Case Reports

Endovascular Treatment of Concomitant Obstructions of a Denver Drainage Catheter and Superior Vena Cava in a Patient With Liver Cirrhosis

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Summary

With the increased use of intravascular catheters and devices, they have become the major non-malignant cause of superior vein cava (SVC) syndrome. We report a patient with liver cirrhosis who had received a peritoneovenous drainage catheter for refractory ascites, and then developed SVC syndrome because of concomitant occlusions of both the SVC and the drainage catheter. The patient regained patency of both the occluded vessel and the drainage catheter through percutaneous transluminal venoplasty, and there was dramatic improvement of clinical symptoms and good performance of the drainage catheter. Percutaneous intervention may be a feasible and effective therapy for SVC syndrome and intra-catheter thrombosis-related dysfunction. (Int Heart J 2017; 58: 447-450)

Key words: Superior vein cava syndrome, Percutaneous intervention, Angioplasty, Indwelling catheters

Superior vena cava (SVC) syndrome is characterized by stenosis or occlusion of the SVC leading to venous congestion of the venous drainage territory affecting the head, neck, upper extremities, and upper chest. The most common cause of SVC syndrome is external compression by malignancies. However, with their increased use, intravascular catheters and devices have become the major non-malignant cause of SVC syndrome.

Herein, we report a patient with liver cirrhosis who had received a peritoneovenous drainage catheter for refractory ascites, and developed SVC syndrome because of concomitant occlusions of both the SVC and the drainage catheter. The patient regained patency of both the occluded vessel and the drainage catheter by means of percutaneous transluminal venoplasty, and showed a dramatic improvement in clinical symptoms with good performance of the drainage catheter thereafter.

Case Report

A 50-year-old male, with known alcoholic liver cirrhosis, stage C in the Child-Pugh classification, who had undergone implantation of a peritoneovenous shunt (Denver shunt) for refractory ascites 4 years before this event, was referred to our cardiovascular department due to progressive swelling of the face and bilateral upper arms, dyspnea on exertion, and increasing waist circumference for 4 months. The Child-Pugh stage had deteriorated from his usual stage B to stage C (11 points: albumin: 2.1 g/dL, total bilirubin: 0.5 mg/dL, prothrombin time prolongation: 6.1 seconds, ascites: severe, no hepatic encephalopathy). A high level of D-dimer (1740 μg/L, normal cut-off value: 550 μg/L) was found and contrast-enhanced chest computed tomography revealed that a huge thrombus had caused complete obstruction of the SVC, which also involved the tip of the Denver catheter. The result was malfunction of the peritoneovenous shunt and blood drainage of the upper venous system through the azygous and collateral veins (Figures 1A and 1B). With the diagnosis of SVC syndrome, intravenous heparin and oral Coumadin were initiated. Biochemical analyses of coagulation/fibrinolytic factors (protein C, protein S, antithrombin III, fibrinogen, factor V Leiden, factor VIII, and anti-phospholipid/-cardiolipin antibodies) were unremarkable, as were the autoimmune serological markers (rheumatoid factor, ANA, dsDNA).

Percutaneous recanalization of the SVC was introduced days later because of the limited improvement with anticoagulation. Initially, venography was performed using the right femoral vein approach, and confirmed the total thrombotic occlusion of the SVC that blocked the outflow of the Denver peritoneovenous shunting catheter. Endovascular therapy to the SVC thrombotic lesion was approached with a Judkins Right 4/6 Fr guiding catheter, which was advanced to the proximal site of the lesion. The lesion was successfully crossed with a Miracle 6 guidewire (Asahi Intecc, Aichi, Japan) supported by a 1.25 × 20 mm over-the-wire balloon catheter, and the lesion site was dilated by using a 4.0 × 20 mm balloon catheter.

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Received for publication June 28, 2016. Revised and accepted August 26, 2016. Released in advance online on J-STAGE May 8, 2017.

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dilations (Figure 1C), an infusion catheter (Fountain, Merit Medical System Inc., South Jordan, UT, USA) was implanted within the lesion site with drug delivering pores between the SVC and the proximal innominate vein (Figure 1D). Thrombolytic therapy through the Fountain infusion catheter with urokinase was conducted by a bolus dose of 240,000 IU initially, followed by continuous infusion with a daily dose of 900,000 IU, for a total of 24 hours.

