RESEARCH ARTICLE

# STUDY OF RECENT TRENDS IN ACUTE PANCREATITIS

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**ABSTRACT**

**Background:** Acute pancreatitis is an acute condition presenting with abdominal pain and usually associated with raise pancreatic enzyme level in the blood or urine as a result of inflammatory disease of pancreas. Pancreatitis is a disease of wide clinical variation ranging from mild discomfort alone to a severe illness with hypotension, metabolic derangement, sepsis, multiple organ failure and death. Mortality from acute pancreatitis has decreased from around 12 to 2 percent, according to a large epidemiologic study of acute pancreatitis. However, mortality rates remain much higher in subgroups of patients with severe disease.

**Aims & Objective:** To study recent trends in acute pancreatitis, to study the various etiological factors for development of acute pancreatitis, assessment of various clinical features, complication, investigations and diagnosis of acute pancreatitis and its complications, to evaluate various modalities of the treatment for acute pancreatitis and its complications.

**Material and Methods:** Material of this study consists of 50 patients of acute pancreatitis who were admitted in our Hospital. The clinical presentation clinical examination was carried out, and physical findings, associated medical conditions, laboratory and radiological investigations, severity, management and outcome were studied and the etiology was determined by reviewing the medical records of all cases documented to have acute pancreatitis during the period of study.

**Results:** Average hospital stay for 50 patients in our study is approximately 7 days. The diagnosis of acute pancreatitis was considered when abdominal pain is associated with elevation of serum amylase level. Abdominal pain (100%), fever (82%), vomiting (86%), abdominal distension (40%) and dyspnoea (50%) are its common clinical symptoms.

**Conclusion:** Gallstones and alcohol abuse are the main causes of acute pancreatitis. There is no mortality in our study. Among all the patients, only 4 patients in the follow-up developed complications approximately after 2 months of duration of attack; and they undergone for operation. Clinical examination, serum amylase and ultrasonography are still the diagnostic tool for pancreatitis. Most cases of pancreatitis can be managed conservatively with early diagnosis, aggressive medical management and by treatment of cause.

**Key-Words:** Pancreatitis; Pseudopancreatic Cyst; Pancreatic Debridement

## Introduction

Pancreas consists of network of fine ducts lined by secretory cells. Exocrine cells (99%) which are glandular epithelial cells and secrete pancreatic juice. Endocrine cells (1%) that are pancreatic islets (islets of Langerhans) and secrete hormones. Pancreatic Juice is alkaline (PH 8.4) bicarbonate rich fluid. Cholecystokinin is responsible for enzyme secretion. Pancreatic enzyme includes trypsin, chymotrypsin, amylase, lipase, phospholipase, ribonuclease, deoxyribonuclease etc.[1] Etiology of Acute Pancreatitis are alcoholism, gall stone, ischemia, drug induced, hyperparathyroidism, hypercalcaemia, trauma, ERCP, mechanical Obstruction, pancreas divisum, autoimmune, hereditary, infectious, malnutrition, scorpion bite, hyperlipoprotenemia, pregnancy. If etiology is gallstone, cholecystectomy is essential.[2]

One of the earlier stages noted is- the colocalization of digestive enzyme zymogen such as trypsinogen with lysosomal hydrolases such as cathepsin B inside cytoprasmic vacuoles. Cathepsin B can activate trypsinogen and trypsin can active the other zymogens known as Colocalization hypothesis. According to the colocalization hypothesis, cathepsin enzyme leads to aciner cell injury and triggers an intrapancreatic inflammatory response.[3] Acute hemorrhagic pancreatitis is haemorrhagic necrosis of pancreas. The enzyme elastase produces disruption of blood vessels causing haemorrhage. The pathologic changes represent a spectrum- with interstitial edema and minimal necrosis at one end and confluent macroscopic necrosis at the other extreme.[4]

