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RESEARCH ARTICLE

INCIDENCE, MANAGEMENT AND OUTCOME OF ACUTE PANCREATITIS IN A TERTIARY CARE HOSPITAL IN NCR

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ABSTRACT

Background: Acute pancreatitis is a common disease having wide clinical variations. It may vary in severity from mild self-limiting pancreatic inflammation to pancreatic necrosis which is a life threatening condition causing to multi organ failure syndrome. **Methodology:** We conducted a retrospective study in Rama Medical College Hospital and Research Centre, Pilkhuwa, Hapur in the Department of surgery from April 2017 to April 2019. All the patients with the diagnosis of acute pancreatitis were included in this study. **Results:** The present study was done in the patients who were admitted in the surgery ward with the diagnosis of acute pancreatitis during April 2017 to 2019. Fifty nine patients of acute pancreatitis were there. Out of these female patients were more in number followed by male patients. The foremost cause of acute pancreatitis was gall stone diseases in female patients and alcoholism in male patients. The mean age was 45-55 years in both sexes. Most patients were managed conservatively in high dependency surgical intensive care unit. Severe acute pancreatitis had poor prognosis. Surgical outcome was also not good. **Conclusion:** Acute pancreatitis is more prevalent in female population followed by male. Gall stone pancreatitis was more common in females and alcohol abuse in males. Conservative management was more fruitful than surgical intervention.

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INTRODUCTION

Acute pancreatitis is defined as acute condition presenting with abdominal pain which may be mild, moderate or severe and is usually associated with raised pancreatic enzyme levels in the blood and urine as a result of pancreatic inflammation. The underlying mechanism of injury is thought to be premature activation of pancreatic enzymes within the pancreas leading to autodigestion. Anything that insults the acinar cells of the pancreas increases the secretion of zymogen granules and damage the duct epithelium triggering acute pancreatitis. Once cellular injury has been initiated, it leads to pancreatic oedema, haemorrhage and eventually necrosis. As inflammatory mediators are released into the circulation systemic complications arise such as haemodynamic instability, bacteraemia, acute respiratory distress, pleural effusion, gastrointestinal haemorrhage, renal failure and disseminated intravascular coagulation.

Clinical manifestation: Pain is the cardinal symptom. It develops suddenly reaching in maximum intensity within minutes rather than hours and persists for hours or even days.

The pain is frequently severe, constant and refractory to usual doses of analgesics. Pain initially begins in epigastrium but may be localised to either upper quadrant or felt diffusely throughout the abdomen. There is radiation of pain in the back in about 50% of cases. Some patients get relief by sitting or leaning forwards. The pain is so sudden that it mimics acute perforated peptic ulcer. If pain is maximal in right hypochondrium it resembles acute cholecystitis or biliary colic. Radiation of pain to chest may simulate myocardial infarction, pneumonia or pleurisy. Acute pancreatitis may mimic most causes of acute abdomen. Nausea and vomiting occurs repeatedly. Retching and hiccough may be there and it is persistent and troublesome. It is due to irritation of diaphragm by gastric distension. On examination the patient may be well or gravely ill with profound shock, toxic and confused. Body temperature could be normal, subnormal or elevated. Mild jaundice will be present due to biliary obstruction. A swinging pyrexia suggests cholangitis. Grey Turner's sign and Cullen's sign may be present due to bleeding in fascial planes of the abdomen. There may be distension of abdomen due to ileus or ascites. An inflammatory mass develops in epigastrium after

few days. Guarding and rigidity of abdomen may be there. Pleural effusion is present in some cases. The patient might be confused and exhibit the signs of metabolic derangement with hypoxemia.

Aetiopathogenesis

The aetiology and pathology of acute pancreatitis have been intensively investigated for centuries worldwide. Most descriptive studies attribute various aetiological factors which varies in different populations. There are several factors including alcohol, gall stones, infections, trauma, drugs and hereditary factors. Gall stones are the leading cause which is followed by alcohol; these two constitute about 75% of all cases (Steinberg, 1994). There is always some insult to the pancreas which leads to the cascade of all events leading to acute pancreatitis and its complications. The following causes have been attributed for the causation of acute pancreatitis.

