A Retrospective Study on Acute Pancreatitis

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Abstract: Pancreatitis means inflammation of pancreas. It could be better divided into acute and recurrent pancreatitis. It is studied extensively because of grave prognosis in which stand at 9.8 per 100000 of the population. Of all the abdominal emergencies this particular condition carries the highest mortality & morbidity. The etiology of pancreatitis is attributable to many factors like alcohol, gall stones, ductal causes, vascular, trauma both blunt and surgical, autoimmune disease, metabolic, parasitic and many more. In this study the prime etiological factor is alcohol. The individuals in lower socio economic strata are the one most affected. Whatever may be the etiological agent, the action of trypsin a pancreatic enzyme on pancreatic cells sets in motion the auto digestion and inflammatory process leading to acute pancreatitis. The understanding of the etiology is essential to strategies therapeutic measures in managing this condition. Here in this series of 115 patients in and around Madurai district of Tamil Nadu were treated conservatively.

Keywords: pancreatitis, alcohol, inflammation.

I. Introduction

The term ‘Pancreas’ means all flesh no bones. The pancreas is an endocrine and exocrine organ which varies in shape and is located in the upper abdomen behind the stomach. It has got a head, neck, body and tail (Figure 1). The head of the pancreas lies within the curve of the duodenal loop, the tail is at the hilum of spleen, the inferior vena cava and right renal vessels lie posteriorly. The common bile duct receives the main pancreatic duct as it passes through the pancreatic head and then drains into the duodenum at the ampulla. It is about 5.75–9.5 cm long. [2]

Pancreatitis

The inflammation of the pancreas is known as pancreatitis. To start with it is not a bacterial infection causing pancreatitis, it is the enzyme namely trypsin and chymotrypsin brings about auto digestion of acinar cells. Further it is overtaken by bacteria. The other pathological agent as mentioned, work directly or indirectly on the acinar cells.

Mild to moderate forms of pancreatitis will resolve with the treatment in about 80% of the patients. When complications develops 20% mortality is reported (Ref.2). The etiopathogenesishas been widely studied
starting from the theory of bile reflux in to the pancreatic duct which triggers pancreatitis.Opei proposed the theory as bile duct reflux is the main cause (1).

Pathophysiology of pancreatitis
The common pathological event in acute pancreatitis is the early activation of zymogen within the pancreatic parenchyma. The activation of trypsinogen by entericpeptidases to trypsin is important as trypsin converts all proteolytic precursor enzymes like phospholipase, chymotrypsin and elastase to their active forms and activates other cascades such as complement, kinin-kallikrein, coagulation and fibrinolysis signaling cascades.(11,12,16) Patient presents with upper abdominal pain radiating to the back assumes Von munshins position (i.e.) forward bent position to get relief. In the abdomen there is distention initially as a result of ileus followed by pancreatic ascites with prune coloured fluid in the abdomen rich in amylase and lipase. The pancreatic enzymes digests smaller capillaries produce microscopic bleed which leads to peritoneal collection. There is reduction in hemoglobin and fluid volume. Shift of fluid into the third compartment, saponification of mesenteric fat by lipase which in turn become calcium soap thereby results in hypocalcemia, tetany and patchy omental calcifications. Hypokalemia, ECG changes, serum amylase and lipase elevation to thousand fold are the other features (Ref2). Patient goes in to hypovolumic and septic shock followed by multi organ failure and the pancreas undergoes necrosis, phlegmon, diabetes mellitus calcification

Mechanism Of Alcoholic Pancreatitis
Alcohol induced acute pancreatitis usually develops in patients who consume large quantities of alcohol for 5-10 years before the first attack. However, it may occur with the consumption of a small quantity of alcohol also (two drinks/day).Environmental factors like smoking and high-fat diet may also contribute to the development of acute pancreatitis in alcoholics.(12)

There Are Three Possible Different Mechanisms Of Alcoholic Pancreatitis. (Ref.4,16)
1. Obstruction of small ductules by proteinaceous plugs: Chronic alcohol ingestion results in the secretion of protein-rich pancreatic fluid, which may result in inspissated protein plugs and obstruction of small pancreatic ducts.
2. Abnormal spasm sphincter of Oddi: Alcohol transiently increases pancreatic exocrine secretion and abnormal contraction of the sphincter of Oddi (the muscle at the ampulla of Vater).
3. Direct toxic effects: Metabolic byproducts of alcohol have direct toxic effects on the acinar cells.

