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From congenital variants to critical clues: A radiologic review of inferior vena cava pathologies

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ABSTRACT

Inferior vena cava (IVC) pathology is often underrecognized on non-dedicated imaging examinations, yet it carries significant diagnostic and clinical implications, particularly in acute emergency settings. This review highlights the complementary roles of ultrasound (US) and computed tomography (CT) in the assessment of IVC abnormalities. The US provides real-time, bedside evaluation of IVC patency, caliber, and hemodynamics, whereas CT offers detailed anatomic and structural characterization critical for diagnosis and management. The review aims to systematically discuss congenital variants, thrombotic, traumatic, systemic conditions, and neoplastic involvement of the IVC, emphasizing their imaging features and clinical relevance.

Keywords: Congenital, Computed tomography, Inferior vena cava, Thrombus, Trauma, Ultrasound

INTRODUCTION

The inferior vena cava (IVC) is a central venous structure with diagnostic importance in both emergency medicine and systemic conditions. Although often less emphasized than the aorta, it serves as an early indicator of a broad range of pathologic conditions, including traumatic injuries, thrombotic events, neoplastic involvement, and congenital anomalies. However, IVC abnormalities frequently go unrecognized on non-vascular imaging examinations, leading to missed opportunities for critical diagnosis.

This review is organized to provide an overview of embryologic development, followed by a discussion of imaging techniques, imaging features, and clinical implications of congenital variations, trauma, thrombosis, and neoplastic conditions. Finally, the clinical utility of functional assessment of IVC caliber and collapsibility with ultrasound (US) is discussed in the context of hemodynamic evaluation and volume responsiveness in acute care scenarios.

NORMAL ANATOMY

The IVC is the largest systemic vein, formed at the level of the fifth lumbar vertebra (L5) by the union of the common iliac veins. It ascends along the right side of the aorta, traverses the diaphragm, and terminates in the right atrium. Along its course, it receives blood from key

tributaries, including the lumbar, renal, gonadal, hepatic, and phrenic veins. Anatomic variants, such as duplicated IVC or left-sided IVC, have clinically important implications for imaging interpretation and interventional planning.[1,2]

IMAGING

Multiple imaging modalities, including US, computed tomography (CT), magnetic resonance imaging (MRI), and catheter venography, are used to evaluate the IVC, each with distinct advantages and limitations. US particularly with color Doppler, is a non-invasive tool for initial assessment of IVC anatomy, patency, blood flow, and thrombus detection. It is particularly useful in distinguishing thrombus from artifactual filling defects observed on intravenous (IV) contrast-enhanced CT or MRI. Using contrast-enhanced US further enhances diagnostic confidence in differentiating bland from tumor thrombus.[3]

CT and MRI offer high-resolution, cross-sectional evaluation of the IVC and its tributaries. These modalities allow for accurate IVC caliber assessment, detection of congenital variants, and detailed characterization of thrombus extent and adjacent pathology.[4] The coronal plane is particularly effective for visualizing the IVC course.[5] IV contrast-enhanced CT performed at 70-90 s or longer offers uniform opacification of the IVC compared with routine portal venous phase imaging at 60-70 s, where mixing of the blood and contrast may challenge thrombosis assessment.[6] In contrast, non-contrast CT can result in a diagnostic pitfall, as IVC variants and collaterals can mimic a mass or lymphadenopathy.

MRI provides an operator-independent alternative to US and avoids the use of ionizing radiation. It can be performed with gadolinium-based IV contrast or using a balanced steady-state free precession sequence, a rapid gradient-based echo sequence that is less susceptible to motion artifacts and flow-related signal loss.[7] This technique is particularly advantageous in patients with renal impairment, where contrast use may be contraindicated.[5,6]

PATHOLOGIES

Congenital anomalies

Congenital anomalies of the IVC arise from disruptions in the normal embryologic development of the posterior cardinal, subcardinal, and supracardinal veins between the 6th and 8th weeks of gestation [Figure 1]. The IVC is composed of four segments: infrarenal, renal, suprarenal, and hepatic segments.[8-10] The congenital variants can complicate interventional procedures, particularly IVC filter placement in common variants such as duplicated and left-sided IVC. Rare variants, including absent infrarenal IVC, hypoplastic IVC, and IVC associated with polysplenia syndrome, may impair venous return and increase the risk of deep vein thrombosis (DVT). Recognition of these variants on imaging is essential to avoid misdiagnosis and guide appropriate management [Table 1].[11]

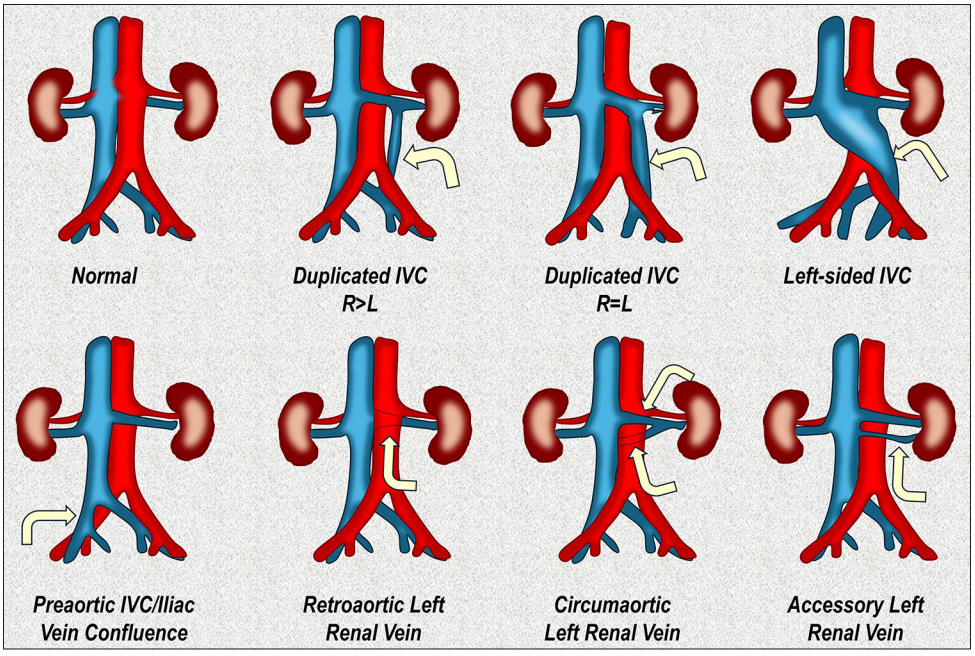

Figure 1: Congenital anomalies and variants of the inferior vena cava (IVC).Schematic illustration demonstrating developmental deviation leading to IVC variants. R>L, right-sided IVC bigger than left; R=L, right and left-sided IVC of similar size.

An IVC anomaly should be considered as a potential differential diagnosis in young patients without an underlying cause for hypercoagulability or malignancy who present with unexplained venous thrombosis, recurrent, refractory lower extremity ulcers, varicose veins, and superficial collaterals.[12]

DUPLICATED IVC

Duplicated IVC is seen in 0.2-3% of the population, resulting from the persistence of the right and left supracardinal veins.[10] The left IVC drains into the left renal vein, which then crosses the midline to join the right-sided IVC. The respective common iliac veins drain into their respective sides of the IVCs.[13] This variant is clinically important in planning filter placement, which may require bilateral infrarenal filters or a single suprarenal filter [Figure 2].

