer
Methyl alcohol
Organophosphate poisoning
Scorpion venom
Ischaemia *Polyarteritis nodosa*, *systemic lupus erythematosus*, *thrombotic thrombocytopaenic purpura*, *cardiopulmonary bypass*
Drugs *Thiazides*, *furosemide*, *azathioprine*, *mercaptopurine*, *oestrogens (oral contraceptives)*, *procainamide*, *sulphonamides*, *erythromycin*, *tetracycline*, *pentamidine*, *metronidazole*, *L-asparaginase*, *phenformin*, *valproic acid*, *paracetamol*, *salicylates*, *ACE inhibitors*, *losartan*, *propofol*, *nucleoside-analogue reverse transcriptase inhibitors*

Table 27.2 Revised Atlanta criteria 2013

Mild acute pancreatitis
Absence of organ failure
Absence of local complications
Moderately severe acute pancreatitis
Local complications and/or
Transient organ failure (<48 h)
Severe acute pancreatitis
Persistent organ failure (>48 h)

Clinical Features

- Mostly patient present with dull, constant, acute onset abdominal pain usually in the epigastric region, sometimes radiating to the back.
- Other symptoms include nausea, vomiting, anorexia and diarrhoea.
- Patient may have tachycardia, fever and/or hypotension.
- A few patients exhibit jaundice.
- On per abdomen examination, abdominal tenderness, guarding and distension may be present.
- Some patients may have dyspnoea, pleural effusion or acute respiratory distress syndrome (ARDS).

Table 27.3 Modified Marshall scoring system for organ dysfunction

	Score				
Organ system	**0**	**1**	**2**	**3**	**4**
Respiratory (PaO_2/FiO_2)	>400	301–400	201–300	101–200	≤101
Renal[a]					
(Serum creatinine, μmol/l)	≤134	134–169	170–310	311–439	>439
(Serum creatinine, mg/dl)	<1.4	1.4–1.8	1.9–3.6	3.6–4.9	>4.9
Cardiovascular (systolic blood pressure, mm Hg)[b]	>90	<90, fluid responsive	<90, not fluid responsive	<90, pH <7.3	<90, pH <7.2
For non-ventilated patients, the FiO_2 can be estimated from below:					
Supplemental oxygen (l/min)	**FiO_2 (%)**				
Room air	21				
2	25				
4	30				
6–8	40				
9–10	50				

A score of 2 or more in any system defines the presence of organ failure
[a]A score for patients with pre-existing chronic renal failure depends on the extent of further deterioration of baseline renal function. No formal correction exists for a baseline serum creatinine ≥134 μmol/l or ≥1.4 mg/dl
[b]Off inotropic support

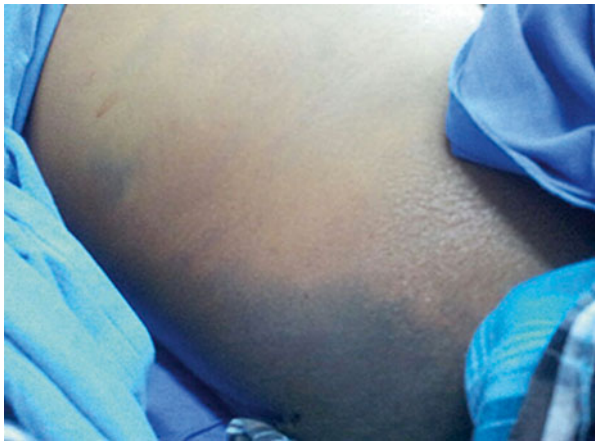

Fig. 27.2 Grey Turner's sign – contributed by Dr. Sandeep David, CMC Vellore, India

- Patients with severe acute pancreatitis may present with haematemesis or melaena and may be haemodynamically unstable.
- Patients with severe necrotising pancreatitis may have the following findings:
  - Cullen sign – bluish discolouration around the umbilicus due to haemoperitoneum
  - Grey Turner's sign – reddish-brown discolouration along the flanks resulting from retroperitoneal haemorrhage (Fig. 27.2)

Differential Diagnosis

Any acute abdomen or sometimes cardiac as well as pulmonary conditions can mimic AP. Some of the common differentials are enlisted in the box below.

- Acute mesenteric ischaemia
- Perforated gastric or duodenal ulcer
- Dissecting aortic aneurysm
- Biliary colic
- Acute myocardial ischaemia
- Ectopic pregnancy
- Intestinal obstruction
- ARDS

Diagnosis

- The diagnosis of AP should be considered in presence of two of the following three criteria:
  I. Typical abdominal pain suggestive of AP
  II. Serum amylase and/or serum lipase more than three times the upper limit of normal value
  III. Characteristic feature of AP in abdominal imaging
- Detailed history should be taken to find out the cause of AP, including history of alcohol consumption, hyperlipidaemia, similar episodes in the past, abdominal trauma and past history of gallstones or ERCP. Medication history should be asked to rule out drug induced AP.
- Apart from serum amylase and lipase, complete blood count including haematocrit, liver function test, serum triglyceride levels, serum calcium, blood urea nitrogen (BUN) and serum electrolytes should be checked to look for aetiology as well as to assess severity of AP.
- Serum triglyceride level of >1000 mg/dl is considered significant as a cause of AP in absence of gallstones and history of alcohol abuse.
- ECG – to rule out acute coronary syndrome.
- Chest x-ray erect view to look for air under diaphragm in case of intestinal perforation and also to aid to diagnosis of any pulmonary pathology, e.g. ARDS.
- In female patients under reproductive age group, bedside urine pregnancy card test should be done to rule out ectopic pregnancy.
- Transabdominal ultrasound should be done in all patients of AP to look for possible causes [17].
- In patients >40 years of age without any identifiable cause of AP, pancreatic tumours should be suspected as a probable cause [18, 19].

