Portal vein thrombosis: Imaging spectrum of etiologies in MDCT.

Poster No.: C-0711
Congress: ECR 2015
Type: Educational Exhibit
Authors: R. Monreal, I. Rubio Marco, I. García de Eulate, J. Saenz Banuelos, C. Sánchez Rodríguez, H. Gómez Herrero, S. Cervantes; Pamplona/ES
Keywords: Embolism / Thrombosis, Contrast agent-intravenous, CT, Veins / Vena cava, Liver, Abdomen
DOI: 10.1594/ecr2015/C-0711

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method is strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Learning objectives

(1) To review the MDCT features of portal vein thrombosis

(2) To review some of the clinical situations associated to portal vein thrombosis with images of cases studied in our institution

Background

The blood flow of the liver implies around 25-30% of the cardiac output, which is about 1500ml/min. The liver receives this blood supply from two different sources: the portal vein and the hepatic artery. The former delivers 75% of the blood volume and the latter supplies only the remaining 25%, however this blood is more oxygenated.

In most patients, the portal vein is formed as a result of the confluence of the superior mesenteric vein and the splenic vein behind the neck of the pancreas. This vessel is further on divided into the left and right branches when reaching the hepatic hilium. The left portal branch vascularises the I, II, III, IV hepatic segments, and the right branch irrigates the V and VIII segments (anterior division) and also the VI-VII segments (posterior division). Apart from this standard portal vein anatomy, other branching patterns can be described, most of them asymptomatic but important to consider within possible future surgical interventions. (Fig. 1 on page 2)

Portal venous thrombosis (PVT) is found in 1% of the autopsies, and is a major cause for non-cirrhotic portal hypertension. The clinical presentation of the disease is usually nonspecific, and variable depending on the moment of the evolution. In acute thrombosis, the patient can suffer from abdominal pain, fever, malaise, and this initial episode may even go unnoticed. Some patients are diagnosed in a chronic stage, presenting symptoms derived from complications of portal hypertension such as trombocitopenia, esplenomegaly, gastrointestinal hemorrhage (due to varicose veins) and jaundice.

The consequences of PVT are mainly related to the extension of the thrombus. The spread of a thrombus into the mesenteric vein and mesenteric arches leads to intestinal ischemia, for which emergency surgery can be required.

Images for this section:
Fig. 1: The four most common branching patterns of the intrahepatic portal vein: A) Normal branching pattern. B) Trifurcation of the main portal vein. C) Right posterior portal vein as first branch of main portal vein. D) The segment VI branch as a separate branch of the right portal vein. (LPV: Left portal vein. RPPV: Right posterior portal vein. RAPV: Right anterior portal vein)
Findings and procedure details

If nonspecific symptoms of PVT are manifested, the diagnosis is based on imaging techniques. Because of its simplicity, the Color Doppler Ultrasound (CDU) is considered the first line in the diagnosis of PVT, with a sensitivity of around 70-90%. Nevertheless, venous phase Computer Tomography (CT) is the best imaging modality to diagnose the disease, allowing an accurate definition of the extension of the thrombosis, determining its chronicity, and elucidating the nature of the thrombus. An alternative diagnosing technique is the Magnetic Resonance Angiography (MRA), which is the most sensitive modality (100%) since it is able to show the presence of portal venous thrombosis. However, it is not always available.

It should be taken into consideration that in both, CT and in Magnetic Resonance (MR) images, there can be artefacts related to laminar flow that can mimic PVT. This "pseudothrombosis", often observed during arterial-dominant phase, is a consequence of the incomplete mixture of opacified blood from the splenic vein and unenhanced blood from the superior mesenteric return. This apparent filling defect is resolved on later phases, demonstrating an homogeneous opacification of the portal vein.

