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Ultrasound in vascular liver diseases

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US in Vascular liver diseases

Budd-Chiari syndrome

Portal vein thrombosis

Congenital vascular malformations

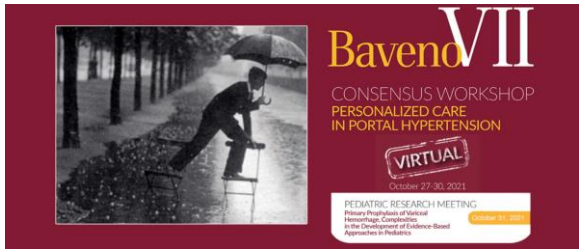
Porto-sinusoidal vascular disorder (PSVD)

Hepatic artery diseases (aneurysm, thrombosis, A-V/A-P fistulas)

Sinusoidal obstruction syndrome

Radiation-induced liver disease

Peliosis hepatis and sinusoidal dilatation



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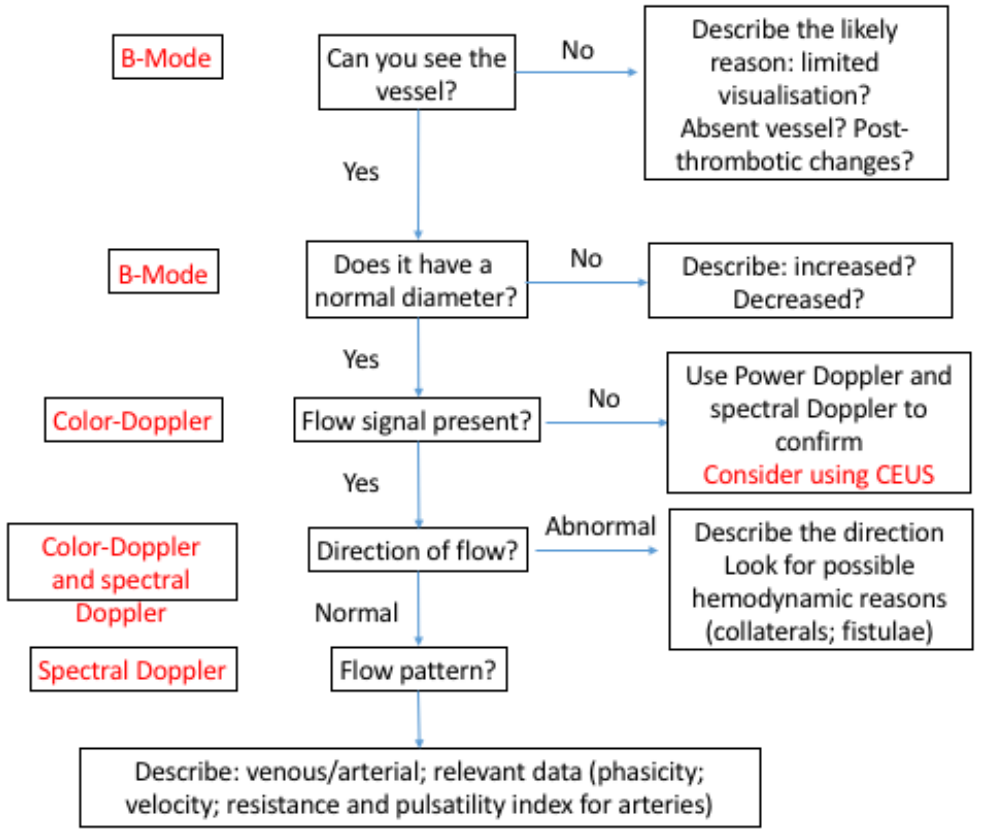
VASCULAR LIVER DISEASE GROUP

European Reference Networks

ERN RARE-LIVER

Systematic US approach to liver vessels



De Gottardi, Berzigotti et al.
Ultraschall Med 2018

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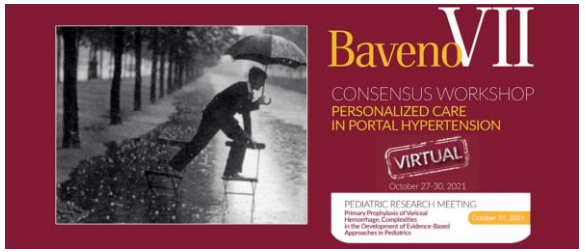

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European
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LIVER

