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Portal Vein Thrombosis a literature review of the clinical outcomes associated with different management strategies in patients with liver cirrhosis

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ABSTRACT

Portal vein thrombosis (PVT) is a frequent complication in patients with liver cirrhosis, often leading to a deterioration in patients' clinical condition. The treatment of PVT relies on anticoagulation therapy. The main key of therapy is to achieve recanalization of the portal vein and reduce thrombus progression, leading to a reduced risk of portal hypertension-related complications. Numerous anticoagulant drugs, such as VKAs, DOACs, and LMWH, have been investigated regarding their effectiveness and safety profiles. This article presents a deep analysis of the effectiveness of various treatments for PVT in cirrhotic patients.

Keywords: Portal Vein Thrombosis, Cirrhosis, Anticoagulation, Recanalization, Transjugular Intrahepatic Portosystemic Shunt

1. INTRODUCTION

Pathogenesis

PVT is a serious vascular condition characterized by the obstruction of the portal vein and is one of the most common thrombotic manifestations in patients with cirrhosis. (Dong et al., 2021) Around 10% of these patients will develop a PVT (Northup et al., 2021). Following that around 25-40% patients waiting for liver transplantation suffers from PVT (Chawla & Bodh, 2015). The management of PVT is determined by its underlying etiology, the extent of thrombotic involvement, and the distinction between acute and chronic presentation. Alcoholic cirrhosis or Metabolically Dysfunctional-Associated Steatotic Liver Disease (MASLD), in correlation with metabolic syndrome, leads to blood stasis in the portal vein due to increased vascular resistance and the diversion of blood from the portal system through collateral vessels, which, according to Virchow's triad, promotes thrombus formation.

Anatomy of the portal venous system

The portal vein system is a specific part of the circulatory system responsible for transporting nutrient-rich blood from the gastrointestinal tract to the liver. This mechanism is important for maintaining metabolic balance and helping the liver function properly. The portal vein is formed in the abdominal cavity by the union of two large veins: the superior mesenteric vein and the splenic vein. The superior mesenteric vein collects blood from the intestines, while the splenic vein collects blood from the spleen and parts of the stomach and pancreas. After joining, they form the portal vein, which ascends within the hepatoduodenal ligament, running next to the proper hepatic artery and the common bile duct, entering the liver at the porta hepatis (Parson, 2009). Inside the liver, the PV branches into right and left portal veins, supplying functional liver segments (Grace et al., 1998). Inside the liver, portal venous blood combines with oxygen-rich blood from the hepatic artery in the sinusoids- special capillary spaces lined with tiny openings in their walls. Mixed blood then drains into central veins, converging into hepatic veins, which empty into the inferior vena cava (IVC) (Parson, 2009).

