Portal hypertension and chylous ascites complicating acute pancreatitis: the therapeutic value of portal vein stenting

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ABSTRACT

Chylous ascites as a consequence of acute pancreatitis is very rare. We present an unusual case of a 73-year-old man who developed refractory chylous ascites one month after an acute severe episode of gallstone pancreatitis, associated with portal hypertension. He was successfully treated with portal vein stenting, which has remained patent to date.

KEYWORDS

Pancreatitis – Chylous ascites – Portal vein stent

Accepted 3 April 2017

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Introduction

Chylous ascites is a rare clinical entity, associated with poor outcomes attributable to malignancy.¹ Pancreatitis is an uncommon cause of atraumatic chylous ascites (4%), while superior mesenteric–portal vein (SMV/PV) thrombosis complicates 7% of acute pancreatitis.^{1,2} We discuss a rare case of a patient who developed refractory chylous ascites secondary to acute pancreatitis and SMV/PV thrombosis, which was treated with portal venous stenting. We postulate that the inflammatory changes associated with pancreatitis and thrombosis of the spleno-mesenterico-portal axis led to non-cirrhotic portal hypertension and refractory chylous ascites in our patient.

Case history

A 73-year-old male was referred to our hepatobiliary and pancreatic surgery centre with a 14-month history of refractory ascites requiring multiple hospital admissions for percutaneous drainage with little or no effect. This was on a background of acute severe gallstone pancreatitis, which necessitated admission to the intensive treatment unit five weeks prior to the first presentation with ascites. His past medical history was significant for essential hypertension, type 2 diabetes mellitus, gastro-oesophageal reflux disease and cholelithiasis. There was no significant history of excess alcohol intake.

On examination, the patient had significant abdominal distension and poor nutritional status, which required nasojejunal and parenteral replacement. He weighed 73 kg on admission (pre-ascites weight was 57 kg).

Investigations and treatment

Laboratory results on admission were as follows: white blood count (wbc) $6.7 \times 10^9/l$, C-reactive protein 36.7 mg/l, sodium 133 mmol/l, total protein 61 g/l, albumin 30 g/l, alanine aminotransferase 17 iu/l and bilirubin 9 $\mu\text{mol/l}$. His serum lipid profile had previously been shown to be normal.

An ultrasound-guided 10-French all-purpose drain was inserted and he was treated with 100 ml 20% human albumin solution for every 2 litres of ascites drained. Approximately 16 litres of milky yellow fluid were drained, which had a similar biochemical profile as the previous samples (wbc 99 cells/mm³, lactate dehydrogenase 48 iu/l, amylase 11 iu/l, albumin 14 g/l, triglycerides 7.1 mmol/l and cholesterol 1.7 mmol/l). Tuberculosis culture was negative. The fluid was transudative (serum ascitic albumin gradient 16); overall, the findings were consistent with a leak of chyle.

The aetiology of the ascites was investigated. Serum tumour marker profile (alphafetoprotein; cancer antigen, CA, 153; carcinoembryonic antigen; CA199); viral and autoimmune hepatitis screens (hepatitis B, hepatitis C, anti-mitochondrial, anti-Sm, anti-LKM, anti-GPC, antinuclear antibodies) were negative. Immunoglobulin levels were within normal limits and protein electrophoresis produced

an inflammatory sample. A liver biopsy showing focal bridging fibrosis and mean portal pressure of 35 mmHg (normal range: 5–10 mmHg) led to a working diagnosis of chronic liver disease with ascites and mild encephalopathy. Serum ammonia level was 33 $\mu\text{mol/l}$ (normal range: 12–41 $\mu\text{mol/l}$). Triple-phase computed tomography (CT) of the patient's liver showed marked attenuation of the portal vein at the portal confluence with dilatation of the SMV and relatively poorly formed collateral vessels. Endoscopic retrograde cholangiopancreatography (ERCP) showed extrahepatic biliary dilatation with diffuse filling defects throughout the biliary tree consistent with common bile duct (CBD) stones.

A lipiodol lymphangiogram was considered, to identify and embolise the source of the chyle leak. However, further progression of his portal venous narrowing, with multiple venous collaterals and segmental portal venous hypertension, was identified on CT. The decision was thus made to attempt portal vein stenting to evaluate the effect of restoring near normal portal flow on the ascitic volume.

