Aspiration thrombectomy of the hepatic veins in Budd Chiari Syndrome

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Abstract

Budd-Chiari syndrome (BCS) is a rare, potentially life-threatening condition characterised by obstruction of the hepatic venous outflow tract due to thrombosis. Treatment typically involves lifelong anticoagulation and relieving the obstruction. This case report introduces hepatic venous thromboaspiration as an additional endovascular technique to achieve recanalisation. (Acta gastroenterol. belg., **2024,** 87, **535-537**).

Keywords: Budd-Chiari syndrome, thromboaspiration, endovascular treatment Budd-Chiari syndrome

Introduction

Budd-Chiari syndrome (BCS) is a condition characterised by obstruction of the hepatic venous outflow tract due to thrombosis. It can occur at any level from the small hepatic veins to the atrio-caval junction. BCS is rare and its clinical presentation ranges from asymptomatic to potentially life-threatening fulminant acute liver failure. Subsequent chronic fibrotic alterations and liver atrophy, when not lethal, develop in the affected liver segments. Treatment typically involves two main strategies: lifelong anticoagulation related to the underlying procoagulant status and relieving the obstruction of the hepatic venous outflow (1,2). Established endovascular decompression techniques in case of BCS consist of angioplasty, stenting or transjugular intrahepatic portosystemic shunt (TIPS) placement. This case report introduces hepatic venous thromboaspiration as an additional endovascular decompression technique.

Case report

A 31-year-old male previously diagnosed with a JAK2-mutation-positive myeloproliferative neoplasm (*i.e.* essential thrombocytosis), presented with epigastric pain. There were no clinical signs of chronic liver disease e.g. jaundice, spider naevi, caput medusa, ascites, etc. except for splenomegaly. The splenomegaly was not clinically palpable and was attributable to the essential thrombocytosis rather than chronic liver disease. Laboratory results showed elevated transaminase levels (alanine aminotransferase (ALT) 159 U/L and aspartate aminotransferase (AST) 271 U/L), International Normalized Ratio (INR 1.32) and total bilirubin (1.7 mg/dL). Cholestatic liver enzymes (alkaline phosphatase (ALP) and gamma-glutamyltransferase (gGT)) were normal (ALP 93 U/L, gGT 61 U/L). The mild peak of ALP starting around D10 is believed to represent liver regeneration, similar to what is observed after hepatectomy (3). There was neither evidence of biliary obstruction on ultrasound. Abdominal doppler ultrasound revealed absent flow in the right and middle hepatic veins. Subsequent urgent computed tomography (CT) confirmed a BCS caused by simultaneous occlusion of the right and middle hepatic vein (Fig. 1a). The left hepatic vein was patent. Low molecular weight heparines (in therapeutic dosage) were promptly administered and an urgent recanalisation procedure was scheduled.

The procedure was performed under general anaesthesia in an angiography suite. After ultrasound-guided puncture of the right jugular vein, a 10 French (Fr.) guiding sheath (Flexor, Cook Medical, Bloomington, Indiana) was advanced in the inferior caval vein. The middle hepatic vein was recanalised using a 5 Fr. Multipurpose catheter (Performa, Merit Medical, South Jordan, Utah), a Glidewire (Terumo Europe, Leuven, Belgium), and the dilator of the guiding sheath. Digital substraction venography showed a spiderweb pattern of collateral vessels, pathognomonic for BCS (Fig. 1b). After predilatation with a 6 mm angioplasty balloon (Metacross 6x40mm, Terumo Europe, Leuven, Belgium) a mechanical aspiration system (Indigo system Lightning 7, Penumbra Inc, Alameda, California) consisting of a 7 Fr aspiration catheter (CAT7) and a corresponding separator device (SEP7) was advanced in the middle hepatic vein. Mechanical thrombectomy was performed using computer aided intermittent aspiration driven by a vacuum pump, while the tip of the catheter was continuously cleared with the separator device. Next, the exact same procedure was successfully performed in the right hepatic vein. The final venography at the end of the procedure showed no residual thrombus, restored in-line flow and absence of collaterals in both the middle and right hepatic veins (Fig. 1d).