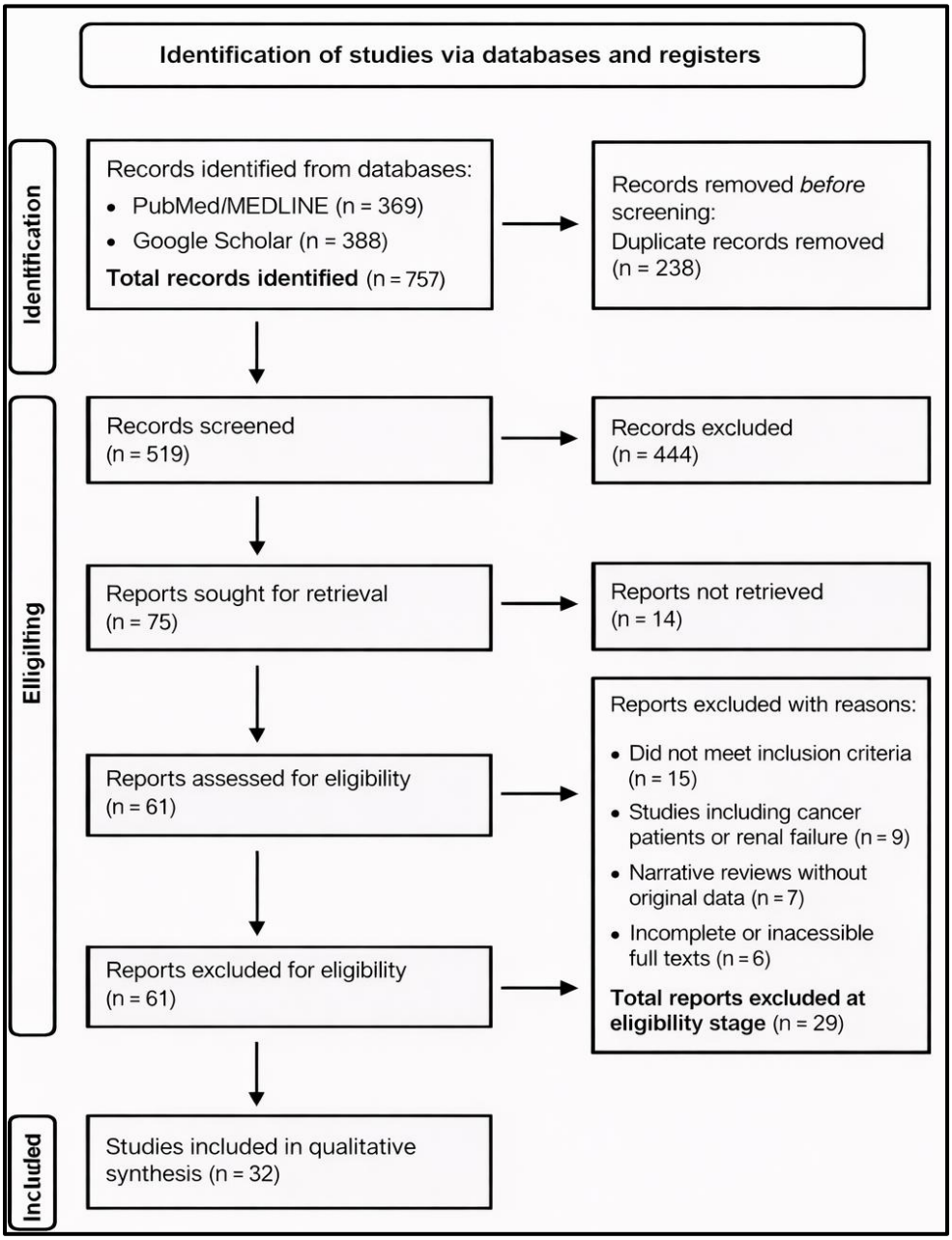

Figure 1 Flow chart

2. REVIEW METHODS

The article provides a review of the effects of treating PVT in patients with cirrhosis using low molecular weight heparins, vitamin K antagonists, and direct oral anticoagulants. We excluded studies describing patients with cancer or renal failure. A literature search was carried out in PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar employing combinations of terms referring to Portal Vein Thrombosis. We have found 32 articles from 1998 to 2024. The study selection process is summarized in Figure 1.

3. RESULTS & DISCUSSION

The study shows that PVT is an important complication of liver cirrhosis. Treatment involves anticoagulants to restore normal blood flow and prevent further enlargement of the thrombus. A TIPS procedure may be required to lower pressure in the portal system, and in rare cases, a liver transplant is needed. Among available treatments, LMWH are favored because they act faster and are safer than VKAs. Still, more studies are needed to confirm these results.

The principles of treatment

Anticoagulation remains the mainstay of treatment, mainly consisting of low molecular weight heparin (LMWH) and direct oral anticoagulants (DOACs) (Giri et al., 2023a). Based on the article 'Natural history of portal vein thrombosis in cirrhosis: A systematic review with meta-analysis', we can conclude that among patients who did not receive anticoagulant therapy, approximately 22% experienced progression of PVT. Around 29.3% of them achieved partial recanalization, and 10.4% of them achieved complete recanalization (Giri et al., 2023b). Patients who used anticoagulant therapy had significant benefits. Several studies show higher rates of partial or complete recanalization of the portal vein, ranging from 53% to 60% (Delgado et al., 2012; Pettinari et al., 2019).

A systematic review of 8 studies and 353 patients with liver cirrhosis and portal vein thrombosis (PVT) showed that complete portal vein unblocking was achieved in 72% of those receiving anticoagulation (LMWH or VKA), compared to 42% in the untreated group (Delgado et al., 2012). Additionally, analyses comparing patients with cirrhosis receiving anticoagulant therapy with those not receiving such treatment showed that the treated group had lower mortality and better improvement in liver function parameters. What is more interesting is that both groups had a similar number of bleeding events (Pettinari et al., 2019). The effectiveness of treatment is better when initiated within six months of PVT diagnosis, and both LMWH and VKA show comparable therapeutic efficacy (Rodriguez-Castro et al., 2019). It is worth mentioning that discontinuation of anticoagulation is highly related to PVT recurrence (Delgado et al., 2012).

