All clogged up: Spectrum of venous thrombosis in the abdomen and pelvis.

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Learning objectives

1. To understand the pathophysiology of venous outflow obstruction in the abdomen and pelvis and its clinical implications.

2. To review the different thrombus types and their most common causes in the abdomen and pelvis.

3. To illustrate the imaging findings of venous thrombosis in the abdomen and pelvis on US, CT, MR and PET.
Background

Pathophysiology of venous outflow obstruction in the abdomen and pelvis.

In general terms, venous outflow obstruction can be compared to a simple plumbing problem: the pipe that carries the flow away from the toilet or sink is not working. It does not matter how or where the obstruction is, the results in our bathroom or kitchen will be the same.

The common mechanism involves a progressive rise in the hydrostatic pressure inside the venous lumen proximal to the obstruction site with retrograde transmission. When a certain threshold hydrostatic pressure is achieved, depending on oncotic pressure and vessel permeability among other factors, fluid leakage will occur. If the pressure continues increasing, it can potentially compete with the perfusion pressure of arterial inflow. This can result in severely diminished or stalled blood flow to the affected parenchyma with ensuing tissue ischemia. The existence and capacity of collateral pathways plays a pivotal role in this setting and can determine the outcome of the patient.

Unfortunately, the basic model explained above is not enough to account for all the spectrum of clinical manifestations of abdominopelvic vein thrombosis. The clinical picture and imaging patterns will be as diverse as the contents of the abdomen and pelvis itself. Depending on the specific vein compromised, different organs or territories will be affected and a complex mix of symptoms, signs and imaging markers will develop from a an initial common physiopathologic mechanism. In other words, the same original problem will generate different results depending on the location.

To add even more complexity to this scenario, it must be noted that the cause of the venous obstruction can arise from different sources, and each individual etiology implies a different clinical and physiopathologic setting. The most common and simple form of venous obstruction is the bland clot. This organizing mix of platelets and fibrin can develop secondary to a myriad of local and systemic process as venous stasis, hypercoagulability states and endothelial damage (Virchow’s triad). Elements in this triad also play a role in non-bland thrombus formation. This is the case in septic thrombophlebitis: in this disease, an infectious process is the initial step in the cascade of endothelial injury and coagulation activation giving birth to a clot that can also include microorganisms. Thus there is not only impeded venous outflow but also the risk of dissemination the septic process to the draining distal parenchymas: liver, lung or heart. The last of the main three types of thrombus is the tumoral one. This “clot” is really an endoluminal extension of a neoplastic process (usually malignant, but may occur in benign, non-cancerous neoplasms) that
can produce venous outflow obstruction. Nevertheless, it must be taken into account that tumoral thrombus can also be associated with variable amounts of bland clot.

There are other sources of venous obstruction, but they are far less common than the first three mentioned. Amniotic fluid in pregnant women, yellow bone marrow in fractured patients and talc particles in central catheter users can also impede venous outflow. Fortunately, all this examples require a very specific clinical setting that usually is well known before venous compromise is established, rising the suspicion index accordingly.

Clinical implications

Depending on the veins affected, the clinical picture of a venous thrombosis will be in direct relationship with the proximal organ suffering from congestion / ischemia. For example, if the superior mesenteric vein is occluded then ensuing mural and luminal changes in the bowel loops drained by this vessel will be responsible for the clinical manifestations of this condition. Also, as mentioned above, the presence of a functional network of venous collaterals can dramatically affect the presentation or outcome of the thrombotic event, even making it asymptomatic. It is important to remember that antegrade embolism or septic extension to the liver or thorax can produce additional clinical and radiologic findings.

Also, it is important to note that the combination of the specific vein affected, the clot burden and extension as well as any parenchymal damage determines the choice of management and potential treatments, whereas it be expectant observation, pharmacological (usually anticoagulant drugs) therapy, endovascular or surgical thrombectomy.
Fig. 2: Positive Lupus anticoagulant patient. Echogenic material is seen within the main portal vein, consistent with thrombosis.

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Fig. 3: (Same patient as figure 2). By adding Color-Doppler examination no flow can be demonstrated within the main portal vein. Flow signal from hepatic artery can be seen alongside the main portal vein.

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Fig. 4: (Same patient as figure 2, 3). Positive Lupus anticoagulant patient. Abdominal CECT. Hypodense filling defects are seen within main portal vein and its right branch, confirming Doppler US findings.

