

DEFINITION

Acute pancreatitis is defined as an acute inflammation of pancreas along with involvement of other regional tissues or organs. Clinically it is presence of two of the following three features: (1) severe and constant epigastric or left upper quadrant pain with radiation to the back, chest, or flanks; (2) serum lipase activity (or amylase activity) at least three times greater than the upper limit of normal; and (3) characteristic findings of acute pancreatitis on contrast-enhanced computed tomography (CECT) and magnetic resonance imaging (MRI) or transabdominal ultrasonography.

MANAGEMENT OF ACUTE PANCREATITIS

There is a mnemonic for components of management of acute pancreatitis:

P: Perfusion

A: Analgesia

N: Nutrition

C: Clinical Assessment

R: Radiological Assessment

E: ERCP

A: Antibiotics

S: Surgery

Perfusion

Maintenance of adequate perfusion is the most important determinant of outcome in acute pancreatitis in the first 72 h. There occurs frequent hypovolemia in patients with acute pancreatitis because of multiple factors including vomiting, reduced oral intake, third space loss of fluids, increased respiratory losses, and diaphoresis that requires aggressive hydration. A combination of microangiopathic effects and edema of the inflamed pancreas decreases pancreatic blood flow leading to increased cellular death, necrosis, and ongoing release of pancreatic enzymes activating numerous cascades. Early aggressive intravenous fluid resuscitation provides circulatory support which prevents serious complications such as pancreatic necrosis. The preferred fluid for resuscitation is Ringer's lactate. Infusion rates during the first 24 hours in hospital should be sufficient to restore circulating volume and urine output (250 – 500 ml per hour). Early aggressive intravenous hydration is most beneficial during the first 12 – 24 h, and may have little benefit beyond this time period. In a patient with signs of severe volume depletion such as hypotension and tachycardia, more rapid repletion may be needed. Aggressive early hydration will require caution for certain groups of patients, such

as the elderly, or those with a history of cardiac and / or renal disease in order to avoid complications such as volume overload, pulmonary edema, and abdominal compartment syndrome. Response to fluid resuscitation should be assessed by non-invasive response monitoring (heart rate <120 bpm, mean arterial pressure 65-85 mm Hg, urine output 0.5-1 mL/kg/h) along with monitoring of hematocrit and BUN.

Analgesia

Pain is the most troublesome symptom of acute pancreatitis which may lead to impaired respiratory function by restriction of abdominal wall movement. Therefore, adequate analgesia is important during the first few days of clinical presentation. It should be according to the need of the patient. Both opiates and non-opiates can be used in spite of some theoretical risks of exacerbation of pancreatitis by morphine, which can increase pressure in the sphincter of Oddi.

Nutrition

Earlier pancreatic rest (NPO) was considered an important part of management of acute pancreatitis based on a hypothesis that stimulation of pancreatic exocrine secretory function by food may lead to worsening of acute pancreatitis. But experimental studies have shown that Acute Pancreatitis leads to decreased pancreatic exocrine function and does not get stimulated by food. Moreover, bowel rest is associated with intestinal mucosal atrophy and increased infectious complications because of bacterial translocation from the gut. Provision of early enteral nutrition leads to shorter hospital stay, decreased infectious complications, decreased morbidity, and decreased mortality. In mild AP, oral feedings can be started immediately once nausea, vomiting and pain has resolved. In mild AP, initiation of feeding with a low-fat solid diet appears as safe as a clear liquid diet. In severe AP, tube feeding should be started as soon as possible to prevent infectious complications. Parenteral nutrition should be used only if enteral route is not available or not tolerated or doesn't meet caloric requirements. Both nasogastric and nasojejunal tube feeding are equally safe and effective except for increased risk of aspiration in nasogastric feeding.

Clinical Assessment

It is based mainly on assessment of the severity of pancreatitis. According to revised Atlanta Classification, severity of acute pancreatitis can be divided into three groups: mild, moderately severe and severe pancreatitis (Table 1).

Several scoring systems are available to predict which

Table 1: Definitions of severity in acute pancreatitis: comparison of Atlanta and recent Revision

Atlanta Criteria (1993)	Atlanta Revision (2013)
Mild Acute Pancreatitis	Mild Acute Pancreatitis
<ul style="list-style-type: none"> Absence of organ failure* Absence of local complications 	<ul style="list-style-type: none"> Absence of organ failure Absence of local complications
Severe Acute Pancreatitis	Moderately Severe Acute Pancreatitis
<ul style="list-style-type: none"> Local complications and/or Organ Failure 	<ul style="list-style-type: none"> Local complications and/or Transient Organ Failure (<48h)
GI bleeding (>500cc/24hr)	Severe Acute Pancreatitis
Shock –SBP ≤ 90mm Hg	Persistent Organic Failure > 48h
PaO ₂ ≤ 60%	
Creatinine ≥ 2mg/dl	

*Definitions of organ failure: Respiratory: arterial oxygen pressure/fractional inspired oxygen ≥300; Circulatory: systolic blood pressure <90 mm Hg and not fluid responsive; Renal: plasma Creatinine concentration ≥1.9 mg/dl

Table 2: Various Scoring System and their significant values

Type of Scoring system	Score
BISAP Score	≥ 3
Modified Marshall Score	> 2
APACHE II Score	≥ 8
Ranson Criteria	>3
HAPS Score	≥ 2

patients will develop severe disease (Table 2). These include clinical scores such as APACHE II, Ranson criteria, HAPS score, BISAP score, modified Glasgow scores and the CT based score (Balthazar score, Mortelet Score). Out of these, BISAP is easiest to apply and can be used on initial presentation. BISAP relies on the BUN level, impaired mental status, SIRS, age over 60 years and pleural effusions to stratify patients, and has a prognostic accuracy similar to the other scoring systems. While Ranson's score is the oldest and most validated but has limitations that require 48 h for accurate scoring.

