Successful Endovascular Treatment of Acute Portal and Superior Mesenteric Vein Thrombosis in a Non-Cirrhotic Patient

Tratamento Endovascular Bem-Sucedido da Trombose Aguda da Veia Porta e da Veia Mesentérica Superior num Doente Sem Cirrose

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Abstract:

Portal vein thrombosis (PVT) in the absence of cirrhosis is an uncommon but potentially life-threatening condition. It is frequently associated with systemic prothrombotic disorders. Early identification of the underlying cause, evaluation of PVT extension and underlying complications, and appropriate therapeutic intervention are crucial for prognosis. This case describes a 62-year-old male presenting with acute abdominal pain, in whom imaging revealed PVT with extension to the superior mesenteric vein, without underlying cirrhosis. Despite prompt anticoagulation, endovascular treatment with catheter-directed thrombolysis and mechanical thrombectomy was conducted, achieving successful recanalization. Etiological investigation excluded major thrombophilic conditions. Long-term anticoagulation was maintained with favourable evolution. Non-cirrhotic PVT requires early diagnosis and individualized management. Endovascular therapy should be considered in selected cases with a dismal prognosis and/or mesenteric involvement.

Keywords: Portal Vein; Radiology, Interventional; Thrombectomy; Thrombolytic Therapy; Venous Thrombosis.

Resumo:

A trombose da veia porta (TVP) na ausência de cirrose é uma entidade rara, mas potencialmente grave, frequentemente associada a estados pró-trombóticos. O reconhecimento precoce da etiologia, a avaliação da extensão da TVP e complicações associadas, assim como a intervenção terapêutica adequada, são determinantes para o prognóstico. Apresenta-se o caso de um homem de 62 anos admitido por dor abdominal aguda, no qual se identificou TVP com extensão à veia mesentérica superior, sem evidência de cirrose. Apesar de anticoagulação inicial, foi necessária intervenção endovascular com trombólise dirigida e trombectomia mecânica. O estudo etiológico excluiu trombofilias major. O doente manteve evolução favorável sob anticoagulação prolongada. Neste contexto, o diagnóstico precoce e abordagem individualizado são fundamentais. A intervenção endovascular deve ser considerada em casos selecionados com um mau prognóstico e/ou envolvimento da VMS.

Palavras-chave: Radiologia de Intervenção; Terapia Trombolítica; Trombectomia; Trombose Venosa; Veia Porta.

Learning Points

1. Non-cirrhotic portal vein thrombosis with mesenteric extension represents a high-risk vascular emergency requiring early multidisciplinary management.
2. Superior mesenteric vein involvement is associated with lower spontaneous recanalization rates and increased risk of intestinal ischemia.
3. Early endovascular intervention combined with anticoagulation can restore porto-mesenteric patency and avoid bowel resection in selected cases.
4. Indefinite anticoagulation should be considered in patients with extensive thrombosis and previous venous thromboembolism, even when major thrombophilia is excluded.

Introduction

Portal vein thrombosis (PVT) in the absence of liver cirrhosis is an uncommon but clinically relevant vascular disorder, associated with significant morbidity and long-term complications. In contrast to cirrhotic PVT, non-cirrhotic portal vein thrombosis is more frequently associated with systemic prothrombotic conditions, including myeloproliferative neoplasms, inherited thrombophilia, paroxysmal nocturnal hemoglobinuria, and inflammatory or infectious states.[1,2] Identification of the underlying aetiology is of paramount importance, as targeted treatment of the causal condition may substantially influence prognosis, such as cytoreductive therapy in myeloproliferative disorders or complement inhibition in paroxysmal nocturnal hemoglobinuria.[1,3]

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Anticoagulation has long been considered the cornerstone of therapy for non-cirrhotic PVT. However, increasing evidence suggests that anticoagulation alone may be insufficient in a relevant subset of patients, particularly in those with extensive thrombosis, complete portal vein occlusion, or involvement of the superior mesenteric vein (SMV). These features are associated with a lower likelihood of spontaneous or anticoagulation-induced recanalization and a higher risk of progression to chronic portal hypertension and its complications.[4] In this context, interventional radiology has emerged as an increasingly important therapeutic option. Endovascular techniques, including catheter-directed thrombolysis, mechanical thrombectomy, or combined approaches, have demonstrated high rates of portal vein recanalization when performed early and in experienced centres. Available data suggest that a combined strategy - anticoagulation plus endovascular intervention - may achieve superior recanalization rates and reduce the incidence of chronic portal hypertension when compared with anticoagulation alone, particularly in patients with extensive thrombosis or mesenteric vein involvement.[1,5,6]

Furthermore, recent evidence has refined long-term management strategies. The study by Plessier et al demonstrated that in selected patients with idiopathic non-cirrhotic PVT and complete recanalization, anticoagulation discontinuation may be considered under strict surveillance, particularly guided by D-dimer levels.[7] We report a case that illustrates the contemporary management of acute non-cirrhotic PVT with SMV involvement, emphasizing the pivotal role of interventional radiology in achieving successful recanalization.

