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CRITICAL CARE MEDICINE

Bench to Bedside



Editors
Bikram Kumar Gupta
Syed Moied Ahmed
Rakesh Garg
Suhail Sarwar Siddiqui
Mohd Saif Khan

Forewords
Rajesh Chandra Mishra
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CRITICAL CARE
MEDICINE
Bench to Bedside

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CHAPTER

24 Severe Acute Pancreatitis

Pankaj Singh Rana, Saurabh Chandrakar

CASE VIGNETTE

A 52-year-old chronic alcoholic male patient with prior history of hypertension presented to the hospital with complaints of abdominal pain for last 4 days. There was a history of multiple episodes of nonbilious vomiting and decrease urine output for last 2 days. Patient was in agony and lying still on bed. His laboratory investigations showed elevated serum lipase (412 IU/L) and serum amylase: (1,428 IU/L) levels, along with raised serum creatinine (6.2 mg/dL). CT Abdomen finding suggestive of acute severe pancreatitis with moderate ascites [modified computed tomography (CT) severity index 8], mild hepatomegaly, and minimal left-sided pleural effusion. A presumptive diagnosis of severe acute pancreatitis (AP) with associated acute renal failure was made.

Acute pancreatitis, the most common inpatient gastrointestinal ailment, is a pancreatic inflammation with wide range of severity as well as local and systemic complications. Patient outcome depends upon its rapidity of onset, severity, and multiorgan involvement. Despite significant improvement in protocolized approach, patient outcome is still poor in severe cases.

Q. 1. How do you define acute pancreatitis?

The revised Atlanta classification requires two or more of following criteria to establish the diagnosis of AP. It includes characteristic abdominal pain suggestive of AP, raised serum amylase or lipase level at least >3 times upper normal limit, characteristic imaging evidence of pancreatitis. Radiological imaging modalities should be done only when the diagnosis is unclear or patient is not improving clinically within the first 48–72 hours after the hospital admission. These imaging modalities should not be used solely to make the diagnosis of AP.

Q. 2. What are different causes of acute pancreatitis?

There are several causes of AP which could be broadly grouped into mechanical-obstructive, vascular, infectious, toxic-metabolic, or miscellaneous category **(Fig. 1)**; however,

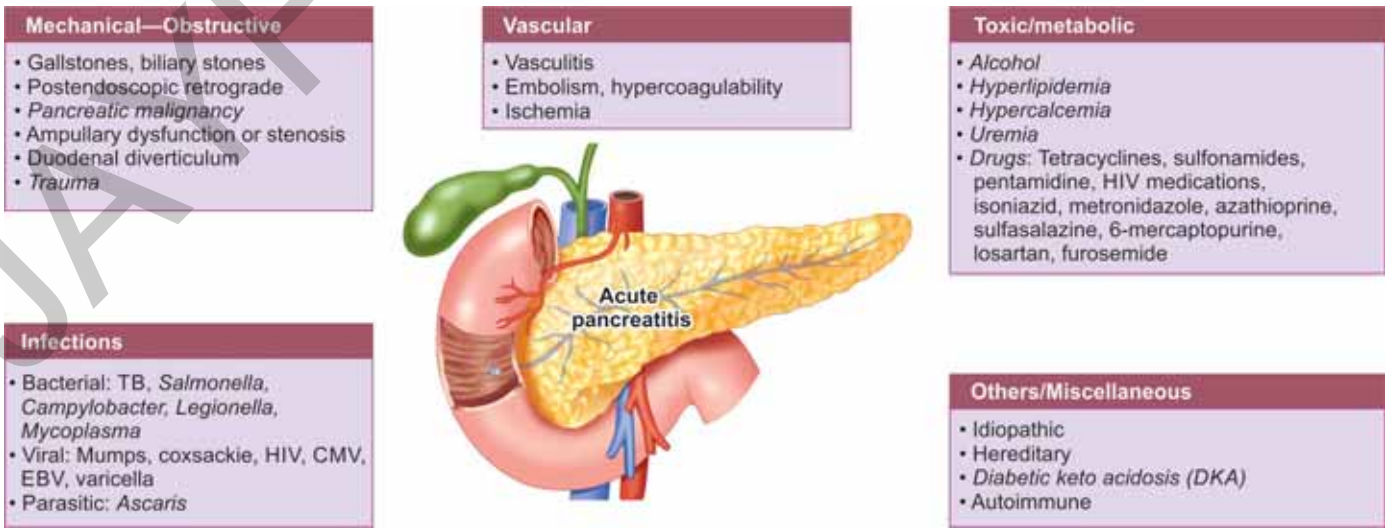


Fig. 1: Etiology of acute pancreatitis. (CMV: cytomegalovirus; EBV: Epstein–Barr virus; HIV: human immunodeficiency virus)