LEFT-SIDED IVC

A left-sided IVC prevalence is estimated between 0.2% and 0.5%. It occurs due to the regression of the right supracardinal vein and persistence of the left supracardinal vein. The left-sided IVC joins the suprarenal IVC through the left renal vein [Figure 3].[10] Although often asymptomatic, this anomaly may mimic retroperitoneal lymphadenopathy on non-contrast imaging examinations and pose technical challenges during nephrectomy or adrenalectomy, particularly when associated with other venous anomalies such as multiple renal veins. In addition, placing an IVC filter through transjugular access can be challenging in patients with common iliac vein thrombosis.[6,8,14]

ABSENT INFRARENAL IVC

The prevalence of an absent infrarenal IVC with preserved suprarenal segment is <1%, with approximately 5% of young adult patients being in the third to fourth decades of life.[15,16] It is often attributed to intrauterine or perinatal thrombosis affecting the posterior and supracardinal veins rather than true agenesis. It is associated with well-developed collateral pathways through azygos, hemiazygos, and lumbar veins and may delay diagnosis due to compensatory collaterals [Figure 4]. The lumbar collateral vessel can sometimes mimic a paraspinal mass.[8]

HYPOPLASTIC IVC

The underdevelopment of the IVC is a rare condition, accounting for <1%.[17] It results from the failure of vein development, including vitelline, subcardinal, and supracardinal veins.[18,19] Most cases are asymptomatic, due to a well-developed collateral system [Figure 5]. They may be incidentally discovered during imaging for chronic venous insufficiency or unexplained DVT.

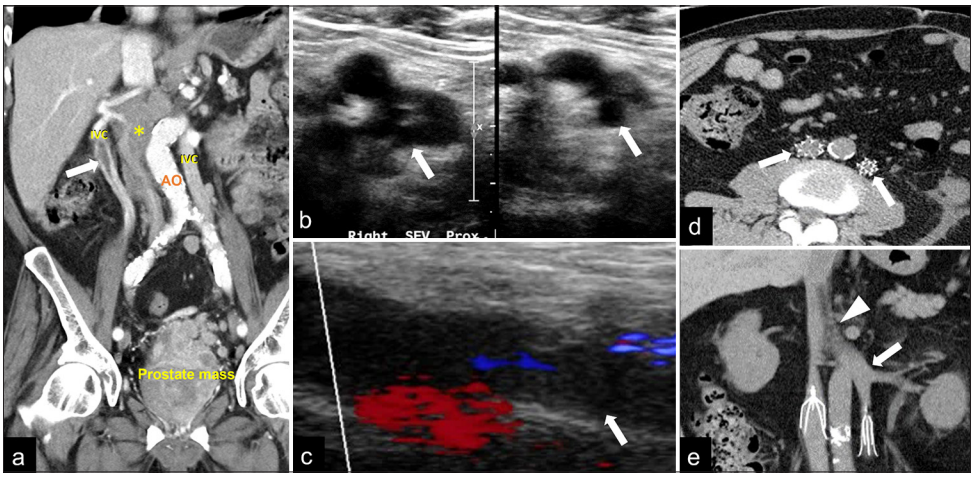

Figure 2: A 77-year-old male with prostate cancer and duplicated IVC. (a) Contrast-enhanced CT (CECT) demonstrates duplicated IVCs, with a near-occlusive thrombus in the right-sided IVC (white arrow) and metastatic nodal conglomerate (yellow asterisk). (b) Gray-scale US in a different case, a 62-year-old man with left flank pain, right leg swelling, and edema, shows non-compressible right femoral and profunda femoral veins (white arrows). (c) Color Doppler US demonstrates absent flow (white arrow). (d) Axial CECT showed duplicated IVCs (not shown). Two infrarenal filters were placed in each IVC (arrows) to prevent pulmonary embolism. (e) Coronal CECT shows the left-sided IVC draining into the left renal vein (white arrow). The left renal vein then crosses to join the right IVC, maintaining a normal suprarenal anatomy. Mixing artifact is noted as the renal vein drains into the right-sided IVC (white arrowhead).

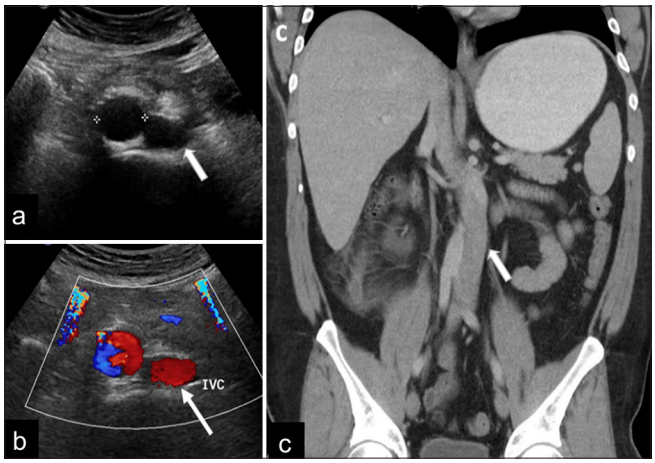

Figure 3: A 65-year-old male presented to the emergency room with epigastric pain and left-sided IVC. (a) The gray-scale and (b) color Doppler US images show a tubular structure (white arrow) to the left of the aorta (calipers). The tubular structure is consistent with left-sided IVC. (c) Coronal contrast-enhanced CT confirms the finding (white arrow).

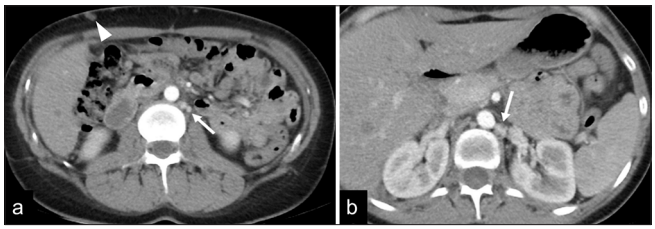

Figure 4: A 27-year-old male with abdominal pain. (a, b) Axial contrast-enhanced CT shows lumbar collaterals (arrows) and superficial collaterals (arrowhead) related to absent infrarenal IVC.

POLYSPLENIA WITH IVC INTERRUPTION

Polysplenia syndrome with IVC Interruption accounts for 0.6% of cases. It involves failure of the right subcardinal-hepatic anastomosis and atrophy of the right subcardinal vein. This results in the absence of the intrahepatic IVC and azygos or hemiazygos continuation [Figure 6].[8,14,18] This variant is associated with other congenital anomalies and should prompt further evaluation for heterotaxy syndrome.[20]

THROMBOSIS AND TUMOR THROMBUS

IVC thrombosis (IVCT) is an uncommon but clinically significant form of venous thromboembolism. It is associated with complications such as post-thrombotic syndrome (90%), venous claudication (45%), pulmonary embolism (30%), and venous ulceration (15%).[21] The development of IVCT involves a combination of factors known as the Virchow triad, which includes hypercoagulability, stasis, and endothelial injury.[22] IVCT may be occlusive or non-occlusive [Figure 7]. In occlusive thrombosis, collaterals develop over time to restore the venous return [Figure 8].