A commonly used classification system (the Atlanta classification) divided acute pancreatitis into two broad categories: Mild (edematous and interstitial) acute pancreatitis and Severe (usually synonymous with necrotizing) acute pancreatitis. The severity of acute pancreatitis can be predicted based upon clinical, laboratory, and radiologic risk factors, various severity grading systems, and serum markers. Some of these can be performed on admission to assist in triage of patients while others can only be obtained during the first 48 to 72 hours or later.[5] Clinical Features of Acute Pancreatitis are abdominal pain, nausea, vomiting, hiccoughs and abdominal distention.[2] Abdominal pain develops quickly, reaching maximum intensity within minute and persist for

hours or even days. Constant in nature, experience first in epigastrium, radiating to back in 50% of cases. It is refractory to the usual dose of analgesics and constant in nature and intensity. Nausea and vomiting are usually marked accompaniments. Vomiting is often frequent and persistence. Hiccoughs can be troublesome and may due to gastric distention or irritation of diaphragm. Abdominal distention as a result of ileus, bowel sounds are usually diminished during an attack of pancreatitis and the abdomen may become distended and tympanic.[2,5] On general examination patient may be gravely ill with profound shock, toxicity and confusion, tachypnea, tachycardia, hypotension, mild icterus. Icterus may be present due to biliary obstruction or in gall stone pancreatitis**.** Grey-turner's sign (bluish discoloration in flank), Cullen's sign (bluish discoloration in periumbilical region) Direct and rebound abdominal tenderness as well as both, voluntary or involuntary guarding are common. These findings may be localized to the epigastrium or they may be diffusely present throughout abdomen. An epigastric mass, reflecting the inflamed pancreas and surrounding tissue, may be felt in the upper abdomen or left upper quadrant. Complications of acute pancreatitis divided in to systemic (general), local and regional complications. During systemic complication, no need of surgery during initial period of resuscitation and stabilization. Once infected necrosis is suspected and confirmed, an appropriate debridement may be performed. Only other indication for intervention is presence of cholangitis, in which case, an endoscopic sphincterotomy should be performed. Once acute phase has been survived, usually by the end of the first week and major organ failure is under control then local complication become pre-eminent.[5] Acute pancreatitis and related complications are systemic inflammatory response syndrome, compensatory anti-inflammatory response syndrome, and multiple organs dysfunction syndrome. Local complications include acute fluid collection, pancreatic necrosis, acute pseudocyst, pancreatic abscess, paralytic ileus, pancreatic effusion**,** and pancreatic ascites**.** Investigations in acute pancreatitis are s. amylase, urinary amylase, s. lipase, liver function test, diagnostic paracentesis, abdominal x-ray, ultrasonography, Computed Tomography.[2] Hyperamylasemia is seen within 24 hours of onset of attack and returns to normal within 7 days. Serum amylase above 3 times normal is indicative of the pancreatitis. Urinary amylase elevation persists for the longer time with greater magnitude than serum amylase elevation. Serum lipase begins to elevate after 48 hours of attack. Elevation of serum bilirubin, serum alkaline phosphatase and transaminase level may be observed in gall stone pancreatitis and impacted stone at the ampulla. Diagnostic paracentesis may reveals elevation in peritoneal fluid amylase and lipase. Chest X-ray may show pleural effusion. Abdominal X-ray may show "sentinel loop" (dilated jejunal loop in upper abdominal), "colon cut off sign" (transverse colon distention with no air beyond splenic flexure). It may show gall stone. It may show pancreatic calcification. Abdominal ultrasonography show air and fluid filled bowel loops over line and obscuring the pancreas. It may show pancreatic edema, peripancreatic fluid collection, alter echo texture and gall stone. Dynamic computed tomography is the most accurate method for diagnosis acute pancreatitis and its complications like pancreatic necrosis and peripancreatic fluid collection as shown below.