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most common ones are gallstones (40%) and chronic alcohol consumption (30%). Certain population groups are at higher risk for AP including male gender, age >60 years, diabetics, obese, high triglyceride levels, chronic alcohol consumption, smokers, autoimmune disease, cystic fibrosis, genetic predisposition, and family history of pancreatitis and/or gallstones.

Q. 3. Describe the characteristics of abdominal pain and other symptoms in acute pancreatitis.

Abdominal pain is cardinal symptom of AP, usually in the epigastric region, sudden in onset, dull, boring, radiation to back, and gradually increases in severity until it reaches to constant ache. It may be associated with nausea, vomiting, anorexia, dyspnea, tachypnea, and abdominal distension. It is aggravated by supine posture, eating and drinking (especially alcohol), and relieved by sitting and leaning forward. Presence of shock with above symptoms indicates a third space fluid loss or ongoing internal hemorrhage.

Q. 4. How do you classify acute pancreatitis?

On the basis of pathophysiological involvement, AP can be categorized as early or late phase. The early phase involves acute systemic inflammatory response and occurs during the first week after symptom onset. During this phase, the clinical severity of AP and its treatment strategies are based of type and severity of organ failure. The late phase, involves persistent organ failure and complications in severe involvement, generally starting from the second week, which may last for even months.

On the basis of severity, AP is divided into mild, moderately severe, and severe AP (revised Atlanta classification 2016). Mild pancreatitis donates no organ failure or local complication, and such patients are discharged within first week, with very low mortality. Moderately severe AP is indicated by transient organ failure lasting <48 hours and/or local or systemic complications. Any organ failure persisting >48 hours, is defined a severe disease. The modified Marshall scoring system is used to define organ failure taking into account parameters of respiratory, renal, and cardiovascular systems **(Table 1)**. A score of ≥2 for any system indicating organ failure.

Based on radiological imaging, AP is categorized as interstitial edematous pancreatitis (IEP) and acute necrotizing pancreatitis. IEP indicates non-necrotizing pancreatic inflammation and a more common variety. It precipitates as focal or diffuse pancreatic enlargement encircled by flimsy peripancreatic inflammation or small fluid collection. Necrotizing pancreatitis may involve either or both pancreatic parenchyma or the peripancreatic tissues. The combined pancreatic and peripancreatic involvement is usually more common.

TABLE 1: Modified Marshall scoring system.

	Score				
Organ system	***0***	***1***	***2***	***3***	***4***
Respiratory*	>400	301–400	201–300	101–200	≤100
Renal: Serum creatinine (mg/dL)	≤1.4	1.5–1.8	1.9–3.5	3.6–4.9	≥5
Cardiovascular: Systolic blood pressure (mm Hg)	>90	<90, fluid responsive	<90, not fluid responsive	<90 with pH <7.3	<90 with pH <7.2

*Partial pressure of oxygen/fraction of inspired oxygen (PaO_2/FiO_2)

Q. 5. How would you approach a patient with acute pancreatitis?

It involves meticulous history taking, careful examination, and laboratory workup and continuous monitoring for better patient outcome **(Tables 2 to 4)**.

TABLE 2: Key clinical findings on physical examination.

Examination	***Key elements/features***
General	Distressed, anxious, fever
Respiratory	Tachypnea, pleural effusion (usually left sided)
Hemodynamic	Hypotension, tachycardia
Abdomen	*Common:* Epigastric tenderness, guarding, rigidity, distension *Uncommon:* Discoloration of flanks due to retroperitoneal hemorrhage (Gray Turner sign), edema and bruising in the subcutaneous fatty tissue around umbilicus (Cullen sign), pain or tenderness in epigastric region (6–7 cm above umbilicus) (Korte sign), pain under xiphoid process (Kamenchik's sign)

TABLE 3: Relevant laboratory investigations in acute pancreatitis.

