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Case Report

SUCCESSFUL TREATMENT OF BUDD-CHIARI SYNDROME WITH PERCUTANEOUS TRANSLUMINAL BALLOON ANGIOPLASTY

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ABSTRACT

Introduction: Budd-Chiari syndrome (BCS) is a relatively rare disease in which an obstruction of hepatic venous outflow causes intrahepatic venous congestion and portal hypertension. Surgical treatment is associated with high morbidity and mortality. Recently, percutaneous transluminal angioplasty (PTA) has been applied to patients with BCS and it has shown a favorable outcome.
Case report: Here we report a case of 50 year old male patient presented with history of insidious onset of abdominal distension and swelling of lower limbs since last six months and yellowish discoloration of eyes since two weeks. He was diagnosed as a case of BCS.
Treatment: Patient underwent successful percutaneous transluminal balloon angioplasty (PTBA). His symptoms significantly improved with patency of IVC at 6 months of follow-up.
Conclusion: PTA is an effective treatment for BCS caused by short-length obstruction of the hepatic portion of the IVC or hepatic veins. However, considering the occurrence of restenosis, regular clinical and ultrasound assessments are necessary after angioplasty.

1. Introduction

Budd-Chiari syndrome (BCS) is a relatively rare disease in which an obstruction of hepatic venous outflow causes intrahepatic venous congestion and portal hypertension. This obstruction of hepatic venous flow can occur at any level, from the small hepatic veins to the junction of the right atrium and inferior vena cava (IVC). Surgical treatment is associated with high morbidity and mortality. Recently, percutaneous transluminal angioplasty (PTA) has been applied to patients with BCS and it has shown a favorable outcome.[1–8] Here we report a case of 50 year old male patient with BCS whom we successfully treated with percutaneous transluminal balloon angioplasty (PTBA).

2. Case report

A 50 year old male patient presented with history of insidious onset of abdominal distension, and swelling of lower limbs since last six months and yellowish discoloration of eyes since two weeks. There was no previous history of jaundice, fever, drug ingestion or abdominal trauma.

2.1. On examination

BP 110/70 mmHg. No signs of anemia and jaundice. Abdomen was distended with dilated subcutaneous veins (Fig. 1) on the abdominal wall and bilateral pitting pedal edema. Per-abdominal examination revealed non tender hepatomegaly, splenomegaly and ascites.

2.2. Investigations

His hematological parameters, renal, liver and thyroid function tests were within normal limits. Viral markers (hepatitis A, B,C & E) were negative. Ultrasound abdomen showed complete obstruction of inferior vena cava (IVC). Upper GI endoscopy revealed esophageal varices. Contrast enhanced computed tomography (CT) revealed complete obstruction of hepatic portion of IVC (Fig. 2).

An IVC venogram from both the distal and proximal sides of the obstruction showed a short, complete obstruction of hepatic portion of IVC (Fig. 3). Length of obstruction was approximately 18–20 mm.

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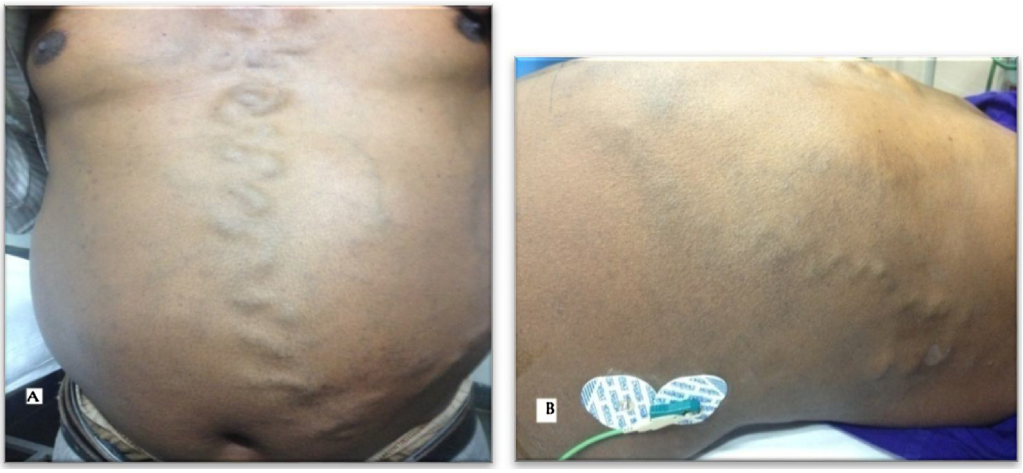

Fig. 1. Photograph showing distended abdomen with prominent abdominal veins.

