DOI: 10.20514/2226-6704-2024-14-3-165-172
УДК 616.36-004:616.36-005.6
EDN: AWPZXZ

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ТРОМБОЗ ВОРОТНОЙ ВЕНЫ ПРИ ЦИРРОЗЕ ПЕЧЕНИ. ЧАСТЬ 1: ЭПИДЕМИОЛОГИЯ, ПАТОГЕНЕЗ, КЛИНИКА, ДИАГНОСТИКА, ВЛИЯНИЕ НА ПРОГНОЗ

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Portal Vein Thrombosis in Liver Cirrhosis. Part 1: Epidemiology, Pathogenesis, Clinic, Diagnosis, Impact on Prognosis

Резюме

Тромбоз воротной вены является самым распространенным тромботическим осложнением у больных циррозом печени, особенно в случаях тяжелых форм. Патогенез носит мультифакторный характер, определяется изменением равновесия между системами свертывания и противосвертывания. Тромбоз часто протекает бессимптомно, обнаруживается случайно, хотя может осложняться варикозным кровотечением, ишемией кишечника, портальной билиопатией. Ультразвуковое допплеровское исследование является методом скрининга, в качестве альтернативы используются компьютерная томография, магнитно-резонансная томография. В обзоре освещены данные об эпидемиологии, факторах риска, клинических особенностях, диагностике тромбоза воротной вены у пациентов с циррозом печени. Приведены данные о влиянии тромбоза воротной вены на прогрессирование цирроза, выживаемость больных, в том числе после трансплантации печени.

Ключевые слова: *тромбоз воротной вены, цирроз печени, эпидемиология, факторы риска, диагностика, прогноз*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

Источники финансирования

Авторы заявляют об отсутствии финансирования при проведении исследования

Статья получена 22.03.2024 г.
Принята к публикации 24.04.2024 г.

Для цитирования: Ягода А. В., Корой П. В., Байсаева Л. С. и др. ТРОМБОЗ ВОРОТНОЙ ВЕНЫ ПРИ ЦИРРОЗЕ ПЕЧЕНИ. ЧАСТЬ 1: ЭПИДЕМИОЛОГИЯ, ПАТОГЕНЕЗ, КЛИНИКА, ДИАГНОСТИКА, ВЛИЯНИЕ НА ПРОГНОЗ. Архивъ внутренней медицины. 2024; 14(3): 165-172. DOI: 10.20514/2226-6704-2024-14-3-165-172. EDN: AWPZXZ

Abstract

Portal vein thrombosis is the most common thrombotic complication in patients with liver cirrhosis, especially in cases of severe forms. The pathogenesis is multifactorial in nature, it determined by a change in the balance between the coagulation and anticoagulation systems. Thrombosis is often asymptomatic and is accidentally detected, although it can be complicated by varicose bleeding, intestinal ischemia, and portal biliopathy. Ultrasound Doppler examination is a screening method, as an alternative, computed tomography and magnetic resonance imaging are used. The review highlights data on epidemiology, risk factors, clinical features, and diagnosis of portal vein thrombosis in patients with liver cirrhosis. The data on the effect of portal vein thrombosis on the progression of liver cirrhosis and the survival of patients, including after liver transplantation, are presented.

Key words: *portal vein thrombosis, liver cirrhosis, epidemiology, risk factors, diagnosis, prognosis*

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Conflict of interests
The authors declare no conflict of interests

Sources of funding
The authors declare no funding for this study

Article received on 22.03.2024
Accepted for publication on 24.04.2024

For citation: Yagoda A. V., Koroy P. V., Baisaeva L. S. et al. Portal Vein Thrombosis in Liver Cirrhosis. Part 1: Epidemiology, Pathogenesis, Clinic, Diag-nosis, Impact on Prognosis. The Russian Archives of Internal Medicine. 2024; 14(3): 165-172. DOI: 10.20514/2226-6704-2024-14-3-165-172. EDN: AWPZXZ

