# *STUDY OF CALCULUS PANCREATITIS*

*Dissertation Submitted for*

**MS Degree (Branch I) General Surgery**

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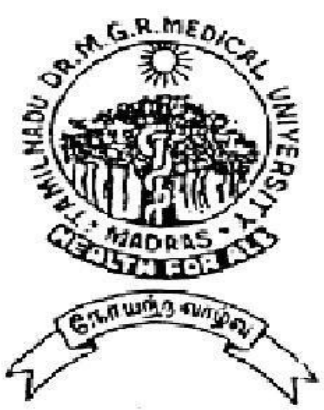


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# CERTIFICATE

This is to certify that this dissertation titled **"*STUDY OF CALCULUS PANCREATITIS*"** submitted by **DR.P.K.PRABU** to the faculty of General Surgery, The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MS degree Branch I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from October 2008 to October 2010.


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# DECLARATION

I**, DR.P.K.PRABU** solemnly declare that the dissertation titled **"*STUDY OF CALCULUS PANCREATITIS*"** has been prepared by me. This is submitted to **The Tamilnadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MS degree (Branch I) General Surgery.

**Place: Madurai** **DR. P.K.PRABU**

**Date:**

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# CONTENTS

# INTRODUCTION

Chronic calcific pancreatitis is a relentlessly progressive fibroinflammatory process, resulting in various amounts of destruction of endocrine and exocrine elements, which may eventually lead to pancreatic insufficiency.

Abdominal pain which is excruciating and recurrent is dominant feature of chronic pancreatitis that initially brings most of the patients to physician's attention. The pathogensis of pancreatic pain is often multifactorial and explains why not all patients respond to same mode of therapy.

In contrast to the quantitatively huge interest stands. The fact that basic problem concerning the disease, the initial steps, the propagation, the mechanisms are still unsolved. This obvious defect in knowledge and understanding of what really going on, when individual get a chronic pancreatitis bears a profound influence on therapeutic approach to the disease.

# AIM OF THE STUDY

- To study the incidence of the chronic calculus pancreatitis at GRH, Madurai
- To study the epidemiological patterns in relation to age, sex and place.
- To study the different etiological factors and pattern of clinical presentations.
- To know the outcome and response of the medical and surgical procedure.

# REVIEW OF LITERATURE

**1. Surgical management of chronic pancreatitis: Long term result in 141 patient. Br.J. Surg 1988.**

**Mannell A; Adson MA ; Mcllrath DC ; Tistrup DM; Department of Surgery Mayo Clinic Rochester, Minnesota**

- 141 patient were operated for chronic pancreatitis at Mayo clinic. The main indication was pancreatic pain and choice of operation was based on anatomical abnormalities in the gland.
- Mean follow up period — 8.5 years
- Number of patient operated — 141 patients

- **Conclusion**

  — 77% of operated patient had lasting pain relief

  — Longitudinal pancreatico jejunostomy in those with dilated duct and whipple operation for disease of pancreatic head gave good results.

**2. Local resection of the head of the pancreas combined with longitudinal pancreatico-jejunostomy in the management of patients with chronic pancreatitis.**

**Ann Surg 1994, Frey CF : Amikura K, Department of S. rgery, University of California, Davis Medical Centre, Sacramento.**

**Material -**The operation was performed on 50 patients, pain relief, endocrine and exocrine insufficiency and weight gain were assessed.

**Conclusion -** The LR — LPJ provides good pain relief with modest increase in endocrine and exocrine insufficiency and a significant increase in weight. Even when relieved of pain, patients seldom return to the work force.

**3. Pain relapse in the first 10 years of chronic pancreatitis, An JSurg 1996.**

**Talamani G ; Bassi C, Falconi ; Sartonin ; Salvia R DiFrancesco V; Frulloni L; Vaona B ; Bovo P; Vanfin I; Pederzoli P; Cavallini G. Department of Medicine University Verora, Italy.**

To evaluate whether the annual number of pain relapse of chronic pancreatitis correlated with sex, type of pancreatitis, drinking and smoking and type of surgery.

Length of follow up - 10 yrs

Number of patients - 2,034 / year

Conclusion - Regardless of surgical treatment patients should be advised to reduce both their alcohol intake and cigarette smoking.

**4. Chronic Pancreatitis, Paul Georg Lankisch MD, sur Opin Gastroenterology 2007.**

Abstract: Purpose of Review is to focuss on the most important new observations in chronic pancreatitis.

**Recent findings —**

- Superiority of Surgery compared with endotherapy for long term pain relief.
- Smoking enhances the risk of chronic pancreatitis
- New insights in Autoimmune pancreatitis

**Study:**

Amsterdam group conducted Randomized trial, Comparing endoscopic and surgical drainage of the pancreatic duct.

39- patients participated

19— endoscopic treatment

20— surgical drainage procedure

Follow up period —24 months

**Result:**

Complete or partial pain relief

32% - Endoscopic group

75% - Surgery group

**5. Surgical Management of CP:**

**Gourgiotis S, Germanos S, Ridolfini MP, Department of hepatobiliary and pancreatic surgery. Royal London Hospital London, UK. Hepatobilary pancreatic Dis mt 2007 Apr 6.**

**Background:**

During the last decade increasing knowledge about pathophysiology of CP, improved results of major pancreatic resections and integration of sophisticated diagnostic methods in clinical practice resulted in significant changes in surgery for CP.

**Conclusion:**

Surgical procedure provide long term pain relief a good post operative quality of life with preservation of endocrine and exocrine function. In addition to available results from randomized controlled trials, new studies are needed to determine which procedure is most effective for the management of patient with CP.

**6. Extended drainage versus resection in Surgery for CP: A prospective randomized trail comparing LPJ + Local pancreatic head excision with pylons preserving panereatoduodenectomy.**

**Ann Sug 1998, Dee, Izbicki JR, Bloechle C, Broering DC, Knoefel WT, Kuechler T, Broelsch CL Department of Surgery University of Hamburg, Germany.**

To analyse the efficacy of LPJ — LPHI & PPPD

Number of patients —61 patients were randomly allocated

**Conclusion:** - Both procedures are equally effective in terms of pain relief and definitive control of complications affecting adjacent organ, but extended drainage procedure provides better quality of life.

**7. Comparative RCT between endoscopic treatment with surgical treatment in painful obstructive CP. Dite et al, endoscopy 2003.**

Number of patients - 140

Surgery - 80% Resection, 20% drainage

Endoscopy — 52% sphincterotomy and

Stenting, 23% stone removal,

Follow up period —5 yrs

**Conclusion:**

Pain relief after surgery is superior to endotherapy in patient with painful obstructive CP.

**8. Chronic pancreatitis : A prospective nationwide study of 1086 subjects from India**

**Balakrishnan V, Unnikrishnan AG, Thomas V, Choidhini Veera raju P, Singh IP, Garg P, Pai CG, Devi RN, Bhasin D, Iayanthi D, Premalatha N, Chacko A..., Amitha Institute of 4edical Science, Cochin India, Sep 2008: pub Med.**

**Study :** Prospective nationwide study of risk factors and clinical profile of Chrome pancreatitis thing: 32 major centres from all over India contributed data on 1086 patients.

**Outcome Measures:** Risk factors, Clinical features, implications and treatment of Chronic pancreatitis.

**Conclusion:** In this first nationwide prospective study of Chronic pancreatitis in India, Idiopathic Chronic pancreatitis was the most common form, followed by alcoholic pancreatitis. The classical form of tropical Chronic pancreatitis is becoming less common.

# HISTORY OF PANCREATIC ANATOMY

The pancreas was first mentioned in the writing of Eristratos (310-250 BC) and given its name by Rufus of Ephesus (Crica 1040). The name pancreas (Greak Pan- all, kreas-flesh almost) was used because the organ contain neither cartilage or bone. Its main duct was described by Wirsung in 1642, where the enlargement of the duct at its junction with the CBD and its projection in to the duodenum as a papilla were first described by Vater in 1720. Santorini in 1734 described the accessory duct that bears his name. It was only after demonstration of digestive enzymes by Claude Bernard in 1850 that the pancreas became a complete organ with an important function and thus a worthy object of study.

Inspite of the apparent accessibility of the pancreas, a number of complex relations combines to make its surgical removal difficult. In 1899 Halsted was first to successfully remove the head of pancreas and portion of duodenum for ampullary carcinoma. Several surgeons developed two staged operations, for removal of head of pancreas. These efforts culminated in 1940 with one stage operation of whipple.

Sir Andrew Watt kay wrote in 1978, “For me, the tiger country is removal of the pancreas. The anatomy is very complex and one counters anomalies”.

**Location:**

The pancreas lies posterior to the stomach and lesser omentum in the retroperitoneum of the upper abdomen. It extends obliquely rising slightly as it passes from the medial edge of duodenal C loop to the hilum of the spleen. It lies anterior to the inferior venacava, aorta, splenic vein and left adrenal gland.

**Regions:**

Pancreas is divided into four regions. The head and uncinate process, neck, body and tail. The head lies within the duodenal loop and its uncinate process extends posteriorly and medially to lie behind the Superior mesenteric vein and SMA. The neck of the gland extends medially from the head to lie anterior to these vessels. The body extends laterally from the neck toward the spleen, where as the tail extends into the splenic hilum.

**Blood supply and Lymphnodes:**

Both the celiac trunk and the SMA provide the arterial supply to the pancreas. Variations are common, but for the most part, the body and tail are supplied by branch of the splenic artery, where as head and uncinate process receive their supply through arcade originating form the hepatic and gastroduodenal branch of celiac artery and from the first branch of the SMA.

Venous drainage is to the splenic vein, SMV and portal vein. The pancreas is drained by multiple lymph node groups. The major drainage of pancreatic head and uncinate process is to the subpyloric, portal, mesenteric, mesocolic and aortocaval node. The pancreatic body and tail for the most part are drained through nodes in the celiac, aortocaval, mesenteric and mesocolic groups and through nodes in the splenic hilum.

