Imaging Evaluation of the Inferior Vena Cava¹

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Abbreviation: IVC = inferior vena cava


SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

■ Classify congenital IVC variants and understand their implications for patient treatment.

■ Identify IVC involvement in malignancy and its potential impact on surgical planning.

■ Describe the appearance of the IVC after intervention and in the context of trauma or infection.

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The inferior vena cava (IVC) is an essential but often overlooked structure at abdominal imaging. It is associated with a wide variety of congenital and pathologic processes and can be a source of vital information for referring clinicians. Initial evaluation of the IVC is most likely to occur at computed tomography performed for another indication. Many routine abdominal imaging protocols may result in suboptimal evaluation of the IVC; however, techniques to assist in specific evaluation of the IVC can be used. In this article, the authors review the spectrum of IVC variants and pathologic processes and the relevant findings from magnetic resonance imaging, angiography, sonography, and positron emission tomography. Embryologic development of the IVC and examples of congenital IVC variants, such as absence, duplication, left-sided location, azygous or hemiazygous continuation, and web formation, are described. The authors detail IVC involvement in Wilms tumor, leiomyosarcoma, adrenal cortical carcinoma, testicular carcinoma, hepatocellular carcinoma, renal cell carcinoma, and other neoplasms, as well as postsurgical, traumatic, and infectious entities (including filter malposition, mesocaval shunt, and septic thrombophlebitis). The implications of these entities for patient treatment and instances in which specific details should be included in the dictated radiology report are highlighted. Furthermore, the common pitfalls of IVC imaging are discussed. The information provided in this review will allow radiologists to detect and accurately characterize IVC abnormalities to guide clinical decision making and improve patient care.

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Introduction

The inferior vena cava (IVC) is the main conduit of venous return to the right atrium from the lower extremities and abdominal viscera. It can be a source of critical information for referring clinicians, and recognition of IVC variants and pathologic characteristics can help guide patient treatment. The purpose of this article is to increase knowledge of both congenital IVC variants and other processes affecting the IVC and to emphasize their effect on patient care. After a brief overview of IVC imaging techniques and embryologic features, cases in which computed tomography (CT) was performed with relevant correlates from magnetic resonance (MR) imaging, angiography, ultrasonography (US), and positron emission tomography (PET) will highlight the spectrum of congenital variants, neoplasms, and other entities related to surgery, intervention, trauma, and infection. Furthermore, potential imaging pitfalls will be discussed.
Increasing the delay after contrast material injection to 70–90 seconds allows more uniform enhancement of the entire IVC at CT. Because of anomalous persistence, regression, and anastomoses of the vitelline, posterior cardinal, subcardinal, and supracardinal veins, several classic congenital variations, including IVC absence or duplication, a left-sided IVC, anomalous continuation of the IVC to the thorax, and a retrocaval ureter, can occur alone or in combination. Identification of duplication of the IVC is important in patient treatment because if the entity is not recognized before routine placement of an infrarenal IVC filter, recurrent pulmonary embolism can develop. Malignant IVC involvement is most often attributable to direct endovascular extension and/or intraluminal thromboembolization. Tumor thrombus, the most common manifestation of IVC involvement, can be differentiated from bland thrombus on the basis of expansion of the vessel lumen and enhancement of the filling defect. Pseudolipoma is a volume-averaging artifact due to prominent pericaval fat above the caudate lobe and is often seen in patients with cirrhosis. Admixture artifact may also occur at the level of the renal veins when contrast-enhanced blood from the kidneys mixes with nonenhanced blood from the lower extremities.

**TEACHING POINTS**

- Increasing the delay after contrast material injection to 70–90 seconds allows more uniform enhancement of the entire IVC at CT.
- Because of anomalous persistence, regression, and anastomoses of the vitelline, posterior cardinal, subcardinal, and supracardinal veins, several classic congenital variations, including IVC absence or duplication, a left-sided IVC, anomalous continuation of the IVC to the thorax, and a retrocaval ureter, can occur alone or in combination.
- Identification of duplication of the IVC is important in patient treatment because if the entity is not recognized before routine placement of an infrarenal IVC filter, recurrent pulmonary embolism can develop.
- Malignant IVC involvement is most often attributable to direct endovascular extension and/or intraluminal thromboembolization. Tumor thrombus, the most common manifestation of IVC involvement, can be differentiated from bland thrombus on the basis of expansion of the vessel lumen and enhancement of the filling defect.
- Pseudolipoma is a volume-averaging artifact due to prominent pericaval fat above the caudate lobe and is often seen in patients with cirrhosis. Admixture artifact may also occur at the level of the renal veins when contrast-enhanced blood from the kidneys mixes with nonenhanced blood from the lower extremities.

