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The Portal Venous System and Portal Vein Thrombosis: Review Article

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Portal Vein Anatomy

Portal vein is formed by the confluence of superior mesenteric and splenic veins at the L2 vertebral level behind the head of pancreas after the opening of inferior mesenteric vein into the splenic vein. It continues for approximately 8–9 cm within the hepatoduodenal ligament until its bifurcation at the hepatic hilum. The portal vein is located partially dorsal and slightly medial to the common bile duct in the hepatoduodenal ligament. At this location, left gastric vein also connects with the portal vein. Variations occur in the branching pattern of the portal vein when it branches in the liver. These variations are classified as follows: Type 1, normal portal vein branching pattern; Type 2, portal trifurcation variant in which anterior posterior branches of the right PV and left PV originate from the main portal vein at the same level; and Type 3: posterior branch of the right PV is classified as the first branch of the main portal vein. Schmidt et al. Portal vein normal anatomy and variants, Portal venous pressure is usually between 5 and 10 mmHg in a healthy person [1,2].

Even though portal vein and hepatic artery work together for delivering nutrients to and storing and removing metabolites from the liver, 75% of the blood supply to the liver originates from the portal vein and 25% originates from the hepatic artery; that is, portal vein blood flow is approximately 1000–1200 mL/min. Blood collected by the capillary system from the gastrointestinal tract is transported through the portal vein to the liver and returns via the hepatic veins. Central veins surrounded by the liver lobules turn into sinusoids during their course along the lobules, and their diameter increases as they form post-lobular vein by converging with the veins originating from the other lobules. These veins converge to form the hepatic veins and then open into vena cava inferior as two or more branches. Portal vein exhibits variations while intrahepatically branching [3].

Introduction

Portal vein thrombosis, which was defined for the first time in 1869, is the blockage of the portal vein or its branches by clot or tumoral infiltration, and it is one of the causes of presinusoidal portal hypertension. The risk factors involved in the etiology of thrombosis described by Wirchov are valid and all conditions resulting in stasis and endothelial damage and hypercoagul ability are considered as risk factors for the development of portal vein thrombosis, the most common cause being liver cirrhosis. At this point, the slowing down of blood flow in the portal vein, disruption in synthesis function of the liver (such as protein C, S and antithrombin 3), and hepatocellular carcinoma (HCC) development on cirrhosis are implicated as the primary predisposing factors. Another reason is the direct invasion of tumor into or external compression to the portal vein by hepatopancreatobiliary system malignancies. Moreover, thrombogenic factors released from the tumor can also cause this condition. Other causes include the occurrence of malignant diseases such as HCC, pancreatic cancer, and bile duct cancers. Numerous etiologic causes have been described to date and are listed in Table 1 [4].

Microanatomy of Liver, Histopathology in PVT

It is imperative to examine hepatic microanatomy (hepatic lobule and acinus structure) to better understand the morphological changes in the liver. In its classical model, “lobule” is a hexagon structure which comprises portal regions at its corners and terminal hepatic venule at its center. In hepatic acinus structure, there are cell groups that receive blood supply from the same branch

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of hepatic artery and portal vein within two adjacent lobules. Within this acinus structure, hepatocytes are divided into three zones with poorly defined borders based on their proximity to supplying vessels. This zonal arrangement is useful in explaining metabolic functions between blood and hepatocytes and the reason why hepatocytes are affected by various agents and diseases at different levels. The cells in zone 1 are the first and cells in zone 3 are the third to respond to blood flow. The cells in zone 1 are the closest to veins, and are thus the first to benefit from or be injured from the oxygen, nutrients, and toxins in the blood and bile duct blockages [5].

Three important anatomic structures in the portal area are as follows: portal venule, hepatic arteriole, and bile duct. The hepatocytes inside the lobule are radially arranged around the central vein as a single row of cells. The adjoining hepatocytes form bile canaliculi. Blood flow is in the sinusoidal gaps which are connected to portal venule and hepatic arteriole and which are between the cords of hepatocytes. Sinusoids are lined by specialized endothelial cells and Kuppfer cells, which are the tissue macrophages of liver, and are found in the sinusoids [6]. The space of Disse stretches between endothelial cells and hepatocytes and the exchange of substances between blood and hepatocyte occurs in these spaces.