CI — confidence interval; CT — computer tomography; MRI — magnetic resonance imaging; RR — risk ratio; OR — odds ratio; PVT — portal vein thrombosis

Portal vein thrombosis (PVT) is a condition, where a blood clot is formed in the portal vein. It can be secondary to a malignancy (tumour invasion of the vein) or can have non-neoplastic nature. Non-neoplastic thrombosis develops in the portal vein trunk and branches and involves splenic or superior mesenteric veins. In the absence of repatency, vein lumen is obliterated, and portoportal collaterals and portal cavernoma are formed. Hepatic cirrhosis (HC) or its absence in non-neoplastic PVT is essential, since aetiology, clinical manifestations and therapy in these patient populations differ [1-4].

PVT epidemiology in hepatic cirrhosis

PVT incidence in this population is not more than 1.1 %; it affects equally men and women [5]. The main causes of portal vein thrombosis are secondary hepatobiliary tumours (44 %), hepatic cirrhosis (28 %), primary hepatobiliary tumour (23 %), abdominal infections or inflammations (10 %), myeloproliferative disorders (3 %) [5].

In hepatic cirrhosis, PVT is the most common thrombotic complication; its incidence varies from 0.6 to 26 % [6-8], with the mean value of 13.92 % [9]. In 1998–2014, the incidence of PVT in cirrhosis patients increased from 0.7 to 2.4 % (annual rate of increase: 9 %), while associated mortality dropped from 11.9 to 9.1 % (annual rate of reduction: 3.0 %) [10].

One-year morbidity in patients with hepatic cirrhosis is 1.6–4.8 % [9, 11-15], three-year mortality — 7.6–9.3 % [9, 11], five-year mortality — 10.7 % [12]. Higher one-year mortality was reported as well: up to 16 % [16].

It is suggested that PVT mortality correlates with hepatic cirrhosis severity; it does not exceed 1–4 % in compensated disease and varies from 7.4 to 16 % in severe cases [6, 17]. In a meta-analysis, PVT incidence was 9.9 %, with Child-Pugh class A, and 18.3 % in patients with Child-Pugh class B and C; higher values were recorded in patients with hepatocellular carcinoma (up to 40 %) [18]. Overall, cirrhosis is associated with 8-fold increase in the risk of PVT in this population, with the odds ration (OR) of 7.9 [5]. Mortality depends on disease aetiology: very often, PVT is associated with non-alcoholic fatty liver disease [19].

PVT pathophysiology in hepatic cirrhosis

In acute PVT, a blood clot completely or partially blocks the portal vein. Acute obstruction of the superior mesenteric vein and mesenteric arcs results in ischaemia and bowel infarction, which are rare in hepatic cirrhosis due to slow development and progression of thrombosis, thus, an alternative venous drainage is formed. An acute complete block of the portal vein causes compensatory vasodilation of hepatic arteries ("arterial rescue"), which stabilises hepatic function [17].

Arterial vasodilation is followed by an arterial rescue phase, where venous portoportal collaterals are formed, which bypass an occluded segment, and, in 3–5 weeks, a cavernoma is formed, which is a distinguishing feature of chronic thrombosis [17, 20].

PVT risk factors in hepatic cirrhosis

PVT pathogenesis in hepatic cirrhosis is multifactorial; it is caused by imbalance between coagulation and anticoagulant systems [2]. A precise contribution of the Virkhov's triad to PVT in cirrhosis (slower blood flow, hypercoagulation and endothelial damage) is still unclear [21].

Venostasis

Hepatic cirrhosis is associated with increased intrahepatic vascular resistance and reduced portal blood flow, which is a predictor of PVT [22]. A high risk of PVT in cirrhosis is associated with a reduced blood flow velocity in the portal vein, the threshold values (15 cm/s) of which were predictors of a high risk of thrombosis [9, 14, 22, 23]. Risk factors of PVT were a larger portal vein diameter, spleen longitudinal axis and presence of large portocollateral vessels [6, 16, 24].

