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Case Report

Direct portal vein recanalization with stenting associated with embolization of esophagogastric varices in a patient with portal vein thrombosis

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ABSTRACT

Introduction: Chronic extrahepatic non-tumoral thrombotic portal vein occlusion in non-cirrhotic patients is a rare condition, affecting 5–10% of patients with portal hypertension.

Presentation of case: The present study reports the case of a young patient without previous comorbidities who presented with portal hypertension secondary to chronic extrahepatic non-tumoral thrombotic occlusion of the portal vein. He underwent portal recanalization with a 12 × 80 mm nitinol self-expandable stent and embolization of esophagogastric varices with fibrous springs and cyanoacrylate via transparieto-hepatic access. Immediate resolution of the *trans*-lesion pressure gradient was obtained transoperatively, while complete remission of esophagogastric varices was verified by endoscopic control during outpatient follow-up.

Discussion: Chronic portal vein occlusion is associated or not with liver cirrhosis. The chronic phase is characterized by cavernomatous transformation of the portal vein, which consists of the formation of multiple collaterals that bypass the lesion. This phase usually courses with portal hypertension and consequent variceal gastrointestinal bleeding. Decompression of the portal system through direct recanalization (angioplasty with stenting) is one therapeutic options.

Conclusion: We conclude that, in the present case, resolving portal hypertension by direct portal recanalization was a good therapeutic option, as it decompressed the portal system while maintaining the hepatopetal flow.

1. Introduction

Chronic extrahepatic non-tumoral thrombotic portal vein occlusion in non-cirrhotic patients is a rare condition, affecting 5–10% of patients with portal hypertension [1]. The standard treatment of variceal bleeding secondary to portal hypertension consists of non-selective beta-blockers associated with serial endoscopic elastic ligation procedures. In cases refractory to clinical/endoscopic treatment, considering the high mortality of surgical venous shunts, the intrahepatic porto-systemic shunts are placed percutaneously; however, this technique diverts the portal flow of the normal liver, which may cause various degrees of postoperative hepatic encephalopathy. To circumvent this undesirable fact, direct percutaneous portal recanalization has emerged as an alternative [2]. This case follows 2020 SCARE guidelines for reporting cases in surgery [3].

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2. Presentation of case

A 33-year-old man presented to the emergency department with abdominal pain in the epigastric region associated with nausea. He reported the symptoms worsening in the postprandial period and denied other associated symptoms. He had no pre-existing chronic diseases. He denied inveterate consumption of alcoholic beverages and/or a previous history of viral hepatitis. The patient had no previous history of abdominal surgeries or pathologies of the upper gastrointestinal tract, such as pathologies of the biliary or pancreatic tracts. There is no previous history of upper gastrointestinal tract neoplasms in his family, nor a history of thrombophilia. He reported, in his epidemiological history, a mild infection with coronavirus disease 2019 (COVID-19) about 45 days before the onset of the abdominal pain.

The initial treatment for dyspeptic syndrome was instituted, but because he did not respond well to the proton pump inhibitors with Omeprazole 40 mg once daily, a diagnostic investigation was followed with an upper digestive endoscopy in which large-caliber esophagogastric varices were identified extending from the distal third of the esophagus to the esophagogastric transition. Therefore, primary prophylaxis (propranolol 20 mg twice daily) for variceal upper digestive hemorrhage and endoscopic elastic ligation sessions were scheduled.

After about 4 months being asymptomatic, he started presenting with high digestive hemorrhage associated with hemodynamic instability that required the transfusion of blood derivatives and multiple endoscopic approaches to control the bleeding by elastic ligation of the varices. Despite the multiple elastic ligations, the patient has recurrent episodes of digestive hemorrhage with hemodynamic instability.

The continuing diagnostic investigation of the portal hypertension syndrome revealed no signs of chronic hepatopathy. A contrast-enhanced computed tomography scan of the abdomen revealed an extrahepatic portal vein with reduced diameter (0.6 cm) as well as laminar filling failure inside and with adjacent collateral vessels (cavernomatous transformation) (Fig. 1).

