CASE REPORT

Therapeutic challenges of pancreatic ascites and the role of endoscopic pancreatic stenting

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SUMMARY

Management of pancreatic ascites poses significant therapeutic challenges. Treatment usually consists of either conservative management or interventional therapy with little consensus between the two options. Conservative therapy is the most common initial treatment option but has high failure rates hence arguing for interventional therapy as a preferred primary treatment option. Endoscopic treatment is particularly appealing due to lower failure rates and mortality than conservative therapy or surgery. We describe a patient with recurrent pancreatic ascites who was successfully managed with endoscopic transpapillary stenting. This report contributes to the limited but growing literature on the management of pancreatic ascites.

BACKGROUND

Pancreatic ascites (PA) is a well recognised but rare complication of pancreatitis defined as the accumulation of pancreatic fluid in the peritoneal cavity due to non-malignant pancreatic disease.¹ Its incidence is not known but thought to be infrequent, occurring more in men and in persons between the ages of 20 and 50 years.¹ Disruption of pancreatic ductal anatomy resulting in direct communication of pancreatic secretions to the peritoneal cavity or indirect communication via a leaking pseudocyst is thought to result in PA. Ductal disruption is most frequently due to chronic pancreatitis (77.8%) or pancreatic trauma (8.6%).¹ Clinical manifestations include abdominal distension, early satiety, weight loss and, less commonly, massive pleural effusions.² Abdominal pain or peritoneal symptoms are typically absent, particularly in alcoholics.^{1,2}

Diagnosis requires demonstration of pancreatic fluid in the peritoneal cavity. PA is typically characterised by elevation in amylase (typically greater than 1000 IU/L) and protein levels (greater than 3 g/dL).^{1,3} Endoscopic retrograde cholangiopancreatography (ERCP) provides definitive diagnosis by visual demonstration of a pancreatic ductal disruption and has the additional therapeutic benefit of reducing intraductal pressure with sphincterotomy or insertion of a transpapillary stent.¹

We describe a case of refractory and recurrent ascites illustrating the therapeutic challenges in a patient with PA. We describe our experience in the successful management of PA with combined medical and interventional therapy.

CASE PRESENTATION

A 39-year-old man with a history of alcohol dependence was referred to our institution for

evaluation of progressive abdominal distension. Two months prior to initial presentation, he developed abdominal pain and was diagnosed with mild acute alcoholic pancreatitis that resolved with supportive care. A month later, progressive abdominal distension and discomfort began and abdominal ultrasonographic evaluation revealed ascites and sequelae of pancreatitis. An abdominal CT scan showed significant ascites and multiple peripancreatic fluid collections consistent with pancreatic pseudocysts (figure 1). A large volume paracentesis (LVP) was performed with ascitic fluid analysis notable for amylase 10 018 U/L, albumin 8700 mg/L, and 1039 white cell count/ μ L with 62% segmented neutrophils. Ascitic fluid cultures were negative. The diagnosis of PA was performed based on the elevated amylase level and imaging evidence of pancreatic pseudocysts. Over the next few days the patient developed rapidly progressive abdominal distension and was transferred to this institution for management of recurrent PA.

At admission to our institution the patient endorsed discomfort due to abdominal distension. He did not describe abdominal pain or tenderness and denied recent fevers, chills or rigours. His medical comorbidities included pancreatitis, gout and alcohol dependence. He had not had any surgeries. He was on an oral prednisone taper for a recent acute gout flare. He reported several years of heavy alcohol consumption, but had been abstinent of alcohol since onset of current symptoms. There was no family history of gastrointestinal disease or malignancies.

At initial evaluation, the patient's temperature was 36.3°C, blood pressure 107/71 mm Hg, pulse 101 bpm, and respiratory rate 16 breaths/minute.

Figure 1 CT of the abdomen with ascites (black arrow) and pancreatic pseudocysts (white arrows) with adjoining pancreatic tissue.

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Figure 2 Endoscopic retrograde cholangiopancreatogram shows accumulation of contrast in cystic structures adjacent to the pancreas (white arrow) and free extravasation from the body of the pancreas (black arrow).