Ultrasound/CDUS is the first line imaging technique
to be used in this setting

High sensitivity (>90%) for thrombosis

Budd-Chiari syndrome

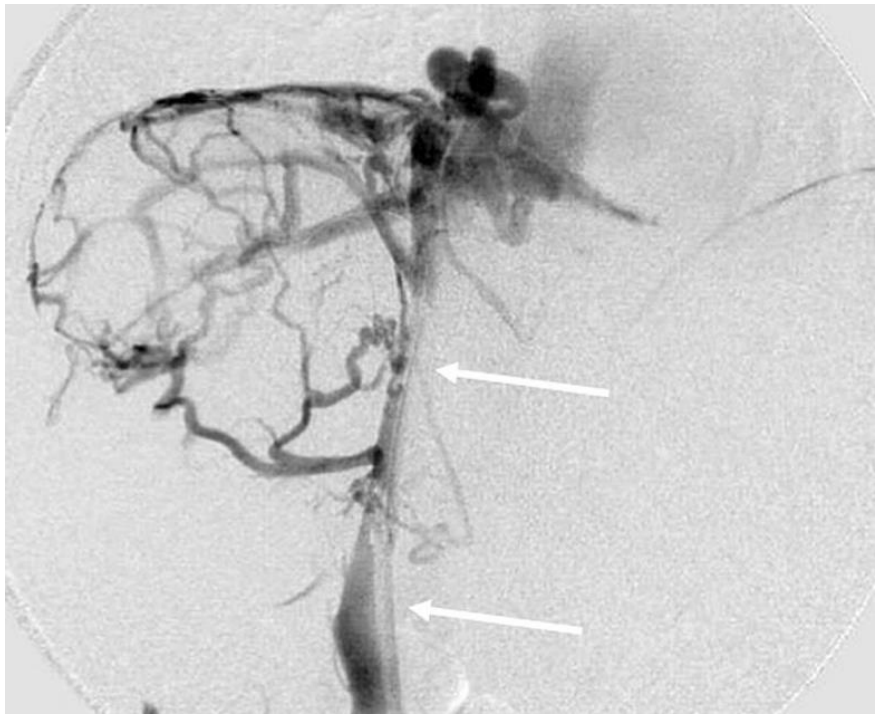
DEFINITION

BCS is defined as the obstruction of hepatic venous outflow regardless of its causative mechanism or level of obstruction

Clinical impact very heterogeneous according to localization and timing.
Hepatic venous web.

EPIDEMIOLOGY

- Incidence 1.5 (0.68-2.17)/million year
- Prevalence 4/million
- F/M=7/3



Potier, Hepato-Gastro, 2018. Van Wettere, Abd Radiol, 2018

Budd-Chiari syndrome

Acute or fulminant forms (7%)

- sudden onset hepatic impairment, ascites, pain, and often kidney failure
- hepatomegaly following obstruction of the three main hepatic veins.