A segment VI portal venous puncture was performed by JJ with a 22-gauge Chiba needle under ultrasound guidance and a tight focal thrombotic narrowing involving the SMV and proximal main portal vein (MPV) was bypassed. Venography within the SMV demonstrated compromised flow with retrograde filling of an attenuated splenic vein and contrast stasis within peripheral SMV branches. Pressure measurements across SMV/MPV thrombosis demonstrated a 15 mmHg gradient. Balloon angioplasty was performed and

a 16 mm diameter, 9 cm long Wallstent (Boston Scientific, Marlborough, MA) was inserted.

Outcome and follow-up

Post-stent venography demonstrated excellent flow into the MPV (Fig 1). The MPV pressure had increased to 5 mmHg (from 1 mmHg), consistent with a reduction in pressure gradient. This resulted in a marked improvement of ascites with an ascitic drain output of 0 ml within 24 hours post-PV stenting. He was then commenced on long-term 75 mg aspirin daily in an attempt to maintain stent patency. The ascites significantly reduced within 2 weeks and his physical and biochemical profile had returned to normal at clinical follow-up 1 year later. He was therefore able to undergo elective cholecystectomy, which on the advice of the anaesthetic team was delayed to allow such recovery prior to surgery. This also allowed for duct clearance of the CBD stones, with serial ERCPs.

He was also followed up with serial CT scans over 1 year, which have demonstrated good opacification of the stent with flow into the right and left portal venous branches and resolution of chylous ascites (Fig 1).

Discussion

Chylous ascites is characterised by the presence of milky triglyceride-rich fluid in the peritoneal cavity; commonly caused by abdominal surgery, trauma, malignancies,

Figure 1 A) Computed tomography (CT) showing focal compression of the portal vein (arrow) and extensive ascites with scalloping of the liver edge. B) Venography with catheter tip showing tight focal thrombotic narrowing of the proximal main portal vein (arrow). C) Post-venoplasty venography demonstrating good flow into the main portal vein. D and E) CT showing patency of SMV/PV stent at the proximal (D) and distal (E) ends, with reduction in amount of ascites at 1-year follow-up.

cirrhosis and mycobacterial infections.¹ Chylous ascites secondary to acute pancreatitis with SMV/PV occlusion is extremely rare, with only seven reported cases of chylous ascites in association with acute pancreatitis.³

Vascular thrombosis and non-cirrhotic portal hypertension are associated with pancreatitis. Obstruction of the spleno-mesenterico-portal axis secondary to thrombosis most commonly involves the splenic vein (90%), followed by the mesenteric vein (72%) and the MPV (24%), resulting in non-hepatic portal hypertension.⁴ The pathogenesis is not fully understood, although several hypotheses exist:

- > pancreatic enzyme erosion and/or portal hypertensive rupture of lymphatics causing a chyle leak;
- > exudation of chyle secondary to lymphatic obstruction and perivascular inflammation; and/or
- > occlusion of veins due to compression by a pancreatic pseudocyst.^{3,4}

Treatment is aimed at reducing the amount of chyle produced and the pressure that augments the chyle leak. Treatment options therefore include diets incorporating high protein and medium chain triglycerides, total parenteral nutrition, somatostatin analogues, anticoagulation and surgical interventions.³ Owing to high operative morbidity and

mortality, endovascular techniques have increasingly been adopted as safe and effective alternatives in the management of SMV/PV thrombosis and stenosis.⁵ Evidence for the use of percutaneous portal venous stenting in benign disease is limited but it has been described in the management of acute and chronic pancreatitis with good outcomes.^{4,5} Our patient demonstrated an immediate and significant response following PV stenting; while dietary measures, parenteral nutrition and diuretics showed little/no improvement.

References

1. Steinemann DC, Dindo D, Clavien PA *et al.* Atraumatic chylous ascites: systematic review on symptoms and causes. *J Am Coll Surg* 2011; **212**(5): 899–905.
2. Ahmed M, Usman Aziz M *et al.* Vascular complications in cases of acute pancreatitis – CT scan based study. *J Pak Med Assoc* 2016; **66**(8): 977–979.
3. Lippey JF, Yong TL. Chyle leak following biliary pancreatitis. *J Surg Case Rep* 2014; **2014**(7): rju052.
4. Banerjee A, Kulkarni AV *et al.* Percutaneous stenting of the portal vein prior to biliary bypass in a patient with chronic pancreatitis and portal biliopathy. *Indian J Gastroenterol* 2015; **34**: 261–263.
5. Maleux G, Vaninbrouckx J, Verslype C *et al.* Pancreatitis-induced extrahepatic portal vein stenosis treated by percutaneous transhepatic stent placement: a case report. *Cardiovasc Intervent Radiol* 2003; **26**(4): 395–397.