INTRODUCTION

Even after more than a century of comprehensive evaluation, 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. Acute pancreatitis is defined as an acute inflammatory process of the pancreas, with variable involvement of other regional tissues or remote organ systems. It may occur as an isolated attack or recur in distinct episodes with reversion to normal histology between attacks. It is distinguished from Chronic Pancreatitis by the absence of continuing inflammation, irreversible structural changes and permanent impairment of exocrine and endocrine function.

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Pancreatitis is a disease, which is unique with protein presentation and difficult to diagnose and manage.

Diagnosis of AP is most often established by clinical symptoms and laboratory testing. Contrast-enhanced computed tomography (CECT) and / or magnetic resonance imaging (MRI) of the pancreas is reserved for patients in whom the diagnosis is unclear or who fail to improve clinically.

It is important to assess the condition of the patient and predict its severity early to minimise the cost of expensive investigations and prevent invasive procedures as a large number of such patients tend to run a benign course. To achieve this a number of scoring procedures have been devised. It thus becomes imperative to study the clinical presentation of AP at time of presentation, impact of investigations, predict the course it would likely to run using various scoring methods, its complications and their outcomes.

Review of Literature

The earliest description of pancreatitis was available only in 1579 by the French Surgeon, Ambrose Pare. Reginald Huber Fitz, a Boston physician and pathologist, in 1889 gave clinical description of acute pancreatitis and reported the pathological findings that allowed him to distinguish hemorrhagic, supplicative, and gangrenous forms of this disease. Prognostication of Acute Pancreatitis was done for the first time in 1974 by Bangalore born, John HCR anson when he was at New York University Medical Center, New York. There were various ill-defined terminologies with regards to Acute Pancreatitis. This lead to a symposium at Atlanta where an universally accepted, clinically based classification system for acute pancreatitis was developed in 1992. It has been noticed in most of the studies that there is an increase in the incidence of the disease by a factor of 10 in the past 3 decades. The reason for the increase is speculated to be due to increase in alcohol abuse and an improved ability to diagnose the disease. The incidence varies from 5.4 to 79.8 per 1,00,000 population and it carries an overall mortality rate of 10–15 %.6 Men are affected much more than women–10 to 30% higher incidence. The reason for male preponderance is probably higher incidence of alcoholic pancreatitis and also because biliary pancreatitis is seen equally in males and females, despite a higher prevalence of gallstones in females. Acute Pancreatitis is related to alcohol or gallstone disease in 80% of cases. The remaining 10 % are related to metabolic factors, drugs and other conditions and10% are idiopathic. The mortality rate approaches 40% in severe cases. The frequency of different forms of pancreatitis varies from source to source and depends on country of origin and the population studied.

The pancreatic juice contains enzymes that are of major importance in digestion and its secretion is controlled in part by a reflex mechanism and in part by gastro intestinal hormones, secretin and CCK. 18 Powerfult protein splitting enzymes of the pancreatic juice are secreted as inactive proenzymes. Trypsin converts Chymotrypsinogens into chymotrypsins and other proenzymes into activezymes. Trypsin can also activate Trypsinogen, therefore once some Trypsin is formed probably by colocolization, there is an autocatalytic chain reaction. The potential danger of the release into pancreas of a small amount of Trypsin is apparent, the resulting chain reaction would produce active enzymes that could digest the pancreas. Another enzyme activated by trypsin is Phospholipase A2. This enzyme splits a fattyacid of lecithin, forming lysosethin. Lysolsethin damages cell membranes. It has been hypothesized that in acute pancreatitis Phospholipase A2 is activated in pancreatic ducts, with the formation of lysosethin from the lecithin that is a normal constituent of bile. This causes disruption of pancreatic tissue and necrosis of surrounding fat.

Small amounts of pancreatic digestive enzymes normally leak into the circulation, but in acute pancreatitis the circulating levels of the digestive enzymes rise markedly. Elevation of serum amylase is observed within 24 hours of the onset of symptoms and gradually returns to normal in the subsequent week. Measurement of plasma amylase or lipase concentration is therefore of value in diagnosing the disease.

Necessity of Objective Stratification

1. To predict the likely course of the disease soon after admission which would be a guide for 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.

Two scoring systems Modified Glasgow criteria and Atlanta classification are being used. Limitation of Glasgow criteria is the need to wait for 48 hours to complete the assessment while Atlanta classification is used to standardise the final diagnosis of the type of Pancreatitis for assessment.

Most cases of AP are managed conservatively but some cases may require minimally invasive procedures such as ERCP.