**Innervation:**

The pancreas is innervated by both sympathetic and para sympathetic components of the autonornic nervous system. The principal and possibly only, pathway for pancreatic pain involves nociceptive fibers arising in the pancreas. They pass through the celiac ganglia to form the

greater, lesser and least splanchnic nerves that pass to cell bodies in the thoracic sympathetic chain. Efferent visceral motor supply to the pancreas is provided by both the sympathetic and parasympathetic systems. The latter involves preganglionic fibers arising form cell bodies in the vagal nuclei that travel through the posterior vagal trunk to the celiac plexus. Postganglionic fibers then innervate pancreatic islets, acini, ducts and blood vessels. In general, the nerves of the pancreas travel with the blood vessels supplying the organ.

**Ducts:**

The main pancreatic duct, or duct of Wirsung arising in the tail of the pancreas and terminates at the papilla of vater in the duodenum. It crosses the vertebral column between T12 and L2. Within the body and tail of the pancreas, the duct lies slightly caphaled to a line drawn midway between the superior and inferior edges. The duct is also more posterior than anterior. In adults, the duct within the head measures 3.1 to 4.8 mm in diameter and gradually tapers to measure 0.9 to 2.4 mm in the tail. With age, the duct diameter can increase. The duct of santorini i.e. The minor or accessory pancreatic duct is smaller than the main duct. It extends from the main duct to enter the duodenum at the lesser papilla.

That Papilla lies about 2 cm proximal and slightly anterior to the major papilla.

## EMBRYOLOGY

### Organagenesis:

During the 4th week of gestation, two endodermal buds arise from the duodenum ; the hepatic diverticulum, which is desired to form the liver, gall bladder and bile ducts, and the dorsal pancreatic bud that forms the body and tail of the pancreas. On the 32nd day of gestation, this hepatic diverticulum gives rise to a ventral pancreatic bud that eventually develops into the uncinate process and inferior part of the head of the pancreas. The dorsal pancreatic bud extends transversely across the abdomen to lies anterior to the portal and mesenteric vessels. With time and as the duodenum rotates to form a C loop configuration the ventral pancreas and distal bile duct undergo clockwise rotation around the back of duodenum to finally, lie on the medial side of the duodenum, inferior and slightly posterior to the dorsal pancreas and posterior to the portal and mesenteric vessels. On the 37th day of gestation, the two pancreatic buds fuse and in 90% of individuals, their duct system also join.

## HISTOLOGY

The mature pancreas is an endocrine organ made up of the islets of Langerhans and an exocrine organ consisting of acinar and ductal cells. The acinar cells, so named because they are clustered like grapes on the stem of a vine, discharge their secretions into a centrally located acinar space that communicates with the major pancreatic duct. Most of the cells in the pancreas are acinar cells and duct cells make up only 5% of pancreatic mass. Histologically, acinar cells have a high content of endoplasmic reticulum and an abundance of apically located eosinophilic zymogen granules. The cells lining the main pancreatic duct are table columnar cells and many contain mucin granules. With progression form the large ducts to the smaller intralobular and interlobular ducts the lining cells become flatter, assuming a cuboidal configuration, and the mucin granules are no longer seen. Centroacinar cells located at the junction between ducts and acini resemble acinar cells in size and shape but lacks Zymogen granules.

# PHYSIOLOGY

About 2.5 liters of clear, colorless, bicarbonate-rich pancreatic juice, containing 6 to 20g of protein, is secreted by the human pancreas each day. It plays a critical role in duodenal alkalinization and in food digestion.

**Protein Secretion:**

With the possible exception of the lactating mammary gland, the exocrine pancreas synthesizes protein at a greater rate, per gram of tissue, than any other organ. More than 90% of that protein consists of digestive enzymes. Most of the digestive enzymes are synthesized and secreted by acinar cells as inactive proenzymes or zymogens that, in health, are activated only after they reach the duodenum where enterokinase activates trypsinogen and the trypsin catalyses the activation of the other zymogens. Some of the pancreatic digestive enzymes are synthesized and secreted in their active forms without the need for an activation step (eg. Amylase, lipase, ribonuclease). Acinar cells also synthesize proteins, including enzymes, that are not destined for secretion but, rather, are intended for use within the acinar cell itself. Examples of this latter group

of proteins include the various structural proteins and lysosomal hydrolases.

Newly synthesized proteins are assembled within the cisternae of the rough endopiasmic reticulum and transported to the Golgi, where they are modified by glycosylation. Those destined for secretion pass through the Golgi stacks and are packaged within condensing vacuoles that evolve into zymogen granules as they migrate toward the luminal surface of the acinar cell. By a process involving membrane fusion and fission, the contents of the zymogen granules are then released into the acinar lumen. Other proteins that are not destined for secretion are segregated away from the secretory pathway as they pass through the Golgi, and they are then targeted to their appropriate intracellular site.

Secretion of protein from acinar cells is a regulated process. At rest, secretion occurs at a low or basal rate, but this rate can be markedly increased by secretory stimulation that, in the pancreas, is both hormonal and neural. Pancreatic acinar cells can express receptors for acetylcholine. Cholecystokinin, secretin, and vasoactive intestinal peptide. Stimulation of secretion by either acetylcholine or cholecystokinin has been shown to involve activation of phospholipase C, generation of inositol triphosphate

and diacyl glycerol, and a rise in intracellular ionized Calcium levels that, by yet unidentified mechanisms, upregulates the raw of secretory protein discharge at the apical cell membrane. In contrast. secretion and vasoactive intestinal peptide activate adenylate cyciase, increase cellular levels of cyclic adenosine monophosphat (AMP), and activate protein kinase A. This also leads to protein secretion at the apical pole. Recent studies indicate that human acinar cells may not possess receptors for cholecystokinin and that in humans, cholecystokinin stimulation of secretion is mediated by intrapancreatic nerves that express cholecystokinin receptors.

**Electrolyte Secretion:**

Although stimulation of acinar cells results in the secretion of a small amount of serum like fluid, most of the fluid and electrolytes secreted from the pancreas arise from duct cells. The earliest step in duct cell electrolyte secretion involves diffusion of circulating carbon dioxide into the duct cell, and that carbon dioxide is hydrated by carbonic anhydrase to yield carbonic acid. Subsequently, the carbonic acid dissociates into protons and bicarbonates ions. The protons diffuse out of the cell and are carried away in the circulation while the bicarbonate

remains inside the cell. The fluid and electrolyte secretagogue sectretion acts, through a cyclic AMP mediated process, to stimulate chloride secretion, at the apical cell surface, through cystic fibrosis transmembrane regulator (chloride) channels. Then, through an apical chloride-bicarbonate exchanger, the actively secreted chloride is taken up again by the duct cell in exchange for bicarbonate. Taken together the result of these events is the secretion of a bicarbonate rich fluid into the duct and the discharge, into the circulation, of protons. In the absence of secretin stimulation, pancreatic juice has a more plasma like composition because it is composed primarily of acinar cell secretions and there is little duct cell secretion of chloride to permit exchange with bicarbonate. With secretin stimulation, chloride secretion is increased, flow rates rise, and chloride bicarbonate exchange results in juice that is rich in bicarbonate and poor in chloride.

**Integrated Physiology:**

During the resting (inter digestive) phase of gastrointestinal function, pancreatic secretion is minimal and may be as low as 2% of that noted with maximal stimulation. The pancreatic response to a meal is a three phase process that includes a cephalic phase, a gastric phase, and an

intestinal phase. The cephalic phase, accounting for 10% to 15°/o of meal stimulated pancreatic secretion, reflects the response to the sight, smell, or taste of food. It is believed to be almost exclusively mediated by peripherally relased acetycholine, which directly stimulates pancreatic secretion of enzymes and gastric secretion of acid. The acid indirectly stimulates pancreatic secretion of fluid and electrolytes by causing duodenal acidification and secretion release. The gastric phase of pancreatic secretion, accounting for 10% to 15% of meal- stimulated pancreatic secretion reflects the response to gastric distention and the entry of food into the stomach. These events can cause release of gastrin and stimulate vagal afferents. By binding to cholecystokinin receptors, gastrin is itself a weak stimulant of pancreatic enzyme secretion. Vagal stimulation also increases enzyme secretion.

More important, however gastrin and vagal stimulation cause gastric acid secretion, and this leads to duodenal acidification, release of secretion from the duodenum, and pancreatic secretion of fluid and electrolytes. The intestinal phase of pancreatic secretion reflects the response to food and gastric secretions entering the proximal intestine. Acidification of the duodenum and the presence of bile in the duodenum

promote secretin release. In addition, in the duodenum and proximal small intestine, the presence of fat and protein, as well as their partial breakdown products, stimulates the release of cholecystokinin, and this cholecystokinin stimulates enzyme secretion from acinar cells. The intestinal phase of pancreatic secretion accounts for 70% to 75% of meal stimulated pancreatic secretion.

# PANCREATIC CALCULI

Though the incidence of pancreatic calculi is still generally considered to be quite low, the reports of the last 15 - 20 years indicate that this condition is much more common than is generally appreciated. The ease with which the condition can be recognized and the possibilities for relief by present-day surgical measures make it quite worth while to bear this disease in mind in the differential diagnosis of abdominal complaints)

The etiology of this disease is not clear. While many have called attention to the biliary tract disease which is associated in some cases 32 most patients show no such associated involvement. The possibility that the pancreatic disease may be secondary to biliary tract involvement seems unlikely, therefore. The consideration of reflux of bile into the pancreatic ductal system as a causative factor also is not widely credited. The calculi are made up almost entirely of calcium carbonate and calcium phosphate; there are occasionally minor additions of organic matter. These salts are not normal constituents of pancreatic juice.

The primary factor in the development of pancreatic calculi is that of an alteration in the composition of the external secretion of the gland. This may result from infection from any source—blood, lymphatic or ductal. A secondary factor of stasis also probably plays a role, but simple ligation of the duct in an experimental animal does not uniformly produce calculi. Attacks of pancreatitis may give rise to areas of calcification in the pancreas but are not the source of most calculi.

