

Portal vein thrombosis in liver cirrhosis

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Abstract

Portal vein thrombosis (PVT) is considered to be a frequent complication of liver cirrhosis. However, unlike PVT in patients without cirrhosis, very few data are available on the natural history and management of PVT in cirrhosis, despite its association with potentially life-threatening conditions, such as gastroesophageal bleeding and acute intestinal ischemia. Moreover, no consensus regarding PVT in cirrhosis exists. Suggested causes of PVT in cirrhosis include reduced portal blood flow velocity, multiple congenital or acquired thrombophilic factors, inherited or acquired conditions, and derangement of liver architecture. However, the understanding of PVT in cirrhosis is incomplete. In addition, information on the management of PVT in cirrhosis is inadequate. The aims of this review are to: (1) assemble data on the physiopathological mechanism, clinical findings, diagnosis and management of PVT in cirrho-

sis; (2) describe the principal factors most frequently involved in PVT development; and (3) summarize the recent knowledge concerning diagnostic and therapeutic procedures.

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Key words: Portal vein thrombosis; Liver cirrhosis; Thrombophilic factors; Anticoagulation; Splenectomy

Core tip: Portal vein thrombosis (PVT) is considered to be a frequent complication of liver cirrhosis; however, very few data are available on the natural history and management of PVT in cirrhosis, despite its association with potentially life-threatening conditions. The understanding and information on the management of PVT in cirrhosis are incomplete. The aims of this review are to: (1) assemble data on the physiopathological mechanism, clinical findings, diagnosis and management of PVT in cirrhosis; (2) describe the principal factors most frequently involved in PVT development; and (3) summarize the recent knowledge concerning diagnostic and therapeutic procedures.

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INTRODUCTION

Portal vein thrombosis (PVT), an obstruction of the portal vein or its branches by a blood clot, is encountered in a variety of clinical settings, such as myeloproliferative disease, cirrhosis, cancer and infection. More patients with cirrhosis are being diagnosed with PVT be-

cause current imaging techniques allow for the detection of asymptomatic PVT during routine ultrasonographic examination. PVT has a variety of clinical presentations, from asymptomatic to life-threatening conditions such as gastroesophageal bleeding and acute intestinal ischemia^[1-3]. Although liver transplantation has altered the prognosis of patients with cirrhosis, the presence of PVT can exclude a patient from a transplant listing or negatively impact post-transplantation survival^[4].

It remains unclear whether PVT is a consequence of severe liver disease, a factor aggravating underlying liver disease, or both. PVT is considered to be a frequent complication of liver cirrhosis; however, unlike PVT in patients without cirrhosis, very few data are available on its natural course and management despite its association with potentially life-threatening conditions. In addition, no consensus regarding PVT with cirrhosis exists. There is a growing need for optimal, evidence-based management of PVT in cirrhosis.

The aims of this review are to: (1) assemble evidence regarding the physiopathological mechanism, clinical findings, diagnosis and management of PVT in cirrhosis; (2) describe the principal factors most frequently involved in PVT development; and (3) summarize the most recent knowledge concerning diagnostic and therapeutic procedures.

DEFINITION OF ACUTE AND CHRONIC PVT

From a clinical point of view, PVT comprises two different entities: acute PVT and chronic PVT. Each represents a successive stage of the same disease. Although they share similar causes, they differ with respect to their management^[5].

Acute PVT is characterized by the sudden formation of a thrombus within the portal vein^[6]. The thrombus can involve variable portions of the mesenteric veins and/or splenic vein. Occlusion can be complete or partial, leaving a peripheral circulating channel.

In patients with chronic PVT, also known as portal cavernoma, the obstructed portal vein is replaced by a network of hepatopetal collateral veins connecting the patent portion of the vein upstream of the thrombus to the patent portion downstream. The number, size and location of these collaterals are extremely variable from patient to patient^[6].

However, in patients with cirrhosis, it may be difficult to establish the “age” of the thrombosis because the criteria commonly used in patients without cirrhosis to define acute or chronic PVT (presence of collateral circulation and signs of portal hypertension) are already features of liver disease^[7,8].