**Table-1:** Computed Tomography Severity Index (CTSI)[6,7]

| Grade | Score |
|---|---|
| **A.** Normal gland | 0 |
| **B.** Focal/diffuse enlargement | 1 |
| **C.** Peripancreatic inflammatory change | 2 |
| **D.** Single pancreatic fluid collection | 3 |
| **E.** Two/more fluid collection | 4 |

| CT grade | Points | Necrosis | Points | CTSI |
|---|---|---|---|---|
| A | 0 | 0 | 0 | 0 |
| B | 1 | None | 0 | 1 |
| C | 2 | <30% | 2 | 4 |
| D | 3 | 30-50% | 4 | 7 |
| E | 4 | >50% | 6 | 10 |

| Score | Morbidity (%) | Mortality (%) |
|---|---|---|
| 0-3 | 8 | 3 |
| 4-6 | 35 | 6 |
| 7-10 | 92 | 17 |

Endoscopic retrograde cholangiography is useful in identifying abnormality such as ampullary stenosis, focal pancreatic ductal abnormality and pancreas divisum. ERCP should not be performed within 4 weeks of an episode of acute pancreatitis or in the presence of pseudocyst. On account of difference between patient with mild and severe disease, it is important to define that group of patient who will develop severe pancreatitis. For prognosis of acute pancreatitis various scoring system have been introduced such as Ranson and Glasgow scoring system. A score of 9 or more indicates a severe attack. Score of 6 or more include 95% of all those who developed complications.[5]

Scoring system is to predict the severity of acute pancreatitis. Ranson and Glasgow score both the systems, is classified as severe when 3 or more factors present and are helpful in prognostic assessment of acute pancreatitis.[2] C-reactive protein level > 210 mg/l in the first 4 days of the attack or 120 mg/l at the end of first week has a predictive performance. C-reactive protein (CRP) assays are readily available, and levels rise with disease severity.[8] Similarly, The Acute Physiology and

Chronic Health Evaluation II (APACHE II) system is as accurate at 24 hours as other systems at 48 hours, and it is now therefore regarded as perhaps the optimal scoring system to access disease severity in pancreatitis.[9] Twelve physiologic variables are measured and weighed based on their degree of abnormality: temperature, mean arterial pressure, heart rate, respiratory rate, arterial oxygen tension, arterial pH, serum sodium, serum potassium, serum creatinine, haematocrit, white blood cell count and Glasgow coma scale. Further points are added for increased age and chronic organ dysfunction. The newer APACHE II system uses an additional five physiologic variables to improve accuracy, although the newer system may be less useful than the APACHE II score in distinguishing mild from severe pancreatitis.[5] A recent modification of the APACHE II system that includes a clinical assessment of obesity (APACHE-O score) has been suggested to further improve predictive accuracy, with a positive predictive value of 74%.[10] Numerous individual markers have been investigated as possible indicators of prognosis in pancreatitis in both laboratory and clinical settings. Haemoconcentration predicts parenchymal necrosis, as well as the presence of organ failure, in acute pancreatitis.[11,12] Other inflammatory markers, including TNF soluble receptors, polymorphonuclear (PMN) elastase, serum procalcitonin, soluble IL-2 receptors, and soluble E-selection, have shown potential in the investigation setting.[13] Trypsinogen-activating peptide (TAP) is an additional marker that may be useful in determining prognosis in acute pancreatitis. TAP is released, with activation of trypsinogen to trypsin, and plasma and urine levels are known to correlate with severity of pancreatitis.[14,15] Eary CT scans often fail to identify developing necrosis until such areas are better demarcated, which may become evident only 4 days after the initial clinical onset of symptoms.[9] It is not possible to differentiate infected from sterile pancreatic necrosis based only on clinical and laboratory data because organ failure, significant leucocytosis, and fever are seen in both the cases. Emphysematous pancreatitis, the demonstration of gas within the pancreatic parenchyma, is diagnostic of infection. Using image-guided precise aspiration of the necrotic pancreas, infected pancreatic necrosis can be diagnosed with a high degree of accuracy. Pancreas is aspirated under CT guidance and samples are sent for aerobic, anaerobic, and fungal cultures. Repeated CT guided aspiration therefore is necessary frequently in the patients in whom a conservative strategy is adopted for sterile pancreatic necrosis until clinical improvement is documented.[16]