Case Report

A 62-year-old male with known cardiometabolic risk factors (class 1 obesity, arterial hypertension, and dyslipidaemia) and a previous history of left popliteal deep vein thrombosis presented to the Emergency Department (ED) with acute-onset abdominal pain. The symptoms had begun one week before admission and were characterized by severe pain localized to the right upper quadrant, which developed after a copious meal. The pain persisted for approximately one week and was consistently exacerbated by food intake. This was associated with vomiting and food intolerance, which, together with the persistent pain, prompted presentation to the ED.

There was no history of recent vaccination before the acute event. The patient had no family history of thrombotic disease and denied alcohol consumption or smoking. On admission, he was hemodynamically stable, with elevated lactate dehydrogenase (LDH) levels, mildly increased C-reactive protein, and slight elevation of liver enzymes. Doppler ultrasound demonstrated portal vein thrombosis. Contrast-enhanced computed tomography confirmed acute thrombosis of the portal vein with extension to the superior mesenteric vein, associated with bowel wall oedema (Fig. 1).

Figure 1: Coronal reconstruction of contrast-enhanced abdominopelvic computed tomography. [**A**] Sub occlusive thrombosis of the portal and mesenteric venous system (before procedure); [**B**] Complete resolution of porto-mesenteric thrombosis after intervention.

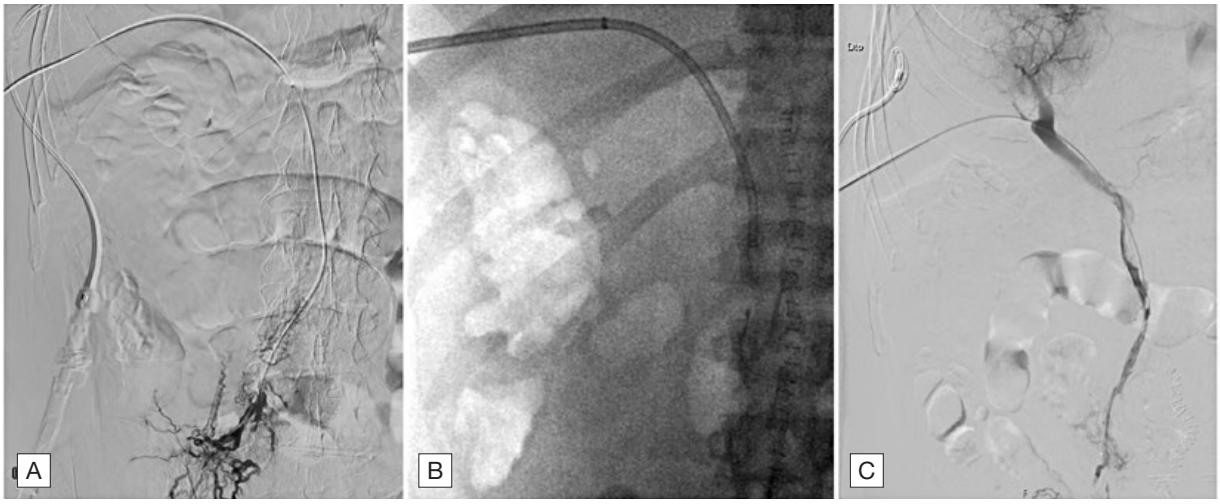

Figure 2: [**A**] Angiographic study with a microcatheter positioned in a distal branch of the superior mesenteric vein (SMV), demonstrating occlusion of this segment; [**B**] Mechanical thrombectomy catheter positioned within the SMV; [**C**] Follow-up angiographic study with a microcatheter in a distal branch of the SMV showing recanalization with residual mural thrombosis.

An exploratory laparotomy was performed, revealing thickened, violaceous intestinal walls involving an ileal segment approximately 70 cm from the ileocecal valve, extending over a length of about 60 cm. As intestinal viability was preserved, surgical resection was not performed, and the patient was referred for endovascular percutaneous transhepatic thrombectomy with intravascular thrombolysis. Local alteplase infusion was maintained for three days. Subsequent angiographic control demonstrated complete recanalization of the superior mesenteric vein, main portal vein trunk, and left portal vein branch, with persistent occlusion of the right portal vein branch (Fig. 2).