Gall stones: Gall stone pancreatitis is caused by gall stones passing through the bile duct. Stones obstructs the sphincter of Oddi causing increase in pancreatic duct pressure. This causes injury to the acinar cells of pancreas activating the digestive enzymes. In patient with no history of alcohol intake increased levels of serum alanine aminotransferase (ALT) up to three times of its normal value is indicative of gallstone pancreatitis (Diehl, 1997), if having gall stones. Smaller gall stones, microlithiasis or biliary sludge are thought to be at higher risk for gall stone pancreatitis. Microlithiasis causes functional obstruction at sphincter of Oddi, which subsequently results in bile and biliary-pancreatic secretion reflux causing injury to the pancreatic duct (Venneman, 2003 and Garg, 2007).

Alcohol: Alcohol consumption is the second common leading cause of acute pancreatitis. Alcohol damages the acinar cells. It is considered the main mechanism for developing acute pancreatitis in alcoholics. Yadav *et al* reviewed the epidemiology of acute pancreatitis based on reported population based studies from 1966 to 2005. Gall stone pancreatitis was more common in females and alcoholic pancreatitis was more common in middle aged males (Yadav, 2006). Genetic studies have suggested that in hereditary pancreatitis mutation of cationic trypsinogen gene and serine peptidase inhibitor Kazal type 1 (SPINK) gene can promote acute pancreatitis in presence of alcohol (Lucrezio, 2008).

Post-operative: Acute pancreatitis can develop in various intraabdominal surgical procedures. There is trauma to the pancreas due to intraabdominal manipulation. Sometime hypotension develop during the operation leading to pancreatitis. The postoperative pancreatitis has more complication rates.

ERCP and acute pancreatitis: After endoscopic retrograde cholangiopancreatography the incidence of acute pancreatitis is around 5%. This is due to manipulation of biliary ductal system causing injuries to the ducts. There is hyperamylasaemia in about 35-70% cases undergoing ERCP. Most of the time this type of pancreatitis is asymptomatic (Freeman, 1996 and Wang, 2009), and recovery is full.

Trauma: Direct injuries to pancreas leads to acute pancreatitis. The injury may be blunt or penetrating. About 5% of all abdominal trauma lead to acute pancreatitis (Lucrezio, 2008).

Drug induced: Drugs strongly associated with acute pancreatitis are azathioprine, sulphonamides, tetracycline, valproic acid, methyl dopa, oestrogens, furosemide, 6-mercaptopurine, pentamidine, 5-aminosalicylic acid compounds, corticosteroids, sulindac (NSAID), didanosine (for HIV/AIDS) (Balani, 2008).

Infections: Common viral infection that cause acute pancreatitis in children are Epstein- Barr virus, Coxsackie virus, echovirus, varicella-zoster, mumps and measles. Mycoplasma pneumoniae and salmonella also cause acute pancreatitis (Parenti, 1996).

Smoking: Smoking has been directly incriminated for occurrence of acute pancreatitis especially in heavy smokers (Lindkvist, 2008 and Tolstrup, 2009).

Tumours: In approximately 14% of cases different tumours cause acute pancreatitis. These tumours are pancreatic ductal carcinoma, ampullary carcinoma, islets cell tumour, solid pseudo tumour of pancreas, sarcoma, lymphoma, cholangiocarcinoma or metastatic tumours.

Hypercalcemia, hyperlipidaemia, autoimmune pancreatitis and scorpion bite are the other causes of acute pancreatitis.

Investigation

Biochemical investigations: The following are the different biochemical tests which are helpful in diagnosis, management and prognosis of acute pancreatitis.

Serum amylase: The measurement of serum amylase is the gold standard for diagnosis and management of acute pancreatitis. The classical teaching is that a serum amylase level that is three to four times greater than the upper limit of normal is diagnostic of acute pancreatitis.

Serum lipase: In acute pancreatitis the pancreatic enzymes lipase, elastase and trypsin are simultaneously released into the blood stream. The clearance of all these enzymes varies with the onset of acute pancreatitis. The timings of sampling will affects the test's sensitivity (Toouli, 2002). Lipase has the higher diagnostic accuracy as compared to amylase because the serum lipase levels remain elevated for a long time (Matull, 2006).

Serum alanine aminotransferase (ALT): The elevation of alanine aminotransferase more than 150 IU/L is a predictive factor for biliary cause of acute pancreatitis (Ammori, 2003). A previous meta-analysis has indicated that this threefold elevation of alanine aminotransferase has a positive predictive value of 95% for diagnosing the acute gall stone pancreatitis (Tenner, 1994).

Urinary trypsinogen peptide: The biochemical measurement of trypsinogen activation peptide (TAP) and trypsinogen-2 is more useful as a diagnostic marker for acute pancreatitis due to their accuracy but their availability is limited (Matull, 2006). Early elevation of urinary TAP have been shown to be associated with severe acute pancreatitis (Toouli, 2002).