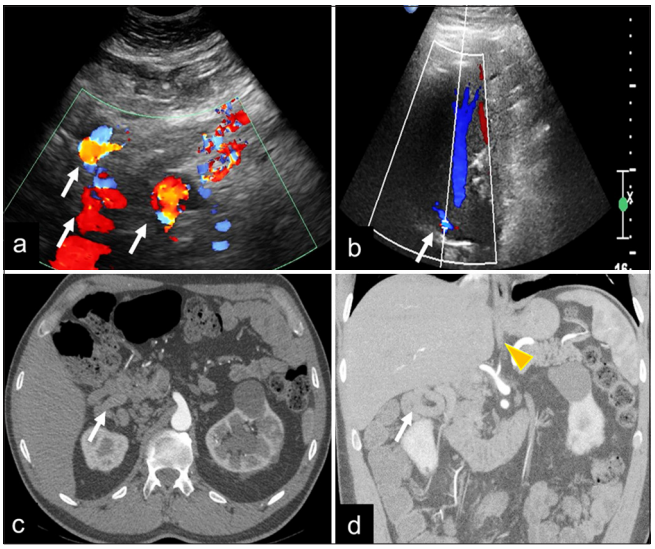

Figure 5: A 37-year-old male with abdominal pain and hypoplastic IVC. (a) Transverse color Doppler US demonstrates multiple collateral veins in the right upper quadrant (white arrows). (b) Sagittal US image shows a diminutive IVC (white arrow). (c) Axial and (d) coronal CECT confirms the presence of numerous collateral veins (white arrow) and a hypoplastic IVC (orange arrowhead).

Several factors predispose individuals to IVCT. These include inherited thrombophilia (e.g., factor V Leiden), hormone therapy, smoking, obesity, pregnancy, and malignancy. Iatrogenic causes from femoral vein catheterization, pacemaker leads, or cardiopulmonary bypass are increasingly implicated. IVC filters, especially retrievable types, are also increasingly linked to IVCT risk, with reported incidence rates ranging from 0.6% to 8% and up to 33% for permanent filters.[21, 23]

CT and MRI are primarily used to assess IVCT, which allows for evaluating the level and extent of thrombosis. Differentiation between a true thrombus and pseudo-thrombus, caused by contrast mixing artifact in the suprarenal IVC, is essential [Figure 9]. Delayed imaging improves diagnostic accuracy by allowing proper mixing of blood and contrast media. US is a useful adjunct to exclude thrombosis in such cases.[24,25] Volume averaging from pericaval fat above the caudate lobe can sometimes mimic occlusion and can be distinguished using coronal reconstructions.[6]

Thrombi are categorized as bland or tumor thrombus based on enhancement patterns and internal vascularity. Bland thrombus is non-enhancing and avascular, while tumor thrombus demonstrates internal enhancement due to neovascularity [Table 2].[6]

Management of bland IVCT primarily involves anticoagulation as the first line of therapy. In acute or

subacute cases (<28 days), catheter-directed thrombolysis or pharmacomechanical catheter-directed thrombolysis may be used when bleeding risk is low. Endovascular interventions, including percutaneous transluminal angioplasty and IVC stenting, are effective in chronic thrombosis (>28 days) or recurrent thrombosis.[21,26] Long-term follow-up imaging is essential to assess stent patency and resolution of thrombus. Tumor thrombus treatment is guided by the underlying malignancy. Surgical resection is often indicated, such as in cases of renal cell carcinoma (RCC) with caval extension.

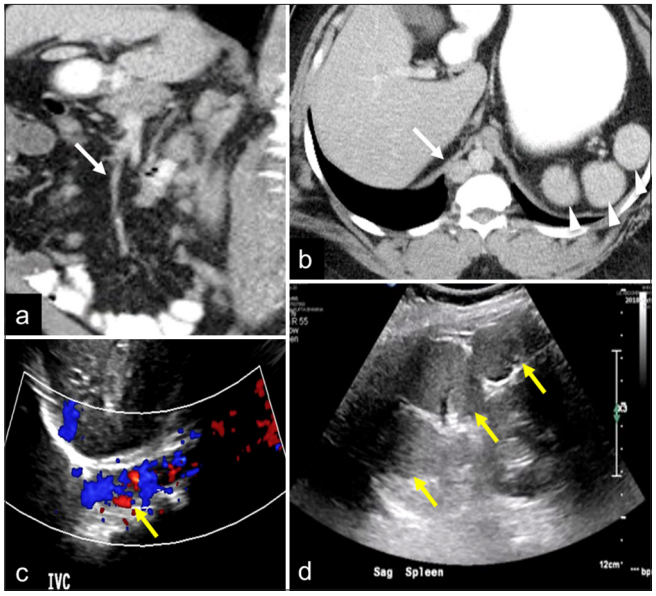

Figure 6: A 37-year-old male with numerous left-sided soft tissue lesions in the abdomen. (a) Coronal contrast-enhanced CT (CECT) demonstrates a diminutive IVC (white arrow). (b) Axial CECT shows the azygous continuation of the IVC (white arrow) and multiple splenules (white arrowheads). (c) Color Doppler US correlate demonstrates a small-caliber IVC (yellow arrow). (d) Grayscale image shows multiple splenules (yellow arrows) in polysplenia syndrome with IVC Interruption.

TRAUMA

Traumatic injury of the IVC is rare, with an incidence of approximately 1% due to blunt trauma and 0.5-5% due to penetrating injuries.[27] These injuries typically result from high-energy mechanisms and are frequently associated with multisystem trauma. Mortality and morbidity are substantially influenced by factors such as the mechanism of injury, the anatomic site involved, accompanying injuries, and the individual's physiological condition. The anatomic level of injury significantly influences prognosis, with reported mortality rates of 100% for suprahepatic, 78% for retrohepatic, and 33% for suprarenal IVC injuries.[28,29]

Contrast-enhanced CT imaging in the trauma setting plays a critical role in detecting IVC injury, especially during venous and delayed phases. Key findings include contour irregularity, active contrast extravasation, pericaval hematoma, intraluminal thrombus, and pseudoaneurysm formation.[10,30]