Non-selective b-adrenoceptor blocking agents, which are prescribed in hepatic cirrhosis, presumably increase the risk of PVT by reducing the portal blood flow and its velocity [25]. According to meta-analyses, the risk of thrombosis is higher in patients treated with non-selective b-adrenoceptor blocking agents (OR 4.62, 95 % confidence interval (CI) 2.50–8.53; $p < 0.00001$) [9, 25]. In other studies, there was no correlation between their

use and PVT, probably due to a favourable effect of non-selective b-adrenoceptor blocking agents on bacteriaemia and endotoxemia [11, 12, 14].

Thrombophilia

Despite thrombocytopenia and low pro- and anticoagulant proteins, patients with hepatic cirrhosis maintain haemostatic balance without susceptibility to haemorrhaging and blood clots due to platelet hyperactivity and higher levels of von Willebrand factor [26, 27]. However, this delicate balance can easily tilt towards pro-haemorrhagic or pro-thrombotic phenotypes.

Hypercoagulation, which includes imbalance in von Willebrand factor/ADAMTS13 and factor VIII/protein C, as well as platelet hyperactivity, stronger ability to generate thrombin and impaired clot lysis [28-30], increase the risk of thrombotic complications in patients with hepatic cirrhosis. Population studies demonstrated that the risk of thrombotic/thromboembolic complications in cirrhosis is at least the same as in the general population [26]. In hospitalised patients with hepatic cirrhosis, the incidence of venous thromboembolism was 1.2–7 % [31], whereas subjects without hepatic pathologies or with mild/moderate pathologies had venous thromboembolism in 2.7, 2.4 and 0.9 per 100 discharged patients, respectively [32]. According to other sources, the risk of venous thromboembolism in cirrhosis was 1.7 times higher vs. patients without cirrhosis [33]; thromboembolic events were recorded in 561.1 and 249.7 per 10,000 person-years, respectively [34]. The 10-year risk of venous thromboembolism in hepatic cirrhosis was higher (2.5 %) than in controls (1.7 %) [35].

These correlations between PVT and factor VIII/protein C and factor II/protein C, as well as proteins C, S, antitrombin III, plasminogen activator inhibitor-1, thrombomodulin resistance, clot lysis markers in systemic and portal blood flow in hepatic cirrhosis, are controversial [14, 36-38]. It is assumed that, in hepatic cirrhosis, platelet hyperfunction, the ADAMTS-13/von Willebrand factor ratio and blood D-dimers are related to PVT [9, 39-41]. According to available information, in hepatic cirrhosis, PVT was associated with mean platelet volume of over 8.9 fL (OR 5.38; 95 % CI 1.95–14.84) [42].

The role of hereditary thrombophilia in PVT is being studied [12, 43, 44]. It is believed that, in hepatic cirrhosis, PVT is not associated with mutations in prothrombin and factor V genes [12]. According to other sources, genetic thrombophilic defects can contribute to PVT pathogenesis [45]. For instance, mutations in genes of factor V Leiden *G1691A*, prothrombin *G20210A*, plasminogen activator inhibitor-1 *4G-4G* and methylenetetrahydrofolate reductase*C677T* due to cirrhosis were more common in patients with PVT vs. subjects without thrombosis [43, 45-47]. Presence of the negative allele of prothrombin gene *G20210A* in patients with PVT was associated with higher factor II values, factor II/D-dimer and factor II/protein C ratios [46]. At the same time, there are no clear guidelines on the need for thrombophilia tests in patients with PVT, although sometimes it is advisable to screen for genetic thrombophilia in patients with hepatic cirrhosis and PVT [1-4].

Overall, the incidence of thrombophilic irregularities (protein S and antitrombin III deficit, mutations of prothrombin gene*G20210A*, factor V Leiden*G1691A*, anticardiolipin antibodies, etc.) in patients with PVT and cirrhosis can reach 5–16 % [48, 49].

Endothelial damage

The role of endothelial dysfunction in PVT is studied inadequately. Endothelial damage from prior abdominal surgery, splenectomy, portal-systemic bypass surgery can be a risk factor, although the resulting changes in portal blood flow can contribute to blood clotting as well [18, 21].