PREVALENCE

The prevalence of PVT in patients with cirrhosis has been reported more frequently in recent years. The re-

ported prevalence of PVT is in the range of 0.6%-15.8% in patients with liver cirrhosis or portal hypertension^[9-15]. The presence of PVT is reportedly 0.6% when evaluated by angiographic studies^[9], 4.4% when evaluated by ultrasound^[10], and 10%-12% when evaluated by computed tomography and magnetic resonance imaging^[11]. Moreover, the prevalence of PVT increases with patient age and liver disease severity, reaching 15% in patients awaiting liver transplantation^[12-15].

The etiology of liver disease influenced the prevalence of PVT in a study of 885 patients who underwent liver transplantation. The prevalence of PVT was 3.6% in primary sclerosing cholangitis, 8% in primary biliary cirrhosis, 16% in alcoholic and hepatitis B virus-related cirrhosis, and 35% in hepatocellular carcinoma (HCC)^[12].

ETIOLOGY OF PVT IN CIRRHOSIS

Inherited and acquired thrombophilic disorders, bacterial infection^[16,17] and sluggish portal flow^[2,18,19] may all play a role in the high prevalence of PVT in patients with cirrhosis.

Cirrhosis was recently considered to be a hypercoagulable state, not a hypocoagulable state. The levels of both pro- and anti-coagulation proteins are reduced under conditions of hepatic synthetic impairment in patients with liver cirrhosis. Coagulation and anticoagulation mechanisms remain balanced but are carried out at a lower level^[20-22]. The net result is a hemostatic balance that is compensated under normal circumstances, with no tendency for bleeding or thrombosis^[22]. In cirrhosis, however, this equilibrium can easily tilt towards either bleeding or thrombosis^[19,23-26]. Some authors have demonstrated that elevated levels of factor VIII (a procoagulant driver) in combination with decreased levels of protein C (an anticoagulant driver), both of which are typically found in patients with cirrhosis (*i.e.*, procoagulant imbalance), are probably related to partial resistance to the *in vitro* anticoagulant action of thrombomodulin^[27-30]. However, in patients with impaired synthetic function and low plasma levels of natural coagulation inhibitors, there is currently no simple way to ascribe such a low level to a pre-existing deficiency^[31].

A thrombophilic genotype, including factor V Leiden G1691A mutation^[32,33], methylenetetrahydrofolate reductase (TT677) mutation^[34,35] and prothrombin (G20210A)^[11,36], is associated with the formation of PVT. However, they may play a minor pathogenic role in the formation of PVT.

Reduced portal flow velocity seems to be the most important predictive variable for PVT development in patients with cirrhosis^[37-39]. Amitrano *et al.*^[38] suggested that portal blood stasis in patients with cirrhosis is the main change favoring thrombosis, even in the presence of other local, systemic, congenital and acquired factors. Kinjo *et al.*^[39] performed Doppler ultrasonographic examinations after splenectomy in patients with cirrhosis and showed that portal venous flow was dramatically decreased by 49.2% in the PVT group but only by 6.6% in

the non-PVT group.

Splenectomy has recently been reported to play a role in the surgical strategy for HCC and interferon-based therapy for hepatitis C^[40-45]. In addition, it can improve the prognosis for patients with cirrhosis by allowing them to receive interferon therapy or undergo treatment for HCC^[44,45]. Despite the good results demonstrated in these studies, the high prevalence of PVT after splenectomy in patients with cirrhosis remains problematic^[39,46]. It has been suggested that blood turbulence or stasis in the stump of the splenic vein after splenectomy might result in increased coagulability, leading to the propagation of splenic venous thrombus formation in the portal system after splenectomy^[47]. Splenomegaly and a large splenic vein diameter are independent risk factors for PVT after splenectomy in patients with concomitant cirrhosis and portal hypertension^[39,46,47].

The role of sclerotherapy as a potential trigger for PVT is controversial^[48,49]. Some recent reports showed that thrombopoietin receptor agonists might be associated with an increased incidence of PVT in patients with cirrhosis^[50,51].