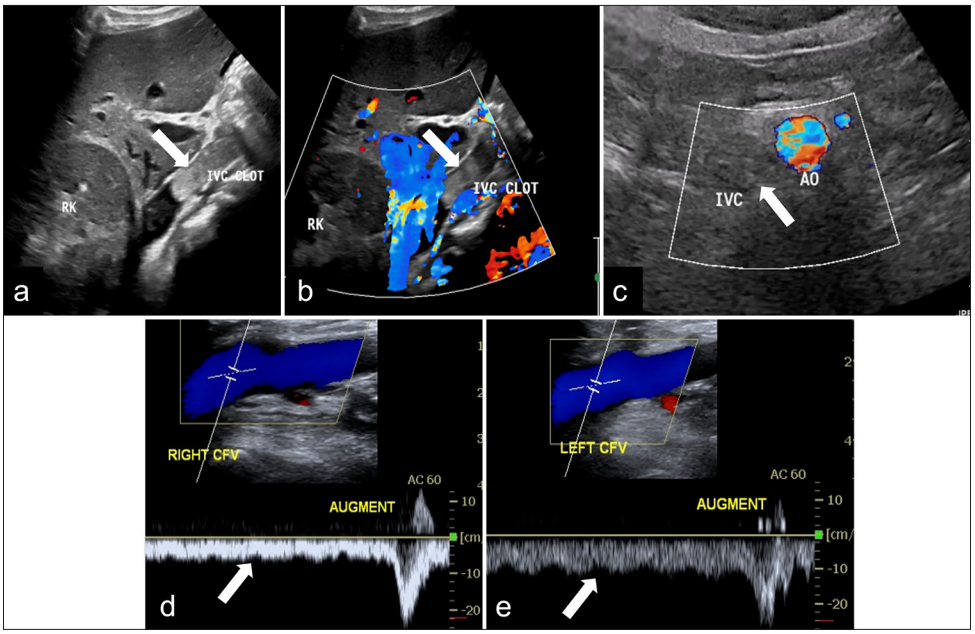

Figure 7: A 36-year-old male with lower extremity swelling due to IVC occlusive thrombosis. (a) Gray-scale longitudinal ultrasound image demonstrates echogenic thrombus in the IVC (white arrow). (b, c) Color Doppler images in two different planes show absent blood flow (white arrows). (d,e) Spectral Doppler of the right and left common femoral veins shows a monophasic waveform (white arrows) secondary to proximal occlusion. In this case, the thrombosed IVC.

Table 1: Congenital anomalies of the IVC.

Variant	Embryologic basis	Prevalence (%)	Imaging findings	Clinical implications
Duplicated IVC	Persistence of both supracardinal veins	0.2-3	Two IVCs; left drains into the left renal vein, which crosses the midline to join the right-sided IVC	Filter placement planning; risk of thrombus recurrence
Left-sided IVC	Regression of the right, persistence of the left supracardinal vein	0.2-0.5	IVC located on the left side, drains into the left renal vein	May mimic lymphadenopathy; surgical planning
Absent Infrarenal IVC	Intrauterine/perinatal thrombosis of the infrarenal segment	<1	Absence of infrarenal IVC, dilated azygos/hemiazygos veins	Increased DVT risk in young patients
Hypoplastic IVC	Incomplete development of vitelline/subcardinal/ supracardinal veins	<1	Small-caliber IVC with prominent collaterals	Often incidental; associated with chronic venous insufficiency
Polysplenia with IVC Interruption	Failure of the right subcardinal–hepatic anastomosis	~0.6	Absent intrahepatic IVC with azygos/hemiazygos continuation	Suggests heterotaxy; consider associated congenital anomalies

IVC: Inferior vena cava, DVT: Deep vein thrombosis

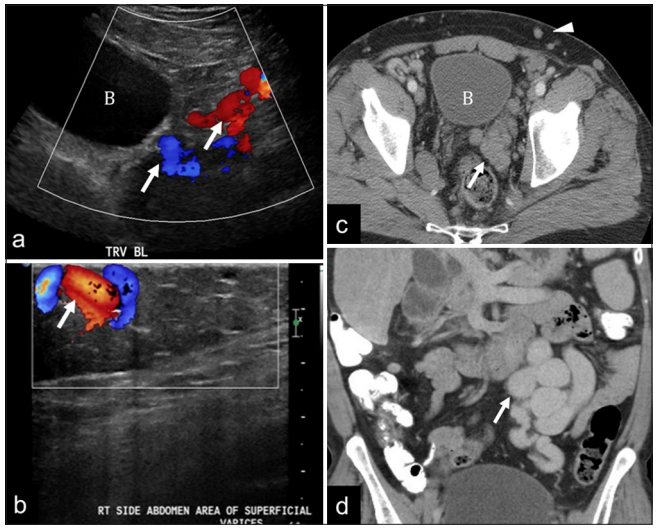

Figure 8: A 67-year-old woman with left-sided discomfort due to chronic IVC occlusion and collateral formation. (a) A transverse plane color Doppler ultrasound image of the pelvis demonstrates collateral veins (white arrows) adjacent to the bladder (B). (b) The color Doppler images also show superficial collateral veins (white arrow). (c) Axial contrast-enhanced CT (CECT) confirms collateral veins (white arrow) adjacent to the bladder (B) and superficial collateral veins (arrowhead). (d) Coronal CECT shows large mesenteric collateral veins (white arrow) from the sequela of chronic IVC occlusion.

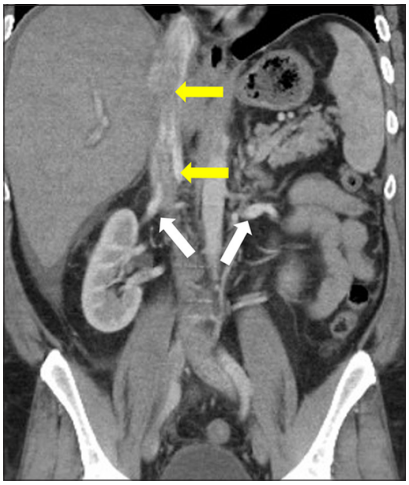

Figure 9: A 45-year-old male with congestive heart failure. Coronal contrast-enhanced CT image demonstrates well-opacified renal veins (white arrows) draining into the non-opacified IVC. The admixture of contrast-opacified and unopacified blood creates a mixing artifact in the suprarenal IVC (yellow arrows).

LACERATION

IVC laceration, though rare, is a surgical emergency and is associated with retroperitoneal hematoma and adjacent organ injury (e.g., liver lacerations). IVC laceration can lead to life-threatening hemorrhage and hemodynamic instability. Timely recognition of trauma, CT, and surgical consultation is crucial to reduce the high associated mortality [Figure 10].[10]

PSEUDOANEURYSM

Pseudoaneurysm typically arises from penetrating trauma, IVC filter removal, or underlying pathologies, including infection or inflammation [Figure 11].[31-33] It can be

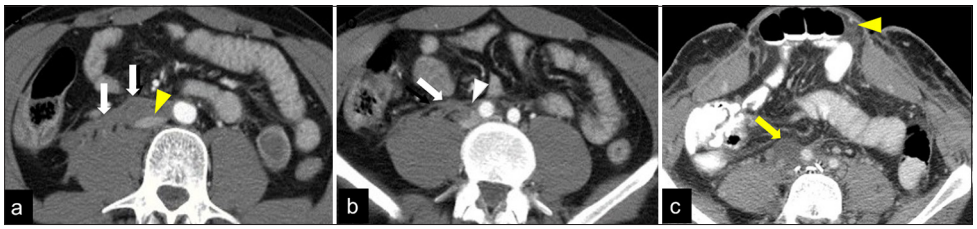

Figure 10: A 34-year-old male after a motor vehicle collision. (a, b) Axial contrast-enhanced CT(CECT) images demonstrate a retroperitoneal hematoma (white arrows) anterior to the infrarenal IVC. The inferior vena cava is flat due to a volume-depleted state (yellow arrowhead) and shows contour irregularity at the level of the aortic bifurcation (white arrowhead) from a laceration, which was confirmed at surgery. (c) Postoperative CECT image demonstrates surgical clips adjacent to the IVC and a small post-surgical seroma (yellow arrow). Additionally, rectus diastasis and a ventral hernia containing loop of non-obstructed bowel are seen (arrowhead).