Laboratory investigations	***Key elements and features***
Serum amylase	Half-life is 10 minutes; levels normalize in 3–5 days, may also get elevated in disease of salivary gland, intestine, gallbladder, and appendix
Serum lipase	Half-life is 7–13 hours; levels normalize in 1–2 weeks, may also get elevated in disease of gallbladder and appendix
Liver function test	Raised SGOT, SGPT, CRP, LDH, and low serum albumin
Renal function test	Blood urea and creatinine levels should be performed to exclude acute renal failure
Complete blood count	Leukocytosis indicates SIRS, hemoconcentration

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Laboratory investigations	***Key elements and features***
Serum calcium level	Decreased indicates hypoalbuminemia or fat necrosis while raised value is observed if hypercalcemia is the causative factor
Blood sugar level	May be raised because of the β-cell dysfunction
Lipid profile	To exclude hypertriglyceridemia as the etiology if serum triglyceride value >1,000 mg/dL
Arterial blood gas	To assess oxygenation, ventilation, and acid-base disorder
Miscellaneous	Raised CRP, procalcitonin, phospholipase A, urine trypsinogen, blood culture

(CRP: C-reactive protein; LDH: lactate dehydrogenase; SGOT: serum glutamic-oxaloacetic transaminase; SGPT: serum glutamic-pyruvic transaminase; SIRS: systemic inflammatory response syndrome)

TABLE 4: Relevant radiological investigations in acute pancreatitis.

Radiological workup	***Key features***
Radiological workup is indicated in doubtful diagnosis, severe acute pancreatitis for first 72 hours, initial improvement followed by deterioration, and to rule out complications	
Chest X-ray	To rule out pleural effusion, collapse, consolidation, infiltrates
X-ray abdomen	May show gallstone, pancreatic calcification. Also done to rule out perforation, obstruction
Ultrasonography abdomen	It has limited value due to gaseous bowel. Gallstones suggest gallstone pancreatitis, and size of the common bile duct helps in diagnosing choledocholithiasis, biliary obstruction, pancreatic pseudocyst
CT abdomen	It aids the diagnosis of complications associated with the pancreatitis. Pancreatic head involvement indicates poor prognosis, while tail involvement has better prognosis. In early stages of disease (<48–72 hours), CECT should not be done just to make the diagnosis. After 48–72 hours, CECT helps in detection of pancreatic and/or peripancreatic necrosis, acute pancreatic fluid collections. Late CECT (after 7–21 days) is helpful in determining the severity and management like aspiration/drainage
Endoscopic retrograde cholangiopancreatography (ERCP)	Early ERCP (<48–72 hours) is desirable only in cases of acute biliary cholangitis pancreatitis. Surgical procedure may be required to manage the cause of CBD obstruction, only when ERCP fails or not feasible

(CBD: common bile duct; CT: computed tomography; CECT: contrast-enhanced computed tomography)

Q. 6. Define various scoring systems used to prognosticate patients with acute pancreatitis.

Several scoring systems are in use to predict patient outcome in AP but none has been proven as ideal bedside tool for assessment of AP. It includes Acute Physiology and Chronic Health Evaluation (APACHE-II) score, Ranson's criteria, modified Glasgow or Imrie criteria, modified CT score index, bedside index for severity in acute pancreatitis (BISAP), modified computed tomography severity index (CTSI), and the harmless acute pancreatitis score (HAPS). These indices are not without limitations and often been criticized for their complexity, cumbersomeness, inadequate sensitivity/specificity, inability to calculate a score on admission and dependence on some variables usually not available at the time of admission. Most accepted systems include Ranson's, APACHE-II, CTSI, and BISAP criteria. The details of each score are as follows:

- The APACHE-II score was initially designed for AP patients requiring intensive care. It uses a total of 15 variables. It has good negative predictive value and average positive predictive value for predicting severe disease. Decreasing trend during first 48 hours is suggestive of a mild disease, while rising trend indicate severe disease. An APACHE-II score >8 is considered high risk for severe disease. Limitations include complex and cumbersome to use could not differentiate interstitial and necrotizing pancreatitis, and have a poor predictive value at 24 hours of admission.
- Ranson's criteria is evaluated at admission and at 48 hours after admission. At admission: Five variables are included, while at 48 hours six variables are included. Each variable is given a single point. A score <3 indicate 1% mortality risk, 3–4 indicate 15%, 5–6 indicate 40% and score >6 indicates 100% mortality from AP, respectively. Limitations include high false-positive rate and poor positive predictive value **(Table 5)**.

