Imaging gallery of Inferior Vena Cava and its Mass Lesions

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Authors: M. Bapat, D. P. Patkar, K. Agawane, S. puranik, M. R. Verma; Mumbai/IN
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Learning objectives

Learning objectives:

1. To describe the etiopathogenesis of inferior vena cava (IVC) involvement by intra or extra luminal masses, affecting the intrahepatic, suprarenal, renal and infrarenal segments.

2. To classify these into neoplastic and non neoplastic lesions with the help of characteristic computed tomography (CT) features.
Background

Background:

IVC is conduit of venous return to the heart from rest of the body so the masses involving IVC can have great impact on the other systems of the body. In this exhibit, we are trying to simplify the imaging approach on CT to classify these masses into neoplastic and non neoplastic and also whether these arise primarily from IVC or as an extension of pathology involving other organs.
Findings and procedure details

Procedure details:

Ultrasonography (USG) and Doppler are usually initial imaging modalities for abdominal pathologies and CECT is done to further investigate the lesion. Computed tomography (CT) is probably most common imaging modality for detection, characterization and determining extent of IVC pathology. 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]. Contrast enhanced CT (CECT) of abdomen and lower thorax is required to evaluate the extent of primary lesion and metastatic survey. Contrast enhanced MRI is advanced imaging technique for IVC lesions. In this exhibit we are discussing the imaging features of IVC masses mainly on CECT.

Embryologic Development and Normal Anatomic Structure:

The IVC is the main channel of venous return from the lower extremities and abdominal viscera. The mature IVC has four segments: the hepatic, suprarenal, renal, and infrarenal IVC [3]. The vitelline vein contributes to the hepatic segment of the IVC. The suprarenal IVC is composed of a segment of the right subcardinal vein. 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 [3, 4].

Imaging Findings:

IVC thrombus was commonly seen as associated finding in patients with hepatic /renal / testicular malignancies as well as incidental finding in other conditions. Extension of DVT into IVC is a common cause of bland thrombus.

IVC masses can be classified into non neoplastic and neoplastic causes. Non neoplastic masses include bland thrombus. Causes of bland thrombus formation are sepsis, trauma, dehydration, immobility, coagulopathy, inflammation, post surgical and vascular devices.

Neoplastic masses include benign and malignant pathologies. Leiomyomatosis come under benign masses. Malignant IVC masses can be primary in origin from IVC or secondary as a result of direct extension or thromboembolism from malignancy involving
other organs. Leiomyosarcoma is the most common primary malignancy involving the IVC [5]. Causes of secondary involvement includes hepatocellular carcinoma, renal cell carcinoma, Wilms tumor, testicular carcinoma, adrenocortical carcinoma and other neoplasms.

Bland thrombus is seen as filling defect in the IVC lumen on CECT. Tumor thrombus can be differentiated from bland thrombus on the basis of expansion of the vessel lumen and enhancement of the filling defect. Scan done in arterial phase also commonly reveals arterial neovascularity of the IVC tumor thrombus pointing to the malignant etiology, differentiating from bland thrombus.

Case 1

Intracaval bland thrombus

A case of a 42 year old male (Fig. 1) with abdominal pain and fever since 5 days. CECT was done after initial clinical and USG evaluation. It reveals multiple liver abscesses and a non enhancing filling defect in IVC suggestive of bland thrombus. Sepsis is the cause of IVC thrombus in this case.

Case 2

Intravenous leiomyoma

A case of a 47 year female (Fig. 2) with history of hysterectomy for uterine leiomyoma few years back. It reveals smooth intracaval portion of the enhancing mass which causes mild luminal expansion. The mass reaches right atrium (Fig 2a). In Fig. 2c, arrow shows filling defect in left common iliac vein. Filling defect was also seen in left internal iliac vein reflecting the pelvic origin of the mass.

Intravenous leiomyomatosis is a rare, benign smooth muscle tumor with an unusual pattern of growth resulting in a clinically aggressive course. It is characterized by intravascular proliferation of benign smooth muscle in the absence of, or beyond the confines of, a leiomyoma [6], and arises either by extension of a leiomyoma into adjacent veins or by vascular intimal smooth muscle proliferation [7,8]. Patients often have a history of hysterectomy or synchronous uterine leiomyomas [9]. Continuity of the intravascular tumor with the pelvic veins may be demonstrated [9].

Case 3

Primary IVC Leiomyosarcoma
A case of 59 year old female (Fig. 3) presenting with gradual onset of pedal edema and abdominal distension since 4 months. Scan reveals heterogeneously enhancing mass replacing the IVC with extraluminal extension (Fig. 3b). Arterial phase scan (Fig 3a) depicts arterial neovascularity in the mass. Also noted is ascites and liver metastasis.

**Case 4**

**Primary IVC Leiomyosarcoma**

MR scan of another 65 yr old female (Fig. 4), coronal TRU FISP (Fig 4a) image shows hyperintense mass involving suprarenal and intrahepatic IVC. Axial and coronal T2 fat saturated images in fig. 4b and 4c show hyperintense mass. Post contrast axial T1 weighted image in fig. 4d shows mild enhancement of the lesion.