Even though several reasons are mentioned in the etiology of PVT, we do not expect a considerably different morphologic appearance in histopathology because of these etiologies. Acute thrombosis of intrahepatic portal vein roots does not cause ischemic infarction, but it rather produces regions with well-defined margins, which is called "Zahn's infarct." Histopathologically, necrosis is not observed but hepatocellular atrophy and apparent dilatation and hemostasis exist in the sinusoids; in addition, a dense fibrosis of intraheptic portal tract formation caused by the obstruction of portal vein branches is known as hepatoportal sclerosis and it is not clear whether this histology is a late stage of a liver damage or an underlying primary progressive disease [7].

Clinical Appearance

The severity of clinical symptoms in portal vein thrombosis depends on the involvement of other vascular structures (Table 2) and both acute and chronic forms can be encountered in clinic practice [8]. In acute PVT, clinical findings depend on the extensiveness of thrombus and the time that has elapsed after symptom onset. In acute form, severe abdominal pain, bloody diarrhea, and epigastric complaints are the main presenting complaints, whereas esophageal variceal hemorrhage may be an initial manifestation in patients with cirrhosis who follow an insidious clinical course. Moreover, congestion, ischemia, necrosis, and perforation may develop in the small bowel of patients with acute thrombosis as a result of the involvement of superior mesenteric vein. The crucial factor negatively affecting prognosis in acute form is the development of intestinal ischemia rather than hepatic ischemia. Furthermore, ascites may develop in acute form.

In the chronic form, patients may have an asymptomatic course or exhibit symptoms associated with portal hypertension that develops secondary to portal vein thrombosis. The main presenting complaints include esophageal variceal hemorrhage, hypersplenism, splenomegaly, and portal biliopathy. Cholestasis and related findings such as ascites, jaundice, fatigue, pruritus, and colic pain are prominent in chronic form accompanied by portal biliopathy. Laboratory tests show elevated liver enzymes, increase in leukocyte count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) level, and inflammatory markers or leucopenia-thrombocytopenia-anemia associated with hypersplenism, hemoconcentration, hyperbillurbinemia, increase in INR and creatinine levels, and hypoabuminemia [9].

Table 1: Causes of PVT.

Cirrhosis (Most common)
Malignancies (Bile duct cancer, pancreatic cancer, HCC)
Hematologic Diseases (Polycythemia vera, etc.)
Hypercoagulability (Protein C/S, Antithrombin 3 deficiency, Factor 4 Leiden mutation, Antiphospholipid synd, etc.)
Infections (Acute Appendicitis, Sepsis, Causes of Acute Abdomen)
Acute Pancreatitis
Rheumatological Diseases (Behçet's Disease, Lupus)
Pregnancy
Surgical Trauma

Table 2: Classification based on the extensiveness of vascular involvement.

Type I	Thrombosis is limited to the portal vein
Type II	Thrombosis of the portal vein that extends into the SMV, but mesenteric veins are patent
Type III	Splanchnic venous system is extensively involved with accompanying collaterals
Type IV	Different from Type III, large collaterals turn into fine collaterals

Diagnosis

With technological advances, Doppler ultrasonography (USG), computed tomography (CT), and magnetic resonance (MR) imaging have replaced invasive methods such as mesenteric angiography or portal venography, which were previously preferred in the diagnostic process and used to detect thrombus. In many clinics, endosonography has been established as part of the diagnostic process [10].

Doppler ultrasonography is the first cheap and noninvasive imaging method used for diagnostic purposes and it easily demonstrates biliary pathologies; the demonstration of thrombus within the portal vein without accompanying cavernous transformation suggests acute PVT. On Doppler USG, the demonstration of enlargement in the portal vein and hypoechoic thrombus is the basic finding of acute PVT. CT with contrast enhancement allows visualization of malignancies that may accompany portal vein thrombosis. Contrast enhancement is also capable of detecting abscess, intestinal necrosis, and perforation. Angiographic methods can be preferred if the diagnosis remains unclear after standard radiologic imaging or a shunt operation is being planned. Angiography can be performed with concurrent therapeutic interventions in patients with acute thrombosis [11,12].

