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Clinical, Radiological Profile Of Pancreatic Ascites & Outcome with Various Treatment Modalities in A Tertiary Care Center

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Abstract
Background: *Pancreatic ascites is defined as exudative ascites caused by nonmalignant pancreatic disease and is characterized by high amylase concentration (>1000IU/L) and protein concentration (>3g/dl)[1,2].It is also defined as syndrome characterized by intraperitoneal pancreatic fluid accumulation due to pancreatic duct disruption or pseudo cyst rupture with high amylase and protein concentration.*
Objectives: *to characterize the clinical and radiological profile of patients presenting with pancreatic ascites and to evaluate the outcome of different treatment modalities and follow up.*
Material and methods: *All the patients with ascites of pancreatic origin attending the outpatient department or inpatients from department of Gastroenterology and also patients from medical and surgical department of Siddhartha Medical College were observed over a period of 24 months i.e November 2017 to October 2019. Symptomatic ascites (ascites or recurs after one or more paracentesis with amylase levels >1000 IU/L and symptomatic ascites with high amylase levels >1000IU/L and ascites with high amylase levels with absence of other explanation for fluid collection were included.*
Results:*In present study of 25 patients 23 were male and 2 were females. The most frequent underlying disease was chronic pancreatitis (84 %), due to alcoholism 76% of cases and one(4%) patient had hereditary etiology and were diagnosed as idiopathic pancreatitis. Only 4 (19.05%) cases of pancreatic ascites are associated with pancreatic pseudocyst.*
Conservative therapy was given to all patients in our study (nil orally, and subcutaneous octreotide followed by abdominal paracentesis). ERCP was done in 10 cases with a success rate of 70 %.In present study 3 patients underwent surgery of which in 2 patients duct disruption was identified on ERCP.
Conclusions: *Conservative treatment can be employed in Pancreatic ascites of acute pancreatic origin. Endotherapy is the best option for treatment of Pancreatic ascites in Chronic pancreatitis patients.*
Keywords: *ERCP, Endotherapy, Paracentesis*

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I. Introduction:

It is defined as exudative ascites caused by non malignant pancreatic disease and is characterized by high amylase concentration (>1000IU/L) and protein concentration (>3g/dl).[1,2]

It is also defined as a syndrome characterized by intraperitoneal pancreatic fluid accumulation due to pancreatic duct disruption or pseudocyst rupture with high amylase and protein concentration.

Because of the low incidence of pancreatic ascites, only case reports and case series have been published, and no randomized controlled trials have been performed to find out the clinical profile and which is the best therapeutic approach.

Indian Data-

Studies on pancreatic ascites in India are very few and all are case reports. Only one study done by D K Bhasin et al [3] from PGI, Chandigarh was published in 2006. Study included 10 patients of pancreatic ascites and pleural effusion with pancreatic duct disruption documented on pancreatogram. In all patients nasal pancreatic drain was placed for 4 weeks. Ascites and/or pleural effusion resolved in all the patients within 4 weeks of placement of NPD. The healing of ductal disruption was demonstrated by nasa pancreatogram as early as 2 weeks and NPD could be removed without necessitating another endoscopy. No major complications related to NPD placement were noted. There was no recurrence of pancreatic ascites and/or pleural effusion at a mean follow up of 39 months.

C Ganesh pai et al [4] analyzed the data on pancreatic ascites with or without pleural effusion treated endoscopically over a ten-years period and concluded that endoscopic therapy is an excellent therapeutic alternative in patients with pancreatic ascites and pleural effusion.

Souad j Taghavi etal [5] open trans duodenal stenting in a chronic pancreatitis patient with pancreatic ascites in whom endotherapy is failed

Prakash Kurumboor et al [6] studied the outcome of pancreatic ascites in in patients with tropical calcific pancreatitis managed using a uniform treatment protocol and concluded thatIn patients with pancreatic ascites in tropical calcific pancreatitis endotherapy and transpapillary stentinghelps in resolution of ascites in nearly half of the patients. In the remaining patients, preliminary conservative management followed by surgical pancreatic ductal drainage provides good relief of symptoms.

ETIOLOGY-

It is reported that pancreatic ascites is more common with Chronic pancreatitis and is around 80%, Trauma 8-10%, Unknown etiology – 10%. Other uncommon etiology- Cystic duplication of bilio-pancreatic duct,Ampullary stenosis,CBD stone contribute to around less than 4 %. Acute Pancreatitis is an uncommon etiology [2]

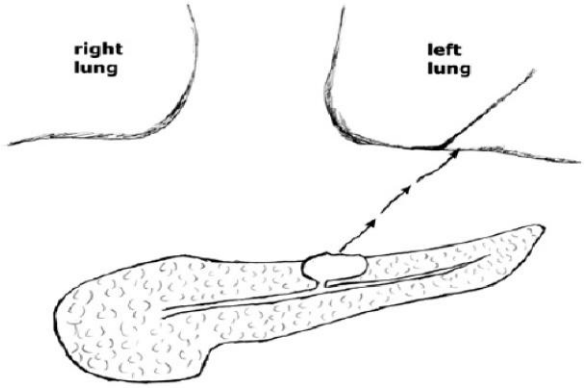
PATHOPHYSIOLOGY OF ASCITES DEVELOPMENT-

Acute pancreatitis is an uncommon etiology: ascites can occur in association with an attack of acute pancreatitis as a result of chemical peritonitis, but the volume is usually small and reabsorbed when the attack resolves. It is unusual for ascites to persist and accumulate in massive amounts after acute pancreatitis [7]

Patients with CP may develop fistulae that reach either the pleural space (producing a pancreatic pleural effusion) or the abdominal cavity (creating pancreatic ascites). This most often occurs when an established pseudocyst ruptures, and the fluid tracks to one of these compartments. It occurs as a consequence of persistent leakage of pancreatic juice from a pseudocyst or the pancreatic duct [8]

The ductal leak is often contained by the back wall of the stomach, transverse colon, and other adjacent structures, which results in a pseudocyst. In those patients where this process is unsuccessful, pancreatic juice may directly reach the peritoneal cavity. Fluid within the abdominal cavity can also track through the esophageal hiatus or aortic hiatus into the mediastinum or erode through the diaphragm into the pleural space. As a consequence, unilateral or bilateral pleural effusions can be found. The majority of patients with these internal fistulae are chronic alcoholics with long-standing CP. In children, trauma is the most common cause of ductal disruption with internal fistulae

Figure 1 :An anterior pancreatic ductal disruption that is not contained and communicates freely with the peritoneal cavity results in pancreatic ascites



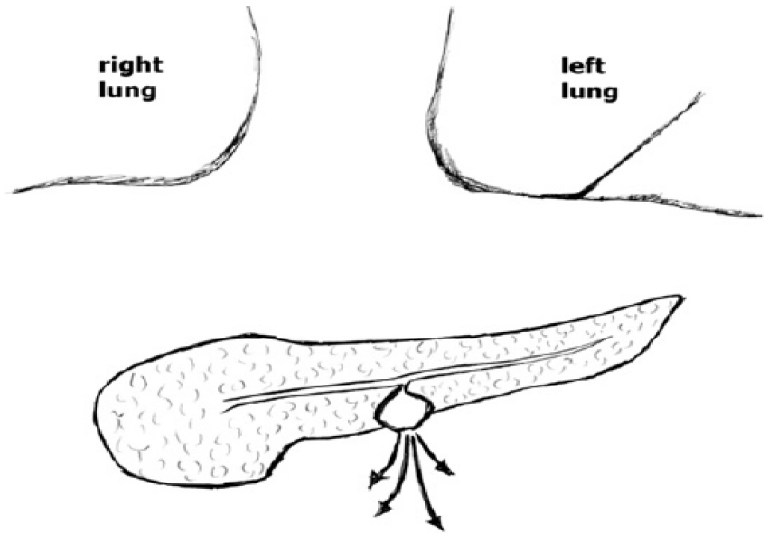


Figure2:A posterior pancreatic ductal disruption tracks through the retroperitoneum into the mediastinum or pleural space to result in a pancreatic pleural effusion

ORIGIN OF LEAKAGE- BASED ON ERCP-

The accumulation of ascitic fluid is caused by leakage of pancreatic juice through a pseudocyst, which communicates with a ductal disruption (43–80%) or a disruption in the pancreatic duct without a pseudocyst (10%); in 10% of the cases, the origin is undetermined [9,10,11]. Pancreatic ascites has been reported to occur in 3.5% of patients with chronic pancreatitis and in 6% to 14% of patients with pseudocysts.