Surgical treatment is usually reserved for management of local complications

Aims & Objectives

1. To study the clinical presentation and complications of Acute Pancreatitis and their impact on outcome.
2. To compare the Glasgow score with Atlanta score for accuracy to predict prognosis.

MATERIAL AND METHODS

This prospective study was conducted between June 2012 to June 2013 on patients admitted to Hindu Rao Hospital, Delhi. 45 patients with acute pancreatitis were enrolled for the study. 5 patients were excluded since they did not fulfil the diagnostic criteria. Therefore 40 patients with pancreatitis (n =40) were available for analysis.

The Diagnostic Criteria Included 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 C.T. scan suggestive of acute pancreatitis.

Inclusion Criteria

Patients referred to or admitted in the Department of General Surgery and fulfil the diagnostic criteria
Exclusion Criteria
1. Acute episodes in patients of chronic pancreatitis.
2. Patients less than 14 years of age.

On admission a detailed history and a thorough physical examination was done. Data collected on admission included age, sex, address and clinical presentation with respect to pain, vomiting, jaundice and distension of the abdomen. History of etiology with respect to alcohol, gallstones, trauma, and drugs was noted. History of previous episodes and co-morbidities was noted. During the first 48 hours, patients were stratified according to the Glasgow criteria. On discharge or death, patients were stratified into mild or severe according to the Atlanta classification. A comparison between classification of patients by Glasgow score at time of admission and by Atlanta score at time of discharge was noted and the two were then compared.

Data was collected on complications, investigations and interventions undertaken, outcome, duration of stay in hospital and ICU and mode of nutritional support. Prediction of severity by Glasgow criteria was compared with severity stratification by Atlanta classification.

Data was collected in the proforma. Descriptive statistics analysis was carried out in SPSS 17 and graph excel, continuous variables are presented as mean, median. Categorical variables are expressed as frequencies and percentages.

RESULTS
Male predominance (67.5%) with a median age of 39 years was observed. Pain was the most common presenting symptom (93%) followed by vomiting (60%). Other symptoms included fever (20%), abdominal distention (15%), and jaundice (7.5%). 28% of the patients were hypertensive while 20% were diabetic. 47.5% had biliary pancreatitis while 25% had alcohol induced pancreatitis. No cause could be found in 15%. Sr. Lipase supported the diagnosis in 80% while for Sr Amylase it was 52%. CECT had a sensitivity of 100%. 20% had acute fluid collection while 17.5% had acute necrosis. Pleural effusion was seen in 30% of the cases.

On comparing Glasgow score with Atlanta score it was found that Glasgow scores predicted 65% of the patients correctly in mild cases while its predictive value was only 35% in severe cases. ARDS was seen in 15% while ARF in 12.5%. 5% patient died.

Of the 19 patients of Biliary pancreatitis, 12 (84%) underwent cholecystectomy and 4 had ERCP with sphincterotomy. Other surgical procedures performed were abscess drainage and necrosectomy (5%). Mean average stay was 13.5 days in severe cases and 10 days in mild cases.

DISCUSSION
The age and sex distribution found in this study is similar to that found in South England audit and the Edinburgh series. While comparing causes of AP it was gall stones which was the cause in 47.5% which too was similar to the North Indian, Swedish and South UK study. About 35% of the patients had severe disease which was similar to South England study. In our study the Glasgow scores when compared to Atlanta criteria predicted the severity in 30 (75%) cases. The individual values of Glasgow score in our series cannot be given importance or used for correlation of outcome, because all investigations were not done uniformly in all cases. There were many constraints including cost and difficulty in convincing patients to have investigations done when they were improving and planned for discharge within a day or two.

In our study the percentage of patients having local complications in the form of necrosis, infected pancreatic necrosis (IPN) and abscess was higher than that of the South England Audit. This higher number may be due to the higher number of severe cases; 42.27% in our study compared to 32% in the South England Audit. Improvement in management has led to a reduction in mortality rates, particularly in specialized units where technical resources and experienced personnel are available. The overall mortality rate in our series was 5% below the recommended rate of 10% by the U.K. guidelines. The median hospital stay was almost equal in both the studies.

CONCLUSIONS
Acute Pancreatitis is found in younger males usually with a biliary pathology. When both Sr. Amylase and Sr. Lipase are used as investigation the sensitivity is about 80%. All patients should be stratified within 48 hours of admission and this helps in identifying patients who are likely to have a severe attack. These patients may require surgical intervention to manage the cause and complications of the disease and may require ICU management to survive. About 5% patients die despite best possible support. Glasgow scores predicted 65% of the patients correctly in mild cases while its predictive value was only 35% in severe cases. Early management of Gall stones and avoiding alcohol can prevent attacks of AP.

Bibilography