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Thrombosis of Portal Vein

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Abstract: ***Cirrhosis and portal hypertension is the most prevalent causes of portal vein thrombosis (PVT) in the general medical population; however, it can also be caused by liver tumors (Plessier, 2021). In most cases, the diagnosis is made as a result of an imaging test finding a thrombus in the portal vein. Symptoms of PVT, such as ischemic hepatitis, liver problems, or small intestine infarction, can be life - threatening if left untreated. Pathophysiology of this illness is discussed in length, with an emphasis on PVT in cirrhotics, and extensive advice is provided on the best diagnostic and treatment options.***

Keywords: Cirrhosis, hypertension, thrombosis, liver tumors, thrombus

1. Introduction

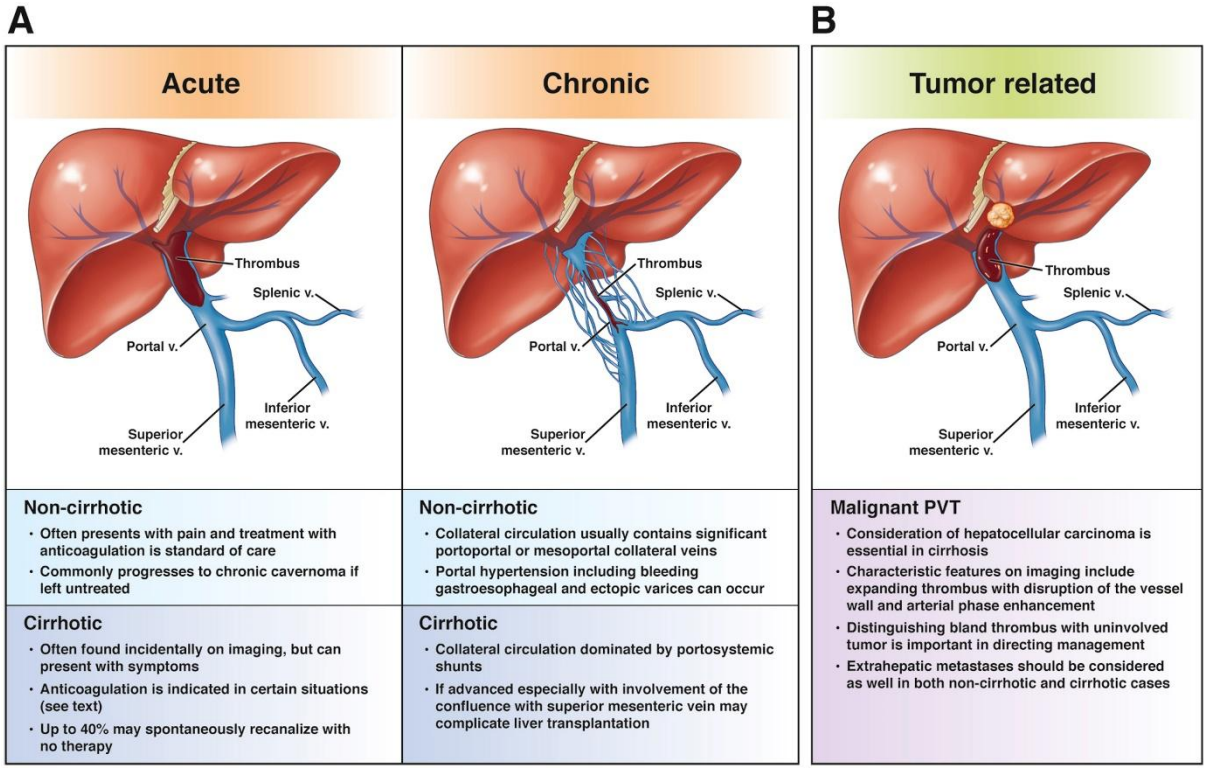
Occlusion of the portal vein and its tributaries' lumen by thrombus development is known as portal vein thrombosis (PVT). The increased the using abdominal imaging (most typically Doppler ultrasonography) during standard medical evaluations and liver cancer surveillance is leading to an increase in the number of PVT diagnoses, many of which are discovered by chance. PVT is associated with three major clinical complications: An extremely high death rate of up to 50% is associated with small intestinal ischemia, which may necessitate a small bowel and multivisceral transplantation if the patient is still alive after a PVT hepatofugal extension.

