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General Surgery

A PROSPECTIVE STUDY OF ACUTE PANCREATITIS

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ABSTRACT 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[1]. 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.

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]. However the frequency of different forms of pancreatitis varies from source to source and depends on country of origin and the population studied. Acute pancreatitis resulting from unregulated activation of pancreatic enzymes which can lead to extra pancreatic complications due to persistence of hypovolaemia, 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].

KEYWORDS : Acute pancreatitis,Multi scoring systems, Single prognostic factors, Pseudo cyst, Necrosis, Cystogastrostomy, Multi organ failure, Mortality.

Aims and objectives of the study

1. To study the incidence of complications developing in patients diagnosed as acutepancreatitis.
2. To study nature of complications due to acutepancreatitis.
3. To evaluate patients who need surgicalintervention.
4. To assess the morbidity andmortality.

A brief resume of 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 Chalkaidon. During 100AD Rugus 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 Newyork university medical centre,Newyork. 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. This system attracted lot of criticism because of its inaccuracies. However, it did serve as a prototype for development of two subsequent systems APACHE I and APACHE II has been widely applied for gradingpancreatitis.

In the field of imaging acute pancreatitis, Emil J Balthazar, professor of radiology, Bellevue medical centre, Newyork, gave the CT grading of acutepancreatitis[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 pancreaticfunction[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 isremoved.

Chronic pancreatitis is defined by the irriversible loss of exocrine pancreatic parenchyma. It is a syndrome involving progressive inflammatory changes in the pancreas that result in permanent structural damage, whichleads 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%). Relative frequency depends on the patient population and prevalence of alcoholism in the population studied.

Causes of acute pancreatitis

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

Obstruction

- Cholidocholithiasis
- Ampullary or pancreatictumors
- Worms or foreign bodies obstructing thepapilla
- Pancreas divisum with accessory ductobstruction
- Cholidochocele
- Periampullary duodenaldiverticula
- Hypertensive sphincter ofoddi

Toxins or drugs

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

- **Trauma**
- Accidental- blunt trauma to theabdomen.
- Iatrogenic-operations around pancreas, ERCP, Endoscopic sphinctorotomy

Metabolic abnormalities

- Hypertriglyceridemia
- Hypercalcemia

Infection

- **Parasitic**: Ascariosis,Clonorchiasis
- **Viral:** Mumps, rubella, Hepatitis A, B, non-A, non-B, Coxsackie virus B, Echo virus, Adeno virus, Cytomegalo virus, Varicella, Epstein- Barr virus, Human- immunodeficiencyvirus.
- **Bacterial:** Mycoplasma, Campylobacter jeguni, Mycobacterium tuberculosis, Mycobacterium avium complex, Legionella, Leptospirosis.

Vascular abnormalities

- Ischemia- hypoperfusion (Post CABG), atheroscleroticemboli
- Vasculitis- systemic lupus eruthematosis, Poly arteritis nodosa, Malignant hypertension

Miscelleneous conditions

- Penetrating peptic ulcer
- Crohn'sdisease
- Reye's syndrome, Cystic fibrosis
- Hypothermia

Pathology

Morphology

The morphology of acute pancreatitis stems directly from the action of activated pancreatic enzymes that are released into the pancreatic substance.

The basic alterations are:-

1. Proteolytic destruction of pancreaticsubstance.
2. Necrosis of blood vessels with subsequenthemorrhage.
3. Necrosis offat.
4. An accompanying inflammatory reaction. The extent and predominance of each of these features depend on the duration and severity ofprocess.

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 interstetial 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 proenzymetrypsinogenintracellularly. 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 pseudocystformation.

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 leftflank.
2. Cullen's sign: Bluish discoloration of periumbilicalregion.
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 rised serum amylase and serum lipase are diagnosticcornerstone.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. Further more 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%.Inpatient so fpancreatitisthisratioisincreasedand 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 abdominalpain.

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- glutamyltransferase, 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 transversecolon with no gas in the spleenic 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.

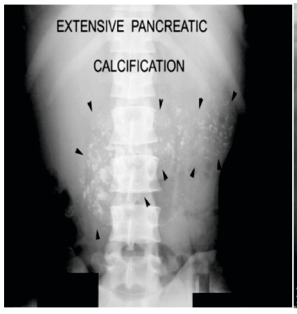

Figure-1:Plain radiograph of erect Abdomen chronic pancreatitis

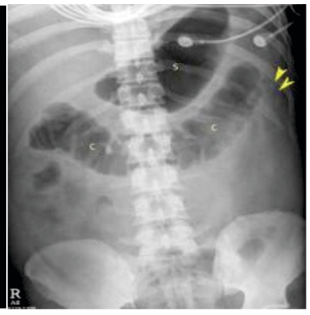

Figure-2:Plain radiograph of erect Abdomen acute pancreatitis

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 pneumoperitonium. 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 suspectedpancreatitis:

1. Confirmdiagnosis
2. Staging of severity of inflammatoryprocess
3. Detection of complications particularly the identification and quantification of parenchymal and peri pancreaticnecrosis.

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

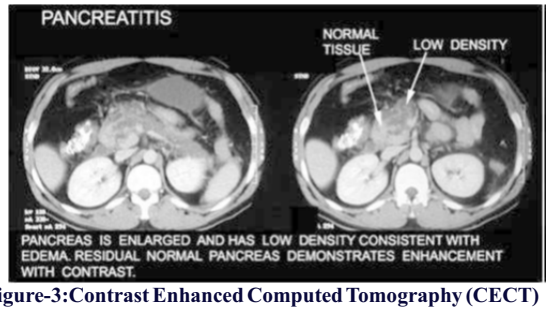

Figure-3:Contrast Enhanced Computed Tomography (CECT)

Treatment of acute pancreatitis

All cases of acute pancreatitis should be stratified into mild or severeduring the first 48 hours using one of the scoring systems. Severe cases need intensive monitoring and resuscitation in ICU.

