Acute portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management

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INTRODUCTION

The portal vein is formed by the confluence of the splenic and superior mesenteric veins, which drain the spleen and small intestine, respectively (figure 1). Occlusion of the portal vein by thrombus (portal vein thrombosis [PVT]) typically occurs in patients with cirrhosis and/or prothrombotic disorders (table 1). Patients with acute PVT have the sudden onset of portal venous occlusion due to thrombus. The occlusion may be complete or partial. In addition to involving the portal vein, the clot may also involve the mesenteric veins or the splenic vein. Patients with acute PVT have not yet developed features of chronic PVT, such as collateral circulation (eg, cavernous portal transformation) or portal hypertension. If it is not known when the clot developed, but the patient does not have features of chronic PVT, the PVT can be referred to as being "recent" [1]. Patients with recent PVT are managed the same as those with acute PVT.

This topic will review the clinical manifestations, diagnosis, and management of acute PVT. The epidemiology and pathogenesis of PVT, as well as the approach to patients with chronic PVT are discussed elsewhere. (See "Epidemiology and pathogenesis of portal vein thrombosis in adults" and "Chronic portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management".)

The approach to patients with PVT has also been reviewed in society guidelines [2-4]. The discussion that follows is generally consistent with those guidelines.

CLINICAL MANIFESTATIONS

The clinical manifestations of acute portal vein thrombosis (PVT) depend on the extent of the obstruction and the speed of its development.

Symptoms — Acute PVT may be clinically silent and diagnosed during a radiologic examination for other reasons (such as acute pancreatitis) (image 1). Other patients may have abdominal pain that develops suddenly or progresses over a few days [5]. Patients may also report fever and dyspeptic symptoms. Patients with cirrhosis may present with variceal bleeding. The presence of spiking fevers, chills, and a painful liver is suggestive of septic PVT (acute pylephlebitis). In addition to symptoms related to the PVT, patients may also have symptoms related to conditions that predispose to the development of PVT, such as acute pancreatitis. (See "Pylephlebitis", section on 'Clinical manifestations'.)

If the superior mesenteric vein is occluded, patients may have colicky abdominal pain and diarrhea [5]. If the proximal mesenteric venous arches are involved, ischemia and ultimately infarction may develop. In such patients, the abdominal pain may radiate to the back, persist beyond five to seven days, be associated with abdominal distension from ascites, and be associated with bloody diarrhea. For some patients with underlying inflammatory bowel disease, the clinical presentation of acute PVT (eg, abdominal pain, diarrhea) may mimic a disease flare.

Physical examination — Most patients with acute PVT do not have abnormal findings on physical examination. However, some patients with acute PVT may have an ileus and thus have abdominal distension without other signs of intestinal obstruction [5]. Patients typically do not have guarding unless the PVT is the result of an intra-abdominal inflammatory process or if intestinal infarction has occurred. Patients with infarction may also have signs of ascites on examination (eg, a fluid wave). Patients with cirrhosis may have stigmata of chronic liver disease, such as palmar erythema, or signs of hepatic encephalopathy. (See "Cirrhosis in adults: Etiologies, clinical manifestations, and diagnosis", section on 'Physical examination' and "Hepatic encephalopathy in adults: Clinical manifestations and diagnosis", section on 'Clinical manifestations' and "Overview of intestinal ischemia in adults", section on 'Clinical features'.)

Laboratory testing — Laboratory testing may reveal elevations in plasma levels of acute phase reactants (sometimes markedly elevated). Liver tests are typically normal because hepatic arterial blood flow compensates for decreased portal inflow, although a transient, moderate increase in serum aminotransferases is seen in some patients. In patients with bowel ischemia, laboratory testing may reveal metabolic acidosis, signs of renal or respiratory failure, a

leukocytosis, and an elevated hematocrit due to hemoconcentration [5]. In patients with septic PVT, blood cultures are often positive for *Bacteroides fragilis* or *Escherichia coli,* although other pathogens have also been cultured. In patients with cirrhosis, laboratory testing may reveal an elevated bilirubin, low platelet count, prolonged international normalized ratio (INR), or renal insufficiency. (See "Pylephlebitis", section on 'Microbiology' and "Cirrhosis in adults: Etiologies, clinical manifestations, and diagnosis", section on 'Laboratory findings' and "Overview of intestinal ischemia in adults", section on 'Laboratory studies'.)