CLINICAL FINDINGS

Clinical findings of PVT in cirrhosis vary from asymptomatic to life-threatening conditions. Partial PVT, which is now often detected by routine ultrasonography or computed tomography, might be associated with few symptoms. However, complete PVT may present as abdominal or lumbar pain with sudden onset or progression over a few days. Rapid, complete obstruction of the portal vein or mesenteric veins without involvement of the mesenteric venous arches induces intestinal congestion, which manifests as severe, continuous, colicky abdominal pain and occasionally as nonbloody diarrhea^[1-3]. The bleeding risk appears to be higher in patients with PVT and cirrhosis than in patients with cirrhosis alone (39% *vs* 27%, respectively)^[52]. In many patients, however, the thrombus is partial and its aspects and location change in follow-up images. Laboratory findings, including the levels of aminotransaminase, fibrin and fibrinogen degradation products, and d-dimers, are often normal in many cases of developing PVT.

Chronic PVT is commonly diagnosed after a fortuitous finding of hypersplenism or portal hypertension. In the majority of patients it is asymptomatic. Gastrointestinal bleeding is better tolerated by patients with chronic PVT than in those with other forms of portal hypertension, probably because patients with PVT are usually younger and have no liver dysfunction. The occurrence of ascites or encephalopathy in patients with chronic PVT is uncommon and is usually encountered only transiently following gastrointestinal bleeding or when unrelated renal failure or marked sepsis is present in older patients^[5]. Liver test results are typically normal in patients with portal cavernoma in the absence of underlying liver disease. Biliary symptoms related to portal

cholangiopathy (jaundice, biliary pain, cholangitis, cholecystitis or pancreatitis) rarely reveal the presence of a cavernoma^[53,54]. Hepatopulmonary syndrome is present in about 10% of patients.

DIAGNOSIS

More of these patients are being diagnosed with PVT because current imaging techniques allow for the detection of asymptomatic PVT during routine ultrasonography in patients with cirrhosis.

Ultrasound and Doppler ultrasound are almost always sufficient for a diagnosis of PVT^[55,56]. In most patients, the diagnosis of acute PVT can be rapidly established using noninvasive imaging. Ultrasound sonography can show hyperechoic material in the vessel lumen with distension of the portal vein and its tributaries. Doppler imaging shows the absence of flow in part or all of the lumen.

Enhanced computed tomography (CT) can show a lack of luminal enhancement in the portal vein, increased hepatic enhancement in the arterial phase, and decreased hepatic enhancement in the portal phase^[57]. CT and magnetic resonance (MR) angiography are more sensitive techniques than Doppler imaging with respect to assessment of the extent of the thrombus within the portal venous system^[56-58]. Definitive diagnosis of PVT can be obtained by MR imaging (MRI) and CT; the former provides a better evaluation of the extent of the thrombosis, particularly in the mesenteric vein, reaching a sensitivity and specificity of 98%-100%. CT provides information not only about the extent of the thrombosis and the development of collateral circulation, but also about the state of the abdominal organs. It is the procedure of choice when intestinal ischemia or hepatocellular carcinoma is suspected^[59,60]. A diagnosis of cavernoma is readily achieved by abdominal imaging with ultrasound, CT or MRI, which shows serpiginous structures while the main portal vein and/or its main branches are not visible.

A recent study showed that positive intrathrombus enhancement on contrast-enhanced sonograms is an accurate predictor of recanalization in patients with recent portal thrombosis^[61].

TREATMENT

Optimal management of PVT in cirrhosis is not addressed in any current consensus publication. There are a few reports about the factors that influence recanalization or the extent of thrombosis; however, the actual impact of PVT treatment on the natural course of cirrhosis has not been investigated. No randomized controlled trials have been performed and most existing evidence concerning PVT treatment is based on case series and is of low quality.