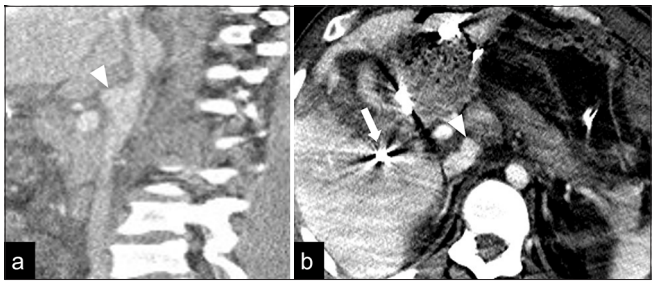

Figure 11: A 29-year-old male with a gunshot wound to the abdomen. (a) Sagittal and (b) axial contrast-enhanced CT image demonstrates a focal outpouching of the IVC (white arrowhead) due to a pseudoaneurysm and a ballistic fragment within the liver (white arrow).

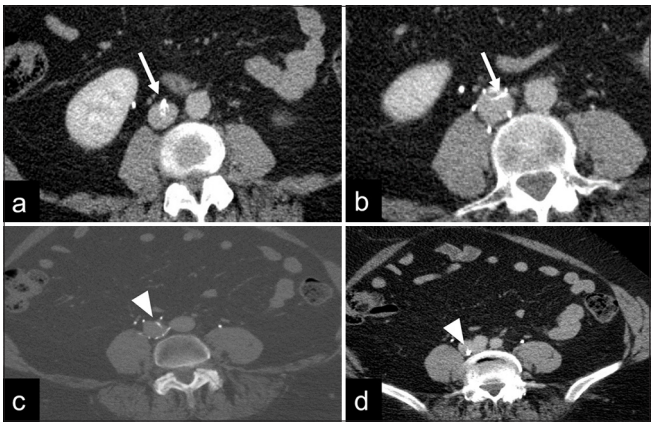

Figure 12: A 62-year-old woman with new shortness of breath after IVC filter placement. (a, b) Axial contrast-enhanced CT images show a low-lying filter located just above the confluence of the common iliac veins. The filter hook extends beyond the IVC wall (white arrows). (c, d) Axial contrast-enhanced CT images show a fractured strut within the IVC (white arrowheads). This filter tilt resulted in breakthrough pulmonary embolism (not shown).

complicated by arteriovenous fistula formation, presenting with lower extremity venous hypertension, bruit, or pulsatile mass. Pseudoaneurysm management includes endovascular repair to prevent complications such as rupture or thrombosis.[34]

IATROGENIC TRAUMA-DISPLACED IVC FILTER

While not strictly "traumatic," IVC filters may cause vascular injury during placement or removal. Complications include filter-related thrombosis, filter tilt, migration, caval wall perforation [Figure 12], and penetration into adjacent structures.[35-38] The complications are managed by repositioning or retrieving the filter. Longer dwell times, increased tilt angles, and hook embedment are associated with increased difficulty in retrieving the filter. Management may involve endovascular retrieval or surgical removal in severe cases.[39,40]

SYSTEMIC PROCESSES

IVC serves as a dynamic marker of hemodynamic status, with its size and respiratory variation reflecting changes in intravascular volume and central venous pressure. In critically ill patients, including those with trauma, sepsis, congestive heart failure, acute kidney injury, and portosystemic shunts, IVC caliber assessment plays a key role in volume evaluation and monitoring response to resuscitation.

Table 2: Imaging features of bland vs. tumor thrombus.

Feature	Bland thrombus	Tumor thrombus
Enhancement	None	Present due to vascularity
Associated mass	Usually absent	Often contiguous with a tumor
Margins	Smooth, sharply defined	Irregular, expanding lumen
Vascular flow on Doppler ultrasound	No internal flow	Internal flow may be detected

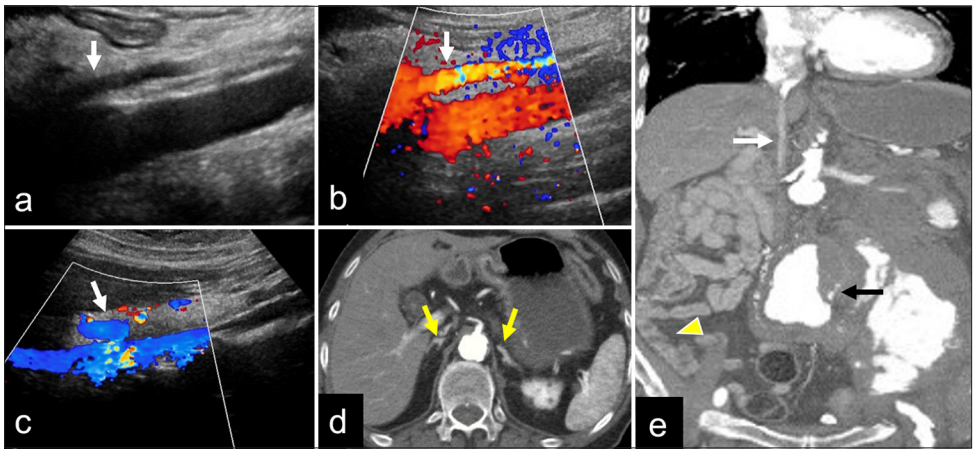

Figure 13: A 72-year-old man with hypovolemia and a ruptured aortic aneurysm with flattened IVC. (a) Longitudinal gray-scale and (b, c) color Doppler US images of the IVC show a small-caliber IVC (white arrows). (d) Axial contrast-enhanced CT (CECT) image shows hyperdense adrenals (yellow arrows). (e) Coronal CECT shows slit-like caliber of the retrohepatic IVC (white arrow), retroperitoneal hematoma with contrast extravasation from the ruptured aorta (black arrow), and bowel mucosal hyperenhancement secondary to ischemia (yellow arrowhead).

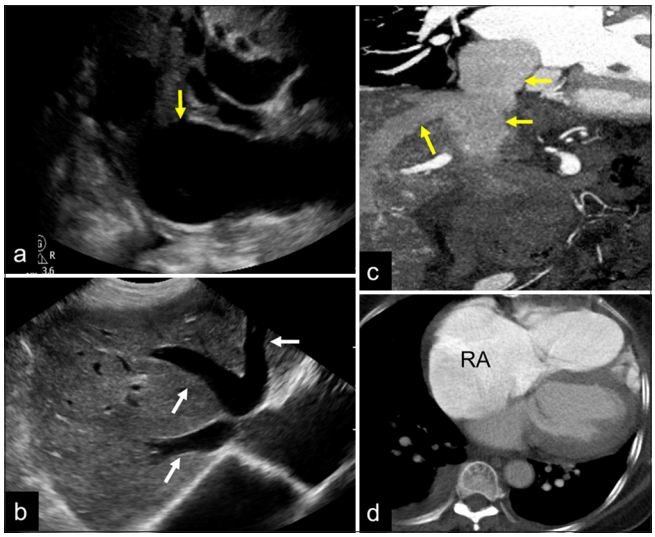

Figure 14: A 63-year-old woman with shortness of breath. (a, b) Gray-scale US image demonstrates a dilated IVC (yellow arrow) and hepatic veins (white arrows). (c) The coronal contrast-enhanced CT image shows the US correlate of dilated IVC and hepatic veins (yellow arrows). (d) Axial contrast-enhanced CT image additionally shows cardiomegaly with a large right atrium (RA) due to congestive heart failure.