TABLE 5: Ranson's criteria.

Criteria	***Parameters***
On admission	• Age >55 years • Total leukocyte count >16,000/μL • Lactate dehydrogenase (LDH) >600 U/L • Aspartate aminotransferase (AST) > 120 U/L • Glucose >10 mmol/L
With next 48 hours	• Hematocrit fall >10% • Urea rise >0.9 mmol/L • Calcium <2 mmol/L • pO_2 < 60 mm Hg • Base deficit >4 • Fluid sequestration >6 L

- *Modified Glasgow score* ***(Table 6)****:* It is performed within 48 hours of patient admission. It includes eight parameters. Each variable is given a single point.

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Score ≥3 indicates severe attack. It is more precise than the Ranson's scoring system in determining severe AP and has good accuracy in predicting need of ICU admission.

TABLE 6: Modified Glasgow score (Glasgow Imrie criteria for acute pancreatitis).

Criteria	*Parameters*
Within 48 hours of admission	• PaO_2 < 8 kPa • Age >55 years • Neutrophils >15 × 10^9/L • Calcium <2 mmol/L • Renal function urea >16 mmol/L • Enzymes LDH >600 IU/L, AST > 2,000 IU/L • Albumin <32 g/L • Sugar/glucose >10 mmol/L

(AST: aspartate aminotransferase; LDH: lactate dehydrogenase)

- *Bedside index for severity in acute pancreatitis* ***(Box 1)****:* It is performed within 48 hours of patient admission. It includes five parameters. Score ≥3 indicates significant risk of mortality. It has lower sensitivity and similar specificity when compared with other coring systems in use. It cannot easily distinguish between transient and persistent organ failure.

BOX 1: Bedside index for severity in acute pancreatitis (BISAP) score.

Criteria parameters:
- Blood urea nitrogen (BUN) > 25 mg/dL (1 point)
- Altered mental status with a Glasgow Coma Score <15 (1 point)
- Evidence of systemic inflammatory response syndrome (SIRS) (1 point)
- Age >60 years old (1 point)
- Imaging modality reveals pleural effusion (1 point)

- *Modified CT severity index* ***(Table 7)****:* It includes various CT findings such as pancreatic inflammation, fluid collections, pancreatic necrosis, and extrapancreatic complications. It is graded between 0 and 10. A CTSI score 0–3: mild; 4–7: moderate; 8–10: indicates severe pancreatic necrosis, respectively. It has a strong positive correlation with the development of complications and mortality in patients with severe disease. It correlates well with the length of hospital stay and the development of organ failure.

TABLE 7: Modified CT severity index (CTSI).

Criteria	*Parameters*
Pancreatic inflammation	*0:* Normal pancreas *2:* Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat *4:* Pancreatic or peripancreatic fluid collection and/or peripancreatic fat necrosis
Pancreatic necrosis	*0:* None *2:* 30% or less *4:* >30%
Extrapancreatic complications	*2:* One or more of pleural effusion, ascites, vascular complications, parenchymal complications, and/or gastrointestinal involvement

- The harmless acute pancreatitis score is based on three parameters and calculated within 30 minutes of admission. According to this, in patients with no sign of peritonitis and with normal hematocrit and serum creatinine levels, risk of mortality, necrosis, need for hemodialysis or ventilatory support is minimal. HAPS has been shown to be 97% specific for identifying a nonsevere disease course, although not sensitive.
- Several serum markers such as C-reactive protein (CRP), procalcitonin, and interleukin-6 are also used as indicator of severe AP. Among these, CRP has high sensitivity, but rises gradually according to severity of AP.

Q. 7. What is the role of lipase in diagnosing acute pancreatitis?

The sensitivity and specificity of lipase is around 90% to diagnose AP and has replaced amylase for the diagnosis of pancreatitis. Lipase levels >3 times the upper normal limit are used to diagnose AP. However, no correlation exists between the lipase levels and the severity of AP. Lipase elevation due to nonpancreatic pathologies tends to be <3 times the upper normal limit.