**IVC Imaging Techniques**

Because CT is used to evaluate a wide variety of abdominal symptoms, it is likely to be the most common imaging modality for initial detection of IVC variants and pathologic findings. Routine abdominal imaging at 60–70 seconds after intravenous administration of contrast material (portal venous phase) shows enhancement in the renal and suprarenal IVC but may also show admixture artifact in the infrarenal IVC (1,2). Increasing the delay after contrast material injection to 70–90 seconds allows more uniform enhancement of the entire IVC at CT (2). Another advanced imaging technique available for IVC evaluation is MR imaging. MR imaging, specifically with breath-hold contrast material–enhanced three-dimensional T1-weighted imaging and balanced steady-state free-precession techniques, is more reliable than CT for evaluation of IVC tumor thrombus (1). US is also useful for initial evaluation when an IVC pathologic condition is suspected, although US is subject to operator dependence and evaluation of the infrahepatic IVC may be limited because of artifact resulting from overlying bowel gas and body habitus (1). Doppler US may show absent or abnormal flow in the IVC at the site of occlusion or narrowing caused by an intraluminal mass (3). In pediatric patients, US is generally the first-line modality for cross-sectional imaging. If US is not sufficient because of operator or patient factors, MR imaging is sensitive for IVC abnormality detection while still not requiring the use of ionizing radiation. However, anesthesia may be required in the pediatric population at MR imaging.

**Embryologic Development and Normal Anatomic Structure**

The IVC is the main conduit of venous return from the lower extremities and abdominal viscera. The mature IVC has four segments: the hepatic, suprarenal, renal, and infrarenal IVC (4). Formation of the IVC involves complex anastomoses and regression of multiple embryonic veins, including the vitelline vein and the paired posterior cardinal, subcardinal, and supracardinal veins (Fig 1). The vitelline vein contributes to the hepatic segment of the IVC. The suprarenal IVC is composed of a segment of the right subcardinal vein that does not regress. The renal segment of the IVC is formed by the anastomosis between the right subcardinal and right supracardinal veins. A segment of the right supracardinal vein persists as the infrarenal segment. The embryonic veins also lead to the azygos, hemiazygos, and common iliac veins (4,5).

**Congenital IVC Variants**

Congenital variations of the IVC are the result of abnormal embryologic development involving the vitelline, posterior cardinal, subcardinal, and supracardinal veins (5). They are present in approximately 4% of the population, and these individuals are often asymptomatic (1,2). Because of anomalous persistence, regression, and anastomoses of the vitelline, posterior cardinal, subcardinal, and supracardinal veins, several classic congenital variations, including IVC absence or duplication, left-sided IVC, anomalous continuation of the IVC to the thorax, and retrocaval ureter, can occur alone or in combination.

**Absence of the IVC**

Congenital absence of the entire IVC or of only the infrarenal IVC (6–10) has been previously described but has an unknown incidence and unclear cause. IVC absence may result from complete failure of embryonic vein development; however, perinatal venous thrombosis and atrophy have also been suggested (8–10). In addition to inability to identify the IVC at imaging, prominent venous collateralization may be a finding (Fig 2). Patients may experience lower extremity venous insufficiency or idiopathic deep vein thrombosis or have prominent lumbar collateral vessels, which can be mistaken for paraspinal masses (6–11).

**Duplication of the IVC**

IVC duplication is the result of persistence of both supracardinal veins forming duplicated infrarenal IVC segments (5). The left infrarenal...
Figure 1. Schematic shows embryologic features of the IVC. The IVC (dark blue) is composed of multiple segments derived from anastomosis (light blue) of embryonic veins. The vitelline vein contributes to the hepatic IVC. The suprarenal IVC is composed of a segment of persistent right subcardinal vein. The subsupracardinal anastomosis persists as the renal segment. A segment of the right supracardinal vein persists as the infrarenal IVC. The caudal aspects of the posterior cardinal veins persist as the iliac veins. The superior supracardinal veins also persist as the azygos venous system (green).

