

# Superior Vena Cava Thrombosis Treated by Angioplasty and Stenting in a Cirrhotic Patient with Peritoneovenous Shunt

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**We report a case of superior vena cava (SVC) thrombosis in a patient with liver cirrhosis and peritoneovenous surgical Denver shunt, successfully treated by angioplasty. In 2005, a 75-year-old man with a cryptogenetic liver cirrhosis and peritoneovenous surgical Denver shunt was admitted to our hospital for chylous ascites. Venography showed a stenosis near the junction of the SVC with the right atrium. Magnetic resonance confirmed an endoluminal filling defect, suggestive of thrombosis, close to the jugular extremity of the peritoneovenous surgical denver shunt. A percutaneous transluminal angioplasty of the SVC thrombosis was successfully performed. Dicumarolic treatment was started. Two and 8 months after percutaneous transluminal angioplasty, a computed tomography scan showed the patency of the SVC. The patient died in June 2006 due to severe liver function impairment and hepatorenal syndrome. The present case shows that percutaneous transluminal angioplasty represents a good choice for primary intervention. (Ann Thorac Cardiovasc Surg 2008; 14: 60–62)**

**Key words:** superior vena cava thrombosis, liver cirrhosis, peritoneovenous shunt, angioplasty, stenting

## Introduction

Refractory ascites occurs in 5 to 10% of decompensated cirrhotic patients. Peritoneovenous shunt is one of the alternative treatments for refractory ascites, however, both early and late complications can occur, including abdominal infections and/or sepsis, disseminated intravascular coagulopathy, acute pulmonary edema, shunt occlusion, peritoneal fibrosis and venous thrombosis.<sup>1)</sup>

In cirrhotic patients with peritoneovenous shunt, total and partial occlusion of superior vena cava (SVC) were

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seen respectively in 53% and 17%.<sup>2)</sup>

We report a case of SVC thrombosis in a patient with liver cirrhosis and peritoneovenous shunt, successfully treated by angioplasty and stenting of the SVC.

## Case Report

In April 2005, a 75-year-old Caucasian man with an history of cryptogenetic liver cirrhosis was admitted to our hospital because of increasing abdominal volume due to ascitic fluid. Two years previously, the patient had been treated for refractory ascites with the placement of a Denver peritoneovenous shunt. The patient also had a diagnosis of chronic atrial fibrillation, not treated with anti-coagulant therapy because of the cirrhosis-related coagulopathy but monitored periodically by echocardiography.

Chemical examination of the ascitic fluid showed the presence of a chylous ascites. The presence of spontaneous bacterial peritonitis and neoplastic cells was excluded

and portal vein thrombosis was not visible by ultrasound.

During hospitalization, dysfunction and/or occlusion of the peritoneovenous shunt were excluded. Moreover, the presence of jugular turgor was seen by physical examination and echocardiography showed turbulence in the right cardiac atrium. A SVC venography was thus performed, showing a stenosis of the SVC, near the junction of the SVC with the right cardiac atrium; a significant pressure gradient (8 mmHg) was found between the SVC and right atrium (Fig. 1). The venography in antero-posterior view showed a stenosis at the junction of the SVC with the right atrium (Fig. 1).

