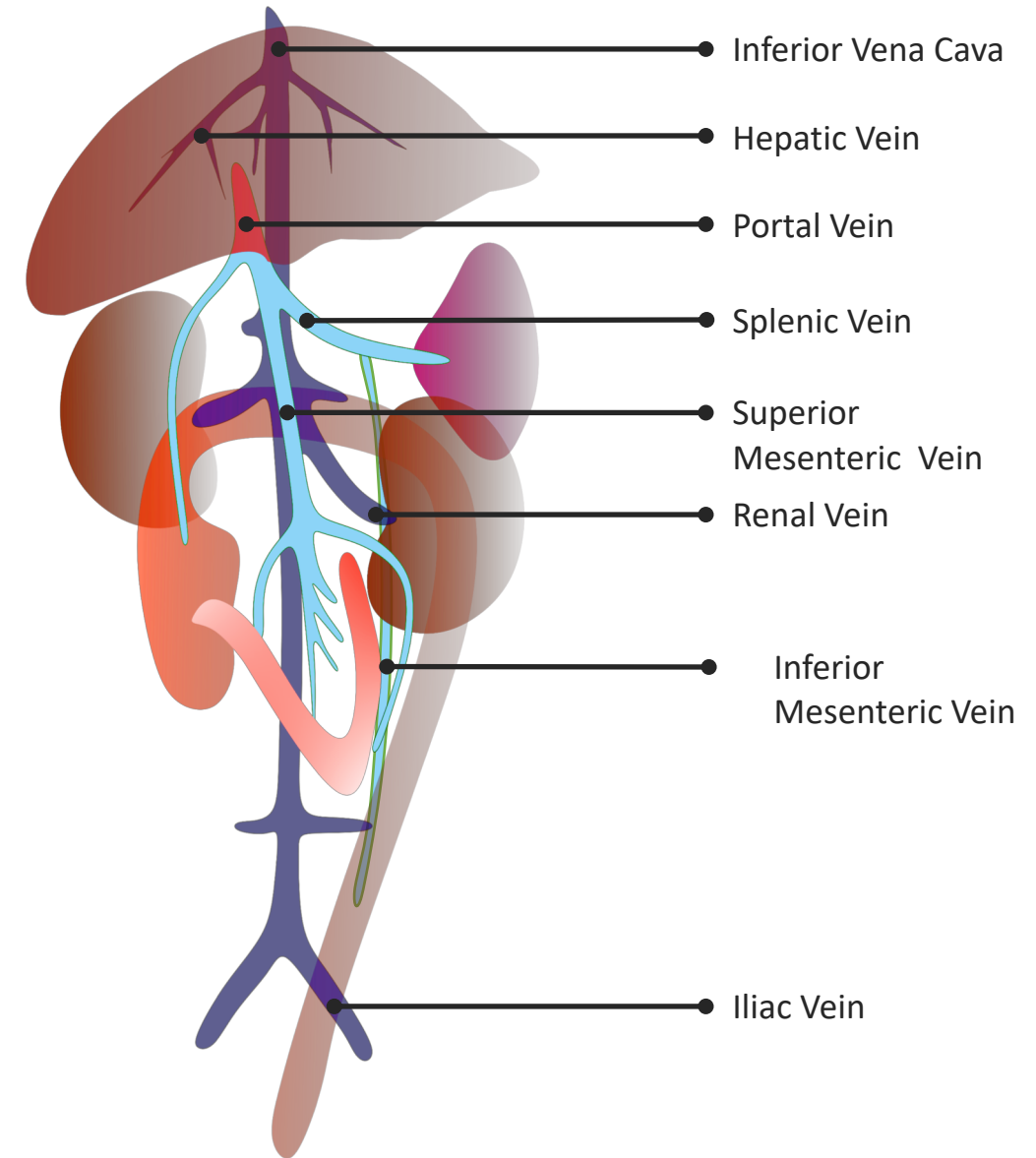


Non-traumatic Venous Emergencies of the Abdomen

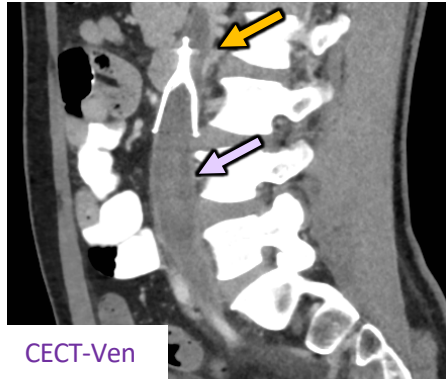
Vinit Baliyan, Anushri Parakh,
Sandeep Hedgire, Anand Prabhakar



Ilio-caval Thrombosis

Cases

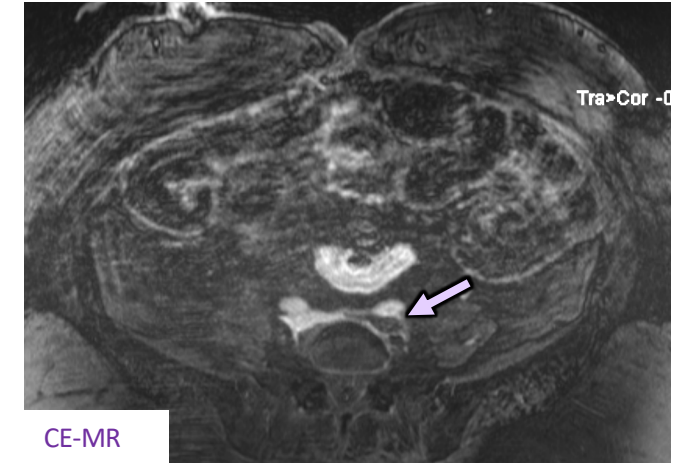
Bland IVC thrombus in setting of inherited hypercoagulopathy with thrombus extending above the filter IVC filter (yellow)



Leiomyosarcoma (*) of the IVC with secondary bland thrombus (arrow)



May-Thurner Syndrome (MTS)



Causes

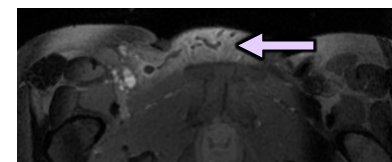
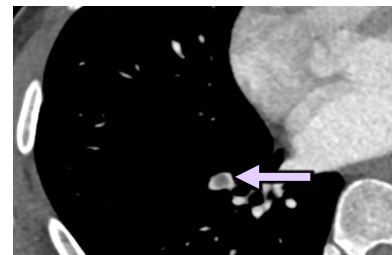
- 1) Inherited coagulopathy
- 2) Acquired coagulopathy
 - i. Immobility
 - ii. Malignancy
 - iii. Myeloproliferative disorders
 - iv. Recent surgery
- 3) Local tumor invasion
 - i. Renal / hepatic cancers
 - ii. Primary IVC leiomyosarcoma
- 4) Compression syndromes
 - 1) May-Thurner (MTS)

Clinical Implications

- 1) Embolism (pulmonary)
- 2) Chronic stasis
 - i. Varices
 - ii. Dermatitis and ulcers
 - iii. Male infertility due to varicocele
- 3) Extension to visceral tributaries

