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Original Research Article

Early surgical management in pancreatic ascites on a background of chronic pancreatitis

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ABSTRACT

Background: Pancreatic ascites can occur as a complication of acute or chronic pancreatitis. In majority of cases, it is associated with pseudocyst or duct disruption. Management is initially conservative with paracentesis with nutritional support. Early surgery has been recently contemplated as primary management for pancreatic ascites.
Methods: A prospective study was done over a duration of three years from November 2017 to October 2019 in patients of chronic pancreatitis presenting with pancreatic ascites. All patients underwent pancreatic protocol contrast enhanced computed tomography abdomen and magnetic resonance cholangiopancreatography, duct diameter and main pancreatic duct (MPD) disruption site. pseudocyst site was identified. After optimizing patients, early surgery was planned. Surgeries included either lateral pancreatic jejunostomy, if MPD were dilated. Pancreaticogastrostomy, cystogastrostomy or cystojejunostomy, if there was pseudocyst with extraneous impression over stomach or on mesocolon, and distal pancreatectomy (and/or) splenectomy.
Results: Out of 20 cases of pancreatic ascites, 6 were of acute pancreatitis and conservatively managed and 14 were subjected to early primary surgery. 8 out of 14 patients underwent LPJ. 2/14 underwent pancreaticogastrostomy. 2/14 underwent cystogastrostomy and 1/14 underwent spleen preserving distal pancreatectomy. 1/14 underwent distal pancreatectomy and splenectomy. None of the patients had postoperative recurrence of pancreatic ascites. One patient developed Postoperative intra-abdominal collection which was drained. Mortality was 2/14 (14.2%), one died immediate postoperatively and another succumb to Pulmonary embolism on post-operative day-4. Pain scores were significantly reduced post-operatively.
Conclusions: Primary early surgery directed towards primary pathology, as guided by MPD status, in selected patients with chronic pancreatitis with ascites leads to faster recovery of patient.

Keywords: Pancreatic ascites, Chronic pancreatitis, Pleural effusion, Lateral pancreatic jejunostomy

INTRODUCTION

Pancreatic ascites was first reported in the literature by Smith in 1953.[1] It can occur as a complication of either acute or chronic pancreatitis. In pancreatic ascites, the duct disruption is anterior and internal pancreatic fistula develops in peritoneal cavity and in pancreatic pleural effusion or pancreatopleural fistula, the disruption site is posterior and tracks through the mediastinum into one or both pleural cavities.[2-5] Approximately 50% of patients who develop pancreatic ascites have a concomitant pseudocyst that is leaking. Approximately 95% of cases of pancreatic ascites are associated with chronic pancreatitis. The leak manifests upstream of a stricture or stone, and the point of least resistance for the pancreatic juice to flow is into the abdominal cavity rather than the

duodenum, where it belongs. Because the pancreas normally produces >1 l of fluid a day, ascites and effusions are typically massive, and the absence of an appropriate inflammatory response in a patient who is often malnourished means that spontaneous closure of internal pancreatic fistula is unlikely. Management is initially conservative with paracentesis, Total parenteral nutrition, octreotide or endotherapy if possible or surgery. Early surgery has been recently contemplated as primary management for pancreatic ascites. Here, we report our clinical experience with early surgical outcomes after early surgical management of pancreatic ascites in chronic pancreatitis patients.