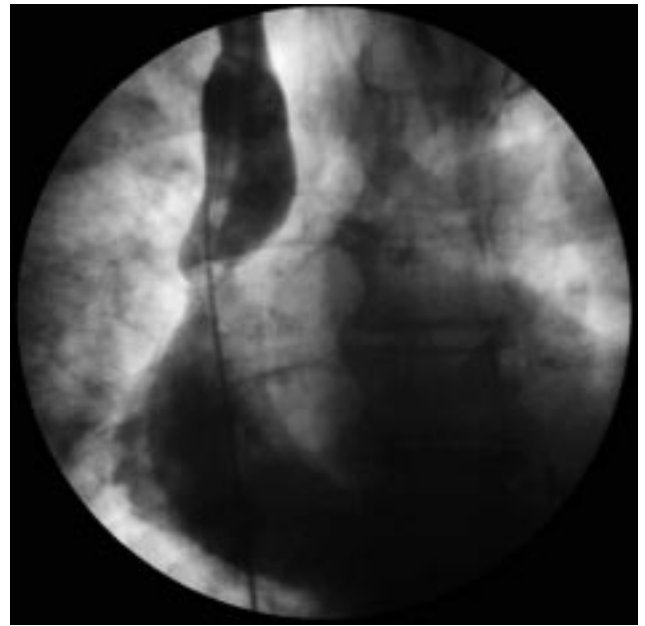
Magnetic resonance imaging (MRI) confirmed the presence of an endoluminal filling defect, suggestive of thrombosis, close to the jugular extremity of the peritoneovenous shunt (Fig. 2).

Endovascular therapy with balloon angioplasty (percutaneous transluminal angioplasty [PTA]) of the SVC thrombosis and stenting of the SVC was performed. A venogram was performed by injecting contrast antegradely through the subclavian sheath. A multipurpose catheter was advanced and the lesion was crossed with a guidewire, dilated and subsequently stented. After this procedure, reduction of the ascites and the disappearance of the jugular turgor rapidly occurred. The presence of thrombophilic factors was excluded and dicumarolic treatment was started. Two (Fig. 3A) and 8 (Fig. 3B) months after the surgical procedure, a computed tomography (CT) scan was performed, showing the patency of the SVC. Subsequently, no massive increase in abdominal volume and/or jugular turgor tested by physical examination nor echodoppler and/or turbulence in the right cardiac atrium test by echocardiography was seen. The patient died in June 2006 due to severe liver function impairment and hepato-renal syndrome.

## Discussion

The present case shows a thrombosis of the SVC in a patient with liver cirrhosis and peritoneovenous shunt, successfully treated by PTA and stenting of the SVC.

The absence of thrombophilic factors in our patient, as well as the particular location of the thrombosis (very close to the jugular extremity of the shunt) suggests that the thrombosis was due to the presence of the shunt extremity in the jugular vein.<sup>2)</sup> Some factors can contribute to this event promoting an intimal injury and favoring the thrombogenic process: (i) the tube itself; (ii) chronic atrial fibrillation<sup>3)</sup>; (iii) chylous ascitis.<sup>4)</sup> In particular, Smadja