EPIDEMIOLOGY-

Pancreatic ascites is more common in males and Male: female 2:1 [2]. It is more common between the ages of 20-50yrs[2]. Pancreatic ascites is most common in chronic pancreatitis around 80%. Alcoholic etiology is the most common etiology around 70 %. In South India tropical pancreatitis is the most common etiology. Of the chronic pancreatitis 40-80% have pseudocysts, 3-5% has pancreatic ascites [12]. Of all the pancreatic ascites 1/3rd of patients have a past history of acute pancreatitis .

CLINICAL FEATURES -

Patients with pancreatic ascites may have no history or signs to suggest an internal fistula. Most often, they note abdominal distension and typically do not have a history of a recent episode of pancreatitis, although they are often known to suffer from CP [12]. In some patients, there may be abdominal pain, weight loss, or dyspnea. Also, in a number of patients, the presentation may mimic intra-abdominal carcinomatosis with weakness, weight loss, and even false-positive ascitic fluid cytology. It has been hypothesized that pancreatic enzymes can induce metaplasia of serosal cells, and this might be mistaken by less-experienced cytopathologists as malignant cells[13]

INVESTIGATIONS -

When the ascites is sampled, the amylase in the fluid is usually greater than 1000 IU/L and averages 4000 IU/L .

The fluid is usually "exudative" with albumin levels typically over 3 g. Serum amylase is often elevated, but a normal serum amylase does not eliminate pancreatic ascites or pancreatic pleural effusion. Those patients with pancreatic pleural effusion also commonly lack a history of a recent flare of pancreatitis and may mainly complain of dyspnea or chest pain rather than pancreatic-type abdominal pain [12,13]. Like pancreatic ascites, there is mainly anecdotal data. Most reviews note that the majority of patients are chronic alcoholics, and at least half involve ruptured pseudocysts.

In those with pancreatic ascites or a pancreatic pleural effusion, a high quality computed tomography (CT) with pancreatic protocol may define the presence or absence of a residual pseudocyst and may occasionally define the fistula tract. There are also reports of accurate localization of the fistula track using magnetic resonance pancreatography. In most cases, however, an endoscopic retrograde cholangiopancreatography (ERCP) is required to localize the site of the leak and assess for downstream Pancreatic duct strictures, both of which must be known for appropriate therapeutic planning

TREATMENT -

Conservative Treatment

Conservative medical management—withholding of oral feedings, total parenteral nutrition, treatment with somatostatin analogs and large volume paracentesis,for up to 4 weeks—has led to successful resolution of pancreatic ascites in only approximately 50% of cases.The use of the somatostatin analog, octreotide, has been used with anecdotal success. Medical therapy by itself has only moderate efficacy but may be worth the attempt in some patients. In one recent review, octreotide or somatostatin was effective in 12 of 17 patients. Efforts at external drainage are usually met with failure and/or recurrence. Depending on the series, conservative therapy for 3 to 4 weeks has resulted in resolution of ascites in 25% to 60% of case, with mortality rates ranging from 15% to 25%.[14,15,16]

Endoscopic Treatment

If medical therapy is undertaken, and ascites or pleural effusions do not resolve, ERCP should be performed if not already done. Endoscopic therapy can be an option at the same setting. The placement of pancreatic duct stents has become more widely used as therapy of these fistulae. In one recent review, stent placement was as successful as surgery. Pancreatic stenting for the treatment of duct disruption may be employed in one of two ways. Firstly, the endoprosthesis can be introduced in a transpapillary fashion and placed across the pancreatic duct disruption to occlude the defect, which may lead to closure of the fistula. Alternatively, a short stent may be placed across the sphincter of Oddi, reducing the pressure gradient between the pancreatic duct and duodenum, facilitating healing of the fistula. In those with a leak from the pancreatic head or body, a stent that covers the site of the leak is often used, but some have advocated that a shorter pancreatic duct stent across the ampulla is adequate. Pancreatic duct sphincterotomy is also used, usually in conjunction with stent placement. Leaks from the tail of the gland are only able to be treated with these shorter stents.

Surgery

Even if endoscopic therapy is not possible, ERCP should be done in all of these patients with pancreatic ascites or pancreatic pleural effusion to assist the surgeon. The anatomy of the duct and site of the leak allow the surgeon to plan the operative approach, shorten operative time, avoid enterotomy if possible, and aid in the design of optimal operation directed at the duct, pseudocyst, or parenchyma alone. If endoscopic therapy is impossible or unsuccessful, the surgical management of a direct leak in the absence of a persistent or residual pseudocyst is to cap the leak with a defunctionalized jejunal limb and roux anastomosis. If the leak is in the tail, distal pancreatectomy may suffice. In those with a dilated pancreatic duct, therapy may include the addition of a duct decompression surgery, such as the Puestow procedure. In the presence of a pseudocyst, it may be possible to perform a cyst jejunostomy or cystogastrostomy. Postsurgical recurrence rates up to 50% have been reported in cases where the ductal disruption was not visualized preoperatively by endoscopic retrograde pancreatography (ERP)[17]. Despite the improved success rate of surgical therapy in cases in which duct disruption is visualized by ERP, the mortality rates for operative and nonoperative management similarly range from 1% to 25%.

Bore & Cameron[49] classified pancreatic ductal leaks in to 4 types- which decide the type of surgery

TYPES OF PANCREATIC DUCT LEAK-

Type I -- Direct Pancreatic duct leak without pseudocyst
Type II --leak from the pseudocyst located in tail
Type III --leak from the obstructed proximal duct
Type IV --leak from pseudocyst located in proximal portion

TYPES OF SURGERIES-

Type I -- Anastomosis of Roux-en-y loop over a hole in the pancreatic duct.
Type II -- Distal pancreatectomy in addition depends on proximal duct obstruction if absent –oversew of pancreatic remnant if present- drainage by pancreatic remnant if present- drainage by pancreaticojejunostomy using Roux-en-Y loop
Type III -- Distal pancreatectomy with Roux-en-Y drainage for remaining pancreas
Type IV --internal drainage- cystogastrostomy, cystoduodenostomy cysto jejunostomy

External drainage not recommended -high recurrence rate of ascites. Total/ Subtotal pancreatectomy- not recommended due to pancreatic dysfunction miley endocrine.