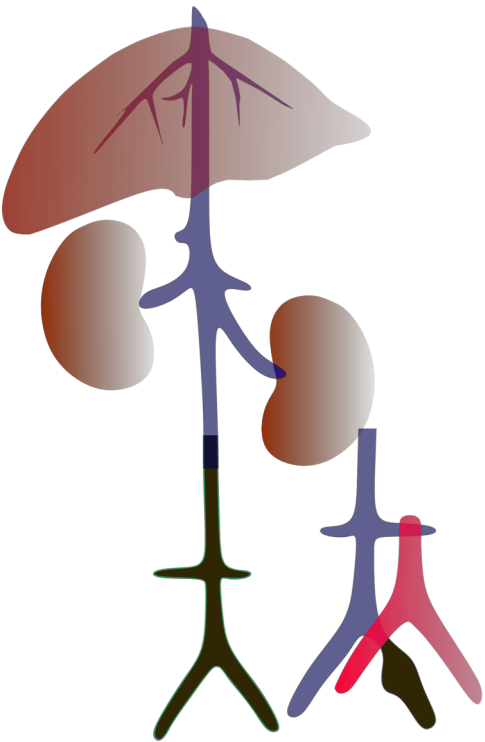
Management

- 1) Anticoagulation, IVC filter
- 2) Mechanical thrombectomy
- 3) Venous stenting/bypass-MTS

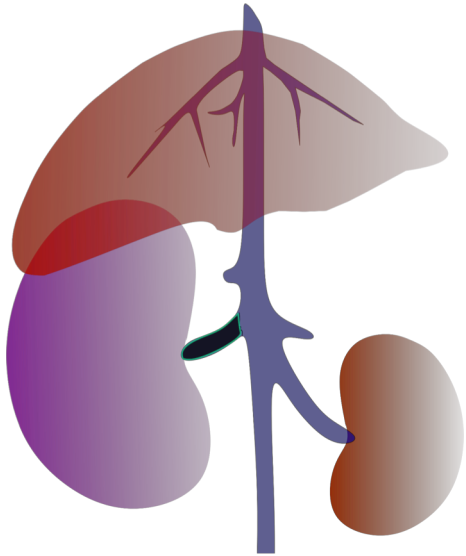


Imaging

- Duplex US for DVT in lower extremities
 - CT and/or MR venography
- 1) Differentiate acute vs chronic**
 - i. *Acute*-expanded vein, wall enhancement, fat stranding
 - ii. *Chronic*-eccentric thrombus, narrow vessel caliber, calcifications, webs, collaterals / varices
 - 2) Bland vs Tumoral thrombus**
 - i. Tumor thrombus-greater degree of venous expansion, thrombus vascularity, diffusion restriction, metabolically active on PET