Endotoxemia

Hepatic pathologies are often associated with bacterial translocation and endotoxemia, resulting from damaged intestinal barrier. Bacterial infection can increase portal pressure, while endotoxemia can activate a coagulation cascade in the portal vein [50]. Use of enoxaparin in hepatic cirrhosis inhibited bacterial translocation due to better intestinal microcirculation and reduction in enterocyte damage, and was associated with lower incidence of PVT [51].

Hepatic cirrhosis severity

Usually, PVT development is associated with severe hepatic damage (Child-Pugh class 3), thrombocytopenia, complications of portal hypertension and prior therapy (endoscopic sclerotherapy of varicose veins, splenectomy, bypass surgery), hepatocellular carcinoma [8, 9, 11, 16]. A high risk of PVT was associated with the degree of oesophageal varices ($p = 0.01$), prothrombin time ($p = 0.002$) [12], low platelets count (77.4 vs. 111.6×10^9/L; $p = 0.001$), a history of ascites (78.9 % vs. 59.2 %, $p = 0.009$), Child-Pugh class ($p = 0.04$), a history of oesophageal haemorrhaging (47.4 % vs. 29.1 %; $p = 0.003$), duration of waiting (8.5 vs. 4.8 months; $p = 0.002$) [52]. According to the available information, in cirrhosis, PTV was associated with Child-Pugh score of over 9 (OR 3.99; 95 % CI 1.59–9.98) and platelet count of less than 56×10^9/L (OR 7.67; 95 % CI 2.33–25.26) [42]. A meta-analysis demonstrated that Child-Pugh classes B and C, high MELD scores, thrombocytopenia, ascites and severe oesophageal varices were predictors of PVT in hepatic cirrhosis patients [9]. Severity of cirrhosis with PVT is partially a result of reduced portal blood flow.

The association of hepatocellular carcinoma with PVT is a result of prothrombotic changes, observed in tumours (platelet activation, higher thrombin expression, hypofibrinolysis, higher levels of prothrombotic microvesicles) [53].

Clinical presentation

PVT in hepatic cirrhosis is often asymptomatic and is diagnosed accidentally during an ultrasound examination or if a hepatic process is decompensated [2]. Symptoms of PVT are non-specific and include nausea, vomiting, mild abdominal pain, diarrhoea, loss of appetite. There is no correlation between intensity of clinical representation and characteristics of PVT: duration, degree of occlusion, stage of hepatic condition [18].

PVT can manifest or can be complicated by variceal bleeding, bowel ischaemia, portal biliopathy [17].

Hepatic cirrhosis patients with PVT are at a higher risk of oesophageal haemorrhaging, recurrent haemorrhage, low efficacy of endoscopic control of bleeding, increased 6-week mortality [6, 54, 55]. Recurrence of oesophageal varices after their ligation was more common in PVT (25.4 and 14.67 %, p = 0.03) [54]. PVT, active haemorrhage seen at endoscopic examination, low haematocrit, highly active aminotransferasees and high Child-Pugh class were predictors of 5-day inefficiency of haemorrhage therapy from upper GIT sections in cirrhosis — uncontrolled or recurrent haemorrhage [56].

The actual incidence of intestinal ischaemia and infarction in PVT with cirrhosis is unknown [2]. 67 % of patients with non-cirrhosis PVT had abdominal pain, caused by physical exercise, and signs of ischaemia (low saturation of the small intestine mucous membrane) [57]. No intestinal infarction in patients with cirrhosis and PVT was reported in prospective studies [16, 58], although if a blood clot moves to the superior mesenteric vein, the risk of intestinal infarction and associated mortality grows [2]. The probability of intestinal infarction is higher in complete occlusion of the portal vein and superior mesenteric vein; however, the risk is still not clear. More rare mesenterial ischaemia in hepatic cirrhosis can be a result of decompression due to portoportal collaterals [3].