C-Reactive proteins: The CRP is a single reliable and easily accessible marker for assessing the severity of acute pancreatitis. It has demonstrated good prognostic accuracy for

severe acute pancreatitis, pancreatic necrosis and hospital mortality when measured at 48 hours following hospital admission (Cardoso, 2013 and Staubli, 2015).

Other markers: IL-6 and IL-8 as well as phospholipase A2 has been used for diagnosis of severe acute pancreatitis, pancreatic necrosis and in hospital mortality measured at 48 hours after hospital admission (Cardoso, 2013 and Staubli, 2015).

Haematocrit – This is another cheap and easily available parameter indicative of severity of acute pancreatitis. An admission haematocrit > 44% or failure of haematocrit to decrease at 24 hours following admission is indicative of severe acute pancreatitis in the early stage of disease (Berger, 2007). Additionally some studies have demonstrated that haemoconcentration has been associated with the risk of developing necrotising pancreatitis and organ failure (Berger, 2007 and Muddana, 2009), but some refute this (Lankisch, 2001 and Gardner, 2006). The absence of haemoconcentration on admission has a high negative predictive value for the development of necrosis (Lankisch, 2001 and Gardner, 2006).

Imaging: Imaging is also very important tool for the diagnosis of acute pancreatitis.

Ultrasonography: Transabdominal ultrasound is the commonest initial investigation. It has the highest sensitivity for detection of gallstones but it is poor for detection of choledocholithiasis and pancreatitis. Because the pancreas is situated posteriorly and it is difficult to visualize it easily. Overlying bowel gases, large body habitus and abdominal pain make examination more difficult.

CECT: In suspected patient of acute pancreatitis dynamic contrast enhanced CT is the imaging modality of choice. CECT is the important tool for the diagnosis, staging the severity and detection of complications (Balthazar, 1989). The staging of severity and detection of complications depend on the time of CT scanning. In the first 24-48 hours CT finding of necrosis may be equivocal as only 25% patients of acute pancreatitis develop necrosis. Pancreatic necrosis may not develop within the first 48 hours. If the patient is not critically ill the initial CT scan should be obtained at least 72 hours following symptom onset. CTSI (CT severity index) has been devised on the basis of CT findings of pancreatic necrosis. It is used to detect pancreatic lesion, assess its extent and define its aetiology (Whitcomb, 2006). Balthazar EJ *et al* proposed severity index for radiological prognostic scoring system. He initially proposed categories A to E in ascending order of severity, based on the presence of inflammatory changes and proportion of pancreatic necrosis. It has been called CT severity index (CTSI) and used for prognostic purposes. This CTSI is most widely used in clinical settings (Balthazar, 1989). This is very helpful for predicting prognosis and complications of acute pancreatitis.

Magnetic Resonance Imaging: Magnetic resonance imaging in the form of magnetic resonance cholangiopancreatography (MRCP) has become a popular imaging modality for evaluation of the bile duct and pancreatic ducts. MRCP is most reliable in diagnosing choledocholithiasis. Contraindication of MRCP is pace maker and other metal objects in the patient's body. But MRI is not readily available. It is costly and time consuming and so it is not advisable in an acutely ill patient.

Endoscopic ultrasound (EUS) -EUS is the most reliable pretherapeutic diagnostic modality for choledocholithiasis. It is very useful for assessing microlithiasis. Microlithiasis has been attributed a cause of recurrent acute pancreatitis in patients with no evidence of choledocholithiasis. EUS also evaluate ductal abnormalities.

Pathophysiology of acute pancreatitis: There are generally three phases of acute pancreatitis. In first phase there is premature activation of trypsin in the pancreatic acinar cells. This activates other injurious pancreatic enzymes. Then there is intra pancreatic inflammation. In third phase there is extra pancreatic inflammation (Berger, 2007) and necrosis.

Mild acute pancreatitis: In this there is minimal or no organ dysfunction and no parenchymal necrosis. There is prompt and uncomplicated recovery.

Moderate and severe acute pancreatitis: It is defined as when one of the respiratory, cardiovascular or renal failure persist for more than 48 hours (Mitchell, 2003 and Papachristou, 2007 and Buter, 2002).

Several methods have been used to assess severity, prognosis and management of acute pancreatitis. These are

- A. Ranson's criteria
- B. Glasgow score
- C. APACHE II
- D. CECT - As already discussed, this is the method of choice in evaluating the severity and prognosis for acute pancreatitis.