Fig 3 : showing Schema of the pancreatic ducts and ruptured sites in pancreatic ascites

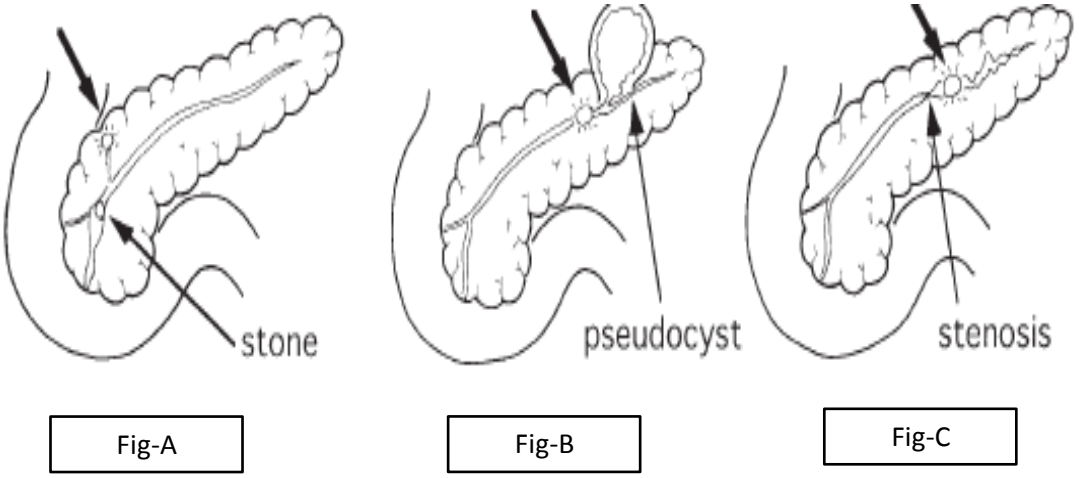


Fig- A - direct disruption of the pancreatic head (arrow).
Fig- B - a pseudocyst of the pancreatic tail and direct disruption of the pancreatic duct at the proximal portion of the pseudocyst (arrow).

Fig- C - Stenosis of the pancreatic duct with direct disruption at the distal portion of the stenosis (arrow)

AIM OF THE STUDY
1.To Know the Clinical & Radiological profile of patients with presenting with pancreatic ascites
2.To Evaluate the outcome of different treatment modalities and follow up

II. Materials And Methods

All patients with ascites of pancreatic origin attending the outpatient Department or inpatients from Department of Gastroenterology and also from patients from Surgery, and General Medicine Department of Gandhi Hospital ,Secunderabad were included in the study.
Period of study- over a period of 24 months i.e November 2009- October 2011
Pancreatic ascites is defined as

1. Exudative ascites with ascitic amylase more than 1000 IU/l and\or
2. Ratio of ascitic amylase to serum amylase 6 and\or
3. Ascites diagnosed by Imaging by U/S abdomen, CECT abdomen and ERCP.

Selection of patients-
INCLUSION CRITERIA-
1.Symptomatic ascites- ascites which persists or recurs after one of more paracentesis with amylase levels >1000IU/L
2.Asymptomatic ascites with high amylase levels >1000IU/L
3.Ascites with high amylase levels with absence of other explanation for fluid collection.

EXCLUSION CRITERIA

1. Age < 12 yrs
2. Pregnant women
3. Patients who are immunocompromised
4. Pancreatic ascites caused by traumatic & surgical causes
5. Pancreatic neoplasms in which the possibility of peritoneal carcinomatosis was discarded
6. Incapacitating Systemic disorders
7. Patients with underlying liver disease were excluded
8. Tuberculous ascites and ascites due to other causes

Patients with pancreatic ascites were initially managed with nil orally and abdominal paracentesis. Patients who did not improve in 1 week or those deteriorating while on conservative treatment were subjected to Endoscopic Retrograde cholangio Pancreatography (ERCP). ERCP was done under aseptic conditions with Olympus 145 series side viewing duodenoscope under conscious sedation with Inj Alprazolam and Inj Fortwin.

Screening Endoscopy was done in all patients to exclude Chronic duodenal Ulcer and possibility of narrowing of the Duodenal lumen and r/o portal Hypertension secondary to Chronic Pancreatitis

In ERCP, Selective Cannulation of pancreatic duct was done, dye injected, any stricture, calculi and duct disruption was identified. Pancreatic sphincterotomy was done in all cases and a pancreatic stent was placed across the fistula where it is possible and a stent was placed across the papilla if the fistula cannot be bridged. Stents were removed after 6 weeks in patients in whom Pancreatic stenting was done.

Therapeutic success was defined as clinical resolution of ascites, a fluid collection and/or fistula. Therapeutic failure was defined as persistence of the PD disruption, ascites, fluid collection, or fistula as demonstrated by radiologic imaging or the need for surgical/radiologic intervention.Patients not improved after ERCP were subjected to surgery in the Department of Surgery.

Patients with pancreatic ascites were initially managed conservatively for 2 weeks with nil by mouth and nasojejunal feeds as per caloric requirement and subcutaneous octreotide 100mcg tid, and abdominal paracentesis, response is assessed after 2 weeks and graded as complete response , partial response, no response. In complete response in whom ascites is completely resolved clinically and radiologically has to keep under follow up. Those with partial response should be subjected to 2 more weeks of conservative therapy. Those without response after 2 weeks and others who got partial response but no response after extended 2 weeks therapy should be subjected to endotherapy - Endoscopic Retrograde cholangio Pancreatography (ERCP). ERCP was done under aseptic conditions with Olympus 145 series side viewing duodenoscope under conscious sedation with Injection Alprazolam and Injection Fortwin.

Screening Endoscopy was done in all patients to exclude Chronic duodenal Ulcer and possibility of narrowing of the Duodenal lumen and r/o portal Hypertension secondary to Chronic Pancreatitis.

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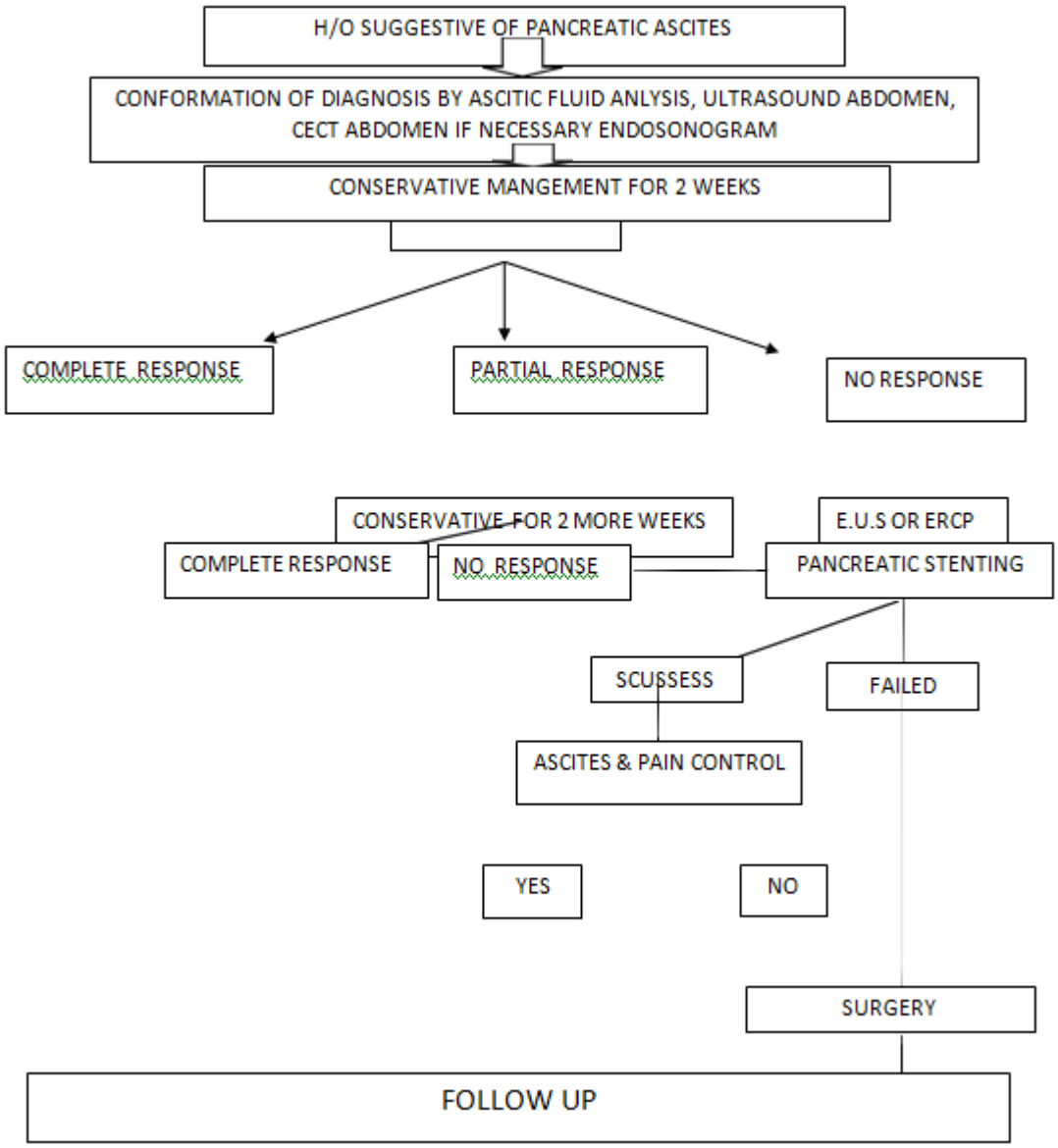
Therapeutic success was defined as clinical resolution of ascites, a fluid collection and/or fistula. Therapeutic failure was defined as inability to cannulate pancreatic duct, persistence of the PD disruption, ascites, fluid collection, or fistula as demonstrated by radiologic imaging or the need for surgical/radiologic intervention.Patients not improved after ERCP were subjected to surgery in the Department of Surgery.

