Inferior vena cava: review of anatomic variants and pathologic processes.

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Learning objectives

The inferior vena cava (IVC) is the large vein responsible for returning blood from the lower extremities and abdomen to the right atrium. It is associated with many congenital variants and pathological processes. However, it is a structure often overlooked in abdominal imaging studies.

We propose addressing the following objectives:

- Reviewing the embryological development of the inferior vena cava (IVC) and its congenital variants.
- Reviewing inferior vena cava (IVC) pathological processes.
- Describing potential imaging pitfalls.

Describing, in particular, radiological findings and clinical implications.
Background

Imaging (CT, MRI, US and Angiography) plays a crucial role in the diagnosis and management of patients with anatomic variants or pathological processes of the IVC.

1. Embryology of the inferior vena cava (IVC):

The mature IVC is formed by four segments: the hepatic, suprarenal, renal and infrarenal IVC.

Complex anastomoses and regression of vitelline, posterior cardinal, subcardinal and supracardinal veins form each of the IVC segments (Fig 1).

Congenital variations of the IVC are the result of abnormal embryologic developments involving these embryonic veins.

2. Congenital IVC variations:

The spectrum of classical congenital IVC variations includes:

2.1 Absence:

Affecting the whole IVC or only the infrarenal IVC (Fig 2). The cause is not clear, it could be a fault in the venous embryological development but also has been suggested perinatal thrombosis or atrophy. Associated with this we can found abundant collateral venous circulation, lower extremity venous insufficiency or deep vein thrombosis.

2.2 Duplication:

Duplicated infrarenal IVC segments are caused by persistent of both supracardinal veins. (Fig 3).

Left-sided IVC drains into the left renal vein. It should therefore display a normal continuation of the suprarenal IVC.

Prevalence is 0.2-0.3%.

It is essential to know the existence of this anomaly in patients who will be treated with vena cava filter. If no, they might suffer a pulmonary embolism.
2.3 Left-sided IVC:

Abnormal persistence of the left supracardinal vein with regression of the right supracardinal vein. Left-sided IVC drains into the left renal vein. Then it continues with a normal suprarenal IVC.

The prevalence is 0.2-0.5%.

Rarely causes clinical involvement. However, it may cause confusion in vascular access.

2.4 Anomalous continuation of the IVC to the thorax:

The suprarenal inferior vena cava continuing with azygos or hemiazygos vein. Blood finally reaches the heart through the superior vena cava. The hepatic veins drain directly into the right atrium due to absence of the intrahepatic segment of the IVC.

In some cases, the hemiazygos vein may drain directly into the coronary sinus or left brachiocephalic vein.

2.5 Retrocaval ureter:

It is one of the few congenital anomalies of the IVC that can be symptomatic (urinary outflow obstruction and recurrent urinary tract infections). Surgical treatment may be necessary.

The IVC develops from the right posterior cardinal vein instead of the right supracardinal vein forming a retrocaval ureter. (Fig 4).

2.6 IVC webs:

These consist of a complete or incomplete membrane in the intrahepatic IVC. It can be caused by a rare congenital anomaly or secondary to thrombosis.

IVC webs are more common in Asian and South African populations.

It generates portal hypertension. Depending on severity, treatment can be angioplasty, placement of a stent or creation of a transjugular intrahepatic portosystemic shunt.

2.7 Extrahepatic portocaval shunt (Abernethy Malformation):

Two types have been described (Fig 5):
• **Type 1 shunt:** It is a complete end-to-side shunt between the portal vein and the IVC, with the main portal vein distal to the shunt being absent. (Fig 6). Type 1 shunts are classified into: **Type 1a** in which the splenic vein and superior mesenteric vein drain separately into a systemic vein. **Type 1b** The splenic vein and superior mesenteric vein drain together after joining to form a common trunk. Type 1 shunts usually occurs in girls and are associated with multiple malformations: Polysplenia, malrotation, biliary atresia and congenital heart defects.

• **Type 2 shunt:** The intrahepatic portal vein is intact, but some of the portal flow is diverted into a systemic vein through a side-to-side shunt. Type 2 shunts evidence no gender preference and have fewer associated malformations than type 1 shunts.

3. **Non-tumoural acquired pathology of the inferior vena cava:**

In addition, the IVC can be affected by trauma (slitlike IVC and aortocaval fistula), surgery (stent, IVC filter, mesocaval shunt) or infections (thrombophlebitis).

3.1 **IVC filter:**

IVC filter placement is a common procedure performed by interventional radiologists. TC is important before placement to rule out congenital variants as duplication of IVC (Fig 7).

3.2 **Stenosis of the IVC:**

A complication frequently appearing after a liver transplantation caused by compression due to postoperative swelling.

Doppler ultrasonography plays an important role in detecting it.

Vascular complications are the second leading cause of graft failure after acute rejection, and treatment includes angioplasty and stent placement (fig 8).

3.3 **Mesocaval shunt:**

Surgical interposition between the superior mesenteric vein and the IVC. This was a popular treatment for uncontrollable variceal bleeding associated with cirrhosis and portal hypertension. However, this treatment has decreased in popularity since the advent of the transjugular intrahepatic portosystemic shunt procedure.

