

## PORTAL VEIN THROMBOSIS — ULTRASOUND IMAGING

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**Abstract:** Portal venous system, apart from the main portal vein, includes its tributaries: superior and inferior mesenteric vein, as well as splenic vein, so the term portal venous thrombosis encompasses a broad spectrum of pathological conditions. Usually, one or more causative factors can be recognized, either local endothelial/flow disturbances, or systemic inherited/acquired conditions. Portal vein thrombosis can be associated with benign or malignant disorders. Whether we are speaking about acute or chronic thrombosis, the clinical presentation is different. Acute thrombosis can be presented in a wide range, from mild abdominal discomfort to a state of intestinal ischemia and life-threatening infarction. Chronic thrombosis is usually recognized when variceal bleeding or other symptoms of portal hypertension express. Fast and accurate diagnosis sometimes is a life-saving procedure, especially in acute vascular alterations. Recently, due to the improvement of imaging procedures the number of patients with diagnosed portal vein thrombosis is increasingly growing. With a negative predictive value of 98% color Doppler ultrasound is considered as imaging modality of choice in detecting portal vein thrombosis. Based on large studies it is presumed that overall risk of getting portal vein thrombosis during lifetime is 1% in general population, but much bigger 5%-15% in cirrhotic patients. Existence of specific ultrasound criteria, if fulfilled, has ensured that diagnosis of portal vein thrombosis is fast and non-invasive. Procedure is convenient for the patient and healthcare providers, and above all, allows prompt treatment preventing further deterioration.

**Key words:** portal vein, portal vein thrombosis, portal hypertension, ultrasound, color-Doppler ultrasound.

### INTRODUCTION

Main portal vein (PV) arises behind the head of pancreas where the superior mesenteric vein (SMV) bringing blood from small intestine and part of pancreas, and splenic vein (SV) bringing blood from the spleen, join together.

Blood running from large intestine through inferior mesenteric vein (IMV) usually drains into SV, while the blood from the stomach and part of pancreas drains directly into the main PV. In the liver hilum, PV divides in two main branches, one for each liver lobe. Consequently, regarding anatomy of the portal venous system, the term portal venous thrombosis (PVT) includes clot formation in the main portal vein, its branches or its tributaries.

Due to common risk factors, thrombosis may appear in one, or in several parts of portal venous system. Accordingly, clinical signs may be confusing, and symptoms may overlap. Splenic vein thrombosis can be present as isolated condition, while thrombosis of superior and inferior mesenteric vein is usually accompanied with thrombus in the main portal trunk.

Occlusion may be complete, involving the whole vessel lumen, or partial, with adherent clot and flow present on the periphery of the vein. Several classifications of PVT nowadays are used.

The anatomical classification into four categories refers to extra hepatic PVT: thrombosis in the main PV behind the confluence of the SMV and SV (grade 1), thrombus extended in the SMV but not in the mesenteric vessels (grade 2), complete thrombosis of splanchnic veins with presence of huge collaterals (grade 3) and diffuse splanchnic thrombosis with only small collateral vessels (grade 4).

Another frequently used classification, especially in planning liver transplantation, proposed by Yerdel identifies 4 different grades of PVT as shown in Table 1 (1, 2).

Both classifications besides anatomical, have important etiological and prognostic relevance, since patients with extensive thrombosis have a greater risk of bowel infarction than those with isolated PVT who are prone to variceal bleeding.

### ETIOLOGY

According to recent knowledge, occurrence of venous thrombosis including PVT results from more than

**Table 1.** Classification of PVT according to Yerdel

Grade 1: Minimal or partial thrombosis of portal vein, the clot is defined on less than 50% of vessel lumen, with/without minimal extension in the SMV
Grade 2: more than 50% occlusion of the portal vein lumen, including total occlusion, with/without occlusion of SMV
Grade 3: complete thrombosis of portal vein and proximal part of SMV (distal part of SMV is clot-free)
Grade 4: complete thrombosis of portal vein and proximal as well as distal part of SMV

one causative factor. Risk factors, local or systemic, may contribute to development of PVT. Changes in any part of Virchow's triad (coagulation disturbances, reduced flow or endothelial lesion) can be responsible for clot formation.