Those who are not able to cannulate pancreatic duct are subjected to MRCP or Endo sonogram and subjected to surgery depending on underlying pancreatic ductal and fistula status. Those patients who responded to any of the treatment options were kept under follow-up.

ALGORITHM FOLLOWED IN STUDY PROTOCOL



Pancreatic ascites is a rare complication of both acute and chronic pancreatitis. Total of 287 patients were admitted in the Department of Gastroenterology, Gandhi Hospital from August 2009 to February 2012. Of these 25 patients were diagnosed to have pancreatic ascites and were included in the study as per inclusion criteria.

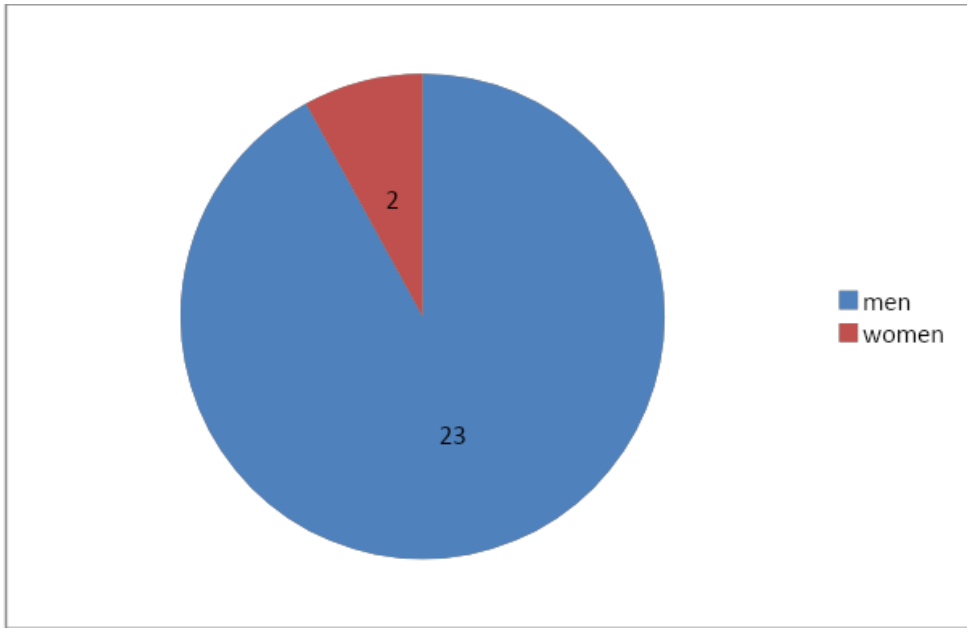
III. Observations And Results

Pancreatic ascites is a rare complication of both acute and chronic pancreatitis. Total of 287 patients were admitted in the department of Gastroenterology, Government General, Vijayawada over a period of 24 months i.e November 2017 to October 2019. Of these 25 patients were diagnosed to have pancreatic ascites and were included in the study as per inclusion criteria.

Sex and Age

Of all 25 patients included in study 23 were male and 2 were female and age ranging from 15 yrs to 58 yrs. means age is of 33.1 yrs and with median of 33.

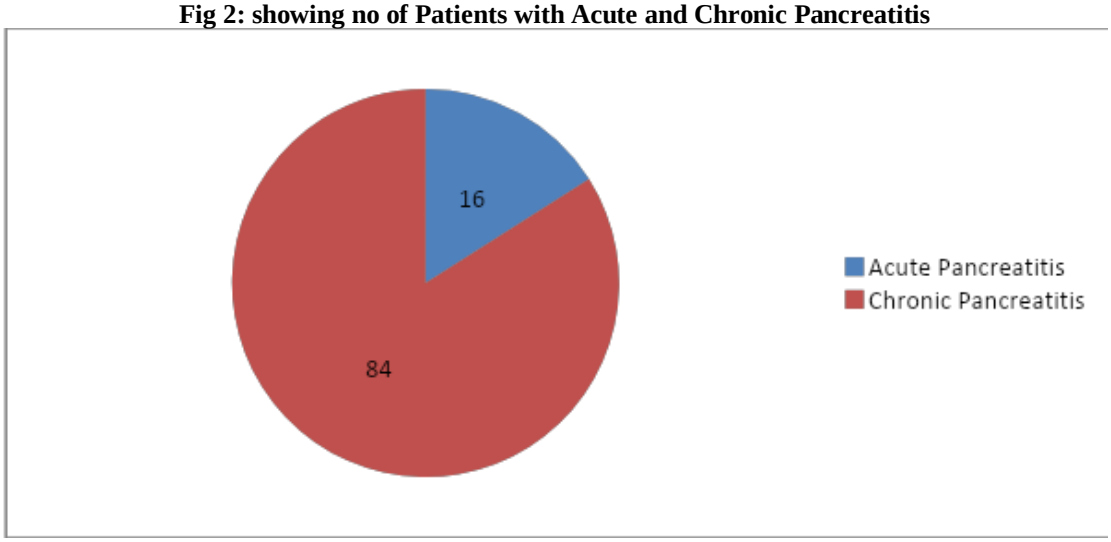
Fig 1: showing Sex wise distribution of patients-