Figure 2. Coronal abdominal CT images of congenital IVC variants. (a) Absence of the intrahepatic IVC (arrow). The superior mesenteric vein and portal venous confluence appear to be normal (arrowhead), but the IVC is absent. (b) Duplication of the IVC (arrows), with a left-sided IVC draining into the left renal vein (black arrowhead) and normal continuation of the suprarenal IVC (white arrowhead). (c) Left-sided IVC (arrow) below the level of the renal veins joins the left renal vein (arrowhead) and continues as the intrahepatic IVC. (d) Azygos continuation of the IVC is indicated by a prominent azygos vein (arrow) running parallel to the aorta and absence of the intrahepatic IVC (arrowhead).
IVC joins the left renal vein and drains into a normal suprarenal IVC (Fig 2). The prevalence of this anomaly is 0.2%–0.3% (5). Identification of duplication of the IVC is important in patient treatment because if the entity is not recognized before routine placement of an infrarenal IVC filter, recurrent pulmonary embolism can develop (5). Therefore, for each patient, a review of prior cross-sectional images is an essential step before filter placement. If no images are available, cavography should be performed through the left iliac vein. A filter can then be placed into each cava. In addition, the IVC lying to the left of the abdominal aorta can be mistaken for a lymph node if not followed along its course (1).

**Left-sided IVC**
A left-sided IVC, as it is classically described, results from regression of the right supracardinal vein, with abnormal persistence of the left supracardinal vein, and has a prevalence of 0.2%–0.5% (5). Similar to a duplicated IVC, a left-sided IVC courses cranially to the left of the abdominal aorta, joins with the left renal vein, and drains into a normal suprarenal IVC (Fig 2). Although the clinical effect is minimal, a left-sided IVC can cause difficult central venous access during interventional procedures if the endovascular operator is unaware of the anatomic structure. Specifically, a left-sided IVC may cause confusion between venous and arterial access, limit access options for IVC filter placement, or complicate pulmonary thrombolysis.

**Anomalous Continuation of the IVC**
Continuation of the suprarenal IVC as the azygos or hemiazygos vein is attributable to embryonic failure to form the right subcardinal–hepatic anastomosis and, in the case of azygos continuation, has a prevalence of 0.6% (4,12). The suprarenal IVC drains either into the azygos vein and returns to the heart through the superior vena cava or into the hemiazygos vein and subsequently into the azygos vein (Fig 2). The hemiazygos vein may also drain directly into the coronary sinus through a persistent left-sided superior vena cava or into the left brachiocephalic vein through the accessory hemiazygos vein (13). Because of the absence of the intrahepatic segment of the IVC, the hepatic veins drain directly into the right atrium. The azygos vein, enlarged to accommodate increased flow, could be mistaken for retrocrural lymphadenopathy and a prominent azygos and superior vena cava confluence for a right paratracheal mass (12). Drainage through the hemiazygos vein may simulate a left-sided mediastinal mass or, in the event of accessory hemiazygos drainage, an aortic dissection (13,14). There is also potential for inadvertent ligation of the hemiazygos vein during thoracic surgery (15).

**Retrocaval Ureter**
Although the genitourinary system develops separately, the spatial relationship between the ureter and the IVC depends on IVC embryologic features. If the infrarenal IVC develops from the right posterior cardinal vein instead of the right supracardinal vein, the result will be a retrocaval ureter, also known as a circumcaval ureter (4). The ureter, usually situated on the right side, courses posterior to the IVC and descends to the right of the aorta (Fig 3). There is potential for partial urinary outflow obstruction and recurrent urinary tract infections (4). Diagnosis of a retrocaval ureter can easily be facilitated with CT urography. If the patient is symptomatic, treatment necessitates surgical relocation of the ureter (1,5).

**IVC Webs**
IVC web formation is an uncommon IVC anomaly that has been described as resulting from a congenital vascular anomaly or the sequel of thrombus formation (16). IVC webs are rare in North American and northern European populations and more common in Asian and South African populations (16). Imaging shows a complete or
fenestrated membrane in the intrahepatic IVC or a segment of fibrotic occlusion that may be of variable length (1,16). Prominent intrahepatic and extrahepatic collateral vessels also develop (Fig 4). Clinically, a web causes hepatic outflow obstruction and can lead to congenital Budd-Chiari syndrome, which may then lead to hepatocellular carcinoma. Inferior venacavography can be performed to help confirm the diagnosis (16). Depending on the severity of the associated liver disease, treatment may include angioplasty, placement of a stent, or creation of a transjugular intrahepatic portosystemic shunt, interventions that would relieve the resultant portal hypertension (17).

