A RETROSPECTIVE STUDY OF MANAGEMENT OF ACUTE PANCREATITIS IN A PERIPHERAL TERTIARY HOSPITAL

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ABSTRACT

BACKGROUND & OBJECTIVES

Acute pancreatitis (AP) is one of the most common diseases in gastroenterology. Two percent of all patients admitted to hospital are diagnosed with AP. During the last decade, an increasing incidence was observed, mostly because of a higher sensitivity of diagnostic tests. Treatment of Acute Pancreatitis is still symptomatic and no specific medication is available today. As a result of popular belief that the pancreas should be put to rest during acute pancreatitis, the parenteral route for nutrition is still predominantly used in Acute Pancreatitis. There has been increasing evidence; however, about gut being main source of microorganisms causing infectious pancreatic complications and multiorgan failure. In patients with severe pancreatitis, oral intake is inhibited by nausea and subileus. Although some reports show that enteral feeding is possible in acute pancreatitis and associated with fewer septic complications. Although the evidence is inconclusive to support enteral nutrition in all patients with severe acute pancreatitis, the enteral route may be used if tolerated. Supportive treatment is the most important line of management in acute pancreatitis. The aim is to study the management of acute pancreatitis in a peripheral tertiary hospital and to assess the outcome of the management.

METHODS & MATERIALS

Data Collection: Patients with acute abdominal pain are admitted in hospital and diagnosed as acute pancreatitis based on blood investigations and radiological findings. Patients categorised- Revised Atlanta Classification. Different medical management modes followed and outcomes recorded, tabulated and analysed.

Research Design: Retrospective study.

Research Settings: Mahatma Gandhi Memorial Government Hospital, Trichy, Tamilnadu.

Duration: 5 yrs. (2010-2015)

Sample Size: 186.

Inclusion Criteria: Patients between 12 and 75 yrs. of age, patients admitted to the hospital as a case of acute pancreatitis, both sexes, patients willing to participate in the study.

Exclusion Criteria: Paediatric age group, >75 yrs. Age, patients not willing to participate, patients absconded without completing treatment.

RESULTS

Chronic alcoholism is the most common cause of acute pancreatitis. Male preponderance is more. Usually in the 4th and 5th decades. Most patients presented with abdominal pain. CT findings were diagnostic and gold standard. All underwent conservative management of which 7 cases succumbed to the disease and the rest were treated.

KEYWORDS

AP (Acute Pancreatitis), SIRS (Systemic Inflammatory Response Syndrome), CT (Computerised Tomography), Injection Octreotide, IV Fluids.

INTRODUCTION: There have been important changes in the definitions and classification of AP since the Atlanta Classification from 1992.

Revised Atlanta Classification 2012: Two distinct phases of AP have now been identified:

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1. Early (Within 1 week), characterised by the systemic inflammatory response syndrome (SIRS) and/or organ failure.
2. Late (> 1 week), characterised by local complications.

It is critical to recognise the paramount importance of organ failure in determining disease severity. Local complications are defined as peripancreatic fluid collections, pancreatic and peripancreatic necrosis (sterile or infected), pseudocysts, and walled-off necrosis (sterile or infected). Isolated extrapancreatic necrosis is also included under the term necrotising pancreatitis. Severity is classified as mild, moderate or severe. Severe acute pancreatitis is defined by persistent organ failure, that is, organ failure >48 hrs.

Based on the revised Atlanta Classification of acute pancreatitis, acute pancreatitis is clinically defined by at least the first two of three features: (a) Epigastric Pain Often Radiating to the Back; (b) Serum Amylase and Lipase Levels Three or More Times Normal; and (c) Characteristic Findings on CT, Magnetic Resonance (MR) Imaging, or Transabdominal Ultrasonographic (Us) Studies. APACHE II scoring.[1] Ranson's criteria.[2] Revised Atlanta classification and Balthazar CTSI.[3] are the various scores available of which Revised Atlanta Classification and Balthazar CT scoring.[4],[5],[6] was used in this study.

Contrast enhanced CT is the gold standard for diagnosis of pancreatic necrosis. The presence of more than 50% pancreatic necrosis on CT is prediction of severe disease. The diagnosis of pancreatic necrosis on CT is prediction of severe disease. The diagnosis of pancreatic necrosis does not predict development of organ failure. Hence several scoring systems for assessment of severity of acute pancreatitis exist. These multiple factor scoring systems help to assess risk of complication in patients with AP and to categorise patients into groups at high risk of complications.[7]

Ranson's Criteria:[2]
On Admission:
- Age: >55 years.
- White Blood Count: >16000/mm.
- Blood Glucose Level: >11.0 mmol/L.
- Lactate Dehydrogenase (LDH): >350 IU/L.
- Aspartate Aminotransferase (AST): >250 U/L.
- At 48 hours.
- Packed Cell Volume: Decrease >10% from Admission.
- Blood Urea Nitrogen (BUN): Increase >1.8 mmol/L from Admission.
- Calcium: <2 mmol/L.
- Oxygen Partial Pressure: <60 mmHg.
- Base Deficit: >4 mmol/L.
- Fluid Sequestration: >6 L.