PVT increases the risk of variceal bleeding and is reportedly an independent risk factor for the inability to

control variceal bleeding^[62]. In addition, PVT can be a life-threatening emergency when it extends to the superior mesenteric vein, leading to intestinal infarction. Anticoagulated patients with cirrhosis have better recanalization rates and PVT extension than non-anticoagulated patients^[63]. Therefore, in patients with concomitant cirrhosis and PVT, a treatment algorithm that includes anticoagulation and transjugular intrahepatic portosystemic shunting (TIPS) provides a good chance of complete repermeation, reduces portal hypertensive complications and decreases the rate of thrombosis progression^[63]. Francoz *et al*^[4] evaluated patients with cirrhosis awaiting liver transplantation and found that survival was significantly lower in those with complete PVT at the time of surgery ($P = 0.04$). Furthermore, the rate of partial or complete recanalization was significantly higher among patients receiving anticoagulation therapy than among those not receiving anticoagulation therapy ($P = 0.002$).

Conversely, some reports have shown that PVT has little influence on prognosis in patients with cirrhosis. Maruyama *et al*^[64] evaluated 150 patients with virus-related cirrhosis but without PVT at baseline; PVT developed in 28% of patients (42/150), with a cumulative incidence of 12.8%, 20% and 38.7% at 1, 5 and 8-10 years, respectively. The natural course of thrombosis was improvement in 47.6% of patients, unchanged in 45.2%, and worsened in 7.2%. Spontaneous resolution or an unchanged appearance was the most common outcome of PVT; therefore, cirrhotic PVT had little influence on prognosis. In their multivariate analysis, Luca *et al*^[65] noted that there was no clear association between progression or regression of partial PVT and clinical outcome and that the Child-Pugh score at the time of diagnosis was the only independent predictor of survival.

In the field of liver transplantation, there is accumulating evidence that PVT, especially thrombus extension to the superior mesenteric vein, may adversely affect the outcome of transplantation. Thus, patients with concomitant cirrhosis and PVT who are on the waiting list for liver transplantation should be treated with anticoagulation therapy^[66,67]. PVT prior to liver transplantation is an independent prognostic factor for post-transplant survival^[68,69] and complete or partial PV recanalization has been associated with a better survival rate after liver transplantation^[4]. It has also been shown that individuals with PVT at the time of liver transplantation are at higher risk of recurrent PVT after transplantation and of requiring retransplantation^[30,70]. The increased mortality and morbidity rates associated with PVT are mostly restricted to the first year after liver transplantation^[4,62] and actuarial survival after 1 year is good. Therefore, PVT cannot be considered to be a contraindication to liver transplantation^[71].

Anticoagulation therapy is of proven benefit in patients with acute deep vein thrombosis^[72]. The optimal anticoagulation regimen for the treatment and monitoring of PVT has not yet been fully explored and no clear recommendations exist regarding this issue in recent

guidelines or consensus publications^[6,28]. Treatment strategies most often include the use of anticoagulation, while thrombectomy and TIPS are considered second-line options.

The goal of anticoagulation therapy for acute PVT is to recanalize the obstructed veins, which will prevent intestinal infarction and portal hypertension. Correction of the causal factors should be achieved as soon as possible.

Vitamin K antagonists (VKA) have been used in some studies to treat PVT in patients with cirrhosis. The rate of PV recanalization in patients with cirrhosis treated with VKA is about 40%^[4,73]. Orally administered VKA is more acceptable to patients; however, treatment with VKA is particularly difficult in patients with cirrhosis, mostly because anticoagulation monitoring is complex in this particular situation. Notably, international normalized ratio (INR) monitoring in patients with liver disease probably overestimates the bleeding risk because this international sensitivity index is determined using plasma from patients taking VKA^[74]. The INR has only been validated in individuals with normal liver function on stable anticoagulation. A 29% variation in the mean INR was reported in patients with cirrhosis in a study in which three different thromboplastin reagents were used^[75]. It is also unclear whether a target INR between 2 and 3 is adequate in individuals with an abnormal INR before anticoagulation therapy^[30].

No consensus exists regarding the optimal duration of anticoagulation therapy in these settings. Complete recanalization can be delayed until the sixth month of anticoagulation therapy^[5,76]. However, whether this is also true for patients with cirrhosis who develop acute PVT remains to be determined.