Given the clinical/endoscopic treatment failure and the risk of hemorrhagic shock, the team formed by three vascular surgeons, two of them also interventional radiologists, opted for direct recanalization of the portal system through percutaneous intraluminal portal vein angioplasty to depressurize esophagogastric varices and maintain the preferential hepatopetal flow.

Under general anesthesia, with the patient in the horizontal dorsal decubitus position, we initially performed indirect portography via selective catheterization of the superior mesenteric artery, demonstrating the integrity of the intrahepatic portal branches and confirming the preferential portal flow for esophagogastric varices in the topography of the small gastric curvature. Next, transparieto-hepatic puncture of the right peripheral portal branch was performed with a 22G × 15 cm Chiba needle, followed by the introduction of a 0.018″ metallic guide wire from an access kit (Neff Percutaneous Access Set 100®; Cook Medical, Bloomington, Indiana, USA) with subsequent progressive dilation of the pathway using the kit's coaxial system and placement of an 8Fr × 11 cm valved introducer. The portal segmental occlusion was then transposed using a 4Fr vertebral angiographic catheter and 0.035″ hydrophilic guide wire. With the tip of the angiographic catheter positioned after portal occlusion was achieved, an image of the direct portography was obtained followed by measurement of the *trans*-lesion pressure gradient (20 mmHg). After systemic heparinization, a hydrophilic guide wire was replaced by an Amplatz guide wire followed by pre-dilation of the portal lesion with a 7 × 40 mm semi-compliant balloon catheter (Fig. 2A and B). After the initial pre-dilation, we embolized the esophagogastric varices through selective microcatheterization (2.8Fr microcatheter) of varicose branches from the splenic vein using biological glue (synthetic cyanoacrylate) and Lipiodol in a 1:8 ratio. The embolization was complemented with fibrinated micro-springs. A second pre-dilation of the portal stenosis was performed with a 12 × 40 mm semi-compliant balloon. We then proceeded to position and release the 12 × 80 mm nitinol auto-expandable stent from the splenic vein (middle third) up to the distal portal vein, followed by its post-dilatation/accommodation with a 12 × 40 mm balloon. The final control showed that the patency of the treated segment and the new measurement of the pressure gradient pre- and post-stenting was equal to zero, confirming adequate depressurization of the portal system (Fig. 3). To conclude the procedure, the 8Fr valve introducer was removed under fluoroscopy guidance with embolization of the hepatic tract with biological glue and Lipiodol (ratio 1:1).

During hospitalization, anticoagulation with enoxaparin dose of 1mg/kg twice daily was maintained. The patient had an uneventful postoperative course and was discharged on the 4th postoperative day with oral rivaroxaban (20 mg/day) and acetylsalicylic acid (100 mg/day). During outpatient follow-up, he remained asymptomatic without new episodes of upper gastrointestinal bleeding. At the control upper digestive endoscopy performed on the 30th day postoperative, complete remission of the esophageal varices was observed; on upper abdominal Doppler ultrasound, a patent stent in the portal vein and monophasic-hepatopetal pattern flow in the portal and splenic veins with a mean flow velocity of 42.7 cm/s were noted, as was monophasic-hepatopetal flow in the right and left portal branches.