3.4 **Slitlike IVC:**
A slitlike IVC is defined as an IVC with a transverse-to-anteroposterior diameter ratio greater than 3:1 seen at multiple levels (Fig 9). In patients with trauma, a slitlike IVC is associated with significant hypotension and impending shock. It is a nonspecific finding in patients with normovolemic and no significant clinical effects.

3.5 Aortocaval fistula:

An aortocaval fistula is a rare but often catastrophic complication of abdominal trauma or abdominal aortic aneurysm. 80%-90% of aortocaval fistula formation is the result of rupture or erosion of an abdominal aortic aneurysm into the IVC. Imaging findings include early contrast opacification in the IVC during arterial phase imaging and loss of a normal fat plane between the aorta and IVC.

3.6 IVC Thrombophlebitis:

Gonadal vein thrombophlebitis commonly involves the ovarian vein in postpartum patients. However, this entity can extend into the IVC; approximately 80% of cases are right-sided. CT findings consist of enhancing the venous wall and perivascular inflammation. Treatment includes anticoagulation drugs and antibiotics.

4. Tumoural acquired pathology of the inferior vena cava:

Both primary (leiomyosarcoma) and secondary malignant neoplasm can involve the IVC.

- **Primary IVC malignancy** is extremely rare; although **leiomyosarcoma** represents less than 1% of all malignancies, it accounts for more than 75% of tumours arising from large veins. (Fig 10).
- **Secondary malignant neoplasm** of the IVC is more common than primary IVC neoplasm. It is frequently caused by direct endovascular extension and/or intraluminal thromboembolization (Fig 11).

**Bland thrombus** and intracaval tumours should be differentiated. Unlike tumour thrombus, bland thrombus lacks luminal expansion and enhancement (Fig 13).

Risk factors for thrombus formation include a hypercoagulable state, malignancy, venous stasis, focal compression, and IVC filters.

Bland thrombus in the IVC most often extends from pelvic and lower extremity deep vein thrombosis.

Correct identification of the extent of IVC involvement is essential to staging and determining surgical intervention.
5. Imaging Pitfalls:

5.1 Pseudo filling defect due to a flow-related phenomenon producing an image of pseudothrombus. It is most often seen at the level of the renal veins in the corticomedullary or portal venous phase (Fig 12 y 13). Contrast-enhanced blood from the kidneys mixes with non-enhanced blood from the lower extremities. This artifact can be seen at both contrast-enhanced CT and contrast-enhanced MR imaging. This pitfall is usually solved in more delayed postcontrast images.

5.2 Pseudolipoma of the IVC is a partial volume averaging artifact caused by juxtacaval fat above the caudate lobe, rather than a true intraluminal lesion. It is often seen in patients with cirrhosis.

5.3 Retrograde flow. 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. (Fig 14).
Images for this section:

**Fig. 1:** Drawing illustrates the embryologic development of the IVC. The IVC segments (dark blue) are formed by anastomoses and regression of multiple embryological veins (light blue).

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Fig. 2: Coronal CT image: A and Axial CT image: B. IVC absence affecting the infrarenal segment.

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Fig. 3: Coronal CT image. Arrows show duplication of inferior vena cava.

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**Fig. 4:** Axial CT image. Arrow shows right retrocaval ureter.

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**Fig. 5:** Drawing illustrates extrahepatic portocaval shunt (Abernethy Malformation).

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**Fig. 6:** Coronal MIP MR image. Abernethy malformation type1. Congenital absence of the portal vein with shunting of portal blood (yellow triangle) into the inferior vena cava (arrow).

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Fig. 7: Cavography image showing IVC filter.

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**Fig. 8:** CT image reconstruction showing IVC stent. Stent was placed because of a hepatic tumour that was compressing the inferior vena cava.

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**Fig. 9:** Axial CT image. Slitlike IVC in a patient with significant hypotension and impending shock (arrow).

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Fig. 10: Coronal CT image. Arrow shows primary leiomyosarcoma of inferior vena cava.
**Fig. 11:** Coronal CT image. Inferior vena cava tumoural thrombus of a renal cell carcinoma (arrow).

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Fig. 12: Axial CT image. Pseudo filling defect (arrow) due to a flow-related phenomenon producing an image of pseudothrombus. It is most often seen at the level of the renal veins in the portal venous phase, contrast-enhanced blood from the kidneys mixes with nonenhanced blood from the lower extremities.

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**Fig. 13:** Axial CT image. Pseudo filling defect (blue arrow) due to a flow-related phenomenon producing an image of pseudothrombus. In the left renal vein there is a filling defect (yellow arrowhead), in this case it is a real thrombus.

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**Fig. 14:** Axial CT image. Retrograde flow of contrast material into the IVC because of right heart failure or a contrast material injection rate faster than 3 mL/sec.

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Findings and procedure details

Vascular structures can be evaluated on contrast-enhanced CT scans. However, in cases in which intravenous contrast material is contraindicated, MR imaging may be used. We review IVC variants and pathological processes, placing special emphasis on radiological findings and potential imaging pitfalls.

We present our most representative cases.
Conclusion

The inferior vena cava (IVC) is an essential but often overlooked structure. IVC evaluation must be a fundamental part of the search pattern.

IVC is associated with a wide variety of congenital and pathological conditions, to recognize and to refer it can be vital to patient care.

The radiologist should be aware of the various pitfalls of IVC imaging and must be able to differentiate true from pseudo filling defects.
References