Case Series

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Extra-splanchnic vascular thrombosis in pancreatic ascites: a case series

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ABSTRACT

Pancreatic ascites is a dreaded complication of acute or chronic pancreatitis. It's a therapeutic challenge with its effect on nutrition and the associated complications. Pancreatic ascites can lead to formation of pseudo aneurysm by direct erosion of vessel, but extra splanchnic vascular complications are rare. In this series of 5 cases, we report an unusual incidence of extra splanchnic vascular complications in cases of pancreatic ascites. Out of the 21 cases of pancreatic ascites treated over a period of 2 years (February 2021 to February 2023) in our centre, we encountered 5 cases with vascular complications in the extra splanchnic circulatory system like pulmonary, cerebral, and cardiac. All these cases didn't show a thrombus in IVC or leg veins in Doppler study. Among these 5 patients, three succumbed to the complications. This highlights the need of targeted evaluation for thrombosis in systemic circulation, especially pulmonary circulation for all patients of pancreatic ascites. Though a causal association cannot be proved by this report, the increased incidence of pulmonary thrombosis in pancreatic ascites patients need further evaluation and work up and detailed analysis. Targeted evaluation and urgent treatment in the form of anticoagulation along with surgical or endoscopic treatment of pancreatic leak may halt the ongoing pro coagulation cascade.

Keywords: Pancreatic ascites, Pulmonary thrombosis, Extra splanchnic vascular thrombosis

INTRODUCTION

Pancreatic ascites is a dreaded complication of acute or chronic pancreatitis. It's a therapeutic challenge with its effect on nutrition and the associated complications.

Vascular complications are commonly seen in acute pancreatitis; it can be local complications like splenoportal thrombosis or distant venous thrombosis like DVT or pulmonary thrombosis.

Pancreatic ascites can also lead to formation of pseudo aneurysm mainly in splenic artery and GDA. Pancreatic ascites also causes a systemic inflammatory situation with a wide range of effects.

CASE SERIES

Case 1

A 49-year-old gentleman admitted with c/o abdominal pain and distension and anorexia of 2-months duration-treated elsewhere. He was a known case of ethanol related CCP. On evaluation he was diagnosed as a case of pancreatic ascites with ascitic fluid amylase level of 23500 u/ml. He presented with features of sepsis, initially managed conservatively with antibiotics and nutritional care. He was optimized and planned for MPD stenting. On cardiac evaluation for he was found to have right atrial thrombus- for which he was started on anticoagulants-further evaluation with cardiac magnetic resonance

imaging (MRI) was not possible due to poor GC (Figure 1). He was put on a therapeutic dose of unfractionated heparin and optimized. He was taken up for MPD stenting. Unfortunately, he suffered cardiac arrest secondary to massive pulmonary embolism from the right atrial clot. After resuscitation and 2 days on ventilator support, he expired.

Case 2

A 31-year-old gentleman complained of pain abdomen and abdominal distension for 1 month with history of jaundice or pedal edema. He also gave history of multiple episodes of ascitic fluid tapping done for abdominal distension with respiratory distress. Abdominal pain radiates to backsevere abdominal pain-insidious in onset- increasing severity on food intake. He had loss of weight and loss of appetite. He was evaluated for headache 2 week back and diagnosed as a case of cortical vein thrombosis and was put on antiplatelets (Figure 2). He was diagnosed as a case of pancreatic ascites, with DT amylase of 8400u/dl and protein content of 3.1/dl. Computed tomography (CT) scan revealed pancreatic duct disruption noted in the distal body. He was initially put on conservative management in view of poor general condition and performance status. He was started on iv heparin. Anaemia was corrected with multiple blood transfusions. Ascitic fluid tapping done whenever it was tense, and patient was symptomatic. He underwent CT angiogram to evaluate for haemorrhagic ascites which revealed right pulmonary artery thrombosis (Figure 3). Ultrasonography (USG) Doppler of leg veins was negative for DVT. After thorough optimization patient was taken for MPD stenting. MPD stenting done on 22 November 2021 post procedure patient improved over course of time (Figure 4). On subsequent imaging there was a resolution of pulmonary thrombosis. After 6 months, MPD stent was removed and is currently on follow up.