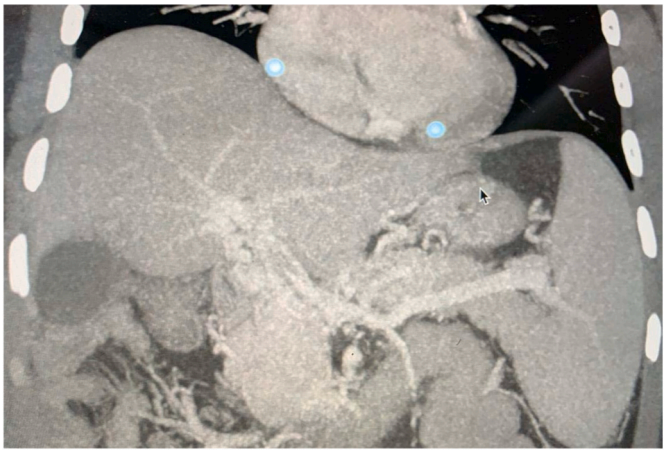

Fig. 1. Abdominal computed tomography angiography showing cavernomatous transformation of the portal vein.

3. Discussion

Chronic portal vein occlusion is associated or not with liver cirrhosis. The causes differ between cirrhotic and non-cirrhotic patients, determining differences in therapeutic rationale. In non-cirrhotic patients, hypercoagulability states and intra-abdominal conditions such as pancreatitis and extrinsic compression are important causes of portal vein thrombosis [4].

The chronic phase is characterized by cavernomatous transformation of the portal vein, which consists of the formation of multiple collaterals that bypass the lesion [5]. This phase usually courses with portal hypertension and consequent variceal gastrointestinal bleeding [6]. Decompression of the portal system through direct recanalization (angioplasty with stenting) is one therapeutic options [7]. Surgical porto-systemic shunts are indicated when less invasive, endovascular, or endoscopic measures are unsuccessful [8].

Consensus in the literature is lacking on which patients with chronic portal vein occlusion would benefit from direct portal recanalization through angioplasty with stenting [9,10]. In 2019, Marot et al. observed the primary patency at 2 years of 15 non-cirrhotic patients undergoing portal recanalization with stenting and proposed a classification that would guide a more precise indication of which patients would benefit most from this approach [2]. Their classification proposes a division into 3 groups, in which type 1 corresponds to occlusion of the origin of the

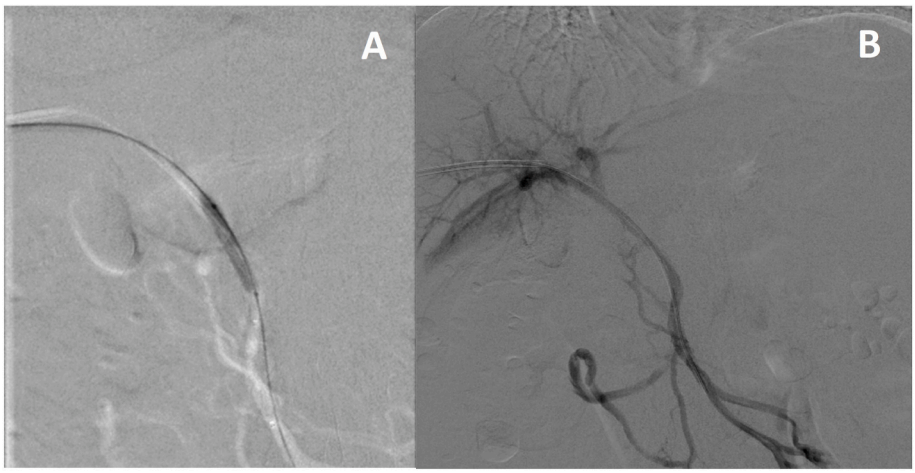

Fig. 2. A. Pre-dilatation using a 7 × 40 mm Advance ATB OTW® balloon catheter. B. Angiographic status after pre-dilation showing residual stenosis of approximately 60% of the lumen of the treated vessel.

