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Study of clinical profile in acute pancreatitis and its management

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Abstract
More than a century after its comprehensive description, acute pancreatitis remains a common disorder with devastating consequences. Although most episodes are mild and self-limiting, up to a fifth of patients develop a severe attack that can be fatal. The presentation of wide spectrum of symptoms gives the clinician a heart-breaking exercise to bring back the patient from the clutches of the disease process. Pancreatitis by itself is a disease, which is unique, protean and extrudes into the diagnostic arena. It cannot be too strongly emphasized that the primary treatment of acute pancreatitis is conservative only, but it is the pandora's box of manifestations, with its inherent complication's surgery comes into play as diagnostic, prognostic and therapeutic endeavors. Because of the frequent emergency, multimodality presentation, difficult preoperative diagnosis and management of complications, this challenging subject is taken up for the present study in which we will be studying the clinical profile and management of acute pancreatitis in our hospital. Inspite of technical advances in medical and surgical fields acute pancreatitis remains a major cause of morbidity and mortality.
Acute pancreatitis is a common and challenging disease that can develop both local and systemic complications. Its hallmark is acute pancreatic inflammation associated with little or no fibrosis. It ranges from a mild self-limiting inflammation of the pancreas to critical disease characterized by infected pancreatic necrosis, multiple organ failure and a high risk of mortality. The clinical outcome has improved over recent decades, even in the absence of specific treatments that target outcome-determining pathophysiology, probably because of a more consistent approach to diagnosis, monitoring and management.
This Present study was conducted. at Bowring and lady Curzon and Victoria Hospital, Bangalore, a tertiary care centre with round the clock radiology and endoscopy services. All endoscopies are done by Gastroenterologists. Surgeons with expertise in pancreatico biliary diseases are available. Here acute pancreatitis was found to be two and a half times more common in males than females.

Keywords: acute pancreatitis, multi scoring systems, single prognostic factors, pseudo cyst, necrosis, cystogastrostomy, multi organ failure, mortality

Introduction
Acute pancreatitis is the most common gastrointestinal discharge diagnosis in the United States (274,119 patients in 2009), an incidence which has increased 30% since 2000, and is associated with the highest aggregate inpatient costs at 2.6 billion dollars per year. The crude mortality rate of 1.0/100,000 ranks it as the 14th most fatal illness overall and the ninth most common noncancer gastrointestinal death. Worldwide the incidence of acute pancreatitis ranges from 5 to 80/100,000 population with the highest incidence recorded in Finland and United States [2]. The racial incidence of acute pancreatitis also shows significant variation related to the prevalence of etiological factors and ethnicity. The annual incidence of acute pancreatitis in Native Americans is 4 per 100,000 population; in whites it is 5.7 per 100,000 population; and in blacks it is 20.7 per 100,000 population [3].
Smoking is an independent risk factor for acute pancreatitis [4]. Acute pancreatitis resulting from unregulated activation of pancreatic enzymes which can lead to extra pancreatic complications due to persistence of hypovolemia, a decreased intravascular volume and multi organ dysfunction [5]. In spite of technical advances in medical and surgical fields acute pancreatitis remains a major cause of morbidity and mortality.
Acute pancreatitis is defined as an acute inflammatory process of the pancreas, with variable involvement of other regional tissues or remote organ systems [6]. It may occur as an isolated attack or recur in distinct episodes with reversion of normal histology between attacks. By definition, acute pancreatitis is reversible. It is distinguished from chronic pancreatitis by the absence of continuing inflation irreversible structural changes and permanent impairment of exocrine and endocrine function [7].

Aim and objectives of the study
1. To study the incidence of complications developing in patients diagnosed as acute pancreatitis.
2. To study nature of complications due to acute pancreatitis.
3. To evaluate patients who need surgical intervention.
4. To assess the morbidity and mortality.