Pancreatic calculi are totally different from biliary calculi. Instead, they resemble very closely those seen in the salivary glands and ducts. They are whitish or grayish-white in color and are very hard, rough and horny in contour. They are rarely faceted even though multiple. They may vary in size from that of a grain of sand up to 2.5 inches in diameter, and may weigh as much as 200Gm[31]. Though solitary calculi have been found, usually there are multiple stones, up to several hundred in number. Their most common location is in the head of the pancreas, with decreasing frequency toward the tail, though in cases with multiple stones the entire gland may be studded with calculi.

Some observers have distinguished two types of pancreatic lithiasis— a diffuse calcification within the gland and calcifications

limited to the larger ducts only. Many of the former actually represent cases with hundreds of calculi, most of which will of necessity occupy the smallest ducts and, there- fore, appear at first glance to lie within the substance of the gland itself.

Though certain small series have given conflicting reports as to sex incidence, it is now well-established that the disease predominates in the male in ratio of 3 or 4 to I. The disease has been reported at the ages of 14 to 72 years, but occurs most commonly in the 4th and 5th decades. An occasional case is asymptomatic and is discovered quite by chance. The common symptom, however, is that of pain. This pain typically is severe epigastric colic resembling that seen in obstruction of the cystic duct of gallbladder. The pain is usually located more in the midline, however, and often radiates to the left upper quadrant and back and occasionally also to the scapula and shoulder. The colic is often severe enough to require opiates relief, although at times the pain is of a duller and more constant nature. As in biliary colic, nausea and vomiting are frequent accompaniments of pancreatic colic. Weight loss is very common and some show frank emaciation and cachexia. Many are alcoholics. This may

represent an attempt to obtain release from the pain rather than a precipitating factor in the development of calcifications.

As a result of obstruction to pancreatic duct by the calculi which precipitate the attack, pancreatic enzymes cannot reach the intestinal tract. This interference may lead to changes in the stools characterized by frequent, pale, frothy, bulky stools containing increased amounts of fat. Also, there is an increase in the amounts of undigested meat fibers in the stools. Many have reported making the diagnosis of pancreatic lithiasis by observing the passage of calculi in the stools following an attack of colic. The calculi often become impacted in the region of the ampulla of Vater. This may lead also to obstruction of the common bile duct; therefore, jaundice has been seen in many cases of pancreatic colic. With recurrent attacks of obstruction, the pancreas gradually undergoes atrophy and develops degeneration of the parenchyma. The first part of the gland to show this change is the acinar tissue. Only after some time are the islet cells also destroyed.

Oser was able to find 24 of 70 cases (34 per cent) of pancreatic calculi showing diabetes. Other authors have reported an incidence of 40 per cent or more. A transient glycosuria may occur during an attack.

Many cases show a diabetic type of glucose tolerance curve even though they do not spill sugar in the urine. These alterations in sugar tolerance at first are limited to the acute attack, but as damage to the pancreas progresses, and permanent changes set in, the diabetic tendency also remains constant. An improvement in the glucose tolerance curve and a disappearance of glycosuria has often followed operative intervention for pancreatic calculi. Painful diabetes should be a warning to search for pancreatic stones. In advanced stages of degeneration of the pancreas secondary to calculi producing obstruction, fatty changes of the liver often develop, apparently the result of loss of lipocaic from the destruction of the pancreas.

Occasionally a mass develops in the upper abdomen during an acute attack as a result of obstruction of the pancreatic duct. in some the pancreas may be outlined as a firm mass, perhaps somewhat tender. Fever, chills and leukocytosis may or may not accompany an attack. The disturbance of sugar metabolism has been discussed above. Elevation of the serum amylase and lipase and urinary diastase during an acute attack has been recorded.

Aside from the history and the character of the pain, by far the most important diagnostic measure available is that of roentgenologic examination. This should consist of a survey film of the abdomen, including both anteroposterior and lateral or oblique views. The pancreas lies transversely in the region of the first to third lumbar vertebrae, the tail rising much higher than the head, up to the 10th to 12th dorsal vertebrae. The lateral view confirms the position of the calcifications in relation to the spinal column. Since these stones are almost pure calcium they cast a denser shadow than most biliary tract stones. However, they may also be confused with renal calculi, calcified mesenteric lymph nodes and calcified plaques in the splenic vessels or aorta. A cholecystogram and a urogram or pyelogram should aid in establishing the location of the calcifications with relation to the biliary and urinary tracts.

Complications of pancreatic calculi—aside from the effects produced by interference with enzyme and hormone production—include the development of cysts, abscesses, carcinoma and hemorrhage. The cysts may vary in size with acute attacks. The abscesses may present on the abdominal wall or rupture spontaneously into surrounding viscera. Occasionally a stone has eroded into a vessel large enough to give rise to

severe or even fatal hemorrhage into the gastro-intestinal tract. A small percentage of cases show malignant change, but this does not necessarily bear any relation to the presence of the calculi.

# ETIOLOGY OF CHRONIC CALCIFIC PANCREATITIS

Newer classification systems, such as the TIGAR-O, categorize chronic pancreatitis based on the various known etiologic factors and mechanisms that are jointly considered risk modifiers (TIGAR-O) toxic, metabolic, idiopathic, genetic, autoimmune, recurrent severe, obstructive.

We discuss the various causes of chronic pancreatitis based on the TIGAR-O system.

Multiple toxic and metabolic etiologies involved in chronic pancreatitis. The association of alcohol and chronic pancreatitis was first described by COMFORT and associates in 1946. Alcohol still the most common cause of chronic pancreatitis in Western industrialized countries, but only 5% to 10% of alcoholics develop clinically apparent chronic pancreatitis, and at autopsy 10% to 20% of alcoholics are found to have evidence of chronic pancreatitis.

Because only a fraction of alcoholics develop chronic pancreatitis, involvements of other factors are actively being investigated. Several

evidence have shown that in addition to direct effects of alcohol, various predisposing factors, including genetics, smoking, intestinal infection, high fat diet, compromised immune function, gallstones, gender, hormonal factors and drinking patterns may render the pancreas more susceptible to alcohol induced tissue injury.

Smoking also is independently associated with increased risk for chronic pancreatitis. Chronic pancreatitis induced by smoking is particularly associated with pancreatic calcification. By mechanisms similar to alcohol, tobacco produces alterations in the secretion and composition of pancreatic juice mainly as a result of decreased pancreatic juice and bicarbonate secretion and induction of oxidative stress.

Calcium plays a central role in trypsinogen secretion and trypsin stabilization. Hypercalcemia caused by primary or secondary hyperparathyroidism results in recurrent acute pancreatitis, which progresses to chronic pancreatitis, likely owing to trypsinogen activation, which results in necrosis and fibrosis of the parenchyma.

Increased serum calcium concentration is believed to induce direct damage to acinar cells, and increased secretion of calcium results in

intraductal stone formation. Hypercalcemia also seems to modify pancreatic secretion, leading to protein plug formation.

**Idiopathic:**

Thirty percent of patients with chronic pancreatitis do not have known risk factors for chronic pancreatitis and are considered to have idiopathic pancreatitis. Mutations of the serine protease inhibitor, Kazal type 1 (spink 1) gene in 25% of patients with idiopathic chronic pancreatitis. Based on the bimodal age of onset of the clinical symptoms, idiopathic pancreatitis is separated into two distinct entities. Early onset idiopathic chronic pancreatitis presents during the first 2 decades of life with abdominal pain being the predominant clinical feature, whereas pancreatic calcifications and exocrine and endocrine pancreatic insufficiency are rare at the time of first diagnosis.

In contrast, the clinical presentation of late onset idiopathic chronic pancreatitis is in patients in their 40s, usually following a rather painless course, but associated with significant exocrine and endocrine pancreatic insufficiency and pancreatic calcifications.

Tropical or nutritional pancreatitis is considered a form of idiopathic chronic pancreatitis. It is the most common form of chronic pancreatitis in certain parts of the world, such as India, sub-Saharan Africa, and Brazil, and affects children and young adults (Schneider et al 2002). The disease is subdivided into tropical calcific pancreatitis, which is characterized by severe recurrent and chronic abnormal pain and extensive pancreatic calcifications, and fibrocalculous pancreatic diabetes, which is characterized by significant pancreatic endocrine insufficiency This form of chronic pancreatitis is related to mutations in the SPINK 1 gene.

Strong association between cystic fibrosis transmembrane conductance regular (CFTR) mutations and idiopathic chronic pancreatitis. One third of all patients with idiopathic chronic pancreatitis have CFTR mutations.

Leading pancreatologists speculate that most chronic pancreatitis might be a genetic disease with multi factorial triggering factors.

**Genetic**

Until more recently few data existed on the genetic basis of chronic pancreatitis. The only known hereditary form of chronic pancreatic insufficiency that was well studied was cystic fibrosis.

Research has focused on the SPJNKI-N34S gene mutation, which also is associated closely with tropical (50%), alcoholic (6%), or idiopathic (20%) c chronic pancreatitis.

One of the major discoveries in chronic pancreatitis was the description of the point mutation in patients with autosomal dominant hereditary pancreatitis. Several variants of the mutation of the cationic trypsinogen gene all lead to a malfunction of trypsinogen. Hereditary pancreatitis presents typically in a bimodal pattern of childhood and adulthood. Hereditary pancreatitis is an autosornal dominant disease associated with trypsinogen gene mutations that carries an 80% penetrance.

Despite great advances in the knowledge of genetics in pancreatitis, currently it is advised to evaluate for mutations only in patients with hereditary pancreatitis.

**Autoimmune**

Autoimmune chronic pancreatitis (AIP) is a rare but distinct form of chronic pancreatitis that is associated with autoimmune features. AIP is characterized by specific histopathologic and immunologic features. The morphologic hall marks are periductal infiltration by lymphocytes and plasma cells and granulocytic epithelial lesions with consequent destruction of the duct epithelium and venulitis.

The pathogenesis of AIP values a cellular CD4+ and CD8+ T cell) and humoral immune mediated attack of the ductal cells and pancreatic ducts resulting in cytokine mediated inflammation and periductular fibrosis, which leads to obstruction of the pancreatic ducts.