At hospital discharge, the patient was asymptomatic and anticoagulated with edoxaban. Etiological investigation for prothrombotic conditions revealed, in addition to class 1 obesity (BMI 30.4 kg/m^2), moderately elevated homocysteine levels (18.3 µmol/L). There were no persistent systemic inflammatory markers or clinical features suggestive of Behçet's disease or other autoimmune disorders. Antiphospholipid antibodies were negative, as were the *JAK2* mutation, factor V Leiden mutation, and the prothrombin gene mutation. Functional assays of coagulation factors, including protein C, protein S, and antithrombin III, were within normal ranges. Heterozygosity for the *MTHFR* gene was the only genetic abnormality identified. Liver elastography demonstrated a controlled attenuation parameter (CAP) of 295 dB/m and liver stiffness of 9.5 kPa, consistent with S3 steatosis and F2 fibrosis, with no evidence of cirrhosis. Six months after the acute event, an upper endoscopy was performed, showing no signs of portal hypertension.

After two years of follow-up following the thrombotic event and endovascular intervention, the patient remains asymptomatic, with patent portal and superior mesenteric veins (Fig. 3), under long-term anticoagulation with edoxaban 60 mg daily. Given the clinical presentation of PVT with SMV involvement and a previous episode of deep vein thrombosis, indefinite anticoagulation was considered appropriate.

Discussion

Identification of the underlying cause is particularly relevant in PVT in the absence of liver cirrhosis, as targeted treatment may directly influence prognosis and recurrence risk. In the present case, however, an extensive etiological work-up failed to identify a major prothrombotic disorder, a finding that is consistent with approximately one-third of reported cases.[1] Although heterozygosity for the *MTHFR* gene was detected, this genetic variant has not been consistently associated with an increased risk of portal vein thrombosis and is not currently recommended as part of routine thrombophilia screening.[2] Obesity, on the other hand, is increasingly recognized as a relevant prothrombotic condition and may have contributed as a cofactor in this patient. Indeed, up to one quarter of patients with non-cirrhotic PVT are obese and present at least one identifiable thrombotic risk factor.[1,8]

Even in the absence of a clearly identifiable triggering factor, the severity of presentation in this patient - namely, extensive PVT with superior mesenteric vein involvement and a previous episode of deep vein thrombosis - supports the indication for long-term anticoagulation. Current evidence suggests that patients with unprovoked PVT, particularly those with extensive thrombosis or recurrent venous thromboembolism, benefit from indefinite anticoagulation to reduce the risk of recurrence.[2,9] Recent data from Plessier *et al* further indicate that, in selected patients with idiopathic non-cirrhotic PVT and complete recanalization, anticoagulation discontinuation may be considered under close surveillance guided by D-dimer levels.[7] Nevertheless, in patients such as the one described here — with mesenteric