In CT we can find some direct and indirect signs of PVT. Within the indirect signs, parenchymal alteration of the liver may be observed, in which a deficit in portal blood flow occurs (Fig. 2 on page 8). This implies hepatic glycogen depletion and an increased hepatocyte fat that results in a decreased hepatic attenuation. Moreover, two phenomena are described related to the bloodflow of the segments affected by the thrombosis: a decreased enhancement in portal venous phase (due to alteration in local portal perfusion), and an enhancement in the hepatic arterial phase (because of the compensatory increase in the arterial flow). Other indirect signs such as the existence of portosystemic collateral vessels, arteriportal shunts and cavernomatous transformation of the portal vein can also exist. Among the direct signs of PVT, we can find a hypodense filling defect in the portal vein lumen with partial or complete occlusion on contrast enhanced scans, which can extend to other vessels (Fig. 2 on page 8). In complete thrombosis it is possible to distinguish a peripheral rim enhancement that is likely due to flow through dilated vasa vasorum. However, in partial thrombosis a similar image can be observed because of the pass of the contrast around the thrombus that occludes the lumen partially.

Some CT findings help differentiating acute from chronic thrombosis. For example, an intraluminal hyperdensity in the portal vein in plain scans suggests acute thrombosis. By contrast, in chronic PVT, collateral circulation is developed in order to alleviate the hemodynamic effects of the obstruction. As a result, varicose veins, a recanalized umbilical vein, and even a cavernous transformation of the portal vein (formation of tortuous collaterals vessels bypassing the obstructive area) (Fig. 3 on page 9, Fig. 4 on page 10, Fig. 5 on page 10, Fig. 6 on page 11) may be found. The major
collateral veins are biliary (cystic and paraahepatic veins) and gastric (left and right gastric veins)

Information about the etiology of the thrombosis is also provided by CT images. In around 70% of patients with PVT a cause is identified, although in 15% of them several factors coexist, supporting the theory of the PVT as a multifactorial disease.

The principal causes are:

-Cirrhosis (Fig. 7 on page 11): One of the most common causes of PVT is the decreased portal flow due to the increased hepatic resistance in cirrhotic livers. In some cases, the thrombus is resolved spontaneously and the patient remains asymptomatic. However, in more than half of the cases, complications such as gastrointestinal bleeding may take place. If PVT is present, the hepatic artery becomes enlarged and, as a result, the major vascular supply to the hepatic parenchyma.

-Neoplastic disease: It is, alongside liver cirrhosis, the most common disorder associated to PVT.

Portal vein involvement by neoplasms is possible by extrinsic vein occlusion, usually originated by pancreatic cancer, gallbladder cancer and enlarged metastatic lymph nodes, but it can also be caused directly from the primary lesion as it occurs with hepatocellular carcinoma (HCC).

HCC is the cancer most frequently associated with PVT (Fig. 8 on page 12, Fig. 9 on page 12). In primary hepatic tumours, the portal thrombosis can be caused directly by the tumour mass and it is also indicative of an advanced tumoural stage. Elucidating the nature of the thrombus is extremely relevant in patients with HCC since tumoural vascular invasion substantially worsens prognosis. It constitutes a pathway for metastatic spread, and may rely on the effect that malignant portal thrombosis has on therapeutic strategies. Portal invasion in HCC implies systemic therapy such as sorafenib if the hepatic function is preserved.

Differentiation between a bland thrombus and a tumoural thrombus is not always possible. However, enhancement within a thrombus during the hepatic arterial phase suggests the presence of portal vein invasion and tumoural thrombosis. Another sign of malignant thrombosis is the identification of the main portal vein greater than 23 mm in diameter due to distension of the venous lumen.

Other techniques may be useful in this setting. Doppler-US may diagnose malignant thrombus by detecting colour on it. Contrast-enhanced US (CEUS) is more powerful than Doppler US and US guided biopsy of the thrombus in diagnosing tumoural thrombus, with sensitivity values of 88%. It is able to detect enhancement on the thrombus.
18F-FDG PET-TC has been recently reported be a promising method for distinguishing between bland and neoplastic thrombus using a cut-off value of SUV maximum > 3.35.

Shrinkage of the thrombus and/or recanalization of the vessel during follow-up are considered criteria of benign nature of the thrombus. On the other hand, enlargement of the thrombus, disruption of the vessel wall and parenchymal infiltration confirm the malignant nature of the thrombus.

Tumoural PVT has also been reported to be associated with various other cancers such as pancreatic cancer (Fig. 3 on page 9, Fig. 10 on page 12, Fig. 11 on page 13), gastric cancer, colorectal cancer (Fig. 12 on page 13, Fig. 13 on page 14) and cholangiocarcinoma (Fig. 14 on page 14) among others.

