International Surgery Journal
Gunturi SRV et al. Int Surg J. 2017 Feb;4(2):564-570
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: http://dx.doi.org/10.18203/2349-2902.isj20170088

Original Research Article

Surgical management of pancreatic ascites

Surya Ramachandra Varma Gunturi[1]*, Konidala M. V. S. Suman[3], Sambi Reddy G.[1], Dama V. L. Narayana Rao[1], Prabhakar B.[2], Malleshwara Rao G. L. N.[1]

[1]Department of Surgery, [2]Department of Medical gastroenterology, Osmania General Hospital, Osmania Medical College, Hyderabad, India
[3]Department of Surgery, Sri Venkateswara Medical College, Tirupati, India

Received: 06 January 2017
Accepted: 11 January 2017

***Correspondence:**
Dr. Surya Ramachandra Varma Gunturi,
E-mail: drsurya777@gmail.com

ABSTRACT

Background: Pancreatic ascites is a challenging problem faced by clinicians. The management requires a multidisciplinary approach. Timely surgical intervention is the key especially in patients with chronic pancreatitis and pancreatic ascites where conservative and endoscopic treatments were not successful.
Methods: The data was retrieved from a prospectively maintained database for a period of 4 years. A total of 14 patients were included. They were initially optimized with ascitic fluid drainage, nutritional supplementation either enteral or parenteral. Endoscopic retrograde cholangio pancreatography (ERCP) was done in patients with failed conservative treatment after 2 weeks. Endoscopic pancreatic stenting was attempted in proximal ductal disruptions. Nonresponders were taken up for surgery. The type of surgery was based on the site of leak and associated pancreatic pathology like pseudocyst.
Results: Initially three patients were responded to conservative management. ERCP was done in 9/14 patients. ERCP demonstrated leak of contrast into peritoneal cavity in 3 (3/9), leaking pancreatic pseudocyst in 3 (3/9) and non-visualisation of distal duct in 2 (2/9). ERCP and stenting of pancreatic duct was attempted in three patients and was successful in resolution of symptoms in one. Nine out of fourteen needed surgical intervention. Surgery was based on site of leak and presence of pseudocyst.
Conclusions: Majority of the patients in our study were ethanolics and a change in life style early in the course can prevent this morbid disease. Nasojejunal tube feeding with blendarized home feeds will improve the nutritional status. CECT abdomen and ERCP will give a road map in deciding the type of intervention. For proximal ductal disruption endoscopic stenting should be tried before going for a major surgical intervention. Surgery provides definitive cure.

Keywords: Internal pancreatic fistulae, Pancreatic ascites, Pancreatic ductal disruption

INTRODUCTION

Pancreatic ascites is an "exudative ascites caused by non-malignant disease of pancreas characterized by very high fluid amylase level over 1000U/L and ascitic fluid protein concentration more than 3gm/dl".[1-3]

The accumulation of Ascitic fluid is caused by leakage of pancreatic juice through a pseudocyst or a disruption in the pancreatic duct which is secondary to chronic pancreatitis.[4,5] It can occur rarely after acute pancreatitis or trauma.

Pancreatic ascites can be diagnosed easily once the possibility of its existence is considered.[5]

The objective of this study was to study the aetiology, clinical features and diagnostic approach in cases of

pancreatic ascites. The optimal uses of different modalities of treatment including various types of surgery were also studied.

METHODS

This study consists of 14 patients those who were diagnosed and treated as pancreatic ascites over a period of 4 years in Osmania Government General Hospital/Osmania Medial College, Hyderabad.

Inclusion criteria

Patients of pancreatic ascites with the background of chronic pancreatitis, pleural effusions associated with pancreatic ascites.

Exclusion criteria

Pancreatic pleural effusions without pancreatic ascites; pancreatic fistulas formed following right and left pancreatic resections with (or) without intestinal anastomosis.

Following symptoms and their duration were noted in all the patients. Pain, abdominal distention (increasing abdominal girth) nausea and vomiting, loss of appetite, weight loss, greasy frothy stools, upper GI bleed, difficulty in breathing. Past history of pain abdomen and personal history of intake of alcohol and duration were noted

Clinical examination included nutritional status, anemia, jaundice, lymphadenopathy and features of shock if any were looked into. Abdomen was examined for free fluid, tenderness, lumps and organomegaly.