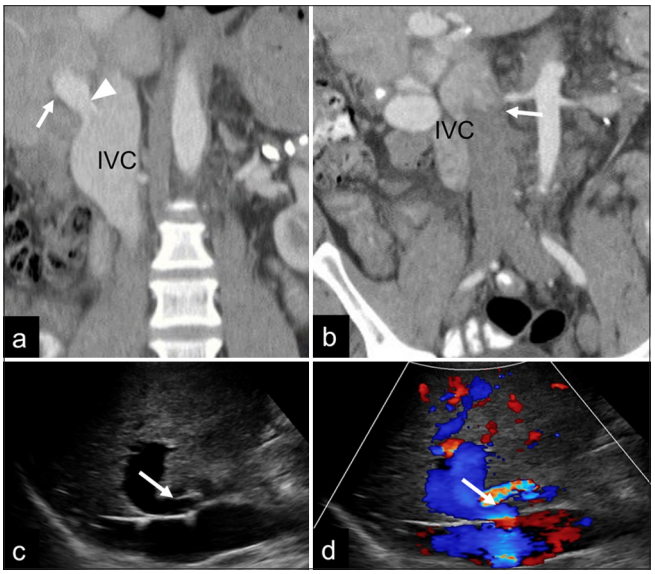

Figure 15: A 28-year-old male with biliary atresia and iatrogenic portocaval shunt. (a) Coronal contrast-enhanced CT (CECT) image demonstrates a shunt (white arrowhead) between the portal vein (white arrow) and the infrahepatic IVC. (b) Coronal CECT shows mixed opacification of the IVC caudal to the anastomosis (white arrow). (c) Gray-scale US image shows the portocaval shunt (white arrow). (d) Color Doppler confirms the patency of the shunt with hepatofugal flow from the portal vein into the IVC (white arrow).

FLATTENED IVC

A flattened IVC, characterized by a transverse-to-anteroposterior diameter ratio of >3:1 at multiple levels, correlates with hypotension and hypovolemia in the trauma setting [Figure 13]. It is frequently observed in "hypoperfusion complex" on CT, often accompanied by decreased aortic caliber, diffuse bowel distention with fluid, peritoneal fluid collections, and hyperenhancement of bowel wall, kidneys, and pancreas. In non-trauma patients, especially the elderly, a flattened IVC may indicate underlying volume depletion.[41,42]

HEART FAILURE AND DILATED IVC

IVC lacks valves and exhibits diameter changes in response to respiratory cycles and intrathoracic pressure shifts. In emergency

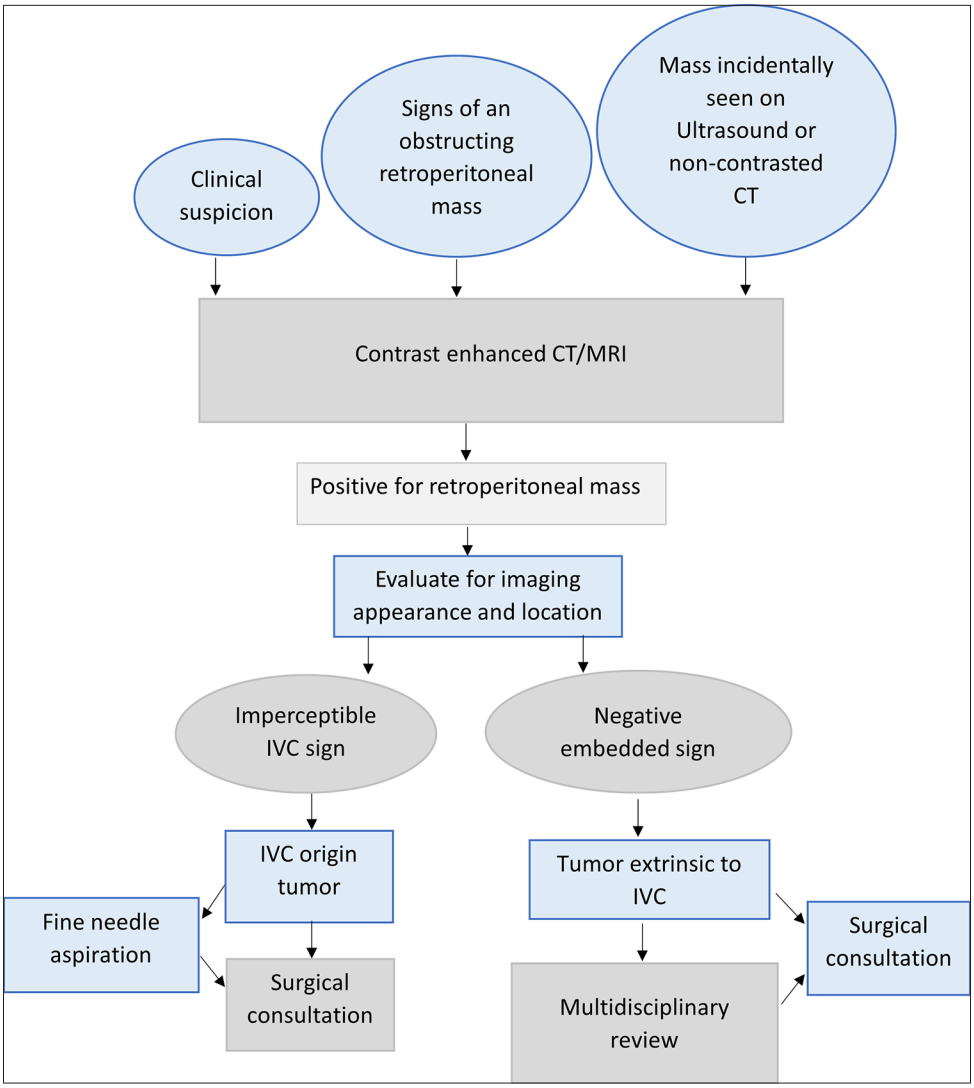

Figure 16: The flow chart shows the imaging-based diagnostic approach to inferior vena cava masses and recommendations. CT: Computed tomography, MRI: Magnetic resonance imaging, IVC: Inferior vena cava.

and critical care settings, point-of-care US is routinely used to assess IVC size and respiratory variation as a non-invasive surrogate for intravascular volume status. A collapsible IVC during inspiration typically suggests volume responsiveness, while a dilated, non-collapsing IVC indicates volume overload or elevated right atrial pressure. These measurements help guide fluid resuscitation and evaluate cardiac function.[43-46]

In heart failure due to coronary artery disease, hypertension, valvular heart disease, or cardiomyopathies, the IVC often appears persistently dilated with minimal or absent respiratory variation [Figure 14].[47,48] This finding correlates with systemic venous congestion and is associated with adverse outcomes, including increased mortality.[49,50]

On contrast-enhanced CT, retrograde opacification of the IVC or hepatic veins is a specific (98%) indicator of right-sided heart dysfunction, although its diagnostic reliability diminishes at higher contrast injection rates.[51]

EXTRAHEPATIC PORTOCAVAL SHUNT (EPCS)