Medical treatment

The initial management is mainly non operative and supportive. Thegoal of initial management are fluid replacement, electrolyte balance, pain management, nutritional support and prevention and treatment of local and systemic complications.

Fluid replacement

Fluid losses can be enormous leading to hemoconcentration andhypovolemia. External fluid losses are caused by repeated vomiting and nausea, which limits fluid intake. Internal losses are caused by fluid sequestration into third spaces. These losses should be replaced by crystalloids and colloids to maintain a CVP of 8-12 cm of water. Definitive treatment of a patient with acute pancreatitis in the initial 72 hrs is fluid transfusion to maintain hemodynamic stability.

Pain management

Pain in acute pancreatitis can be very severe and usually requiresparenteral analgesics. Narcotics are the therapy of choice. There is no definitive human study to support the wide spread belief that morphine exacerbates pancreatitis by contracting sphincter of oddi.

Nutritional support

In mild cases there is no evidence that either enteral or parenteralnutrition has a beneficial effect on patient outcome[26]. Oral re-feeding can be started once the pain is controlled. In severe cases either total parenteral nutrition (TPN) or enteral feeding is employed and should be started early. Enteral feeding also prevents bowel mucosal injury and translocation of gut bacteria thus reducing chances of infection of pancreas.

Role of prophylactic Antibiotics

Infectious complications are regarded as the leading cause ofmortality in severe pancreatitis. In about 30% of cases the infection tends to be polymicrobial. E.coli was the commonest organism followed by Klebsiellapneumoniae, Enterococci, Staphylococcus, and Pseudomonas. The use of prophylactic broad spectrum antibiotics reduces infection rates in CT proven necrotizing pancreatitis but may not improve survival.Imipenem significantly reduces the incidence of pancreatitc sepsis in patients with necrotizing pancreatitis &imipenem – Cilastin 500mg three times a day for 2 weeks is recommended[27].

Other modalities include Octreotide (Somatostatinanalog), Lexipafant (Platelet activating factor inhibitor) and Gabexatemesilate (antiprotease).

Role of ERCP in Gallstone Pancreatitis

Gall stones are the leading cause of acute pancreatitis in many western and Asian countries. The working party Report (BangkokmWorld Congress of Gastroenterology 2002) has given following guideliness.

1. Urgent ERCP should be performed in patients with acute pancreatitis of suspected or proven gall stone etiology when criteria for severity are met, and / or there is co-existent cholangitis, jaundice, dilated CBD, or when there is clinical deterioration in patients with initial mildprognostic signs.
2. Endoscopic sphincterotomy is recommended in patients with severe gallstone pancreatitis with significant local and / or systemic complications, dilated bile duct without demonstrable stones, and a gall bladder containing stones if cholecystectomy is neither possible nor contemplated.

Surgical treatment

The operative procedures include the following:

1. Necrosectomy
2. Percutaneous drainage
3. Endoscopic (Minimally invasive) procedures
4. Cholecystectomy

Treatment of complications

Infected pancreatic necrosis can cause significant damage toperipancreatic viscera. Longer the infective process is allowed to persist, the higher are the chances of damage to the peripancreatic structures. Perforations of stomach are usually on the posterior wall and are technically easy to close, but the chances of breakdown are high. Interposition of omentum between the stomach and the pancreas may be a useful adjunct to avoid breakdown of suture line.

Erosions of the medial wall of the duodenum are very difficult to manage and require pancreatic oduodenectomy. Perforations of the lateral aspect of duodenum are easier to manage, a serosalonlay patch using a jejunal loop gives good results.

About 2% of patients develop colonic complications in the course of the illness, which include ileus, fistula, perforation and colonic necrosis (bad prognostic factor). Resection of non viable colon and proximal colostomy is needed.

Perforation of the gall bladder or the bile duct is extremely rare. These patient have biliary peritonitis and require cholecystectomy or Biliary enteric bypass. Awareness of the existence of such complications can prevent delay in surgical intervention, a factor which adversely affects the outcome.

Outcome of the disease

Studies of exocrine function show persistent functional insufficiency inthe majority of patients up to 2 years after severe acute pancreatitis. The long term clinical endocrine and exocrine consequences of acute pancreatitis depend on the following factors:

1. Cause (alcohol or non alcoholic)
2. Whether patient continues to consume alcohol.
3. Severity of necrosis
4. Degree of surgical pancreatic debridement

The main quality-of-life outcomes up to 2 years after treatment are similar to those obtained with coronary artery bypass grafting.

MethodologyThis prospective study will be conducted on patients admitted to General Surgery ward Sri Venkateswara Ram Narayan Ruia Government General Hospital, Tirupathi.

Materials and methods

The diagnostic criteria include at least one of the following:

1. Serum amylase more than 4 times the upper limit of normal.
2. Serum lipase more than 2 times the upper limit of normal.
3. Ultrasound or CT scan suggestive of acute pancreatitis.

This is based on the UK guidelines for the management of acute pancreatitis. After approval of institutional ethical committee and written informed consent from the patients, 90 patients will be studied.

Inclusion criteria

Patients admitted to General Surgery ward and diagnosed to have acute pancreatitis.
All patients should fulfill the diagnostic criteria.
Patient giving written informed consent
Patient age above 14 years and below 60 years.

Exclusion criteria

Pediatric age group (<14yrs).
Age group above 60 yrs.
Patients not giving consent

On admission history will be collected and thorough physical examination will be conducted. Data collection on admission includes age, sex, address and clinical presentation with respect to pain, vomiting, jaundice and distention of abdomen. History of previous episodes and comorbidities will be noted.

