# Portal Vein Thrombosis in Cirrhosis: Challenges in Management and Approach, a Case Report

## Trombose da Veia Porta na Cirrose: Desafios na Abordagem e Tratamento, Relato de Caso

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## Abstract:

Portal vein thrombosis is a common complication in cirrhosis, especially in patients with decompensated disease, associated hepatocellular carcinoma, or those awaiting liver transplantation. Anticoagulation is the first-line therapy to improve transplant feasibility and outcomes in this context, although bleeding risk, particularly in the presence of portal hypertension, adds a notable challenge and complexity to its management.

We report the case of a patient with cirrhosis who presented with gastrointestinal bleeding due to gastroesophageal variceal rupture and hepatic encephalopathy, complicated by porto-mesenteric thrombosis. While undergoing pre-liver transplantation workup and shortly following the beginning of anticoagulation, he developed a subdural hematoma, highlighting the complexity of risk-benefit assessment and therapeutic decision-making. Thus, this case report highlights the delicate balance between haemorrhagic risk and thrombotic benefit and supports individualized, multidisciplinary management of PVT in cirrhotic patients.

**Keywords:** Anticoagulants; Hemorrhage; Hypertension, Portal; Liver Cirrhosis; Liver Transplantation; Portal Vein; Portasystemic Shunt, Transjugular Intrahepatic; Venous Thrombosis.

## Resumo:

A trombose da veia porta é uma complicação frequente no contexto de cirrose, particularmente no contexto de cirrose descompensada, carcinoma hepatocelular associado e/ou em doentes candidatos a transplante hepático. A anticoagulação é o tratamento de primeira linha, nomeadamente no que concerne a uma melhoria da viabilidade e dos *outcomes* do transplante, embora o risco hemorrágico, particularmente na presença de hipertensão portal, adicione complexidade significativa à sua gestão.

Os autores apresentam o caso clínico de um doente com diagnóstico recente de cirrose, que se manifestou inicialmente por uma hemorragia gastrointestinal devido à ruptura de varizes gastroesofágicas e encefalopatia hepática. Durante o estudo pré-transplantação hepática, uma trombose porto-mesentérica é diagnosticada e num curto período após o início da anticoagulação, o doente desenvolveu um hematoma subdural. Com este caso clínico, os autores pretendem demonstrar a complexidade e o difícil balanço entre o risco e o benefício desta abordagem terapêutica, reforçando a importância de uma gestão individualizada e multidisciplinar da trombose da veia porta no contexto de cirrose.

**Palavras-chave:** Anticoagulantes; Cirrose Hepática; Derivação Portossistémica Transjugular Intra-Hepática; Hemorragia; Hipertensão Portal; Transplantação de Fígado; Trombose Venosa; Veia Porta.

### Learning Points

1. Portal vein thrombosis is a frequent complication in patients with cirrhosis, particularly in decompensated cirrhosis and liver transplantation candidates.
2. Anticoagulation is the first-line medical therapy, despite its challenging management.
3. Bleeding risks associated to anticoagulation in portal vein thrombosis management can be portal hypertension related or not.
4. Anticoagulation intends to improve survival and reduce portal hypertension-related bleeding through portal vein recanalization and prevention of thrombosis progression
5. Transjugular intrahepatic portosystemic shunt can also be an alternative when anticoagulation is contraindicated or if portal vein recanalization is not achieved with anticoagulation therapy.