Q. 8. What is the role of procalcitonin in pancreatitis?

Procalcitonin is often conceptualized as a marker for bacterial sepsis, but it may be elevated in other conditions such as pancreatitis, renal failure, and other infection. In AP, procalcitonin may be used for:

- *Risk stratification:* Greater procalcitonin elevation indicates more severe inflammation, and thus predict a more severe disease
- Procalcitonin level has a good negative predictive value to rule out infected pancreatic necrosis; low levels are against infected necrosis, whereas an elevated level is nonspecific. It may avoid unnecessary antibiotic therapy or invasive procedures.

Q. 9. Describe approach for management of severe acute pancreatitis?

Patients suffering with severe AP, often requires admission in intensive care unit. Overall management of severe AP is conservative and includes supportive therapy. The salient points are mentioned below:

- *Initial resuscitation:* It includes assessment management of airway, breathing, and circulation as per general standard protocols elsewhere used in emergency settings. Patients with severe AP have large fluid shift to the third space, which leads on to hemoconcentration, and relative pancreatic hypoperfusion, which can further precipitate

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pancreatic necrosis. So rapid fluid restoration remains a priority. Choice of IV fluid is crystalloids, however, vasopressors and invasive hemodynamic monitoring is indicated in patients hypotensive despite adequate fluid resuscitation. Target is to meet urine output above 0.5 mL/kg/h and hematocrit below 30%. Any underlying electrolyte imbalance or coagulation abnormalities should be correct following the standard protocols.

- *Pain control:* An effective pain regimen in AP varies from conventional analgesics for mild cases to highly potent NSAIDs alone or in combination with opioids and even interventional procedures like thoracic epidural analgesia in severe AP. Current literature suggests that opioids are well tolerated in AP and there is no evidence higher morality or morbidity due to opioids. Though literature is limited for thoracic epidural analgesia, it provides rapid pain relief and reduces need for opiates in AP.
- *ICU admission:* Patient's with end-organ involvement including unstable hemodynamics, respiratory distress, reduced urine output, or impaired mentation indicates severe course and should be admitted to the ICU.
- *Antibiotics:* Prophylactic prescription of antibiotics in patients with AP should be avoided. In a patient with sterile necrosis, there is no role of antibiotic prophylaxis to prevent the development of infected necrosis. The only definitive indication is in cholangitis due to common bile duct (CBD) stones; antibiotic choice should cover gram-negative and anaerobic microorganism. In infected pancreatic necrosis, or other suspected infectious source, an antibiotic with better pancreatic tissue penetration including carbapenem and quinolones are recommended.
- *Other adjuvants:* Anti-inflammatory mediators may have a role if administered early. However, due to altered microcirculation and frequent lag time between symptom onset and drug administration, their role is still questionable. Vasoactive agents such as somatostatin and octreotide also decrease inflammatory mediators such as tumor necrosis factor-α (TNF-α), interferon, and decrease phagocytic activity of monocyte. However, their potent splanchnic obstruction effect renders them of no use in AP.
- *Nutrition:* Patients with AP are at moderate to high risk of malnutrition due to hypercatabolic state, which can lead to severe nutrition deficiencies, if not addressed early. Traditional concept of pancreatic rest is no longer validated and oral feeding should be instituted within 24 hours after hospital admission as tolerated. Thus, enteral nutrition (EN) (low-fat soft oral diet) shall be initiated as soon as clinically tolerated, with no consideration to serum lipase concentrations in patients with suspected mild AP. When choosing type of enteral formula, a standard polymeric diet is preferred. Even in patients unable to tolerate feed orally due to nausea and vomiting, EN should be started early, within 24–72 hours of admission and still preferred to parenteral nutrition (PN). EN should be initiated within first 24 hours after procedure like minimally invasive necrosectomy, if parameters such as hemodynamic stability, sepsis, and gastric emptying favor it. EN should be administered via a nasogastric tube. Nasojejunal feed in condition of gastric intolerance. In patient who still do not tolerate enteral feed or it is contraindicated, PN should be administered to target the nutritional requirements. Intra-abdominal pressure (IAP) and the vitals of patients should be monitored continuously. In severe AP or if IAP > 15 mm Hg, EN should be initiated via nasojejunal route starting at 20 mL/hour (trophic feeding), and titrated as tolerated. In severe AP and IAP > 20 mm Hg or abdominal compartment syndrome, EN should be temporarily stopped and PN should be started. For ongoing PN, parenteral L-glutamine should be supplemented at 0.20 g/kg/day. Probiotics or pancreatic enzyme supplementation is not recommended for AP.
- *Surgery:* In patients with gallstones, cholecystectomy surgery may be required to prevent repeated episodes of AP. However, it should be deferred in necrotizing biliary pancreatitis, till active inflammation resolves. In a stable patient with infected pancreatic necrosis, image-guided drainage of pancreatic collection should be differed for >4 weeks to allow for walled-off necrosis. For a symptomatic patient with infected necrosis, minimally invasive methods for drainage and necrosectomy are preferred over open surgical procedures. Other indications include pancreatic hemorrhage and acute abdomen.