EPCSs can be either congenital or iatrogenic. Congenital EPCS are classified into type I (absent intrahepatic portal vein) and type II (intrahepatic portal vein present).[52] These shunts divert portal blood away from the liver, potentially causing hepatic encephalopathy and liver dysfunction. Iatrogenic shunts, typically created to manage portal hypertension, carry similar risks.[53]

Doppler US in EPCS helps determine the patency and flow direction within the anomalous vessels [Figure 15]. Additional imaging findings may include reduced liver size, increased periportal echogenicity, and, over time,

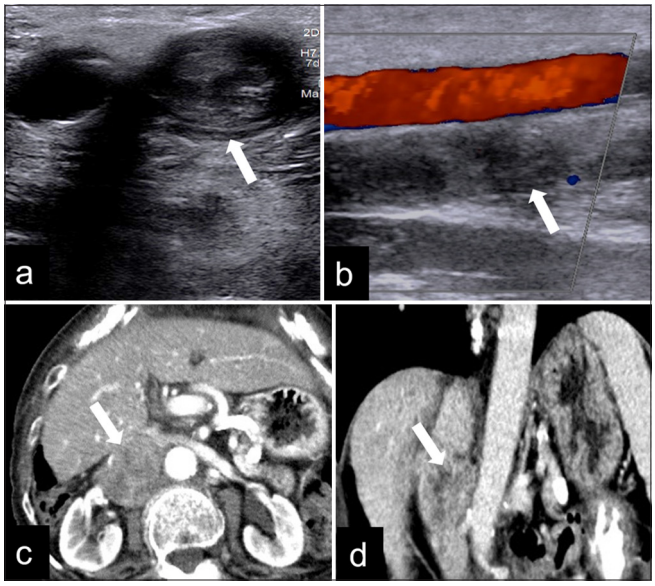

Figure 17: A 67-year-old male with abdominal pain and leg swelling. (a) Gray-scale US image demonstrates a distended common femoral vein with a mixed echogenicity intraluminal filling defect (white arrow). (b) Color Doppler image demonstrates a complete absence of venous flow (white arrow) consistent with an occlusive thrombus. (c) Axial and (d) coronal contrast-enhanced CT image shows a heterogeneously enhancing mass distending the infrarenal IVC (white arrow) secondary to a biopsy-proven leiomyosarcoma and a cause for the distal venous thrombosis.

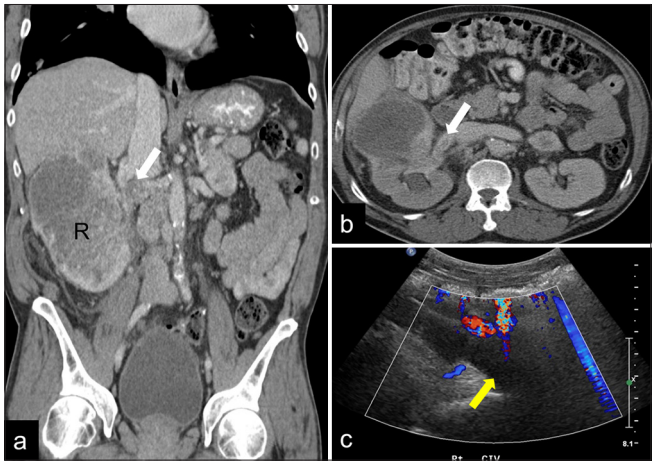

Figure 18: A 55-year-old male with flank pain and hematuria due to renal cell carcinoma. (a) Coronal contrast-enhanced CT (CECT) image demonstrates a right-sided renal mass (R) extending into the IVC (white arrow). (b) Axial CECT shows thrombus in the right renal vein (white arrow). (c) Color Doppler US shows absent venous flow in the right common iliac vein due to an occlusive thrombus (yellow arrow) resulting from stasis and proximal occlusion.

development of benign regenerative nodules. These nodules can be further characterized with contrast-enhanced US and cross-sectional imaging.[52-54]

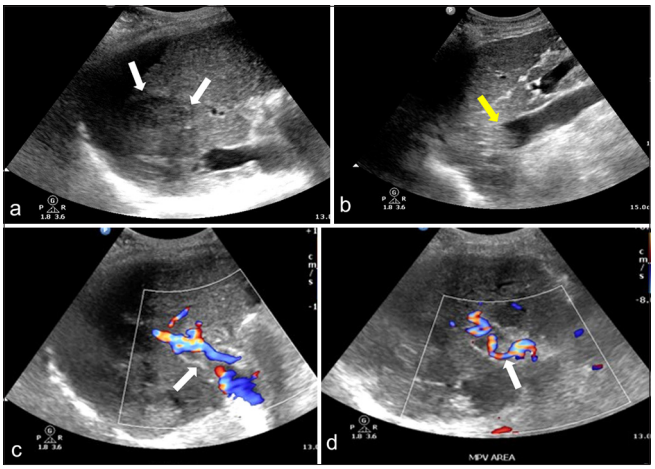

Figure 19: A 43-year-old woman with cirrhosis and right upper quadrant pain secondary to hepatocellular carcinoma with IVC and portal vein invasion. (a) Gray-scale US image of the liver demonstrates a hypoechoic mass (white arrows) against a heterogeneous, hyperechoic liver parenchyma caused by cirrhosis. (b) Gray-scale US image of the IVC demonstrates tumor extension into the retrohepatic IVC (yellow arrow). (c) Color Doppler images of the main portal vein show non-occlusive thrombus (white arrow). (d) Color Doppler images of the main portal vein show flow within the non-occlusive thrombus secondary to tumor invasion (white arrow).

NEOPLASMS

Neoplastic involvement of the IVC may arise from primary tumors, such as leiomyosarcoma, or more commonly from secondary invasion by adjacent malignancies, including RCC, hepatocellular carcinoma (HCC), adrenal cortical carcinoma, metastasis, and lymphoproliferative disease.[6] The imaging appearance, route of extension, and clinical implications vary with tumor type [Figure 16]. Multiplanar reconstructions are essential for delineating the extent of luminal invasion and assessing resectability. Contrast enhanced ultrasound (CEUS) can be an adjunct when findings are equivocal.[6]

LEIOMYOSARCOMA

Primary IVC leiomyosarcoma, though rare (<1%), remains the most common primary malignancy of the IVC.[6] It typically presents in females in the fifth to sixth decade and presents with vague symptoms such as abdominal pain, lower extremity edema, or DVT. Diagnosis relies on cross-sectional imaging, most commonly CT and MRI, to assess tumor location, extent, and vascular involvement.[55] On contrast-enhanced CT or MRI, it manifests as a lobulated, enhancing soft-tissue mass that may be intraluminal or extraluminal, the latter occurring in approximately two-thirds of cases [Figure 17].[6] Imaging findings suggesting IVC origin include the "imperceptible IVC sign," where the IVC wall is not visualized at the site of maximal contact with the mass,