Initial Management For Acute Pancreatitis is nil per oral, aggressive fluid resuscitation, NGT for ileus, enteral feeding when tolerated, ERCP for biliary obstruction, TPN if enteral feeding not tolerated, CT scan (after 72h) if there is deterioration. To inhibition of pancreatic secretion anticholinergics, glucagon, 5-fluorouracil, acetazolamide, cimetidine, propylthiouracil, calcitonin, somatostatin can be given. CT-guided FNA can be done if clinical deterioration (WBC, hypotension, organ failure, fever). Operative measures in the form of biliary procedures, pancreatic drainage, pancreatic resection, pancreatic debridement, drainage of pancreatic infection can be done as and when required.[5]

## Materials and Methods

All were indoor patients with diagnosis of various breast diseases. Each patient was study in detail with relevant clinical history, examination, laboratory investigations and management. The study comprised of total 50 patients treated by various modalities. The selection criterions for the patient were based on (1) complain of abdominal pain; (2) on examination- abdominal tenderness; (3) Ultrasonography finding; and (4) serum amylase more than threefold rise than normal. Ultrasonography is a very safe investigation which can be repeated. CT scan used in cases when there is clinical deterioration. The selected patients had been treated thereafter in form of different modalities like, (a) simple analgesic; (b) fluid resuscitation; and (c) surgical treatment as and when required.

## Results

Our study is being compared with “Acute Pancreatitis in multi-ethnic population “, publised by P Kandasami et al.[17] Minimum age in our study is 21 years and maximum age is 73 years having mean age 40.3 years. In case study mean age is of 43.5 years. Maximum number of patients (32) is below 45 years of age (Table 2). In selected case study, acute pancreatitis occurs in 26.32 % in Male patients of Malay and 73.68% in Female patients of Malay. Same ratio in Chinese population is 42.11% in male and 57.89% in female, while in India; ratio is 77.33% in male and 22.67% in female. In our study, 70% male patients developed acute pancreatitis and 30% of females, which correlates with Indian ratio of multi-ethnic study (Table 3). Abdominal pain is present in all 50 (100%) cases and vomiting in 43 (86%) which is comparable with Maingot’s Abdominal Operations – “Acute Pancreatitis”, in which it is stated that abdominal pain was present in 85-100% and vomiting in 92% of their patients. Abdominal tenderness is present in all 50 cases and bowel sound is absent in 1 case. Guarding / rigidity are present in 30 patients. So, these two

**Table-2:** Age Distribution

| Age | No of Cases | In Our Study (%) | In Case Study (%) |
|---|---|---|---|
| <45 | 32 | 64% | 68% |
| 45-54 | 10 | 20% | 22% |
| 55-64 | 2 | 4% | 7.4% |
| 65-74 | 6 | 12% | 2.1% |
| >74 | 0 | 0% | 0.35% |

**Table-3:** Gender Distribution

| Gender | In Our Study | In Case Study |
|---|---|---|
| Male | 35 (70%) | 76 (57.58%) |
| Female | 15 (30%) | 56 (42.42%) |

**Table-4:** Signs of Acute Pancreatitis

| Signs | No. of Cases |
|---|---|
| Abdominal tenderness | 50 (100%) |
| Guarding/Rigidity | 30 (60%) |
| Ascites | 10 (20%) |
| Absent bowel sound | 1 (2%) |
| Jaundice | 7 (14%) |

**Table-5:** Serum Amylase in Acute Pancreatitis

| Level | No. of Cases |
|---|---|
| <130 (normal) | 1 (2%) |
| 130-260 | 2 (4%) |
| 261-390 | 3 (6%) |
| >390 | 44 (88%) |

**Table-6:** Serum Lipase in Acute Pancreatitis

| Level | No. of Cases |
|---|---|
| <60 (normal) | 0 |
| 60-200 | 3 (6%) |
| 201-340 | 5 (10%) |
| >340 | 42 (84%) |

