Royal College of Surgeons
ADVANCING SURGICAL CARE

ONLINE CASE REPORT

Ann R Coll Surg Engl 2017; **99:** e145–e147
doi 10.1308/rcsann.2017.0058

Spontaneous central venous thrombosis and shunt occlusion following peritoneovenous shunt placement for intractable ascites

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ABSTRACT

A 43-year-old man had a peritoneovenous shunt inserted for the treatment of chylous ascites secondary to myelofibrosis. Despite being on anticoagulation for superior mesenteric vein thrombosis, he developed shunt dysfunction within two weeks of insertion. Superior venacavography showed multiple filling defects in the right axillary vein, no filling of the right brachiocephalic and right subclavian vein, and thrombotic occlusion of the internal jugular veins bilaterally. The shunt was removed 11 days after insertion, and there was extensive thrombosis of the venous end of the shunt and the compressible pump chamber. Shunt thrombosis is known to occur but remains a rare complication, with 87% of such obstructions being due to a thrombus at the tip of the venous end of the shunt. Extensive thrombosis of the shunt (as in the present case) is very rare.

KEYWORDS

Peritoneovenous shunt – Thrombosis – Occlusion – Ascites – Chylous

Accepted 5 February 2017

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The insertion of peritoneovenous shunts[1] for the treatment of intractable ascites results in an increase in cardiac output, plasma volume and an improvement in renal function, with an enhanced response to diuretics. However, shunt occlusion or malfunction, with recurrence of ascites, are well described complications of the procedure. We present the details of a patient who developed the rare complication of extensive thrombosis of the venous end and compressible pump chamber of a peritoneovenous shunt inserted for the treatment of chylous ascites secondary to myelofibrosis.

Case history

A 43-year-old man with refractory chylous ascites requiring weekly paracentesis was admitted with hypovolaemic acute kidney injury. He had presented initially six months earlier with a history of alcoholism (20–50 units/week) and oesophageal varices (grade 2 seen on upper gastrointestinal endoscopy). Computed tomography had revealed hepatosplenomegaly, a mass in the porta hepatis compressing the portal vein and superior mesenteric vein thrombosis. Bone marrow and liver sampling confirmed myelofibrosis with extramedullary haematopoiesis and he had tested positive for the Janus kinase 2 mutation (associated with myeloproliferative neoplasms). He was anticoagulated, and treated with hydroxycarbamide and then ruxolitinib with resultant reduction in splenomegaly. However, accumulation of ascites escalated with subsequent renal impairment.

After optimisation of renal function, an 11.5 Fr Denver® (peritoneovenous) shunt (CareFusion, Waukegan, IL, US) was placed under general anaesthesia. The abdominal portion was inserted surgically intraperitoneally between the liver and costal margin; the venous end was inserted into the right subclavian vein under fluoroscopic guidance. Anticoagulation was continued, and both ascites and electrolyte disturbance resolved. Unfortunately, in the second postoperative week, the ascitic fluid started to reaccumulate.

The patient's nutritional needs mandated placement of a central venous catheter but ultrasonography demonstrated thrombosis of the axillary vein and complete occlusion of the right subclavian vein, raising the suspicion of extensive central venous occlusion. Superior venacavography showed multiple filling defects in the right axillary vein, no filling of the right brachiocephalic and right subclavian vein, and thrombotic occlusion of the internal jugular veins bilaterally (Fig 1). Eleven days after insertion, the Denver® shunt was removed under local anaesthesia, revealing thrombosis extending from the venous end of the shunt to the compression chamber (Fig 2). Parenteral nutrition was abandoned because of venous occlusion while the presence of myelofibrosis precluded multivisceral transplantation. The patient was transferred to the palliative care physicians for end-of-life care and he died three weeks after the removal of the shunt.