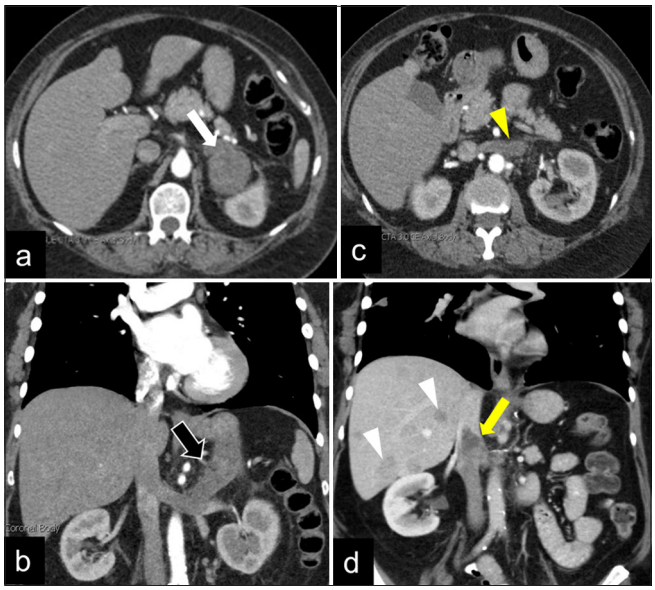

Figure 20: A 67-year-old woman with worsening abdominal pain due to left adrenal cortical carcinoma. (a) Axial contrast-enhanced CT (CECT) image demonstrates a left adrenal mass (white arrow). (b) Coronal CECT image shows the adrenal mass extends into the left adrenal vein (black arrow). (c) Axial CECT shows progression of tumor thrombus from the adrenal vein into the left renal vein (yellow arrowhead). (d) Coronal CECT shows tumor thrombus extending all the way into the IVC (yellow arrow), and there are multiple metastases (white arrowheads) in the liver.

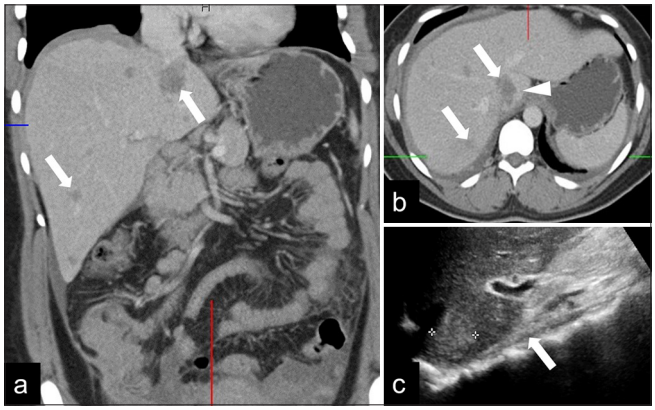

Figure 21: A 53-year-old male with abdominal pain and anemia secondary to colorectal cancer with metastases and IVC extension. (a) Coronal and (b) axial contrast-enhanced CT images demonstrate multiple liver metastases (white arrows) from a cecal carcinoma, with one metastatic lesion seen extending into and invading the IVC (white arrowhead). (c) Gray-scale US confirms the intravascular extension of the metastasis, with thrombus within the IVC (white arrow). Calipers shows the metastasis.

whereas the "negative embedded sign" indicates displacement of the IVC by the mass, suggesting an extrinsic tumor origin.[56] Because the IVC is easily visualized with US, image-guided biopsy can be performed for tissue confirmation.[57]

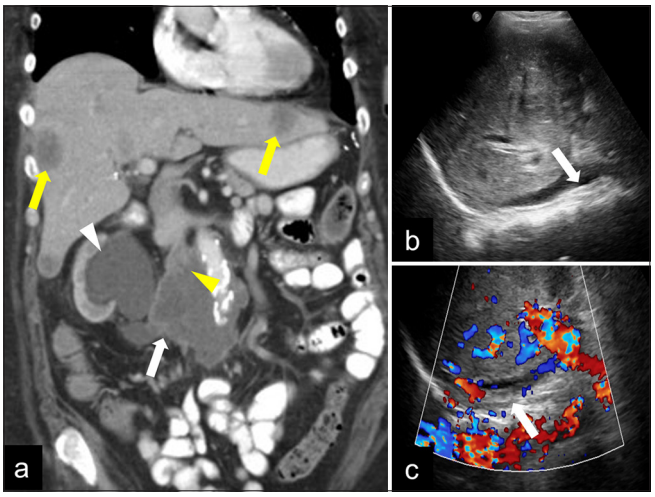

Figure 22: A 45-year-old woman with worsening hydroureteronephrosis secondary to uterine cancer with retroperitoneal lymphadenopathy and IVC extension. (a) Coronal contrast-enhanced CT image demonstrates right hydroureteronephrosis (white arrowhead) secondary to a large necrotic retroperitoneal nodal mass compressing the proximal ureter (white arrow) and invading the IVC (yellow arrowhead). Multiple metastases are present in the liver (yellow arrows). (b) Gray-scale and (c) color Doppler confirm intraluminal tumor invasion (white arrow).

IVC involvement is classified into three surgical levels: infrarenal, perihepatic, and suprahepatic, with higher levels correlating with increased surgical complexity and poorer prognosis.[58] Complete surgical resection with or without vascular reconstruction remains the mainstay of treatment, and adjuvant radiation or chemotherapy may be considered depending on stage and metastatic burden.[58-60]

SECONDARY IVC NEOPLASMS

Secondary involvement of the IVC by malignant neoplasm is more common than the primary tumor. On cross-sectional imaging, neoplastic involvement of the IVC exhibits distinct yet overlapping patterns based on tumor origin and extension route. The segment involved in the IVC demonstrates distension of the lumen and enhancement of the filling defect.[3] RCC is the most frequent cause, with venous extension occurring in 4-10% of cases [Figure 18]. Tumor thrombus may extend from the renal vein into the IVC or even reach the right atrium, categorized as T3b and T3c, respectively, in TNM staging.[6,10,14] HCC extends through hepatic veins, with reported IVC or right atrial involvement in 4.5-5.9% of cases [Figure 19]. This carries a poor prognosis and may lead to Budd-Chiari syndrome, presenting with ascites, abdominal pain, and hepatomegaly.[6,61] Adrenal cortical carcinoma, though rare, can directly invade the IVC through the adrenal vein or exert mass effect, displacing the vessel [Figure 20].[10,61]

Various other malignancies with metastasis to the liver pose a risk of extending to the IVC and hepatic vasculature [Figure 21]. Retroperitoneal lymphadenopathy, benign or malignant, may cause extrinsic IVC compression [Figure 22].[5,62] This, in turn, leads to impaired venous return, lower extremity edema, thrombosis, and risk for pulmonary embolism. On CT and MRI, such mass effect manifests as narrowing, displacement, or complete effacement of the lumen.[5] Although less sensitive for deep retroperitoneal disease, US may detect altered Doppler waveforms when hemodynamically significant compression is present.[6] Management ranges from treating the underlying cause with chemotherapy, irradiation, or immunosuppressive therapy to endovascular stent placement or thrombectomy.

CONCLUSION

The IVC is often overlooked, but a clinically important source of pathology on non-dedicated imaging examinations, especially in emergency and acute care settings. US and CT offer a comprehensive multimodal assessment. The US provides dynamic physiological data such as collapsibility and flow direction, while CT delivers high-resolution structural details and broad anatomical context. Early detection of IVC abnormalities is essential, as it can significantly influence patient management and prognosis. Looking ahead, the use of artificial intelligence in automated IVC segmentation and classification, along with increased application of contrast-enhanced US, shows promise for improving diagnostic accuracy and streamlining workflows in both emergency and routine practice.

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