CASE REPORT

Open Transduodenal Stenting as an Option to Failed Endotherapy in Pancreatic Ascites

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ABSTRACT

Context Pancreatic ascites is rare and thought to be due to rupture of a pancreatic pseudocyst, main pancreatic duct disruption or leakage from an unknown site.

Case report We report the case of a 33-year-old gentleman with a history of chronic alcoholic pancreatitis who presented with pancreatic ascites and was successfully treated with open transduodenal stenting and pancreatic and biliary sphincteroplasty.

Conclusion Open transduodenal stenting is an option to failed endotherapy in pancreatic ascites.

INTRODUCTION

Pancreatic ascites is rare and its causes are not fully understood. It is thought to result from the rupture of a pancreatic pseudocyst, disruption of the main pancreatic duct (MPD), or possibly leakage from an unknown site. The case of a young adult presenting with pancreatic ascites with a history of chronic alcoholic pancreatitis is presented here. ERCP confirmed the presence of MPD disruption secondary to a calculus in the region of the head/neck of the pancreas. Endoscopic stent insertion was unsuccessful and the patient underwent a laparotomy and transduodenal exploration with stenting of the pancreatic duct and pancreatic and biliary sphincteroplasty. Whilst cases of pancreatic ascites treated by endoscopic stenting have been reported, this type of clinical presentation is rare.

CASE REPORT

A 33-year-old gentleman presented with abdominal pain and distension. He also reported significant weight loss. He had a past history of chronic alcoholic pancreatitis. He was a smoker and had consumed 180-225 g of ethanol per day for the past ten years but denied any consumption in the last two months.

On examination he was cachectic and his abdomen was tense with ascites and generally tender. On admission his serum amylase was normal (87 IU/L; reference range: 20-110 IU/L). It was unclear whether the ascites was related to his alcoholic liver disease, but on tapping the amylase levels (greater than 10,000 IU/L) were highly suggestive of pancreatic ascites.

Initially, management was conservative with nutritional support using naso-jejunal feeding in combination with a somatostatin analogue (octreotide, 200 micrograms tid). A MR with MRCP was performed but in view of the ascites and the patient’s inability to cooperate in the scanner the films were of poor quality and could not be interpreted. ERCP
showed that the MPD diameter was 5 mm and the distance to the site of the fistula from the papilla was approximately 2.5 cm (Figure 1). The MPD that was visualised did not show any evidence of stricturing. The calculus was seen to be possibly outside the MPD raising the possibility of a parenchymal calcification causing duct rupture or a MPD calculus eroding outside into the parenchyma with the resultant fistula. Although the calculus did not appear to completely obstruct the MPD, as a result of the duct disruption a distal pancreatogram could not be obtained. Despite a biliary and pancreatic sphincterotomy, pancreatic stent insertion was unsuccessful because access was difficult and the distal MPD could not be visualised. Hence it was decided to proceed to open surgical intervention. The laparotomy was technically extremely difficult as the patient’s entire abdominal contents were encased in a cocoon and he had significant portal hypertension with features of chronic liver disease. Access to the MPD via the lesser sac was very difficult despite extensive mobilisation. As a result, a transduodenal approach was used to gain access to the MPD and bile duct. The stone was in fact outside of the MPD within the parenchyma of the pancreas with MPD disruption and fistula formation. A pancreatogram with a cholangiogram were obtained. The cholangiogram was normal. The pancreatogram again did not visualise the distal MPD satisfactorily. Hence a pancreatic and biliary sphincteroplasty was undertaken. Stone extraction was unsuccessful. A guide wire was passed into the MPD past the fistula and a pancreatic stent was inserted over the wire. A plastic infant feeding tube of 8 Fr and 5 cm long was used and anchored to the sphincteroplasty. A completion pancreatogram was obtained which confirmed placement of the stent beyond the fistula. The patient made a good post operative recovery and was well enough to be discharged 10 days later with complete resolution of the ascites. A liver biopsy was also performed at the time of the operation and this confirmed chronic liver disease secondary to alcohol. The patient has been well at one year follow-up with weight gain and no recurrence of pain or ascites. The stent was not removed and was spontaneously discharged. An ultrasound scan in the post-operative period showed no recurrence of the ascites.