Case 3

A 33-year-old gentleman admitted with c/o abdominal pain and distension and anorexia of 20-days duration-treated elsewhere. He was a known case of ethanol related

chronic pancreatitis. On evaluation he was diagnosed as a case of pancreatic ascites with ascitic fluid amylase level of 9600 u/ml and protein level of 2.8 gm/dl. He developed hemiplegia secondary to massive CVA in ward. Subsequent evaluation with CT scan brain and angiogram revealed, left MCA territory massive infarct and had right segmental pulmonary thrombosis (Figure 5).

Case 4

A 19-year-old teenager a k/c/o CCP from the age of 7 years, presented with pain abdomen and distension for one month, diagnosed to have acute exacerbation of CCP and was treated conservatively. He also had breathing difficulty. He was diagnosed to have chronic pancreatitis with pancreatic ascites with pancreato-pleural fistula after ascitic and pleural fluid analysis (Figure 6). On subsequent evaluation he was found to have extensive thrombosis involving neck and upper limb veins and pulmonary vasculature (Figure 7). Central venous Doppler (on admission) showed acute thrombosis involving Right internal jugular, subclavian, brachiocephalic veins, extending to axillary and brachial veins (Figure 8). He was initially managed conservatively with Right chest drain tube, nutritional measures- later taken up for definitive surgery. In view of chronic pancreatitis features, Frey's procedure was done. Post-surgery, recovery was uneventful. Repeat scan after 1 month of surgery showed partial recanalization of the thrombosed veins with good flow.

Case 5

A 50-year-old male was referred with a diagnosis of pancreatic ascites in sepsis. He had history of abdominal pain and distension for 10 days and diagnosed as pancreatic ascites with an ascitic fluid amylase of 24000 U/ml. On examination he was in shock and his general condition was poor. Evaluation revealed he was having right pulmonary artery thrombosis along with features of pancreatic ascites and duct disruption in the body of pancreas. He was started on heparin but sustained cardiac arrest on 2nd day of his admission and expired.

Table 1: Case list.

S. no.	Age/sex	Diagnosis	Vascular complication	Clinical course
1	32/male	Acute pancreatitis with pancreatic ascites	Right atrial clot	Expired due to pulmonary embolism
2	33/male	Acute pancreatitis with pancreatic ascitis and pancreato-pleural fistula	Cortical vein thrombosis and right pulmonary artery thrombosis	Recovered well with MPD stenting
3	49/male	Acute pancreatitis with pancreatic ascites	Left MCA territory massive CVA	Expired
4	19/male	Chronic calcific pancreatitis with pancreatic ascites	Right segmental pulmonary artery thrombosis, B/L IJV, SCV thrombosis	Improved with Frey's procedure
5	50/male	Acute pancreatitis with ascites	Pulmonary artery thrombosis	Patient expired due to sepsis



Figure 1: 2-D echo showing right atrial clot.

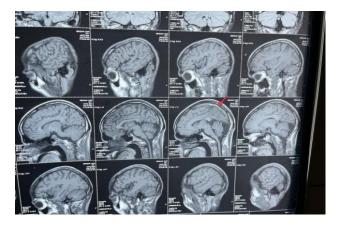


Figure 2: Cortical vein thrombosis.

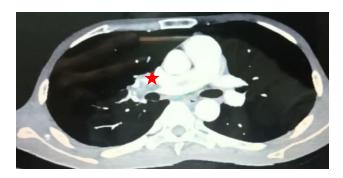


Figure 3: Right pulmonary artery thrombosis. Note: Thrombus is denoted by symbol.



Figure 4: MPD stent in situ.



Figure 5: Left MCA territory massive infarct.

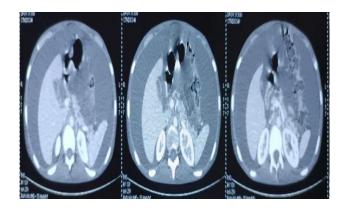


Figure 6: CT scan showing duct disruption and gross ascites.

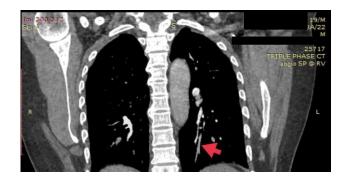


Figure 7: Segmental pulmonary artery thrombosis.



Figure 8: Ultrasound showing non-compressible vein due to thrombosis.