This endosvascular intervention resulted in prompt improvement of symptoms and of liver biochemistry

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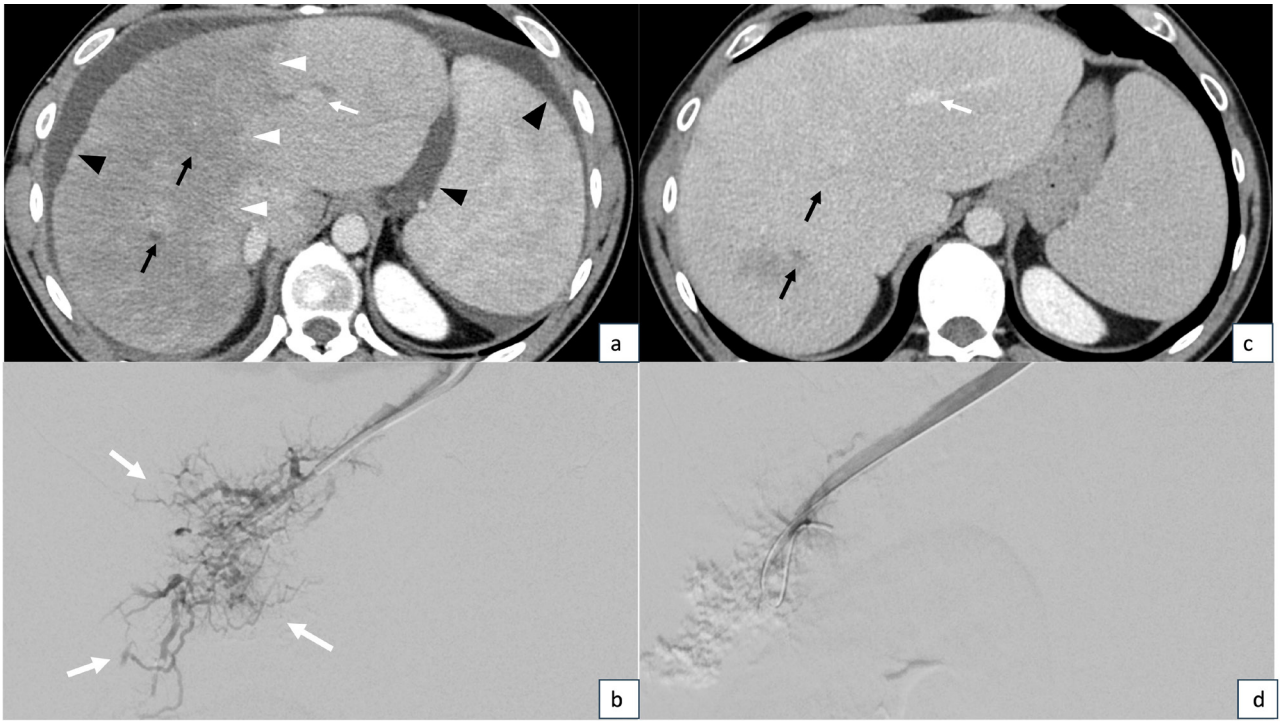

Figure 1. — a) An axial computed tomography image shows thrombotic occlusion of the right and middle hepatic vein (black arrows), and a patent left hepatic vein (white arrow). This outflow obstruction led to acute perfusion changes in the right liver lobe (white arrowheads). The presence of ascites (black arrowheads) indicates acute portal hypertension. b) Angiography trough a 5 French catheter advanced in the occluded middle hepatic vein shows spiderweb like venous collaterals (arrows), indicating Budd-Chiari Syndrome. c) An axial computed tomography image 4 months after the procedure shows reocclusion of the right and middle hepatic vein (black arrows), and a patent left hepatic vein (white arrow). There is compensatory hypertrophy of the left liver lobe, and there are no residual signs of portal hypertension. d) Completion angiography after aspiration thrombectomy shows restored in-line flow in the middle hepatic vein, with absence of venous collaterals.