During the first 48hrs patients will be stratified according to the Glasgow criteria as recommended by the UK guidelines. All investigations will not be done in patients who already have Glasgow score equal to or more than 3, investigations will not be repeated in patients who are obviously improving and not affordable.

No steps will be taken to suggest changes in decisions made by the treating unit regarding investigations or treatment. Patients with complications and operated patients will be managed in the ICU.

On discharge or death patients will be stratified into mild or severe according to Atlanta Classification. Data on complications, investigations, interventions undertaken, outcome, duration of stay in hospital and ICU and mode of nutritional support will be collected. This will be followed by comparison between prediction of severity by Glasgow criteria and Atlanta classification.

Patients with Biliary pancreatitis were offered cholecystectomy as needed. Patients with alcoholic pancreatitis were urged to stop consuming alcohol and deaddiction was attempted with the help of Psychiatrist in a few cases.

Observation andresults

Statistics

Data of 90 patients, who satisfy the diagnostic criteria were collected and processed, the observations are represented as below.

Age distribution

The figure-4 shows peak incidence of age distribution is in the 4th decade.The mean age of the study group was 37.86 years.

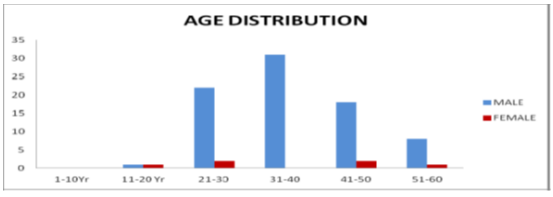

Figure-4: Frequency of age distribution

Sex distribution

The figure-5 shows, of the 90 patients 85 (94.4%) were males and 5 (5.6%) females. Of these 31(36.47%) males had a severe disease and 1 (20%) females had severe disease.

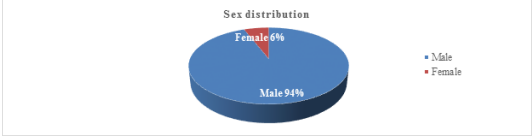

Figure-5: Frequency of sex distribution

Etiology:

The figure-6 confirmsAlcohol consumption is the most common etiology with history of alcohol consumption present in 76 (82.6%) patients. 6 (6.7%) patients had Biliary pancreatitis, with majority of them havingmilder disease. 1 (1.1%) patient had pancreatitis due to blunt injury to the abdomen. 1 (1.1%) patient had drug induced pancreatitis, a case of RVD and on ART. 6 (6.7%) patients had no cause

identified.

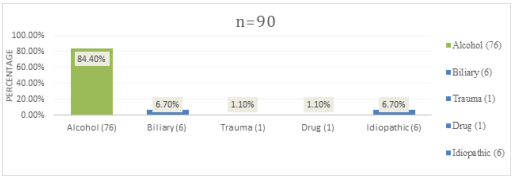

Figure-6:Etiological distribution

Clinical features

In the observations of figure-7 pain abdomen and vomiting are commonest presentation. Pain in abdomen is present in 89 (98.9%) patients and vomiting in 68 (73.9%) patients. Other clinical features included, distention of abdomen in 17 (18.5%) cases, fever in 28 (30.4%) cases, and jaundice in 5 (5.4%) cases.

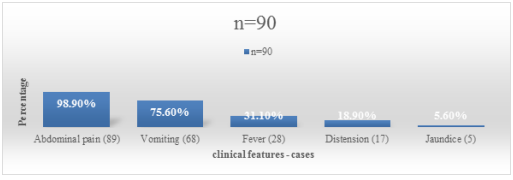

Figure-7:Clinical features

Co-morbitities

20 patients out of 90 had history of pre existing co-morbidities in the form of diabetes (12), hypertension (9), ischemic heart disease (3). 6 out of 12 diabetics had a severe disease.

Diagnostic investigations

From the diagnostic investigations as shown in the table-1, while serum Amylase supported the diagnosis in 69 cases (Sensitivity 76.67%) and serum Lipase supported the diagnosis in 80 cases (sensitivity 88.8%), both Serum Amylase and serum Lipase together picked up 86 cases (sensitivity 95.5%). X-rays of abdomen and Ultrasonography (USG) of the abdomen was done in all cases and USG supported the diagnosis in 77 (85.5%) cases. Computer Tomography (CT) was done in 80 patients and it supported the diagnosis in all the 80 cases.

Table-1:Diagnostic investigations

Test	Done in	Suppoted diagnosis	Didn't support diagnosis
Serum Amylase	All	69 (76.67%)	21
Serum Lipase	All	80 (88.8%)	10
Both	All	86 (95.5%)	4
USG	All	77 (85.5%)	13
CT	80	80 (100%)	0

Severity stratification and co-relation of glasgow scores

At the time of discharge or death all cases were classified into mild orsevere according to the Atlanta classification.

From the observations of figure-8 shown as 58 (64.5%) patients had a mild disease while 32 (35.5%) had a severe attack. During the first 48 hours patients were predicted to have severe ormild disease according to Glasgow criteria. According to Glasgow criteria 65 out of 90 patients were predicted to have mild disease and 25 out of 90 patients were predicted to have severe disease.

SEVERE CASES: 18 cases out of 25 were correctly predicted to be severe by the Glasgow scores.

MILD CASES: 52 cases out of 65 were correctly predicted to be mild by the Glasgow scores.

Therefore a total of 70 (77.77%) cases were correctly predicted to have mild or severe disease. Hence positive predictive value of Glasgow criteria found to be 77.77%.