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## Introduction

Portal vein thrombosis (PVT) is defined as a non-malignant obstruction involving the portal vein, its branches, its radicles or any combination of these, that can extend into the mesenteric or splenic veins. PVT is a frequent complication in patients with cirrhosis, particularly in those with decompensated disease, hepatocellular carcinoma, and/or awaiting liver transplantation.[1-3]

In patients with cirrhosis, systematic screening of PVT is indicated while screening for hepatocellular carcinoma (HCC). In the case of a confirmed diagnosis, anticoagulation is recommended regardless of the degree of occlusion or extension of PVT, to improve the feasibility and outcomes of LT. However, therapeutic management can be challenging and requires an individualized, multidisciplinary approach.[4,5] Anticoagulation is the first-line medical therapy to improve transplant feasibility and outcomes. Concerns regarding bleeding complications, especially in the setting of portal hypertension, increase the therapeutic challenge of managing these patients. However, current evidence suggests that prophylactic anticoagulation could prevent decompensation, portal vein thrombosis and improve survival.[1,4]

## Case Report

A 65-year-old man was referred to our Hepatology Department for a pre-liver transplantation evaluation due to cirrhosis associated with a metabolic dysfunction-associated steatotic liver disease (MASLD), complicated by a clinically significant portal hypertension (oesophageal variceal rupture episode as first presentation 3 years before) and grade 2 hepatic chronic encephalopathy. The patient presented the following associated comorbidities: a glucose-6-phosphate dehydrogenase deficiency and a metabolic syndrome including type 2 diabetes (with a 25-year evolution, requiring insulin therapy), dyslipidaemia, obesity (with a body mass index value of 32.3 kg/m$^2$) and arterial hypertension. His usual treatment comprehended rifaximin 550 mg twice daily (2d), lactulose 30 mL 2d, insulin NovoMix® 48U 2d, propranolol 20 mg 2d, rosuvastatin 20 mg daily (1d) and sitagliptin 100 mg 1d.

In the context of pre-liver transplant workup, the laboratory tests showed a normocytic anaemia (haemoglobin 8.3 g/dL), thrombocytopenia (platelets 64x109/L), INR 1.4, alanine aminotransferase 40 U/L (normal value (N) < 45), aspartate aminotransferase 70U/L (N< 35), γ-glutamyl transferase 70 U/L (N< 55), alkaline phosphatase 85U/L (N< 130), bilirubine 25 µmol/L (N< 17) and creatinine 133 umol/L (1.50 mg/dL). Other causes of liver disease were also excluded: serologies for hepatitis B, C and E virus were negative, the autoimmune study was negative (including antinuclear antibodies, anti-LKM1, anti-SLA, anti-smooth muscle, antimitochondrial antibodies, anti-sp100, and anti-gp210 antibodies); serum protein electrophoresis and immunoglobulin levels showed no abnormalities, and alpha-1 antitrypsin and ceruloplasmin levels were also normal. No alcohol consumption was reported by the patient. A computed tomography (CT) revealed a partial porto-mesenteric vein thrombosis (Fig. 1).

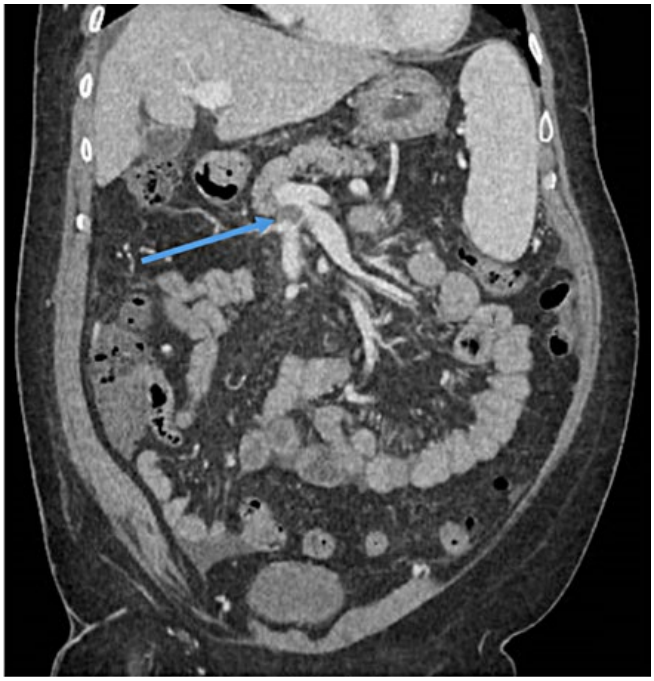


**Figure 1**: Abdominal and pelvic CT scan demonstrating partial thrombosis of the superior mesenteric vein (SMV) (blue arrow), contacting the origin of the portal trunk (blue arrow).