Most frequent risk factors are hypercoagulable disorders, inherited or acquired prothrombotic conditions. Decreased flow velocity in portal hypertension is most common cause among cirrhotic patients, while local precipitating risk factors, such as inflammation or infection, results in endothelial disturbances and thrombus formation.

In one big retrospective study involving 225 patients with PVT, based on patients medical history, risk factors were divided in a low grade risk factors (hypertension, diabetes mellitus, tobacco or alcohol use, SLE or sarcoidosis and illicit drug use) and high grade risk factors (preexisting liver disease in 69%, and regional

cancer present in 47% of PVT patients, pancreatitis, hereditary thrombophilia, family history of thrombosis) (3). Another study clearly supports the hypothesis that concurrence of several prothrombotic factors favors development of PVT. Studying influence of several disorders : protein C, protein S and antithrombin deficiency, antiphospholipid syndrome; factor V-Leiden, factor II and methylene-tetrahydrofolate-reductase (MTHFR) gene mutations, French group concluded that PVT "should be regarded as an index for one or several prothrombotic disorders, whether or not local precipitating factors or oral use of contraceptives is found"(4). Risk factors for portal vein thrombosis are shown in Table 2.

## PATHOPHYSIOLOGY

PVT has several consequences on systemic and splanchnic hemodynamic and their relevance depend on the extension of the clot. If clot is limited on the main portal vein, upstream of the thrombus there are no consequences on the intestines. As a result of portal vein obstruction, liver is left without two thirds of its blood supply; circumstances in which two "buffer" mechanisms intervene to compensate the loss.

The first one is almost immediate increase in arterial blood flow through dilation of hepatic artery. The second is so called "venous rescue"; development of collateral vessels with aim of bypassing the obstructed part of the vein. The appearance of "collaterals" is very rapid; it starts after occlusion, becoming visible almost in several days.

Imaging methods are revealing portal vein as a tiny cord, surrounded by irregular dilated vessels, mimicking vascular tumor. First time when this phenomenon was noticed, it was named "cavernoma" since it was considered as development anomaly in children. Collaterals are present in porta hepatis and in adjacent organs like gallbladder wall, duodenal wall, surrounding pancreatic head (5, 6).

Liver is rarely suffering from decreased portal flow, although transient signs of decompensated liver disease may be present. In experimental models, after portal vein ligation in mice, histology revealed marked apoptosis of hepatocytes, with increased mitotic acti-

**Table 2.** Risk factors for portal vein thrombosis

Hypercoagulable states:	
Inherited:	antithrombin deficiency
	Protein C deficiency
	Protein S deficiency
	Heterozygous factor V Leiden
	Prothrombin mutation
	MTHFR mutation
Acquired:	Malignancy
	Myeloproliferative disorders
	Use of oral contraceptive pills
	Paroxysmal nocturnal hemoglobinuria
	Inflammatory bowel disease
	Antiphospholipid syndrome
Reduced flow/portal hypertension:	Cirrhosis
	Hepatobiliary malignancies
Endothelial disturbance:	Local inflammation/infection
	- pancreatitis
	- cholangitis
	- diverticulitis
	- appendicitis
	- neonatal omphalitis
	Abdominal surgery
	TIPS
	Fine needle aspiration biopsy
	Glue treatment of gastric varices

vity and compensatory hypertrophy in other, well-perfused lobe.

Same model of compensatory hypertrophy is seen after surgical resection of liver segments in patients with liver tumors. At this stage portal hypertension develops, in order to support enough flow through collateral vessels into the liver. Simultaneously, systemic vascular resistance decreases with an increase in cardiac output leading to hyperdynamic circulation. In case where thrombosis is extended to the mesenteric vessels, mesenteric arches cannot drain the blood, functioning as collaterals. Meanwhile, arterial vasoconstriction as a reflex mechanism if prolonged can lead to intestinal wall infarction, one of the most detrimental events following PVT (7, 8, 9).