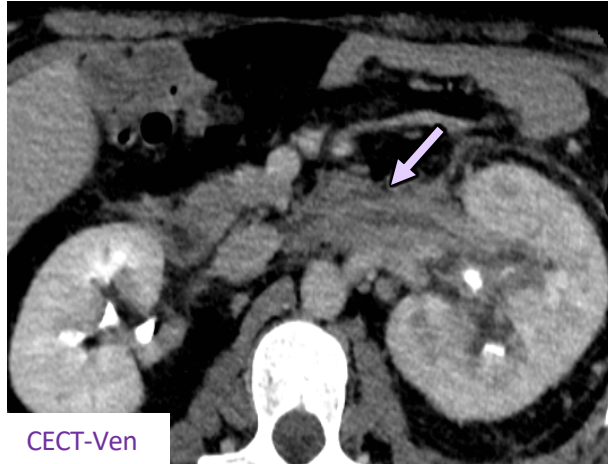


Visceral Caval Tributary Thrombosis



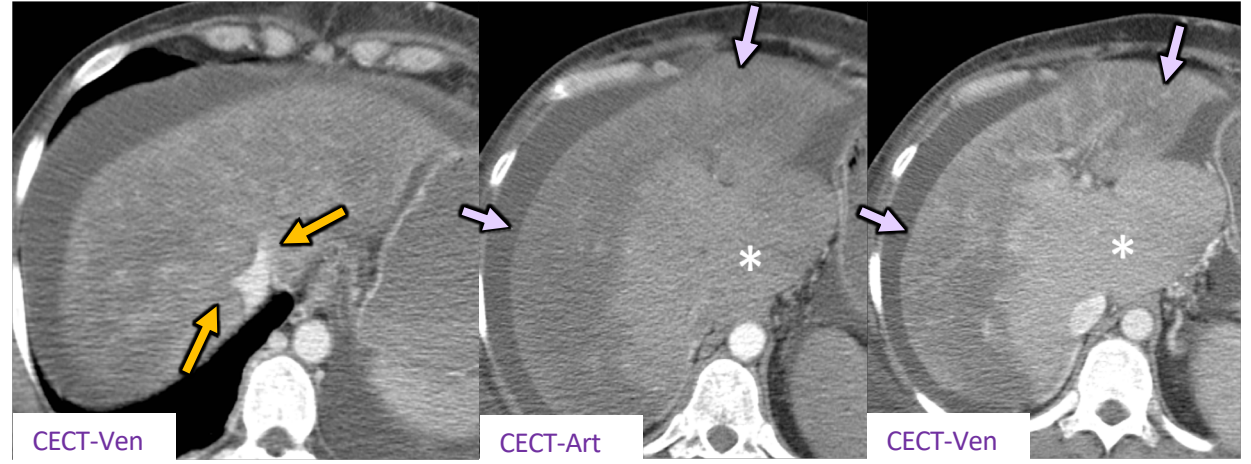
Cases

Renal Vein Thrombosis in Behçet Syndrome



CECT-Ven

Budd Chiari Syndrome with hepatic vein attenuation (yellow), reduced peripheral enhancement (purple), increased central enhancement (*) and ascites



CECT-Ven

CECT-Art

CECT-Ven

Causes

General

- 1) Inherited coagulopathy
- 2) Acquired coagulopathy
 - i. Dehydration
 - ii. Vasculitis
- 3) Local tumor invasion
 - i. Renal / hepatic cancers

Specific

- 1) Budd Chiari Syndrome – Congenital (webs), myeloproliferative disorder, local tumors and abscesses
- 2) Renal Vein Thrombosis – nephrotic syndrome, sickle cell, renal abscess

Clinical Implications

Budd Chiari

- 1) Hepatic dysfunction, hepatic failure
- 2) Cirrhosis, regenerative nodules, hepatocellular carcinoma
- 3) Portal hypertension and its sequelae

Renal Vein Thrombosis

- 1) Acute renal failure
- 2) Renal atrophy
- 3) Papillary necrosis

Imaging

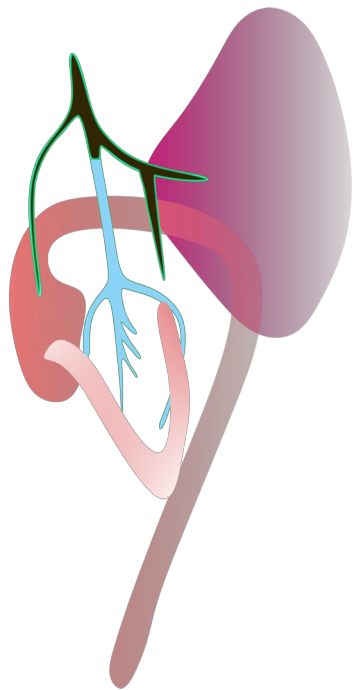
Budd-Chiari- Multiphase CT and/or MR

- i. Acute-Venous occlusion, hepatosplenomegaly, ascites, decreased peripheral parenchymal and increased central parenchymal enhancement*
- ii. Subacute/chronic-Atrophy with caudate hypertrophy, arterialized peripheral blood flow, collaterals, regenerative nodules, venous calcifications*