Abdominal imaging — Patients with acute PVT who undergo abdominal imaging have evidence of portal venous occlusion and may have findings suggestive of intestinal ischemia. In addition, imaging may reveal an underlying focus of infection or multiple, small liver abscesses in patients with septic PVT. (See 'Diagnosis' below and "Pylephlebitis", section on 'Imaging studies'.)

DIAGNOSIS

Acute portal vein thrombosis (PVT) is diagnosed with abdominal imaging that demonstrates portal venous occlusion without the radiographic findings of chronic PVT (eg, cavernous portal transformation). (See "Chronic portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management", section on 'Diagnosis'.)

In patients with suspected acute PVT, we typically obtain a contrast-enhanced abdominal computed tomography (CT) to confirm the diagnosis, evaluate for predisposing conditions (eg, intra-abdominal infection), assess the extent of the thrombosis and the anatomy of collaterals, and detect evidence of intestinal infarction (table 1). (See "Epidemiology and pathogenesis of portal vein thrombosis in adults", section on 'Epidemiology'.)

If suspicion for PVT is not high (eg, the patient does not have cirrhosis or other risk factors for thrombosis), a Doppler ultrasound is a reasonable initial approach. If the ultrasound suggests acute PVT, an abdominal CT can then be obtained. (See "Epidemiology and pathogenesis of portal vein thrombosis in adults", section on 'Epidemiology'.)

Abdominal magnetic resonance imaging (MRI) is an alternative in patients who cannot undergo CT or in whom radiation exposure from CT is a concern. If CT and MRI are contraindicated or not available, ultrasound with Doppler is an alternative, although ultrasound may not detect predisposing conditions or ischemia. Finally, a diagnosis of PVT can be made with portal venography or superior mesenteric angiography, but angiography is invasive and is generally not required [6].

Abdominal CT or MRI — In patients with acute PVT, a CT scan without contrast may show hyperattenuating material in the portal vein, though contrast-enhanced imaging is often needed to make the diagnosis because the density of the thrombus may be difficult to differentiate from the vessel wall [7-9]. Imaging after injecting intravenous contrast may reveal lack of luminal enhancement, increased hepatic enhancement in the arterial phase, and decreased hepatic enhancement in the portal phase [10,11]. On MRI angiography, PVT appears as a filling defect that partially or completely occludes the vessel lumen in the portal venous phase [12]. Data are lacking on the sensitivity and specificity of CT and MRI for detecting PVT. In a study with 36 patients, MRI had a sensitivity of 100 percent and a specificity of 99 percent for detecting PVT [13].

Abdominal ultrasound with Doppler — Abdominal ultrasonography in patients with acute PVT may show hyperechoic material within the portal vein, with distension of the portal vein and its tributaries [14]. The portal vein diameter is typically greater than 13 to 55 mm in diameter and does not vary in diameter during respiration [7]. Because a thrombus may not be visualized on ultrasound in up to a third of patients with PVT, Doppler imaging should also be used. In patients with PVT, Doppler imaging will show the absence of flow in some or all of the vessel lumen. Ultrasonography with Doppler imaging has been estimated to be 89 to 93 percent sensitive and 92 to 93 percent specific for diagnosing PVT [10,15]. The accuracy of Doppler ultrasound depends in part on the expertise of the ultrasonographer and whether PVT was suspected prior to the examination [5,16]. The sensitivity of Doppler ultrasound for PVT may be increased by adding contrast-enhancing agents [17].