There is no definitive consensus as to which scoring system should be used. All have a good negative predictive value but low positive predictive value. However, it is recommended that risk assessment be performed for all patients with pancreatitis to stratify them into higher and lower risk categories. Apart from this scoring system, single parameters like hematocrit > 44%, raised BUN (>20 mg/dl) may also help to predict severe pancreatitis.

Radiological Assessment

It has two roles in management of acute pancreatitis: to diagnose and evaluating local complications. While

Table 3: Local Complications of Acute Pancreatitis

- Pseudocyst
- Sterile necrosis
- Infected necrosis
- Abscess
- Gastrointestinal bleeding
- ✓ Pancreatitis-related
- Splenic artery or splenic artery pseudoaneurysm rupture
- Splenic vein rupture
- Portal vein rupture
- Splenic vein thrombosis leading to gastroesophageal variceal bleeding
- Pseudocyst or abscess hemorrhage
- Postnecrosectomy bleeding
- ✓ Nonpancreatitis-related
- Mallory-Weiss tear
- Alcoholic gastropathy
- Stress-related mucosal gastropathy
- Splenic complications
- Infarction
- Rupture
- Hematoma
- Fistulization to or obstruction of small or large bowel
- Right-sided hydronephrosis

ultrasound is the most common modality used for the diagnosis of pancreatitis but in doubtful cases CECT abdomen may be required for confirming the diagnosis. USG and CECT may also indicate the etiology of pancreatitis like gallstones.

The second major role of radiological investigations is to evaluate local complications. Based on CECT or MRI findings local complications are acute peripancreatic fluid collection, pancreatic pseudocyst, acute necrotic collection and walled-off necrosis (Table 3).

ERCP

ERCP in acute pancreatitis is related to the management of choledocholithiasis. In the absence of cholangitis and / or jaundice, MRCP or EUS rather than diagnostic ERCP should be used to screen for choledocholithiasis if highly suspected.

Antibiotics

Antibiotics should be given for an extrapancreatic infection, such as cholangitis, catheter-acquired infections, bacteraemia, urinary tract infections, and pneumonia. Routine use of prophylactic antibiotics in patients with severe AP is not recommended. Serum Procalcitonin is the best single marker for predicting infection in pancreatitis. In patients with infected necrosis, antibiotics known to penetrate pancreatic necrosis, such as carbapenems, quinolones, and metronidazole, may

308 be useful in delaying or sometimes totally avoiding intervention, thus decreasing morbidity and mortality. Routine administration of antifungal agents along with prophylactic or therapeutic antibiotics is not recommended..

Surgery/Interventions

Step up approach ,in management of acute pancreatitis should be followed as

1. Catheter drainage
2. Minimal invasive necrosectomy
3. Open necrosectomy

PROGNOSIS

Early evaluation and risk stratification for patients with acute pancreatitis are important to differentiate patients with mild versus severe disease because patients with severe disease often need intensive care treatment (Figure 2). The overall mortality in patients with acute pancreatitis is 10-15%. Patients with biliary pancreatitis tend to have a higher mortality than patients with alcoholic pancreatitis. This rate has been falling over the last 2 decades as improvements in supportive care have been initiated. In patients with severe disease (organ failure), who account for about 20% of presentations, mortality is approximately 30%. Approximately 20-25% of patients with acute pancreatitis have a severe form of the disease which usually necessitates high dependency or intensive care management in the first week or two of illness. The measurement of C-reactive protein is also helpful and it has recently been shown that the combining

of the Glasgow scoring system with CRP results in 80% or better sensitivity and specificity for those who develop major clinical complications. The body mass index (BMI) when over 30 kgs/m² is also a useful marker of an adverse outcome, and CT scanning is another method of grading severity. In patients with pancreatic necrosis without organ failure, the mortality approaches zero.

REFERENCES

1. Tenner S, Baillie J, DeWitt J, Swaroop Vege S. American College of Gastroenterology Guideline: Management of Acute Pancreatitis. *Am J Gastroenterol Advance* 2013; 1-15.
2. Banks Peter A, Bollen L Thomas, Dervenis C, Gooszen G, Johnson D Colin, Sarr G Michael et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. *Gut* 2013; 62:102–111.
3. Johnson C D, Besselink M G, Carter R. Acute pancreatitis. *BMJ* 2014; 349:1-8.
4. Petrov MS, Windsor JA. Classification of the severity of acute pancreatitis: how many categories make sense? *Am J Gastroenterol* 2010; 105: 74–6.
5. Santvoort, HCV, Besselink MG, Bakker OJ, Hofker HS, Boermeester MA, Dejong CH et al. A Step-up Approach or Open Necrosectomy for Necrotizing Pancreatitis. *N Engl J Med* 2010; 362:1491-502.
6. Ahuja A, Virk S, Sood A, Sidhu SS, Chhina RS. Acute Necrotising Pancreatitis: Referral Pattern and Surgical Management. *Pancreas* 2008; 36:221-22.
7. Singla SK, Jain NP, Gupta S, Virk SS, Attri PI, Sethi P. Clinical profile and outcome of severe acute pancreatitis. *NJIRM* 2013; 4:71-4.