The existing situation
Acute pancreatitis is one of the common cause of hospital admissions presenting with pain abdomen. Acute

pancreatitis is a condition that has a varied presentation, etiology, obscure pathogenesis and varied clinical outcome from mild self-limiting episode to severe life threatening multiorgan failure. The pathological spectrum varies from edematous pancreatitis which, is uncomplicated and self-limiting to necrotizing pancreatitis in which degree of pancreatic necrosis correlate the severity of attack and systemic complication which involve renal, lung, GIT, brain and may lead to multi system organ failure. Despite decades of research and clinical trials, treatment remains essentially supportive. Improved outcomes are clearly linked to advancements in supportive care. This study evaluates the prognosis of acute pancreatitis with conservative treatment, how much percentage of patients are ultimately required surgery on follow up, apart from the management of acute pancreatitis. This might help in evaluating what type of patients might need surgical intervention.

Review of literature

The earliest description of pancreas dates back to 300 BC, given by Herophilus of Chalcedon. During 100AD Rufus of Ephesus thought that pancreas acts as a cushion for stomach and named it as "PANCREAS" meaning "all flesh" because the organ contains neither cartilage nor bone [8]. In 1642 Johann Wirsung described main pancreatic duct and in 1734 G B Santorini described accessory pancreatic duct which go by their names [9]. Operative intervention on pancreas, which was first attempted by Le Dentu in1862. In 1901 Eugene Opie, a pathologist at John Hopkins hospital in Baltimore, documented a gallstone impacted in ampulla of Vater during the postmortem examination of a patient, (operated on by Halsted) who had died of gallstone pancreatitis and there by described the pathogenic mechanism of gallstone pancreatitis [10]. The importance of pancreas and severity of its inflammatory disease were only recognized in 1925 when Berkeley George Andrew Moynihan (lord Moynihan of Leeds) Professor of clinical surgery, Leeds, England, descried Acute Pancreatitis [11]. In 1929 Elman. R, described the association between elevated Serum Amylase levels and Acute Pancreatitis. Watts in 1963 reported survival of a patient who was treated by total pancreatectomy for acute pancreatitis [12]. The prognostication of Acute Pancreatitis [13] was for first time in 1974 by John H C Ranson when he was at New York university medical centre, New York. He was born in Bangalore, India (1938). In 1978 from the department of surgery, Royal Infirmary, Glasgow, Clement W Imrie devised a grading system similar to Ranson's where only nine factors need to be assessed, this system is also well known as Glasgow scoring system. He further modified this system to include only eight factors, also called Modified Glasgow Scoring system [14]. William A Knavs, in year 1981 developed a system to quantify severity of illness in ICU patients called APACHE (Acute physical and clinical health evaluation) system. However, it did serve as a prototype for development of two subsequent systems APACHE I and APACHE II has been widely applied for grading pancreatitis.

In the field of imaging acute pancreatitis, Emil J Balthazar, professor of radiology, Bellevue medical centre, New York, gave the CT grading of acute pancreatitis [15]. There were various ill defined terminologies with regards to acute pancreatitis. This lead to the symposium at Atlanta where in an universally accepted clinically based classification system for acute pancreatitis was developed in 1992, all the terminologies related to acute pancreatitis were clearly defined and a sound basis for future studies was established [16].

Definition of pancreatitis

Pancreatitis is an inflammation in the pancreas associated with injury to the exocrine and endocrine (at times) parenchyma, resulting in clinical manifestations ranging in severity from a mild, self-limited disease, to a life threatening acute inflammatory process, the duration of which can range from transient attack to a permanent loss of pancreatic function [17].

Classification of pancreatitis

Acute pancreatitis is defined as an inflammatory process of the pancreas and possible peripancreatic tissue with multiorgan involvement including multiorgan dysfunction syndrome (MODS) causing an increased mortality rate [18]. In this, the gland can return to normal if the underlying cause of pancreatitis is removed.

Chronic pancreatitis is defined by the irreversible loss of exocrine pancreatic parenchyma. It is a syndrome involving progressive inflammatory changes in the pancreas that result in permanent structural damage, which leads to impairment of exocrine and endocrine function. Recurrent episodes of acute pancreatitis may lead to chronic pancreatitis overtime [19].

Etiology

Alcohol intake and biliary tract disease account for majority of cases (90%).