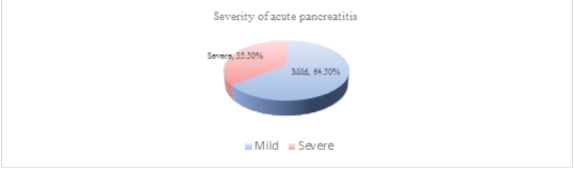

Figure-8: severity of acute pancreatitis

Local complications

The figure-9 shows Pancreatic Ascites was present in 17 (18.9%) patients. All of them were treated conservatively. Organised fluid collection in the form of Pseudocyst detected by eitherUSG or CT scan was present in 7(7.8%) patients. Most of these were treated conservatively and by follow up but two of them with thick cyst wall was treated with cystogastrostomy during the same hospital admission.

Five (5.6%) patients had acute necrosis confirmed on C.T scan with 2 of these patients developed Pancreatic abscess which was drained. Out of three other patients two underwent necrosectomy and one was treated conservatively.

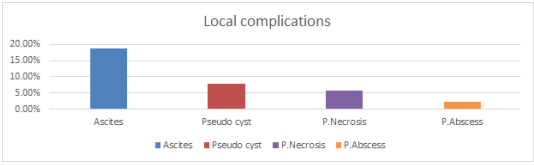

Figure-9: local complications

Other complications

7 (7.8%) patients had pleural effusion, mainly on the left side. None of them required therapeutic intervention. 3 (3.3%) patients had basal Atelectasis. 1patient had wound dehiscence and 1 patient had deep vein thrombosis (DVT).

Organ failure and mortality

The figure-10 shows following observations,in which2(2.2%) patients had ARDS evident on the X-ray of chest and required mechanical ventilation. 4 (4.4%) patients had acute renal failure (ARF), 1 required haemodyalsis. 3 (3.3%) patients died; 2 of these died secondary to ARDS and 1 patient due to ARF.

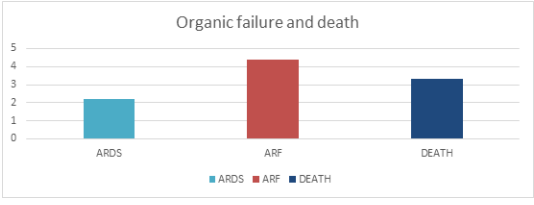

Figure-10:Organ failure and death

Surgical procedures

The figure-11 confirms following observation, in which Necrosectomy was performed on 3 (3.3%) patients with pancreatic necrosis. Two patients with pancreatic abscess underwent external drainage of abscess. One patient with traumatic pancreatitis underwent exploratory laparotomy with debridement of pancreas. Open cystogastrostomy was performed in two patients with matured pancreatic pseudocyst.

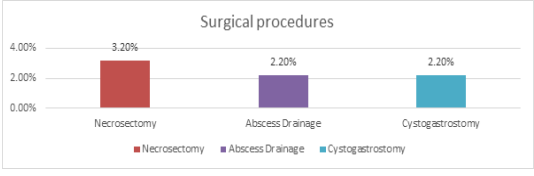

Figure-11: Surgical procedure

Nutritional support

Figure-12 shows that Nutritional support was given to 18 (20%) patients with severe acute pancreatitis. 12 (13.33%) patients had enteral nutrition (EN) by naso-jejunal(NJ) feeding while 6 (6.67%) patients were given total parenteral nutrition (TPN).

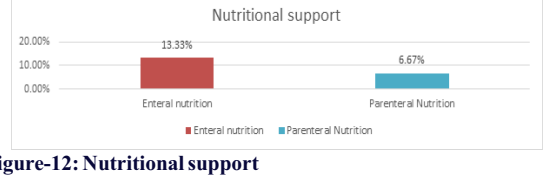

Figure-12: Nutritional support

Hospital stay and ICU care

The mean hospital stay was 12.13 days (Range – 6 to 34 days). The mean hospital stay in severe cases was 18.33 days while in mild cases was 8.79 days. Where the standard deviation for the mild and severe cases is observed to be 2.215 and 6.981 respectively.

The statistically analysed data by unpaired t-test observed as 7.877 and the p-value is found to be 0.001, whichis significant. Hence there is significant difference in hospital stay between mild and severe cases.

Discussion

Patients with severe acute pancreatitis demand considerable resourcesin the form of imaging, endoscopy, surgery and intensive care. This study was conducted on patients admitted in General Surgery ward in Sri Venkateswara Ram Narayan Ruia Government General Hospital (Sri Venkateswara medical college), Tirupathi.

In this study, analysis of clinical presentation of acute pancreatitis wasdone. Relevant investigations were carried out and patients appropriately managed depending upon the etiology and severity of acute pancreatitis.

Age

According to the table-2, the mean age of presentation in our study was 37.86 years and iscomparable to the study by Kashid A et al[28] which is 35 years. Studies conducted by Pupelis G et al[29] had late presentation of 47years and study conducted by Buchler MW et al[30] had a mean age of presentation of 55.1.

This is probably because alcohol was the main etiological factor inour study which presents usually in the younger age group.

Table-2:Comparison of Mean Age Presentation of various studies

MEAN	Kashid A et al	Pupelis G Et al	Buchler MW et al	Present Study
AGE	35	47	55.1	37.86

Sex

As per the data shown in the table-3, there was a male preponderance in our study with a M:F ratio is16.5:1. Male patients accounting for 94.4% and female patients accounting for 5.6%. The other studies like study conducted by Kashid A et al[28] had male patients of 70.91% and female patients of 29.9%. Pupelis G et al[29] study had 73.7% male patients and 26.3% female patients. Study conducted by BuchlerMW et al[30] had 61% male patients and 39% female patients. The other studies although had a higher percentage of males but the ratio of M:F was low. This again is attributed to alcohol which was the main etiologic agent and which is more common in male population of low socioeconomic status in India.