DISCUSSION

Pancreatic ascites is a condition in which pancreatic secretions leak into the abdominal cavity. It causes severe inflammatory reaction in the peritoneal cavity and massive fluid shifts. It is a protein rich exudative ascites. The severity of pancreatic ascites depends on the level of duct disruption and background parenchymal and ductal pathology, associated sepsis, and malnutrition.^{1,2} It is a condition which is difficult to manage as it causes severe abdominal pain, distension, and anorexia.

Diagnosis is by demonstrating high protein content and amylase or lipase level in ascitic fluid.³ Further work up is done by CT scan to show the ductal and parenchymal changes of acute or chronic pancreatitis. MRCP evaluation will show pancreatic ductal anatomy and the level of disruption. The management depends up on the general condition and nutritional status of the patient.⁴

Initial management is conservative in the form of medical management with anti-secretary agents and nutritional support. Spontaneous resolution is not unknown, but extremely rare.⁵ Underlying ductal and parenchymal pathology plays a significant role in the success of medical management and resolution of disease.⁵ Larsen et al has shown that patients with proximal MPD stricture or blockage due to a stone may fail to improve spontaneously, and MPD stenting is an attractive option to manage cases of pancreatic ascites especially if the ductal disruption is in the proximal part of the pancreas and we can bridge across the ductal disruption.6

Surgical options include doing distal pancreatectomy, thereby removing the pathological area if the ductal disruption is in the left half of pancreas.⁷ One can also perform Frey's procedure or lateral pancreatico-jejunostomy including the disrupted duct to the anastomosis. On the other hand, if it is a leaking pseudocyst, we can do cysto-gastrostomy or cystojejunostomy. Most performed surgery is either Frey's procedure or distal pancreatectomy, with high success rate.8 In literature search we could find studies showing the importance of doing early surgery in pancreatic ascites and the benefits of the same approach.^{7,8}

In our series, 2 patients underwent MPD stenting and the other one underwent surgical management. Among the patients who underwent MPD stenting, one recovered well and the other had a cardiac event at the time of the procedure and succumbed. In the 3rd case, patient sustained massive CVA while admitted in ward. Fourth case was a case of tropical pancreatitis with ductal calcifications and dilatation. In view of CCP changes he was taken up for Frey's procedure, recovered well with surgery. One patient died of aspiration pneumonia and sepsis and other one died of massive pulmonary thromboembolism. Two patients who recovered, showed halt of prothrombotic cascade and resolution of thrombus. Both patients recuperated well and currently under follow up.

This series shows a high incidence of extra-splanchnic vascular thrombosis but cause of the thrombosis is not clearly understood. This case series shows the importance of pulmonary evaluation as pancreatic ascites patients have a significant risk of extra splanchnic thrombosis especially in the pulmonary and cardiac. Literature search revealed it is a rare association that is only reported in a limited number of case reports, with reported series showing high risk of thrombosis predominantly in the pulmonary circulatory system. 9 However, there are reports on acute pancreatitis complicated with deep vein thrombosis and pulmonary embolism.¹⁰

In our own institute, out of the 21 pancreatic ascites patients treated during the last 2 years, we identified 5 patients with extra splanchnic vascular complications, and this accounts for approximately 20%. Among these patients, all were males and were in the young or middle age. Four were alcohol related acute pancreatitis and one was a young boy with tropical pancreatitis. All these patients suffered from either arterial or venous thrombosis or both. As evident in this series, early treatment of pancreatic ascites leads to resolution of this thrombotic event. We assume pancreatic ascites as a prothrombotic state with the pancreatic enzymes getting absorbed from the peritoneal cavity, causing systemic inflammation and endothelial injury and a procoagulant state. State or fluid shift causing hemoconcentration and thrombosis. This association makes treatment also challenging by increasing the risk for surgical procedure and anaesthesia. This assumption needs further workup and a large-scale study to prove the association.

CONCLUSION

This rare association of pulmonary thrombosis and pancreatic ascites needs targeted evaluation and urgent treatment to halt the ongoing pro coagulation cascade. However, we need more detailed studies to answer the questions like, is routine pulmonary angiogram necessary for all cases, or routine anticoagulation needed for all cases. Early successful treatment shows resolution of the thrombotic event and full recovery. This ascertains the need of doing early surgery in pancreatic ascites.

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