Prognosis:
- 0-2 Criteria: <1% Mortality.
- 3-4 Criteria: 16% Mortality.
- 5 or More: >40% Mortality.

Grade	CT feature
A	Normal pancreas consistent with mild pancreatitis
B	Focal or diffuse enlargement of the gland but without peripancreatic inflammation
C	B + peripancreatic inflammation
D	C + associated single fluid collection
E	C + two or more peripancreatic fluid collections or gas in the pancreas or retroperitoneum
Table 1.1: Balthazar CTSI (CT Severity Index) Scoring[3]	

When pancreatic necrosis has developed, differentiating between sterile and infective necrosis is essential for management of patients. Infection is suspected in patients who develop signs of sepsis. These patients should undergo FNA to differentiate between them. FNA is an accurate, safe and reliable approach. Only patients with clinical signs of sepsis should undergo FNA because FNA bears potential risk of secondary infection.[7] Various modes of management are available for acute pancreatitis. But the most commonly employed is the medical management.[8],[9],[10],[11],[7],[12],[13],[14] Initially nil oral, IV fluid supplementation and inj. Octreotide, followed by liquid diet once symptoms reduce.[15],[16],[17],[18] and slowly bringing the patient back to normal diet with analogous correction of effects of pancreatitis such as diabetes mellitus (DM) and hypocalcaemia.[10],[11],[12],[14],[15]

RESULTS AND OBSERVATIONS: The most common cause of acute pancreatitis in our study was alcoholism accounting to 84% of all causes found.[13]

Causes	No.	Percentage
Chronic Alcoholic	156	84
Gallstones	11	6
Post ERCP	4	2
Blunt Trauma	4	2
Not Known	11	6
Table 1.2: Causes of Acute Pancreatitis[8],[9],[10],[13]		

Almost all patients presented with acute abdominal pain with stable vitals. 19 cases presented with severe acute pancreatitis with stable vitals. 7 patients came with shock due to? necrotising pancreatitis.

Presenting Symptom	No.	Percentage
Abdominal pain	160	86
Severe acute pancreatitis (Stable vitals)	19	10
Shock (?nec. pancreatitis)	7	4
Table 1.3: Presenting Symptoms[9],[10],[11]		

Patients came with various signs and symptoms such as abdomen pain, fever, vomit, abdominal distension, abdomen guarding/rigidity of which abdominal pain was prevalent in 100% of all patients.

Signs & Symptoms	No.	Percentage
Fever	89	48
Abdominal pain	186	100
Vomit	97	52
Abdominal distension	11	6
Abd. guarding & rigidity	26	14
Table 1.4: Signs & Symptoms		

Enlargement of gland without peripancreatic inflammation is the most common CT finding.[3]

CT Finding	No.	Percentage
Grade A	38	20
Grade B	74	40
Grade C	48	26
Grade D	19	10
Grade E	7	4
Table 1.5: CT Findings[3]		

All 186 cases were treated with conservative management of which 7 cases succumbed. These 7 cases presented with shock and treated conservatively with SICU care. Of the 179 cases that were treated, hospital stay was categorized as follows.

Hospital stay	No.	Percentage
<7 days	42	23
7-10 days	118	66
>10 days	19	11
Table 1.6: Hospital Stay		

DISCUSSION: The clinical course of Acute Pancreatitis varies from a mild transitory form to a severe necrotising disease. Most episodes of acute pancreatitis (80%) are mild and self-limiting, subsiding spontaneously within 3 to 5 days. Patients with mild pancreatitis responds well to medical treatment and generally do not need ICU treatment or pancreatic surgery. In contrast severe pancreatitis is associated with organ failure or local complications, such as necrosis, abscess formation or pseudocysts or both. Severe pancreatitis may be observed in 15-20% of all cases.[7]

The commonest cause of Acute pancreatitis in this series is Chronic alcoholism/Alcohol abuse.[8],[9],[10], Second most common cause being gallstones. The mildest pathological change observed in the pancreas during acute pancreatitis consists of oedema of the gland. This may be accompanied by infiltration of the intralobular septa by inflammatory cells. Microscopic examination also may show areas of fat necrosis in the pancreas and surrounding tissues. If such necrosis is more extensive, it becomes grossly recognizable as characteristic whitish yellow plaques. Finally, vascular thrombosis or disruption may result in pancreatic necrosis or gross haemorrhagic infarction. Increased levels of active pancreatic enzymes have been observed within the pancreas in the peritoneal exudate and in the bloodstream of patients with pancreatitis and usually are implicated in the multiple systemic and local complications of the disease.[11] Hypovolaemia occurs owing to losses from intravascular space – both systemic loss and into retroperitoneal space.