Q. 10. What are the complications related to acute pancreatitis?

Complications related to AP can be categorized as local, peripancreatic, and/or systemic involvement **(Table 8 and Fig. 2)**. One should have high suspicion for complications in patients with persistent or recurrent symptoms, or secondary rise of serum markers, and/or signs of sepsis.

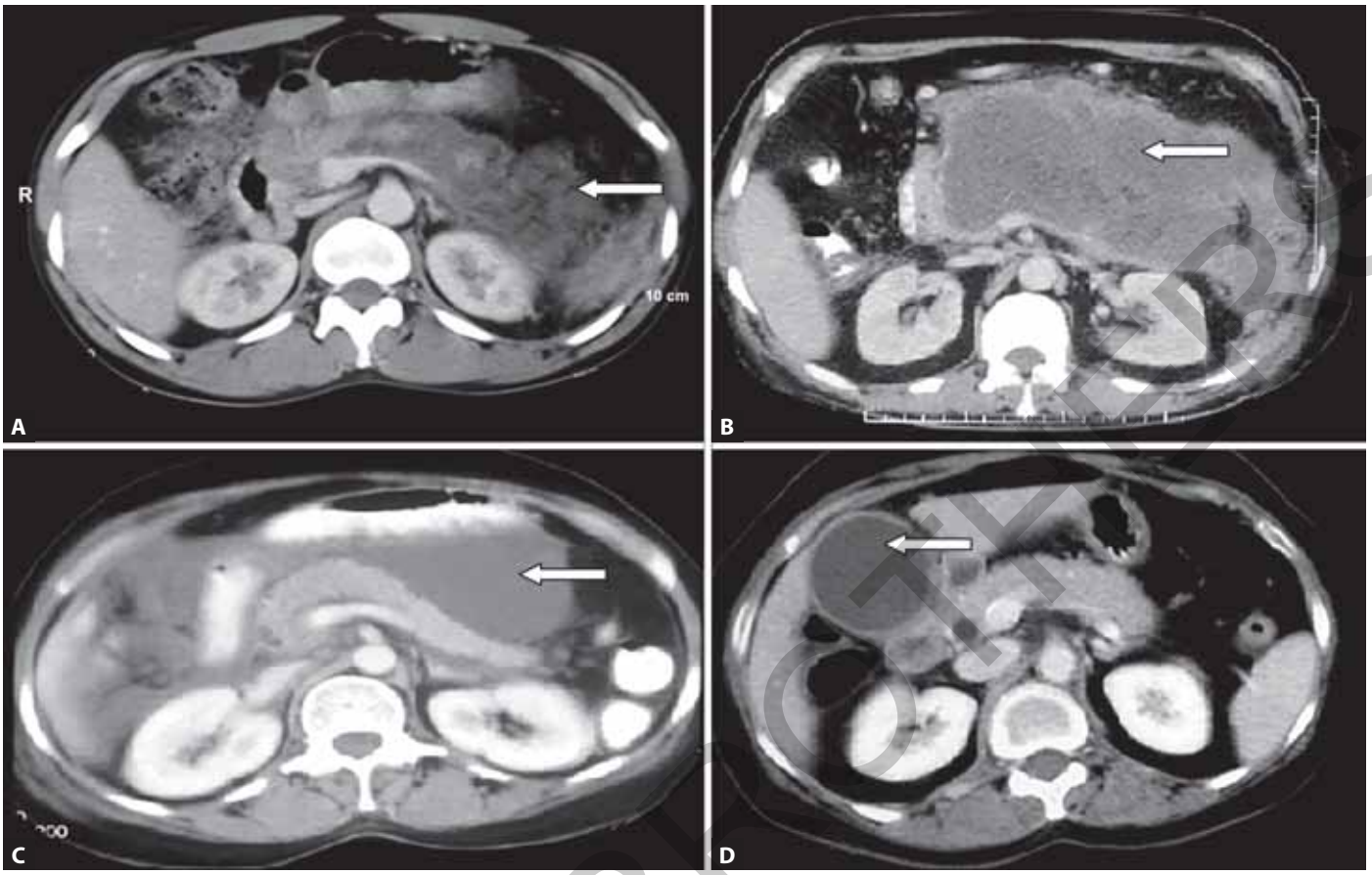
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TABLE 8: Complications of acute pancreatitis.