**Hypercoagulable states:**

Myeloproliferative disorders are often associated with thrombosis, and PVT may be the first sign of the disease due to the masking of the typical blood characteristics (polyglobuly, leukocytosis, thrombocytosis) caused by the haemodilution and hypersplenism.

Other diseases are associated with portal thrombosis such as acquired thrombophilic states (antiphospholipid syndrome, paroxysmal nocturnal hemoglobinuria or hyperhomocysteinemia) but also several inherited thrombophilias (protein C or S deficiency or factor V Leiden thrombophilia)

**Inflammatory and Infectious Diseases:**

Pylephlebitis is a septic thrombophlebitis of the portal venous system (Fig. 6 on page 11, Fig. 15 on page 15). It is usually a side effect due to an infection in the region drained by the portal system or in the portal vein contiguous structures. Nevertheless, there are some patients in which the origin cannot be identified. Diverticulitis is the most common cause of pylephlebitis, but there are many others, including appendicitis, pelvic infections, biliary diseases, inflammatory bowel (Fig. 16 on page 15) disease and necrotizing pancreatitis (Fig. 17 on page 16). Pylephlebitis begins with thrombophlebitis of the small veins that drain the infected area; subsequently, it extends to larger veins and it finally leads to septic thrombophlebitis of the mesenteric vein and it can even affect the portal vein. This septic thrombophlebitis of the portal venous system can be associated with liver abscesses (the majority of them situated in the right hepatic lobe due to the greater flow of the right portal vein branch). Such abscesses may result from the direct extension of the bacteria from the biliary system in patients with cholangitis or they may arrive from a distant focus, for example from the gastrointestinal tract as it occurs in appendicitis and diverticulitis.
Liver abscesses occasionally mimic the appearance of malignant tumours on CT, since they may have a solid appearance with venous thrombosis associated. Hence, the differential diagnosis between liver abscesses or hepatocellular carcinoma should be made, and the clinical symptoms, the evolution of the lesions and CT images can help the diagnosis. The thrombosis associated with the abscesses are seen as a non-enhancing hypoattenuating structures without distension of the lumen, different from the malignant thrombosis, which shows substantial enhancement with neovascularity and distension of the venous lumen.

**Damage of the splenoportal axis: transplantation, splenectomy and other surgeries** *(Fig. 18 on page 17, Fig. 4 on page 10).*

Portal vein abnormalities as thrombosis develop in about 1-3% of orthotopic liver transplantations. Some predisposing causes have been described, such as deficient surgical techniques, mismatches in size between donor and recipient portal veins, previous portal vein thrombosis or calcification and hypercoagulable states.

PVT is no longer considered an absolute contraindication to liver transplantation, as there are surgical techniques for such cases. However, it remains as such for diffuse chronic thrombosis of the portal vein and the superior mesenteric vein.

Splenectomy is another case of surgery in which PVT can appears as a complication in a range from 0.5% to 22% of the cases. It is most often seen following myeloproliferative disorders and almost never after trauma. Another predictor factor of portal thrombosis in this type of surgery is the spleen weight, since bigger spleens are related with more risk of PVT. In recent years, the laparoscopic technique has become the election procedure for splenectomy, even if it increases the risk of developing PVT as it reduces the blood flow in the portal system due to the pneumoperitoneum.

**Chemotherapy and Radiotherapy** *(Fig. 19 on page 17)*:

Chemotherapy damages the organs as a result of microvascular injury and endothelial damage, venous thrombosis, and tissue fibrosis. Arterial and venous thrombosis have been described during antineoplastic chemotherapy, and oxaliplatin and bortezomib have been associated more specifically with PVT. Radiotherapy is also related to vascular damage, usually involving the intima in acute setting and the entire vessel in chronic stage leading to fibrosis. In the liver the radiation produces a vennooclusive disease in the small branches of the hepatic and portal veins.

**Aneurysm of portal vein:**

It is a rare vascular anomaly that may be congenital or acquired, as a consequence of portal hypertension, pancreatitis and/or trauma. The most common locations are the
splenomesenteric confluence, main portal veins and intrahepatic portal vein branches at bifurcation sites. It is diagnosed if the diameter of the vessel is significantly larger at a given point than in the remainder of the vessel, especially if it is saccular or fusiform.