Identification of predisposing conditions — Patients with PVT who do not have cirrhosis or who have compensated (Child class A or B) cirrhosis should be evaluated for conditions that may have predisposed to thrombosis, such as prothrombotic states. Because PVT is common in patients with decompensated cirrhosis, it is reasonable not to pursue an evaluation for other predisposing conditions in such patients. The approach to the evaluation of patients with established venous thrombosis is discussed in detail elsewhere. (See "Evaluating adult patients with established venous thromboembolism for acquired and inherited risk factors" and "Epidemiology and pathogenesis of portal vein thrombosis in adults", section on 'Epidemiology'.)

DIFFERENTIAL DIAGNOSIS

The primary considerations in the differential diagnosis of acute portal vein thrombosis (PVT) are invasion of the portal vein by an abdominal malignancy (most frequently hepatocellular carcinoma) or, less often, constriction of the portal vein within a tumor (typically pancreatic

cancer or cholangiocarcinoma) [5]. In cases of tumor invasion or external constriction, a thrombus within the portal vein may develop as a secondary event (malignant PVT). Imaging characteristics can help differentiate benign PVT from malignant PVT. Differentiating benign from malignant PVT is particularly important in patients with cirrhosis and hepatocellular carcinoma who are being considered for liver transplantation since malignant PVT is a contraindication to liver transplantation.

Findings that suggest a malignant PVT include [18,19]:

- Elevated alpha fetoprotein
- Portal vein diameter >23 mm
- Enhancement of endoluminal material during the arterial phase of contrast injection
- Arterial-like pulsatile flow seen with Doppler ultrasound
- Disruption of the vessel walls or tumor encroaching on the portal vein

MANAGEMENT

The primary management of acute portal vein thrombosis (PVT) is anticoagulation and, when possible, treatment of predisposing conditions (table 1). The goal of anticoagulation is to prevent extension of the clot and to allow for recanalization so that intestinal infarction and portal hypertension do not develop. Unlike chronic PVT, where the role of anticoagulation in patients with cirrhosis is unclear, studies suggest anticoagulation for acute PVT is beneficial for patients with cirrhosis. However, because patients with cirrhosis may have esophageal varices, we typically screen for varices prior to initiating anticoagulation. (See 'Efficacy of anticoagulation' below and "Chronic portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management", section on 'Anticoagulation'.)

Patients should be started on low molecular weight heparin to achieve rapid anticoagulation, with a switch to an oral anticoagulant (warfarin or possibly a direct-acting oral anticoagulant [DOAC]) once the patient's condition has stabilized and no invasive procedures are planned (if warfarin is used, our goal international normalized ratio [INR] is 2 to 3) [5]. The role and safety of anticoagulation in patients with cirrhosis and severe liver dysfunction is not well established, but studies indicate that the outcomes for patients with acute PVT are not worse compared with patients without thrombosis [20].

Data from mostly observational studies have indicated that direct oral anticoagulants may also be used to treat PVT successfully [21-24]. However, for patients with cirrhosis, there are reports suggesting decreased efficacy of rivaroxaban and apixaban [25]. In addition, one small

retrospective series suggested that the bleeding risks with these agents are comparable to those seen with traditional anticoagulation [26]. More data are needed to make evidence-based recommendations on the use of these agents in patients with acute PVT, particularly for those with cirrhosis.

Treatment is generally recommended for at least three to six months, though long-term treatment is recommended for most patients with permanent thrombotic risk factors that cannot be corrected [2,4]. Long-term therapy may also be appropriate for patients with acute PVT that extends into the mesenteric veins, given the risk of intestinal infarction in such patients. In patients with cirrhosis, non-warfarin-based therapy may be preferable since the INR may not reflect the patient's level of anticoagulation. (See "Warfarin and other VKAs: Dosing and adverse effects" and "Direct oral anticoagulants (DOACs) and parenteral direct-acting anticoagulants: Dosing and adverse effects" and "Hemostatic abnormalities in patients with liver disease", section on 'Portal vein thrombosis (PVT)'.)

In addition to anticoagulation, patients with septic PVT require antibiotic therapy. Patients who develop infarction in the setting of acute PVT require surgical exploration. Failure to do so is associated with high mortality rates. (See "Pylephlebitis", section on 'Treatment'.)