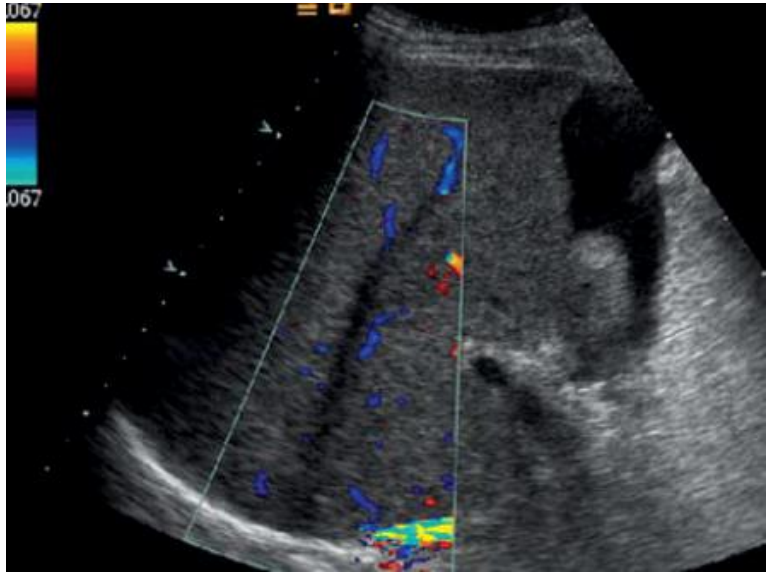
Chronic or subacute forms

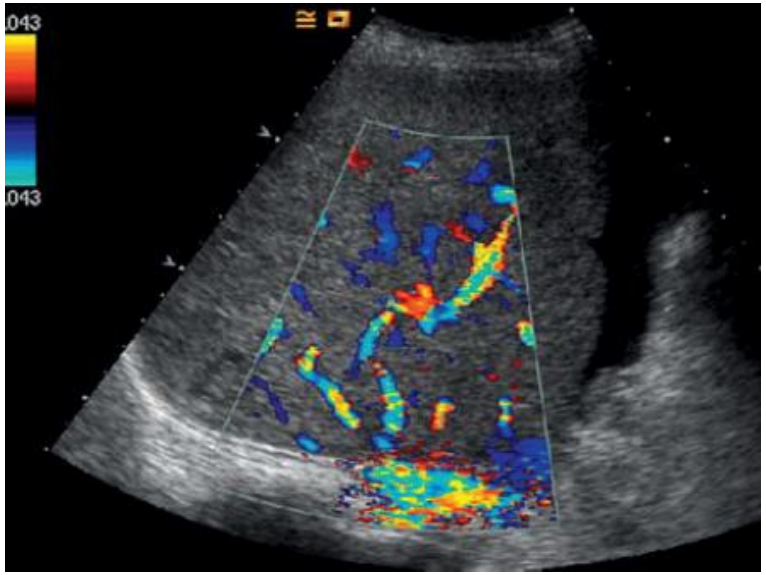
- most frequent presentation
- onset of the disease is slower in most patients
- few symptoms
- frequently impaired liver function
- portal hypertension is often present

The clinical diagnosis of BCS is difficult and should be suspected in the presence of:

- sudden development of ascites with increased liver volume and upper abdominal pain
- abundant high-protein ascites contrasting with moderately abnormal hepatic tests
- liver disease in a patient with a known thrombogenic condition
- fulminant hepatic failure accompanied by increased liver volume and ascites
- unexplained chronic liver disease

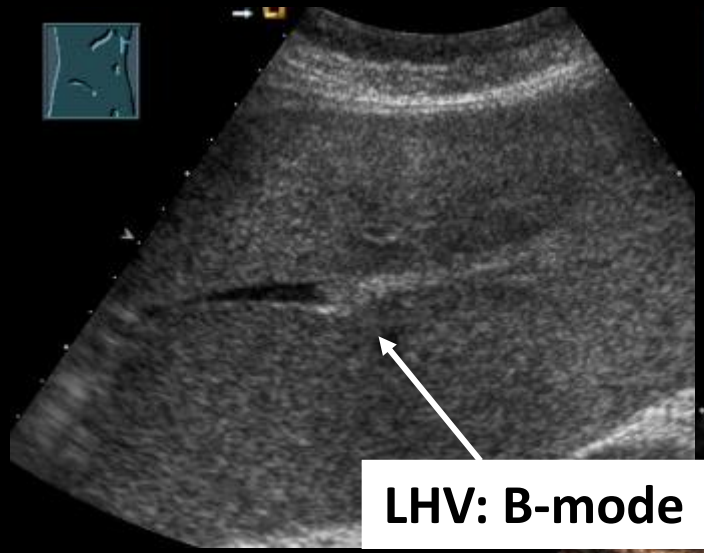
Budd-Chiari syndrome can be diagnosed by ultrasound



