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A Rare Case of Pancreatitis with Pancreatic Ascitis &Pancreatic Pleural Effusion

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Biliary pancreatitis, pancreatic pleural effusion, portal hypertension, ductwork lithiasis

Abstract
Background: Pancreatic ascites is an uncommon condition characterised by a large accumulation of pancreatic fluid in the peritoneal cavity. The most prevalent cause is chronic alcoholic pancreatitis 1. Ascites is a typical symptom of alcoholic liver disease, and it is mainly caused by portal hypertension. The other causes include ductal lithiasis 2, ampullary stenosis, cystic duplications of biliopancreatic channels, biliary pancreatitis 5, and pancreatic trauma.

1. Case Report

A 36yr old male patient came with complaint of abdominal distension for 2 months which is insidious in onset ; gradually progressive , associated with pain in umbilical region relieved on forward bending, complaint of breathlessness on exertion for 1week; grade 2 breathlessness; sudden onset,no history of orthopnoea & PND ; relieved on rest

Complaint of bilateral lower limb swelling for 1 month, complaint of high coloured urine for 2weeks. No h/o nausea,vomiting, loose stools,constipation,chest pain , Palpitations ,burning micturition, decreased urine output,loss of consciousness ,ent bleed.

Patient is known alcoholic for 11 years- brandy and whiskey 280ml/day

Known smoker 10 beedis/day, no other known comorbidities

O/E - patient is conscious,oriented, afebrile

S/E

CVS -s1;s2 +

Rs - BAE +; basal creps+

P/A- soft; non tender; fluid thrill+ ; shifting dullness+

Cns - nfnd

Vitals- stable

Investigations

TLC-17040

PCV-37.8

U/R -plenty of Rbcs

Pus cells-8-10

Urine c/s -no growth

FLP-normal

RFT-normal

LFT-Sr.albumin. -2.2

Alkaline phosphatase-239

PT-18.9(elevated)

PTT-34.3(elevated)

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INR-1.7(elevated)

ASCITIC FLUID TAPPING DONE BOTH DIAGNOSTIC & THERAPEUTIC AND ITS ANALYSIS AS FOLLOWS

Culture & sensitivity - no growth

ascitic fluid chloride -102.0

Ascitic fluid for LDH - 557(increased)

Ascitic fluid for protein- 3.2(increased)

Ascitic fluid for ADA -16.41(normal)

Ascitic fluid for glucose - 52(normal)

Ascitic fluid for total count - 906(N-71%,L-29%)

Ascitic fluid for amylase -21024(increased)

Ascitic fluid For AFB-negative

Pleural tapping done & pleural fluid analysis as follows

Pleural fluid for ADA- 12.80(n)

Pleural fluid for LDH-530(increased)

Pleural fluid for glucose-74(n)

Pleural fluid for protein-3.4

Pleural fluid for amylase-10040(elevated)

Pleural fluid for lipase-77(elevated)

Pleural fluid for tc-327(L-83%,N-15%,E-2%)

Pleural fluid for AFB-negative

CECT ABDOMEN DONE AND IMPRESSION IS

Cystic lesion in the pancreatico-duedenal groove arising from head of pancreas

with associated peri pancreatic fat stranding.

Fat stranding around D2 segment of duodenum.

Moderate ascites,

Features of groove pancreatitis with pancreatic pseudocyst.

Right renal calculus.

USG ABDOMEN DONE AND IMPRESSION IS

Liver shows heterogenous parenchymal echoes with nodular surface.

Moderate-severe ascites,

Bilateral pleural effusion

-Features suggestive of liver parenchymal disease

SGE opinion sought in view of pancreatic ascites and advised for ERCP with pancreatic stenting or cysto duodenostomy.

Urology opinion taken sought in view of renal stones and advised for intervention after resolving of Acute pancreatitis.

Ascitic tapping done and pigtail catheter drainage for 3 days and ERCP done then pancreatic stenting was done and supportive treatment given and patient symptomatically improved

2. Background

Hepatic cirrhosis is frequently the cause of massive ascites in a persistent alcoholic patient [1]. Patients with prolonged drinking and pancreatitis who arrive with ascites should be suspected of having pancreatic ascites3 [2]. The most probable cause is a leakage or ductal rupture of a pancreatic pseudocyst [3]. The diagnosis is made based on the presence of ascitic fluid amylase (> 1000 U/L). Acute pancreatitis (8.6%), chronic pancreatitis (83%) and trauma are the most frequent causes of ductal disruption (3.6 percent). Some of the medical therapies include withholding oral feedings, total parenteral nutrition 4 (TPN), paracentesis, and octreotide injection [4]. In the event that medical treatment is unsuccessful, interventional therapy, such as endoscopic transpapillary pancreatic duct stenting, or surgery, such as cystogastrostomy, cystenterostomy, pancreatic sphincterectomy, or partial pancreatic resection, may be needed. We describe a case of significant ascites in a patient with chronic pancreatitis brought on by ongoing alcohol consumption. To effectively manage the patient, a mix of medical and interventional treatment was applied.