## EPIDEMIOLOGY

In a big epidemiological study based on autopsies, population prevalence of PVT is 1%. 28% of PVT cases had underlying cirrhosis, 23% primary and 44% secondary hepatobiliary malignancies, while 10% had major abdominal infections or inflammatory disease, and the rest 3% had myeloproliferative disease (10, 11).

Although low in general population, prevalence among cirrhotic patients ranges between 4, 4% and 15%. According to some investigators PVT is responsible for 5%-10% of overall cases of portal hypertension, up to 40% in developing countries (12, 13). The prevalence among the patients candidates for orthotopic liver transplantation ranges between 0.6%-16%, and around 6,5% among the patients with hepatocellular carcinoma (14, 15, 16).

## CLINICAL PRESENTATION

PVT presentation depends on severity of onset, therefore it is divided in two rather huge categories: acute and chronic. Sometimes it can be difficult to make a difference between the two types on the basis of symptoms. Likewise, both acute and chronic PVT may be covered by symptoms of underlying disease/condition.

Acute PVT can be presented from mild abdominal discomfort, through classical image of intestinal ischemia with colicky pain, nausea, diarrhea and fever. Depending on the extent of PVT and involved mesenteric vessels, bowel ischemia will give rise to occult stool bleeding in 50% of cases, and hematemesis, hematochezia or melena in 15% of cases. Signs of peritonitis and sepsis develop in approximately one to two thirds of patients. Sometimes ascites may appear due to venous congestion, while splenomegaly is almost always present. If the condition is not recognized and treated on time, very fast signs of intestinal perforation and shock will emerge. Physical examination will re-

veal abdominal distension, tenderness or guarding in case of intestinal perforation or peritonitis caused by inflammation. Prognosis of acute PVT depends on the grade of thrombosis and underlying disease. Consequently, mortality varies from 0-76%, among patients with mesenteric vein thrombosis from 20-50% of cases (17, 18, 19).

Chronic PVT can stay unrecognized in absence of liver disease, until the onset of variceal bleeding (hematemesis, melena) which is the first sign of PVT in 20-50% of cases. Patients with PVT without cirrhosis will present with signs of portal hypertension (bleeding, splenomegaly), but without ascites and encephalopathy, due to preserved liver function. Portal hypertensive billiopathy is present in almost 80% of patients with extrahepatic portal vein obstruction. Due to the compression of "cavernoma" or periportal varices on common bile duct, peridochal fibrosis or ischemical strictures with proximal dilation of billiary tree are frequently recognized. However, billiary stasis and choledochal stone formation will cause jaundice, cholangitis and abdominal pain in less than 30% of cases. Physical signs in chronic PVT are those of portal hypertension, liver cirrhosis and hypertensive billiopathy.

Prognosis of chronic PVT depends more on underlying disease then on PVT itself. Patients with cirrhosis and PVT according some studies have approximately hundred times greater risk of bleeding episode. Mortality is rather small and varies from less than 10% to 26% in patients with cirrhosis or malignancy (20-25).

Regarding PVT and survival in patients with cirrhosis, in one single center big retrospective cohort study (a total of 3295 patients; 4, 5% had PVT) "the presence of PVT at the time of transplantation was associated with an increased risk of liver death at 30 days" (26).

## DIAGNOSIS

Establishing diagnosis of PVT is based mainly on imaging and laboratory findings. Ultrasound (US), magnetic resonance angiography (MRA), computed tomography (CT) and catheter angiography (only in case when invasive-shunt is planned) are imaging procedures used in patients with suspected PVT.

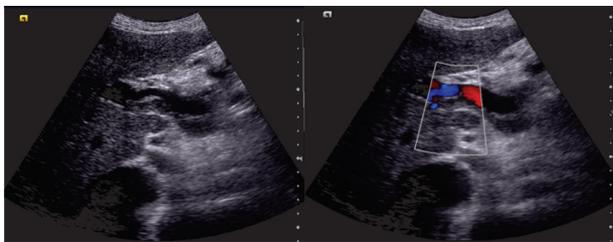
Ultrasound is the first imaging modality which is reliable with high degree of accuracy in detecting and follow-up of PVT. During the years, refinement of the method and development of duplex and color Doppler US, the sensitivity and specificity is improved, ranging from 60-100%. Contrast-enhanced US added an extra quality in distinguishing benign from malignant PVT (27, 28, 29).