Etiology

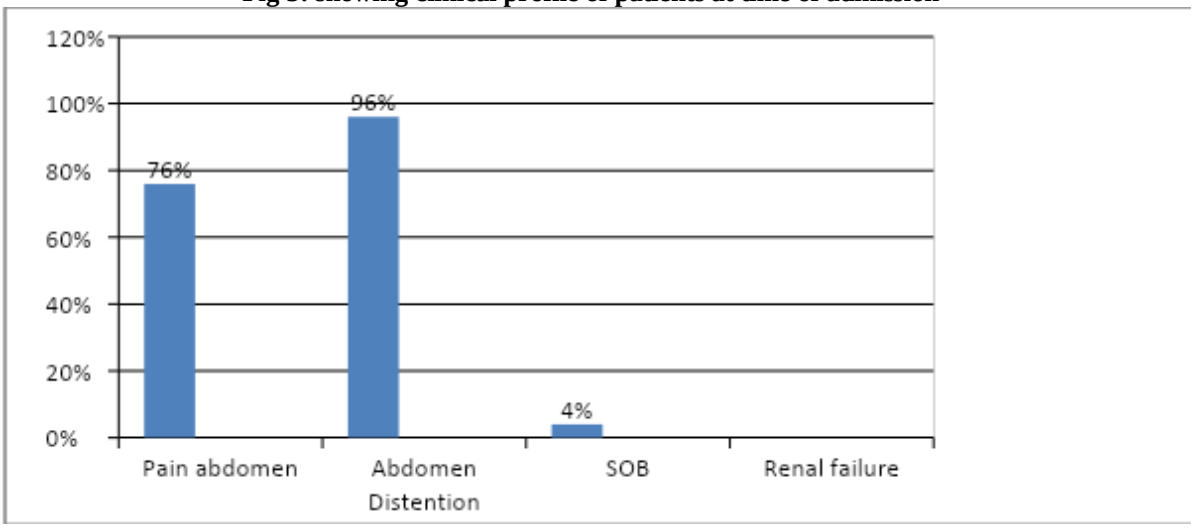
Of 25 patients, 4 (16%) patients did not show any changes of chronic pancreatitis radio logically [ultrasound (all 25 patients) or CT scan abdomen(10 patients)] and were diagnosed to have acute pancreatitis where as other 21 (84 %) patients were diagnosed to be chronic pancreatitis. In 16 (64 %) cases pancreatic ascites are associated with pancreatic pseudocyst. 1 patients had associated pleural effusion.

Fig 2: showing no of Patients with Acute and Chronic Pancreatitis



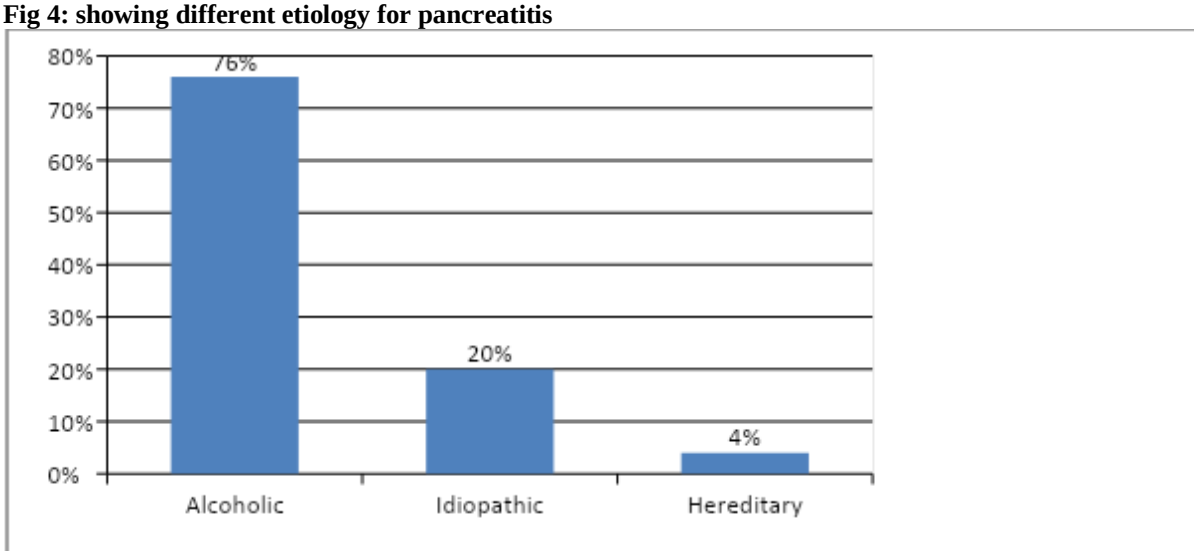
Of 25 patients included in the study 19(76%) patients presented with pain in the abdomen and at time of presentation abdomen distention was noted in 15(60%) patients and developed after admission in 9(36%). Shortness of breath and chest pain was noticed in 1(4%) patient. None of them had renal failure or signs of septicemia at time of admission. 3(12%) developed septicemia in the 2nd week and died .

Fig 3: showing clinical profile of patients at time of admission-



Of 25 patients 19 (76%) were diagnosed to be alcoholic pancreatitis with significant intake of alcohol (150g/day for 5 years), one patient (4%) had significant family history with history of pancreatitis. In remaining 5 (20%) no significant history and biochemical abnormalities including serum calcium and triglycerides and was diagnosed as idiopathic pancreatitis

Fig 4: showing different etiology for pancreatitis



After investigations underlying pathology for formation of ascites, cause was not identified in 4(16%) patients and in 16(64%) pseudocyst was identified and in 5(20%) disruption of pancreatic duct was identified.