Causes of acute pancreatitis

The main causes of acute pancreatitis [20] are discussed below

Obstruction

- Cholidocholithiasis
- Ampulla of pancreatic tumors
- Worms or foreign bodies obstructing the papilla
- Pancreas divisum with accessory duct obstruction
- Choledochal
- Periampullary duodenal diverticulum
- Hypertensive sphincter of oddi

Toxins or drugs

- **Toxins:** alcohol, scorpion venom, organophosphorus, insecticides
- **Drugs:** Azathioprine, Mercaptopurine, valproic acid, estrogens, tetracycline, metronidazole, nitrofurantoin, furosemide, sulfonamides, methyldopa, cimetidine, ranitidine, sulindac, didanosine, acetaminophen, erythromycin, salicylates.

Trauma

- Accidental- blunt trauma to the abdomen.
- Iatrogenic- operations around pancreas, ERCP, Endoscopic sphincterotomy

Metabolic abnormalities

- Hypertriglyceridemia
- Hypercalcemia

Infection
- **Parasitic**: Ascariasis, Clonorchiasis
- **Viral:** Mumps, rubella, Hepatitis A, B, non-A, non-B, Coxsackie virus B, Echo virus, Adeno virus, Cytomegalovirus, Varicella, Epstein- Barr virus, Human- immunodeficiency virus.
- **Bacterial:** Mycoplasma, Campylobacter jejuni, Mycobacterium tuberculosis, Mycobacterium avium complex, Legionella, Leptospirosis.

Vascular abnormalities
- Ischemia- hypoperfusion (Post CABG), atherosclerotic emboli
- Vasculitis- systemic lupus erythematosus, Poly arteritis nodosa, Malignant hypertension

Miscellaneous conditions
- Penetrating peptic ulcer
- Crohn's disease
- Reye's syndrome, Cystic fibrosis
- Hypothermia

Pathology

Morphology

The basic alterations are:
1. Proteolytic destruction of pancreatic substance.
2. Necrosis of blood vessels with subsequent hemorrhage.
3. Necrosis of fat.
4. An accompanying inflammatory reaction. The extent and predominance of each of these features depend on the duration and severity of process.

In the very early stages, only interstitial edema is present. Soon after, focal and confluent area of frank necrosis of endocrine and exocrine tissue are found.

The peritoneal cavity contains a serous and slightly turbid fluid in which globules of oil can be identified. Foci of fat necrosis may be found in any of fat depots.

Histopathology

Focal areas of fat necrosis occur in pancreatic and peripancreatic fat. Following enzyme destruction, adipocytes are transformed into shadowy outlines of cell membranes filled with pink, granular opaque precipitates amorphous basophilic calcium precipitates may be visible with in the necrotic focus. Neutrophilic infiltration and interstitial hemorrhage eventually ensure.

Pathophysiology of acute pancreatitis

The central event in the pathogenesis of acute pancreatitis is the premature activation of trypsinogen in the pancreatic acinar cells. One of the most widely accepted theories to explain this is the colocalization hypothesis.

The Cathepsin B contained in the lysosome, activates the proenzyme trypsinogen intracellularly. This causes cellular autodigestion and local extrusion of acinar cell contents. This non- infective destruction of pancreatic parenchyma induces an inflammatory reaction.

Clinical presentation of acute pancreatitis

An accurate history and thorough clinical examination will often raise clinical suspicion of acute pancreatitis.

Presentation

Pain abdomen, Nausea, vomiting, Abdominal distention and jaundice.

On examination

Presentation is usually as an anxious and apprehensive patient with fear of death.

Tachypnoea, Tachycardia, Elevated temperature

Tenderness: Epigastric and right hypochondriac tenderness is present, may present through out the abdomen.

Abdominal distension: Initially localized to the upper abdomen and later a generalized distension is seen with peripancreatic fluid collection, ascites and pseudocyst formation.

Severe pancreatitis associated with hemorrhage into the retroperitoneum may produce few distinctive signs in about 3% of patients with pancreatitis.
1. Grey turner's sign: Bluish discoloration in the left flank.
2. Cullen's sign: Bluish discoloration of periumbilical region.
3. Fox sign: Bluish discoloration below the inguinal ligament or at the base of thepenis.