Efficacy of anticoagulation — The use of anticoagulation in patients with acute PVT is supported by studies that suggest it leads to increased recanalization rates of the portal vein compared with no treatment. Recanalization is important because failure to restore venous drainage of the small intestine puts patients at risk for intestinal ischemia and infarction, as well as complications from chronic PVT. In addition, the use of anticoagulation for acute PVT is indirectly supported by numerous trials that show a benefit of anticoagulation patients with deep vein thrombosis. (See "Chronic portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management" and "Overview of the treatment of proximal and distal lower extremity deep vein thrombosis (DVT)".)

Limited data suggest that patients diagnosed with acute PVT are unlikely to spontaneously recanalize their portal vein [1,27,28]. However, retrospective studies have found that if patients are treated with anticoagulation, at least partial recanalization of the portal vein occurs in 63 to 93 percent of patients, with complete recanalization in 34 to 45 percent [1,27-30]. As an example, in a series of 95 patients with acute PVT followed for a median of 234 days, anticoagulation restored patency of the portal vein in 39 percent of patients. Failure to recanalize the portal vein was independently associated with the presence of ascites or an occluded splenic vein [27].

Anticoagulation also appears to be beneficial for patients with acute PVT in the setting of cirrhosis. In a series of 55 patients with cirrhosis and acute or recent PVT who received anticoagulation, 33 (60 percent) achieved partial or complete recanalization, with complete recanalization occurring in 25 (45 percent) [31]. (See "Hemostatic abnormalities in patients with liver disease", section on 'Portal vein thrombosis (PVT)'.)

Duration of anticoagulation — Our approach is to treat patients with anticoagulation for six months provided there is no indication for long-term anticoagulation, such as a permanent thrombotic risk factor that cannot be corrected or a thrombus that extends into the mesenteric veins. If recanalization occurs, it typically does so within six months of starting anticoagulation [29,32]. In patients who do not have an indication for life-long therapy, such as an underlying hypercoagulable state, it is not clear whether to continue anticoagulation beyond six months if complete recanalization has not occurred by that time. Our practice is to discontinue anticoagulation after six months in such patients. We continue to monitor patients for recurrence of symptoms (eg, abdominal pain), and we repeat imaging in three months after discontinuing anticoagulation.

Complications of anticoagulation — Anticoagulation may lead to bleeding in some patients. Studies in patients with acute PVT who do not have cirrhosis have reported bleeding rates of 0 to 6 percent (often minor bleeding, but sometimes severe) [1,28-30]. The risk may be higher in patients with cirrhosis. In a study of 55 patients with cirrhosis and acute PVT who were treated with anticoagulation, five (9 percent) developed bleeding [31]. Bleeding was more common among patients with a platelet count less than 50,000. (See "Management of warfarin-associated bleeding or supratherapeutic INR", section on 'Mitigating bleeding risk'.)

Alternatives to anticoagulation — Several case reports have documented successful lysis of acute PVT using streptokinase or tissue plasminogen activator administered locally by a catheter passed via a transjugular transhepatic or percutaneous transhepatic route [33-38]. In a systematic review that included 71 patients undergoing thrombolysis (with or without thrombectomy), recanalization of the portal vein was complete in 29 patients (41 percent), partial in 32 (45 percent), and absent in 10 (14 percent) [37]. However, the benefit of this approach is uncertain, since the natural history of this condition is not well defined. In addition, serious complications have been reported including significant bleeding and death [37]. In one study, 12 of 20 patients (60 percent) had major complications [38].

Another alternative to anticoagulation is surgical thrombectomy. It is typically reserved for patients who are undergoing surgery for intestinal infarction. In such patients, surgical thrombectomy can be carried out during the laparotomy.