Fig 5: showing different underlying causes for ascites

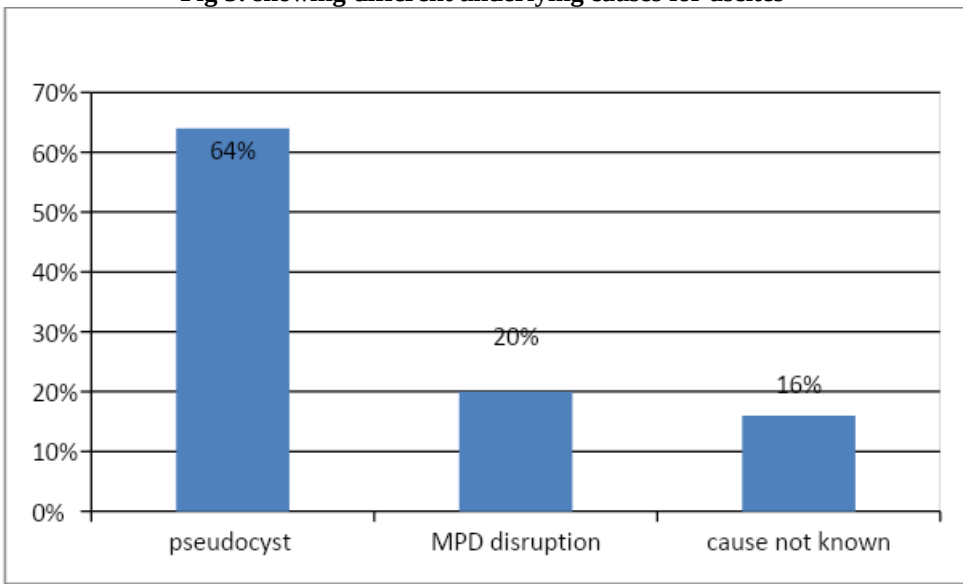


Fig 6: showing radiological profile of acute pancreatitis(C.T SCAN)-

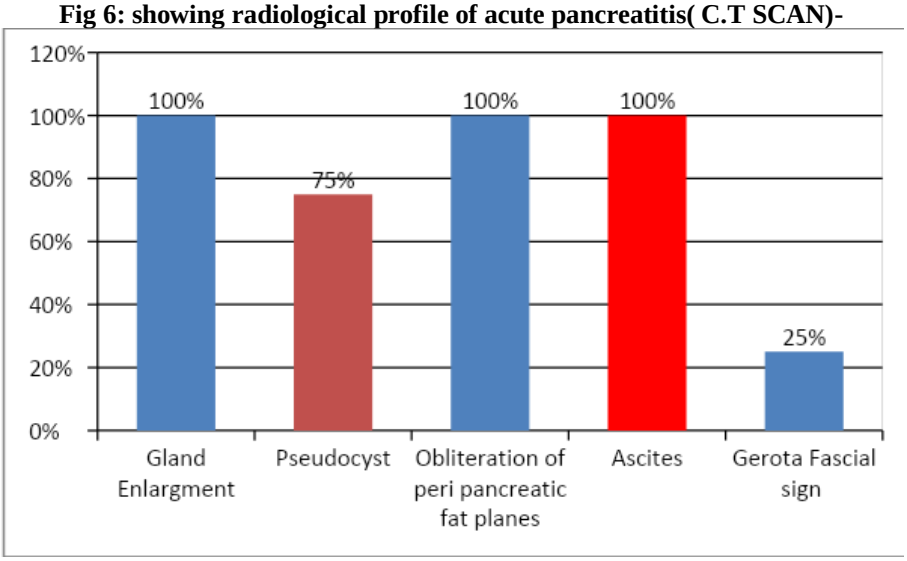
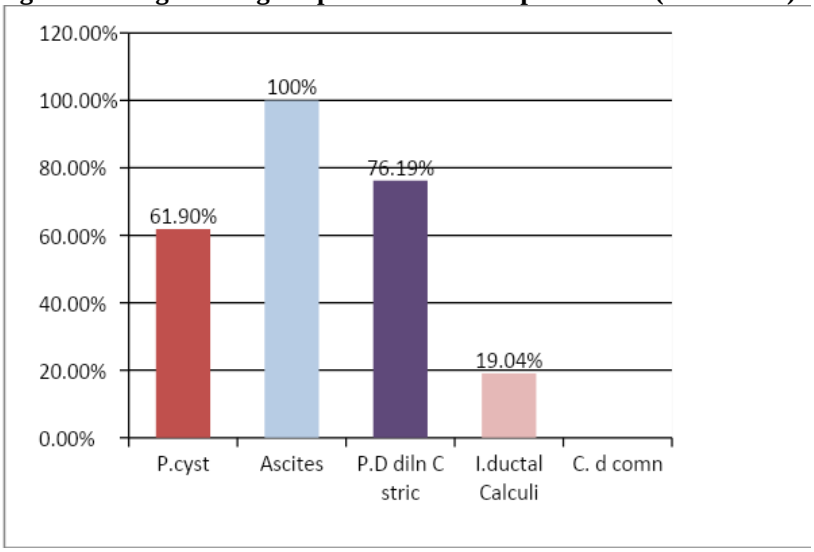


Fig 7 : showing radiological profile of chronic pancreatitis(C.T SCAN) -



Treatment

All these 25 patients were initially treated conservatively with nil orally, nasojejunal feeds as caloric requirement and octreotide 100 micrograms subcutaneously thrice daily in addition abdominal therapeutic paracentesis was done. Patients were observed for decrease in abdominal girth after 14 days of conservative treatment. Response to conservative management was assessed clinically and radiologically after 14days and graded in three categories. Those patients with complete response were discharged and kept under follow-up. Those with partial response were given 2 more weeks of conservative management and observed. Those with no response clinically or radiologically and those who were not tolerating conservative management were subjected to endotherapy in the form of ERCP followed by pancreatic duct stenting.

Table 1: Success with conservative treatment

Pancreatitis	Total number of patients	No. of patients responded	Successes %
Acute pancreatitis	4	4	100
Chronic pancreatitis	21	8	38.09
Total	25	12	48

By the end of 2 weeks in total of 25 patients, treated conservatively 12 (48 %) improved. 3 patients died during the course of conservative management due to multiorgan failure. Remaining 10 patients were subjected to ERCP because of failure of conservative treatment by the end of 2 week.

Table 2: Success with ERCP

Pancreatitis	ERCP success (Ascites relieved)	ERCP failure (Ascites not relieved)	Total
Chronic pancreatitis	7 (70 %)	3 (30 %)	10

In total 10 patients underwent ERCP, ERCP was successful in 7 (70 %) cases (i.e. disappearance of ascites by 3rd week after ERCP). ERCP failed in 3 (30 %) patients. Of the 7 patients, duct disruption was identified in 5 patients. Of 5 cases disruption is seen in the body in 2 (%) patients, in 1 (%) in the tail and in 2 (%) patients in the genus.

Of a total of 25 patients 3 patients required surgery who did not improve with conservative treatment and ERCP. All of them were chronic pancreatitis, one had stricture with calculi and the other 2 had pseudocyst. In patients with pseudocyst cystogastrostomy with lateral pancreaticojejunostomy was done whereas in remaining 1 patients only lateral pancreaticojejunostomy was done. All the 3 patients ascites disappeared after surgery

On subanalysis of the results, of a total of 25 pancreatic ascites patients conservatively treated, 4(100%) out of 4 acute pancreatitis patients improved with complete disappearance of ascites by the end of 2 weeks. In chronic pancreatitis, out of 21 cases only 8 (32 %) cases improved.

Fig 8: showing success rate of conservative treatment in acute and chronic pancreatitis

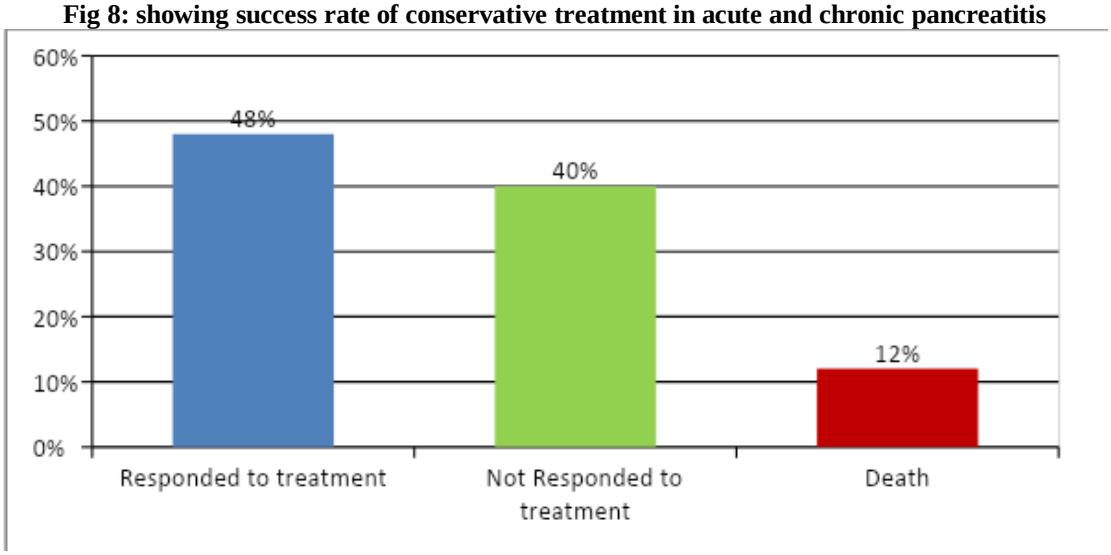
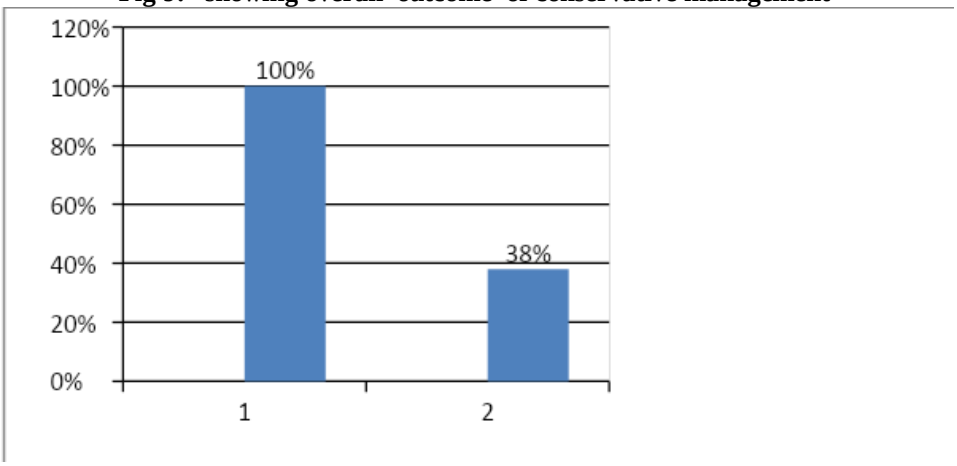


Fig 9: showing overall outcome of conservative management



Of 21 chronic pancreatitis patients,3 died during conservative management due to multiorgan failure, out of remaining 18, 8 responded to conservative management and 10 required ERCP. In only 3(30%) cases ERCP failed, that is, ascites were not relieved by end of 3rd week after ERCP, and in remaining 7(70 %) ascites completely relieved. In 3 patients in whom ERCP failed were subjected to surgery, all patients were improved after surgery.