**-Others: Pregnancy and oral contraceptives pills (OCP's)**.

The majority of studies suggest that PVT does not take place in a pregnant woman if there is no other prothrombotic factor associated. The prevalence of PVT in women who take OCP's is similar to the one of the women who don't. Therefore, it is considered that these two factors isolated are not enough to develop PVT, although they can contribute as enabling factors.

**-Idiopathic:**

Approximately in 30% of patients with PTV the cause is not identified and remains as idiopathic.

**Images for this section:**
Fig. 2: CT findings of portal vein thrombosis A) Parenchymal signal alterations. B) Filling defect in the portal lumen. C) Esophageal varices (arrow). D) Cavernous transformation of portal vein.

Fig. 3: Patient with pancreatic cancer A) Portal vein thrombosis. Parenchimal signal alteration is also present. B) Cavernous transformation of portal vein.
Fig. 4: Patient with antecedent of portal vein thrombosis in the postoperative of sleeve gastrectomy and duodeno-jejunal bypass. Cavernous transformation of portal vein was developed.

Fig. 5: Cavernous transformation of portal vein.
Fig. 6: Patient with Streptococcus Anginosus bacteremia. A) Portal vein thrombosis. B) Cavernous transformation of the portal vein two months after the acute thrombosis.
**Fig. 7:** Portal vein thrombosis (arrow) and parenchymal signal alterations in patient with cirrhosis.

![Image of portal vein thrombosis and cirrhosis](image1)

**Fig. 8:** A) Liver lesions with nodular enhancement which corresponded to multifocal hepatocellular carcinoma (arrowheads) B) Filling defect in the portal vein lumen (arrow)

![Image of liver lesions and portal vein](image2)

**Fig. 9:** A) Focal parenchymal hypodensity and nodular enhancement corresponding to hepatocellular carcinoma (arrowhead). B) Thrombosis of a right portal vein branch (arrow).

![Image of focal hypodensity and thrombosis](image3)
Fig. 10: Patient with metastatic pancreatic cancer A) Thombosis of the main portal vein (arrow). Hydatid Cyst(*). B) Liver metastasis(arrowhead).

Fig. 11: Pancreatic head tumour with portal thrombosis and liver parenchymal signal alteration.
Fig. 12: Patient with colon cancer, liver metastases and portal thrombosis.

Fig. 13: Thrombosis of the right portal vein branch (arrow) and liver metastases(*) in a patient with colon cancer.
Fig. 14: Tumoral thrombosis (arrow) in a patient with cholangiocarcinoma.

Fig. 15: Patient with cholecystitis. A) Ecogenic clot within the lumen of a portal vein branch. B) Wall thickening of the gallbladder and lithiasis.
Fig. 16: Patient with pylephlebitis secondary to E.Coli enterocolitis: Thrombosis of portal vein branches (arrow) and parenchymal signal alteration.
Fig. 17: Patient with acute pancreatitis. A) Portal vein thrombosis. B) Splenic vein thrombosis associated.

Fig. 18: Patient with liver transplantation A) Portal vein thrombosis and ascites. B) Esophageal varices.

Fig. 19: Patient with gastric adenocarcinoma in treatment with oxaliplatin and capecitabine. Thrombosis of left portal vein branches and parenchymal signal alterations.
Conclusion

Liver cirrhosis, neoplastic disease, inflammatory/infectious intraabdominal processes, hipercoagulable states, surgical damage of the spleno-portal axis, chemo-radiotherapy and some anatomic variants predispose to PVT. Radiologist should be aware of these situations in order to detect it when evaluating CT images. The disease course depends on the degree of thrombosis, extent of collateralization and duration of the thrombus. Therefore, it is important to define these features on imaging techniques.

Personal information

References


- Ze-Zhou Song, Min Huang, Tian-An Jiang, Qi-Yu Zhao, Lei Yao, Yun Mou et al. Diagnosis of portal vein thrombosis discontinued with liver tumors in patients with liver


-Zafer Koc, Levent Oguzkurt and Serife Ulusan. Portal Venous System Aneurysms: Imaging, Clinical Findings, and a Possible New Etiologic Factor. 2007; 189 (5)