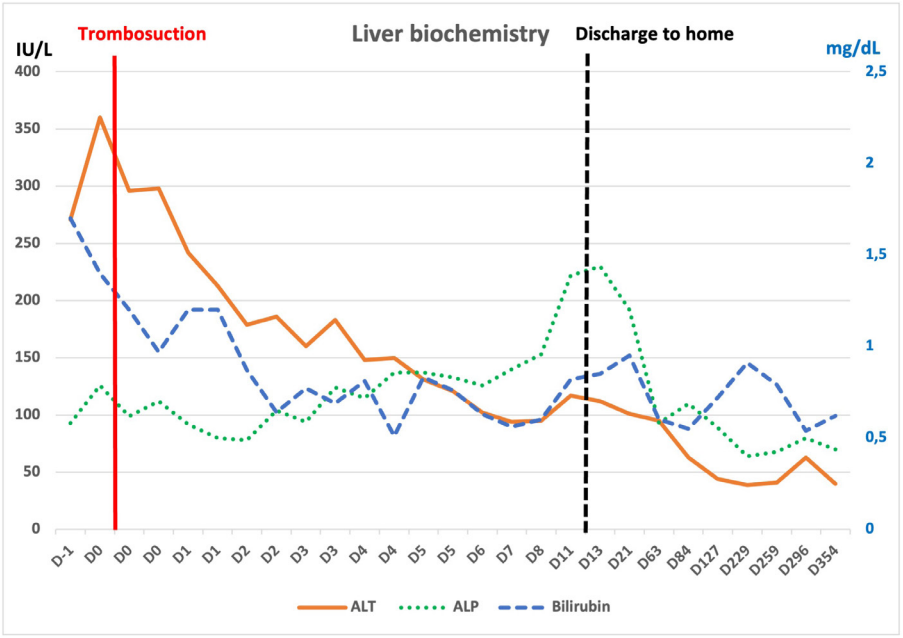

Figure 2. — Evolution of liver function first days after intervention and during follow up.

(Fig. 2), and the patient was discharged after 7 days. A CT-scan 4 months after the procedure showed re-occlusion of the right and middle hepatic veins, but absence of ascites or other signs of portal hypertension, except for splenomegaly, which is attributed to his myeloproliferative disease (Fig. 1c) and was already present at the initial CT. Both repeated ultrasounds as well as the CT-scan showed a gradual development of

atrophy of the affected segments in the right liver and a compensatory hypertrophy of the left liver lobe. The patient remained stable and asymptomatic until now, one year later.

Discussion

The computer-aided intermittent aspiration system applies a strong and constant suction only when the catheter tip is occluded by thrombus, thus minimizing blood loss. It has been shown to remove blood clots efficiently and safely in patients with deep venous thrombosis and pulmonary embolism (4). In BCS, endovascular decompression by anatomic recanalisation should be favoured over TIPS due to its association with superior outcomes (5). In addition to hepatic vein angioplasty or stenting, thromboaspiration offers the potential to not only clear thrombus from the main hepatic veins but also from smaller venules located further upstream in the hepatic venous tree. We estimate that this early restoration of both larger and smaller veins' patency by the added value of computer-aided thromboaspiration, has contributed to the favourable outcome in this patient.

Despite the unfortunate absence of long-term main hepatic vein patency in the current case, there was no worsening of liver function, nor evolution to acute liver failure. Portal hypertension resolved and the need for repeat endovascular intervention, *e.g.* angioplasty (± hepatic vein stenting) or TIPSS-placement, and liver transplantation was successfully avoided up to one year of follow up.

Even though one case-report refrains from firm conclusions, the immediate and rapid improvement of both clinic as well as biochemistry (Fig. 2) supports the added value of this technique in acute BCS as compared to conservative management alone, and probably also to balloon angioplasty without effective clearance of thrombotic material from the veins.

Conclusion

Hepatic venous thromboaspiration expands the endovascular armamentarium in patients with acute BCS, allowing effective clearance of thrombus and restoration of blood flow in both larger and smaller veins. Using this technique, more invasive and irreversible procedures such as TIPS placement (with inherent risk of e.g. encephalopathy or cardiac decompensation) and severe outcomes (including liver transplantation) can be avoided, even in the absence of long-term vein patency.

Conflict of interest

Prof. dr. Wilhelmus Kwanten has a payment and/or honoraria for lectures, presentations, speakers, manuscript writing and educational events form the PanNASH initiative. He had the travel and attendance of EALS ILC 2022 sponsored by Norgine. He has a co-inventor patent on the use of lipopigment imaging for disease (filed by MGH/MIT: US 20190307390).

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The other authors declare that they have no conflict of interest.

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