Biochemical investigations

Ascitic fluid biochemistry including amylase and protein, ascitic fluid for adenosine deaminase (ADA) levels. Ascitc fluid for pathology includes its appearance, colour, coagulum present or absent, TC, DC, presence or absence of RBC, any other cells. Ascitic fluid was evaluated for malignant cells. Serum amylase, liver function tests, prothrombin time and other routine blood biochemical investigations were carried out

Radiological investigations

Plain X-ray abdomen in erect posture was done to rule out pneumoperitonium (as some cases were presented with pain abdomen and ascites). Chest X Ray PA view was done to see for pleural effusion, to rule out pulmonary Koch's. Ultrasound scan abdomen was done to look for the Pancreas size, texture, and calcification, pancreas duct size, abnormal dilatation and calculi, pseudocyst location and size, biliary tract assessment, ascites and other organs like liver and kidneys

Contrast Enhanced Computed Tomography (CECT) scanning of abdomen for confirmation of diagnosis of chronic pancreatitis along with findings of ascites and pancreatic pseudocysts and to rule out space occupying lesions in pancreas. Magnetic resonance cholangio pancreatography (MRCP) done in few cases for non-invasive assessment of ductal system. Upper GI endoscopy was performed in patients where symptoms of acid peptic disease were present.

ERCP (Endoscopic Retrograde Cholangio Pancreatography) was done to know the pancreatic ductal anatomy, for site of leak, size, strictures, dilatations and filling defects, associated pseudocysts (communicating), bile duct size, any stricture, dilatation and filling defects. It is preferred especially in cases where therapeutic stenting has a role. Patients were initially started on conservative management after confirmation of diagnosis of pancreatic ascites basically with an aim to improve the nutritional status for probable intervention while on constant observation for the resolution of ascites.

After diagnosis of pancreatic ascites nutritional improvement is by oral high protein and low fat feeds. If the patient does not tolerate oral feeds Naso jejunal feeds/TPN were given. Injection octreotide at a dose 100µgs 3 times a day was given for a period of 2 weeks along with percutaneous drainage of ascites. After a trail of conservative therapy ERCP was performed. Endoscopic intervention attempted for proximal pancreatic ductal disruptions. The type of surgery was planned depending on the site of leak and associated pancreatic abnormality as demonstrated by ERCP and CECT.

Statistical analysis

The results of our study were expressed in terms of percentages. The P value is calculated by using chi-square test for comparison of treatment outcome between conservative treatment and surgical treatment. $\chi^2 = \Sigma (O-E)^2$ for the value of chi-square above 3.84 with 1 degree of freedom will have a significant P value.

RESULTS

The age of 14 patients ranged from 12-50 years with a mean age of 41.5 years (Table 1).

Table 1: Patterns of age incidence.

Age Group (years)	No. of cases	Percentage of cases
5-15	2	14.28
16-25	2	14.28
26-35	2	14.28
36-45	5	35.71
46-55	3	21.42

Majority of the patients were male (12 out of 14).

Etiology

12 out of 14 patients were chronic alcoholics with more than 8-10 years duration of regular consumption of alcohol.

The presenting symptoms on admission were related to the presence of ascites. On examination mild tenderness was present in all patients but marked signs of peritonitis (or) pancreatitis were absent. The time gap from the onset of symptoms to hospitalization averaged 35 days (range 4 days -120 days). Weight loss was seen in 12 patients out of 14 (Table 2).

Table 2: Clinical presentation.