METHODS

This was a prospective observational study conducted between 1st January 2012 to 31st December 2015 at the Institute of Surgical Gastroenterology and Liver Transplantation, Stanley Medical College and approval from the Institutional Ethical Committee had been obtained. An informed consent had been obtained from all the patients participating in the study. The inclusion criterion was that all patients were with chronic pancreatitis with ascites and an ascitic fluid amylase level >1000 IU/l. We have treated a total of 20 cases of pancreatic ascites over a three years period from November 2017 to October 2019 of which 14 cases (70%) were due to chronic pancreatitis (CP). In 13 (92.8%) of 14 cases the aetiology was ethanol induced CP and all were males. One female patient had idiopathic CP. The age of the patients ranged between 7 to 44 years. 3 out of 14 patients (21.4%) had previous episodes of pancreatitis in past. Most common presenting symptom was abdominal distension followed by abdominal pain and weight loss. 5 out of 14 patients (35.7%) were diabetics. 2 out of 14 patients underwent endoscopic retrograde cholangiopancreatography (ERCP) or main pancreatic duct (MPD stenting earlier which failed to resolve and then underwent surgery as definitive treatment. All the 14 patients were hospitalised and evaluated with the following investigations: complete blood count (CBC), renal function test (RFT), liver function tests (LFT), serum amylase, ascitic fluid amylase and albumin levels (Table 1), plain X-ray chest to rule out coexistent pleural effusion, electrocardiogram, ultrasound of the abdomen, a pancreatic protocol multidetector computed tomography of the abdomen (Figure 1 and Figure 3) and an MRI abdomen with MRCP (Figure 2). All patients were subjected to cardiac evaluation with an echocardiography and a pulmonary function test (PFT). All patients were encouraged to do incentive spirometry to improve the pulmonary compliance. All patients were encouraged for enteral hyperalimentation with high protein diet. 2 patients who presented with severe weight loss and muscle loss were given Total parenteral nutrition. Abdominal paracentesis was not done in any of the patients as none were having respiratory distress and to prevent protein rich fluid losses. Surgery was tailored to the individual case with a combination of internal doct or pseudocyst drainage and distal resection.

Table 1: Demographic characteristics.

Age (years)	1-10	11-20	21-30	31-40	41-50
Male	-	4	3	4	1
Female	1	-	1	-	-

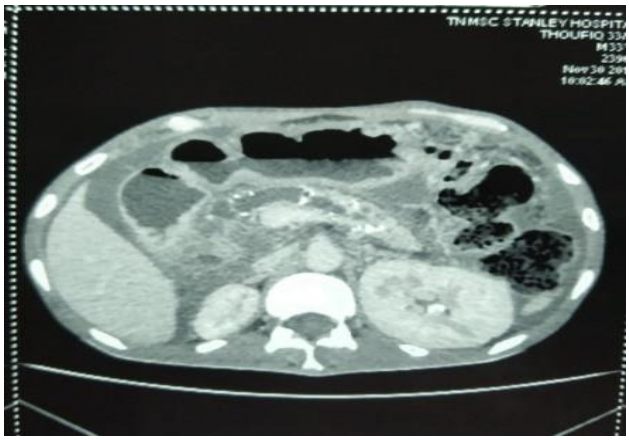

Figure 1: CECT showing dilated MPD and CCP featured with moderate ascites.

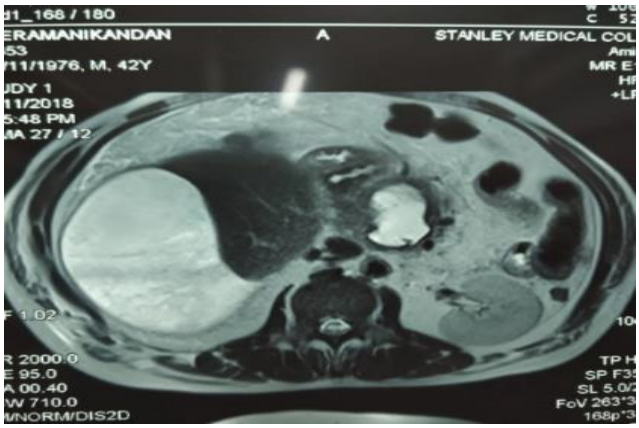

Figure 2: MRCP showing ruptured pancreatic pseudocyst with right subdiaphragmatic collection.

Figure 3: Showing the duct disruption site in MPD with opened (pink) communicating pseudocyst.

RESULTS

All 14 patients were subjected to surgery after optimization. MPD was found to be dilated in 9/14 cases (64.2%) (Table 2). There was associated pseudocyst in 2/14 cases (14.2%). The site of MPD disruption was identified in 4/14 cases (28.57%), three of which were in the distal pancreas to the left of the superior mesenteric artery and one in head for which PG done. The 14 cases underwent primary surgery as follows: LPJ-8, Lateral pancreaticogastrostomy-2, cystogastrostomy-2 (MPD not very dilated and communicating pseudocyst in MRCP), distal pancreatectomy with splenectomy (DP+S)-1 (dense adhesions), spleen preserving distal pancreatectomy-1. Two patients underwent a lateral pancreaticogastrostomy (LPG)- one case had purulent abdominal fluid collections and was in sepsis even after pigtail external drainage, so LPG was done to avoid the hazards of two anastomoses if LPJ was contemplated. In 8/14 cases that underwent an LPJ, MPD dilation was identified in all cases.