His abdomen was markedly distended, hyperresonant to percussion and mildly tender but without rebound tenderness or guarding. Shifting dullness and positive fluid wave were present. No hepatosplenomegaly was appreciated. There were no stigmas of chronic liver disease. Admission studies revealed leucocytosis (22 000 white cells/mL), anaemia (haematocrit 35.1%), hyponatraemia (sodium 128 mmol/L) and hypoalbuminaemia (serum albumin 2.2 g/dL). Elevated plasma amylase of 869 U/L and plasma lipase of 624 U/L was noted. International normalised ratio was 1.18. A LVP was performed and ascitic fluid analysis surprisingly was notable for normal amylase of <5 IU/L, albumin 1.1 g/dL and total protein 2.1 g/dL. The serum-ascites albumin gradient was calculated at 1.1 g/dL. To consider secondary portal vein thrombosis and resultant portal hypertension as a cause for low amylase ascites a liver ultrasound with Doppler flows was performed that demonstrated normal venous waveforms and appropriate flow directions. The lack of ascitic amylase was perplexing given the recent significant elevation in amylase and in consultation with gastroenterology was interpreted to possibly signify spontaneous resolution of PA.

TREATMENT

Octreotide and bowel rest were initiated. After several days of clinical stability a trial of pancreatic stimulation by oral feeding was attempted. Within 3 days recurrence of tense ascites developed. A repeat LVP was performed and ascitic fluid analysis was now significant for elevated amylase of 474 IU/L, albumin less than 1.0 g/dL and protein 1.7 g/dL, suspicious for recurrence of PA. An ERCP was performed and showed an internal pancreatic fistula in the proximal body that communicated with one of several pancreatic pseudocysts as well as a completely transected pancreatic duct at the distal body that freely emptied into the peritoneal cavity (figure 2). Sphincterotomy and placement of a transpapillary 12 cm 5 F pancreatic stent extending to the mid-body was performed. Given potential for secondary infection of ascitic fluid, prophylactic antibiotics were initiated.⁴ In an attempt to decrease need for paracentesis and to increase patient comfort a trial of oral diuretics was initiated.

Three days later the patient developed tachycardia and spiking fevers. Suspicion for postprocedural infection was high despite extensive negative infectious work-up. Antibiotics treatment was escalated to include empiric meropenam. Forty eight hours later the patient improved with resolution of the fever. Meropenam was empirically continued for 2 weeks. While awaiting improvement after ERCP, total parenteral nutrition (TPN) was continued due to severe malnutrition and the patient's inability to tolerate enteral nutrition. Octreotide was also continued after careful risk benefit discussions with local gastroenterology experts. Over the next 3 weeks the PA gradually improved but ability to tolerate oral intake was still poor. Subsequently, oesophagoduodenoscopy was performed to remove the pancreatic stent, and the patient was discharged home on diuretics, TPN and, as tolerated, oral intake with close outpatient follow-up.

OUTCOME AND FOLLOW-UP

At the patient's 6-week follow-up appointment, his abdominal distension and ascites had resolved. All medications and TPN were discontinued. He has since been sober and has not had any additional complications at 14 months after treatment. He has gained 11 kg in weight and his quality of life continues to improve.

DISCUSSION

No randomised clinical trials exist to guide treatment for PA.⁵ Treatment is instead guided by case reports, case series and meta-analyses with no established treatment algorithm. Our experience demonstrates the therapeutic challenges of PA and contributes to the limited literature of successful management with endoscopic transpapillary stenting.