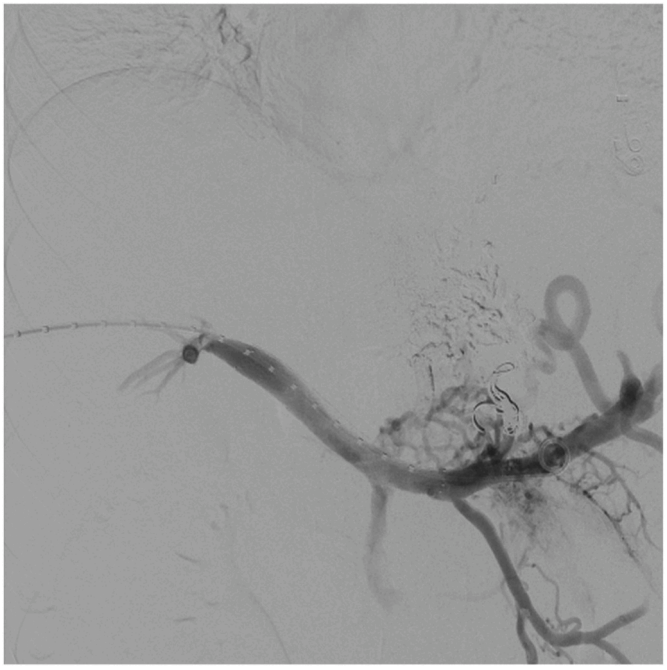

Fig. 3. Control angiography after angioplasty with stenting using a pigtail angiographic catheter with tip positioned in the splenic vein.

portal vein with or without extension to the right and/or left portal branch, type 2 corresponds to type 1 with extension to the main segmental branches, and type 3 corresponds to cases with extension to the distal branches. The study concluded that patients with type 1 seem to benefit more from the technique employed.

Transparieto-hepatic access to reach the portal vein lumen is the approach of choice among non-cirrhotic patients. It allows a variety of endovascular procedures for portal recanalization and allows selective catheterization of esophagogastric varicose vessels for embolization at the same surgical time [11]. As our patient had no chronic hepatopathy and no past (or family) history of thrombotic venous disease, we hypothesized a causal relationship with the previous COVID-19 infection [12].

Angioplasty of the portal vein with stenting was first described using expandable balloon stents [13]. Stenting for direct portal recanalization is recommended in cases of residual stenosis greater than 30% (post-balloon angioplasty), stenosis of neoplastic or inflammatory etiology, and elastic recoil after balloon angioplasty [7,13]. A *trans*-lesion pressure gradient greater than 10 mmHg has also been reported as an indication criterion for stenting, with reports of an incidence of about 30% among patients with portal stenosis after liver transplantation [14]. In our patient, after balloon angioplasty, a residual luminal stenosis rate of approximately 60% was observed, and a self-expanding stent was placed at that moment.

Most of the data available on the endovascular approach to portal stenosis are derived from studies of patients undergoing liver transplantation. In case series including non-transplanted patients, successful portal recanalization was achieved in about 80% of cases. Symptomatic relief after stenting was achieved in 72% in some series [15,16].

Outpatient follow-up with upper gastrointestinal endoscopy and Doppler ultrasonography of the upper abdomen for evaluating primary assisted patency revealed that the treatment initially achieved its proposed goals: resolution of portal hypertension and symptom improvement.

4. Conclusion

We conclude that, in the present case, resolving portal hypertension by direct portal recanalization was a good therapeutic option, as it decompressed the portal system while maintaining the hepatopetal flow.

Ethical approval

As the manuscript is not a research study, we only have the patient consent for writing and others forms of publication. Also, the ethical approval for this case reports has been exempted by our institution.

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We do not have any funding source, this manuscript is just a case report, not a research.

Author contribution

Renan da Rocha, Paulo Diniz, Alessandra Leão and Juan Rodriguez contributions to conception, design, collected the patient details and wrote the paper. Priscilla Ribeiro, José Souza and Leonardo Cavalcante made contributions to patient management. Leonardo Cavalcante, Priscilla Campelo and Marcos Velludo critically revised the article. All authors read and approved the final manuscript.

Trial register number

The manuscript is a case report, not considered a formal research involving participants.

Guarantor

Renan Danilo Lima da Rocha.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Declaration of competing interest

We do not have any conflicts of interests.

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