Case Report

A Man with Progressive Swelling of Abdomen : Uncommon Presentation of a Common Disease

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Ascites is a rare manifestation of chronic Pancreatitis. Patients usually present with progressive ascites with past history of Pancreatitis. But sometimes Pancreatic Ascites may present without any history of pain abdomen suggestive of Pancreatitis. We will discuss a case of painless Pancreatic Ascites without any history of hematemesis or melaena, pedal oedema, respiratory distress, abdominal pain or trauma. CT scan of abdomen revealed atropic pancreas with calcification, peripancreatic collection and ascites. Amylase level in ascitic fluid was high. MRCP showed Cholecystitis with dilated common bile duct with complete loss of pancreatic duct architecture. Patient was initially managed medically followed by surgical intervention.

Pancreatitis should be suspected as etiology of progressive ascites in chronic alcoholic patients with or without typical abdominal pain as early management both medically as well as surgically is the cornerstone of treatment success.

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Ascites is a rare presentation of chronic Pancreatitis. Patients present with progressive ascites with prior history recurrent pain abdomen. But sometimes Pancreatic Ascites may also present without any prior history of typical abdominal pain suggestive of acute or Chronic Pancreatitis. Pancreatic Pseudocyst leakage or ductal disruption are among the commonest etiologies Chronic Pancreatic Ascites. It is more common in chronic alcoholic patients. Pncreatic Ascites may present with recurrent or refractory ascites leading to confusion with malignant ascites. Here we are going to present a case of pancreatic ascites without any prior abdominal pain in an alcoholic person.

Case Report

A 55-year-old non-diabetic, non-hypertensive man from West Bengal, India presented with gradually progressive distension of abdomen for past 2 months with progressive loss of weight. He had history of regular alcohol intake for past 20 years but no history of smoking. There was no history of Hematemesis or Melaena, Clay-coloured stool, Pedal oedema, Shortness of breath, Pain abdomen, Fever or Abdominal Trauma. On examination mild pallor and huge ascites were present. Vitals were stable. No Spider Navei, Venous Prominence, Palmer Erythema, Gynaecomastia or Flapping Tremor were present. Complete hemogram showed Hemoglobin (Hb) - 9.2 gm%, Total Leukocyte count - 4300 cells/ cmm, Platelet count - 2,30,000 cells/ cmm. Liver Function Test (LFT) showed Bilirubin (total) – 0.3 mg/dL, Albumin - 3.2 gm/dL, Globulin - 3.4 gm/dL, Serum Glutamic-Oxaloacetic Transaminase (SGOT) – 29 IU/L, Serum Glutamic-Pyruvic Transaminase (SGPT) – 11 IU/L, Alkaline Phosphatase – 158 IU/L, Gamma Glutamyl Transferase – 36 U/L. Enzyme-linked Immunosorbent Assay (ELISA) for HIV I & II was non reactive, Hepatitis B Surface Antigen (HBsAg) and anti HCV were also non reactive. Ultrasonogram of whole abdomen showed normal size and echotexture of Liver, Spleen- 9.5 cm, diameter of Portal vein - 11.2 mm and huge ascites, no pseudocyst of pancreas. Ocult blood in stool was absent. Upper GI endoscopy did not show any varices or portal gastropathy. Ascitic fluid study revealed, Cell count – 200/ cmm , 95% Lymphocytic, Total Protein – 3.1 gm/dL, Sugar – 85 mg/dL (Corresponding blood sugar was 110 mg/dL), Serum Ascites Albumin Gradient (SAAG) – 0.9, Adenosine Deaminase (ADA) – 6.0 U/L, Cartidge Based Nucleic Acid Amplification Test (CBNAAT) – negative. Patient was having shortness of breath for which Therapeutic Paracentesis was done which was followed by rapid reaccumulation of fluid. Thus repeated ascitic fluid Paracentesis had to be done. Malignant ascites was suspected and for which tumour markers were ordered. Tests revealed only mild elevation of CA 19.9 (serum α-feto protein - 5.9 ng/ml, β-hCG – 2.1 mIU/ml, Carcino Embryonic Antigen (CEA) – 1.61 ng/ml,

Editor's Comment :

- When an alcoholic patient presents with Ascites, Pancreatic Ascites should be kept in mind as a differential beside more common entities like Chronic Liver Disease or Tuberculosis.
- Rarely patients may present as painless abdominal distension and even without any past history of Acute or Chronic Pancreatitis.

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CA19.9 –337.18 U/ml). For further evaluation of organ involvement, contrast enhanced CT scan of whole abdomen was done, which ultimately clinched the diagnosis, showing atropic pancreas with calcification, peripancreatic collection and ascites (Fig 1). Ascitic fluid amylase was found to be very high (5379 U/L). Serum amylase was 340 U/L [Normal range 40-120 U/L]. MRCP showed Cholecystitis with dilated common bile duct traced upto distal end with complete loss pancreatic duct architecture (Fig 2). Patient was treated conservatively with nothing per mouth, intravenous fluid and antibiotics, diuretics, therapeutic paracentesis and octreotide. After initial stabilisation, patient was referred to the Gastroenterological Surgery Department for Definitive Surgical Management and was underwent Pancreatic Duct Stenting and Reconstructive Surgery. Patient was counselled for alcohol cessation and advised for follow-up regularly. After 3 months in the follow-up clinic, patient was found in stable condition without any significant ascites or persisting pain abdomen.