Extrahepatic Portocaval Shunt (Abernethy Malformation)

The Abernethy malformation is classified into two categories (18). Type 1 is characterized by complete shunting of portal blood into the IVC and congenital absence of the portal vein. It is more common in females and is associated with polysplenia and biliary atresia. A type 2 shunt is a partial end-to-side anastomosis between an intact portal vein and the IVC and is most commonly seen as an isolated finding in males (19) (Fig 5). These extrahepatic portocaval shunts are thought to be attributable to either excessive involution of the vitelline vein or failure of the vitelline vein to establish an anastomosis with the hepatic sinusoids or hepatic veins (20,21). The presence or absence of the portal vein is an important imaging finding because it helps distinguish between the two types. The Abernethy malformation is associated with focal nodular hyperplasia and hepatocellular carcinoma; MR imaging with a hepatobiliary contrast agent may be beneficial for distinguishing between the two liver lesions because focal nodular hyperplasia will retain such contrast material (19).
IVC Involvement by Neoplasms

Both primary and secondary malignant neoplasms can involve the IVC and often have similar imaging features. Primary IVC malignancy is extremely rare; although leiomyosarcoma represents less than 1% of all malignancies, it accounts for more than 75% of tumors arising from large veins (22,23). Primary IVC leiomyoma is also possible, and this benign tumor accounts for 15% of tumors arising from large veins (23). Secondary involvement of the IVC in abdominal malignancy is more common than primary IVC neoplasms. Malignant IVC involvement is most often attributable to direct endovascular extension and/or intraluminal thromboembolization. Tumor thrombus, the most common manifestation of IVC involvement, can be differentiated from bland thrombus on the basis of expansion of the vessel lumen and enhancement of the filling defect (24,25). Correct identification of the extent of IVC involvement is essential to staging and determining surgical intervention.

Primary IVC Leiomyosarcoma

Leiomyosarcoma is the most common primary malignancy involving the IVC (26). It arises from the smooth muscle cells in the vessel wall. Seventy-four percent of cases of IVC leiomyosarcoma occur in women, and women aged 40–60 years are the most frequently affected (22,27). The initial growth of an IVC leiomyosarcoma is intramural (3,28). Two-thirds of tumors will demonstrate predominantly extraluminal growth, and one-third will demonstrate predominantly intraluminal growth (3,22,28). The intraluminal tumors may cause venous obstruction. At imaging, the mass may appear as a heterogeneously contrast material–enhancing filling defect of the IVC that may show cystic necrosis (Fig 6). Extraluminal tumors can invade adjacent structures and should be differentiated from neoplasms arising from the surrounding organs or directly from the retroperitoneum (26). The level of IVC involvement is important because tumors involving the renal and suprarenal IVC (42%–50%) are associated with the most favorable prognosis. Involvement of the intrahepatic IVC is associated with the worst prognosis and is seen in 6%–20% of cases. The remaining 37%–44% of tumors involve the infrarenal IVC (3,27–29). Complete surgical resection is required for cure, and en bloc resection of the IVC with a subsequent IVC graft may be necessary, depending on location and characteristics (30). Overall, 10-year survival is 14% and more than 50% of patients develop recurrent disease (3,28).

Renal Cell Carcinoma

Renal cell carcinoma is the most common malignancy that extends into the IVC, with 4%–10% of cases involving venous invasion (31,32). Frequently, patients with malignant tumor thrombus are asymptomatic and the thrombus is first identified at imaging. The appearance at imaging is similar to that of other tumor thrombi, with expansion of the IVC lumen and enhancement of the thrombus, findings suggestive of a malignant process (Fig 7). CT shows IVC extension of renal cell carcinoma with 96% accuracy and is the first choice for imaging renal cell carcinoma because it also allows simultaneous metastatic survey (33). Although the superior extension of tumor thrombus may be underestimated, accurate description of the tumor thrombus is essential because it affects surgical intervention (31). IVC involvement changes TNM system staging to T3b for infradiaphragmatic involvement and T3c for supradiaphragmatic extension.
Figure 8. Adrenal cortical carcinoma. Coronal (a) and axial (b) contrast-enhanced CT images show a large left-sided adrenal cortical carcinoma (*), with extension of the tumor thrombus through the left renal vein (arrowhead in b) and into the IVC (arrows in a).