**DISCUSSION**

Chronic pancreatitis is a progressive, irreversible inflammatory disease and can be at times associated with pain and ascites. Pancreatic ascites is rare and was first reported in the literature in 1953 when Smith described two cases of ascites associated with chronic pancreatitis [1]. In 1982, Bore and Cameron reviewed 182 cases of pancreatic ascites, 130 of which were treated surgically [2]. The exact incidence of pancreatic ascites remains unknown and the causes are not well understood. It has been reported that ruptured pseudocyst exists in approximately 80% of cases, a disruption of the pancreatic duct exists in 10%, and an obscure leakage site exists in the remaining 10% [3]. Pancreatic ascites primarily develops in patients with chronic alcoholic pancreatitis who have pancreatic duct stenosis or stones. It occurs during an attack and an acute inflammatory reaction is usually observed around the leakage. These clinical
characteristics suggest that the rupture or disruption is caused by increased pressure within the pseudocyst or pancreatic duct [4]. Initially, patients with pancreatic ascites should be treated conservatively. Conservative therapy is described as the use of one or more of the following treatments: elemental diet, parenteral nutrition, paracentesis, continuous percutaneous drainage, or the more recently used somatostatin analogues. The aim of these treatments is to reduce pancreatic exocrine secretion, or evacuate the ascites and thus facilitate the closure of the fistula by approximating the peritoneal surface to the point of leakage [5]. Although many patients with pancreatic ascites have been successfully treated with careful management and TPN therapy, there have been several reports of sudden “unexpected” death among these patients during conservative management [6, 7]. We used nasojejunal feeding as this reduces the incidence of sepsis and is well tolerated.

Transpapillary pancreatic duct stents are an alternative option when a ductal disruption is demonstrated by ERCP [8]. Intraductal stents facilitate the healing of ductal disruptions because of partial obstructions of the MPD. This effect may not only be achieved by bypassing the point of leakage, but also by bypassing the pancreatic sphincter without reaching the point of leakage. The latter would reduce intraductal high pressure, and thus lowering pressure by pancreatic duct drainage might be enough to spontaneously heal the leakage point. Bracher et al. obtained complete resolution of the ascites six weeks after placing a polyethylene stent in seven out of eight patients [8]. Adverse effects are uncommon if the stent is removed in three to six weeks; neither infections, acute pancreatitis, occlusion of the stent, nor alterations of pancreatic duct morphology were described after a 14-month follow-up period. The long-term complications of expandable metallic coil stents for the treatment of pancreatic ascites have not been sufficiently studied, and therefore they should not be considered unless other therapies are contraindicated or polyethylene stents have been unsuccessful. Therefore, metallic stents should only be used as a palliative option in patients in whom the main objective is short-term maintenance of quality of life [8]. In this case, as endotherapy was unsuccessful we resorted to transduodenal insertion of a stent. We believe that as a complete pancreatogram was not obtained at ERCP the endotherapy failed. Access was also difficult and this could have been as a result of duodenal distortion as was evident at open surgery with all intra-abdominal contents encased in a cocoon. Nasopancreatic drainage in combination with ESWL was not thought to be an option, as with the likelihood of the calculus being outside the MPD the fistula would not heal.

CONCLUSION

In the face of failed endotherapy, pancreatic and biliary sphincteroplasty along with open transduodenal stenting of the MPD is an option to allow for healing of pancreatic fistulae with a successful outcome.

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Keywords Pancreatic Diseases; Pancreatic Ducts; Pancreatic Pseudocyst

Abbreviations MPD: main pancreatic duct

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