Clinical Presentation And Diagnosis
The diagnosis of acute pancreatitis is most often established by the presence of 2 of the 3 following criteria:
1) abdominal pain consistent with the disease
2) serum amylase and / or lipase greater than three times the upper limit of normal
3) characteristic findings from abdominal imaging. (Ref.5)

II. Materials And Methods
115 cases of acute pancreatitis in and around from Madurai district of Tamil Nadu were admitted to Madurai Medical College with symptoms of acute pancreatitis over a period of two and a half year between 2012 and 2014. The age group is 15 – 70 years and mean age is 39.92. Among them 112 (97.39%) were male patients only 3 (2.61%) were females, all of them were laborers by occupation. Only 9 (7.8%) of them showed preexisting diabetes. All these patients were non vegetarians. 79 (68.6%) of them were smokers smoking about 20 cigarettes per day for a period of 6 months to 35 years. 98 (85%) of them were consuming alcohol ranging from a period of 2-35 years. The quantity varies from 120 ml to 250 ml per day. None of them had any associated gall bladder disease. 98 (85%) of them had upper abdominal pain, 7 (6.08%) had gaseous distention, 50 (43.4%) of them had vomiting, 5 (4.3%) of them had previous episode of pancreatitis. Amylase ranging from 25to4409 and lipase ranging from 19.7 to 32
In our study 97% of males are affected. Most of them are alcoholic, smoker and non-vegetarian. Females in India hardly consume alcohol.

**Agewise distribution**

<table>
<thead>
<tr>
<th>Age group</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>13 - 20</td>
<td>4</td>
</tr>
<tr>
<td>21 – 30</td>
<td>19</td>
</tr>
<tr>
<td>31 – 40</td>
<td>45</td>
</tr>
<tr>
<td>41 – 50</td>
<td>30</td>
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<tr>
<td>51 – 60</td>
<td>11</td>
</tr>
<tr>
<td>61 – 70</td>
<td>6</td>
</tr>
</tbody>
</table>

**Table.1**

This table shows the age group between 21 to 50 mostly affected by acute pancreatitis. This group is the most hardworking population in our society. So, its impact on our society is unbearable.

**Investigations**

Serum amylase, Lipase, Blood Sugar, HbA1C, Urea, Creatinine, Uric Acid, Calcium, Phosphorous, Total Bilirubin, Total Albumin, Globulin, SGOT, SGPT, ALT, ALP, AST, Protein, Sodium, Bicarbonate, Chloride, Potassium, Cholesterol, Lipid profile, Blood Gas, Acid Base, PCV, TC, DC, WBC, ESR, Platelet, Bleeding time, Clotting time, OGD, Urine routine, Hb, X-Ray Chest, USG, CT, MRCP were done.

**Treatment**

Judicious management of fluids, electrolytes, usage of octerotide, pain relief with opiates along with atropine, antibiotic, total parenteral nutrition and to treat other symptoms when it arrives.

**III. Discussion**

Acute pancreatitis is prevalent in the district of Madurai. Socioeconomic factors play a major role as this disease seems to affects the daily wage earners and with minimal educational background. Acute alcoholic pancreatitis confers a heavy financial burden on the health care system and significant physiologic and economical stress on the patient. It is imperative on the medical fraternity to impress upon the vulnerable section...
of the society of its ill effects of alcohol and other intoxicants. As in the western literature, the bile acts upon the duodenal juice and when it enters the pancreatic duct, it sets in motion of the action of trypsin which digests the pancreatic tissue. When it escapes the boundaries of the pancreas it digests the neighbouring structures. Since, in this particular study, it is proved that alcohol is the main etiological agent in the causation of pancreatitis. Lot of sociological inputs in the society can reduce the incidents of pancreatitis in this particular geological area.

IV. Conclusion

With the better understanding of the pathophysiological conditions of acute pancreatitis the outlook is better and the complications could be minimized. So the crux of the issue is that pin pointing the etiology and critical care management will make the scenario brighter. For the prevention of acute pancreatitis caused especially by alcohol abuse, one has to create a social awareness especially among the culpable section of the society (i.e.) the weaker section. If we could achieve this, to a great extent we can prevent the occurrence of this acute abdominal emergency.

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