All those patients who responded to different modalities of treatment were kept under follow up, with weekly visits for one month and later once in month. Till date all those patients responded were under follow up for 5 months. None of these patients had recurrence of ascites.

Fig 10 : showing success rate of ERCP in chronic pancreatitis

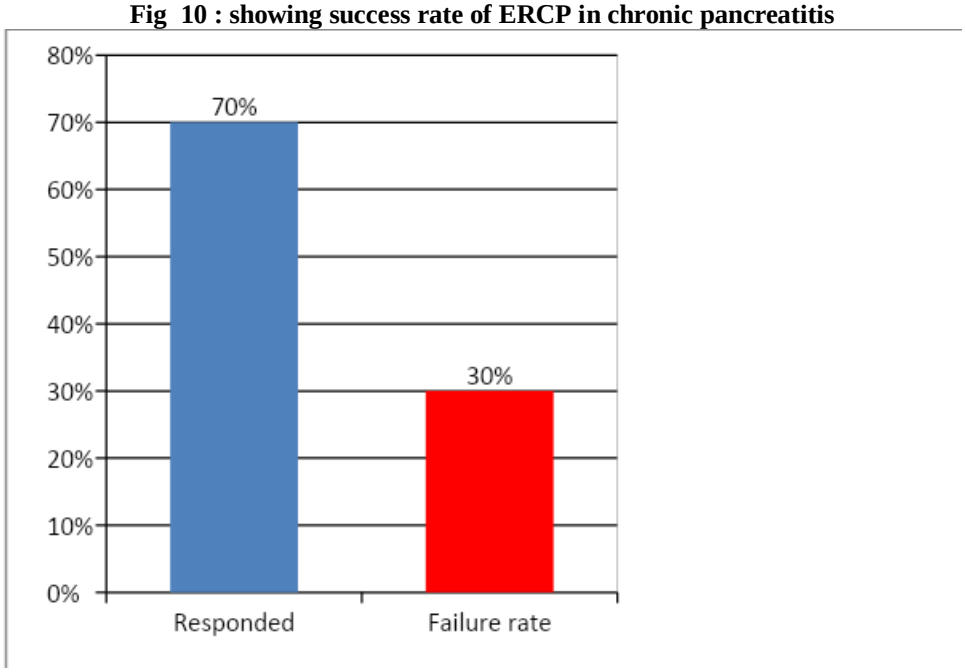


Fig 11 : showing success rate of ERCP in relieving ascites in stenting success and stenting failure(only sphincterotomy)