	No. of cases	Percentage
Abdominal distention	14	100
Pain:moderate to severe mild	3 6	21.42 42.85
Weight loss	12	85.72
Nausea and vomiting	4	28.57
Subcutaneous fat necrosis	1	7.14
Dyspnoea	2	14.28

There was a history of recurrent attacks of pain prior to ascites in 7 patients (50%)

Ascitic fluid analysis: In all 14 patients, the Ascitic fluid for amylase was greater than 1000 somogyi units/dl ranging from 1280 SU/dl to 28000 SU /dl. Ascitic fluid protein was elevated above 3g/dl (range3.1-7g/dl in all cases except in one whose value was 1.34 gms/dl). Ascitic fluid colour was yellow to red. Ascitic fluid cell count showed more no. of lymphocytes than neutrophils in majority of the patients. Ascitic fluid was negative for malignant cells in all. Ascitic fluid ADA levels were found to be within normal limits (normal up to 30 U /dl).

Serum amylase was slightly elevated in the majority of the cases except in one where it was elevated upto1, 790 SU/dl (range 28-1790 SU/dl). In our study serum proteins ranged from 2.2g/dl to 9.5g/dl (normal value 6-7.5g/dl)

Ultra sonogram abdomen demonstrated ascites in all cases, pseudocysts in 3 cases and chronic pancreatic changes in 2 cases.

CECT abdomen was done in 7 out of 14 cases. Presently our philosophy is to do a minimum pancreatic protocol CT including lower chest cuts in all cases and CECT chest if there is associated pleural effusion. Chronic pancreatitis changes along with ascites was seen in all 7 cases. Pseudocysts were seen in five cases (Figure 1).

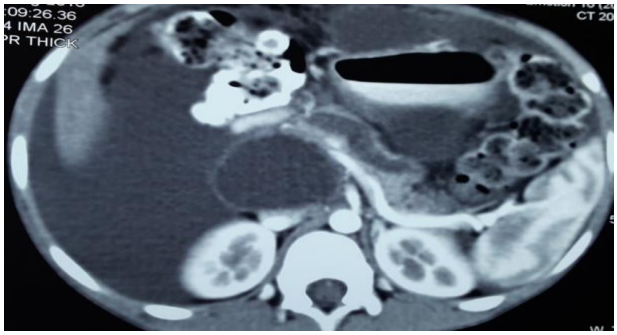

Figure 1: CECT abdomen demonstrating the dilated pancreatic duct with disruption at body and tail junction with ascites.

Initial conservative treatment with low fat, high carbohydrate and high protein diet orally was given to five patients. Recently we have adopted nasojejunal tube feeding with blenderized home feeds for patients who are not having adequate oral intake because of nausea. In 3 out of 4 patients in our study to whom NJ feeds were given improvement in nutritional status in terms of weight gain along with improved general well-being is seen. Total parenteral nutrition was given in three cases.

Ascitic fluid drainage was done in all cases 13/14 (except one), tube drain / PCD catheter was introduced for drainage in 8 out 13 cases. Repeated paracentesis in 5 out of 13 cases. In some patients as high as 3-6 liters were drained per day to relieve distress (range from 100ml to 6 liters / day) and especially when draining large amounts of Ascitic fluid supportive treatment was given in the form of albumin and fluids. In 6 cases injection octreotide was given at a dosage of 100µgs 3 times a day (for a period of 10 to 14 days).

Table 3: ERCP findings.

ERCP Findings	No of cases (n = 9)	Percentage
Ductal distuption	3	33.33
Pseudocyst	3	33.33
Tail is not filled and the region of leak not identified	2	22.22
No significant findings	1	11.11

The conservative treatment is carried out for different periods (2 weeks - 8 weeks) 36% (5) patients managed conservatively 14% (2 patients) expired during conservative management , 14% (2 patients) responded to conservative therapy, 7% (one case) endoscopic stenting was done in follow up. ERCP was performed in 9 cases out of 14 where conservative treatment was failed. It was performed in 8 out of 9 cases that were taken up for surgery (Table 3).

Table 4: Details of patient subjected to surgery.