AIP is characterized clinically by minimal abdominal pain and diffuse enlargement of the pancreas without calcifications or pseudocysts.

On laboratory examination, these patients have hypergammaglobulinemia and autoantibodies, such as antinuclear and anti-smooth muscle antibodies.

**Obstructive:**

Obstruction of the main pancreatic duct is well known to result in chronic pancreatitis. The most common etiologies include scars of the pancreatic duct, tumors of the ampulla of Vater and head of the pancreas, and trauma.

Main pancreatic duct obstruction may lead to stagnation and stone formation of pancreatic juice (stone and duct obstruction theory) or acute recurrent pancreatitis and periductular fibrosis (necrosis fibrosis theory) Histopathologic characteristics of human chronic pancreatitis resulting from obstruction include uniform distribution of interlobular and intralobular fibrosis and marked destruction of the exocrine parenchyma in the territory of obstruction, without significant protein plugs and calcifications.

Chronic pancreatitis results from plugging of the pancreatic duct. The origin of chronic pancreatitis was within the lumen of the pancreatic ductules in contrast to the origins of acute pancreatitis which tends to be inside the acinar cell. Increased lithogenicity of pancreatic fluid leads to

the formation of eosinophilic proteinaceous aggregates, which precipitate and obstruct the pancreatic ductules.

Alcohol decreases the formation and the secretion of pancreatic juice, making it more viscous; low in bicarbonate ; rich in protein, enzymes and caiclium crystals ; and deficient in lithostatin.

Alcohol also has been shown to mediate the release of gastrointestinal hormones by increasing cholecystokinin releasing factor, which affects pancreatic juice formation and flow. The pancreatic stones and plugs are believed to produce ulceration of the ductal epithelial cells resulting in inflammation, fibrosis. obstruction, stasis and further stone formation.

Mechanism of chronic pancreatitis was a dysregulation and overactivity of the hepatic mixed function oxidases leading to oxidative stress. This theory places the acinar cell at the major area of injury by oxidative stress, usually as a result of steady exposure of xenoiotics that induce the cytochrome P-450 enzymatic system, while depleting glutathione.

Pancreatitis is triggered through interference of the methionine to glutathione transsulfuration pathway, resulting in diversion of free radicals in to the pancreatic tissue, with consequent activation of inflammation and fibrosis of the ductules with low flow of pancreatic juice, inhibition of lithostatin, and precipitation of proteins and calcium (Braganza 1998 ; Wilson et al, 1990). Alcohol also may contribute to increase the oxidative stress resulting from depletion of scavengers, such as selenium, vitamin E and C and riboflavin, and help to induce or propagate the damage.

Alcohol and its toxic metabolites cause accumulation of intracellular lipids and fatty acid ethyl esters, which produce damage to the acinar cell. The alterations of intracellular lipid metabolism lead to fatty degeneration, apoptosis and scarring of the pancreatic parenchyma with impairment of the pancreatic microcirculation.

It was shown that these fat cells exists in the human pancreas, can migrate into the periacinar spaces, and are activated by alcohol and acetyl aldehyde, transforming into scar producing cells. The necrosis fibrosis hypothesis views the development and course of chronic pancreatitis as a consequence of severe pancreatitis, emphasizing that fibrosis is a late

development resulting from repeated attacks of acute (alcoholic) pancreatitis, which initially lead to inflammation and necrosis.

The necrosis fibrosis hypothesis has significant supporting evidence from epidemiologic and large follow up studies, which showed that chronic pancreatitis results from recurrent attacks of acute pancreatitis.

The recurrent attacks of acute pancreatitis in hereditary pancreatitis also support the necrosis fibrosis hypothesis. One important aspect that partially negates this hypothesis is the fact that the type of fibrosis that follows acute attacks of pancreatitis involves short lived collagen type III and procollagen type IV and not the long lasting collagen types I and IV (Casini Ct al 2000).

The primary pathogenic factor leading to chronic pancreatitis is an outflow obstruction likely resulting from duct inflammation, destruction and fibrosis which likely are the result of an immunologic attacks on a specific genetic, structural or acquired antigen of the periductular epithelium. The target of this attack may be some specific genetic or acquired antigen on the duct epithelium.

Chronic pancreatitis seems to be an autoimmune or duct destroying disease, analogous to primary sclerosing cholangitis. The assumption is supported by several observations, such as the radiologic and histologic similarity of chronic pancreatitis and primary sclerosing cholangitis, the activation of cytotoxic T.lymphocytes in the periductular areas of the pancreas in patients with alcoholic chronic pancreatitis, and the occasional association of chronic pancreatitis and primary scierosing cholangitis.

The SAPE hypothesis tries to provide a “final common pathway” for the many etiologies for pancreatitis. The basic aspect is that there needs to be susceptibility (genetic or through ongoing insult, such as alcohol toxicity). The critical sentinel event appears and triggers the process causing acute and chronic pancreatitis. Further activation of the immunologic system and the stellate cells propagates chronic pancreatitis, and the end result is fibrosis and calcifications.

This hypothesis has the merits of placing several of the previous theories under “one umbrella”.

# MATERIALS AND METHODS

This prospective study of calculus pancreatitis was conducted in 77 patients admitted in GRH, Madurai, General Surgery and Surgical Gastroentereology department from 2008 to 2010.

Informed consent was obtained from all patient who were included in the study.

**Inclusion Criteria:**

Study group include both male and females between the age group of 13 to 60 yrs with signs symptoms and radiological evidence of chronic calculus pancreatitis.

**Exclusion criteria:**

Patient with chronic pancreatitis associated malignancy were excluded from the study.

**Study Design:**

Each patient in the study was subjected to detailed clinical examination correlating with a detail history. Investigations in the form of routine

hemogram, Liver Function Test, Pancreatic Function Test, and imaging studies like X rays, USG, CT abdomen and other investigations relevant to the suspected disease system involved were done. From the above clinical data and imaging studies chronic pancreatitis was diagnosed. Patients who requires surgical intervention were prepared and taken up for surgical procedure after satisfying the inclusion and exclusion criteria. The results were tabulated and analysed.

# ANALYSIS

## AGE DISTRIBUTION

**Table : 1**

| Age Group ( Years) | No. of Cases | Percentage |
|---|---|---|
| 1-13 | - | - |
| 13-23 | 16 | 20.77% |
| 24-33 | 15 | 19.42% |
| 34-43 | 25 | 32.46% |
| 44-60 | 21 | 15.58% |
| Total | 77 | 100% |

## SEX DISTRIBUTION:

**Table -2**

| Sex | No. of cases | Percentage |
|---|---|---|
| Male | 62 | 81% |
| Female | 15 | 19% |
| Total | 77 | 100% |

Male : Female = 4:1

## CLINICAL DATA

**Table -3**

| Clinical Data | No.of cases | Percentage |
|---|---|---|
| Pain Abdomen | 69 | 90% |
| Diabetes | 36 | 47% |
| Steatorrhea | 52 | 68% |
| Weight loss | 66 | 85% |
| Alcoholic | 24 | 31% |
| Jaundice | 3 | 4% |

## SURGICAL OUTCOME:

### Table – 4

| i) Pain Relief | No. of cases | Percentage |
|---|---|---|
| Yes | 53 | 69% |
| No | 20 | 36.4% |
| ii) Diabetic Improved | 9 | 10% |
| iii) Exocrine insufficiency | - | 0% |
| iv) Relapses | 5 | 7% |

## CLINICAL FEATURES – CHRONIC PANCREATITIS

**Table – 5**

| | |
|---|---|
| No. of Patients | 77 |
| Mean Age | 35 |
| Sex (M:F) | 4.1 |
| Pain | 90% |
| Diabetes | 47% |
| Steatorrhea | 68% |

# DISCUSSION

**Age Distribution:**

The youngest patient in this study was 13 years old female and oldest patient was 60 years old male. Most of the patients presented in the $4^{th}$ decade of life in this study group i.e 30 to 40 years (33%).

This current study correlates with the study conducted by E.L.Eliason etal who also found that pancreatic calculus occurred commonly is patients between 30-40 yrs.

**Sex Distribution:**

Males are affected more than female patient in the ratio of 4.1 in this study group. Which correlates with E.L.Eliason etal study with male to female ration of 4:1.

**Etiological distribution:**

In this study (31%) of the patients are alcoholic. The etiological factor in this group of patients are undetermined that may be attributed to nutritional, idiopathic, hereditary etc.

Chronic alcoholism is one of the important etiological factor in chronic pancreatitis. Alcohol abuse also affects the clinical feature, course and prognosis of disease. In upto 70% of adult patient chronic pancreatitis appears to be caused by alcoholism. This form is more common in men than women between age of 30 to 40.

Heriditary pancreatitis usually begins in childhood but may not be diagnosed for several years A determining factor is two or more family members with pancreatitis in more than one generation.

Idiopathic chronic pancreatitis was the most common from followed by alcoholic pancreatitis in India. **This was published by Amritha Institute of Medical Science, Cochin, India. (chronic pancreatitis : prospective nationwide study of 1086 subjects from India on Sep 2008 in pub-med.)**

**CLINICAL PRESENTATION:**

In my study, most of the chronic pancreatitis patients had principal symptom of abdominal pain radiating to back or to left. Most of the patients were emaciated. Most of them were chronic alcoholic some were with diabetes mellitus. Repeated pain attack is characteristic of chronic

pancreatitis Pain free intervals becomes shorter and the pain eventually occurs everyday. Pain is mainly due to increased intraductal pressure upto 30 to 50cm of H20. (Normal upto 20cm of H20).

Potential cause of pain is chronic pancreatitis

1. Pancreatic ductal hypertension

2. Inflammation of intrapancreatic nerves

3. Loss of protective perineural sheath in pancreatic nerves

4 Pancreatic ischemia

5. Pseudocyst

6. Pancreatic & peripancreatic inflammation

7. Cholangitis.

This current study correlates with the E.L.Eliason and Robert F.Welty etal study shows most common clinical presentation of pancreatic calculus was abdominal pain 90%.