Table-3: Comparison of Male Gender Predominance of various studies

SEX	Kashid A et al	Pupelis G Et al	Buchler MW et al	Present Study
Male(%)	70.91	73.7	61	94.4
Female(%)	29.09	26.3	39	5.6

Etiology

From the observations of table-4 reported as alcohol is the most common etiological agent causing acute pancreatitis.

Alcohol

The exact mechanism of alcohol related injury is unknown. Several theories exist

1. It promotes secretion of pancreatic juice that is high in proteolyticenzyme content but low in enzyme inhibitor content. Enzyme activation could occur in these circumstances and cause pancreatic injury.
2. Secretion of an enzyme rich fluid deficient in enzyme inhibitors could also lead to protein precipitation and the formation of intraductalplugs, leading to intraductal hypertension.
3. Transient state of Hypertriglyceridemia induced by alcohol ingestion causes toxic levels of free fatty acids and their ethyl ester metabolites produced from lipolysis.
4. Acetaldehyde, a byproduct of ethanol metabolism induces microtubule disruption and increases acinar cell membranepermeability.
5. Sphincter of Oddi spasm may be caused by ethanol ingestion resulting in ductal hypertension.

Gall stones pancreatitis

Pancreatic injury can be initiated by the edema and inflammation caused by the migration of a gall stone and not necessarily by impaction. Although the bile reflux theory, often referred to as the common channeltheory' was initially favoured, most observers now believe that it is stone induced pancreatic duct obstruction and ductal hypertension, rather than bile reflux that triggers acute pancreatitis.

Hypercalcemia

Hypercalcemia due to hyperparathyroidism cause acute pancreatitis.

1. Calcium stimulate pancreatic hypersecretion.
2. Calcium induced trypsinogen activation with subsequent parenchymal auto destruction.
3. Calcium associated stone precipitation in the pancreatic duct causing ductal obstruction.

Hyperlipidemias

Hyperlipidemias alone without alcohol abuse can cause acute pancreatitis.

Primary hyperlipidemias – Ferdrickson's type 1 and type v notable for hyper-triglyceridemia and chylomicronemia.

Secondary hyperlipidemias-extraneous estrogen administration, nephritis, and castration may also be the cause of acute pancreatitis.

Idiopathic pancreatitis

Recent studies have clarified the etiology of acute pancreatitis inmany patients once classified as having idiopathic pancreatitis. 60% of these patients were identified to have biliary sludge termed microlithiasis.(suspension of cholesterol monohydrate crystal or calcium bilirubinategranules). Cholecystectomy or endoscopic sphincterotomy prevented relapse of pancreatitis in these patients.

By comparing etiology of acute pancreatitis the table-4 shows Alcohol was the main etiological factor in our study and present inabout 81.1% of patients. This was comparable to the study by Sand J et al[31]at Finland which was 70%. Study conducted by Kashid A et al[28] had etiology of Alcohol in 29.1% and Biliary etiology in 36.4%. Study conducted by Pupelis G et al[29] had etiology of alcohol 54% and 19% Biliary. Other study conducted by Buchler MW et al[30] had etiology of Alcohol 33% and45% Biliary.

Table-4:Comparison of etiology

SEX	Kashid A et al	Pupelis G Et al	Buchler MW et al	Present Study
Alcohol (%)	29.1	54	33	81.1
Biliary (%)	36.4	19	20	5.6

Clinical features

From the observations of table-5 confirms that pain abdomen is the most common complaint**.**

Pain abdomen

Mild to severe epigastric, right and left hypochondriac pain dependingon the location of acute inflammation, with radiation to the back especially interscapular region classically, the pain is characterised as constant , dull and boring, and worse when the patient is supine and may lessen when the patient assumes a sitting bending forwards. A heavy meal or drinking binge often triggers the pain. Our study had 98.9% 0f patients presenting with pain abdomen and study conducted by Kashid A et al[28] had 92.73% of patients presenting with pain abdomen.

Nausea and vomiting

Nausea and non-feculent vomiting are presentin 75 to 90% of patients.vomiting may be severe and protracted. Our study had 75.6% of patients presenting with nausea/vomiting where as study conducted by Kashid A et al[28] had 60% of patients presenting with nausea or vomiting.

Abdominal distention

This is due to result of paralytic ileus araising from retroperitonealirritation or ascitis, or it may occur secondary to a retro peritoneal phlegmon. Our study had 18.9% of patients presenting with abdominal distention which is comparable with study by Kashid A et

al[28] which had 16.36% of patients presenting with abdominal distention.

Jaundice

Jaundice may be occasionally seen in cases of gallstone pancreatitis, itrepresents distal CBD obstruction by gall stones In the present study patients presenting with jaundice were 5.6% which is comparable to the study by Kashid A et al[28] which had 7.27%. In present study 31.1% patients had fever and study conducted by Kashid A et al[28] had 20% patients with fever.

Table-5: Comparison of clinical features

Clinical features	Kashid A et al	Present study
Abdominal pain	92.73	98.9
Nausea /Vomiting	60	75.6
Fever	20	31.1
Abdominal distension	63.3	18.9
Jaundice	7.27	5.6

Serum amylase sensitivity

The elevation of serum amylase is observed within 24hrs of the onsetof symptoms and gradually returns to the normal in subsequent week. Persistent elevation of serum amylase beyond initial week of illness reflects ongoing pancreatic inflammation or development of complication,

pseudocyst or abscess. Serum amylase determination has high sensitivity(>95%). But overall specificity is low (70%), since elevated serum levels occur in many conditions(intra abdominaland extra abdominal). Furthermore, amylase level is not raised in 5% of cases being hyperlipidemic pancreatitis, extensive pancreatic necrosis, and chronically diseased pancreas.