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Findings and procedure details

Multimodality imaging findings of different thrombus types in the abdomen and pelvis using US, CT, MR and PET-CT

Bland clot

Venous bland clot formation in the abdomen and pelvis can be local or result from ascending lower extremity thrombosis. Whereas local conditions as recent trauma or systemic conditions as hypercoagulability states can initiate the biochemical pathway that leads to platelet aggregation and thrombus formation, it can occur with no recognizable cause, and thus categorized as idiopathic.

On US, bland clot can be identified as endoluminal echogenic material. Depending on the age of the thrombus, it can be hypo, iso or hyperechogenic. Also, when acute, it usually determines expansion of the affected venous segment. It does not not show signal on Doppler-Color or vascular flow waves with spectral Doppler techniques. If this is the case, bland clot diagnosis must be discarded in favor of tumoral thrombus.

On non-contrast enhanced CT, bland clot can be seen as endoluminal content that may show greater density than non-thrombosed vessels or soft tissues, specially when the thrombus is acute. As days pass, bland thrombus turns isodense to normal vessels. When contrast is used, the typical finding is an endoluminal hypodense segmental filling defect, either central or eccentric, that totally or partially impedes contrast media flow. It is important to note that a simple bland clot should note increase in density with the use of contrast media. If that is the case, tumoral thrombus should be considered as the likely cause of venous occlusion. The very rare hematoma/thrombus organization could also explain thrombus enhancement and may be considered in the differential diagnosis list.

On MRI, bland clot can be recognized as endoluminal filling defect that typically is iso or hyperintense on T1WI and intermediate signal or mildly hyperintense on T2WI. With the use of gadolinium, no enhancement should be identified.

Septic thrombophlebitis

This kind of thrombus shares most of the characteristics of bland clot on all imaging modalities, except for subtle changes that depend on the nature and virulence of the initial infectious process. Also, the development of distal septic embolism is a well documented complication in septic thrombophlebitis. Embolism of bacteria-laden clots down-stream can generate foci of infection in diverse locations, the most common organs affected
being the liver and lungs. Septic emboli in the pulmonary parenchyma present as multiple nodules that can cavitate.

On US, infected thrombus can show a more heterogeneous echogenicity than regular bland clots. On CT or MRI, the presence of thickened and hyperenhancing venous wall plus coexisting inflammatory changes of the adipose tissue adjacent to the vein help differentiation from bland clot. This complication must be searched for in all cases presenting with an intraabdominal septic process, but specially in patients with postpartum endometriosis, acute appendicitis or diverticulitis.

**Tumoral thrombus**

This type of thrombus has very specific imaging markers that can help us make a correct prospective diagnosis. On US, if the primary lesion is visible, the echogenicity must be similar between the neoplasm and the venous endoluminal content. The defining hallmark however is the presence of vascular flow on Doppler-color or Doppler-spectral examinations.

**Other types**

Besides bland clot, septic thrombus and tumoral thrombus, there are other materials that can go inside the venous lumen and impede the outflow. Fortunately, this cases usually require a very specific clinical context in order to develop.

In the setting of lower extremity fracture, orthopedic surgery or cardiac surgery, fat particles can be released in blood circulation and embolize, impending venous outflow through mechanical tamponade and inciting a local inflammatory response.

Other of this less frequent etiologies is amniotic fluid. It happens when amniotic fluid is enters into the bloodstream through small disruptions in uterine veins during labor. However, the placenta can also be disrupted by trauma or surgery.

Gas bubbles can also embolize in the venous system. The most common cause is iatrogenic, secondary to diverse diagnostic and therapeutic procedures. Trauma can also determine the entering of air or gas bubbles through venous wall defects. And finally, in the setting of decompression illness nitrogen can form bubbles in the bloodstream due the fast pressure decrease.
Specific causes and complications of venous thrombotic disease in the abdomen and pelvis

Portal vein thrombosis

This entity can be seen in a myriad of different clinical settings. Bland clot can develop in the context of portal hypertension, hypercoagulable states, surgery (particularly bariatric surgery), trauma and almost any kind of inflammatory process in the digestive system. Imaging will show typical findings. On US, endoluminal echogenic material and flow absence on Doppler-color and Doppler-spectral images. The caliber can be augmented or can be normal. It must be noted that as early as two weeks are enough to develop porto-portal collateral (cavernous transformation). One of this collateral vessels can be big enough to be confused with a patent main portal vein, not only in US but in other imaging modalities. On NECT hyperdense material in the trajectory of the portal vein can be visualized, but this will depend on the thrombus age. As time goes by, the clot density will decrease. In CECT, hypodense endoluminal filling defects are the hallmark. In the acute setting, central location in the vessel is typical. In chronic cases, peripheral location, endoluminal webs and a diminished caliber are the most common findings. In MRI, the acute thrombus is commonly hyperintense on both T1WI and T2WI. Acute thrombosis can also show water restriction on diffusion weighted images. As the thrombus ages, T2WI intensity will drop. And last, PET-CT has showed promising results regarding its ability to differentiate between bland clot and tumoral thrombus, with an increased 18F-FDG uptake in the latter.