Anticoagulation treatment with tinzaparin 175 U/kg daily was initiated, followed by a bridging period with overlapping warfarin therapy, until targeting an INR of 2–3. A hemodynamic evaluation confirmed a clinically significant portal hypertension with a hepatic venous pressure gradient of 20 mmHg and the liver fragment histology was compatible with cirrhosis, probably associated with a MASLD. It should be noted that, in the meantime, the patient developed ascites, which became refractory due to diuretic intolerance secondary to renal insufficiency.

Twelve days after starting the anticoagulation therapy, the patient developed a clinic of right hemiparesis, and a cerebral CT scan revealed an acute right frontoparietal subdural hematoma resulting in a mass effect on the adjacent sulci and consequent leftward midline shift (Fig. 2). No traumatic head injury preceded these symptoms. The patient's management consisted of embolization of the middle meningeal artery and discontinuation of anticoagulant therapy.

Three months later, a radiological reassessment showed an improvement in the subdural hematoma. A CT abdominal scan showed a worsening of the porto-mesenteric thrombosis extension, following discontinuation of anticoagulant therapy (Figs. 3A and 3B), that could certainly compromise access to liver transplantation.

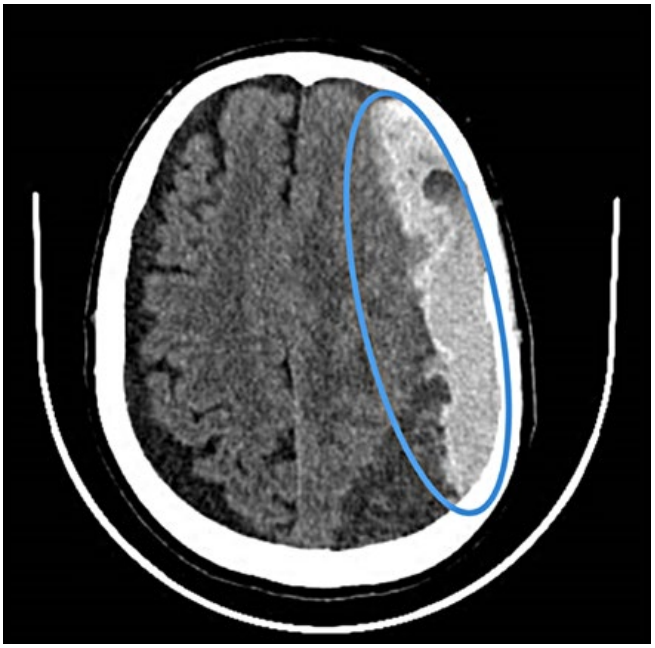


**Figure 2**: Cerebral CT scan showing an acute left subdural hematoma, causing mass effect on the adjacent sulci with a 7 mm midline shift (blue oval).