Randomized controlled trials of anticoagulation therapy for the prevention of recurrent thrombosis are lacking in cirrhotic PVT. In patients with deep vein thrombosis, a lack of complete recanalization indicates a high risk of recurrence after cessation of anticoagulation therapy^[77]. The frequent association with permanent prothrombotic disorders and the risk of intestinal infarction support the use of anticoagulation. However, an increased risk of bleeding secondary to portal hypertension raises concerns. Delgado *et al*^[75] reported that re-thrombosis after complete recanalization occurred in 38.5% of patients with cirrhosis after anticoagulation therapy was stopped. Thatipelli *et al*^[78] stated that prolonged anticoagulant therapy does not appear to be justified based on the low rate of recurrence and substantial rate of major hemorrhage. To avoid the extension of thrombosis to the splanchnic vessels, prophylactic anticoagulation should be continued in patients with underlying thrombophilic conditions or in patients who are likely candidates for future liver transplantation^[4,63].

The choice of the anticoagulation regimen must also account for the potential need to reverse the effect of anticoagulation. There is no current consensus or guideline on whether nonselective beta blockers, endoscopic

variceal ligation or combination therapy is better for variceal bleeding prophylaxis^[7,79].

An attractive alternative to oral anticoagulants could be the use of low-molecular-weight heparin (LMWH), the dosing of which is weight based and thus does not necessitate screening. A 50%-80% portal vein recanalization rate was recently reported with the use of LMWH in 38 patients with cirrhosis, with only a few episodes of non-severe variceal bleeding^[62,80]. Villa *et al*^[81] performed a randomized controlled trial to evaluate the safety and efficacy of enoxaparin, a LMWH, in preventing PVT in patients with advanced cirrhosis. They demonstrated that the actuarial probability of PVT was lower in the enoxaparin group ($P = 0.006$). The actuarial probability of survival was higher in the enoxaparin group ($P = 0.020$). No relevant side effects or hemorrhagic events were observed in their study. Enoxaparin appeared to delay the occurrence of hepatic decompensation and improve survival. However, an increased volume of distribution, such as that produced by ascites and edema, in patients with cirrhosis makes it difficult to determine the optimal dose of LMWH^[82].

The administration of antithrombin III (AT-III) could be an attractive alternative to PVT in cirrhosis. Kawanaka *et al*^[83] demonstrated that the low AT-III activity and further decreases in this activity are associated with PVT after splenectomy in patients with cirrhosis and that treatment with AT-III concentrates is likely to prevent the development of PVT in these patients.

TIPS and anticoagulation therapy are considered to be optimal treatment choices for PVT in cirrhosis^[84]. TIPS completely recanalized the portal venous system in 57% of patients with cirrhosis and resulted in a marked decrease in 30% without major procedure-related complications. Despite problems associated with patency (bare stents, 38% in 12 mo and 85% in 24 mo; covered stents, 21% in 12 mo and 29% in 24 mo) and encephalopathy (27% at 12 mo, 32% at 24 mo), the long-term outcome of TIPS placement for cirrhotic PVT is excellent^[85]. In addition, PVT prior to liver transplantation is an independent prognostic factor for post-transplant survival and TIPS prevents total portal vein occlusion in liver transplantation candidates with partial PVT^[86].

In patients with both cirrhosis and chronic PVT, there is no consensus on the indication for anticoagulant therapy. As described in the consensus for PVT in patients without cirrhosis, therapy for chronic PVT can be separated into prevention and treatment of gastrointestinal bleeding, prevention of recurrent thrombosis and treatment of portal cholangiopathy. When PVT is longstanding and cavernous transformation has occurred in the portal vein, prophylactic anticoagulation is reversed only in patients with thrombophilic conditions and/or a high risk of thrombus extension into the superior mesenteric vein. There is still sufficient evidence in favor of interventional therapy such as TIPS^[87]. Data on endoscopic ligation are lacking in adult patients with chronic PVT.

CONCLUSION

PVT is a common problem in patients with cirrhosis, mostly in individuals with advanced liver disease. However, many unknown pathophysiological aspects of PVT and unresolved issues encountered in everyday practice remain to be addressed. The most optimal, most efficient and safest modalities for treatment, screening and monitoring must be established in future controlled trials.

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