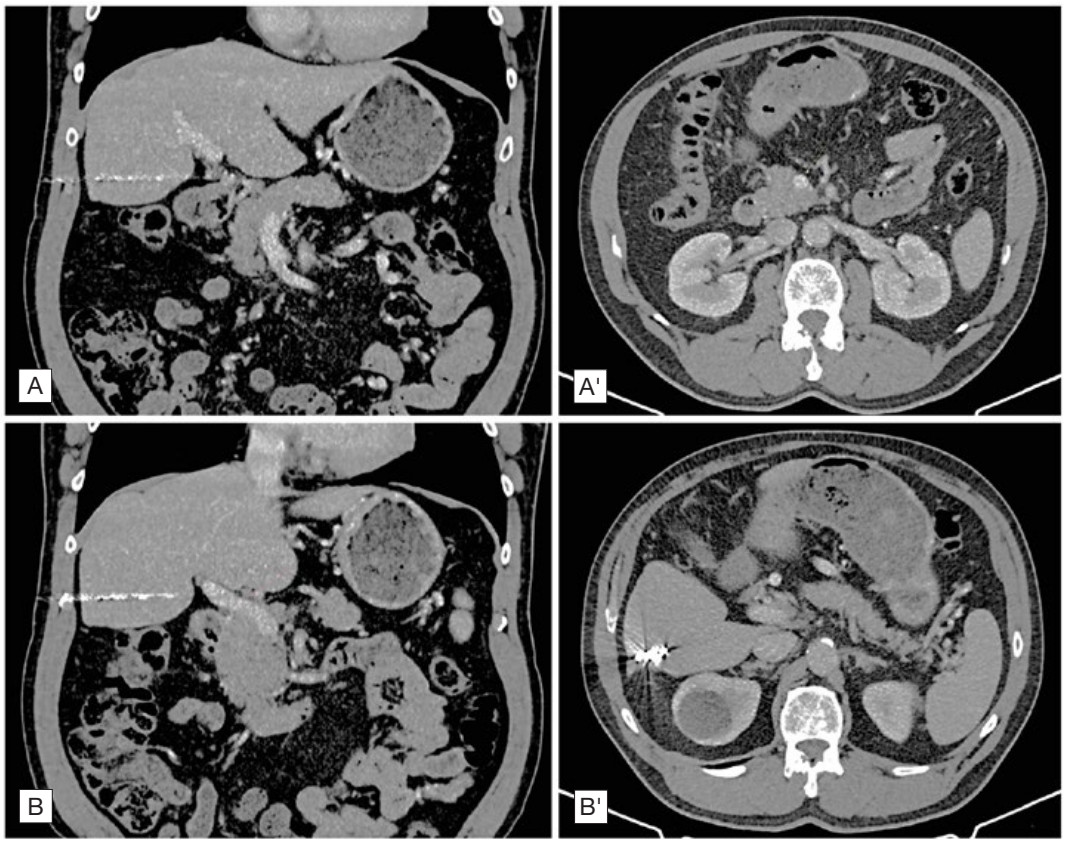

Figure 3: Two-year follow-up CT scan. Images [**A**] and [**A'**] depict the superior mesenteric, while images [**B**] and [**B'**] depict the portal vein. Images A and B correspond to coronal reconstructions, whereas images A' and B' correspond to axial sections.

vein involvement and a history of venous thromboembolism — long-term anticoagulation remains the most appropriate therapeutic strategy.

A fundamental step in the diagnostic approach to PVT is determining the presence or absence of underlying cirrhosis, as this distinction has major implications for etiological investigation and therapeutic decision-making. Imaging findings, including preserved liver morphology and absence of portal hypertension, together with liver elastography, play an important role in excluding cirrhosis in doubtful cases.[2] In the present patient, the absence of morphological signs of chronic liver disease and elastographic findings supported a non-cirrhotic etiology.

The extent of thrombosis, particularly involvement of the superior mesenteric vein, is a key determinant of prognosis and management. Mesenteric extension is associated with a higher risk of intestinal ischemia, lower rates of spontaneous recanalization, and increased morbidity, such as bowel ischemia.[1,2] In this case, clinical features suggestive of intestinal suffering, including prolonged postprandial abdominal pain, elevated lactate dehydrogenase levels, and radiological signs of bowel involvement, justified surgical exploration. Given the preserved intestinal viability, an endovascular approach was favoured over bowel resection. The decision to proceed with endovascular therapy was supported by accumulating evidence indicating that anticoagulation alone leads to incomplete recanalization in a substantial proportion of patients with non-cirrhotic PVT, particularly in cases of extensive or occlusive thrombosis. Reported rates of failure to achieve recanalization with anticoagulation alone reach up to 40%, especially when the superior mesenteric vein is involved.[1,4] In this setting, catheter-directed thrombolysis and mechanical thrombectomy have emerged as effective therapeutic options, particularly when performed early and in experienced centres. These approaches are associated with higher recanalization rates, improved preservation of intestinal viability, and a reduced risk of progression to chronic portal hypertension.[10,11] In the present case, combined endovascular therapy achieved complete recanalization of the portal and mesenteric venous system, restoring mesenteric perfusion and avoiding intestinal resection, thereby underscoring the pivotal role of interventional radiology in selected patients with acute non-cirrhotic PVT.

Finally, this case highlights the importance of a multidisciplinary approach in the management of acute portal vein thrombosis. Optimal care requires: i) identification and treatment of underlying etiological factors whenever possible; ii) prompt initiation of anticoagulation, preferably with low-molecular-weight heparin followed by direct oral anticoagulants, except in specific situations such as triple-positive

antiphospholipid syndrome; and iii) careful evaluation for endovascular intervention in cases with extensive thrombosis, low probability of spontaneous recanalization, or evidence of intestinal ischemia. ■

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FN – Conceptualization of the study, clinical management, data interpretation, manuscript drafting, and critical revision.
MTG – Critical review of the manuscript and intellectual content revision.
PS – Manuscript drafting, critical revision of the article, and contribution to data interpretation and imaging analysis.
All authors approved the final version to be published.

Declaração de Contribuição

FN – Conceptualização do estudo, gestão clínica, interpretação dos dados, redação do manuscrito e revisão crítica.
MTG – Revisão crítica do manuscrito e do conteúdo científico.
PS – Redação do manuscrito, revisão crítica do artigo e contributo para a interpretação dos dados e análise das imagens.
Todos os autores aprovaram a versão final a ser publicada.

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