Vol 9|Issue 2| 2019 | 147-151.

e-ISSN 2248 – 9142
print-ISSN 2248 – 9134

International Journal of
Current Pharmaceutical & Clinical Research

www.ijcpcr.com

TO STUDY ON ACUTE PANCREATITIS IN SOUTH INDIA.

[1]Mallela Nagi Reddy, T. Syama Sundara[2*]

[1]Associate Professor of Orthopaedics, Sri Lakshmi Narayana Institute of Medical Sciences, (Affiliated to Bharath University, Chennai), Pondicherry, India.
[2]Associate Professor of General Surgery, Sri Lakshmi Narayana Institute of Medical Sciences, (Affiliated to Bharath University, Chennai), Pondicherry, India.

ABSTRACT
Clinically pancreatitis is a 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 ones 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 therapeautic measures in managing this condition. Here in this series of 135 patients in and around pondicherry was treated conservatively.

Key words: Bile duct, Pancreatitis, Inflammation, Fibrinolysis.

INTRODUCTION

The term ‘Pancreas ‘means all flesh no bones. The pancreas is an endocrine and exocrine organ which varies in shape 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 (1-2)

Pancreatitis

The inflammation of the pancreas is known as pancreatitis. To start with it is not a bacterial infection causing pancreatitis, it is the chemical, 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 develop 20% mortality is reported (2-6).

The etiopathogenisis has 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

Corresponding Author: - **Dr. T. Syama Sundara** Email:- syam.t.sundar@gmail.com

all proteolytic precursor enzymes like phospholipase, chymotrypsin, and elastase to their active forms and activates other casecades such as complement, kinin-kallikrein, coagulation and fibrinolysis singnaling cascads.(11-12,16)

Patient presents with upper abdominal pain radiating to the back assumes Von munshins position ie forward bent position to get relief. In the abdomen there is distention initially as a result of ileus followed by pancreatic ascites with prune color 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, suponification of mesenteric fat by lipase which in turn become calcium soap thereby hypocalcemia, tetani and patchy omental calcifications. Hypokalemia, ECG changes, serum amylase and lipase elevation to thousand fold (12-16). 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. (6-12)

There are three possible different mechanisms of alcoholic pancreatitis (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 AP is most oft en established by the presence of 2 of the 3 following criteria: (i) abdominal pain consistent with the disease, (ii) serum amylase and / or lipase greater than three times the upper limit of normal, and / or (iii) characteristic findings from abdominal imaging. (Ref.5)

Materials and methods:

135 cases of acute pancreatitis in and around from Sri Lakshmi Narayana Institute of Medical Sciences with symptoms of acute pancreatitis over a period of two and a half between 2012 and 2014. The age group is 15 – 70 years and mean age is 39.94. Among them 132 (97.38%) were male patients only 3 (2.61%) were females**,** all of them were laborers by occupation. Only 9 (7.8%) of them showed pre existing diabetes. All these patients were non vegetarians. 89 (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. 108 (85%) of them had upper abdominal pain, 7 (6.08%) had gascious distention, 60 (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

Investigations

Amylase, Lipase, Blood Sugar, HB/A1C, 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,

Treatment.

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

Table 1: Agewise distribution

Age group	No. of Patients
13 - 20	**4**
21 – 30	**19**
31 – 40	**55**
41 – 50	**40**
51 – 60	**11**
61 – 70	**6**

Figure 1:

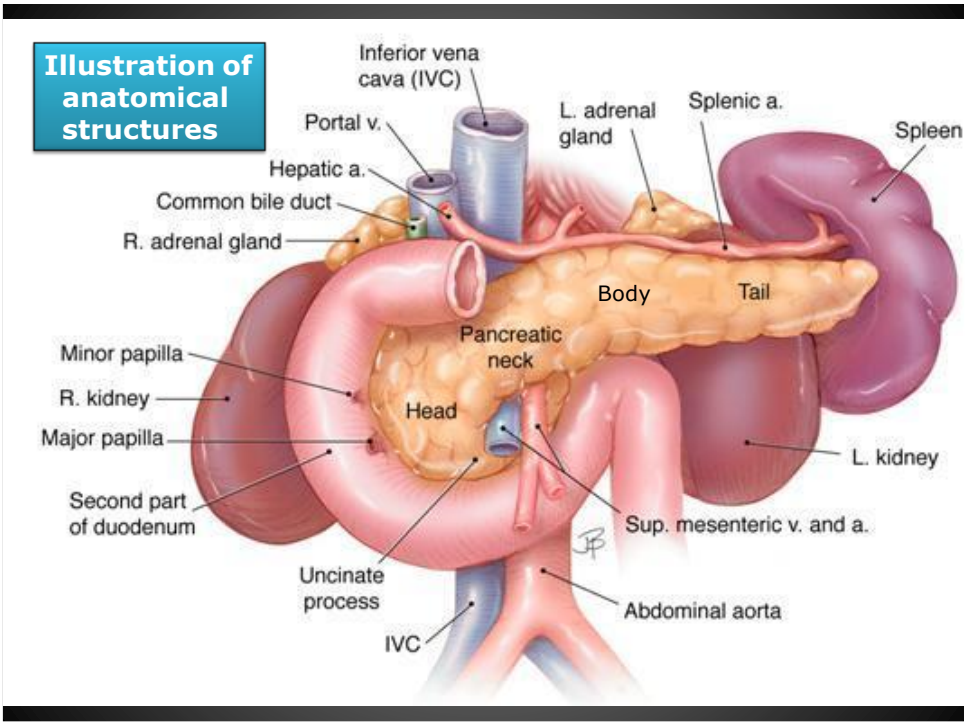
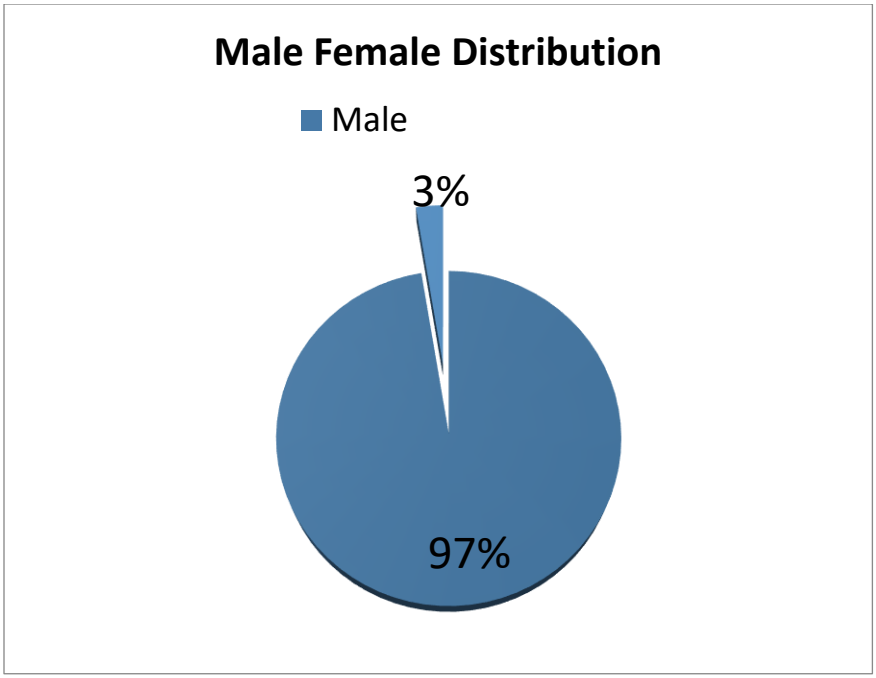


Diagram.1: In our study 97%of males are affected. Most of them are alcoholic, smoker and non vegetarian. Females in India hardly consume alcohol.



Discussion

Acute pancreatitis is prevalent in the district of Pondicherry. 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 neighboring 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.

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 ie the weaker section. If we could achieve this, to a great extent we can prevent the occurrence of this acute abdominal emergency.

REFERENCE:

1. EL Opie; The etiology of acute hemorrhagic pancreatitis. Bull Johns Hopkins Hosp, 12 (1901), pp 182-188
2. Ali Nawaz Khan, John Karani, "Chronic Pancreatitis Imaging". Medscape.
3. New developments in understanding of pathophysiology, diagnosis and treatment of severe acute pancreatitis A. Izakson1, T. Ezri2*, Dana Weiner3, Diana Litmanovich4, E.V. Khankin4 Jurnalul Român de Anestezie Terapie Intensivã 2012 Vol.19 Nr.1, 39-50
4. Pathophysiology of Acute Pancreatitis.Madhav Bhatia Fei Ling Wong Yang Cao Hon Yen Lau Jiali Huang Padmam Puneet Lakshmi Chevali.Department of Pharmacology, National University of Singapore, Singapore. Pancreatology 2005;5:132–144,DOI: 10.1159/000085265.
5. American College of Gastroenterology Guideline:Management of Acute Pancreatit Scott Tenner , MD, MPH, FACG1 , John Baillie , MB, ChB, FRCP, FACG 2 , John DeWi MD, FACG3 and Santhi Swaroop Vege , MD, FACG4, Am J Gastroenterol advance online publication, 30 July 2013; doi: 10.1038/ajg.2013.218.
6. Acute Pancreatitis: Pathophysiology, Clinical Aspects, Diagnosis e Treatme Raffaele Pezzilli Pancreas Unit, Department of Digestive Diseases and Internal Medicine, Sant'Orsola-Malpighi Hospital, Bologna, Italy. lucia.zoppi@pagepress.org
7. In the Clinic Acute Pancreatitis,Annal of internal medicine.americam college of physiciansSection Editors Darren Taichman, MD, PhDBarbara J. Turner MD MPBechiMPHhttp://pier.acponline.org,http://www.acponline.org/products_services/mksap/15/?pr31
8. ACUTE PANCREATITIS Severity Classification,Complications and Outcome, BODIL ANDERSSON, M.D.Sweden 2010 university of lund.
9. Emerging concepts in the pathophysiology of acute pancreatits Mittal,Anubhav: http://hdl.handle.net/2292/6025 https://researchspace.auckland.ac.nz/docs/uoa-docs/rights.htm
10. How We Predict the Etiology of Acute Pancreatitis.
11. Miguel Pérez-Mateo JOP. J Pancreas (Online) 2006; 7(3):257-261. Department of Internal Medicine, Gastroenterology Unit, Hospital General Universitario de Alicante, Miguel Hernández University. Elche, Alicante, Spain. Pathophysiology and Treatment of Acute Pancreatitis:
    New Therapeutic Targets – A Ray of Hope? Peter Felderbauer1, Christophe Müller2, Kerem Bulut1, Orlin Belyaev2, Frank Schmitz1, Waldemar Uhl2 and Wolfgang E. Schmidt1.1.Department of Internal Medicine I, St. Josef-Hospital, Ruhr University, Bochum, and 2Department of Surgery,St. Josef-Hospital, Ruhr University, Bochum, Germany Basic & Clinical Pharmacology & Toxicology 2005, 97, 342–350.
12. Acute Pancreatitis: Etiology, Clinical Presentation, Diagnosis, and Therapy Mitchell S. Cappell, MD,PhD Division of Gastroenterology, Department of Medicine, William Beaumont Hospital, MOB 233, 3535 West Thirteen Mile Road, Royal Oak, MI 48073, USA .Med Clin N Am 92 (2008) 889–923
13. Etiology and diagnosis of acute biliary pancreatitisErwin J. M. van Geenen, Donald L. van der Peet, Pranav Bhagirath, Chris J. J. Mulder and Marco J. Bruno. van Geenen, E. J. M. et al. Nat. Rev. Gastroenterol. Hepatol. 7, 495–502 (2010); published online 10 August 2010; doi:10.1038/nrgastro.2010.114
14. The Clinical Pattern Of Acute Pancreatitis: The Al Kharj Experience. The Internet Journal of Surgery. 2006 Volume 11 Number 1.
15. Update to the Management of Pediatric AcutePancreatitis: Highlighting Areas in Need of Research.Maisam Abu-El-Haija, Tom K. Lin, and Joseph Palermo (JPGN 2014;58: 689–693)
16. Acute Pancreatitis – The Current Concept in Ethiopathogenesis, Morphology and Complications B. Suresh Kumar Shetty1, Ramdas Naik2 , Adithi S. Shetty3,Sharadha Rai4, Ritesh G. Menezes5 and Tanuj Kanchan1 1Department of Forensic Medicine and Toxicology Kasturba Medical College,Mangalore, Manipal University,2Department of Pathology, Kasturba Medical College, Mangalore, Manipal University,3Department of Obstetrics & Gynaecology, K. S. Hegde Medical

Academy,Mangalore, Nitte University,4Department of Pathology, Kasturba Medical College, Mangalore, Manipal University,5Department of Forensic Medicine and Toxicology, Srinivas Institute ofMedical Sciences & Research Centre, Mangalore,India. www.intechopen.com