Renal vein thrombosis- Doppler, CT/MR angiography

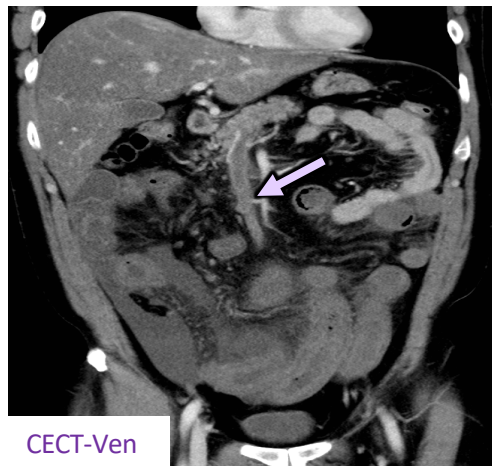
- i. Acute-Nephromegaly, reversal of diastolic arterial flow, increased RI, vein thrombus, venous infarction, persistent cortical enhancement and reduced/lack of enhancement*
- ii. Chronic-Atrophy, collaterals, venous calcifications*

Porto-splanchnic Thrombosis



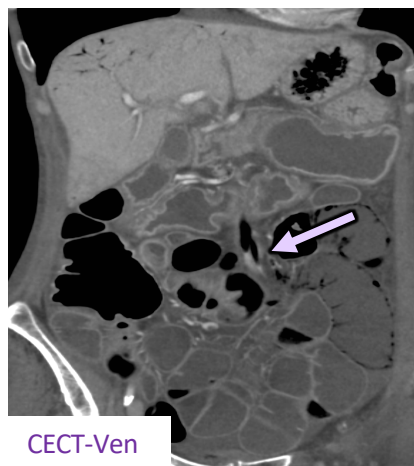
Cases

SMV thrombus with bowel ischemia



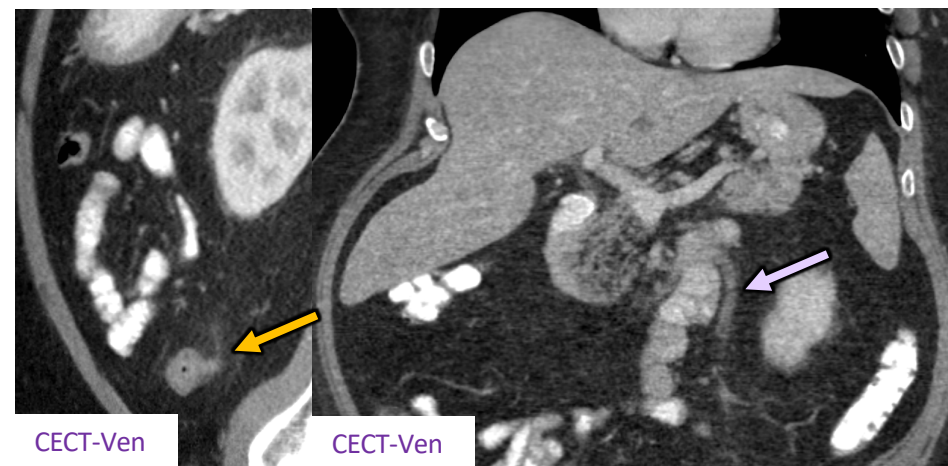
CECT-Ven

Secondary venous gas due to bowel ischemia



CECT-Ven

IMV thrombosis (purple) secondary to sigmoid diverticulitis (yellow)



CECT-Ven

CECT-Ven

Causes

- 1) Inherited coagulopathy
- 2) Acquired coagulopathy
 - i. Idiopathic
 - ii. Cirrhosis
 - iii. Pancreatitis
 - iv. Myeloproliferative disorders
 - v. Abdominal surgery
- 3) Local tumor invasion
 - i. HCC*

*Tumor thrombus- vein expansion greater degree, thrombus vascularity, diffusion restriction, hot on PET

Clinical Implications

Superior mesenteric vein (SMV)

- 1) Mesenteric congestion and bowel wall edema
- 2) Bowel ischemia

Portal vein Thrombosis

- 1) Findings of SMV thrombosis
- 2) Splenic congestion
- 3) Porto-systemic collaterals