Treatment

The first-line treatment after the diagnosis of acute PVT is anticoagulant therapy. The primary purpose of anticoagulant therapy is the prevention of the growth of the existing thrombus and extension into other veins, particularly SMV, and the allowance of recanalization. The ultimate goal is the prevention of intestinal ischemia that may cause mortality, while protecting the liver. In chronic form, treatment planning should be based on the presence of accompanying esophageal and gastric varices. If necessary, beta-blocker therapy and endoscopic band ligation are important in prophylaxis against the risk of hemorrhage. Low-molecular-weight heparin (DMAH,

2 x 0.01/kg) which has today replaced heparin therapy and has a lower complication rate, should be preferred in first-line therapy. Subsequently, warfarin treatment should be commenced. Target INR value should be between 2 and 3. The treatment is expected to last 3–6 months. In patients with hypercoagul ability, a longer treatment period may be required. The rate of recanalization in patients with acute PVT is 50%–75%. Thrombolytic therapy (tissue plasminogen activator and streptokinase) can also be used in the acute phase. Furthermore, surgical thrombectomy is not routinely performed but can be considered in cases with venous involvement which causes diffuse intestinal ischemia [13].

Conclusion

There has been significant improvement in the understanding of surgical anatomy of the liver. Developments, especially in liver transplant and hepatobiliary surgery, are ongoing. PVT is a significant challenge which may be encountered in hepatobiliary pathologies. Various risk factors are involved in the etiology of the disease and the fundamental treatment approaches involve varice eradication and anticoagulant therapy.

References

1. Kan Z, Madoff DC. Liver anatomy: microcirculation of the liver. Semin Intervent Radiol. 2008; 25: 77-85.
2. McCuskey RS. A dynamic and static study of hepatic arterioles and hepatic sinusoids. Am J Anat. 1966; 119: 455–478.
3. Oda M, Yokomori H, Han JY. Regulatory mechanisms of hepatic microcirculation. Clin Hemorheol Microcirc. 2003; 29: 167–182.
4. Ponziani FR, Zocco MA, Campanale C, Rinninella E, Tortora A, Di Maurizio L, et al. Portal vein thrombosis: insight into physiopathology, diagnosis, and treatment. World J Gastroenterol. 2010; 16: 143-155.
5. Putnam CW, Porter KA, Weil R 3rd, Reid HA, Starzl TE. Liver transplantation of Budd-Chiari syndrome. JAMA. 1976; 236: 1142-1143.
6. Kan Z, Ivancev K, Lunderquist A, et al. *In vivo* microscopy of hepatic metastases: dynamic observation of tumor invasion and interaction with Kupffer cells. Hepatology. 1995; 21: 487–494.
7. Abu El-Makarem MA, El-Akad AF, Elian MM, Sherif TM, El-Shaheed RA, Abd El Fatah AS, et al. Non-neoplastic Portal Vein Thrombosis in HCV Cirrhosis Patients: Is it an Immuno-Inflammatory Disorder? Ann Hepatol. 2017; 16: 574-583.
8. Webster GJ1, Burroughs AK, Riordan SM. Review article: portal vein thrombosis -- new insights into aetiology and management. Aliment Pharmacol Ther. 2005; 21: 1-9.
9. Al Hashmi K, Al Aamri L, Al Lamki S, Pathare A. Portal Vein Thrombosis in Adult Omani Patients: A Retrospective Cohort Study. Oman Med J. 2017; 32: 522-527.
10. Lai L, Brugge WR. Endoscopic ultrasound is a sensitive and specific test to diagnose portal venous system thrombosis (PVST). Am J Gastroenterol. 2004; 99: 40-44.
11. Sacerdoti D, Serianni G, Gaiani S, Bolognesi M, Bombonato G, Gatta A. Thrombosis of the portal venous system. J Ultrasound. 2007; 10: 12-21.
12. Tessler FN, Gehring BJ, Gomes AS, Perrella RR, Ragavendra N, Busuttil RW, et al. Diagnosis of portal vein thrombosis: value of color Doppler imaging. AJR Am J Roentgenol. 1991; 157: 293-296.
13. Huard G, Bilodeau M. Management of anticoagulation for portal vein thrombosis in individuals with cirrhosis: a systematic review. Int J Hepatol. 2012; 2012: 672986.