Septic thrombus usually arises from infectious intestinal compromise like appendicitis or diverticulitis, traveling through the superior/inferior mesenteric vein (A.K.A. Pylephlebitis). In addition to the bland clot imaging findings, thickening and mural venous enhancement as well as peri-vascular inflammatory changes can be detected and, in some cases, liver abscess can develop. Although the intestinal inflammatory focus can be present at time of septic thrombophlebitis diagnosis, most commonly the initial event has occurred weeks before and can be resolved at time of imaging.

Tumoral portal vein thrombus is a common pathway of neoplastic dissemination by hepatocellular carcinoma. Less frequently, tumour growth in the portal system can occur in intrahepatic cholangiocarcinoma and liver metastasis. In these cases, there are key imaging findings that will establish the diagnosis with great accuracy. On US, the presence of intrathrombotic vessels on Doppler-color and, more specifically, arterial waveforms on Doppler-spectral examinations are enough to certify the neoplastic nature of the thrombus. The CT and MRI counterpart of these findings is thrombus enhancement with the use of contrast media.

The acute occlusion of the portal vein can lead to pre-sinusoidal portal hypertension or ischemic bowel syndrome, specially if the superior mesenteric vein is also involved.
For portal vein thrombosis cases refer to figures 5 to 12.

**Hepatic veins thrombosis**

Thrombosis of hepatic veins is the main cause of Budd-Chiari syndrome and is associated with a classical clinical triad of abdominal pain, ascitis and hepatomegaly in the acute setting. It is important to note that thrombosis of the hepatic segment of the inferior vena cava can produce the same physiopathological phenomena with no thrombus inside the hepatic veins.

Apart from the common risk factors for bland clot formation, almost 25% of hepatic vein thrombosis is caused by extrinsic compression of an intrahepatic lesion, primary or secondary. Also, it is not uncommon to find concurrent portal vein thrombosis.

In terms of venous imaging findings, it shares the pattern of other locations. It is important to recognize secondary findings of liver parenchyma congestion associated with this condition. Non-homogeneous mottled early liver enhancement and delayed enhancement in the periphery and around the hepatic veins produce the so-called "nutmeg liver".

Tumoral thrombosis of the hepatic veins can also occur, with hepatocellular carcinoma the most frequent culprit.

For hepatic veins thrombosis cases refer to figures 13 to 14.

**Superior mesenteric vein thrombosis**

This entity shares risk factors and imaging findings with portal vein thrombosis, being the superior mesenteric vein one of the two principal tributaries of the portal system alongside the splenic vein. Nevertheless, the most crucial aspect to recognize is its specific clinical context. Superior mesenteric vein thrombosis is responsible for 15% of mesenteric ischemia cases. Because it is primary a venous occlusion, on the first stage arterial supply is not impaired and the so-called "red ischemia" appears, with intestinal mural thickening characterized by prominent submucosal edema producing a "target" wall appearance. Edematous changes can also be seen in the mesentery with associated engorgement of the respective vessels. Sometimes, intraparietal bowel hemorrhage develops, which is nicely demonstrated on NECT as spontaneous parietal hyperdensity. If occlusion persists, arterial supply will be undermined and "white ischemia" will establish, with diminished mucosal enhancement to frank intestinal wall
non-enhancement. Pneumatosis intestinalis is rare in intestinal venous ischemia. It must be noted that there is no linear relationship between the extent of mesenteric venous thrombosis and the degree of bowel ischemia. Some patients may present with extensive thrombotic occlusion of venous mesenteric branches and minimal or no bowel abnormality. Thus, the risk of bowel necrosis is more related to the clinical presentation than the extent of thrombosis: patients with abdominal symptoms severe enough that make them seek medical attention within 24 hours of onset have a higher incidence of transmural ischemia / necrosis as opposed to those with moderate symptoms that present days later.

For superior mesenteric vein thrombosis cases refer to figures 15 to 17.

**Inferior vena cava thrombosis**

This entity also shares risk factors and local imaging findings with portal vein thrombosis, but it is anatomical location makes it susceptible to a wider range of thrombus etiologies. In fact, in the context of an endoluminal filling defect, the main goal will be to differentiate between bland clot and tumoral thrombus and, if possible, to assess the origin of the neoplastic process. Bland clot can arise more often from deep venous thrombosis that travels with the bloodstream. Other specific cause is the extrinsic compression by an abdominopelvic mass or nodal conglomerate, that can stenose the IVC slowing or impeding venous flow with subsequent clot formation.