Therapeutic options for PA include conservative and interventional options. Conservative therapy includes serial LVP, elemental diet, parenteral nutrition, diuretics and somatostatin analogues.^{1 2 4-7} These interventions are thought to decrease pancreatic secretions facilitating fistula closure and promote serosal apposition and healing.⁸ This approach has high failure rates of 40–60%,^{9 10} mortality rates of 17%^{1 2} and may be costly.¹¹ Risk factors for failure of conservative therapy include severity of pancreatic duct disease seen on ERCP including the presence of more than one site of contrast extravasation and degree of biochemical abnormalities on admission labs including low serum sodium and serum albumin levels and a higher ratio of ascitic fluid protein to total serum protein levels.¹⁰ Interventional therapy includes either surgical or endoscopic options encompassing a wide variety of procedures including sphincterotomy, transpapillary stent placement, distal pancreatectomy, cystogastrostomy, cystojejunostomy and cystoduodenostomy.² These procedures facilitate fistula closure by promoting flow of pancreatic fluids into the digestive tract.²

The usual therapeutic strategy involves an initial trial of conservative therapy followed by invasive options in refractory cases.^{1 2 8} Two to 3 weeks of initial conservative therapy has been suggested as a cut-off point to decide on interventional therapy based on the increased observed mortality after this point.¹¹⁻¹³ However, it is debatable whether management should begin with interventional therapy. A meta-analysis to evaluate the utility of different treatment modalities reported that only surgery and transpapillary stents were related to successful treatment where failure of treatment was defined as failure to achieve resolution of ascites, relapse after initial improvement or death.¹ The study concludes that treatment should begin with interventional therapy and conservative

therapy should only be used as an adjunct.¹ Endoscopic pancreatic duct stenting is an appealing initial choice due to a lower failure rate (14.3%) and mortality rate (0%)⁶ compared with conservative therapy (40–60% failure rate, 12% mortality rate)^{1 2 9 10} and surgery (12.8% failure rate, 7.7% mortality rate).² Additionally, ERCP is diagnostic and therapeutic and mandatory prior to surgery to identify the site of leakage. Careful consideration to the size of the pancreatic duct and distance to the site of leak should be made while selecting the appropriately transpapillary stents. These stents facilitate healing of ductal disruptions either by partial obstruction of the main pancreatic duct or bypassing the pancreatic sphincter to reduce intraductal pressure and create preferential flow through the stent.^{1 6} ERCP has the potential complication of secondary infection of the ascites or pseudocysts and adequate precautions to minimise risk should be taken including limiting volume of contrast used and postprocedure antibiotic prophylaxis.¹¹ A study by Bracher *et al*⁶ describes seven of eight cases that underwent endoscopic placement of a transpapillary pancreatic duct stent with complete resolution of PA within 6 weeks of initial stent placement and no recurrence during a 14-month follow-up period. The failure rate was 14.3% and mortality rate was 0%. Although no complications related to stent placement including infection or alterations in ductal morphology were described in these patients, ERCP itself carries an estimated 6.85% incidence rate of post-ERCP complications including duodenal perforation, postprocedural pancreatitis and infections such as cholangitis.¹⁴ A recent case series had similar success with first line endoscopic therapy. The authors describe their experience with endoscopic sphincterotomy and stent placement as first line treatment of 28 patients who had PA or pleural effusions for at least 3 weeks prior to treatment decision. Their cohort of patients achieved a success rate of 92.9% for complete resolution of fluid collections by 3–8-week follow-up. There were complications in 25% of the patients including self-limited abdominal pain (7.1%), fever (17.9%) and sepsis (10.7%). There were no deaths.¹² These studies are limited by small size, but given the high success rate and lower risk with endoscopic treatment as well as the overall limited success of alternative therapies, endoscopic treatment should be considered as the initial therapy.

This case adds to the limited but promising literature reporting successful management of PA with endoscopic transpapillary stenting. In this case, complete resolution of PA was seen within 9 weeks of initial stent placement with no recurrence over a 14-month period. This case was possibly complicated by a post-procedural infection that resolved with antibiotics. In summary,

Learning points

- ▶ Pancreatic ascites is a rare cause of new onset ascites and should be considered if there is a history of alcoholism, pancreatitis or abdominal trauma.
- ▶ Interventional therapy should be strongly considered as the initial treatment option.
- ▶ Endoscopic transpapillary stenting is an increasingly well-described treatment approach due to lower failure rates and mortality than surgery or conservative therapy.^{2 5}

endoscopic intervention should strongly be considered prior to surgery or conservative therapy in the management of PA.

Competing interests None.

Patient consent Obtained.

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