Vomiting is the next commonest symptom and is frequently described. Bilious vomiting with increased salivary flow.

Eating may increase the pain, so many patients avoid food and lose weight. Significant exocrine insufficiency with occur if 90% of secretory capacity of pancreas is lost. The major consequences are steatorrhoea and creatorrhoea. They may complain of bulk, offensive, fatty and oily stools.

Jaundice was present 11 of 59 operative cases studied (14%) and tend to recur with each attack.

Associated gallstones were present in 2 cases (3%) which argues against initial disease of billary tract as being the main cause of pancreatic stone.

Glycosuria is reported in 36 of the 77 cases. It is especially likely to occur with attacks being absent in the intervals. It is more common in the case of long standing where considerable fibrosis of the gland has taken place.

**Diagnosis of chronic calcific pancreatitis:**

In this study diagnosis of chronic pancreatitis is based on the thorough history, physical examination laboratory data or imaging abnormalities. Imaging methods done in this study were plain abdominal radiographs which revealed the presence of focal or diffuse pancreatic calcifications in

25 to 30% of the cases, transabdominal ultrasound which showed pancreatic duct dilatation irregularly of main pancreatic duct, loss or reduction of pancreatic parenchymal echogenicity, calculi and calcifications. CT scan abdomen findings showed pancreatic duct dilation, calcification and cystic lesions.

Imaging, modalities like ERCP and EUS are more sensitive and specific in diagnosis of chronic pancreatitis. They are expensive and the cost factor and non availability remains a constrain.

Diagnosis of chronic pancreatitis is based on a thorough history and physical examination, laboratory data and imaging studies. Today pancreatic function tests play a minor and only complementary role in the diagnosis of chronic pancreatitis. The two main reasons for this minor role are that

i) Non invasive test of exocrine pancreatic function show high sensitivity only in advanced stage of chronic pancreatitis.

ii) Clinical manifestations of an exocrine pancreatic insufficiency occurs late in the course of disease after approx 90% of exocrine parenchyma is destroyed.

**Imaging methods:**

Non invasive imaging methods are method of choice for diagnosis of chronic pancreatitis in clinical situations. Currently ERCP is still the "Gold standard" among the all imaging methods. But in the future it may be replaced by further significant refinement of magnetic resonance imaging cholangiography.

**Plain Abdominal Radiography:**

Shows focal or diffuse pancreatic calcifications in 30 to 40% of cases makes the diagnosis of advanced chronic pancreatitis.

Transabdominal USG is an essential tool to visualize the entire pancreas. It is inexpensive, simple, noninvasive. widely distributed, well tolerated and often first imaging method in patient with abdominal complaint. In routine clinical situation, IJSG is the easiest method to detect the complication of chronic pancreatitis and to follow patients with chronic pancreatitis. Use of ultrasound for diagnosing chronic pancreatitis is limited to advanced stage.

CT scan is as specific as ultrasound but more sensitive. CT scan cannot detect early parenchymal changes and effects on small pancreatic

ducts, but advanced stages and complications of the disease can be evaluated with high reliability. CT is most sensitive to detect calculi. Chronic pancreatitis is excellent method to detect advanced stage but not for early stage of chronic pancreatitis.

ERCP is still “gold standard” imaging modality. An ERCP staging system based in pancreatic ductal changes has been developed for diagnosis of chronic pancreatitis which was published in 1984 as the Cambridge criteria. Changes of early chronic pancreatitis may not be seen on ERCP. ERCP is invasive method (post ERCP pancreatitis of 3 to 7%), expensive, specialised equipment and trained personal are necessary to perform the produce and to interpret the pancreatograms. ERCP may be useful in distinguishing chronic pancreatitis from pancreatic cancer. The advantage of ERCP are standardization and evaluation method in multi center trials and possibility of intervention. The advantages are complications, costs and invasiveness.

Magnetic Resonance Imaging pancreatography an imaging method is created that enables clinicians to visualize ductal of chronic pancreatitis. The advantage of this modality is noninvasiveness. The major disadvantage is that changes of side branches are not visualized

with same accuracy as in ERCP and not sensitive to detect early stages of chronic pancreatitis.

Endoscopic ultrasound visualize the pancreatic duct and the parenchyma and has the ability to detect chronic pancreatitis in patients with early stages of the disease and with advanced chronic pancreatitis. Major advantage of EUS when compared to other imaging modalities is that its ability to detect early stages of chronic pancreatitis without any complications. The disadvantage of this method is need for expert EUS endoscopist and dedicated EUS unit.

A few diagnostic criteria of pancreatic stones are listed briefly:

1) Colic – like pain referred to the back or left epigastrium.
2) Slight preponderance in males.
3) Average age of patients around 40.
4) Long intervals between attacks.
5) Bilious vomiting and increased increased salivary flow.
6) Midepigastric tenderness.

7) Steatorrhea when present.

8) Relative absence of jaundice ; present in 27.6 per cent.

9) Expulsion of calcium carborate or phosphate stones in the stools.

10) Moderate loss of weight and strength.

11) Glycosuria when present.

12) Typical appearance of stones roentgenologically.

In my study, 1 have come across 77 cases of chronic calcific pancreatitis of which 59 cases are of obstructive type. The course of obstruction of pancreatic duct in my study was mainly due to calculi :-

2 cases with pseudocyst

2 cases with gall stone

Remaining 16 cases with diffuse calcification of pancreas.

**MANAGEMENT:**

The treatment of pancreatic calculi is surgical removal except where these is diffuse calcification throughout entire pancreas. In my study about 16 cases presents with diffuse calcification of entire pancreas those cases planned for non – operative management.

**Non operative management of chronic pancreatitis are**

1. Control of abdominal pain

2. Treatment of endocrine insufficiency

3. Treatment of exocrine insufficiency

**Control of abdominal pain**

- Advise to stop alcohol intake (about 50% of patients has some pain relief when they stop led alcohol)
- Advise to consume semi solid or liquid diets instead of solids,
- Carbohydrate more
- Fat & protein less
- H2 receptor antagonists

- Oral pancreatic enzyme supplements.
- Parentral somatostatin analogue to inhibit pancreatic secretion e.g.octreortide
- Attempts to control pain often require early use of non narcotic analgesics followed later by narcotic analgesics.

**Treatment of exocrine insufficiency**

1 Dietary restriction of fat is important.

2. Pancreatic enzyme replacement eg. cotazym, ilozyme, viokase

3. H2 receptor blocking agent

**Treatment of endocrine insufficiency**

1. Mild elevations of blood sugar do not require treatment fasting level >250 mgm/dl should be managed with insulin.

2. Maintain the fasting level around 200 mgm/dl

**Surgical Treatment**

In this study, out of 77 patients of chronic calculus pancreatitis, 59 patient presented with calculi in the pancreatic duct. All 59 patients were treated surgically, The different procedures are as follows.

| | |
|---|---|
| Pancreaticolithotomy followed y Roux-en-Y pancreaticojejunostomy and jejunojejunostomy | - 50 cases |
| Pancreaticolithotomy with cholecystectomy followed y Roux-en-Y pancreaticojejunostomy and jejunojejunostomy | - 1 Case |
| Frey's Procedure | - 3 cases |
| Frey's with cholecystectomy | - 1 case |
| Denervation Procedure | - 2 cases |

The primary goal of operative management is relief of pain, the secondary consideration is to preserve maximal endocrine and exocrine function. prior to surgical intervention ERCP &CT study is must to study the ductal anatomy

1. Drainage procedure — for dilated duct
2. Pancreatic resection — for Normal (or) Narrow duct

**Drainage procedure (pancreatico jejunostomy)**

The main pancreatic duct has a normal diameter of 4 to 5 mm in the head, 3 to 4 mm in the body and 2 to 3mm in the tail. if the diameter is more than 7 to 8 mm in body and head, a pancreatico jejunostomy (modified puestow) is technically feasible with adequate stroma length is 6-10cm.

**Modified puestow's procedure**

A longitudinal pacreatomy typically discloses segmental stenosis of the pancreatic duct and the presence of intraductal calculi in a patient with chronic calcific pancreatitis . Following mobilization of a Roux limb of jejunum , a longitudinal pancreatico – jejunostomy is performed to permit extensive drainage of pancreatic duct system.

In this current study correlate with prinz etal showed lateral pancreatico – jejunostomy better procedure for chronic calculus pancreatitis and has good results in pain relief post operatively to 70% in this study group

1. Operative mortality is about 0%

2. Drain will close spontaneously

3. Patient may gain weight

4. Pain relieved in about 80 to 85% patients

5. Sometimes stenosis of pancreaticojejunal anastomoss may occur, then pancreatic resection is advisable.

**Frey's procedure:**

This procedure done for those patients pancreatic calculus associated with dominant mass in the head of the pancreas.

The extended lateral pancreaticojejunostomy with excavation of pancreatic head provides complete decompression of the entire pancreatic ductal system. Reconstruction is performed with side to side Roux – en – Y pancreaticojejunostomy.

In this current study correlates with Anderson and Topazin study et al showed that Frey's procedure may provide better protection against recurrent stenosis.

**Denervation procedure:**

In patients who have persistent and diabling pain, but who are poor candidates for resection or drainage procedures, denervation procedure provides symptomatic relief.

In our study, denervation procedure was done for 2 patients on operative, celiac ganglionectomy or splanchninectomy.

**Methods:**

1. Neural ablation to block afferent sympathetic nociceptive pathways.
2. Direct infiltration of the celiac ganglia with long action analgesies or neurolytic agents.
3. Operative celiac ganglionectomy or splanchninectomy.
4. Transhiatal splanchninectomy.
5. Transthoracic splanchnic with or without vagotomy.
6. Videoscopic transthoracic splanchninectomy.