Case	Period of conservative management	*ERCP	Surgery	Post op period (POD = post op day)	Post opl stay in hospital	Follow up	Follow up period
1	6 weeks	Main pancreatic duct(MPD) cut off at neck indicating ductal disruption	Distal pancreatectomy and splenectomy	Abdominal collection- USG guided aspiration done	2 weeks	Yes	3 years
2	6 weeks	Freely communicating pseudocyst in the body	Cystojejunostomy	Wound infection	3 weeks	Yes	3 years
3	4 ½ weeks	Not done	Distal pancreatectomy+ splenectomy +pseudocyst excision	Death on 3rd POD	-	-	-
4	3 weeks	Small pseudocyst in the head of pancreas MPD not filled	Small duct Disease, pancreas found inflammed, peritoneal lavage done	-	1 week	No	-
5	3 weeks	Disruption of pancreatic duct In head region with dilated PD	LPJ+ feeding jejunostomy	On 9th POD shock due to secondary haemorrhage	1 month	Yes	2 ½ years
6	2 months	Tail part of the PD not filled	Distal pancreatectomy+ splenectomy	Stress induced hypertension	1 week	Yes	2 ½ years
7	3 weeks	Pseudocyst at tail of pancreas	Distal pancreatectomy+ excision of pseudocyst+ splenectomy	-	10 days	Yes	1 ½ year
8	3 weeks	Leak is not demonstrated, MPD appears normal	Distal pancreatectomy+ splenectomy	-	8 days	Yes	1 year
9	2 weeks	Tail of pancreatic duct is not filled	Distal pancreatectomy+ splenectomy	-	8 days	No	-

*In all cases in ERCP-The biliary system was normal.

In 66.7% of cases, definite pathology was identified on ERCP (Figure 2).

In 11.1% of cases ERCP was not conclusive. In 22% cases ERCP revealed partial information which was supplemented with CT scan findings which demonstrated pseudocysts in the tail region in those cases.

In pancreatic ascites ERCP will display the ductal anatomy and CECT delineates the pancreatic pathology especially in non-communicating pseudocysts and calcifications which are not demonstrated by ERCP. In cases where intervention is not planned immediately MRCP is an option (Figure 3).

Both CECT abdomen and cholangiography are complimentary to each other and correlation with the help of these two investigation helps in the assessment of patient especially while planning for the type of surgery.

Nine patients out of 14 underwent operative therapy. Surgical treatment was individualized and surgery depended on the ductal anatomy as outlined by pre-

operative ERCP (8 OUT OF 9) and associated pancreatic pathology (Table 4).

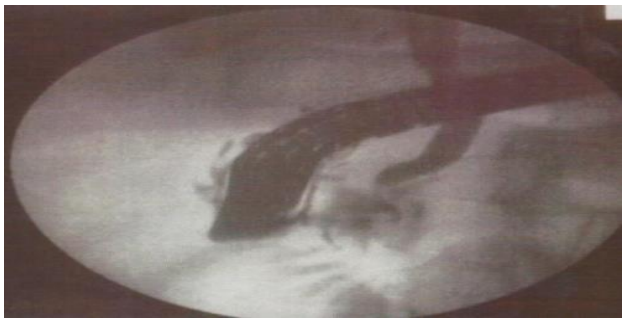

Figure 2: ERCP delineating the leakage of contrast without opacifying the MPD.

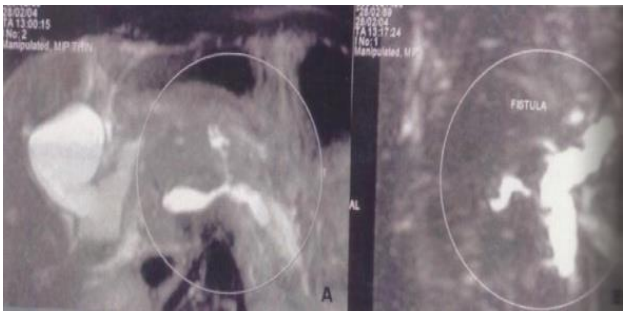

Figure 3: MRCP delineating the ductal disruption.

Distal pancreatectomy was done in 6 /9 (66.66%) LPJ in 1 (11.11%), cystojejunostomy in 1 (11.11%), definitive procedure was not done in 1 (11.11%).while doing distal pancreatectomy after resection of the distal pancreas proximal patency of the duct is verified by cannulating head and neck and was found satisfactory in all 6 cases the cannula was removed and duct was over sewn with 3.0 prolene without anastomosis with jejunal loop. Spleen sparing is not feasible due to inflammation.