3. Discussion

Pancreatic ascites refers to the buildup of pancreatic fluid in the peritoneal cavity. One percent of the population suffers from pancreatic ascites, which strikes men more often than women (2:1) and strikes them between the ages of 20 and 50 [8]. An common alcoholic liver disease consequence is ascites, which is often brought on by portal hypertension6. The

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remaining instances are brought on by cystic duplications of biliopancreatic channels7, ductal lithiasis, ampullary stenosis, biliary pancreatitis, pancreatic trauma, or biliary pancreatitis [9]. It affects around 4% of persons with chronic pancreatitis and between 6 to 14 percent of patients with pancreatic pseudocysts. Additionally, it may occur as a consequence of abdominal physical trauma or acute pancreatitis-related duct dehiscence. For this diagnosis, patients with persistent ascites and a history of drinking, chronic pancreatitis8, or abdominal injuries should be assessed [8]. In 80% of instances, leakage from a pseudocyst connected to the ductal system causes pancreatic ascites; in the other 20% of cases, ductal rupture without a pseudocyst causes the condition [5].

Pancreatic ascites often manifests as mild stomach discomfort, a diminished appetite and feeling of fullness, weight loss, and expanding ascites. Common symptoms include ascites that has just developed, a recent episode of acute pancreatitis, or a history of chronic pancreatitis. However, among alcoholics, these symptoms can not be present, leading to the diagnosis being mistaken for cirrhosis. A paracentesis may be performed, and the fluid analysed for amylase and protein levels to get a diagnosis. Exudative ascites 9from pancreatic disease may be distinguished from those caused by cirrhosis, tuberculosis, or carcinomatosis by the presence of a high amylase content in the ascitic fluid (often above 1000 IU/L) and a protein concentration of > 3 g/dl.In a small percentage of cases, the origin is unknown. Pleural effusion, in addition to ascites, may be noted in such instances [10].

To rule out pseudocysts, an abdominal CT scan should be performed once the diagnosis has been established. Endoscopic retrograde cholangiopancreatography (ERCP) 10 has been suggested as a tool for locating ductal obstruction or leakage so that stenting can be performed if necessary. For candidates who are unable to undergo ERCP, magnetic resonance cholangiopancreatography (MRCP) 11can be used to determine the structure of the pancreatic duct and any anomalies [11]. However, due to a lack of evidence, the usefulness of MRCP in the diagnosis of pancreatic ascites remains unclear.

The treatment of pancreatic ascites is debatable [8]. Due to the rarity of the illness, there are no randomised control studies .] The cautious management of a patient with pancreatic ascites involves stopping oral nutrition and starting TPN. They assist in lowering pancreatic secretions. Only about a third of individuals benefit from this conservative approach, and other patients could need somatostatin analogues like octreotide 12, diuretics, or recurrent paracentesis. In 18 patients with pancreatic adenomas or external pancreatic fistulas, Segal et al. undertook a prospective study to assess Sandostatin (octreotide), a long-acting somatostatin analogue, for its effectiveness (12). The ascites disappeared after nine out of ten patients, on average, after 22 days (+/- 3 days). Seven out of the eight patients had their external fistulas repaired, and they were all high-output fistulas. The importance The overall duration of therapy is uncertain, although a 4- to 6-week trial is recommended, with interventional therapy considered if the condition does not improve. This method resulted in less than half of the patients healing, 15% overall mortality, and 15–25 percent recurrence [9].

Thankfully, our patient responded well to cautious treatment. Patients need interventional therapy once conservative treatment fails, which might be endoscopic or surgical. While large-volume paracentesis is the gold standard for evaluating patients with pancreatic ascites, endoscopic retrograde cholangiopancreatography (ERCP) may assist pinpoint the source of the leak and facilitate the placement of a transpapillary stent 12to bypass the blockage. Patients can also have endoscopic or percutaneous pseudocyst drainage at the same time [8].

When conservative therapy fails to work after 3–4 weeks, surgical surgery is advised. ERCP and contrast-enhanced CT are used to determine the location of the leak and any related pancreatic abnormalities. Pseudocysts are frequently removed through distal pancreatectomy when the pancreatic tail is the source of the leak or are drained with cystogastrostomy, cystojejunostomy, or cystoduodenostomy. Research indicates that for patients with pancreatic ascites and/or pleural effusion who have not responded to pharmacological treatment, internal pancreatic drainage is the most effective surgical alternative. In cases when pancreatic resection is not an option, external drainage may be employed [7]. Roux-en-Y jejunal loops are often used to drain pancreatic duct fistulas.Patients who underwent surgical surgery without ERCP had recurrence rates ranging from 50 to 64 percent [8]. The mortality rates for surgical and medicinal treatments have been shown to be similar (15–25%) [8].Patients who underwent surgical surgery without ERCP had recurrence rates ranging from 50 to 64 percent [8]. The mortality rates for surgical and medicinal treatments have been shown to be similar (15–25%) [8].