Local complications	
Acute necrotic collections (ANCs)	• Occur within the first 4 weeks • Intrapancreatic/extrapancreatic • Well-defined wall is absent • May mature into walled-off necrosis in a month • Usually sterile and resolve with conservative treatment
Infected pancreatic necrosis (IPN)	• ANC may convert into infected pancreatic necrosis, peaks about 1–2 weeks after onset of AP • Usually due to bacterial translocation across gut • Commonly due to gram-negative microorganism • Indicated by clinical deterioration, fever, and leukocytosis after initial improvement with worsening sepsis • *For minimal involvement:* Image-guided fine needle aspiration and bacterial culture sensitivity obtained. Antibiotic initiated as per culture report. If negative, procedure repeated in a week • *For abscess:* Percutaneous or endoscopic drainage • Endoscopic technique has lower risk of fistula formation • Stepping up to surgery, if recurrence
Walled-off necrosis (WON)	• Necrotic collection with a well-defined inflammatory wall • Usually occur after first 4 weeks • May be intrapancreatic/extrapancreatic • Noninfected necrosis does not require any intervention • Management includes percutaneous/endoscopic drainage and stepping up to more invasive therapy, if indicated
Hemorrhage	• May be intrapancreatic/extrapancreatic • Occurs due to gastroduodenitis, peptic ulcer, pancreatitis-induced enzymatic damage to adjacent vessels including splenic, renal, or gastroduodenal arteries, or development of an aneurysm in these vessels • Diagnosed by angiography or angio-CT • *Management:* Angioembolization
Pancreatic fistula	• Due to disruption of the pancreatic duct secondary to pancreatic necrosis • Leads to pseudocyst, ascites, pleural effusion, or pancreatocutaneous fistula • *Diagnosis:* ERCP, MRCP, and CT-guided wirsungraphy • *Management:* Conservative treatment with total parenteral nutrition or ERCP-guided placement of a stent. If persistent, require surgery
Peripancreatic complications	
• Pseudocyst	• Refer to leaked peripancreatic collections which may further get infected, perforated or develop hemorrhage. Require image-guided endoscopic percutaneous drainage
• Thrombosis	• Splenic vein, portal, and/or superior mesenteric vein
• Intraperitoneal hemorrhage	• Detailed above
• ACS	• Due to fluid sequestration
• Bowel obstruction	• Surgical intervention
• Pseudoaneurysm	• Detailed above
Systemic complications	
• *Renal:* Acute kidney injury • *Pulmonary:* Pleural effusion, acute respiratory distress syndrome (ARDS) • *Cardiovascular:* Hypotension, pericardial effusion • *Hematologic:* Coagulopathy • *Metabolic:* Hyperglycemia, hypocalcemia • *CNS:* Psychosis, encephalopathy	

(ACS: abdominal compartment syndrome; AP: acute pancreatitis; CNS: central nervous system; CT: computed tomography; ERCP: endoscopic retrograde cholangiopancreatography; MRCP: magnetic resonance cholangiopancreatography)

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Figs. 2A to D: (A) Acute necrotizing pancreatitis; (B) Walled-off necrosis; (C) Acute peripancreatic fluid collection; (D) Pancreatic pseudocyst.

HIGH-YIELD POINTS

- Early diagnosis and adequate resuscitation (targeted fluid therapy and vasopressor use) within the first 48 hours of admission. AP is the key to better outcome.
- Traditional concept of pancreatic rest is no longer validated, and oral feeding should be instituted within 24 hours after hospital admission as tolerated.
- Prophylactic antibiotics should be avoided in patients with AP.
- Current literature suggests that opioids are well tolerated in AP and there is no evidence higher morality or morbidity due to opioids.

SUGGESTED READING

1. Arvanitakis M, Ockenga O, Bezmarevic M, et al. ESPEN guideline on clinical nutrition in acute and chronic pancreatitis. Clin Nutr. 2020;39(3):612-31.
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