Diagnostic work up

The direct inspection of pancreas at laparotomy with microscopic examination of pancreatic tissue is only way to confirm the diagnosis of acute pancreatitis. In routine clinical practice clinical feature particularly pain abdomen, nausea/ vomiting and raised serum amylase and serum lipase are diagnostic cornerstone. Hyperamylasemia Can be seen in various other conditions like Biliary tract disease, intestinal diseases, salivary disorders, renal failure, macroamylasemia.

Urinary amylase levels remain elevated longer than serum levels. Furthermore, elevated serum amylase levels secondary to macroamylasemia may be detected by decreased urinary amylase levels. The ratio of amylase clearance to creatinine clearance (ACCR) varies from 2-4%. In patients of pancreatitis this ratio is increased and may exceed 10%. The ratio varies from 1-5%. A ratio more than 60% is consistent with the diagnosis of acute pancreatitis. This has low specificity [21]. because the amylase creatinine clearance ratio may be raised in diabetic ketoacidosis, burns renal insufficiency, perforated peptic ulcer, pancreatic carcinoma etc.

Serum lipase elevation is a more specific and sensitive indicator of acute pancreatitis than serum amylase because lipase circulating in the serum is mostly pancreatic origin [21]. Lipase is elevated for longer periods and hence useful in patients who present late. The simultaneous determination of amylase and lipase offers a sensitivity and specificity of 90 to 95% for detecting acute pancreatitis in patients presenting with acute abdominal pain.

Diagnostic paracentesis and analysis of peritoneal fluid for elevated amylase and lipase combined with serum elevations of the same has been strongly correlated with acute pancreatitis [22].

Hemoconcentration, leukocytosis, hyperglycemia, hypocalcemia, mild azotemia, hyperbilirubinemia, elevation of aminotransferases, alkaline phosphatase and gamma-

glutamyl transferase, coagulation abnormalities marked by hypercoagulability, hypofibrinogenemia and DIC are the other hematological changes seen in acute pancreatitis.

Radiological procedures

- **Plain radiograph of Abdomen**

Plain radiograph of Abdomen shown in figure-1 and figure-2 reveal paralytic ileus, sentinel loop (dilated proximal jejunal loop), colon cut- off sign (distension of the colon to the level of transverse colon with no gas in the splenic flexure), obliteration of psoas margins [23]. (due to retroperitoneal irritation and pancreatic calcifications). Plain radiograph also rules out potential abdominal emergencies like hollow viscous perforation.

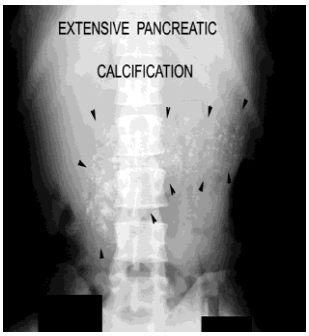

Fig 1: Plain radiograph of erect Abdomen - chronic pancreatitis

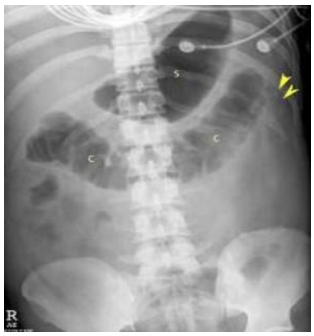

Fig 2: Plain radiograph of erect Abdomen - acute pancreatitis

A chest radiograph may show left pleural effusion, elevated left hemidiaphragm, basal atelectasis and also delineates other causes of pain abdomen like left lower lobe pneumonia or pneumoperitoneum. In multiorgan failure if lung is affected ARDS changes are seen on chest x ray.

- **Abdominal Ultrasonogram**

Abdominal Ultrasonographic evaluation of pancreas may show increased size and decreased echogenicity as well as possible fluid collections. It is the test of choice for diagnosis of gallstones, and sludge.

- **Contrast enhanced computer tomography (CECT)**

Contrast enhanced computer tomography (CECT) is the imaging modality of choice. It has three major roles in the evaluation of patients with known or suspected pancreatitis:

1. Confirm Diagnosis
2. Staging of severity of inflammatory process
3. Detection of complications particularly the identification and quantification of parenchymal and peripancreatic necrosis.