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With a conservative strategy, we were able to properly manage our patient. Our example emphasises a few key messages. Given the great prevalence of ascites, we frequently characterise it as cirrhotic or related to portal hypertension; nevertheless, pancreatic ascites should also be included in the differential. It's critical as a clinician not to overlook cases with pancreatic ascites. A history of acute abdominal trauma, recurring emergency department visits due to stomach discomfort in chronic drinkers, or past or present pancreatitis may all be useful in spotting such circumstances. Additionally, a precise assessment of the SAAG may be made to limit the diagnosis. Paré et al. [13] state that calculating SAAG provides the most definitive separation between ascites caused by liver illness and ascites caused by a tumour. Second, such circumstances are rare, and if they do arise, they may be handled with medication and/or interventional treatment.

4. Conclusion

The most prevalent cause of pancreatic ascites is alcoholic chronic pancreatitis, which is a rare condition. The other instances are brought on by ampullary stenosis 1, ductal lithiasis, biliary pancreatitis, pancreatic trauma, cystic duplications of biliopancreatic channels, or biliary pancreatitis.. In the presence of pancreatic illness, high ascitic fluid amylase (>3 times that of plasma), raised total ascitic protein level (> 3 g/dl), and low SAAG (1.1 g/dl) usually confirm the diagnosis, and such cases can be handled conservatively.

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ETHICAL CONSENT

Patients included in the study had provided informed consent

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CONFLICT OF INTREST

The authors declare that was no conflict of intrest

References

[1] Gomez-Cerezo J., Cano A.B., Suarez I., Soto A., Rios J. Pancreatic ascites: Study of therapeutic options by analysis of case reports and case series between the years 1975 and 2000. Am. J. Gastroenterol. 2003;98:568–577. doi: 10.1016/S0002-9270(02)06032-X. [PubMed] [CrossRef] [Google Scholar]

[2] Bracher G.A., Manocha A.P., DeBanto J.R., Gates L.K., Slivka A., Whitcomb D.C., Martin S.P. Endoscopic pancreatic duct stenting to treat pancreatic ascites. Gastrointest. Endosc. 1999;49:710–715. doi: 10.1016/S0016-5107(99)70287-7. [PubMed] [CrossRef] [Google Scholar]

[3] Smith E.B. Hemorrhagic ascites and hemothorax associated with benign pancreatic disease. AMA Arch. Surg. 1953;67:52–56. doi: 10.1001/archsurg.1953.01260040055008. [PubMed] [CrossRef] [Google Scholar]

[4] Dhar P., Tomey S., Jain P., Azfar M., Sachdev A., Chaudhary A. Internal pancreatic fistulae with serous effusions in chronic pancreatitis. Aust. N. Z. J. Surg. 1996;66:608–611. doi: 10.1111/j.1445-2197.1996.tb00830.x. [PubMed] [CrossRef] [Google Scholar]

[5] Kozarek R.A. Management of Pancreatic Ascites. Gastroenterol. Hepatol. 2007;3:362–364. [PMC free article] [PubMed] [Google Scholar]

[6] Schoefl R., Haefner M., Pongratz S., Pfeffel F., Stain C., Poetzi R., Gangl A. Endoscopic treatment of fistulae and abcess in pancreatitis: Three case reports. Endoscopy. 1996;28:776–779. doi: 10.1055/s-2007-1005604. [PubMed] [CrossRef] [Google Scholar]

[7] Moosa A.R. Surgical treatment of chronic pancreatitis: An overview. Br. J. Surg. 1987;74:661–667.

[8] Cameron J.L., Keiffer R.S., Anderson W.J., Zuidema G.D. Internal pancreatic fistulas: Pancreatic ascites and pleural effusions. Ann. Surg. 1976;184:587–593. doi: 10.1097/00000658-197611000-00009. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[9] Parekh D., Segal I. Pancreatic ascites and effusion. Risk factors for failure of conservative therapy and the role of octreotide. Arch. Surg. 1992;127:707–712.

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CODEN: JCLMC4
Journal of Coastal Life Medicine

doi: 10.1001/archsurg.1992.01420060083012. [PubMed] [CrossRef] [Google Scholar]

[10] Burgess N.A., Moore H.E., Williams J.O., Lewis M.H. A review of pancreatico-pleural fistula in pancreatitis and its management. HPB Surg. 1992;5:79–86. doi: 10.1155/1992/90415. [PMC free article] [PubMed] [CrossRef] [Google Scholar]

[11] Da Cunha J.E., Machado M., Bacchella T., Penteado S., Mott C.B., Jukemura J., Pinotti H.W. Surgical treatment of pancreatic ascites and pancreatic pleural effusions. Hepato-Gastroenterology. 1995;42:748–751. [PubMed] [Google Scholar]

[12] Eckhauser F., Raper S.E., Knol J.A., Mulholland M.W. Surgical management of pancreatic pseudocysts, pancreatic ascites, and pancreatopleural fistulae. Pancreas. 1991;6:S66–S75. doi: 10.1097/00006676-199101001-00011. [PubMed] [CrossRef] [Google Scholar]