Two days later, the follow-up venography revealed some residual unresolved thrombus and a stenosis near the tip of the catheter (Figure 1E). Furthermore, it was suspected that the thrombus occupied the inner lumen of the Denver catheter. Initially, in order to fix the Denver catheter, a 4.0 × 20 mm balloon was anchored between the narrowing SVC and the Denver catheter, and a Miracle 3 guidewire was advanced to cross the occluded segment of the Denver catheter (Figure 1F). Distal injection through the 1.5 × 20 mm over-the-wire balloon (Sprinter OTW, Medtronic, USA) confirmed the distal occlusion of the Denver catheter (Figure 2A). Then, in addition to sequential intra-catheter balloon dilations (1.5 mm), the inner lumen was evacuated by pulling back an inflated 4.0 × 20 mm balloon (Figure 2B). The contrast injection via the tip of the Denver catheter revealed a fast flow and confirmed the patency of the inner lumen (Figure 2C). After this, the critical stenosis of the SVC and innominate veins were dilated with 12 × 40 mm and 14 × 40 mm balloons sequentially, which finally led to full luminal expansion with less than 20% residual stenosis (Figure 2D). Good final blood flow return to the right atrium was subsequently noted (Figure 2E). During the days following the procedure, the patient experienced dramatic symptomatic relief from the edema of the face and bilateral arms. The patient’s abdominal girth was decreased significantly from 94 cm to 87 cm, body weight reduced from 52.8 kg to 47.9 kg, and the Child-Pugh stage improved gradually from stage C to B (9 points: albumin: 2.3 g/dL, total bilirubin: 0.7 mg/dL, prothrombin time prolongation: 4.2 seconds, ascites: mild, no hepatic encephalopathy). The patient was discharged 3 days after the endovascular therapy and treated with anticoagulation therapy with warfarin to maintain a prothrombin time INR of 2 to 3 times the control. Three months later, the TC-99m MAA scan showed good functioning of the Denver catheter (Figure 2F).

**Discussion**

We describe here a case of both thrombosis of the SVC and distal peritoneovenous shunt in a patient with liver cirrhosis successfully treated with percutaneous intervention.

Over the years since the initial discovery of SVC syndrome, multiple varying etiologies have been identified. Nowadays, more than 80% of cases are caused by the invasion or ex-

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**Figure 1.** Computer tomography (CT) and percutaneous venography showing SVC thrombosis. A: CT showing a large thrombus in the SVC and around the distal site of the Denver shunt (arrow). B: CT revealing many collateral veins being recruited by the SVC thrombosis. C: Distal venography confirmed the SVC occlusion (arrow). D: Fountain infusion catheter was placed to cover the whole thrombotic site (arrow pointing to the markers of the distal and proximal ends of the infusion catheter). E: Two days later, angiography showed residual thrombus (arrow) and a stenosis near the tip of the Denver shunt (arrowhead). F: Using an anchoring balloon (arrow) to fix the Denver shunt, a Miracle wire could be advanced into the Denver shunt (arrowheads).
Intrinsic compression of the SVC by malignancy, while the others are caused by “benign” etiologies. The increasing use of indwelling venous catheters, such as dialysis catheters, and implantable cardiac pacemakers, has been proposed to be the main “benign” cause of central venous thrombosis, stenosis, and total occlusion. The absence of thrombophilic factors in our patient, and the location of the thrombosis (both around the jugular site of the shunt and within the shunt) suggest that the thrombosis was due to the presence of the shunt within the SVC.

In cirrhotic patients with a peritoneovenous shunt, SVC thrombosis represents a possible complication, even many years after the placement of the shunt. It should be borne in mind that cirrhotic patients with a peritoneovenous shunt can present increased abdominal girth (ascitic fluid) and/or edema of the face or upper extremities, in which case an imaging survey and possible pharmacological therapy with/without endovascular intervention should be initiated.

With the advances in the field of intervention devices, endovascular techniques with/without a stent have now become the primary treatment for patients with SVC syndrome due to a benign cause. Surgical intervention is reserved for those patients refractory to repeat angioplasty or recurrent restenosis. Some studies have shown that in patients with SVC syndrome due to a benign cause there is a comparable patency rate between endovascular intervention and surgical intervention.

For patients without a fistula, recurrence free survival with endovascular intervention was 91% at 1 year and 73% at 3 years. Assisted patency of the SVC system was 100% at 1 and 3 years. Furthermore, in our case, the failure of the Denver catheter was caused by both SVC thrombosis and intra-catheter thrombosis. Although in the past, surgical removal and replacement were needed in this situation, the endovascular intervention we performed (intra-catheter balloon dilations and a Fogarty-like procedure) was also effective in eliminating the intra-catheter thrombosis and in completing immediate recanalization with good results. In our opinion, endovascular intervention may be an alternative therapy with less trauma and more immediate symptomatic relief for intra-catheter thrombosis-related catheter dysfunction.

Conclusion: We believe we have provided a valuable experience in dealing with catheter failure due to indwelling catheter-related SVC thrombosis and intra-catheter thrombosis. In addition to anti-coagulant therapy, endovascular intervention was found to be effective for this condition with less trauma and more immediate symptomatic relief.
DISCLOSURES

Conflict of interest or external source of funding: None.

REFERENCES