Also, PVT manifests as portal biliopathy, which is partial or complete obstruction of intra- and extrahepatic bile ducts, gall bladder as a result of their compression by paracholecystic and paracholedochal venous plexuses, which appear as a response to blood clotting. Biliopathy is asymptomatic or has signs of cholestasis, biliary sludge, gallstone disease and even secondary biliary cirrhosis [59].

Diagnosis

Very often, PVT is diagnosed during Doppler ultrasound examination in asymptomatic patients or if the process is decompensated. Ultrasound examination in PVT is a screening method [1, 4]. Its sensitivity and specificity are 73–93 % and 99 %, respectively; its positive prognostic value is 86–97 %, while its negative predictive value is 98 %, which is comparable with angiography and computer tomography (90 %, 99 %, 95 %, and 97 %, respectively) [20, 60], showing a hyperechoic signal in the vessel lumen, dilated portal vein, absence/reduced blood flow in a part or all vein lumen; it is able to measure blood flow velocity and direction. Advantages of Doppler sonography include low costs, affordability and absence of radiation. However, this method is dependent on operator's experience; it is less reliable in bloating, obesity, partially occluded portal vein and where a blood clot moves to the splenic and superior mesenteric vein. Ultrasound examinations are hardly able to differentiate between soft blood clots and malignant portal vein invasion [17, 20], therefore it is recommended to perform a contrast-enhanced imaging examination after an ultrasound examination, also in order to rule out hepatocellular carcinoma [1-4].

An alternative to ultrasound examinations is computer tomography (CT), which is a diagnostic method for PVT and cavernoma [20]. Ultrasound examinations are reliable in detecting a blood clot in the portal vein trunk and branches; CT is better in assessing superior mesenteric vein, hepatic veins and inferior vena cava, presence of portal-systemic bypasses, involvement of other veins; it is more useful in diagnosing hepatocellular carcinoma and intestinal ischaemia. CT signs of PVT include a hypoechogenic and hypodense blood clot, more intense blood flow attenuation in the portal vein, more dense parenchyma during the arterial phase and reduced density during the portal phase. Blood clot calcification and cavernoma indicate chronic thrombosis [4, 18].

Contrast-enhanced magnetic resonance imaging (MRI) is an alternative to CT; however, it is not precise in ascites. It is more useful in identification of blood flow irregularities in the portal vein and thrombosis. It is safer than computer tomography, but is limited by movement and flow artefacts, it is less available, more expensive and associated with technical issues in patients with metal implants or surgical clips [4]. Usually, it is performed for additional imaging in young patients in order to reduce radiation [17].

CT, MRI or blood clot biopsy are used to rule out PVT caused by hepatocellular carcinoma invasion [2]. Ruling out malignant invasion of the portal vein (12–20 % of patients with hepatocellular carcinoma) is essential for making a decision on tumour therapy and whether a liver transplant is an option [18]. Signs of malignant invasion include an increase in the portal vein diameter, more contrast blood clot during the arterial phase, neovascularisation, the distance between tumour and blood clot of not more than 2 cm, tumour size of max. 5 cm. A-VENA criteria include the same criteria, save for tumour size, and also include a recommendation to use alpha-fetoprotein 1,000 ng/dL. Malignant invasion can be diagnosed on the basis of 3 criteria (100 % sensitivity, 94 % specificity, 80 % positive prognostic value, and 100 % negative prognostic value) [61].

A less known method is ultrasound-guided endoscopy, with 81 % sensitivity and 93 % specificity in PVT.

However, due to its invasive nature and the inability to reliably identify hepatocellular carcinoma or mesenteric infarction, this method is not recommended [20].

Due to the risk of esophageal varices in PVT, endoscopic screening should be performed as well [17].

Classification

The terminology and classification of PVT have been developed mostly for patient with liver transplant [3]. In the assessment of a spontaneous process and/or response to therapy, it is recommended to specify the initial localisation and spread of the blood clot, extent/degree of obstruction of a vessel lumen, involvement of intrahepatic branches, trunk of the portal vein, splenic and/or superior mesenteric vein, chronic process [1, 3].