In patients with early thrombosis involving small branches of the intrahepatic portal vein or when the clot narrows the vessel by less than 50%, the preferred treatment is follow-up with Doppler ultrasound every three months to monitor possible disease progression (Martens et al., 2022). Pharmacological anticoagulation should be initiated only when there is confirmed progression of the thrombus. On the other hand, in patients with partial (>50%) or complete occlusion of the portal vein and superior mesenteric vein, anticoagulant therapy should be initiated to prevent the clot from worsening (Amitrano & Guardascione, 2009). This treatment is continued for six months. If the patient is awaiting liver transplantation, it should be continued until the procedure is performed. In patients with liver cirrhosis and chronic deep vein thrombosis, anticoagulant therapy is usually reserved for specific indications, such as coexisting hereditary thrombophilia, progression of thrombosis, or current or previous intestinal ischemia (Yao et al., 2023).

Besides the drugs used, other factors influence the treatment process. Regression of PVT is less likely in patients with a high MELD score, ascites, or a higher Child–Pugh class (Wang et al., 2021). What is interesting is that the Factor V Leiden mutation or G20210A mutation significantly reduces the risk of occurrence and improves the prognosis of a patient with PVT and cirrhosis (Qi et al., 2014). The precise molecular mechanisms by which these mutations influence the risk of PVT in cirrhosis have not yet been fully elucidated.

Challenges in treatment

In patients with liver cirrhosis, low-molecular-weight heparins (LMWH) are considered the best anticoagulant agents. Clinically, they are advantageous because they act swiftly yet moderately on the coagulation cascade, and their effects can be reversed in the event of hemorrhage (Wang et al., 2016). Liver failure leads to decreased antithrombin production and, consequently, to a decrease in its concentration in the blood, while the activated partial thromboplastin time (APTT) is often prolonged in this population. Under these conditions, the use of unfractionated heparin (UFH) is not ideal because there is a risk of giving too low a dose, and it is hard to monitor its anticoagulant effect reliably.

Moreover, in patients with liver cirrhosis, the antithrombotic efficacy of direct oral factor Xa inhibitors, such as monoamine oxidase inhibitors (LMWH) or VKAs, may be lower than that of monoamine oxidase inhibitors (LMWH) or VKAs, while the risk of bleeding remains similar (Roberts et al., 2022). Direct oral anticoagulants (DOACs) should not be used by patients classified as Child-Pugh class C or advanced class B (≥ 8 points) (Tadokoro et al., 2024). If the patient is waiting for liver transplantation, dabigatran may be considered, assuming that idarucyzumab a specific reversal agent for bleeding, is available. Before initiating anticoagulant therapy, it is essential to exclude the presence of large esophageal varices. If one of these is reported, prophylactic measures, administration of a non-selective beta-blocker, or endoscopy should be considered to reduce the risk of bleeding (De Mattos et al., 2021).

The role of TIPS in supporting the treatment of PVT

For patients with chronic PVT complicated by recurrent hemorrhages or refractory ascites, the implantation of a transjugular intrahepatic portosystemic shunt (TIPS) with portal vein access is worth considering- yet TIPS placement may be challenging and potentially high-risk in cases of complete portal vein occlusion, particularly when cavernous transformation is absent (Hepatobiliary Disease Study Group, 2021).

A distinct and controversial issue is the role of prophylactic anticoagulation in cirrhotic patients with significant portal hypertension. Evidence from studies by Villa et al. suggests that anticoagulant therapy may not only prevent the development of PVT, but also slow the progression of liver disease and lower portal pressure (Villa et al., 2012), supporting the concept that microthrombi forming in small intrahepatic portal vessels plays a role in ongoing hepatic architectural distortion and disease advancement.