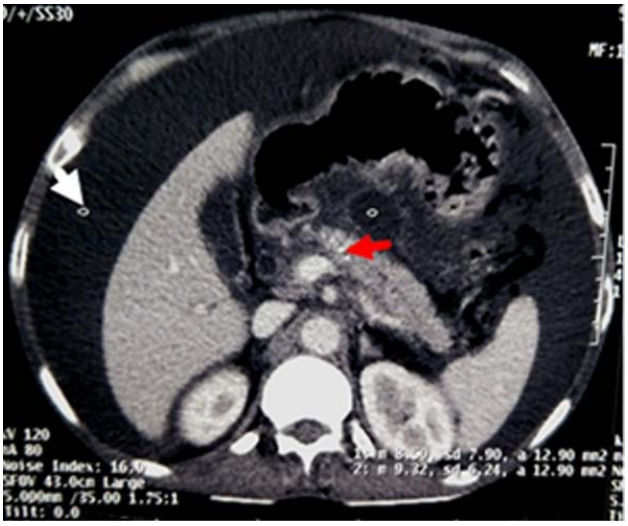

Fig 1 — Contrast enhanced CT scan of whole abdomen showing huge ascites (white arrow), atrophic pancreas with loss of architecture and multiple hyper-dense opacities in the body and head of pancreas suggestive of calcifications (red arrow)

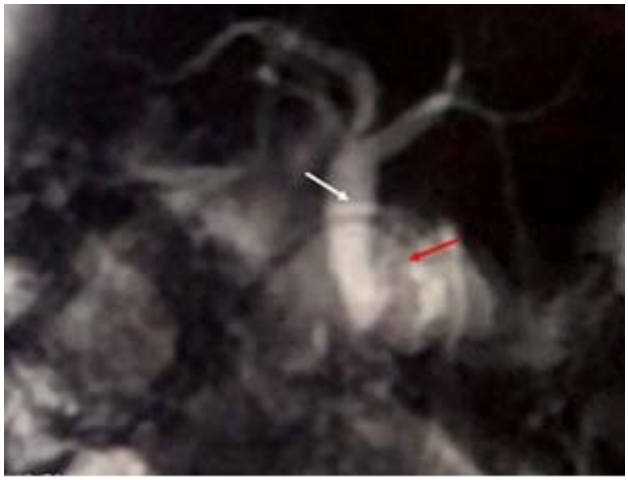

Fig 2 — MRCP showing dilated common bile duct traced up to distal end (white arrow) with loss of normal pancreatic duct architecture suggestive of duct disruption (red arrow)

Discussion

Ascites is one of the commonest manifestations of Chronic Liver Disease and is usually consequence of the two of its common entities- portal hypertension and hepatocellular failure. Pancreatitis is one of the rare etiologies of ascites in patients with habit of long-standing alcoholism and prior history of pain abdomen. But Pancreatic Ascites without any prior history of pain abdomen is a rarest option. Pancreatic ascites is characteristically exudative in nature, with high amylase concentration in ascitic fluid (more than 1000 IU/L) and protein concentration of more than 3 gm/dL[2] that differentiate it from Malignant Ascites and ascites secondary to Cirrhosis of Liver or Tuberculosis. Pancreatic ascites is a rare entity and is reported in around 3-4% of patients with chronic pancreatitis and 6-14% of patients with Pseudocyst of Pancreas[3,4].

Common entities causing Pancreatic Ascites are Chronic Pancreatitis, Ductal Lithiasis, Ampullary Stenosis, Pancreatic Trauma, Ductal Disruption and Cystic Duplications of Biliopancreatic Ducts[2]. Pancreatic ascites is more common in men (male : female = 2:1) and between the age of 20-50 years[2]. The clinical manifestations are progressive ascites, weight loss with abdominal pain. Pancreatic ascites should be thought as differential in patients with progressive ascites with history of Alcoholism, Chronic Pancreatitis or Abdominal Trauma[5].

Therapy for Pancreatic Ascites has many controversies in part of both Medical as well as Surgical management[6]. No randomized control studies are available regarding management of the condition probably due to the rarity of the entity. Though no consensus is available on the management, Chebli *et al*[7] proposed that the management should be guided by the Ductal Anatomy defined by the Endoscopic Retrograde Pancreatography (ERCP). Conservative therapy includes the somatostatin analogues[8,9] and keeping the patient Nothing Per Mouth (NPM) to reduce pancreatic secretion. Initial continuous octreotide infusions followed by subcutaneous injections may be suggested. Patient may be managed with repeated ascitic fluid drainage for symptomatic relief and parenteral nutrition. Long acting Intra-muscular Octreotide may be given once a month as maintenance therapy.

Surgical intervention is also recommended when there is no response to conservative medical management for consecutive 3 - 4 weeks. Recurrence rate has been reported around 60% in patients undergoing surgical intervention without prior ERCP[10,11]. Mortality rates have been reported to be comparable with both surgical and medical therapies (15-25%)[12].

Conclusion

Pancreatic ascites should be considered in the differentials of progressive ascites in chronic alcoholic patients with or without typical pain abdomen suggestive of pancreatitis as early diagnosis and treatment leads to favorable outcome.

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