Adrenal Cortical Carcinoma
Adrenal cortical carcinoma is a rare malignancy, with a reported prevalence of 0.5–2 cases per million persons. Adrenal cortical carcinoma may develop at any age, but there is a bimodal age distribution during the 1st and the 4th–5th decades of life. Sixty-two percent of adrenal cortical carcinoma cases involve functional tumors and may lead to Cushing syndrome, virilization, or feminization (36). The imaging feature of adrenal cortical carcinoma is a heterogeneous mass replacing the entire adrenal gland and often displacing the adjacent kidney, liver, or spleen (Fig 8). Calcifications are common (31). Intravascular extension into the IVC may be seen in up to 30% of cases and is more common in right-sided tumors and tumors that are larger than 9 cm (33). The differential diagnosis should include renal cell carcinoma, pheochromocytoma, and metastatic disease (31). Adrenal cortical carcinoma is more aggressive and has more rapid disease progression in adults than in children. Approximately 50% of adults will have a relatively advanced disease stage at presentation. Local recurrence is common, and the most common sites of metastasis are the liver, lungs, lymph nodes, and bone (36).

Hepatocellular Carcinoma
Invasion into and thrombosis of the portal venous system is typical in patients with hepatocellular carcinoma, but invasion into hepatic veins and the IVC occurs in 4.0%–5.9% of patients (23,24) (Fig 9). Right atrial involvement is also possible because of the right atrium’s proximity to the hepatic venous confluence (37).
Expansion of the hepatic veins and an enhancing thrombus are the typical imaging findings. Occlusion of the IVC and hepatic veins may lead to Budd-Chiari syndrome, and patients may have the classic triad of ascites, abdominal pain, and hepatomegaly at presentation (38). Systemic venous invasion by hepatocellular carcinoma is associated with an extremely poor prognosis, and patients with symptomatic intrathoracic extension of tumor thrombus have a median survival of 1–4 months (37). Invasion of the systemic venous system also predisposes the patient to distant metastasis (23).

**Transitional Cell Carcinoma**

Although microscopic vascular invasion commonly occurs in transitional cell carcinoma, extension into the IVC is rare (fewer than 20 cases have been reported in the literature) (39,40) (Fig 10). Imaging characteristics include a filling defect of the renal collecting system at CT urography, lack of renal contour distortion, and a filling defect in the IVC and/or renal vein. Aggressive surgical intervention is required, including nephroureterectomy (39,41). Invasion of the IVC wall is more common in transitional cell carcinoma than in renal cell carcinoma and may precipitate a need for segmental IVC resection. The prognosis associated with this type of carcinoma is poor; in one study, eight of 14 patients died within 6 months after surgery (39).

**Wilms Tumor**

Wilms tumor is the most common renal tumor in children and involves IVC invasion in 4%–8% of cases (33,42). Wilms tumor manifests as a large heterogeneous mixed solid and cystic mass arising from the kidney and is often first identified at US (Fig 11). Although US can show vascular extension, CT and MR imaging are better for evaluation of metastatic disease (42). Recognition of IVC involvement is important because it advances the tumor staging from I to II, and a stage II tumor may necessitate neoadjuvant
Figure 11. Wilms tumor. (a) Coronal CT image shows heterogeneously contrast-enhancing tumor in the right kidney (*), with tumor thrombus extending into the right atrium (arrow). (b) Transverse US image shows thrombus in the IVC (arrowhead). * = tumor.

Figure 12. Metastatic mixed germ cell testicular carcinoma. (a) Coronal contrast-enhanced CT image shows para-aortic lymphadenopathy (*) and tumor thrombus invading the IVC (arrow) and extending cranially in the IVC lumen (arrowhead). (b) Coronal PET/CT image confirms fluorodeoxyglucose-avid adenopathy (arrowhead) and tumor thrombus (arrow). (c) Gray-scale US image of the right testicle confirms testicular origin of the tumor thrombus.

chemotherapy or radiation therapy. IVC extension is also associated with increased morbidity during nephrectomy (33,42).