CECT has been shown to have a sensitivity of 87% and an overall detection rate of over 90% of pancreatic gland necrosis [24].

Computed Tomography findings in Acute Pancreatitis

Acute pancreatitis were observed through computed tomography [25] as

Pancreatic changes

Parenchymal enlargement- diffuse, focal; Parenchymal edema; Necrosis

Peripancreatic changes

Blurring of fat planes; Thickening of fascial planes; Presence of fluid collection

Non- specific signs

Pleural effusion; Bowel distension

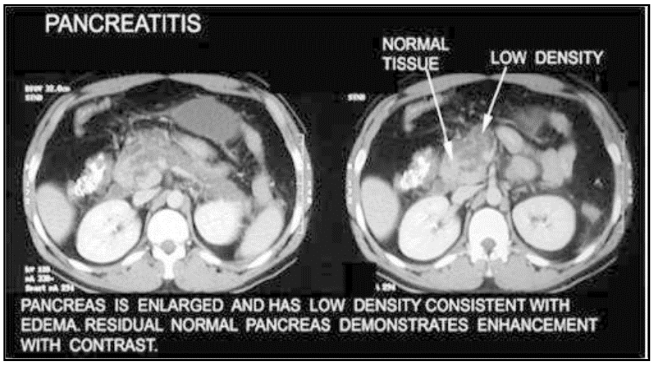

Fig 3: Contrast Enhanced Computed Tomography (CECT)

Treatment of acute pancreatitis

All cases of acute pancreatitis should be stratified into mild or severe during the first 48 hours using one of the scoring systems. Mild cases can be managed in the ward with non-invasive monitoring. Severe cases need intensive monitoring and resuscitation in ICU. Depending on local resources available and patient's condition consideration should be given to referring a patient to specialized centre

Medical treatment

The initial management is mainly non operative and supportive. The goal of initial management is fluid replacement, electrolyte balance, pain management, nutritional support and prevention and treatment of local and systemic complications. The need for patients to avoid factors that may have caused pancreatitis such as alcohol or drugs is self-evident. Studies on the penetration of antibiotics into necrotic tissue found that pefloxacin, metronidazole and imipenem registered good diffusibility. Many other modalities of treatment which showed promising results in animal models and few human studies have been shown to have no benefit in clinical practice. These include Octreotide (Somatostatin analog) Lexipafant (Platelet activating factor inhibitor) and Gabexate mesilate (antiprotease).

Observations and Results

A total number of 45 patients with 49 episodes were entered in the study. All had an admission diagnosis of acute pancreatitis. 5 patients were excluded from the final analysis; 3 patient did not satisfy the diagnostic criteria and 2 patients were diagnosed as chronic pancreatitis. 5 patients had recurrent episodes during the study period. Therefore 40 patients with 45 episodes of acute pancreatitis (n = 40) were analyzed.

Sex and age distribution

Figure-4 depicts that of the 40 patients 28 (70%) were males and 12 (30 %) females. Of these 15(56.41 %) males had a severe disease compared to 3 (25 %) females. The median age of the study group was 35 years (Range 14 - 80 yrs).

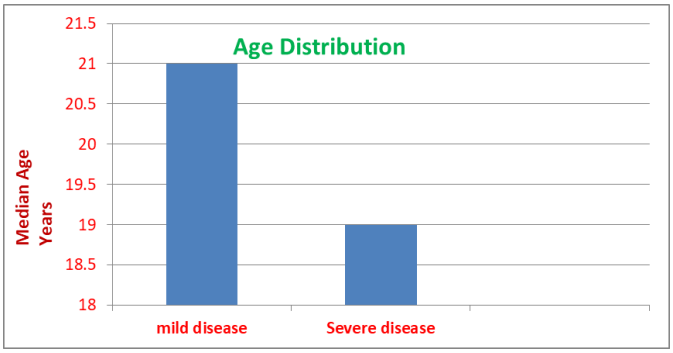

Fig 4: Median age distribution

Clinical features

Most commonly observed was with pain in the abdomen 37 (92.73 %) and vomiting 24 (60 %). Other clinical features included distention of abdomen in 6 (15%) cases, fever in 8 (20%) cases and jaundice in 3 (7.5 %) cases.