Tumoral thrombosis of the IVC obstruction can be varied. The most common neoplasm to reach the IVC is renal cell carcinoma, invading first the renal vein and following its path. It can even reach the heart. Other neoplasms that have a predilection for IVC extension are hepatocellular carcinoma, adrenocortical cancer and Wilm's tumor in the pediatric population. Most of these carcinomas tend to present with a hypervascular enhancement pattern, and neof ormation vessels are easily identifiable in Doppler-color and contrast enhanced CT or MRI within the caval lumen. Direct extension into the IVC can also occur from large nodal masses in the retroperitoneum such as those resulting from metastatic testicular cancer or lymphoma. A primary retroperitoneal leiomyosarcoma arising from the muscular layer of the venous wall can present as tumoral thrombus without a dominant extravascular mass. Tumor thrombus can also extend from pelvic mass via the iliac veins. Intravascular leiomyomatosis is a form of disseminated benign uterine leiomyomas that can grow in the deep venous system, even reaching the heart.

If IVC occlusion is complete, inferior vena cava syndrome will appear, clinically presenting with inferior extremity edema, tachycardia and orthostatism.

For inferior vena cava thrombosis cases refer to figures 18 to 24.
Renal veins thrombosis

Renal veins thrombosis can be bland or tumoral, most frequently occurring on the left side. In adults, the main causes of renal vein thrombus include nephrotic syndrome, glomerulonephritis, diabetes, renal sepsis, tumoral thrombus (renal cell carcinoma, lymphoma or adrenal carcinoma) or trauma. The nephrotic syndrome is one of the most common causes of bland thrombus. In this case, the treatment includes steroids and immune-suppression therapy. In other cases of bland thrombosis, anticoagulation is the primary medical therapy.

The patients can present with flank pain, nausea, vomiting, hematuria or acute renal failure.

The long term sequelae of renal vein thrombosis depend on duration of venous occlusion, recanalization, and collateralization. Chronic thrombosis leads to renal atrophy and development of multiple enlarged venous collaterals. These patients may develop renal failure and hypertension, or they can be asymptomatic.

For renal veins thrombosis cases refer to figures 25 to 28.

Gonadal veins thrombosis

This entity occurs more frequently in early postpartum patients who present with acute lower abdominal or pelvic pain and fever. In this case there is a septic thrombophlebitis of the ovarian veins. It usually occurs as a result of endometritis post caesarian section. Vaginal delivery appears to be a protecting factor against the development of this disease. The right ovarian vein is compromised in 90% of cases, as the the left vein is protected by retrograde flow from the left renal vein. 14% of the patients have bilateral ovarian vein thrombosis. Bland gonadal vein thrombosis may occur in malignancy, pelvic surgery, trauma or inflammatory bowel disease.

The main differential diagnosis of septic gonadal vein thrombosis is acute appendicitis. Both share a similar clinical presentation and physical examination as the right gonadal vein ascends through the right iliac fossa. On CT, both may appear as a tubular structure with thickened, hyperenhancing walls. In this setting, following the course of the gonadal vein ascending from the uterus/adnexa ventral to the psoas muscle and reaching the inferior vena cava at the level of the right renal hilum helps to make the distinction between them.

For gonadal veins thrombosis cases refer to figure 29.
Fig. 5: Liver US examination. The main portal vein presents echogenic endoluminal material that expands the lumen. Tumoral thrombus can not be excluded with this information only.

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**Fig. 6**: (Same patient as figure 5). Liver Doppler US examination. Small motion foci are found on Color-Doppler. When explored with Spectral-Doppler, arterial waveforms are demonstrated. This is pathognomonic for tumoral thrombus, probably hepatocellular carcinoma.

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**Fig. 7:** Abdominal MRI, T2WI. There is thrombus in main and left portal veins. Perisplenic ascites can be seen.

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Fig. 8: Abdominal CECT images show a large, enhancing mass replacing the body and tail of the pancreas. There is tumor invasion of the splenic vein and tumor thrombus extending into the dilated portal vein.

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Fig. 9: (Same patient as figure 8). Post-contrast T1WI images show a large, enhancing mass replacing the body and tail of the pancreas. There is tumor invasion of the splenic vein and tumor thrombus extending into the dilated portal vein.

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Fig. 10: Patient with acute appendicitis. Abdominal CECT, portovenous phase. There is a filling defect within the main portal vein. No changes in caliber are identified.