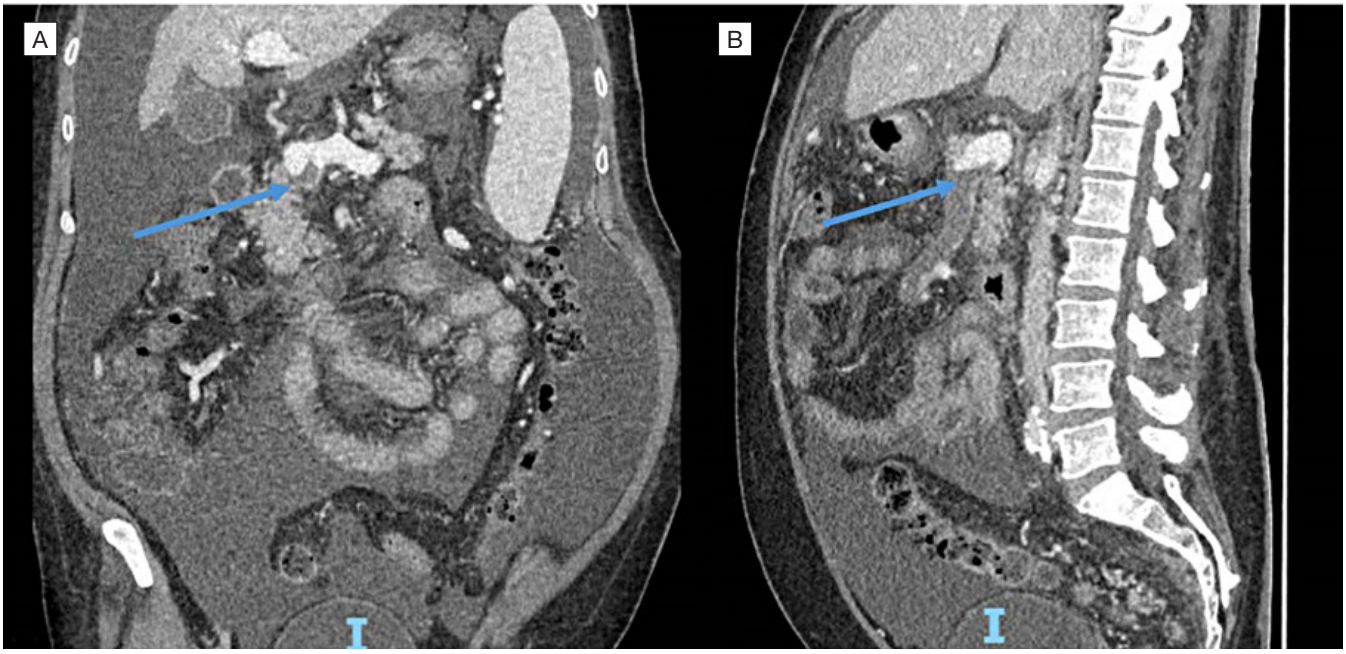
During this period, there was a progressively unfavourable evolution concerning the liver disease and its complications, with worsening of the refractory ascites, several episodes of exacerbation of hepatic encephalopathy and two other episodes of gastrointestinal bleeding due to oesophageal variceal rupture, reinforcing the need and urgency for liver transplantation. Transjugular intrahepatic portosystemic shunt (TIPS) could not be performed due to severe hepatic encephalopathy. Considering all the above and the risk-benefit balance, it was decided to reintroduce anticoagulation to assure the patient's transplant eligibility through improvement of the thrombosis.

Anticoagulation with low-molecular-weight heparin (enoxaparin) at a therapeutic dose of 1 mg/kg/day was administered for three months and then reduced to 4 000 IU/day, without haemorrhagic complications. The patient ultimately underwent liver transplantation eight months after the resumption of anticoagulation. Five years later, the patient presents a favourable evolution after liver transplant.

## Discussion

PVT is a rare entity in the general population (incidence of 2 to 4 per 100 000 inhabitants), but is common in patients with cirrhosis, with an estimated prevalence between 5% to 24% of patients with cirrhosis, particularly in decompensated cirrhosis (17%) and liver transplantation candidates (26%).[6] Regarding the origin of the hepatopathy, MASLD-associated cirrhosis is associated with an increased risk of PVT development.[6,7]

PVT can be classified according to the time of evolution (recent if presumed to be present for less than 6 months or chronic if present for more than 6 months and/or cavernous transformation) and to the percentage of occlusion (minimally occlusive if obstruction of less than 50% of the vessel lumen, partially occlusive if obstruction superior to 50% of the vessel lumen, completely occlusive if total occlusion and no persistent lumen and cavernous transformation if presence of porto-portal collaterals without original portal vein seen).




**Figures 3A and 3B**: Abdominal and pelvic CT scan demonstrating a marked increase in the extent of the superior mesenteric vein (SMV) thrombus, with more evident extension to the origin of the portal trunk and into the jejunal and ileal branches of the SMV (3 months after discontinuation of anticoagulation therapy).