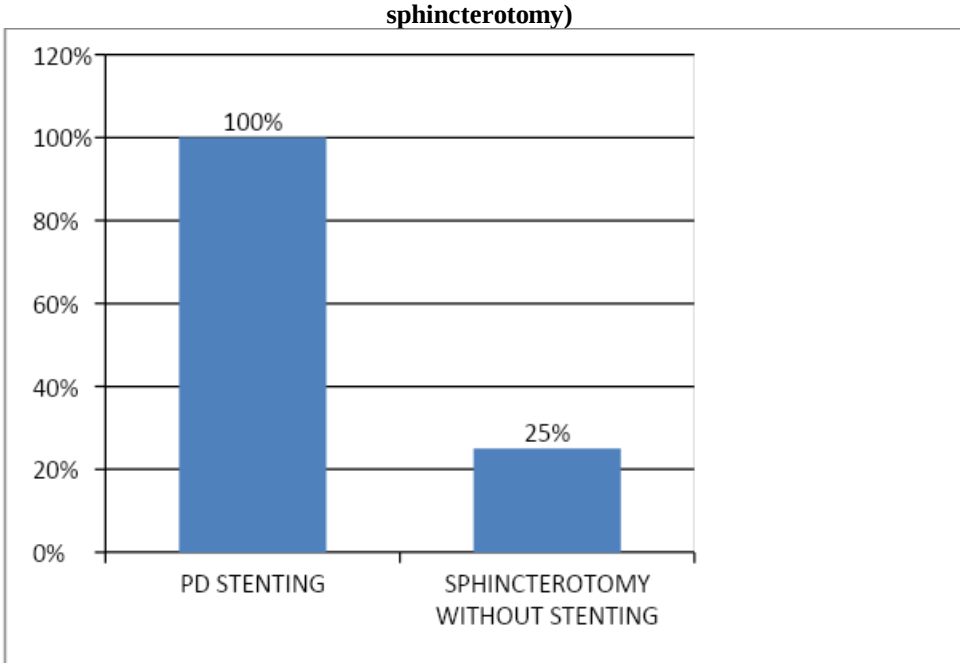


Fig 12 : Figure showing success rate of stenting and only sphincterotomy

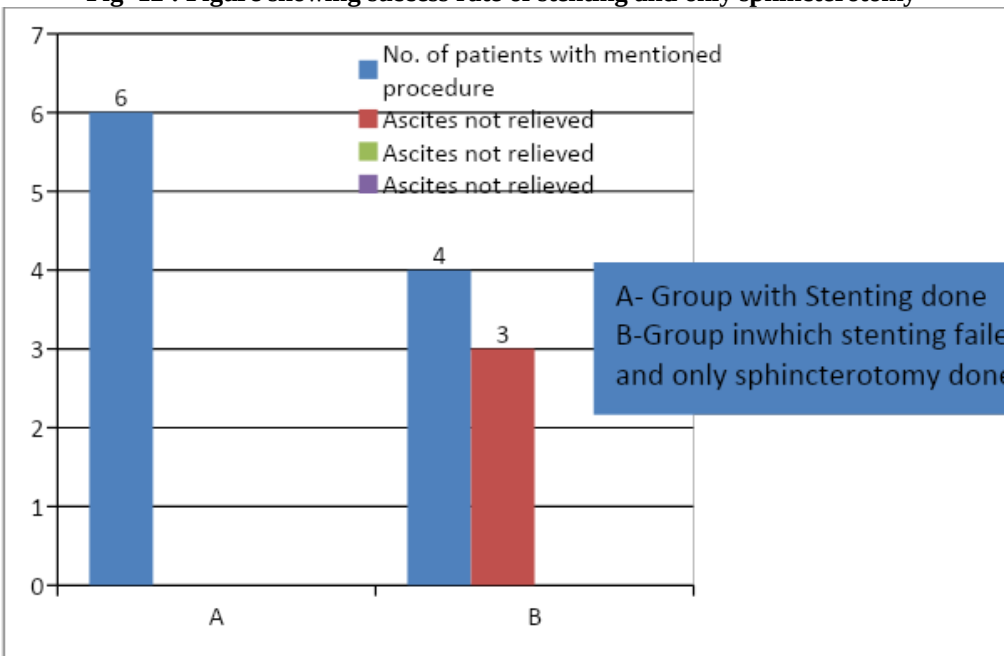


Fig 13 : showing overall success rate of treatment in combination

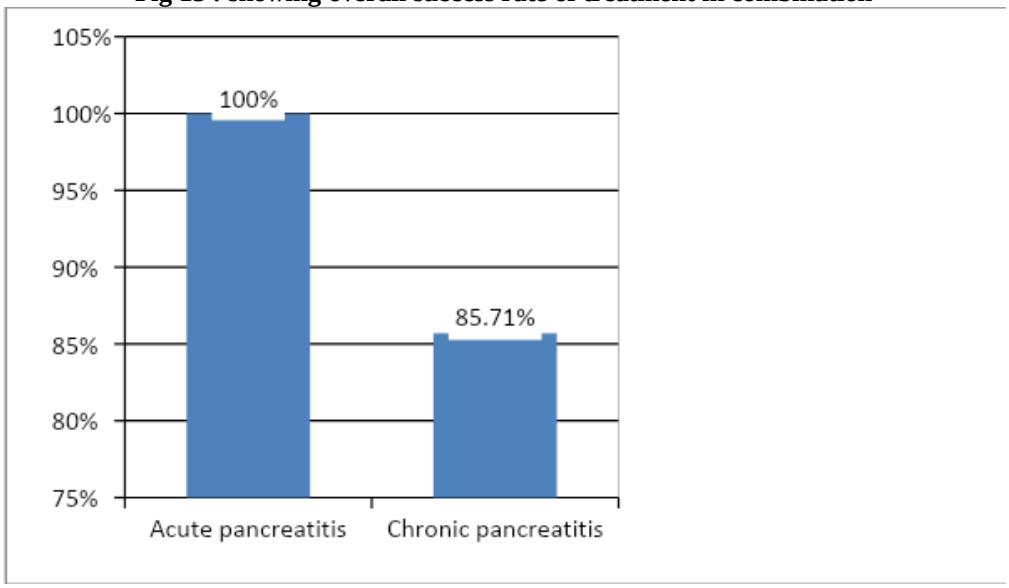


Table 3: Stenting in ERCP

Ascites	Ascites relieved	Ascites not relieved	Total
Stenting done	6 (100%)	0 (0%)	6
Stenting not done	1 (25%)	3 (75%)	4
Total	7 (70 %)	3 (30 %)	10

All the 4 patients underwent sphincterotomy with a sphincterotome. 4 patients had stricture and calculi in main pancreatic duct, only 1 patient got ascites relieved and remaining 3 not benefited with only sphincterotomy and needed surgery.

Of 10 patients who underwent ERCP, stenting was done in only 6 cases (7 Fr 10CM – 2 , 7 Fr 7 CM - 1, 7 Fr 5 CM - 1, 5 Ft 7 CM – 1 pancreatic stents were used). In all these 6 (100%) patients, ascites were relieved by the end of 3 weeks after ERCP. Of 4 patients in whom only sphincterotomy was done, in 1(25 %) patients ascites relieved, in remaining 3(75 %) patients, 3 patients required surgery.

Of these 6 patients in whom stenting was done, in 5 patients stent was removed after 6 weeks. In the remaining 1 patient's stent fell down spontaneously. In 1 patient, along with ascites there is also pleural effusion. This particular patient is having chronic pancreatitis and responded with ERCP, both ascites and pleural effusion was completely responded.

Complications of ERCP and mortality: Of the 10 patients who underwent ERCP, no patient developed major complications like pancreatitis, bleeding, and perforation

Table 4 : Patient characteristics with reference to ERCP success rate

Patient Characteristics	ERCP Success	ERCP Failure
Age (Mean)	30.2 years	32.25 yrs
Acute Pancreatitis (%)	Not needed	-
Chronic Pancreatitis	7 (70 %)	3 (30 %)
ERCP Findings		
Stricture and calculi (4 patients)	3	1
Duct Disruption (5)		
Genu	2	0
Body	2	0
Tail	1	1
Stenting done (6 patients)	6 (100%)	0
Stenting across disruption	3	
Only Sphincterotomy (4 patients)	1 (25%)	3 (75%)

IV. Discussion

Because of the low incidence of pancreatic ascites, only case reports and case series have been published, and no randomized controlled trials have been performed to find out which is the best therapeutic approach. The aim of this study was to know the clinical picture of Pancreatic ascites, and radiological profile and to evaluate the utility of the different treatments for pancreatic ascites.

We found that in our present study of 25 patients 23 were male and 2 were females . The most frequent underlying disease was chronic pancreatitis (84 %), which was caused by alcoholism 76% of cases and one(4%) patient had hereditary etiology and remaining in the remaining 20% no significant etiology was identified and diagnosed as idiopathic pancreatitis . Three of four patients (75%) in the Kozarek *et al* [18] series and six of eight patients (75%) in the Bracher *et al.* series had pancreatic ascites because of underlying alcoholic chronic pancreatitis. In a review of all case reports of patients with pancreatic ascites described in the literature, Gomez-Cerezo *et al* . noted 92/139 patients (66.2%) with underlying chronic alcoholic pancreatitis. Acute pancreatitis is an infrequent cause of pancreatic ascites/pleural effusion because the pancreatic duct disruption in the setting of acute pancreatitis is usually walled off by the surrounding inflammatory reaction. Only 12/139 patients (8.6%) with pancreatic ascites described by Gomez-Cerezo *et al*. had underlying acute pancreatitis. In our study 16 % patients of pancreatic ascites had underlying acute pancreatitis.

Table 1 : Comparison of percentage of chronic pancreatitis in different studies

Study	Chronic Pancreatitis (%)
Kozarek et al study	75%
Bracher et al study	75%
Gomez-Cerezo et al study	66.2%
Present study	84 %

In present study only 4 (19.05%) cases of pancreatic ascites are associated with pancreatic pseudocyst.

Conservative therapy was given to all patients in our study (nil orally, and subcutaneous octreotide followed by abdominal paracentesis). These combined conservative treatments had a considerable outcome in acute pancreatitis and had a high failure rate of 62% with 12% mortality rate in our study, as in previous series which reported disappointingly high rates of 40–60%.

ERCP was done in 10 cases of which duct disruption is identified in 5 cases. Duct disruption is more commonly seen in the body and genus . Stenting was done in only 6 cases of which all patients improved that is 100% success. In 4 cases stenting was not able to be done and only sphincterotomy in these patients' success rate is 25%.

In our study the success rate of ERCP is 70 % which is comparable with the previous studies.

There is only one single study in India done by D K Bhasin et al [13] in which they showed that in 10 cases of pancreatic ascites and pleural effusion, nasopancreatic drainage relieved ascites and effusion completely in 4 weeks.

Shyam Varadarajulu et al showed that stent insertion is successful in 55% of pancreatic disruption patients

In our study in 4 patients stenting was not done and in them 25% improved.This can be explained by mechanism whereby a ductal disruption heals after transpapillary pancreatic stent placement may be obliteration of the high-pressure gradient at the pancreatic sphincter, allowing preferential flow of pancreatic secretions along a low resistance path to the duodenum. It may not be necessary to bridge the site of ductal disruption

An ERCP is mandatory to identify the site of leakage before considering surgical treatment because the type of surgical intervention is decided according to the findings on ERCP. The identification of the point of leakage before surgical intervention has a major influence on the surgical outcome. The surgical failure rate is 12–18% if a point of leakage is found on ERCP before surgery, but it can be as high as 50% if the adequate surgical technique cannot be planned because of the absence of an identifiable site of leakage on ERCP[19] In present study 3 patients underwent surgery of which in 2 patients duct disruption was identified on ERCP before undergoing surgery. All 3 patients improved after surgery.

V. Summary And Conclusions

1. Pancreatic ascites is a rare complication associated more with chronic pancreatitis than acute pancreatitis.
2. From the data available including in the present study Conservative treatment can be employed in Pancreatic ascites of acute pancreatic origin
3. Conservative management will be tried in all patients with chronic pancreatitis with close monitoring.
4. Endotherapy is the best option for treatment of Pancreatic ascites in Chronic pancreatitis patients.

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