- Contrast-enhanced computed tomography (CECT) and/or magnetic resonance imaging (MRI) of the abdomen should be done only in patients in whom diagnosis is not certain or in those patients who do not show any signs of improvement within 48–72 h of hospital admission [20].

Management

- Assess and stabilise airway, breathing and circulation.
- Early aggressive intravenous hydration [21] with isotonic crystalloids to be started for all patients to correct hypovolaemia due to third spacing of fluids, vomiting, reduced oral intake, increased respiratory loses and/or diaphoresis. Special precaution to be taken in patients with renal and/or cardiac disease.
- Lactated Ringer's solution is the preferred crystalloid over 0.9 % normal saline for fluid replacement [22].
- Adequate analgesia should be given at the earliest. Inj. morphine at a loading dose of 0.1 mg/kg body wt. followed by 0.05 mg/kg body wt. every 5 min can be administered until the pain is relieved [23].
- Nasogastric (NG) tube to be inserted and patient to be kept nil per orally (NPO) to give rest to the inflamed pancreas; however prolonged fasting should be avoided. Early oral feeding in acute pancreatitis is beneficial in terms of shorter hospital stay, decreased infectious complications and decreased morbidity and mortality [24].
- Prophylactic antibiotics should not be given for severe AP and sterile necrosis [25]. Antibiotics should be given only if there is evidence of infected necrosis, extrapancreatic infection, cholangitis, bacteraemia, catheter-acquired infections, urinary tract infection and/or pneumonia.
- ERCP should be done within 24 h of admission in patients with concurrent acute pancreatitis and acute cholangitis [26].
- Patients with moderately severe or severe acute pancreatitis should be admitted to an intensive care unit.

Summary and Algorithm (Fig. 27.3)

Acute pancreatitis is associated with emotional, physical, as well as financial burden on the society [3] with significant morbidity and mortality. Early diagnosis and early aggressive intravenous hydration can reduce morbidity and mortality as well as prevent complications. Contrast-enhanced computed tomography (CECT) and/or magnetic resonance imaging (MRI) should be reserved for patients who fail to improve clinically or in whom diagnosis is not confirmed. Patients with moderately severe or severe acute pancreatitis should be admitted to intensive care unit whenever possible. It is important to rule out other life-threatening differential diagnosis of acute pancreatitis before shifting the patients from the emergency department.

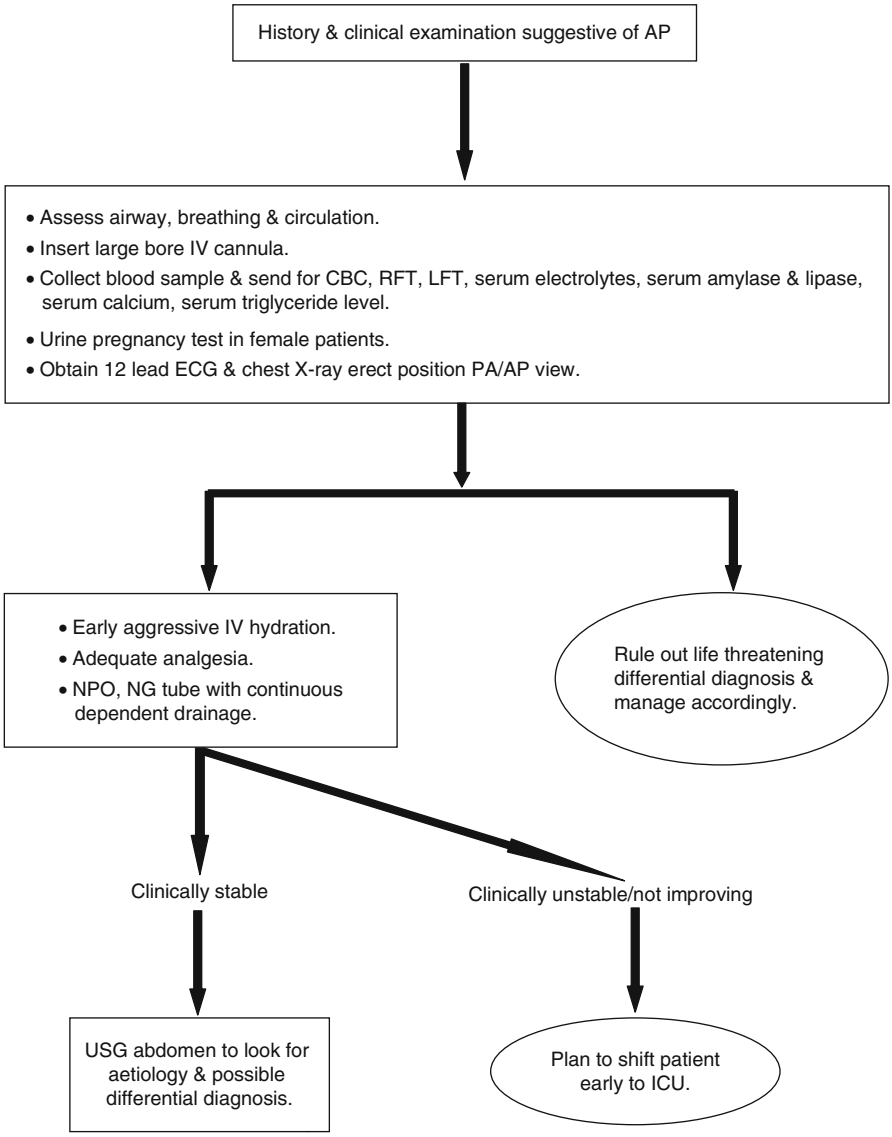

Fig. 27.3 Approach algorithm for acute pancreatitis in ED

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