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Fig. 11: (Same patient as figure 8). Abdominal CECT, arterial phase. Multiples hypodense focal lesions are seen in the right liver lobe. These lesions present peripheral enhancement. Some perilesional edema is identified. This pattern is consistent with liver abcesses, demonstrated at subsequent surgery.

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Fig. 12: PET-CT. There is significant tracer uptake within the portal vein. This is pathognomonic for tumoral thrombus.

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**Fig. 13:** Abdominal post-contrast T1WI MRI. Budd-Chiari syndrome with thrombosis of the hepatic veins. Note the heterogeneous intensity of the parenchyma and surrounding ascites.

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**Fig. 14:** Post-contrast T1WI MRI. Mild hepatomegaly is seen with heterogeneous intensity in the parenchyma. No focal lesions are identified. Thrombus can be seen within inferior vena cava and right hepatic vein.

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**Fig. 15:** Abdominal CECT. No opacification with contrast media is seen in the superior mesenteric vein. Some bowel loops show parietal thickening. Note the expanded caliber of the vein.

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**Fig. 16:** Abdominal CECT. Patient with no opacification of mesenteric vein on upper images. Multiples bowel loops with parietal thickening are found. There is fluid and engorgement of vessels in the mesentery. This changes are consistent with mesenteric ischemia, "red type ischemia", more frequent when the first stage is produced by venous outflow obstruction.

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Fig. 17: Abdominal CECT, portovenous phase. Filling defects can be identified within superior mesenteric vein and main portal vein. There is bowel wall thickening in the left upper quadrant with submucosal edema. These changes are consistent with bowel ischemia.

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Fig. 18: Thorax, abdomen and pelvis CECT, sagital reconstruction. A large filling defect is identified in almost all the course of the inferior vena cava, even entering in the right atrium. It enhances moderately with contrast media, finding consistent with a tumoral thrombus. No associated neoplastic process was found. Subsequent biopsy demonstrated a IVC leiomiosarcoma.

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**Fig. 19**: Abdominal US, transverse view. The inferior vena cava is completely filled with material isoechoic to liver parenchyma. The caliber is expanded.

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Fig. 20: Abdominal US, longitudinal view. The inferior vena cava is filled with material isoechoic to liver parenchyma and the caliber is expanded. The filling defect has a free convexous margin.

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Fig. 21: Abdominal US, transverse view. Patient with large filling defect on inferior vena cava during the same exam. Cefalic to superior pole of right kidney, a large heterogeneous mass is identified. Subsequent biopsy showed an adrenocortical carcinoma with IVC invasion.

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Fig. 22: Abdominal CECT, portovenous phase. A large mass arising from the left adrenal gland can be seen. There is invasion into the inferior vena cava. No difference in caliber between the normal and the invaded segments.

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Fig. 23: Abdominal MRI with Gadolinium. There is a mass in the right liver lobe which invades the adjacent inferior vena cava. There are signs of chronic liver disease. This is a typical presentation of hepatocellular carcinoma.

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**Fig. 24:** Abdominal CECT, arterial phase. There is expansion of the inferior vena cava with endoluminal material. There are arterial vessels within the filling defect. This is seen in tumoral type thrombus. Note the perisplenic ascites.

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**Fig. 25:** Abdominal CECT, arterial phase. A hypervascular mass arising from the right kidney is seen, consistent with renal cell carcinoma. There is invasion of the right renal vein. Note the arterial vessels within the tumoral thrombus.

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**Fig. 26**: Abdominal CECT, portovenous phase. Same patient from figure 22. The tumoral thrombus in the right renal vein invades the inferior vena cava up to the right atrium.

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**Fig. 27:** A large hypervascular mass is seen arising from the left kidney, consistent with renal cell carcinoma. There is a filling defect in distal left renal vein and inferior vena cava with the same appearance than the renal lesion. This is consistent with tumoral thrombus invasion. No the hypervascular enhancement pattern of the thrombus.

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Fig. 28: Patient with left renal cell carcinoma. Abdominal MRI. An intermediate signal thrombus is identified within left renal vein and inferior vena cava. These findings are consistent with tumoral thrombus invasion.

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**Fig. 29:** Abdominal CECT. There is a central filling defect in the gonadal vein. The vein itself is enlarged. It is importante to identify the adjacent ureter. A dilated ureter can present with similar appearance.

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Conclusion

Venous outflow obstruction in the abdomen and pelvis has a myriad of causes, but the primary physiopathologic mechanism is shared by all.

It is important to know the different clinical settings that are associated with various venous obstruction etiologies.

There are specific imaging findings associated with certain thrombus location and causes.
Personal information

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