MATERIALS AND METHODS

A retrospective study of patients suffering from acute pancreatitis was conducted in Rama Medical College Hospital and Research Institute, Pilkhwa, Hapur, Uttar Pradesh. We collected two years data from April 2017 to March 2019 from the Department of Surgery. There were 59 cases of acute pancreatitis. The criteria for diagnosis was non-relenting severe abdominal pain with nausea and vomiting, raised serum amylase, raised serum lipase, ultrasound findings and contrast enhanced computed tomographic findings. Detailed history and clinical examination was done. Other relevant laboratory tests were done e.g. haemogram, serum amylase, serum lipase, liver function tests, serum triglycerides, blood urea nitrogen, serum creatinine, blood glucose, lactate dehydrogenase, serum calcium and arterial blood gas analysis. All the patient were managed as per standard guidelines. Patients with mild pancreatitis were treated in the surgical ward, with moderate to severe pancreatitis were managed in Intensive Care Unit. Step up approach and surgery was done in patients who did not improve with intensive medical management.

RESULTS

The following are results of our retrospective study.

Pt diagnosed with acute pancreatitis	
April 2017-March 2018	27
April 2018-March 2019	32
=====	
No. of pts in 2 years	59
=====	

1. Gender distribution:

Female	43
Male	16

2. Age

Female patients: 43

20 - 30 yr.	6
31 - 40	11
41 - 50	23
51 - 60	2
61 - 70	1

Male patients: 16

20 - 30	1
31 - 40	3
41 - 50	5
51 - 60	6
61 - 70	1

3. Cause for acute pancreatitis: 59 patients

Gall stones	41	69.5%
Alcoholism	11	18.64%
Post op ERCP etc.	3	5.0
Post infection	2	3.38
Idiopathic	2	3.38

4. Incidence of severity :59 patients

Mild to moderate acute pancreatitis – 47

Severe acute pancreatitis -- 12

5. Outcome of severe acute pancreatitis

Pseudocyst 7
 ARDS, MODS, pleural effusion, shock 5
 Death -All patients with ARDS, MODS, Open necrosectomy, ascitis died (5patients)

A total number of 59 patients of acute pancreatitis were admitted in Surgery department of Rama Medical College Hospital and Research Centre, Pilkhua, Hapur, Uttar Pradesh. Female patients were more in number about 73% as compared to males 27%. This study shows preponderance of acute pancreatitis in female population due gall stone disease. The mean age was around 45-55yrs. for both male and female patients. This is the most active and productive age during life. The most common cause for acute pancreatitis in males was alcoholism and in female patients it was gall stone diseases. Other causes e.g. post-operative, ERCP, trauma and idiopathic were less in number. Out of 59 patients 47 had mild to moderate disease and 12 were severely afflicted. Out of these 12 patients 5 developed fatal disease and all these patients died. Most of the patients were treated conservatively in high dependency surgical intensive care unit under anaesthesiologist, surgical specialist and physician. Surgical outcome of necrosectomy was poor.

DISCUSSION

The causative aetiology of acute pancreatitis is multifactorial and poorly understood. The mechanisms that trigger the clinical picture of acute pancreatitis is not well known. The responsible enzymatic process and inflammatory response is

still continued to be studied. It is known that alcohol alone does not cause pancreatitis. Many people who consume alcohol do not develop pancreatitis. Gall stone diseases are most common to cause acute pancreatitis in women. Patients with gall stones smaller than 5mm, microlithiasis or biliary sludge are thought to be at higher risk of gall stone pancreatitis. Microlithiasis causes a functional obstruction at sphincter Oddi which subsequently results in reflux of bile and biliary-pancreatic secretions that injures the pancreatic duct (Venneman, 2003). But some does not accept this theory. Other speculate that duodenal content reflux is a more causative factor for pancreatic injury than bile reflux (Sakorafas, 2000). There are multiple theories implicated in the pathogenesis of acute pancreatitis and all remains controversial. The inappropriate release and activation of pancreatic enzymes induce acute pancreatitis. Trypsin activate the pancreatic enzymes. The lack of prompt pancreatic clearance of this active trypsin result in pancreatic inflammation and other harmful processes (Whitcomb, 2006) Cytokines including interleukin (IL)-1, IL-6, IL-8, Tumour necrosis factor a, and platelets-activating factor are released in circulation. These induce the hepatic synthesis of acute phase reaction proteins like C-reactive protein (CRP). Leucocyte migration and activation produce local and systemic complications (Toouli, 2002).

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