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Commentary

Portal Vein Thrombosis in Patients with Chronic Liver Diseases

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DESCRIPTION

The portal vein is formed by the confluence of the splenic and superior mesenteric veins, which drain the spleen and small intestine, respectively. Obstruction of the portal vein by a blood clot (portal vein thrombosis) (PVT) usually occurs in patients with cirrhosis and/or thrombogenic disorders. Portal vein occlusion due to thrombosis develops suddenly in patients with acute PVT. Obstruction may be complete or partial. In addition to the portal vein, thrombi can also affect the mesenteric or splenic veins. Patients with acute PVT have not yet developed features of chronic PVT, such as collateral circulation (eg, portal spongiosis) or portal hypertension. If the time of thrombus development is unknown, but the patient has no features of chronic PVT, PVT can be described as 'recent'. Patients with recent her PVT are treated in the same manner as those with acute PVT.

Portal vein thrombosis causes upper abdominal pain and may be accompanied by nausea and enlarged liver and/or spleen. The abdomen may be filled with liquid. Persistent fever may be due to systemic inflammation. If a clot forms suddenly, abdominal pain may come and go, but long-term clot formation can also occur without causing symptoms, leading to portal hypertension before it is diagnosed.

Other symptoms may appear, depending on the cause. For example, portal vein thrombosis from cirrhosis may cause bleeding and other signs of liver disease. People who develop portal vein thrombosis due to portal vein thrombosis may have signs of infection, such as fever, chills, and night sweats. Delayed blood flow due to underlying cirrhosis or congestive heart failure is often implicated. Some estimate that it affects 1 in 1, while others believe it affects closer to 1 in 4.

Thrombotic tendency (including genetic disorders such as factor V Leiden deficiency, protein C or S deficiency, or antiphospholipid antibody syndrome) is another common cause. Nearly one-third of patients have myeloproliferative disorders (eg, polycythemia Vera or primary thrombocytosis), most commonly due to Janus Kinase 2 (JAK2) gene mutations. Oral contraceptive use or pregnancy is other non-hereditary thrombotic tendencies.

Alternatively, the portal vein may be damaged as a result of pancreatitis, diverticulitis, cholangiocarcinoma, Hepatocellular Carcinoma (HCC), or abdominal surgery/trauma. Warning signs of causative cancerous growth include elevated alpha-fetoprotein levels, portal vein diameter greater than 2.3 cm, pulsatility on Doppler ultrasonography, or high blood pressure on contrast-enhanced CT scans. Include the Hepatic Arterial Phase (HAP) signal.

PVT is also a known complication of surgical removal of the spleen. In recent years, Myeloproliferative Neoplasms (MPN) has emerged as a major systemic cause of visceral venous thrombosis (including PVT).

Cirrhosis is the most common cause of PVT. In non-cirrhotic livers, PVT is primarily due to congenital or acquired thrombotic conditions. Primary Myeloproliferative Disease (PMD) is the most common procoagulant condition. Other thrombogenic disorders that cause PVT include hereditary thrombogenic disorders such as paroxysmal nocturnal hemoglobinuria (PNH), antiphospholipid syndrome, hyperhomocysteinemia, protein C, S, and antithrombin III deficiency. Included, rare, factor V Leiden mutations, factor II mutations, and Methylenete Tra-Hydro-Folate Reductase (MTHFR) gene mutations. Rare conditions associated with PVT are pregnancy, chronic inflammatory disease, oral contraceptives, and malignancies with or without the above prothrombotic causes. Malignant tumors account for approximately 25% of PVT cases.

Intraperitoneal inflammatory conditions leading to vascular endothelium damage can lead to PVT. These include pancreatitis, cholangitis, appendicitis, and liver abscess. Local injury to the portal vein axis after splenectomy, laparoscopic colectomy, or abdominal trauma with acquired or hereditary thrombotic conditions described above can cause PVT.

The prevalence of PVT in cirrhosis has been reported to range from 0.6% to 16% and is more common in patients awaiting liver transplantation. PVT is observed in up to 35% of patients with cirrhosis and hepatocellular carcinoma. Lifetime risk of PVT in the general population is given as 1%.

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