Recent studies indicate that DOACs provide similar effectiveness with fewer bleeding complications compared to traditional warfarin therapy. On the other hand several studies using DOACs showed complete recanalization rates between 0% and 82.4% (for example, 12.8% at 3 months and 28.2% at 6 months in one study, and 46.0% to 82.4% in others) (Ai et al., 2020). The main complication of treatment is bleeding, which has been addressed in multiple studies. DOACs generated reported major bleeding rates ranging from 0% to 17.6%, while traditional agents reached up to 30% in one cohort (Joseph & Rejeski, 2020). In one analysis, LMWH produced a significant bleeding rate of 7.2% compared to 9.3% for VKAs and 7.9% for DOACs (Mohan, 2020).

Based on these data, we can come to the conclusion that DOACs have been shown to promote recanalization and, in some cases, provide a better bleeding risk profile in comparison to VKAs and LMWH. The selection of the appropriate anticoagulant drug depends mainly on the individual characteristics of the patient, such as the stage of cirrhosis, risk of bleeding, and kidney function, which often constitute additional problems in people with liver disease (Ali et al., 2021).

The Role of Portal Vein Thrombosis in Liver Transplantation

Portal vein thrombosis (PVT) is no longer considered an absolute contraindication to liver transplantation, and its presence does not appear to significantly impact survival rates for patients awaiting transplantation. Furthermore, partial PVT has not been found to worsen early postoperative outcomes (Stine, 2015). Yet, published data indicate that the one-year mortality following transplantation is higher in patients with PVT (13.5%) compared to those without it (9.9%) (Englesbe et al., 2010). The risk of post-transplant complications is highest in patients requiring non-anatomical vascular reconstruction, especially in cases using autologous or synthetic interpositional grafts, which raises the risk because of the potential for thrombosis within the graft (De Franchis, 2015; Miñano & Garcia-Tsao, 2010).

Deep vein thrombosis before liver transplantation is a well-known risk factor for recurrent portal vein thrombosis after liver transplantation, which may occur in up to 40% of patients (Rodriguez-Castro et al., 2019). Moreover, PVT may develop postoperatively (even in individuals without preexisting thrombosis) especially, in those with obesity, diabetes mellitus, MASLD, or ascites. Although there is a risk of thrombosis, a systematic review found no clear benefit from the use of low molecular weight heparin (LMWH) after transplantation, whether at prophylactic or therapeutic doses, in patients at intermediate risk of thrombosis (Wang et al., 2018). Acetylsalicylic acid is used to prevent hepatic artery thrombosis after liver transplantation (Prakash et al., 2023). In patients whose portal vein blood flow drops below 15 cm/s, regular Doppler ultrasound examinations are recommended. This test allows for early detection of thrombosis and other blood flow disturbances (Shakeel & Perveen, 2025). The study summary is presented in Table 1.

Table 1. Key clinical conclusions regarding portal vein thrombosis management in liver cirrhosis

Area	Concise Conclusion
Anticoagulant choice	LMWH are preferred first-line agents due to predictable effect, reversibility, and acceptable bleeding risk
Role of DOACs	DOACs may offer comparable or improved recanalization with similar or lower bleeding risk in carefully selected patients
Bleeding risk management	Anticoagulation requires prior screening and prophylaxis of esophageal varices to minimize bleeding complications
Interventional therapy	TIPS may be considered in chronic or complicated PVT but carries higher procedural risk in complete portal vein occlusion
Individualized treatment	Anticoagulant selection should be tailored to cirrhosis severity, bleeding risk, renal function, and transplant status
Liver transplantation	PVT is not an absolute contraindication to transplantation, although it is associated with higher post-transplant mortality

4. CONCLUSION

Portal vein thrombosis (PVT) is a common condition in patients with liver cirrhosis and is a serious health problem. It can lead to poor blood circulation and serious complications. Treatment aims to improve blood flow in the portal vein and stop the clot from growing. For patients with cirrhosis and PVT, liver transplantation continues to be a key treatment option. Low-molecular-weight heparins (LMWH) and vitamin K antagonists (VKA) are effective, but LMWH are preferred because of rapid effect, good toleration, and they can be easily reversed in case of bleeding. It is also worth mentioning that treatment discontinuation will probably lead to disease recurrence.

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Informed consent

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Ethical approval

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Conflict of interest

The authors declare that they have no conflicts of interest, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data and materials availability

All data associated with this study will be available based on reasonable request to the Corresponding Author.

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