In the post-operative period, four patients had biochemical leak- ISGPS grade BL pancreatic fistula, which settled with conservative management. Five had surgical site infection and settled with appropriate antibiotics. One developed postoperative incisional hernia. Two patients died- one of sepsis with Pulmonary embolism on postoperative day-4 and other, of postoperative cardiac arrest probably due to chronic malnutrition associated cardiomyopathy. One patient received Pneumococcal vaccine preoperatively to DP+S. The overall duration of hospital stays ranged from 15-30 days. A snapshot of the pathological morphology of the disease and the surgery done is given in Table 3.

At one year follow up there was no recurrence of ascites in all the 12 patients who underwent primary surgery and 3/14 patients had mild intermittent abdominal pain- maximum VAS score was 3. All 13 male patients with Ethanol as cause were advised for de-addiction therapy after surgery.

Table 2: Clinical characteristics.

Clinical features	No. of patients
Chronic calcific pancreatitis	**7**
pseudocyst	4
Duct disruption	4

Table 3: Mean values-ascitic and serum albumin and amylase analysis (n=14).

	Serum	Ascitic fluid
Amylase	746	4630
Total proteins	6.2	4.23
Albumin	2.8	2.27

Table 4: MPD morphology on MRCP.

MPD morphology	No. of patients	Intervention done
Dilatation	8	Surgery
Dilatation and partial disruption	2	Stenting followed by surgery and pancreaticogastrostomy
Complete disruption	1	LPJ
Complete obstruction	1	LPJ
Prominent pseudocyst	2	Cystogastrostomy

Table 5: Type of interventions.

Intervention	No. of patients
ERCP + surgery	2
Direct surgery	12

DISCUSSION

Pancreatic ascites is defined as an exudative ascites caused by non-malignant pancreatic disease and is characterised by a very high amylase concentration in ascitic fluid (usually over 1000 IU/l) and albumin concentration over 3 gm/dl. The pancreatic enzymes in the ascitic fluid are inactive and do not lead to digestion of tissues, but instead causes inflammation and exudation leading to an albumin rich fluid.[6] In patients with severe hypoproteinaemia, the ascitic fluid albumin levels might be less than 3 gm/dl. In 40-80% of cases the ascites is due to a leaking pseudocyst communicating with the MPD, in 10% due to MPD disruptions without a pseudocyst and in another 10% the site of disruption cannot be identified. Surgery for pancreatic ascites is often difficult due to the inflammatory process in the peripancreatic tissue, mesentery and due to the presence of pseudocysts and abscess.[4] However, good results of ductal drainage and LPJ in patients with pancreatic ascites have been reported. Selection of patients for surgery should be carefully done.

Conservative therapy for pancreatitis consists in keeping the patient nil per oral and use of somatostatin analogues to decrease the pancreatic secretions. Repeated large volume paracentesis is done to improve patient discomfort and also with a premise that this might promote approximation of peritoneal surfaces of the lesser sac to the leaking site thereby, sealing the site of ductal or pseudocyst disruption.[8] In our study, none were subjected to paracentesis. Early enteral hyperalimentation was given orally 12/14 cases and 2 cases received TPN. 20% human albumin infusion was selectively used in patients with severe cachexia and decreased plasma oncotic pressure.

Endotherapy with pancreatic duct stenting is a viable option in patients with PA complicating CP and also for post traumatic MPD disruptions.[9,10] The various reported series claim success rates from 80-100%. However, this is limited by availability of expertise, feasibility of stenting the disruption and also the location of the ductal disruption. Moreover, pancreatic duct stenting has its own risks. The side flap of the stent can induce ductitis and cause duct stenosis as a result of fibrosis. Above all MPD stenting leads to bacterial colonisation and infectious complications. This leads to infected pseudocysts presenting as increased WBC counts, persistent tachycardia, persistent abdominal pain or fever. This was observed in two of our patients after MPD stenting. Also, it might make future surgery difficult in case endotherapy fails. In patients with advanced CP with multiple duct strictures and intraductal calculi the role of endotherapy is limited. When the MPD is dilated sufficiently to do a safe internal ductal drainage procedure, surgery addresses both the ductal disruption and the associated ductal pathology (strictures and calculi). In patients with intractable pain due to CP and PA surgery again addresses both.