**Table-7:** Serum Bilirubin in Acute Pancreatitis

| Level | No. of Cases |
|---|---|
| Normal | 43 (86%) |
| Raised | 7 (14%) |

**Table-8:** Abdominal Ultrasound (USG) in Acute Pancreatitis

| USG Findings | No. of Cases |
|---|---|
| Partially Obscured Pancreas | 5 (10%) |
| Oedematous Pancreas | 30 (60%) |
| Peripancreatic Fluid Collections | 13 (26%) |
| Gall Bladder Stone | 15 (30%) |
| Ascites | 6 (12%) |
| Pancreatic Duct Calculus | 1 (2%) |
| Pseudocysts | 5 (10%) |
| Alter echo texture | 19 (20%) |

**Table-9:** Etiological Factors in Acute Pancreatitis

| Etiology | No. of Cases | | Total Percentage |
|---|---|---|---|
| | Male | Female | |
| Biliary disease (Gall Bladder) | 5 | 8 | 26 |
| Alcoholism | 28 | 0 | 56 |
| Idiopathic | 3 | 2 | 10 |
| Others (post ERCP, Main Pancreatic Duct Calculi) | 1 | 3 | 8 |

**Table-10:** Etiological Factors in Other Studies of Acute Pancreatitis

| Etiology | Malay | Chinese | Indian | Total |
|---|---|---|---|---|
| **Gallstones** | 10 (26.3%) | 6 (31.6%) | 5 (6.7%) | 21 |
| **Alcoholism** | 3 (7.9%) | 5 (26.3%) | 55 (73.3%) | 63 |
| **Idiopathic** | 23 (60.5%) | 7 (36.8%) | 6 (8%) | 35 |
| **Others** | 2 (5.3%) | 1 (5.3%) | 9 (12%) | 13 |
| **Total** | 38 (100%) | 19 (100%) | 75 (100%) | 132 |

**Table-11:** Complications and their Management in Acute Pancreatitis

| Complications | No. of Cases | Management |
|---|---|---|
| Severe Necrotising Pancreatitis | 1 | Conservative |
| Pseudocysts | 4 | Drainage of cyst |
| Acute fluid collection | 13 | Conservative |

are the most common signs in our study. 7 patients show jaundice as a sign of acute pancreatitis. Ascites (10 patients) may present as a sign of acute pancreatitis (Table 4).

In our study, diagnosis of pancreatitis is made by the clinical presentation or other modalities like USG. 29 (96.67%) patients show hyperamylasemia, while only 1 patient (2%) of our cases had normoamylasemia, who had positive urinary amylase (Table 5). Normal serum lipase value is 60 Units/L. In present study, 50 (100%) patients show raised serum lipase level. Among them, 42 (84%) patients have three fold than the normal value (Table 6). Out of 7 jaundice patients, 6 had acute pancreatitis due to alcoholism and only 1 patient due to GB calculi. Most of the cases of hyperbilirubinemia were those associated with alcoholism (Table 7). Ultrasonography of Abdomen is done in all 50 cases. Maximum number of the patients (60%) showing pancreatic oedema as a characteristic of acute pancreatitis (Table 8).

In our study, alcohol was identified as the most important etiologic factor associated with acute pancreatitis, accounting for 56% of the cases, more than half of the patients. This also correlates with results of India in selected case study. Also the incidence of alcohol association with acute pancreatitis has significantly increased in the male, while gallstone pancreatitis is predominantly a disease of the female. Occurrence of percentage of biliary pancreatitis in our study is 26%, correlated with that of Malay study (26.3%). 10% of patients developed acute pancreatitis due to Idiopathic etiological factor, which is matched again with results of India (8%) (Table 9 & 10). Complications were occurred in 18 patients. The most common complication that developed after an acute attack of Pancreatitis was an acute fluid collection (13 patients – 26%), four patients developed pancreatic pseudocyst, two patients underwent Cysto-Gastrostomy, and one patient underwent external drainage of pseudo pancreatic cyst (Table 11).