In one case direct drainage of the disrupted pancreatic duct was done. It was wide opened and anastamosed to jejunal loop (longitudinal pancreatic jejunostomy). In one patient who had a large communicating pseudocyst anterior to the pancreas cystojejunostomy was performed. In one patient definitive procedure was not done because of small duct disease and severe inflammation in lesser sac.

In our study pleural effusion was seen in 6 cases along with pancreatic ascites. In one patient tube thoracostomy was done and in 2 patients it was resolved when definitive surgery for pancreatic ascites was done

One patient died on the third post-operative day due to respiratory complications along with poor nutritional status and sepsis. One patient developed shock with secondary hemorrhage on 9th post op day, and was managed successfully. Post-operative stay in the hospital was usually one week to three weeks in our series except one patient who was discharged after 4 weeks.

Follow up: six patients out of eight are under follow up (range 1 year to 3 years).

The overall results of our treatment were presented in tabular form (Table 5).

Table 5: The results of treatment in our study.

	No. of patients	Percentages
Non operative (n = 14)		
Success	3	21.42%
Failure	9	64.28%
Death	2	14.28%
Operative (n = 9)		
Success	7	77.7%
Definitive surgery was not done	1	11.1%
Death	1	11.1%

In statistical analysis the patient in whom definitive surgical procedure was not done was also considered as failure. The patient who was dead was also included in failure group (Table 6).

Table 6: Statistical analysis of results of treatment.

	Success	Failure and death	Total
Conservative	3	11	14
Surgery	7	2	9

The P value is calculated by using chi-square test for comparisons of results between conservative treatment and surgical treatment.

$\chi^2 = \Sigma (O-E)^2 / E$
$\chi^2 = 7.07654$

The value of P with I degree of freedom for a value of chi-square 7.0764 is less than 0.1. i.e. $p < 0.1$ which is having significant value. So, according to our study the surgical procedures for pancreatic ascites will give definitive cure and were superior to conservative management.

DISCUSSION

In the present study the mean age of the patients was 41.5 years which was similar to others series.[3-8] The male to female ratio is 6 :1 which was similar to Cameron J et al series.[1,7]

In our series 12 (85.7%) patients are chronic alcoholics. (Consumption of alcohol for more than 8-10 years is noted). In all the studies majority of the patients with pancreatic ascites were ethanolics.

Clinical features

Patients with pancreatic ascites present primarily with a history of slowly increasing abdominal girth and it was seen all of our cases. Because most of the patients have a significant history of alcoholic intake along with poor nutritional status, cirrhosis with ascites is often the admitting diagnosis.

In our series 9 out of 14 abdominal pain was present, in three patients it was moderate to severe in intensity and the rest of the six patients had mild pain. The mild pain was usually a reflection of rapidity with which ascites accumulates.[5] weight loss despite the massive ascites is common and was seen in 12 out of 14 patients. Subcutaneous fat necrosis was seen in one case in our series and it was seen in 2 out 34 patients in john Cameron series.[1,7] (Table 7) All the clinical features in our study were consistent with features described in the literature.[1,5-8]

Table 7: Comparison of clinical features.

Signs and symptoms	Present Study	Sankaran and Walt[5]	Parekh et al[8]	Kaman[6] et al PGI Chandigarh, India
Increasing abdominal girth	14 (100%)	31 (100%)	17/23 (73.91%)	5/6 (83.31%)
Weight loss	12 (85.72%)	14 (45%)	17/23 (73.91%)	-
Pain	9 (64.28%)	20 (65%)	3/23 (13%)	4/6 (66.66%)

The diagnosis of pancreatic ascites was made based on elevated amylase>1000 SU/dl and protein levels> 3gms/dl in the fluid obtained by paracentesis. Only one patient had an Ascitic fluid protein level<3 (1.4) Inspite of his Ascitic fluid amylase 5,628 somogyi units/dl. This could be due to poor nutritional status and low serum protein levels of this patient (total protein, 3.5 gms/dl, albumin 0.7 gms/dl, globulin 2.8 gms/dl).