Leiomyosarcoma 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 [10, 11]. The initial growth of an IVC leiomyosarcoma is intramural [3, 12]. Two-thirds of tumors will demonstrate predominantly extraluminal growth, and one-third will demonstrate predominantly intraluminal growth [3, 10, 12]. The intraluminal tumors may cause venous obstruction.

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, 11-13]. 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 [14].

**Case 5**

**Hepatocellular Carcinoma**

Axial CECT images in a patient with HCC (Fig. 5), shows arterial neovascularity of the hepatic lesion as well as of IVC tumor thrombus (Fig. 5a). Hepatic lesion in segment VII also shows early venous wash out. Expansion of IVC lumen is noted (Fig. 5b and 5c). The IVC tumor thrombus is heterogenously enhancing (Fig. 5b and 5d) and extends upto right atrium (Fig. 5c).

Occlusion of the IVC and hepatic veins may lead to Budd-Chiari syndrome. Systemic venous invasion by hepatocellular carcinoma is associated with an extremely poor prognosis, and patients with symptomatic intra-atrial extension of tumor thrombus have a
median survival of 1-4 months [15]. Systemic venous involvement predisposes the patient to distant metastasis.

**Case 6**

**Renal cell carcinoma**

A 62 year male with painless hematuria (Fig. 6). CECT Scan of abdomen reveals a large heterogeneously enhancing mass arising from left kidney. The mass forms enhancing tumor thrombus which extends via left renal vein (Fig. 6b) upto suprarenal IVC (Fig. 6a).

Renal cell carcinoma is the most common malignancy that extends into the IVC, with 4%-10% of cases involving venous invasion [16,17]. IVC involvement changes TNM system staging to T3b for infradiaphragmatic involvement and T3c for supradiaphragmatic extension and thereby affects management.

**Case 7**

**Nonseminomatous testicular germ cell tumor**

CECT scan images of a 21 year old male (Fig. 7) show right testicular heterogeneously enhancing non seminomatous germ cell tumor (Fig. 7a). Extensive para aortic lymphadenopathy is seen (Fig. 7b). Enhancing tumor thrombus is seen in IVC lumen in infrarenal and renal segments, causing mild luminal expansion (Fig. 7c and 7d).

Some studies have shown that 3%-11% of nonseminomatous testicular tumors involve the IVC [18, 19]. The tumor thrombus may result from intravascular spread through gonadal veins, or bulky retroperitoneal lymphadenopathy may invade directly through the IVC wall [20].

**Limitations:**

The pitfalls of IVC imaging on CECT involve artifacts mistaking 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 8). 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].
Fig. 1: Intracaval bland thrombus: Coronal (Fig 1a) and axial (Fig 1b) CECT scan images.
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Fig. 2: Intravenous leiomyoma: Coronal (Fig. 2a) and axial (Fig. 2b and 2c) CECT scan images of the abdomen and pelvis.

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Fig. 3: Primary IVC Leiomyosarcoma: Axial CECT scan images of abdomen in arterial (Fig. 3a) and venous phase (Fig. 3b) at the level of infrarenal IVC.

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**Fig. 4:** Primary IVC Leiomyosarcoma: MR scan of another 65 yr old female, coronal TRU FISP (Fig 4a) image, axial and coronal T2 fat saturated images (Fig. 4b and 4c), post contrast axial T1 weighted image (Fig. 4d)

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Fig. 5: Hepatocellular Carcinoma: Axial CECT images in arterial (Fig. 5a) and venous (Fig. 5b, 5c and 5d) phases.

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Fig. 6: Renal cell carcinoma: Coronal (Fig. 6a) and axial (Fig. 6b) CECT images.

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Fig. 7: Nonseminomatous testicular germ cell tumor: Axial (Fig. 7a, 7b and 7d) and coronal (Fig 7c) CECT scan images of a 21 year old male.

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**Fig. 8**

**Fig. 8:** Axial CECT image at the level of renal vein (Fig. 8) depicts admixture artifact.

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Conclusion

USG and Doppler are first line of investigation for IVC masses. It is operator dependent modality and is inadequate for determining the extent of the lesion.

Characteristic CT imaging features help us to classify IVC masses into neoplastic and nonneoplastic pathologies and thus guide patient management. Specific CECT imaging features of tumor thrombus are luminal expansion, enhancement of the thrombus and arterial neovascularity (in malignant tumor thrombus).

CT is an optimum imaging technique, to diagnose IVC masses as well as to describe their extent longitudinally and circumferentially, determining the surgical approach. Accurate description of the tumor thrombus and its extent is essential because it changes the staging and thereby affects surgical intervention.

Mild delay in scanning from 60-70 s to 70-90 s can help avoid admixture artifact because of relatively more uniform enhancement of IVC lumen.

The CECT involves risk of exposure to ionizing radiation; however it is most commonly used modality and also helps in metastatic survey.

Though there is no radiation hazard in MRI, but cost of investigation and poor availability in remote areas are limiting factors. MRI in pediatric age group mostly requires anesthesia for adequate scanning.
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