PROGNOSIS

If treatment for acute portal vein thrombosis (PVT) is initiated prior to the onset of intestinal infarction, the patient's prognosis is good [1,39-43]. Abdominal pain and systemic inflammatory response syndrome will start to subside within hours to days after the initiation of anticoagulation and intestinal infarction is prevented if the superior mesenteric vein remains patent or recanalizes. In addition, portal hypertension is prevented if the portal trunk and at least one of its branches remains patent or is recanalized. In a prospective study of 105 patients with acute PVT, 44 percent achieved patency of portal venous flow, with a mortality rate of 2 percent at one year [44].

Untreated, patients with acute PVT may develop intestinal infarction, though the frequency with which this occurs is unclear. Infarction leads to intestinal perforation, peritonitis, shock, multiorgan failure, and death if treatment is not provided promptly [45].

Acute PVT may become chronic among patients who do not develop intestinal ischemia and who do not receive treatment (eg, patients with asymptomatic acute PVT). Limited data suggest that patients diagnosed with acute PVT who do not receive treatment with anticoagulation therapy rarely have spontaneous recanalization of the portal vein [1,27,28]. However, it is not known how often patients with acute PVT who do not come to medical attention spontaneously recanalize.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Hepatic, portal, and splenic vein thrombosis".)

SUMMARY AND RECOMMENDATIONS

- **Background** – Patients with acute portal vein thrombosis (PVT) have the sudden onset of portal venous occlusion due to thrombus. (See 'Introduction' above.)
- **Clinical manifestations** – Acute PVT may be clinically silent or patients may report symptoms such as abdominal pain that develops suddenly or progresses over a few days. Patients with cirrhosis may present with variceal bleeding. The presence of spiking fevers, chills, and a painful liver is suggestive of septic PVT (acute pylephlebitis). Intestinal

infarction should be considered in patients who have abdominal pain that radiates to the back, persists beyond five to seven days, is associated with abdominal distension, or is associated with bloody diarrhea. (See 'Clinical manifestations' above and "Pylephlebitis".)

- **Diagnostic evaluation** – In patients with suspected acute PVT, our initial approach is to obtain a contrast-enhanced abdominal computed tomographic (CT) scan or Doppler ultrasound. In addition to confirming the diagnosis of acute PVT, CT can identify potential predisposing conditions (eg, intra-abdominal infection), assess the extent of the thrombosis, and detect evidence of intestinal infarction. (See 'Diagnosis' above.)

  Patients with PVT who do not have cirrhosis or who have compensated (Child A or B) cirrhosis should be evaluated for conditions that may have predisposed to the development of the clot, such as prothrombotic states. Because PVT is common in patients with decompensated cirrhosis, it is reasonable not to pursue an evaluation in such patients unless they also have a history of thrombotic episodes at another location or other clinical clues suggestive of a hypercoagulable state. (See 'Identification of predisposing conditions' above.)

- **Management** – We recommend anticoagulation therapy for patients with acute portal vein thrombosis rather than expectant management, provided that there is no excessive bleeding risk (eg, large esophageal varices) (**Grade 1B**). In addition, when possible, predisposing conditions should be treated. Patients should be started on low molecular weight heparin to achieve rapid anticoagulation, with a switch to an oral anticoagulant once the patient's condition has stabilized and no invasive procedures are planned (if warfarin is used, our goal international normalized ratio is 2 to 3). In patients with cirrhosis, non-warfarin-based therapy may be preferable since the INR may not reflect the patient's level of anticoagulation. (See 'Management' above.)

  For patients with transient or correctable thrombotic risk factors (eg, pancreatitis) or in whom no thrombotic risk factor is identified, we suggest six months of anticoagulation rather than long-term anticoagulation (**Grade 2C**). For patients with permanent thrombotic risk factors that cannot be corrected, we suggest long-term anticoagulation rather than six months anticoagulation (**Grade 2C**). In addition, long-term therapy may also be appropriate for patients with acute PVT that extends into the mesenteric veins, given the risk of intestinal infarction in such patients. (See 'Duration of anticoagulation' above.)