The management of PVT can be challenging and complex, both in terms of its indications and its evolution under treatment, as well as potential complications. Importantly, partial PVT can spontaneously regress. Thus, anticoagulation therapy is recommended in:

- potential candidates for liver transplant, as anticoagulation will increase the feasibility and eligibility for liver transplant, but also reduce post-transplant outcomes;

and/or

- patients who are or not liver transplant candidates but have a minimally occlusive PVT that progresses in 3-6 months, PVT and hepatocellular carcinoma, symptomatic PVT or complete or partially occlusive PVT.

In all other patients, decisions for anticoagulation should be individualized based on the expected benefit-to-risk balance.[4,5]

The choice of anticoagulation depends on liver dysfunction and liver transplantation timing. Indeed, direct oral anticoagulants are contraindicated in child C cirrhosis, renal failure, and should be used with caution in child B cirrhosis. Similarly, it may be difficult to manage when a liver transplant is imminent. The development of bleeding complications is frequently a major concern in patients with liver cirrhosis, especially when it is complicated by portal hypertension, and more specifically in those presenting with gastroesophageal varices.

However, current evidence suggests that the risk of bleeding does not appear to be further increased by anticoagulant therapy. In fact, Valeriani et al showed that the risk of major bleeding was lower in patients receiving anticoagulation compared with those not receiving this therapy (6% *vs* 13%, respectively). Moreover, bleeding related to portal hypertension may be reduced by anticoagulant treatment for portal vein thrombosis (PVT). This effect may be explained by higher rates of portal vein recanalization, reduced thrombosis progression, and overall lower mortality compared with untreated patients. By improving portal vein recanalization, the pressure load on gastroesophageal varices may be reduced, and consequently the risk of gastroesophageal varices bleeding (70.7% of major bleeding in anticoagulated patients *versus* 75% in untreated patients).[8,9]

Conversely, not related to portal hypertension, cerebral bleeding occurs more frequently in patients undergoing anticoagulant therapy (10.3% *vs* 5%).[8] Risk factors for severe bleeding are: advanced age, severe thrombocytopenia (< 50x109/L), coagulopathy, systemic infection and renal impairment.[10]

Managing anticoagulation with a dedicated anticoagulation clinics may be helpful to reduce the risk of major bleeding as it has been shown to improve time in therapeutic range (TTR) in patients with coumarin derivatives, and to improve safety and quality of care in all patients treated with any anticoagulation.[11]

TIPS can also be alternative, when indicated for cirrhosis complications, or when recanalization is not obtained with anticoagulation, or in patients for whom anticoagulation is contraindicated. Unfortunately, severe HE limited TIPS possibility in that setting. The patient presented by the authors in this case report illustrates the dichotomy between the risks and benefits of anticoagulant therapy in patients with cirrhosis complicated by PVT. Shortly after the initiation of anticoagulation, a major haemorrhagic event occurred, with the development of a subdural hematoma, occurring within one week of treatment. On the other hand, it is noteworthy that the three episodes of gastrointestinal bleeding associated with portal hypertension, due to rupture of gastroesophageal varices, occurred outside the period of anticoagulation (the last of them after the PVT diagnosis).

Following the reintroduction of anticoagulant therapy —initially at therapeutic doses for three months and subsequently at prophylactic doses — no further intercurrent events were observed, particularly haemorrhagic complications, nor was there any progression of portal or mesenteric thrombosis. On the contrary, thrombosis regression was documented and permitted to perform liver transplantation.

This clinical course and challenging therapeutic decision-making ultimately allowed the patient to access liver transplantation, with a favourable outcome five years later. ■

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PAA, AP – Manuscript drafting, literature review, and critical revision

All authors approved the final version to be published.

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PAA, AP – Redação do manuscrito, revisão da literatura e revisão crítica

Todos os autores aprovaram a versão final a ser publicada

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