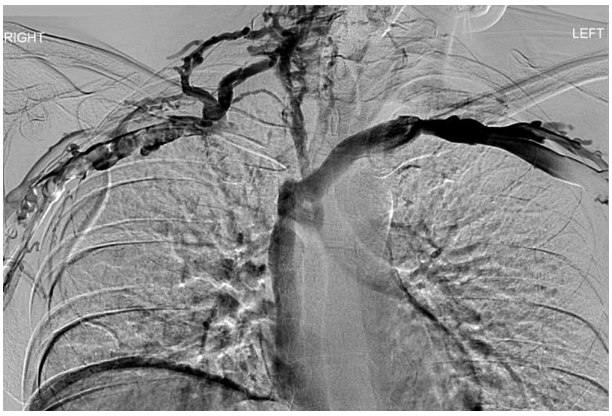

Figure 1 Filling defects are seen in the right brachial and axillary veins with complete occlusion of the right subclavian and brachiocephalic veins. Right upper limb venous drainage is via the thyrocervical trunk and azygos vein. The left axillary, subclavian and brachiocephalic veins as well as the superior vena cava are patent.

Discussion

The use of a pressure activated one-way valve in a device to shunt fluid from the peritoneal cavity to the superior vena cava for the treatment of intractable ascites was described by LeVeen *et al* in 1974.[1] The shunt facilitates drainage of ascitic fluid from the high pressure peritoneal cavity to the low pressure central venous system, and flow in the shunt is maintained if the difference in pressure between the high and low pressure ends of the shunt is 30–50mm of water, as occurs with inspiration. The valve closes when the gradient across the shunt falls below 30mm of water, preventing reflux of blood into the venous end of the tubing.

The Denver® shunt is one such modification of the original design.[2] The shunt is primed with saline and the peritoneal end is placed in the peritoneal cavity, usually between the right lobe of the liver and the ribs, with care being taken to prevent omentum from wrapping around the tip. The shunt is then tunnelled to the neck subcutaneously, with the compressible pump chamber being placed over the lower ribs to aid manual compression of the chamber, and the venous end is then inserted into the superior vena cava via the internal jugular or subclavian veins under fluoroscopic guidance. Shunts may also be placed percutaneously.[3]

Medically intractable ascites secondary to chronic liver disease is the most common indication for insertion of a peritoneovenous shunt[4] while other indications include malignant ascites,[5] chylous ascites[6] and ascites secondary to portal vein thrombosis or renal disease. Primary shunt patency averages three months[5] and a review of a series of studies has shown that the perioperative mortality rate can be as high as 18%, with a 46% survival rate at 21 months and effective palliation of ascites in 59% of survivors at 18 months.[4] When inserted for malignant ascites, mean survival has been shown to be 3.0 ± 1.7 months, with shunts providing effective palliation in up to three-quarters of patients.[5]

Figure 2 Denver® shunt showing extensive thrombosis of the venous end and compressible pump chamber

The best results are seen in patients with milder liver disease and the loss of ascites does not necessarily correlate with a functioning shunt.[4] Abstinence from alcohol is associated with hepatic functional recovery and may decrease renal sodium retention, resulting in shunt occlusion due to low flow.[4] Shunt occlusion is the most common complication, with only 18.6% of shunts remaining functional in survivors at two years.[4] Disseminated intravascular coagulation has also been reported in up to 9% of patients after shunt insertion[5,7,8] and may be caused by activators found in ascitic fluid, such as tissue factor, which is a transmembrane surface protein known to initiate thrombogenesis.[9] Coagulopathy and fluid overload may be preventable by total ascitic fluid drainage at the time of surgery.[4] Perioperative infections with staphylococcal and Gram-negative organisms can occur, and bacterial

peritonitis or septicaemia necessitate shunt removal. Other complications include shunt fracture, displacement and leakage.

Shunt thrombosis is known to occur but remains a rare complication,[10,11] with 87% of such obstructions being due to a thrombus at the tip of the venous end of the shunt.[12] Extensive thrombosis of the shunt (as in the present case) is very rare. Furthermore, in such cases, it has been reported that cavography disclosed a complete obstruction of the superior vena cava or one of its branches in 65% of patients.[12]

Endovascular approaches to treating central venous occlusions have been described[10] while the use of streptokinase to maintain short-term shunt patency has been shown to be successful in cases of shunt thrombosis.[11] In the present case, thrombosis of the right brachiocephalic, subclavian and axillary veins occurred within two weeks of insertion of Denver® shunt, and extended into the venous end of the shunt despite anticoagulation. The cause for extensive venous and shunt thrombosis in this case remains unexplained although a hypercoagulable state is often associated with myelofibrosis. This may have been compounded by the disseminated intravascular coagulopathy associated with shunt insertion.[5,7,8]

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