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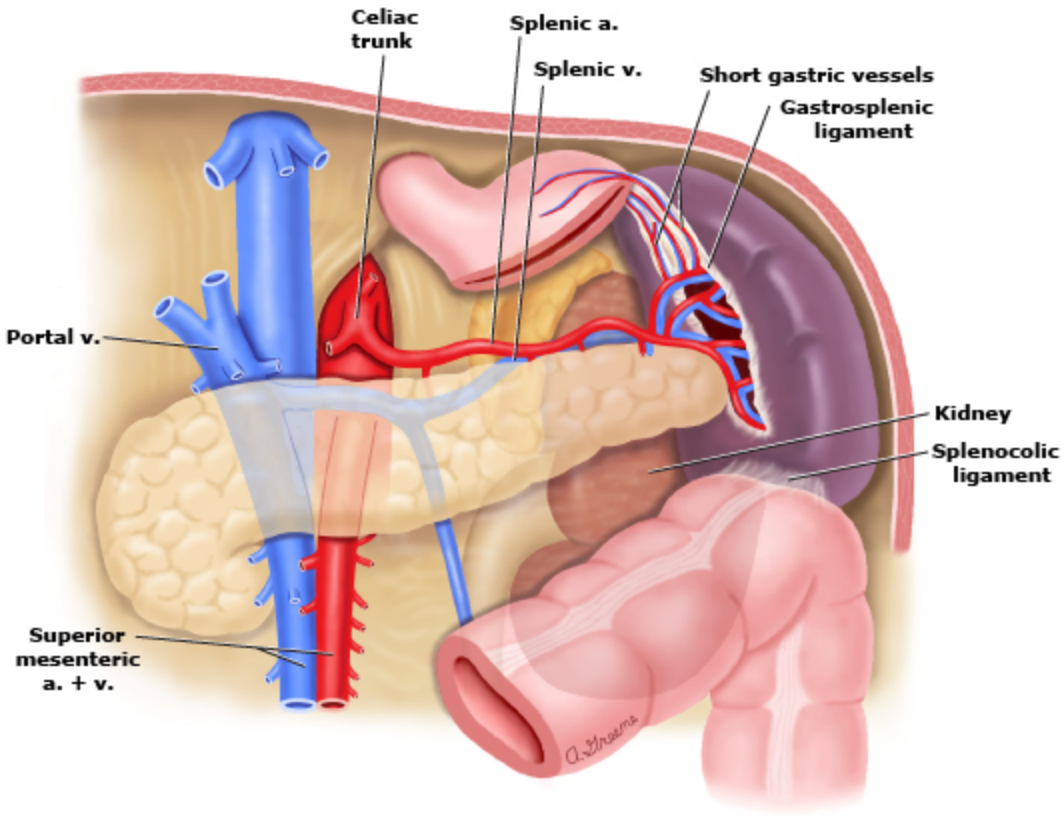
GRAPHICS

Vascular supply of the spleen



The splenic artery is a branch of the celiac trunk. It traverses along the superior margin of the pancreas toward the spleen, branching to form up to six major arteries before entering the spleen. The splenic vein joins the superior mesenteric vein to form the portal vein.