## Discussion

There is a male preponderance (35 out of 50 cases) in our study which coincides with most standard series. It can be concluded that males are affected more often by acute pancreatitis than females. According to our study, Normal value of serum amylase in our laboratory is up to 130 Units/L. Any rise above normal values combined with positive clinical features and radiologic data was taken as a proved case of acute pancreatitis. A consecutive series of 352 patients with acute pancreatitis admitted to Clinic of Digestive Surgery, Geneva University Hospital, revealed 67

patients (19.03%) as normoamylasemic. Thus, hyperamylasemia is a highly sensitive but not specific biochemical indicator of the disease. Values three times above normal are almost specific. In our study, 44 (88%) patients have serum amylase level more than three times the normal value. Serum lipase is only secreted by the pancreas and thus better specificity and sensitivity. In acute pancreatitis, serum lipase level may be elevated more consistently and for longer half-life than serum amylase. USG is the cost effective and reliable investigation for diagnosis of acute pancreatitis as well as its complications. Computed Tomography revealed all the features of Acute Pancreatitis and also, complications like necrotizing pancreatitis. Its greatest advantage is its utility when Retroperitoneum cannot be visualized on Ultrasonography due to bowel gas. Historically, gallstones were recognized to be the predominant factor responsible for acute pancreatitis, representing 40 to 60 per cent of cases. However, more recent reports suggest alcohol as the most common etiological factor. Acute pancreatitis is protean disease, capable of wide clinical variation ranging from a mild, self-limiting disease to a severe disease with devastating consequences. Studies on acute pancreatitis based on the Western population disease reveals that severe disease is characterized by organ failure, 20% to 30% of all patients will have a severe clinical course of the complications such as necrosis, pseudocyst or abscess and 95% of deaths will occur in this subset. In this study, a significant number of patients (25.0%) developed organ dysfunction or local complications. In our study, 36% of patients developed local complications. The characteristics of acute pancreatitis in the patients studied were not different when compared to the West with regards to the severity of the disease and the nature of complications.[17]

## Conclusion

Acute pancreatitis (AP) is a serious disease with a frustrating mortality rate, but with a very good quality of life reported among survivors justifies an optimized allocation of 'therapy intensity'. Incidences of AP are increasing nowadays. Severity of pancreatitis is determined early on in the disease process most cases of pancreatitis have a self-limiting course. There is a slight male preponderance, with maximum age incidences below 45 years. Alcoholism ranks first as the etiological factor (56%) followed by biliary tract disease (26%). Among the males, alcoholism is the most common etiological factor, because addiction of it is more common in males, while biliary tract disease is the most common factor in females, because gall bladder stone is more common in fatty, fertile females of forty. Serum amylase is the best biochemical indicator of the disease, supplemented by serial ultrasound examinations of the abdomen. Computed Tomography is not used routinely due to its high cost. The prognostic factors for this disease are Hyperamylasemia, Hyperglycaemia and Hypocalcaemia, reflecting a poorer outlook. Prognosis can also be assessed by Ultrasonography and Computed Tomography, delineating local complications. Peripancreatic fluid collection and necrotizing pancreatitis is predictor of severity of pancreatitis on USG and CT scan finding. So, serum amylase levels more than threefold of normal level and peripancreatic fluid collection are main predictor of severity of pancreatitis in our study. The initial management for an acute pancreatitis attack should be conservative; with surgery reserved for cases having uncertainty of diagnosis, trauma, very severe attacks not responding to medical therapy and complications of the disease. The severity of acute pancreatitis is variable. The ability to predict the severity can help identify patients at increased risk for morbidity and mortality, thereby assisting in appropriate triage and selection of patients for specific interventions. This topic review will summarize methods for predicting the severity of acute pancreatitis. This study of Acute Pancreatitis is an effort to recount and record each battle which the surgeon fought to treat the illness.

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