Also from vomiting and nasogastric suction. Hypocalcaemia occurs as a reflection of hypoalbuminaemia and to binding of calcium in areas of fat necrosis. Due to hypovolemia, hypotension, tachycardia, increased total peripheral resistance, decreased cardiac output and renal failure occur. Abnormal liver function, elevation of S. bilirubin, alkaline phosphatase and transaminase levels have been attributed to biliary obstruction, hepatic parenchymal necrosis and pericholangitis.[11]

Abdominal pain is the most common presenting symptom. In the series, 7 cases presented with shock features and deteriorated despite intensive management and could not be revived. Male to female ratio is 9:1. Higher preponderance in the male gender. S. amylase/S. lipase values have a high sensitivity to disease severity.[9],[10] CT abdomen is the gold standard for diagnosis of acute pancreatitis and gives a clear picture of all stages and severity of the disease. Enlargement of gland without peripancreatic inflammation is the most common CT finding.[3] in the series.

Management of acute pancreatitis is still symptomatic and no specific medication is available today.[7] Supportive treatment is the main modality of treatment. The most important supportive treatment is adequate and prompt fluid resuscitation with IV fluids and supplemental O_2 or controlled ventilation to guarantee optimal oxygenation of organs. Cardiac ionotropic drugs, haemofiltration or dialysis also may be needed to allow optimal fluid therapy despite acute renal failure or hypoperfusion. Secondary causes of organ failure, such as hypovolaemia, tissue hypoperfusion and hypoxaemia, also must be identified and treated promptly. There is some evidence that vigorous fluid resuscitation may be associated with resolution of organ failure. Because plasma expanders are more effective and long acting, colloids should be preferred compared to crystalloids.[7]

Prophylactic antibiotic treatment is the only treatment in acute necrotising pancreatitis in which significant benefit has been shown by several meta-analyses which represent the highest level of evidence available. Imipenem is antibiotic agent of 1st choice because it reached higher pancreatic tissue levels and higher bactericidal activity against most bacteria present in pancreatic infections. The advantage was limited; however, to patients with severe pancreatitis who received broad spectrum antibiotics that achieved therapeutic pancreatic tissue levels. There was strong evidence that IV antibiotic prophylactic therapy for 10 days to 14 days decreased the risk of superinfection of necrotic tissue and mortality in patients with severe acute pancreatitis with proven pancreatic necrosis at CT.[7]

Indications for Surgical Treatment In Acute Pancreatitis.[7]

- Infected Pancreatic necrosis.
- Sterile pancreatic necrosis (Selected cases).
- Persistent necrotising pancreatitis.
- Fulminant acute pancreatitis.

- Complications of acute pancreatitis (Bowel perforation, bleeding).

Four principle methods of surgical treatment have been advocated.[7]
1. Necrosectomy Combined with Open Packing.
2. Planned, Staged Re-laparotomies with Repeated Lavage.
3. Closed Continuous Lavage of Lesser Sac and Retroperitoneum.
4. Closed Packing.

Various complications of acute pancreatitis are pancreatic fluid collections, pseudocyst, abscess, pancreatic fistulae, biliary leakage and haemorrhages. According to studies, approximately 80% of patients recover without life threatening complications. Mortality has fallen to 5 to 10%. Most deaths are related to septic complications of pancreatic and peripancreatic necrosis.[11] All 186 cases in the study, were treated by conservative measures of which 179 (96.2%) cases revived and were symptom-free and were discharged. 7 cases (3.8%) succumbed to it because of the severity and late presentation of the disease in stage of shock. None of the cases in the study required surgery. Cases with necrotic changes were started on prophylactic antibiotic cover. Most cases were administered IV Meropenem as drug of choice as Imipenem is not available in our setup.

CONCLUSIONS: Chronic alcoholism is the most common cause of acute pancreatitis. Male preponderance is more. Usually in the 4th and 5th decades. Most patients presented with abdominal pain. CT findings were diagnostic and gold standard. All underwent conservative management of which 7 cases succumbed to the disease and the rest were treated.

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