The mean measured serum amylase was 544SU/dl (range 28-1790 SU /dl). Raised Serum amylase may be due to absorption of amylase from the Ascitic fluid into the circulation. Serum proteins of our patients were low which indicates poor nutritional status due to chronic alcoholism and due to exudative ascites. All these biochemical findings of our study were similar to the findings described in the literature.

Injection octreotide at a dose of 100µgs three times a day subcutaneously is administered for varying duration in 6 out of 14 patients. Due to economic reasons, it was not continued for longer durations in these patients. Segal et al obtained resolution of the pancreatic ascites in 8 of 9 patients with octreotide 100µgs TID subcutaneously after a mean duration of treatment of 23days.[8] In our study the role of octreotide was not studied effectively.

Patients who responded to conservative therapy responded early (within 10 days). So in our opinion long standing conservative therapy more than 2 weeks is not advisable to the patients of pancreatic ascites.

The outcomes of our results were comparable with the literature (Table 8 and Table 9).

It is better to approach the problem of pancreatic ascites with the attitude that the initial steps of conservative management will most likely be in preparation for an operation.

Table 8: Comparison of results of conservative management.

	Present study	Cameron et al[7]
Success	3 (21.42%)	7 (41.17%)
Failure	9 (64.28%)	6 (35.29%)
Death	2 (14.28%)	4 (23.52%)
Total patients	**14**	**17**

Table 9: Comparison of results of surgical management.

	Present study	Cameron et al
Success	7 (77.77%)	8 (100%)
Failure	1* (11.11%)	0
Death	1 (11.11%)	0
Total patients	**9**	**8**

*considered as failure because definitive surgical procedure was not done.

In pancreatic ascites surgery gives definitive cure with less morbidity and mortality. Because it has been shown in most of the published literature and also in our study that most cases ultimately required definitive surgery for cure if endoscopic stenting is not feasible.

CONCLUSION

Majority of the patients were ethanolics and a change in life style early in the course can prevent this morbid disease. Nasojejunal tube feeding with blendarized home

feeds will improve the nutritional status. CECT abdomen and ERCP will give a road map in deciding the type of intervention. For proximal ductal disruption endoscopic stenting should be tried before going for a major surgical intervention. Surgery provides definitive cure and type of surgery is determined by the site of ductal disruption and associated pseudocysts.

ACKNOWLEDGEMENTS

Authors would like to sincerely thank Mr. Satyanarayana Raju G., Prof. A. Subodh Kumar, Dr Suneetha, Sindhu, Sanjana, Ujwal and other team members for their support. Authors would like to profusely thank Prof. G. Nagaiah and Prof. Prakash Bhatia for their statistical analysis.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Broe PJ, Cameron JL. Pancreatic ascites and pancreatic pleural effusion in Bradley EL III (Ed), Complications of pancreatitis medical and surgical management WB Saunders, philadelphia Co; 1982:245-64.
2. Cabrera J. Ascitis De origin pancreatico. Med clin Barc. 1986;86;369-72.
3. Gomez- Cerezo J, Barbado Cano A, Suarez I, Soto A. Pancreatic ascites: study of therapeutic options by analysis of case reports and case series between the years 1975s and 2000. American J Gastroenterol. 2003;98(3):568-77.
4. Cameron JL, Anderson RD, Zuidema G. Pancreatic ascites, Surg Gynecol Obstet. 1967;125:328.
5. Saankaran S, Walt AJ, Pancreatic ascites. Arch Surg. 1976;111:430.
6. Kaman L, Behera A, Singh R, Katariya RN. Internal pancreatic fistulae with pancreatic ascites and pancreatic pleural effusions: Recognition and management. ANZ J Surg. 2001;71(4):221-5.
7. Cameron JL. Chronic pancreatic ascites and pancreatic pleural effusion. Gastroenterol. 1978;74:134-40.
8. Parekh D, Segal I. Pancreatic ascites and effusion. Risk factors for failure of conservative therapy and the role of octreotide. Arch Surg. 1992;127:707-12.

Cite this article as: Gunturi SRV, Suman KMVS, Reddy G S, Rao DVLN, Prabhakar B, Rao GLNM. Surgical management of pancreatic ascites. Int Surg J 2017;4:564-70.