2.3. Procedure

The procedure was performed under local anaesthesia. The right femoral vein and right internal jugular vein were accessed percutaneously with 8 F and 7 F introducer sheaths, respectively. Intravenous heparin 100 IU/kg and antibiotic were given. An IVC venogram was performed both distal and proximal to obstruction.

An 8 F mullins introducer set was advanced over a 0.032 × 145 cm j tip guide wire in to IVC up to the level of obstruction (Fig. 4A). The guide wire was exchanged for straight tipped brokenbrough needle (Fig. 4B). The needle was pushed slowely and carefully until the tip penetrated the obstruction part and was advanced to above the caval entrance in to the right atrium (Fig. 4C). Now the mullins introducer set was advanced over the needle in to the right atrium and the needle was exchanged with amplatz superstiff 0.035 × 260 cm j tip guide wire which was positioned in superior vena cava (SVC)(Fig. 4D).Then mullins introducer set removed and the obstruction was dilated using TYSHAK II balloon 12 × 40 mm and TYSHAK II balloon 16 × 60 mm (Fig. 4E). An IVC venogram immediately after balloon dilatation angioplasty showed restoration of IVC patency (Fig. 4D). The patient was discharged on oral anticoagulation and Aspirin 75 mg OD.

2.4. Follow-up

Patient was followed up at 6 months and one year. His symptoms dramatically reduced. Prominent abdominal veins disappeared (Fig. 5), Ultrasound abdomen showed marked reduction in liver and spleen size and ascites. Upper GI endoscopy revealed disappearance of esophageal varices. An IVC venogram done at 6 months showed patency of the IVC (Fig. 6).

3. Discussion

Budd-Chiari syndrome (BCS) is a relatively rare disease in which an obstruction of hepatic venous outflow causes intrahepatic venous congestion and portal hypertension. It can be primary or secondary depending on its pathologic features. The primary type is due to congenital obstruction of the hepatic veins or the hepatic portion of the IVC. The secondary type is due to obstruction of the same anatomic structures by a tumor or, more commonly, thrombus or thrombi in patients with some systemic diseases, usually myeloproliferative disorders.[9]

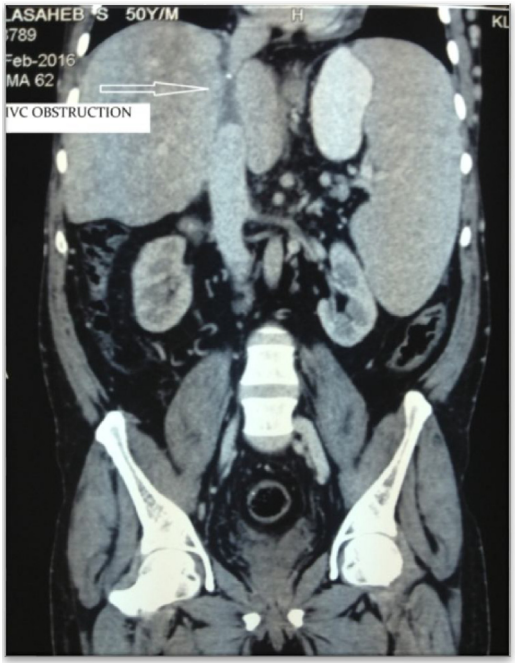

Fig. 2. Contrast enhanced CT showing complete obstruction of hepatic portion of IVC.

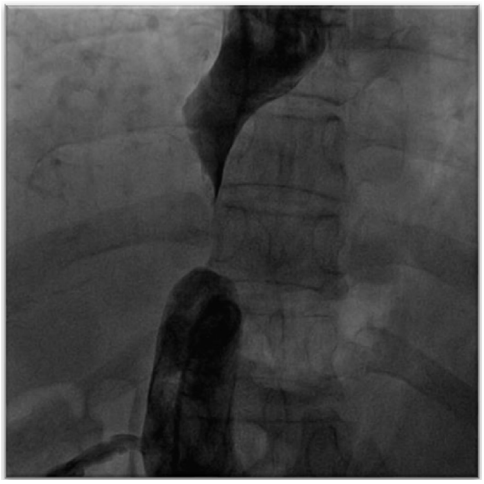

Fig. 3. IVC Venogram showing complete obstruction of hepatic portion of IVC.

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