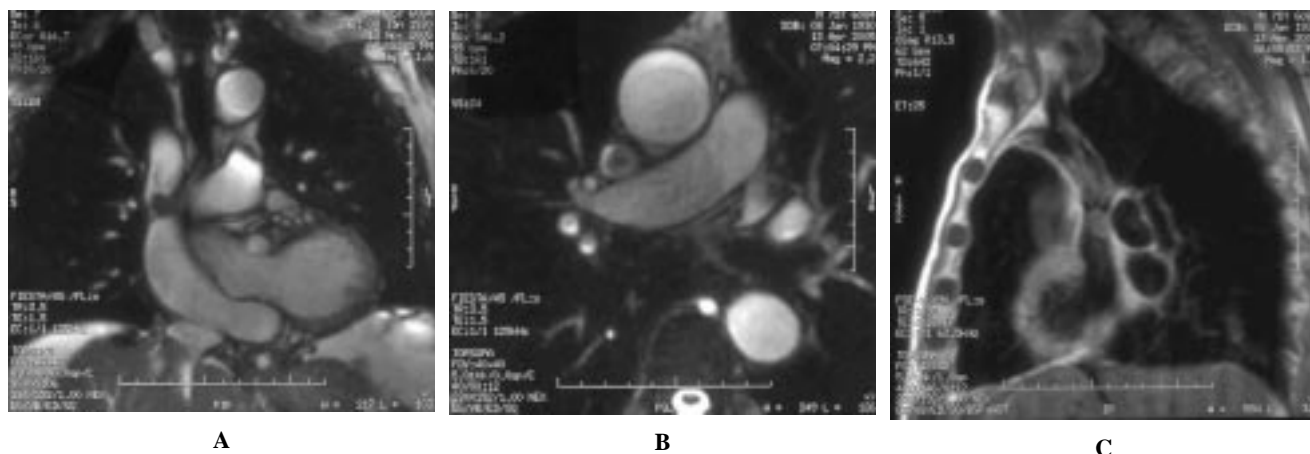


**Fig. 1.** Venography in antero-posterior view showing a stenosis (ST) at the junction of the superior vena cava (SVC) with the right atrium (RA). White lines delineate the cannula (CA) which drains ascitic fluid at the junction level.

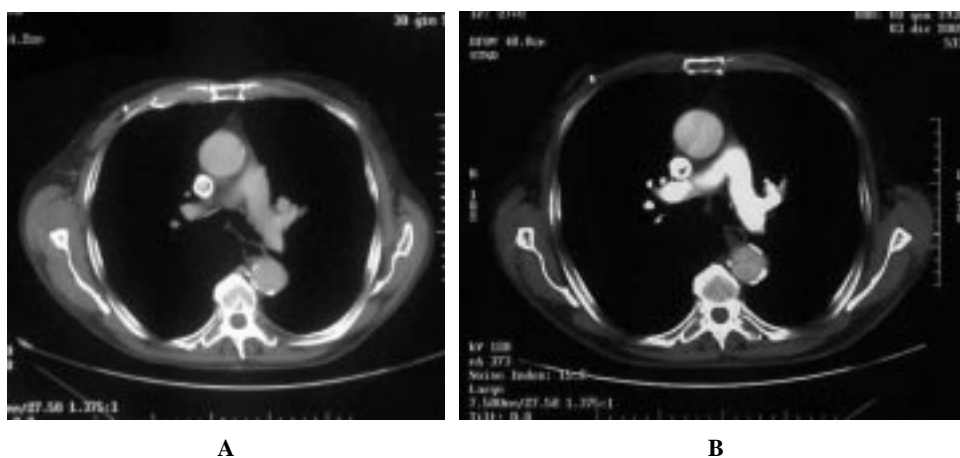
and Franco point out that the obstruction at the level of the venous catheter is due to a sheath of fibrin around the catheter, or to a large clot obstructing the SVC.<sup>5)</sup>

In cirrhotic patients with peritoneovenous shunt, SVC thrombosis represents a possible complication, even some years after the placement of the shunt and should be borne in mind in those cirrhotic patients with peritoneovenous shunt presenting increased abdominal ascitic fluid, in order to start possible pharmacological and/or surgical treatment.

Surgical treatment seems to be the most frequent choice to treat SVC occlusion and seem to be more effective than local ones, including cases unsuccessfully treated by pharmacological treatments. Moreover, among the possible surgical treatments, PTA represents a good choice for primary intervention, as shown in the present case, because it is effective in the short term and does not compromise future open surgical reconstruction. In this respect, the present case is in line with Kalra et al.<sup>6)</sup>; according to these authors, traditional open surgery with veno-venous bypass for benign SVC syndrome seems to be effective over the long term, with possible secondary endovascular interventions to maintain the perfusion, while PTA is a good choice for primary intervention, and eventually to maintain graft patency in surgical treatments.



**Fig. 2.** MR sagittal FSE images (A), MR coronal and axial FIESTA images (B, C) show a hypointense endoluminal filling defect of the superior vena cava 2 cm above the right cardiac atrium, suggestive of thrombosis.



**Fig. 3.** Post-treatment CT.

Two month (A) and 8 month (B) thoracic MDCT images demonstrate no hypodense endoluminal filling defects of the superior vena cava (SVC). The peritoneovenous shunt (hyperdensity around the SVC) is also visualized.

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