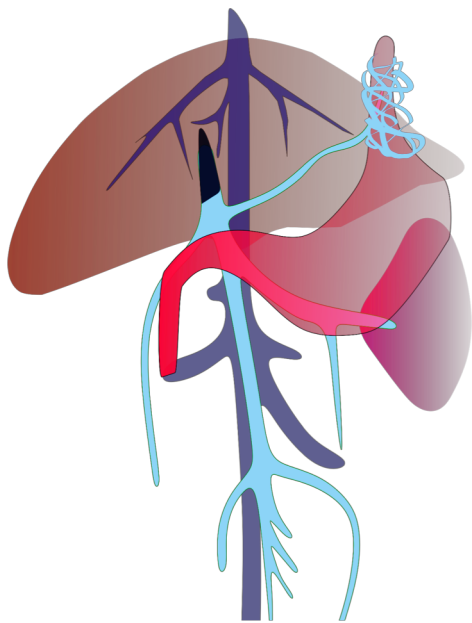
Splenic vein thrombosis

1. Isolated splenomegaly with gastric varices

Imaging

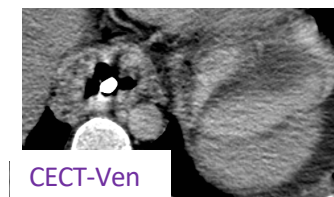
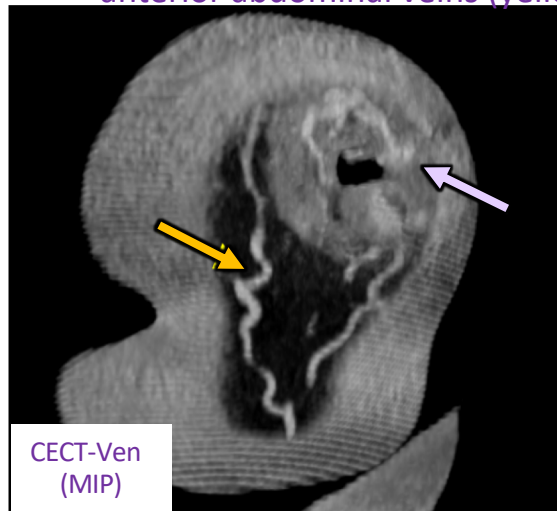
- Multiphase CT and/or MRI
- SMV thrombosis**
- i. *Acute* – Venous occlusion, bowel wall thickening, dilation, mesenteric fat stranding, ascites and hyper-enhancement. Bowel infarction with pneumatosis, venous luminal air and lack of enhancement
 - ii. *Subacute/chronic* –Recanalization of vessel, collateral formation, venous calcifications
- Portal vein thrombosis**
- i. *Acute* –thrombosis, splenomegaly, vein thrombus, absent/decreased flow
 - ii. *Chronic*- Cavernoma, venous calcifications, Porto-systemic collaterals

Variceal Hemorrhage

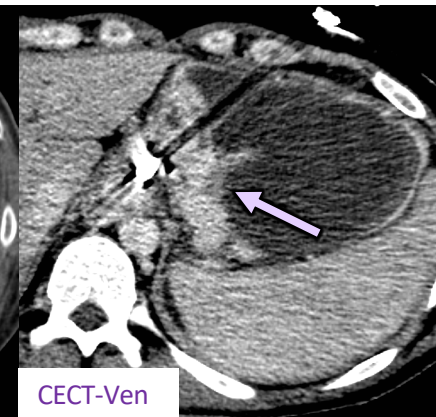
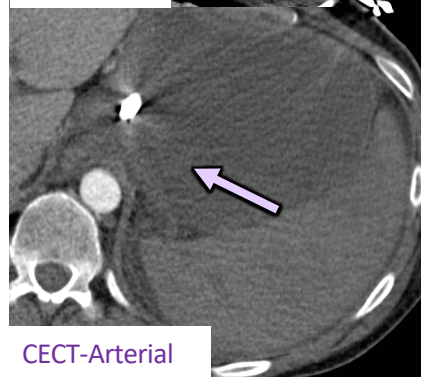


Cases

Peristomal venous hemorrhage in portal hypertension with dilated anterior abdominal veins (yellow) and dilated SMV tributary



Bleeding esophageal varices



Causes

Esophageal/gastric varices are commonly seen in the setting of chronic portal hypertension. These can bleed.