Real-time or gray-scale US demonstrates portal vein, its caliber and presence of thrombus within the lumen. Apart from portal vein and liver parenchyma, appearance

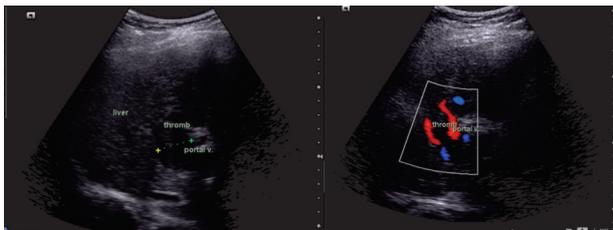
of concomitant changes during the onset of PVT, such as splenomegaly and ascites, can be revealed by gray-scale.

In acute PVT ultrasound will demonstrate dilation of portal vein diameter ( $> 13$  mm) and thrombus apparition which is virtually anechoic. Also, dynamic scan will confirm absence of respiratory variations of the vessel lumen. In chronic PVT thrombus is mainly hyperechoic, sometimes even accompanied by calcifications on the periphery (30).

Color-Doppler US must be performed in patients with suspected PVT, where gray-scale has not succeeded to visualize the thrombus inside the vessel. Sometimes in fresh thrombosis the echogenicity of the thrombus is rather the same as one of the flowing blood, in such cases (10-33%) during Doppler-imaging there will be a lack of signal in part of PV filled with thrombotic material (31). According to Yerdel Doppler US has a sensitivity of 73%, specificity of 99%, positive predictive value of 86% and negative predictive value of 98% (2). Similar results were published by Tessler et al. with sensitivity, specificity, positive predictive value and negative predictive value of 89%, 92%, 62% and 92% respectively (32). When Doppler-US has failed to discover thrombus and there is a clinical suspicion of PVT, or vice-versa: there is lack of Doppler-US in part of vessel due to sluggish flow but no clinical signs of PVT, it is necessary to use other imaging modality (Figure 1).



**Figure 1.** *Ultrasound and color Doppler findings in patient with liver cirrhosis and portal vein thrombosis. Portal thrombosis represented by the presence of echogenic material inside the portal vein on gray-scale; flow revealed on the periphery of the vessel*



**Figure 2.** *Ultrasound and color Doppler findings in patient with portal vein thrombosis. Enlarged portal vein fulfilled with echogenic material on gray-scale; Color Doppler demonstrated the presence of flow in periportal collaterals*

Likewise in both, acute or chronic PVT, Doppler-US will reveal collaterals, disturbances in flow-pattern and hepatic artery changes. Collaterals, portoportal and portosystemic are detectable even by gray-scale, but when tortuous vessels are so big mimicking abdominal masses, color-Doppler US has decisive role. Perisplenic and retroperitoneal varices are most frequently found. Not so rare finding, especially in cirrhotic patients are enlarged paraumbilical vein and superficial collaterals extending distally. In acute PVT development of collaterals can be detected several days (from 7 days to 3 weeks) after thrombus appearance, while in chronic PVT collaterals are present during the first US examination (Figure 2).

PVT can be incidental finding, when “network of small tortuous vessels” with duplex/color-Doppler features of portal flow, is discovered instead of normal portal vein. This “cavernous transformation” is often accompanied with collaterals around common bile duct, gallbladder wall, hepatoduodenal ligament and pancreas.

Disturbances in flow pattern discovered on duplex/color-Doppler are slow flow (below 15 cm/sec), reversed (hepatofugal) and sometimes bi-directional (hepatoportal and hepatofugal) flow. Flow reversal is result of portal hypertension and development of collaterals, and is usually present in certain vessels like intrahepatic portal branches, splenic vein or coronary vein.

Hepatic artery changes as enlarged vessel diameter, flow increase and increased velocity, are concomitant finding due to “arterialization” of liver blood supply.