Improved accuracy in diagnosis of acute pancreatitis can be achieved by measuring amylase isoenzyme components. P- typeisoenzyme which arises from pancreas is more specific than total amylase and may persist forlonger time.

From the table-6, comparing with other studies shown that the sensitivity of serum amylase was 76.67% in the present study. Inthe study by Thomson[32] it was 95.6% sensitive and this can be attributed to the late presentation of patients to our institution, and also because alcohol is a main etiological agent, where raising serum amylase is less compared to Biliary pancreatitis.

Table-6:Comparison of serum Amylase sensitivity

Serum Amylase	Thomson et al	Present study
Sensitivity (%)	95.6%	76.67%

Accuracy of ultrasound abdomen

The table-7 shows that theUltrasonography of abdomen was diagnostic in 85% of patients in our study and this was comparable to the study by Ammori et al[33] . It was diagnostic in 66.67% of patients in the study by Kashid A et al[28] and this may be because ultrasonography is operator dependant and also because the view can be obscured by overlying bowel gas.

Table-7:Comparison of accuracy of ultrasonogram of Abdomen

Ultrasonography Abdomen	Kashid A et al	Ammori BJ et al	Present study
Diagnosis (%)	66.67	86	85
Non diagnostic (%)	33.33	14	15

Severity of acute pancreatitis

Early assessment and prediction of severity are of outstandingimportance to avoid costly and invasive monitoring and treatment in the largest group of patients, who tend to run a benign course.

Necessity of objective stratification

1. For practicing clinicians, a method for predicting the likely course of the disease soon after admission would be a guide to the need for more intensive monitoring or transfer to a specialist centre or serve as justification for any proposed therapeutic intervention.
2. Objective grading of disease severity would allow comparison of outcomes between centers, a necessity for both effective clinical audit and comparison of differing therapeutic approaches.
3. An accurate assessment of disease severity at hospital admission enables selection of patients for clinical trials.

Multifactor scoring system

Many multifactor scoring systems have been described in an attempt toaccurately predict the outcome of the disease.

1. Ranson's criteria
2. Glassgow criteria
3. APACHE I II III – (acute physiology and chronic health enquiry)
4. Balthazar's score- depends on CT scan findings
5. MRCS score-(Medical Research Council Sepsis)
6. SAP score- (Simplified Acute Physiology)

Ranson's criteria

One of the early systems for judging severity was developed byRanson in 1974.

From the table-8 shows the five Initial criteria assess the severity of the acute inflammatory process, where as the six criteria measured at 48 hrsdetermine the systemic effects of circulating enzymes and toxins .The presence of 3 or more Ranson's signs usually indicate severe pancreatitis.

Table-8:Ransons criteria

On admission to hospital	Within 48 hours
Non gall stone pancreatitis:	
Age>55 years	Decrease in PCV>10 points
WBC count>16000 per mm3	Increase in BUN>5mg/dl
Glucose>200mg/dl	Serum calcium<8mg/dl
LDH>350U/L	Arterial PO2<60mm/Hg
Aspartate aminotransferase>250U/L	Base deficit>4mmol/l Fluid sequestration>6l
Gall stone pancreatitis:	
Age>70 years	Decrease in PCV>10 points
WBC count>18000 mm3	Increase in BUN>2mg/dl
Glucose>220mg/dl	Serum calcium<8mg/dl
LDH>400U/dl	Base deficit>5mmol/dl
Aspartate aminotransferase>250U/L	Fluid sequestration>4l

Mortality increases with the number of Ranson's signs

The table-9 shows death rate according to Ranson criteria

Table-9: Death rate

Criteria	Death rate
2 or < 2	<1%
3-4	<16%
5 or >5	>40%

Modified glasgow criteria

Further modification of this system in Glasgow by Imrie and hiscolleagues in 1978 led to the Glasgow system where only 9 factors need to be assessed. A further refinement of this system by Blamey and Imrie led to modified Glasgow system shown in table-10 where only 8 factors need to be assessed.

Table-10:Modified glasgow criteria

Within 48 hours of admission
Age >55 years
WBC count>15000/mm3
Glucose >180mg/dl
BUN>45mg/dl(no response to I.V. fluids)
Lactate dehydrogenase>600 U/l
Albumin < 3.2 gm/dl
Arterial PO2<60 mm Hg
Serum calcium < 8 mg/dl

A limitation of the commonly used scoring systems such as Ransonsor Glasgow criteria is the need to wait for 48 hours to obtain a complete assessment despite refinements, the above scoring systems were found to have low sensitivity and specificity.

Apache system

The APACHE (acute physiology and chronic health evaluation) system, was reported by Knaus and Colleagues in 1981. In the original form, APACHE contained 34 potential physiologicandlaboratory measurements and included many continuous variables. A valueof 0 (normal) to 4 (most abnormal) was assigned to each variable, according to its degree of abnormality.

To this was added an assessment of the patients pre admissionstatus (A-fit to D-severely compromised health) to give the overall APACHE score. Shortly after its introduction APACHE I system was disfavored, because of practical problems like collection of large number of variables. a much simplified modification, the APACHE II system was reported in 1985, which utilized only 12 routinely available physiologic and laboratory measurements, with an additional weightage to age and pre admission health status. An increasing APACHE II score was found to correlate with subsequent risk of hospital deaths. A score of 8 or more predicts a severe disease.this system has the advantage of continually quantifying the patient.

Balthazar's ct severity index (CTSI)

The morphological severity of acute pancreatitis can be definedprecisely using this index developed by balthazar and co workers. The severity of the acute inflammatory process is categorized into stage A through E, corresponding to scores of 0 to 4 respectively as shown in the table-11.