Variceal hemorrhage is a life threatening emergency and imaging is not typically performed in patients with suspected variceal bleed.

Variceal hemorrhage is rarely seen on cross sectional imaging.

Clinical Implications

Esophageal varices

- 1) Life threatening hemorrhage

Stomal varices

- 1) Rare entity
- 2) Bowel ostomies recruit mesenteric vasculature (branches of SMV) in close proximity with systemic veins in the abdominal wall. Therefore, in long standing cases, this is a potential site for development of portosystemic shunts
- 3) Peristomal varices are a rare cause of stomal site bleed

Imaging

- Multiphase CT and/or MRI

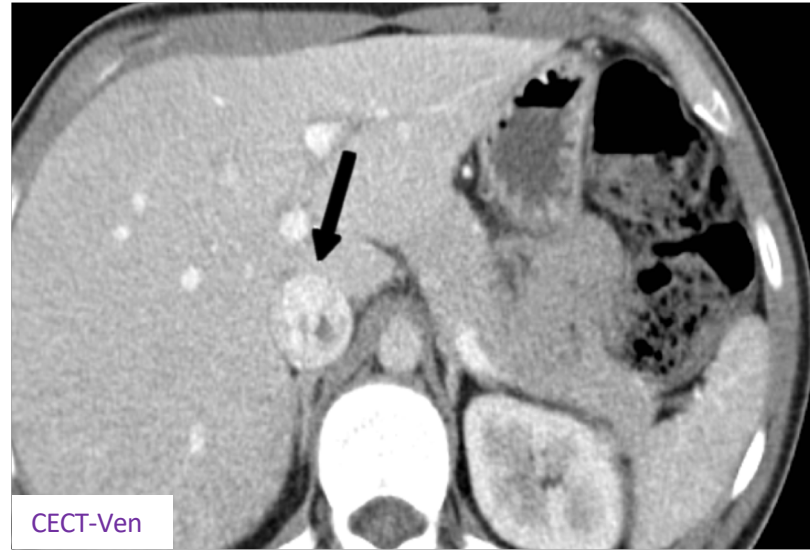
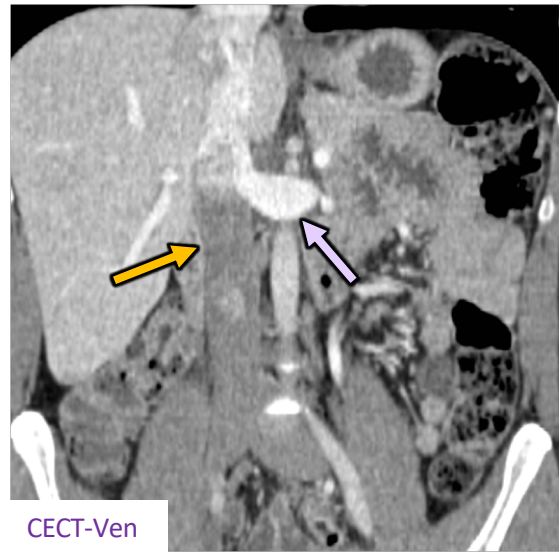
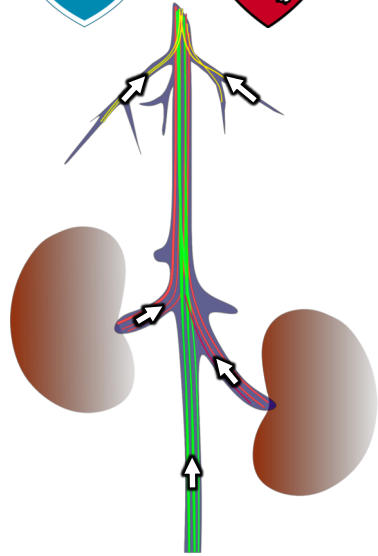
Esophageal varices