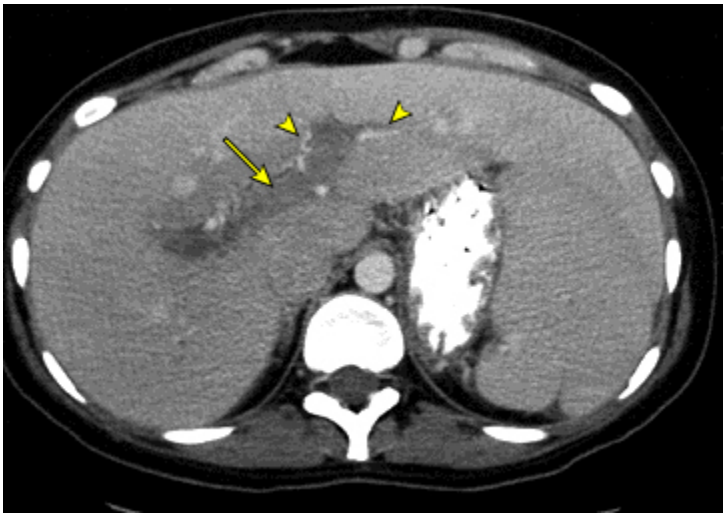
Graphic 68680 Version 4.0

Causes of portal vein thrombosis

Abdominal sepsis
Abdominal surgery
Behçet's syndrome
Cirrhosis
Collagen vascular diseases (eg, lupus)
Compression or invasion of the portal vein by tumor (eg, pancreatic cancer)
Endoscopic sclerotherapy
Hepatocellular carcinoma
Inflammatory bowel disease
Inherited thrombophilias
Myeloproliferative syndromes
Omphalitis
Oral contraceptives
Pancreatic islet cell transplantation
Pancreatitis
Paroxysmal nocturnal hemoglobinuria
Pregnancy
Retroperitoneal fibrosis
Transjugular intrahepatic portosystemic shunt
Trauma

Graphic 73799 Version 5.0

Acute portal vein thrombus



Contrast-enhanced CT scan of the liver in a 27-year-old woman with a systemic coagulation disorder demonstrates a large thrombus in the portal vein (arrow) which is preventing contrast from entering the main portal vein. The liver is being perfused via hepatic arteries (arrowheads).

CT: computed tomography.

Courtesy of Jonathan Krukal, MD.

Graphic 78815 Version 3.0

Contributor Disclosures

Arun J Sanyal, MD Equity Ownership/Stock Options: Durect [NASH]; Exhale NZ [Helicobacter pylori]; Genfit [NASH]; HemoShear [Rare liver diseases]; Indalo [NASH]; NorthSea [NASH]; Rivus [NASH]; Sanyal Bio [Animal testing]; Tiziana [NASH]. Grant/Research/Clinical Trial Support: Alnylam [NASH]; Amgen [NASH]; Boehringer Ingelheim [NASH]; Bristol Myers [NASH]; Covance [Lipoproteins]; Echosens Sandhill [NASH]; Fractyl [NASH]; Genentech [NASH]; Gilead [NASH]; HistoIndex [NASH]; Immuron [Alcoholic hepatitis]; Inventiva [NASH]; Lilly [NASH]; Madrigal [NASH]; Mallinckrodt [Portal hypertension]; Merck [NASH]; Novartis [NASH]; Novo Nordisk [NASH]; Owl [NASH]; Path AI [NASH]; Pfizer [NASH]; ProSciento [NASH]; Regeneron [NASH]; Roche [NASH]; Salix [Hepatic encephalopathy]; Second Genome [Microbiome]; Siemens [NASH]. Consultant/Advisory Boards: 89 Bio [NASH]; Albireo [NASH]; Amgen [NASH]; Amra [Metabolism]; AstraZeneca [NASH]; BiocellVia [NASH]; Boehringer Ingelheim [NASH]; Bristol Myers [NASH]; Conatus [NASH]; Fractyl [NASH]; Galectin [NASH]; Genentech [NASH]; Genfit [NASH]; Gilead [NASH, COVID-19]; HemoShear [Rare diseases]; HistoIndex [NASH]; Immuron [Alcohol-associated liver disease]; Intercept [NASH]; Janssen [NASH]; Lilly [NASH]; Madrigal [NASH]; Mallinckrodt [Portal hypertension]; Merck [NASH]; NGM Bio [NASH]; NorthSea [NASH]; Novartis [NASH]; Novo Nordisk [NASH]; PathAI [NASH]; Perspectum [NASH]; Pfizer [NASH]; Poxel [NASH]; ProSciento [NASH]; Regeneron [NASH]; Roche [NASH]; Salix [Cirrhosis]; Sanofi [NASH]; Sequana [Cirrhosis]; Siemens [NASH]; Takeda [NASH]; Terns [NASH]. All of the relevant financial relationships listed have been mitigated. **Sanjiv Chopra, MD, MACP** No relevant financial relationship(s) with ineligible companies to disclose. **Kristen M Robson, MD, MBA, FACG** No relevant financial relationship(s) with ineligible companies to disclose.

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Conflict of interest policy

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