Vein lumen occlusion can be complete (no lumen), partial (> 50 % of vessel lumen) or minimal (< 50 % of vessel lumen). It is essential not only for therapeutic decisions and evaluation of response to therapy, but also for establishing a correlation between blood clot localisation and clinical presentation. For instance, involvement of the superior mesenteric vein can cause intestinal ischaemia, while splenic vein thrombosis can result in venous dilation in the fundic stomach section [1, 3].

In terms of duration, PVT is classified as recent and chronic (less/more than 6 months, respectively). The term "recent" is more preferable than "acute", because the latter involves presence of clinical symptoms, while PVT is often asymptomatic and is diagnosed accidentally; it is impossible to establish the precise onset of this condition in some patients. In case of cavernous transformation, the latter term is preferable, although cavernomatosis is not a synonym of chronic PVT, because it develops 3–5 weeks after its onset [1, 3].

In spontaneous condition and/or for evaluation of response to therapy, PVT is classified as progressive (a blood clot grows, or complete occlusion is observed), stable (no changes in size or degree of occlusion) or regressive (a blood clot becomes smaller, or occlusion regresses) [1, 3].

Effect of PVT on the course of cirrhosis and prognosis

The course of hepatic cirrhosis with PVT is controversial [62]; it is challenging to identify whether thrombosis is a sign of poor prognosis, or a cause of cirrhosis progression [3]. According to a number of studies [12, 13, 16, 58, 63], PVT is not associated with disease progression or higher mortality rates; however, there is a completely opposite opinion [7, 10, 64, 65].

Effect on survivability

PVT is associated with poor outcome of hepatic cirrhosis and increases the risk of death [10, 64]. Patient with cirrhosis and PVT have lower 1-year survival rates (OR 0.12; 95 % CI 0.14–0.75; p = 0.008) and comparable 3-year survival rates (OR 1.04; 95 % CI 1.00–1.08; p = 0.06), 5-year survival rates (OR 1.33; 95 % CI 0.71–2.48; p = 0.38) and 9-year survival rates (OR 1.24; 95 % CI 0.79–1.93; p = 0.35) [65]. It is assumed that PVT in cirrhosis patients affects long-term rather than short-term survival rates [66].

Also, there is an opinion that PVT correlates with hepatic cirrhosis outcomes. For example, 2 years after PVT, survival rates in groups with process aggravation/stabilisation or improvement were 84.2 and 60.9 %, respectively (p > 0.05) [58]. Unlike subjects without thrombosis, patients with hepatic cirrhosis and PVT had lower mortality rates (OR 0.88; 95 % CI 0.81–0.96) and comparable risk of liver transplantation (OR 0.95; 95 % CI 0.89–1.02) [63]. 3-year survivability without transplantation in patients with PVT and without thrombosis was 100 and 82.8 %, respectively, while survival predictor was MELD value and not thrombosis [11, 63]. Cumulative survival rates in patients with viral hepatic cirrhosis were similar in groups with and without thrombosis [16].

Effect on disease progression

PVT induces or aggravates complications associated with portal hypertension: haemorrhage, hepatic encephalopathy, ascites [58, 66, 67]. According to a meta-analysis, PVT in patients with hepatic cirrhosis increases the risk of functional decompensation (OR 2.52; 95 % CI 1.63–3.89, p < 0.001) [64], acute kidney injury (OR 1.75; p < 0.001) and hepatonephric syndrome (OR 1.62; p < 0.001) [10]. PVT was associated with longer endoscopic therapy of oesophageal varices [68].