Serpiginous vessels around gastroesophageal junction. Additional collaterals at porta hepatis, perisplenic, periumbilical and perirectal locations. In active hemorrhage, brisk extravasation is seen on venous/delayed phases that is absent on arterial phase

Stomal varices

Serpiginous vessels around the stoma with dilated anterior abdominal wall collaterals and dilated adjacent SMV tributary

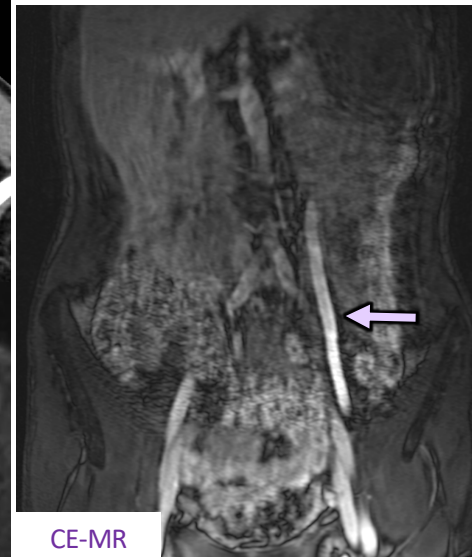
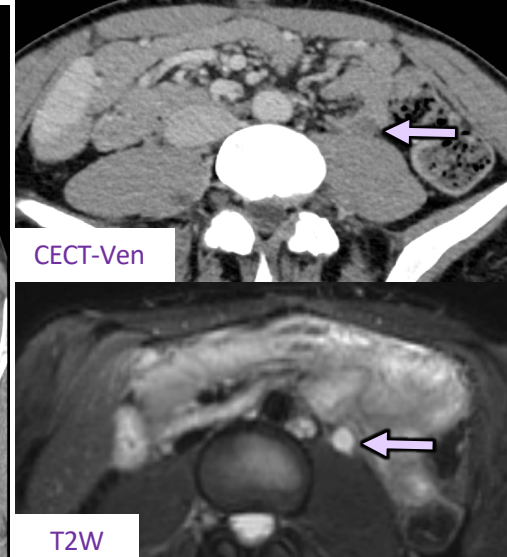
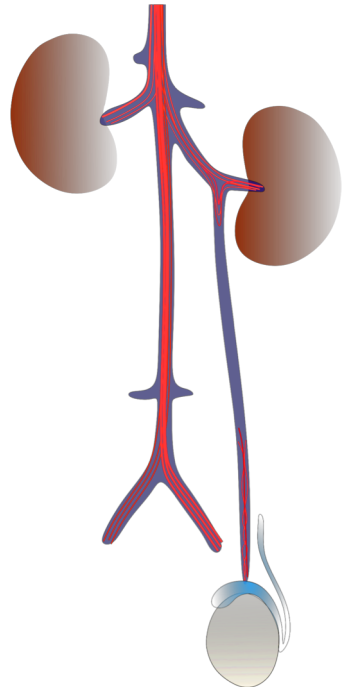
Pitfalls and Artifacts



Case: Apparent filling defect in intrahepatic IVC (black) due to admixing of unenhanced blood from infrarenal IVC (yellow) and enhanced blood from renal veins (purple).

Clinical Implication: May be falsely called as thrombus

Mitigate: By performing delayed imaging, typically at 2 minutes, to differentiate from true thrombosis



Case: Nutcracker syndrome; Post renal vein bypass. Apparent thrombosis of a large gonadal vein (purple) on venous phase CT, that is bright on T2W. Delayed phase MR post contrast administration demonstrates enhancement.

Mitigate: Additional delayed phases in a setting of a large venous channel fed by a small organ/territory.

Thank You