**Pancreatic resections**

1. Pancreatico duodenectomy
2. Pylorus preserving pancreatico duodenectomy
3. Pancreatic Head resection
4. Distal pancreatectomy

The main indication of pancreatic resection for a pancreatitis patient is

1. To relief pain

2. Failure of drainage procedure

3. When the pathological changes involve one part of the gland and the rest is less diseased.

4. When the diagnosis between chronic pancreatitis and pancreatic cancer is in doubt

**PSEUDOCYST OF PANCREAS**

In my study, 2 cases pseudocyst of pancreas, associated with pancreatic calculus. All the patients were chronic alcoholic. They presented with

abdominal pain, vomiting and upper abdomen mass. Two patients presented with enlarged GB with signs of obstructive jaundice.

All the patients had raised serum amylase leveL Ranging from 426 to 910 U/L Few patients show elevated alkaline phosphatase and serum bilirubin and elevated SGOT and SGPT levels. CT scan was useful in assessing the age of the cyst, ERCP can define the pancreatic ductal anatomy and cyst-duct communication. In some patients OGD was also done, OGD showed extra luminal impression of the stomach due to a lesion situated posterior to the stomach.

Surgical treatment depends on the size, duration of the cyst, maturity of the cyst wall and presence (or) absence of infection of cyst contents.

- Cystogastrostomy for a cyst of size 20x15cm situated in the region of Head of pancreas.
- Cystojejunostomy for a cyst of size of 15x15cm situated in the region of tail of pancreas.

**Post operative complications**

In my study bcidience of wound infection about – 11%.

In my study the incidence of Pancreatic Fistula is about 9%

Fistulae following the removal of pancreatic calculi usually close within a few weeks (or) months. In our case drainage averaged about 300 cc. daily over a period of five months, with exception of a week during which the fistula was apparently closed. There is usually little avtodigestion of the skin.

Craft has shown expeximentally that ephedrine definitely reduced the output of external pancreatic secretion. This is due to its vasoconstricting and that fact that the pancreas is very sensitive to changes in the volume of blood flow.

A diet rich in fats andproteins and low in carbohydrates to aid in reducing pancreatic secretion. Clinical reports shown as almost equal difference of opinion regarding the effectiveness of diet and alkalis in reducing the total output of the fistula. In our case there was no appreciable reduction during the period that the patient was taking this diet.

# FOLLOW UP STUDY

In the patients followed up, the current study shows:

1. 70% of the patients had better pain relief.
2. 9% of the patient had improvement in diabetic status.
3. 6.5% of the patient had relapse.
4. No patient had improvement in exocrine deficiency.

# CONCLUSION

In this prospective study which was conducted in 77 patients with chronic calculus pancreatitis at Government Rajaji Hospital, Madurai between 2008-2011 the following conclusions were derived out.

1. Panacreatic calculi occur much more commonly than is generally appreciated.
2. Most of patients presented in their $4^{th}$ decade of life.
3. Males are affected more than females in the ratio of 4:1
4. In this study 31% of the patients are alcoholic the etiological factor in the remaining group of patients are undertermined, that may be attributed to nutritional, idiopathic, hereditary etc.
5. Surgical relief can be obtained, and this is the procedure of choice in severe cases.
6. Decompression surgery done in cases with ductal dillation more than 7mm.

7. Modified Puestow's (PARTINGTON – ROCHELLE) is the most commonly performed surgical drainage procedure and has good results in pain and relief post – operatively to 69% in this study group.

8. Small duct disease resection (or) denervation procedure can be done.

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# PROFORMA FOR A STUDY OF CALCULUS

# PANCREATITIS

NAME : OCCUPATION:

AGE : DATE OF ADMINSSION :

SEX : DATE OF DISCHARGE :

## CLINCAL PRESENTATION

H/O. Jaundice – Progressive / Non Progressive

H/o. Pain Abdomen

H/o. Alcoholism

H/o. Vomiting

H/o. Weight Less

INVESTIGATIONS

Urine – Bile Salts Haemoglobin

Heamatocrit

Bile pigments Blood grouping

Blood – Urea BT

Sugar CT

Serum Creatinine

Serum amylase

LFT

**RADIOLOGICAL INVESTIGATIONS**

Plain X-ray Abdomen :

Ultra Sound :

CT Scan :

ERCP :

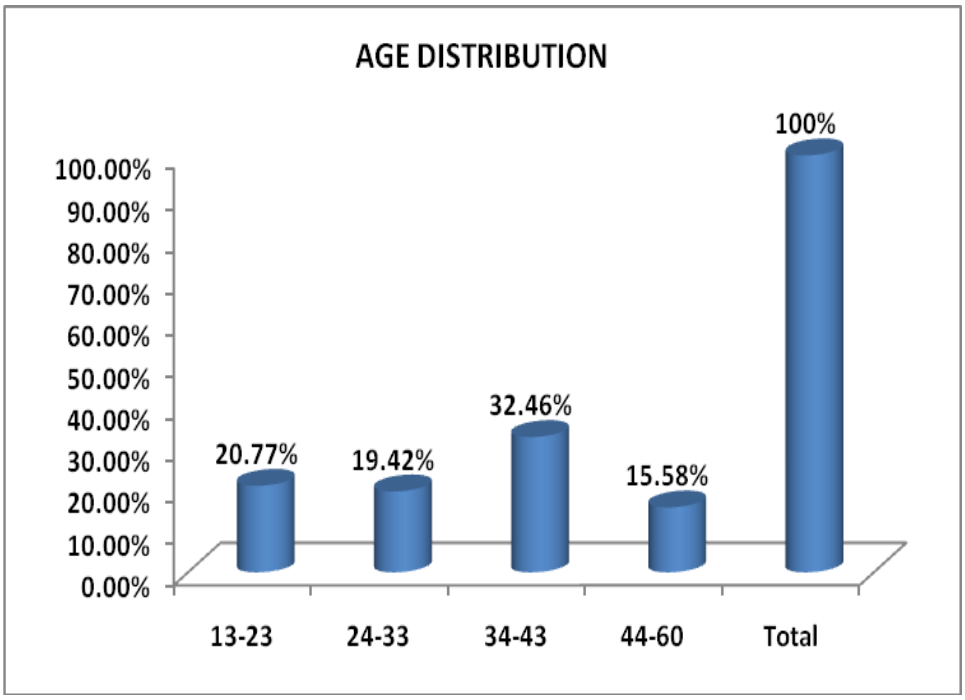
MRCP :

Operative Findings :

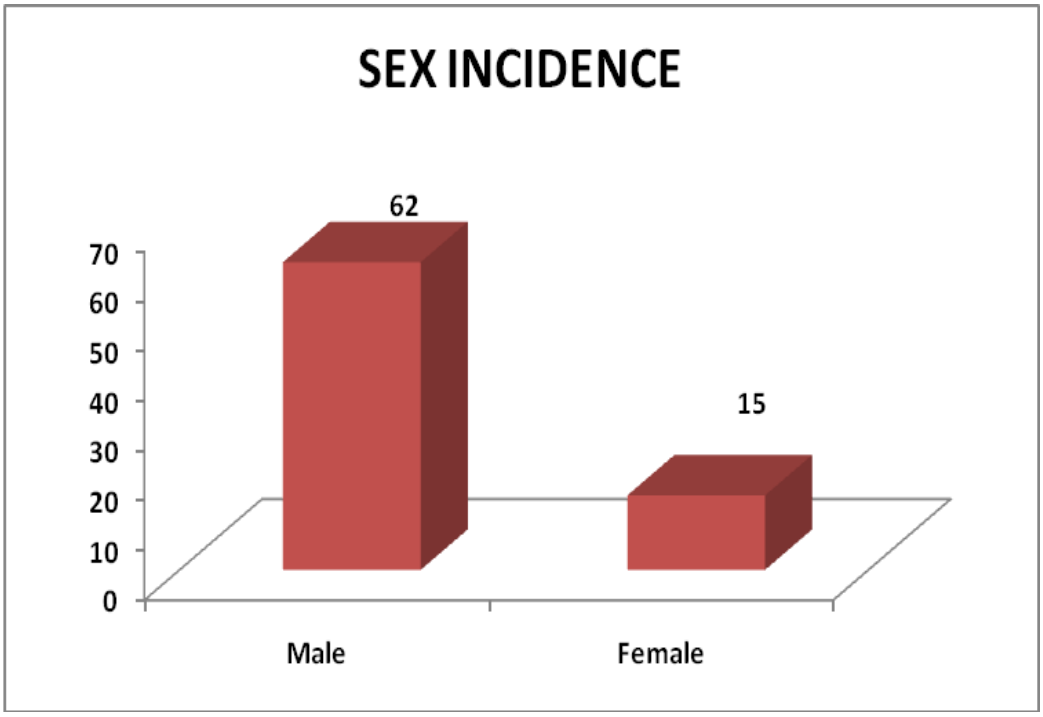
Procedure Done :

Post Operative Period :

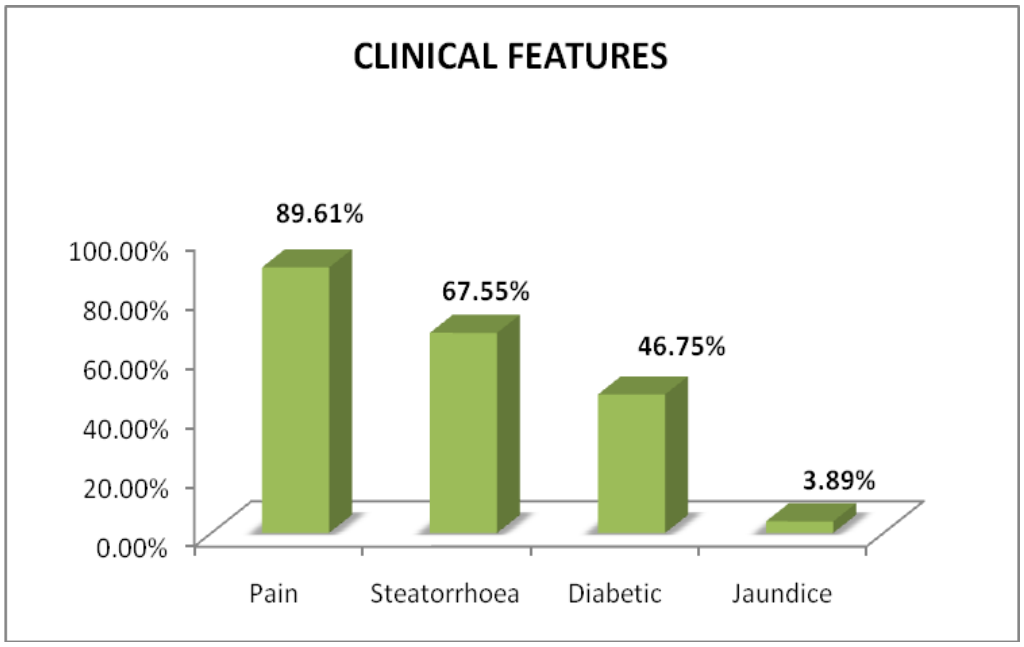
Complication and its Management :




The youngest patient in this study was 13 years old female and oldest patient was 60 years old male. Most of the patients presented in the fourth decade of life in this study group 30 to 40 years (33%).