2. Classification

Acute or chronic onset is possible with PVT. In clinical practice, it can be difficult to distinguish between acute and chronic PVT because within 60 days of being admitted to the hospital, symptoms such as abdominal discomfort, nausea, and fever may begin to occur (Jegadeesan et al., 2017).

To differentiate between four different types of primary portal vein thrombosis, we can look at how far it extends into the mesenteric system, the size of the collateral vessels, and the extent of the enlarged vein. We can also classify PVT as having large collateral vessels, small collaterals, or none at all, depending on the extent of the thrombosis. By utilizing this classification, one may determine a patient's operability as well as their clinical outcomes. It is more dangerous to have thrombosis in both the portal and mesenteric arteries, even if the variceal incidence is minimal. haemorrhage.

Grade I	<50% of light, with no or minimal obstruction of the superior mesenteric vein
Grade II	Grade I with obstruction > 50%, including total obstruction
Grade III	Complete obstruction of the portal vein and proximal superior mesenteric vein
Grade IV	Complete obstruction of the portal vein and superior mesenteric vein



3. Causes

Cirrhosis and congestive heart failure, both of which can cause blood flow to slow, are frequently to blame. There is a lack of consensus on the incidence of PVT in cirrhotic patients, with several studies reporting varying rates of occurrence (Castaneda et al., 2002).

Thrombophilia (containing diseases like factor V Leiden, protein C, and S deficiencies, or acquired immune syndrome) is another frequent cause. The majority of myeloproliferative disorders are caused by a mutation in the Janus kinase 2 gene. Other non - inherited risk factors for thrombosis include oral contraceptive use and pregnancy.

Additionally, the portal vein can be damaged by pancreatitis, diverticulitis, cholangiocarcinoma, hepatocellular carcinoma (HCC), and abdominal surgery/trauma. On a CT scan with contrast, abnormally high levels of alpha - fetoprotein or an abnormally large portal vein diameter (more than 2.3 cm) are red flags for malignant development.

A complication of surgery to remove the spleen is also known as PVT. Myeloproliferative neoplasms (MPNs) has emerged as the primary systemic source of splanchnic vein thromboses in recent years (including PVT)

Symptoms

Few or even no symptoms are common in many cases with PVT (Plessier, 2021). The following are among the most prevalent signs of a less serious blood clot:

- The ache in the upper abdomen
- Extra fluid in the abdomen causes a swollen abdomen

You may get portal hypertension, meaning high blood pressure inside the portal vein if your incidence of portal vein thrombosis is severe enough. Splenomegaly, or perhaps an enlarged spleen, is the result of pressure interfering with normal blood flow in this illness. Increased risk of infection as the spleen grows. Varices (enlarged blood flow) in the esophagus or stomach can also be caused by portal hypertension, which can lead to bleeding.

Aside from these, other serious signs for portal vein thrombosis include high fevers, chills, liver pain, vomiting, and yellowing of the skin called jaundice.

Risk factors

There is a greater risk of blood clots forming if blood flow in the body is erratic. There seem to be various risk factors for getting portal vein thrombosis, even though doctors often don't know what causes it (Lankarani, 2021).

These include pancreatic inflammation, appendicitis, naval infection from of the umbilical cord stump, and polycythemia, or extra red blood cells in babies orally administered cancer prevention contraceptives the liver disease cirrhosis of a liver.

Pregnancy and surgery can further increase the risk of pulmonary venous thrombosis. Blood clots are more prone to form in each of these scenarios, limiting blood flow to the rest of the body. These variables can lead to life - threatening consequences in more severe cases.