Nonseminomatous Testicular Carcinoma

Bulky retroperitoneal lymphadenopathy is a common finding in metastatic testicular cell carcinoma in which an aggressive neoplasm may be in proximity to the IVC (Fig 12) (43). Some studies have shown that 3%–11% of nonseminomatous testicular tumors involve the IVC (44,45). The tumor thrombus may result from intravascular spread through gonadal veins, or bulky retroperitoneal lymphadenopathy may invade directly through the IVC wall (43). Testicular cancer is the most common cancer in males aged 15–34 years. Because the testes are not routinely included at CT imaging, scrotal US should be recommended in young men with bulky retroperitoneal lymphadenopathy and IVC tumor thrombus.

Other Sources of Tumor Thrombus

Metastatic disease in the liver, kidneys, and adrenal glands may involve the IVC through
intravascular spread. As previously described, retroperitoneal lymphadenopathy may also invade directly through the IVC wall. It is important to differentiate tumor thrombus from bland thrombus by using contrast enhancement and luminal expansion, because a neoplasm predisposes patients to coagulopathy and development of bland deep vein thrombosis that can propagate to the IVC (24,25).

Surgery, Intervention, Trauma, Infection, and Imaging Pitfalls

Noncongenital and nonneoplastic processes can also affect the IVC. Knowledge of postsurgical and postprocedural changes is necessary for correct interpretation. In the context of an emergency, the appearance of the IVC can alert the clinician to impending hypovolemic shock. Recognition of infection can allow timely and appropriate treatment, and recognition of imaging pitfalls can prevent inappropriate treatment.

Postsurgical Changes

Creation of a mesocaval shunt is a surgical interposition between the superior mesenteric vein and the IVC. This was a popular treatment in the 1970s and 1980s for uncontrollable variceal bleeding associated with cirrhosis and portal hypertension; however, the treatment has decreased in popularity since the advent of the transjugular intrahepatic portosystemic shunt procedure. Despite this decrease in popularity, surgical creation of shunts is still relevant for decompression of variceal bleeding, because portal vein occlusion can make creation of transjugular intrahepatic portosystemic shunts technically difficult or impossible. Although shunt creation was traditionally an open vascular procedure, recent advances in intravascular US guidance allow endovascular shunt creation (46). A mesocaval shunt may be detected incidentally, or imaging may be performed intentionally to assess for patency (Fig 13).

IVC filter placement is a common procedure performed by interventional radiologists, vascular surgeons, and even interventional cardiologists. Careful evaluation of cross-sectional imaging is very important before IVC filter placement to ensure appropriate protection from deep vein thrombosis. However, many of the complications after placement of IVC filters can also be assessed at cross-sectional imaging (47) (Fig 14).

Continued advancements in liver transplantation result in more frequent postoperative imaging and treatment of complications. Stenosis of the IVC after liver transplantation is a complication caused by compression due to postoperative swelling (48). IVC anastomoses differ between cadaveric donor and living donor transplantations. In recipients of cadaveric transplants, the donor IVC is anastomosed end to end with the recipient IVC or is “piggybacked” onto the recipient’s hepatic vein. Conversely, in recipients of living transplants, the donor hepatic veins are anastomosed to the recipient IVC. Doppler US plays an important role in detecting IVC complications. For example, there is a three- to four-fold increase in IVC velocity at the site of stenosis at spectral Doppler imaging. There is also loss of phasicity of the hepatic veins that is normally the result of atrial modulation (Fig 15). Vascular complications are the second leading cause of graft failure after acute rejection, and treatment includes angioplasty and stent placement (48).

En bloc surgical resection of the vena cava may be a treatment option for patients with retroperitoneal sarcoma arising from or invading the IVC. The optimal treatment of the IVC after resection is controversial and options include ligation or reconstruction. Choices for resection include primary repair if luminal narrowing of less than 50% will result. A bowel or venous graft can also be used if the resection site is localized. If a large segment of the IVC is resected, circumferential replacement with a ringed polytetrafluoroethylene graft may be performed (49,50) (Fig 16).
Other Abnormalities

The IVC can be particularly important in emergency imaging, with two entities requiring immediate recognition and treatment: slitlike IVC and aortocaval fistula (Fig 17). A slitlike IVC is defined as an IVC with a transverse-to-anteroposterior diameter ratio greater than 3:1 that is seen at multiple levels. In patients with trauma, a slitlike IVC is associated with significant hypotension and impending shock. However, it is a nonspecific finding in patients without a history of trauma, and up to two-thirds of patients with this imaging finding can be euvoletic and normotensive (1,2).