Co - Morbidities

13 patients had co-morbidities in the form of Diabetes (8), Hypertension (11), Ischemic heart disease (3), Rheumatic heart disease (1) and Hypothyroidism (1) Three of the seven diabetics had a severe disease.

Etiology

Table 1: Etiologies of acute pancreatitis at our centre

Etiology	N= 40
Gallstone	35.7 (15%)
Alcohol	31 (13%)
Idiopathic	16.7 (11%)
Miscellaneous	16.6 (11%)

Table-1 shows that, 15 patients had biliary pancreatitis, with majority (10) of them having a mild disease. One patient of these had hereditary spherocytosis with pigment stones in the gallbladder and common bile duct.7 of the 11 alcoholic patients had a severe disease. Two of these patients died. 3 patients had pancreatitis due to blunt injury to the abdomen. There was disruption of the main pancreatic duct in 1 of them who presented early. One patient underwent pancreatic jejunostomy. Distal pancreatectomy was done in the second patient. The Third patient was referred with infected pancreatic necrosis three weeks after the accident. 1 patient had Hypertriglyceridemia and 1 was post ERCP. One was attributed to Hypercalcemia. No cause was found in 7 cases. (2 patients had undergone Laparoscopic suspected to be a neoplasm or focal edema of the pancreas. On follow up US G pancreas was found to be normal.

Diagnostic investigations

Serum Amylase was done in patients. It was raised more than four times the upper limit of normal in 18 cases (Sensitivity 50.98 %). It was not done in 4 patients since they were referred with a diagnosis of acute pancreatitis. Serum Lipase was done in 25 cases and it supported the diagnosis in 20 cases (Sensitivity 77.42 %). It was done on an average third to fourth day after symptom onset.

Both Serum Amylase and Serum Lipase were done in 20 cases and both together picked up 16 cases (Sensitivity 80 %). X-rays of the abdomen were routinely done. Ultrasonography (USG) of the abdomen was done in 36 cases and it supported the diagnosis in 28 cases. Contrast Enhanced Computed Tomography (CECT) was done in 15 patients and it supported the diagnosis in all the cases in which it was done. In 5 cases the diagnosis was made only by C.T scan where Sr. Amylase, Sr. Lipase and USG did not support the diagnosis. One patient had undergone exploratory laparotomy for suspected duodenal ulcer perforation and was found to have inflamed and edematous pancreas. The abdomen was closed and he was referred for ICU care.

Severity Stratification and Co-relation of Glasgow scores

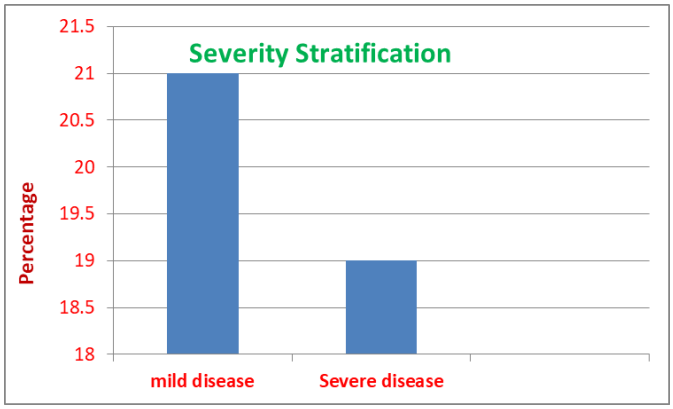

Fig 5: Severity stratification

At the time of discharge all cases were classified into mild or severe according to the Atlanta classification as shown in figure-5, 21 (52.5 %) patients had a mild disease while 19 (47.5% had a severe attack. During the first 48 hours, according to Glasgow criteria Only 6 cases out of 16 patients were predicted to have severe and 20 cases out of 24 patients were predicted to have mild disease. Therefore a total of 26 (65 %) cases were correctly predicted to have mild or severe disease

Local complications

16 (40 %) patients had only acute fluid collections detected by either USG or C.T. scan. All were treated conservatively. 8 (20 %) patients had acute necrosis confirmed on contrast enhanced C.T. scan. 4 (10 %) patients had infected pancreatic necrosis and 2 (5 %) cases had Pancreatic Abscess. All these patients underwent surgery.