Table-11: Balthazar's ct severity index

CT grade	CT Scan description	Score
A	Normal pancreas	0
B	Intrinsic changes <3cm of necrosis enlargement intrapancreatic fluid collection.	1
C	Intrinsic and extrinsic inflammatory changes	2
D	Extinsic changes-not >1 peripancreatic fluid Collection	3
E	Multiple or extensive extra pancreatic fluid collection or abscess.	4

Table-12: CT Severity Index

Necrosis	Score
None	0
0-30%	2
30-50	4
>50%	6

Index	Morbidity	Mortality
0-3	8%	3%
4-6	35%	6%
7-10	92%	17%

Drawbacks of CT scan are the expence, limited availability, limitedspecificity and inconvenience for severely ill patients.

The table-12 shows secondly the presence of and extent of gland necrosis is assessed. The CT grade score is added to the necrosis score.

Single prognostic factors

Several clinical signs, biochemical markers and imaging procedureshave emerged in an attempt at early identification of pancreatic necrosis monitoring of its progression and assessment of the response to therapy. In particular C-reactive protein, leokocyteelastase, trypsinogenactivation peptide and interleukin -6 have shown promise as simple markers of disease severity.

The table-13 shows that interleukin-6 is most sensitive marker of acute pancreatitis

Table-13: Single prognostic factors for early (day 1) prediction of severity in acute pancreatitis.

Factor	Sensitivity	Specificity
Interleukine-6	100	71
Phospholipase A2	75	78
TAP(Trypsinogen activation peptide)	58	73
SPINK/HPSTI Serine protease inhibitor kazal type / Human pancreatic secretory trypsin inhibitor	71	77
Trypsinogen-2	91	71
Hong kong criteria	79	67
Hepatocyte growth factor	71	86
Neutrophil elastase	77	92
Neopterin	21	93
Procalcitonin	67	89

Atlanta classification

An international symposium was conducted September 11 through 13,1992, at Atlanta, and an unanimous consensus on a series of definitions and a clinically based classification system for acute pancreatitis was achieved by a diverse group of forty international authorities from six medical disciplines and fifteen countries.

The Atlanta symposium defined terms like acute pancreatitis (severe and mild) acute fluid collections, necrosis, pseudo cyst and abscess. The present study makes use of these definitions while describing the patient outcome.

Definitions

Acute pancreatitis

It is an acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems.

Severe acute pancreatitis

It is associated with organ failure or local complications such asnecrosis, abcess or pseudocyst. This is characterized by three or more ransons criteria or eight or more APACHE II points.

Mild acute pancreatitis

It is associated with minimal organ dysfunction and an uneventful recovery and it lacks the described features of severe acute pancreatitis.

Acute fluid collections

It occurs early in the course of acute pancreatitis, are located in ornear the pancreas and always lack a wall of granulation or fibrous tissue. It represents a early point of development of acute pseudocyst or pancreatic abscess.

Pancreatic necrosis

It is a diffuse or focal area(s) of non viable pancreatic parenchymawhich is typically associated with peripancreatic fat necrosis. Diffuse or focal well marginated zones of non enhanced pancreatic parenchyma that are larger than 3 cm or involve more than 30%of the pancreatic area are requisite criteria for CT diagnosis.

Acute Pseudocyst

A Pseudocyst is a collection of pancreatic juice enclosed by a wall offibrous or granulation tissue which arises as a consequence of acutepancreatitis, pancreatic trauma or chronic pancreatitis. Formation of a pseudocyst requires 4 or more weeks from the onset of acute pancreatitis. In this regard an acute pseudocyst is a fluid collection that arises in association of an episode of acute pancreatitis, is of more than 4 weeks duration and is surrounded by a well defined wall.

Pancreatic Abscess

It is a circumscribed intra abdominal collection of pus usually inproximity to the pancreas, containing little or no pancreatic necrosis, which arises as a consequence of acute pancreatitis or pancreatic trauma. Pancreatic abscess occurs lately in the the course of severe acute pancreatitis, often 4 weeks or more after onset. Phlegmon, infected pseudocyst, hemorrhagic pancreatitis and persistent acute pancreatitis are non specific terms in general usage, these should be discarded and specific terms as defined above should be used.

Organ failure

It is defined as

1. Shock-systolic B.P.< 90MM of Hg
2. Pulmonary insufficiency – PaO2 60 mm Hg or less
3. Renal failure – creatinine >2mg/dl (after rehydration)
4. Gastrointestinal bleed >500 ml/24 hrs

From the comparison of severity of acute pancreatitis table-14 shown as, 65% of the patients had a mild disease and 35% patients had a severe disease in our study, whereas in study by Kashid A et al[28] 52.73% patients had mild disease and 47.27% patients had severe disease. In study by Buchler MW et al[30] 58% patients had a mild disease and 42% of patientshad a severe disease.

Table-14:Comparison of severity of acute pancreatitis

Severity	Kashid A et al56	Buchler MW et al58	Present study
Mild disease (%)	52. 73	58	65
Severe disease (%)	47.27	42	35

Complications

The clinical course of acute pancreatitis can be divided into twooverlapping phases.

1. The early 'Toxemic' phase (0-15 days) characterized by distant organ damage leading to multi organ failure.
2. The late 'Necrotic phase' (after 2nd week) characterized by locoregional complications.

The systemic complications that characterize the early stage of theattack, particularly cardiovascular and respiratory failure, are mainly due to spilling of active pancreatic enzymes and toxic substances into systemic circulation.