An aortocaval fistula is a rare but often catastrophic complication of abdominal trauma or abdominal aortic aneurysm. Patients often present with acute symptoms related to
high-throughput congestive heart failure, and 80%–90% of aortocaval fistula formation is the result of rupture or erosion of an abdominal aortic aneurysm into the IVC. The remaining cases are attributable to posttraumatic fistulization, although neoplastic and inflammatory causes are also possible (51). The imaging findings of an aortocaval fistula include early contrast opacification in the IVC during arterial phase imaging, loss of a normal fat plane between the aorta and IVC, and enlarged IVC (due to the high flow state). Prompt recognition and early surgical or endovascular repair are important for improved patient outcomes (51).

Bland thrombus is the leading cause of IVC obstruction, which places the patient at high risk for pulmonary embolism. Risk factors for thrombus formation include a hypercoagulable state, malignancy, venous stasis, focal compression, and IVC filters. Bland thrombus in the IVC may be isolated but most often extends from pelvic and lower extremity deep vein thrombosis. Unlike tumor thrombus, bland thrombus lacks luminal expansion and enhancement (1). Anticoagulation drugs are the mainstay of therapy. However, IVC filters can be placed if anticoagulation therapy is contraindicated (1,2).

Gonadal vein thrombophlebitis commonly involves the ovarian vein in postpartum patients; approximately 80% of cases are right sided, but the entity can extend into the IVC (52). The ovarian vein is expanded by bland thrombus; this expansion results in enhancement of the venous wall and perivascular inflammation (Fig 18). Treatment includes anticoagulation drugs and antibiotics (52).

Calcified IVC thrombus was first reported in 1961 and is thought to occur primarily in pediatric populations. Potential causes are abdominal malignancy, adrenal hemorrhage, coagulopathy, and infection. IVC calcification in adults is exceptionally rare (53).

### IVC Imaging Pitfalls

The pitfalls of IVC imaging involve mistaking artifacts for IVC thrombus. Pseudolipoma is a volume-averaging artifact due to prominent pericaval fat above the caudate lobe and is often
seen in patients with cirrhosis. Admixture artifact may occur at the level of the renal veins when contrast-enhanced blood from the kidneys mixes with nonenhanced blood from the lower extremities (Fig 19). Admixture artifact can also result from retrograde flow of contrast material into the IVC because of right heart failure or a contrast material injection rate faster than 3 mL/sec (1,2).

**Conclusion**

Recognition of IVC processes is essential to patient treatment. The spectrum of abnormalities involving the IVC include congenital anomalies, which may be mistaken for adenopathy or may cause difficulty in vascular access and IVC filter placement; neoplasms, which may cause venous occlusion and alter staging and surgical management; and other postsurgical, traumatic, and infectious causes. Knowledge of these processes can markedly affect patient care, and evaluation of the IVC should be a fundamental part of the search pattern for abdominal radiologists. This will allow detection of a congenital or postsurgical abnormality. An accurate and informative description of the IVC is particularly important in the context of abdominal neoplasm, recent liver transplantation, and trauma. Radiologists should be aware that routine abdominal CT with a delay of 60–70 seconds after injection of contrast material may lead to admixture artifact in the infrarenal IVC, potentially resulting in the misdiagnosis of a filling defect. A delay of 70–90 seconds will yield more uniform contrast enhancement. In pediatric patients, US may be a valuable first-line modality if a caval pathologic condition is suspected, because Doppler US can be used to evaluate IVC thrombus without the risk of radiation exposure.

**References**


**Figure 18.** Right gonadal vein thrombophlebitis in a postpartum patient. Axial (a) and coronal (b) CT images show an enlarged well-defined right-sided tubular structure with peripheral enhancement and a low-attenuation filling defect (arrowhead), findings consistent with thrombus and inflammation of the right ovarian vein. The vein drains directly into the IVC, and caval extension of the thrombus (arrow in b) is visible.
Figure 19. Pitfalls of IVC imaging. (a–c) Axial CT images show a pseudolipoma, which is an IVC filling defect (arrow in a) caused by partial volume averaging of prominent pericaval fat (arrowhead) that may simulate IVC thrombus. (d, e) Axial CT images show admixture artifact created by contrast-enhanced blood from the renal veins (arrowhead in d) mixing with nonenhanced venous blood from the lower extremities (arrowhead in e) to simulate a filling defect at the level of the renal veins (arrow in d).