Males are affected four times more than female patients.



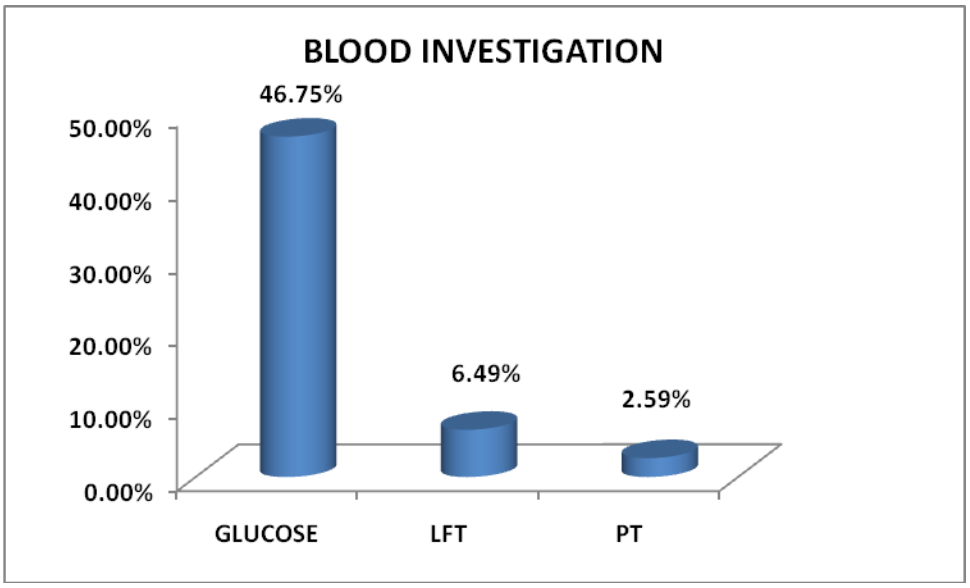

Pain - 89.61%

Steatorrhoea - 67.55%

Diabetic - 46.75%

Jaundice - 3.89%

In my study, most of the chronic clacific pancreatitis patients had principal symptom of abdominal pain (90%).



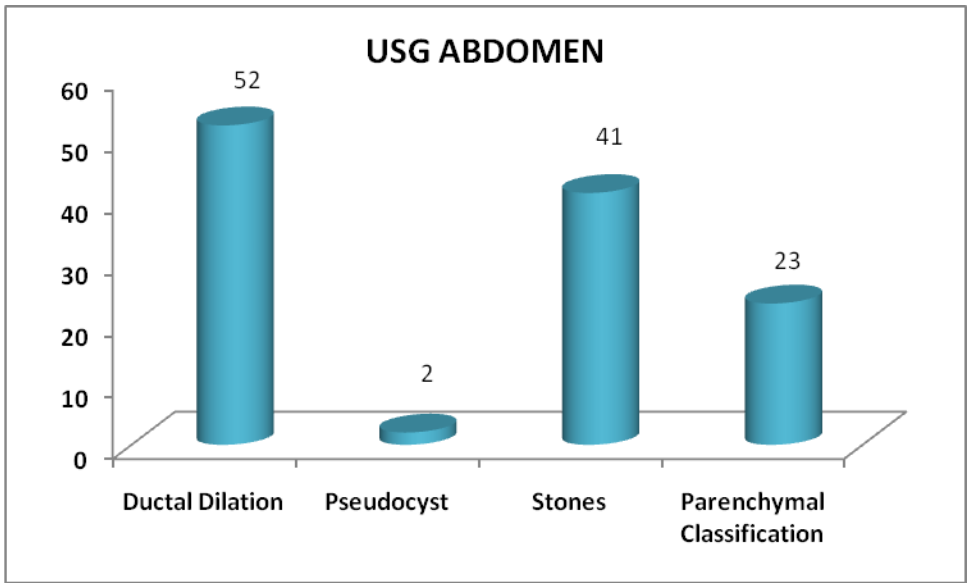

GLUCOSE - 46.75%

LFT - 6.49%

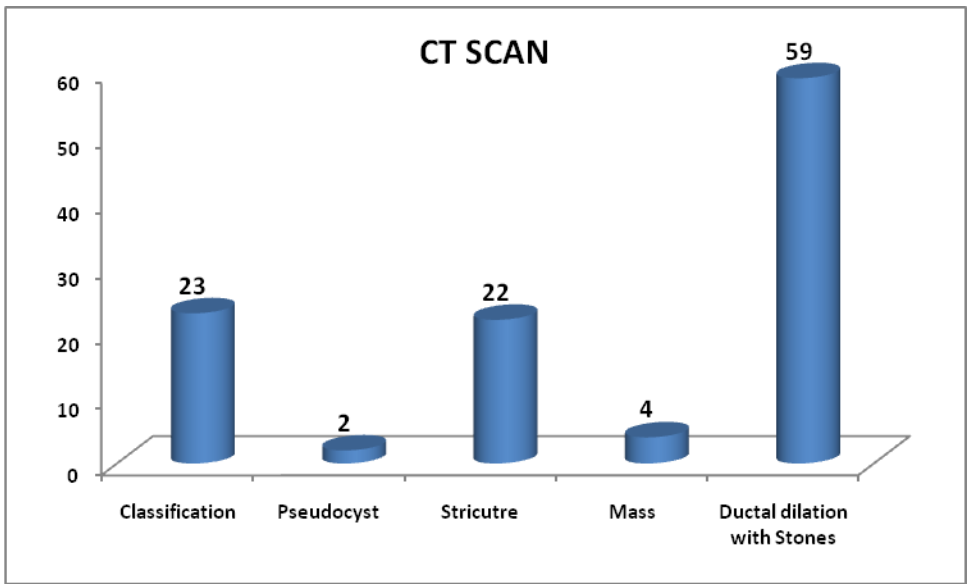
PT - 2.59%

Glycosuria is reported in 36 of the 77 cases (47%). It is especially likely to occur with attacks, being absent in the intervals. It is more common in the case of long standing where considerable fibrosis of the gland has taken place.