The hemodynamic response is similar to gram negative sepsis, whichincludes increased resting energy expenditure, elevated protein catabolism, hepatic gluconeogenesis and peripheral insulin resistance. Respiratory complications arise from reactive changes like pleuraleffusion, elevated diaphragm due to pain, atelectasis, hypoxemia and pulmonary infection. About 20% suffer from early ARDS. Activation of phospholipase A2 releases an enzyme with potent cytolyticproperties that can cause acute pulmonary injury by degrading the surfactant.

Hypovolemia plays a major role in renal dysfunction. Some patientsshow evidence of membranous glomerulopathy and acute tubular necrosis. The release of trypsin activates complement and kinin, possibly playing a part in disseminated intravascular coagulation, shock and renal failure. Kallikrein is also activated by circulating trypsin leading to the release of bradykinin and kallidin, which may be instrumental in causing vascular instability.

Gastrointestinal and retroperitoneal hemorrhage can occur due togastric ulcers, esophagealvarices, erosion of visceral vessels and pseudo aneurysms. The activation of elastase leads to the erosion of elastic components of pancreatic blood vessels contributing to intrapancreatic hemorrhage.

Hyperglycemia may be a consequence of impaired endocrine functionof pancreas as a result of extensive necrosis. Raised levels of LDH in acute pancreatitis are mainly of extrapancreatic origin and are indicative of distal organ damage. Hypocalcemia may result from sequestrating of circulating calcium and albumin in extravascular space owing to increased microvascular permeability.

Local complications

Local complications like infected pancreatic necrosis (IPN) andpancreatic abscess adversely affect the outcome. The most important route of bacterial infection occurs via translocation from the gut. Other modes of infection are microperforations of transverse colon and hematogenous. Reflex from the CBD or duodenum into the main pancreatic duct seems to be a rare cause of infected necrosis. The risk of pancreatic sepsis is maximum in the 3rd week. Bacteriological analysis shows predominantly gram negative microbes derived from the gut.

Escherichia coli are the most frequent pathogen followed by Enterococcus and Klebsiella. Enterobacter, Staphylococci, Anaerobes and Fungi are found in fewer than 20% cases. Infected pancreatic necrosis (IPN) in which necrotic tissue predominates presents early and has higher mortality. Pancreatic abscess consists of pus enclosed by inflammatory walls resulting from infection of liquified necrotic areas. Pancreatic abscess presents after the active phase is over and runs a more indolent course with fewer complications.

Pancreatic pseudocyst prevalence after an attack of acute pancreatitis ranges from 16 to 50% and is more common in alcoholic pancreatitis. Acute pseudocyst consists of an effusion of pancreatic juice rich in amylase that lacks epithelial lining and has become gradually enclosed by fibrous walls after a period of 4 weeks. It is possible that a small ductal leak could initiate cyst formation and seal with passage of time. Most of the pseudocysts resolve spontaneously in 1-2 months, unless they contain large amounts of necrotic material or they get infected. Some of them fistulize into the peritoneal or pericardial or pleural cavity (pancreaticopleural or pericardial fistula) where secondary infection or bleeding can occur.

Colonic complications include mechanical obstruction, bleeding, andcolonic necrosis with perforation and fistula formation. colonic damage is attributed to the direct toxic effect of pancreatic enzymes (pericolitis) or colonic vascular ischaemia (due to DIC, thrombosis of superior mesenteric vein, hypotention).

From the comparison of complications of acute pancreatitis as shown table-15 says that 18.9% of patients in the present study had ascites which ishigher compared to other studies, the rate of pancreatic necrosis was more in other studies as against 5.6% in our study. Organ failure was seen in 6.67% of our patients whereas it was much higher in other studies and this is because most patients in our study had mild disease.

Table-15:Comparison of complications

Complications	Kashid A et al	Buchler MW et al	Present study
Pseudocyst (%)	0	2.45	7.8
Ascites	0	-	18.9
Pancreatic necrosis	18.18	42.15	5.6
Organ failure	29	36.28	6.67
Pancreatic abscess	5.45	0.5	2.2

Duration of hospital stay

The table-16 shows that the mean duration of stay in mild cases being 8.79 days and insevere cases being18.33 days were comparable to other studies.

Table-16:Comparison of duration of hospital stay

Mean Hospital Stay (days)	Kashid A et al	Buchler MW et al	Present study
Mild disease	10	13	8.79
Severe disease	13.5	44.1	18.33

Mortality

Following the observations in table-17 confirms that the mortality rate in our study standing at 3.3% is comparable toKashidA et al[28] and Buchler MW et al[30] studies which are 5.45 and 4.4 respectively.

Table-17: Comparison of mortality

Mortality	Kashid A et al	Buchler MW et al	Present study
Percentage	5.45	4.4	3.3

Summary

This prospective study conducted at SVRRGGH, Tirupathi, included 90 patients with acute pancreatitis, 85 males and 5 females (M:F ~ 6.5:1). The peak incidence was in fourth decade with the mean age of 37.86 years. The commonest etiology was alcohol accounted for 82.6% of cases followedby gall stone disease (6.7%).Pain and vomiting were the commonest presenting complaints. 5 patients had jaundice. Serum amylase and serum lipase together gave high sensitivity (95.5%) 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 12 patients and total parenteral nutrition was given to 6 patients. The mean hospital stay was 12.13 days (Range – 6 to 34 days). Out of 90 patients, 64% had a mild disease while 36% had a severe attack. The overall mortality rate was 3%.

Conclusion

The incidence of acute pancreatitis was found to be in a younger agegroup in our study. Serum Amylase and Lipase both were (95% sensitivity) used for diagnosis wherever 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 specialists in Radiology, Endoscopy and Intensive care unit is essential. Timely intervention by endoscopists and surgeons are crucial to reduce morbidity and mortality. Further attacks should be prevented by early cholecystectomy and avoiding alcohol.

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