Whether we are speaking about benign or malignant thrombosis, Doppler US is reliable in defining the thrombus nature. Finding of “pulsatile flow” (inside the thrombus) on power Doppler, is accepted criterion of malignancy, with overall sensitivity of 82,5 % and specificity of 100%. Apart from power Doppler, contrast-enhanced ultrasound has been increasingly used in differentiating benign from malignant thrombosis. Positive enhancement of portal vein thrombus, when contrast agent is applied during US examination, yielded overall sensitivity and specificity of 100% in determining malignancy (31, 32).

When different ultrasound modalities are not able to determine the nature of the thrombus, biopsy can be helpful. Although there are studies where trans-abdominal fine needle aspiration (FNA) of portal thrombus was safely performed (81, 3% sensitivity), this procedure has not been accepted widely. Recently, endoscopic-ultrasound FNA of PVT was proposed with few advantages over classic trans-abdominal approach. Better visibility of the thrombus, avoiding the collateral vessels and above all shorter needle path and lower potential of seeding malignant cells, are promising features of this technique (33, 34, 35).

## CONCLUSION

Portal vein thrombosis has small prevalence among general population (1%), but it is responsible for 5-15% of overall cases of portal hypertension. Clinical picture can vary in a broad spectrum, from mild abdominal discomfort through fulminant hepatic failure, variceal bleeding or bowel infarction with high mortality rate. Regarding possibility of fatal consequences, fast and accurate diagnosis of PVT is an implication. Recent development of imaging techniques, especially non-invasive, have allowed increasing detection of PVT. Among them, color-Doppler US (including contrast enhanced and endoscopic US), with negative predictive value of 98% is imaging procedure of choice. Of course, this technique has certain limitations; in such cases other modalities like MRA and CT-angiography are used.

## Conflict of interest

The authors declare that there are no conflicts of interest.

## Abbreviations

CT — computed tomography  
 EUS — endoscopic ultrasound  
 FNA — fine needle aspiration  
 IMV — inferior mesenteric vein  
 MRA — magnetic resonance angiography  
 PV — portal vein,  
 PVT — portal vein thrombosis  
 SV — splenic vein,  
 SMV — superior mesenteric vein  
 US — ultrasound

## Sažetak

# PORTNA VENSKA TROMBOZA – ULTRAZVUČNI IMIDŽING

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Portni venski sistem, pored glavne portne vene, uključuje i njene pritoke: gornju i donju mezenteričnu venu kao i spleničnu venu, zbog čega termin portna venska tromboza obuhvata širok spektar patoloških stanja. Obično se može prepoznati jedan ili više uzročnika, kao što su: lokalni poremećaji endotela krvnih sudova/protoka krvi ili sistemske nasledne/stečene bolesti. Portna venska tromboza može biti povezana sa benignim i malignim bolestima. Klinička prezentacija je drugačija kod akutne i hronične tromboze. Akutna tromboza može biti predstavljena u širokom opsegu, od blage abdominalne nelagodnosti do stanja crevne ishemije i tromboze koja ugrožava život. Hronična tromboza se obično prepoznaje kada nastane krvarenje zbog varikoziteta ili drugih simptoma portne hipertenzije. Brza i tačna dijagnoza ponekad znači spašavanje

života, naročito kod akutnih vaskularnih stanja. Zbog poboljšanja radioloških-vizuelnih procedura, broj pacijenata sa dijagnozom portne venske tromboze je u porastu. Kolor dopler se smatra dijagnostičkom metodom izbora u otkrivanju portne venske tromboze, sa negativnom prediktivnom vrednošću od 98%. Na osnovu velikih studija pretpostavlja se da je ukupni rizik od dobijanja portalne venske tromboze tokom života 1% u opštoj populaciji, ali je mnogo veći, od 5-15% kod pacijenata sa cirozom jetre. Ako su ispunjeni posebni ultrazvučni kriterijumi, dijagnoza portne venske tromboze je brza i neinvazivna. Metoda je pogodna za pacijenta i lekara, i što je najvažnije omogućava brz tretman i sprečava dalje pogoršanje bolesti.

**Ključne reči:** portna vena, portna venska tromboza, portna hipertenzija, ultrazvuk, kolor Dopler ultrazvuk.

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