Rare disease

Pancreatic ascites in the setting of portal hypertension

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Summary

Pancreatic ascites is a rare cause of ascites and develops largely as a complication of chronic pancreatitis or sometimes due to duct injury during surgical procedures. The entity may mimic spontaneous bacterial peritonitis or ascites due to portal hypertension. Here, the authors discuss a case of pancreatic ascites developing in the setting of alcoholic liver disease with portal hypertension. The patient had features of chronic pancreatitis with pancreatic duct fistula and was managed with stenting of the pancreatic duct.

BACKGROUND

Pancreatic ascites accounts for only 1% cases of ascites. Being rare and peculiar entity, its diagnosis and management differs considerably.

CASE PRESENTATION

A 30-year-old male presented with generalised abdominal distension associated with mild vague abdominal pain for 15 days. He had weight loss of 3 kg despite abdominal distension. There was no history of haematemesis, pedal oedema, oliguria, liver disease or cardiac symptoms. He gave history of an episode of vague epigastric pain 3 months back but had not taken any treatment for it. He used to drink around 500 ml of country liquor daily for the past 5 years. His family history was not contributory.

Physical examination revealed an averagely built male without any signs of liver cell failure. Jugular veins were normal. Abdominal examination revealed tense ascites. The liver and spleen were not palpable. There was mild tenderness throughout the abdomen without any guarding or rigidity.

INVESTIGATIONS

Investigations revealed haemoglobin of 14.5 g/dl (13.3–16.2 g/dl), white blood cell count 10 900/mm³ (4000–10 000/mm³), platelet count 1 56 000/mm³ (1 50 000–4 50 000/mm³), serum aspartate amino-transferase 41 IU/l (12–38 IU/l), serum alkaline phosphatase 248 IU/l (20–140 IU/l), total bilirubin 0.6 mg/dl (0.3–1.3 mg/dl), serum albumin 2 g/dl (4–5 g/dl), serum amylase 311 IU/l (20–96 IU/l) and serum lipase 241 IU/l (3–43 IU/l). Ascitic fluid showed, 200 cells/ml; 70% polymorphs, 30% lymphocytes, total protein 2.21 g/dl and albumin 1.2 g/dl. There were no pus cells, organisms or malignant cells. Culture did not grow any organisms. Ascitic fluid adenosine deaminase was 24 IU/ml (14–40 IU/ml). Ascitic fluid amylase was 4845 IU/l (normally half of serum amylase value).

Ultrasonography revealed altered echo-texture with fatty infiltrates and severe ascites, suggestive of portal hypertension. Oesophago-gastro-duodenoscopy revealed

two columns of small oesophageal varices with portal hypertensive gastropathy.

Contrast enhanced CT of the abdomen revealed atrophied pancreatic parenchyma with heterogenous enhancement and calcification, suggestive of chronic pancreatitis (figure 1). There was a rent in pancreatic duct at the body of the pancreas (figure 2) communicating with the retroperitoneum, with pseudocyst formation. There were multiple infarcts in spleen with thrombus in portal vein.

DIFFERENTIAL DIAGNOSIS

- ▶ Ascites with portal hypertension
- ▶ Spontaneous bacterial peritonitis
- ▶ Budd–Chiari syndrome
- ▶ Abdominal tuberculosis.

TREATMENT

Patient was kept nil per oral for 2 days, until ultrasound confirmed absence of bowel injury/infarction and he passed stools. Following this, he was started on oral sips with salt restriction (less than 5 g a day). He received spironolactone 100 mg/day with frusemide 40 mg/day (intravenous) with repeated large volume paracentesis (5 l in 1 week). Octreotide could not be used due to financial constraints. Patient had rapid filling ascites that did not respond to this optimum medical treatment and repeated paracentesis. Endoscopic retrograde cholangiopancreatography (ERCP) was subsequently done that revealed a rent in pancreatic duct at junction of body and tail which was cannulated with a 7 french catheter, and a stent was placed.

OUTCOME AND FOLLOW-UP

A final diagnosis of pancreatic ascites secondary to pancreatic duct fistula due to chronic pancreatitis with underlying alcoholic liver disease with portal hypertension was made. The patient responded to the stenting procedure with progressive decline in abdominal girth. Serum amylase decreased to 862 IU/l at the end of first week. Patient was discharged after 2 weeks.

Figure 1 CECT abdomen showing atrophied pancreatic parenchyma with heterogenous enhancement and calcification, suggestive of chronic pancreatitis.

Figure 2 CECT abdomen showing a rent in pancreatic duct at the body of the pancreas.

DISCUSSION

Pancreatic ascites accounts for 1% of all ascites cases. History of chronic pancreatitis is common aetiology in approximately 80% of cases.^{1 2} Pancreatic ascites occurs only in 3.5% of chronic pancreatitis and 6–14% of patients

with a known pancreatic pseudocyst.³ Pancreatic ascites requires ascitic fluid amylase greater than 1000 IU/l, a high fluid protein concentration (usually greater than 3 g/dl).¹ The polymorph count in pancreatic ascites is often over 250 cells/ml⁴ due to which pancreatic ascites can be

confused with spontaneous bacterial peritonitis, as was the case with our patient. Serum amylase may also be mildly elevated. Apart from ascites, imaging studies may reveal evidence of chronic pancreatitis such as pancreatic calcifications or a dilated pancreatic duct; given that 80% of cases are due to chronic pancreatitis.³ Pancreatic pseudocysts, as site of the leak are common finding on imaging in about 80% of cases.³ Our patient had pancreatic calcifications and a pseudocyst. Endoscopic features shows typical finding of extravasation of contrast into the peritoneum from a pseudocyst that communicates with the pancreatic duct. Pseudocyst is seen in 80% of cases and leakage of contrast from the pancreatic duct is seen on ERCP.³ Leakage occurs at equal frequency in each portion of the pancreas.² Pancreatogram may be completely normal or may show common findings of chronic pancreatitis, including strictures, pancreatic ductal dilatation and abnormal side branches.

Conservative management consists of keeping the patient nil per oral, giving parenteral nutrition and repeated paracentesis. Failure rate with conservative treatment is about 55%.¹ Either somatostatin or octreotide can be used in treatment of pancreatic ascites with patients who had failed conservative management.^{1 5 6} Octreotide decreases pancreatic secretions, allowing a chance for the leak to seal. In recent years, ERCP is an emerging treatment for pancreatic ascites; cannulation of duct and placement of stent may help in healing of the rent, thereby avoiding a major surgery.

Learning points

- Pancreatic ascites is rare and may be superimposed on portal hypertension.
- A history of pancreatitis as an underlying cause may not always be elicitable.
- ERCP offers both diagnostic as well as therapeutic advantage in the management and may help avoid major surgery.

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Competing interests None.

Patient consent Obtained.

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