There are various surgical options that must be tailored to the individual case depending on the pathological morphology of the disease and fitness of the patient for surgery. In majority of the cases there is an associated communicating pseudocyst which is usually small, possibly due to the constant leakage of pancreatic juice into the peritoneal cavity. When there is a communicating pseudocyst in the tail a distal pancreatic resection or internal drainage of the pseudocyst can be done. The proximal remnant pancreatic duct is drained internally when diseased and obstructed. When there is distal ductal disruption alone, a distal resection will suffice. The extent of parenchyma resection should always be kept to a minimum to delay the onset of endocrine or exocrine insufficiency in an already diseased organ. When there is a diffuse MPD dilation with disruption, an LPJ is the most appropriate procedure irrespective of the site of disruption. The cavity of a small pseudocyst communicating with the MPD can be included in the LPJ (Figure 4 and Figure 5). In one out of 14 patients, Pancreaticogastrostomy was done due to dense adhesions between pancreas and infracolic compartment precluding an LPJ.[11] In case of proximal ductal disruptions without a dilated MPD a Roux-en Y fistulojejunostomy can be done. When a pseudocyst alone is present an internal drainage procedure to the stomach, duodenum or Roux loop of jejunum is appropriate. If the pseudocyst wall is thin and not safe for an anastomosis an external drainage procedure can be done.[13] The reported success rate with the various series is >90%.[7,12] The overall mortality rate is 9%. In our series, we had two mortality and the rest 12/14 patients had no disease recurrence at the end of one year of post-operative follow up. The duration of hospital stays ranged from fifteen to 30 days.

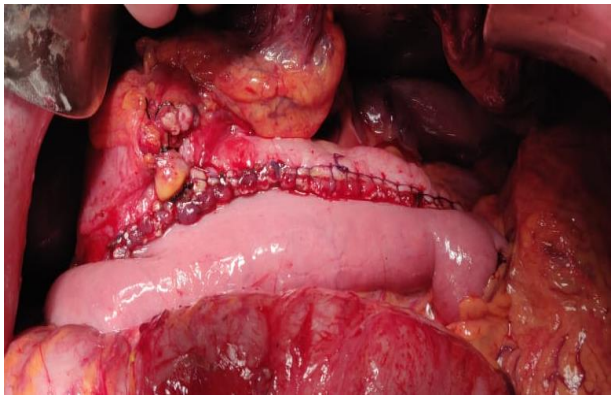

Figure 4: LPJ.

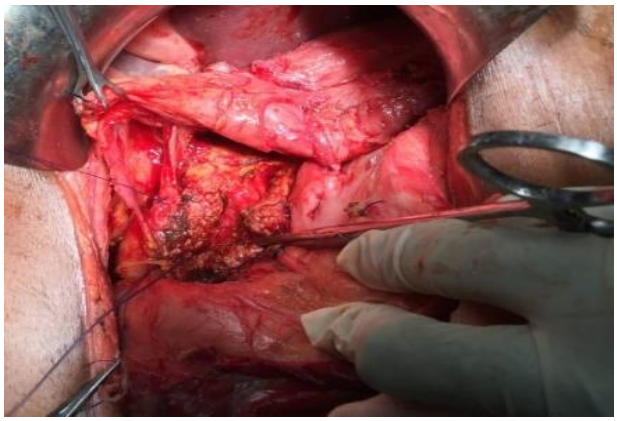

Figure 5: Pancreaticogastric anastomoses.

CONCLUSION

In conclusion, an algorithmic approach based on MRCP findings for pancreatic ascites and planning early surgery for internal drainage provides a single definitive step, as endotherapy may not be effective in all cases. Primary early surgery guided by MPD morphology in selected patients with chronic pancreatitis leads to faster recovery and also removes the ductal pathology and prevents the long-term malnutrition associated with pancreatic ascites.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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