However, it is possible that PVT, unlike initial Child-Pugh or MELD values, was not associated with cirrhosis progression [11]. The probability of hepatic decompensation within 2 years in groups with PVT worsening or improvement/stabilisation was 68.4 and 60.9 %, respectively; cases of hospitalisation for hepatic decompensation were 63.2 and 47.8 % [58]. Cirrhosis progression (probability of ascites, hepatic encephalopathy, variceal bleeding, manifestation of hepatocellular insufficiency) was associated with the patient's age (OR 1.55; 95 % CI 1.11–2.17), body mass index (OR 1.40; 95 % CI 1.01–1.95), prothrombin time (OR 0.79; 95 % CI 0.70–0.90), serum albumin level (OR 0.97; 95 % CI 0.94–0.99), oesophageal varices (OR 1.70; 95 % CI 1.21–2.38), but not with PVT (OR 1.32; 95 % CI 0.68–2.65) [12].

Effects on surgical aspects of liver transplantation

PVT, especially complete PVT, affects the rate of complications and survival rates of patients with liver transplant and has been considered a contraindication to transplantation [17].

Intraoperatively diagnosed PVT is associated with longer surgical support, risk of severe bleeding and longer cold ischaemia of the transplant. In stage I–III PVT (classification by M. A. Yerdel et al., 2000), thrombectomy with portoportal anastomosis is a standard method. In portal vein luminal narrowing, a donor iliac vein transplant is used [17]. In some cases of stage III and IV PVT, complex vessel reconstruction is performed using mesoportal jump grafts from donor veins or synthetic vessel transplants with a portocaval bypass or portal vein arterialisation; however, these procedures are associated with a high risk of post-transplantation hypertension [7, 69].

In case of liver transplant from a living donor, transplants have a short portal vein, whereas anastomosis requires an adequately long recipient vein, which is not always possible in patients with PVT. Therefore, liver transplantation in patients with complete PVT from a living donor is technically more complex and is associated with high mortality rates. In complete PVT, the portal vein can be replaced with a recanalised umbilical vein, subcutaneous vein from a donor or recipient, as well as with hepatic veins from a cirrhotic liver. In recurrent thrombosis, surgical outcomes are similar to those in recipients without thrombosis.

Effects on liver transplantation outcomes

PVT has negative impact on the survival rates of patients with a liver transplant [58, 66, 67, 70], which depends on the extent of thrombosis during surgery [71]. 30-day (13 % vs. 7 %, OR 2.29; 95 % CI 1.43–3.68; $p < 0.0001$) and 1-year mortality rates after transplantation (13.5 % vs. 9.9 %, OR 1.38; 95 % CI 1.14–1.66; $p < 0.0001$) in patients with PVT are higher in patients with PVT vs. patients without thrombosis [67]. Similar data on 30-day (10.5 % vs. 7.7 %) and 1-year (18.8 % vs. 15.4 %) mortality were reported in another study [8]. It is assumed that only complete PVT increases 30-day and 1-year mortality after transplantation [8, 52, 67]. High post-transplant mortality in patients with PVT is observed only during the first year after surgery (OR 1.32; $p = 0.02$) [72].

The highest post-surgery mortality rates, including early mortality (25 %), were recorded in patients with stage IV PVT (classification by M. A. Yerdel et al., 2000) and were associated with portal hypertension severity [73].

There is also some evidence that PVT does not affect survival rates of post-transplant patients and is associated with longer surgery [74]. Patients with PVT and patients without thrombosis did not have any differences in 1-year (85 % and 86 %) and 5-year survival rates (68 % and 73 %) [75]. In non-occlusive PVT, the rate of post-transplant mortality was similar to that in patients without thrombosis [8].

Usually, post-transplantation PVT develops in the area of anastomosis if the diameter of the donor vein and recipient vein mismatches. The incidence of PVT in patients without a history of thrombosis is 0–2 %, while in patients with pre-existing PVT, this value is 2–3 % [74]. Often, PVT development soon after surgery is associated with a poor prognosis [17].

Conclusion

The article describes the epidemiological data, risk factors, clinical manifestation and diagnostic search in portal vein thrombosis in patients with hepatic cirrhosis. The correlation between portal vein thrombosis and cirrhosis progression, survival rates, outcomes of hepatic transplantation is demonstrated.

Вклад авторов:

Все авторы внесли существенный вклад в подготовку работы, прочли и одобрили финальную версию статьи перед публикацией

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