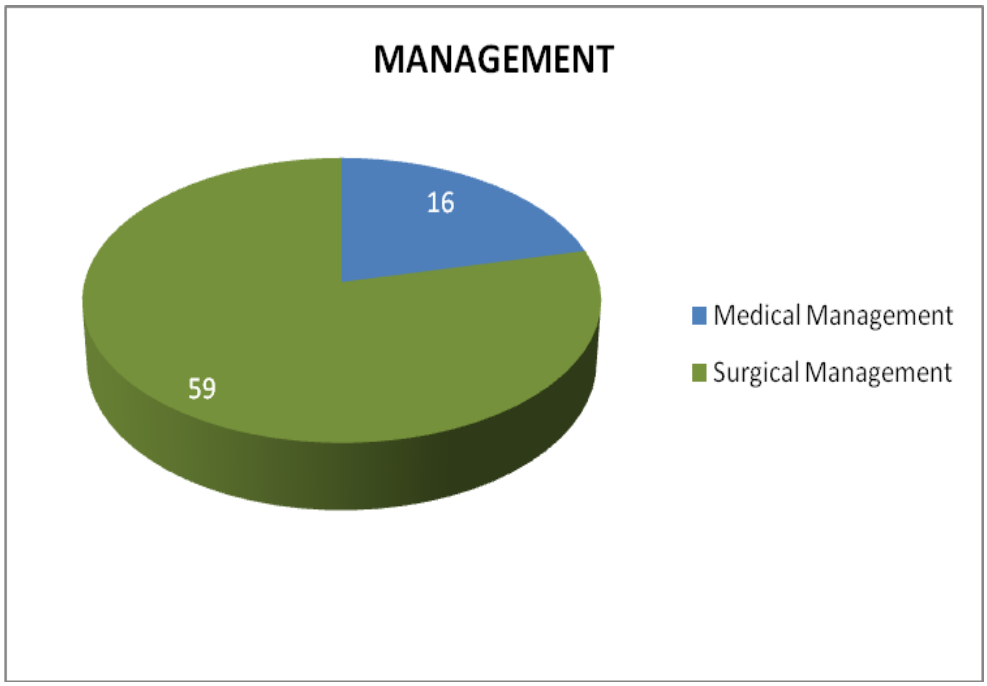
## USB ABDOMEN



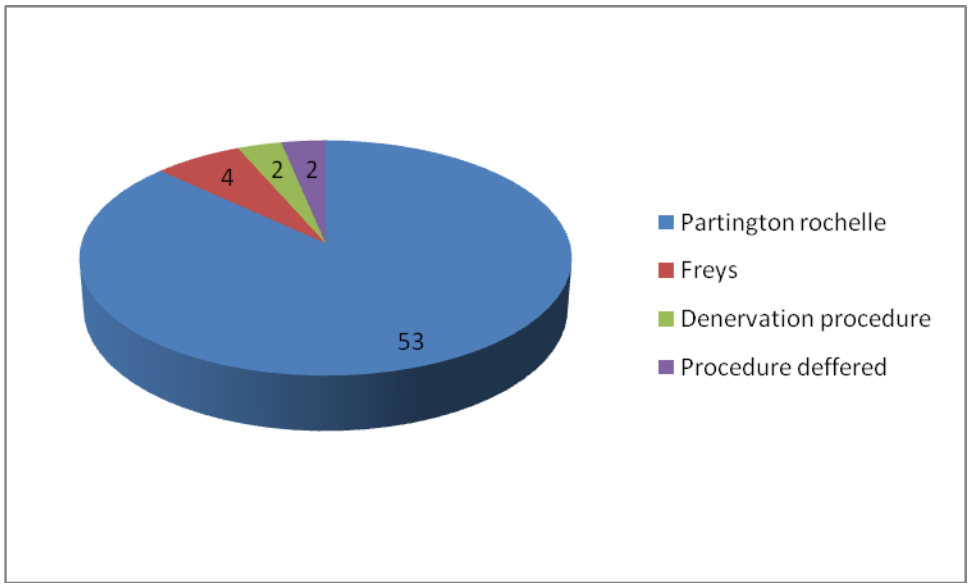

## CT SCAN

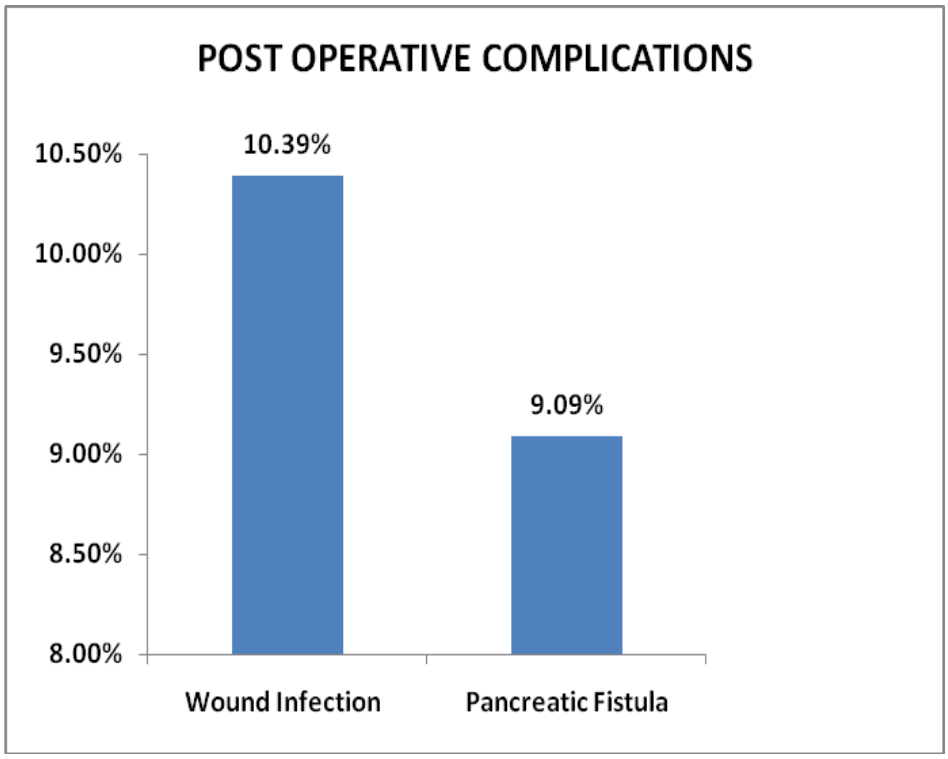


## MANAGEMENT




## SURGERY

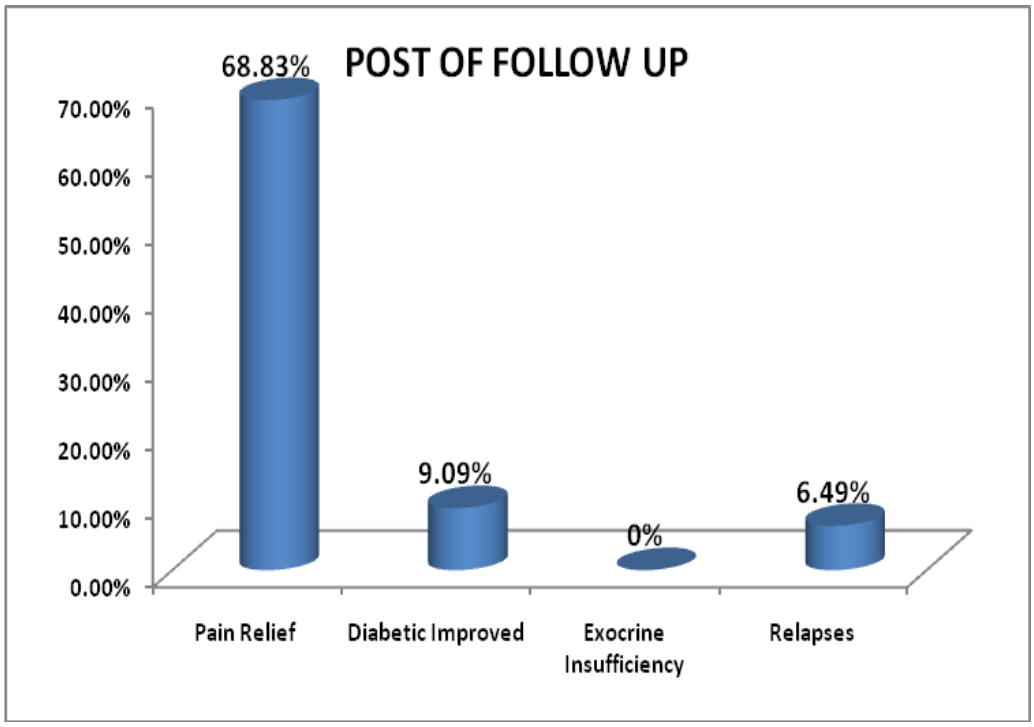





Wound Infection - 10.39%

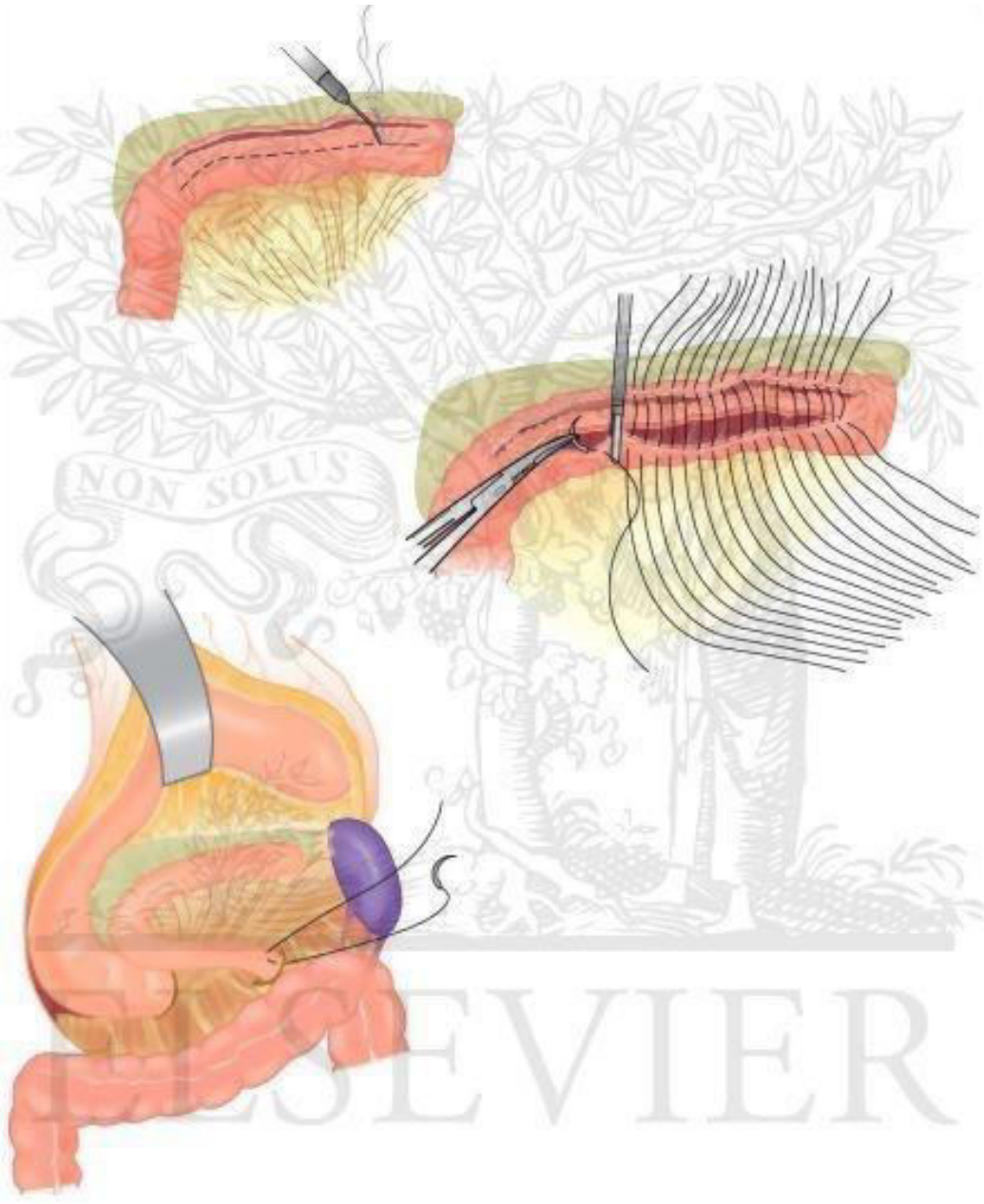
Pancreatic Fistula - 9.09%

## FOLLOW UP STUDY




| | | |
|---|---|---|
| Pain Relief | - | 68.83% |
| Diabetic Improved | - | 9.09% |
| Exocrine Insufficiency | - | 0% |
| Relapses | - | 6.49% |

# LATERAL PANCREATICO JEJUNOSTOMY



# NORMAL FUNCTIONS OF PANCREAS