Other Complications

15 patients had pleural effusion, mainly on the left side. None of them required aspiration. 8 patients had basal atelectasis. 2 patients had pancreatic fistulae; both closed spontaneously with conservative management for 4 to 6 weeks. 2 patients had wound dehiscence and 1 patient had deep vein thrombosis (DVT).

Organ Failure and Mortality

6 patients had ARDS evident on the X – rays of chest and required mechanical ventilation. 5 patients had acute renal failure (ARF); 3 of which required haemodialysis. 1 patient had severe gastrointestinal (GI) bleed evident by large amount of melena. An emergency colonoscopy was done for this patient after which he developed a caecal perforation. 3 (7.5 %) patients died; 1 within 7 days and another after a long waning and waxing course of ICU stay. The third

patient had necrosectomy 4 times.

Procedures

9 patients 0f biliary Pancreatitis underwent cholecystectomy. 6 open Cholecystectomy with CBD exploration;3 laparoscopic cholecystectomy; 1 in the same admission (12 days after symptom onset) and others on follow up. One underwent laparoscopic converted open Cholecystectomy within a month when he was readmitted with features of obstructive jaundice. 4 patients with biliary pancreatitis had ERCP and sphincterotomy as there were limited facilities. 3 of them who had CBD stones were stented. 1 patient had a limited facilities. 3 of them who had CBD stones were stented. 1 patient had a traumatic disruption of the main pancreatic duct; an ERCP was done and stenting of the pancreatic duct attempted. Since it failed he underwent pancreatico – jejunostomy. 2 patients with traumatic disruption of pancreatic duct underwent pancreatico – jejunostomy. The third case of pancreatic trauma had distal pancreatectomy. 2 patients had necrosectomy on an average 3 weeks after symptom onset. 2 patients had pancreatic abscess drainage with closed cavity continuous lavage.

Nutritional support

Nutritional support was given to 16 patients with severe acute pancreatitis. 12 patients had nasojejunal (NJ) feeding ranging from 6 to 55 days and 1 patient had jejunostomy feeds. 3 patients were given total parenteral nutrition (TPN) ranging from10 to 44 days.

Hospital stay and ICU care

The median hospital stay was 12 days (Range – 3 to 65 days). The median hospital stay in severe cases was 13.5 days while in mild cases was 10 days. 25 patients were managed in the ward while 12 required ICU care ranging from 2 to85 days.

Conclusion

The incidence of acute pancreatitis was found to be in a younger age group in our study. Serum Amylase and Lipase both were (80 % sensitivity) used for diagnosis where ever possible. Ideally all cases should be stratified during the first 48 hours according to one of the scoring systems. Scoring systems help to identify patients who are more likely to have a severe attack. Severe cases should be managed in well-equipped ICU, since they may require, massive fluid resuscitation, mechanical ventilation and haemodialysis. Support of specialist in radiology, endoscopy and intensive care unit are essential. Timely Intervention by endoscopist and surgeons are crucial to reduce morbidity and mortality. Further attacks should be prevented by early cholecystectomy and avoiding alcohol.

This prospective study conducted at Bowring and Lady Curzon Hospital and Victoria Hospital Bangalore, included 40 patients with acute pancreatitis, 28 males and 12 females (M : F ~ 2.44 : 1). The peak incidence was in the fourth decade with, the median age of 35 years. The commonest etiology was gall stones disease, accounted for 35.7% of cases followed by alcohol (31%) and idiopathic (16.7 %). Pain and vomiting were the commonest presenting complaints. 3 patients had, jaundice. Serum Amylase and Serum Lipase together gave high sensitivity (80 %) for diagnosis. Computed Tomography was very sensitive, non-invasive tool for diagnosis and imaging of complications. The enteral route was used for nutritional support in 13patients and total parenteral nutrition was given to 3 patients. The median hospital, stay was 12 days (Range 3 to 85 days) and 23 patients required ICU care (2 to 56 days). Out of 40 patients 53 % had a mild disease while 47 % had a severe attack. The overall mortality rate was 7% and mortality rate among severe cases was 28.33%

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