Complications

The severity of liver as well as other organ involvement should be examined when the diagnosis is made. The severity of the obstruction In addition to clinical and laboratory assessments, imaging should be used to examine the condition. There are just a few symptoms that can be linked to a partial thrombus. Isolated intestinal congestion can be caused by a full blockage of both portal and mesenteric veins without the involvement of a mesenteric vein arch, which results in a distributed thickness of an intestinal wall. Most patients do not experience additional organ failures, and liver function is often unaffected by increased hepatic arterial blood circulation. It took 2 to 3 days for the gallbladder thrombosis to cause collateral circulation to grow through pre - existing veins in the porta hepatis. Regardless of whether there is a natural recanalization or even a cavernomatous change, all of these symptoms can be reversed. It can lead to intestinal infarction if a thrombus extends to the mesenteric venous arches. The weakening of an intestinal wall as well as the appearance of enhancement defects following intravenous contrast administration is common radiographic findings.

Diagnosis

Ultrasound is indeed the least invasive way to diagnose portal vein thrombosis, and the inclusion of the Doppler technique displays a filling deficiency in blood flow as evidence (Glick & Jamouz, 2019). Based on the presence or absence of blood flow all around the clot, PVT can be characterized as occlusive or nonocclusive. Type 1 clots are located in the main portal vein; Type 2 clots are found in a portal vein branch, and Type 3 clots are found in both regions. Computed tomography (CT) using contrast, magnetic resonance imaging (MRI), with Mr angiography may be used to determine the severity of the problem (MRA). A gastroscopy or esophageal endoscopy may be recommended for patients with persistent PVT to check for the presence of any further dilated veins. For the most part, liver function tests show no abnormalities aside from modestly increased transaminases. A rise in D - dimer level in blood may well be caused by fibrinolysis.

Table

Thoracic risk variables in a series of routinely screened, non - cirrhotic, acute, or chronic PVT patients in adulthood.

Risk factor	PVT patients (%)
Multiple myelodysplastic syndromes	30 - 40
Atypical	14
Classical	17
a lack of antithrombin	0 - 26
A deficit in protein C	0 - 26
A deficit in the protein S	Feb - 30
a mutation in factor V that causes Leiden disease	Jun - 32

Pathophysiology

Systemic & splanchnic hemodynamics is significantly altered as a result of portal vein occlusion (Valla & Condat, 2000). The liver loses around two - thirds of its blood supply when the portal blood supply is cut off. In contrast to the severe hepatic dysfunction that can be deadly when an abrupt artery blockage occurs, this condition is frequently easily tolerated by patients and often leaves them with no symptoms. The loss of the portal vein's contribution to liver blood flow may be supplemented by the rapid activation of two compensatory processes. Hepatic artery "arterial vasodilation," analogous to portal vein clamping on liver resection, is the first mechanism. For those suffering from PVT, this "arterial rescue" vascular reflex can help keep the liver functioning even if blood flow has been disrupted by the disease. Venous rescue is a second compensatory mechanism that involves the quick formation of collaterals to avoid the restriction in the blood flow. It takes about 3 to 5 weeks from when the portal vein is blocked for this vascular neo - formation to commence. It's for this reason that a "cavernoma" of collateral veins connects the two patent areas close to and far away from a thrombus in its place, which is known as a "thrombosed portal vein." It is difficult to see the actual portal vein because it has thickened and fibrotic. High cardiac output and low blood pressure are prominent characteristics of hyperkinetic circulations at this time.

Disruption of portal blood flow causes significant effects on liver tissue, notwithstanding the elaborate support system that has been activated. An animal study has shown that portal vein obliteration in rat's results in an increase of mitotic activity and death of hypoperf used and normal perfused cells, respectively. In respective liver surgery, this latter effect is used therapeutically. As a result of this process, the liver's ability to synthesize hormones may be impaired in more severe stages with portal vein obstruction.

Treatment

Despite reports of clinical improvement of PVT in the literature, appropriate therapeutic therapy is required to remove the obstruction of the portal vein and prevent catastrophic consequences (Kokubu, 2019). Both acute and chronic PVT are treated similarly, with the primary goal of correcting causative causes, preventing extending the thrombosis, and making sure the portal vein is open. Concurrent evaluation of treatment for issues associated with portal hypertension and portal cholangiopathy is required in cases with long - standing thrombosis. Anticoagulant therapy is currently the best option for reopening the portal vein, although there is no unanimity on how to use it. Just if PVT does not resolve, or only partially, should further treatment options be considered. Another factor that should be taken into account when considering anticoagulant therapy is whether or not there has been recent or long - standing embolism.

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