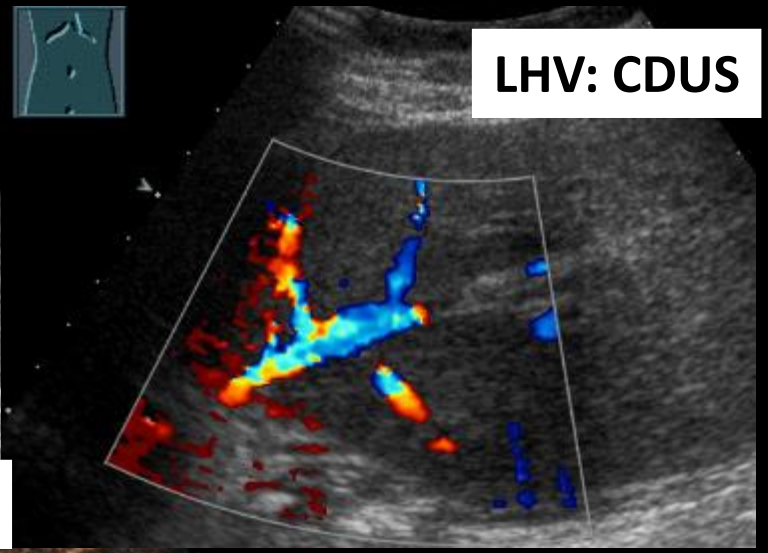


De Gottardi, Berzigotti, Ultraschall in Med, 2018

Direct signs of BCS: HV thrombus

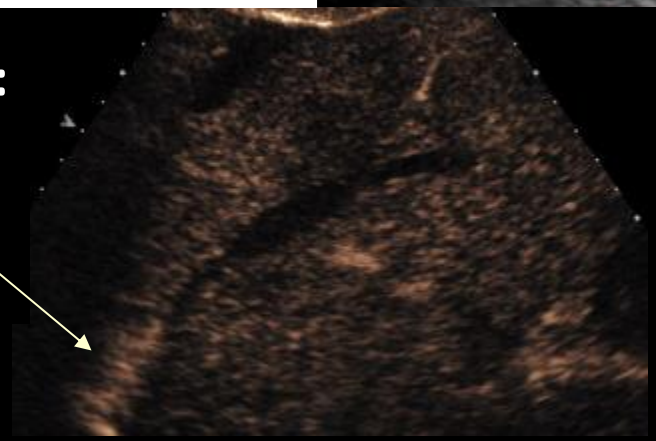


LHV: B-mode

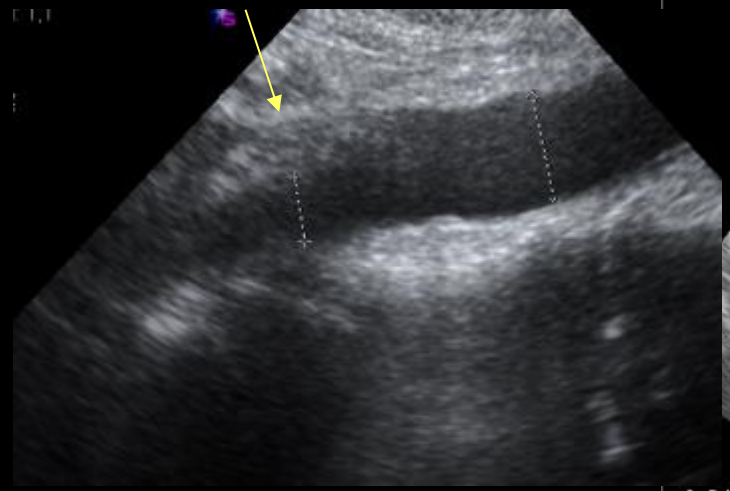


LHV: CDUS

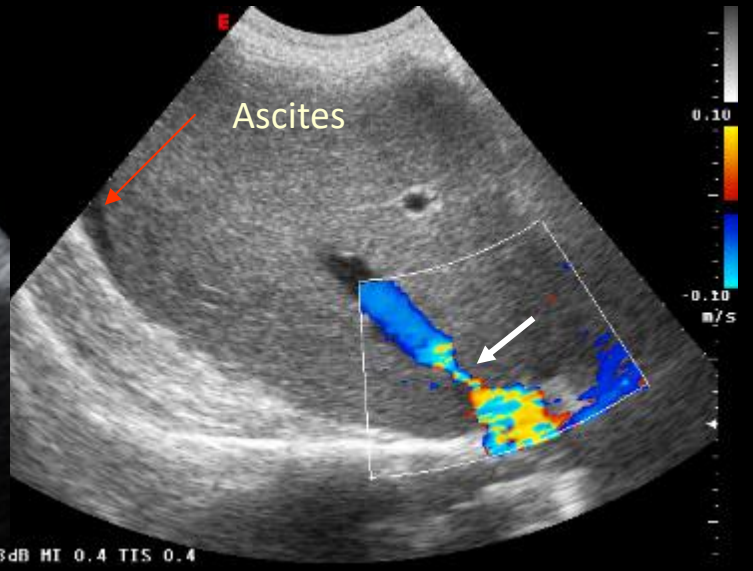
CEUS of left HV: patency of proximal tract



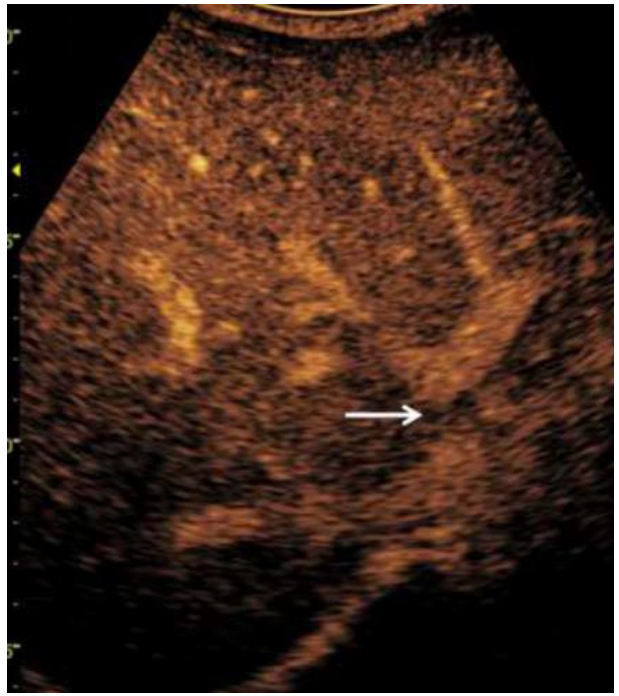
Direct signs of BCS: IVC/hepatic vein system



Thrombosis of IVC



Short-length
stenosis
of the right HV

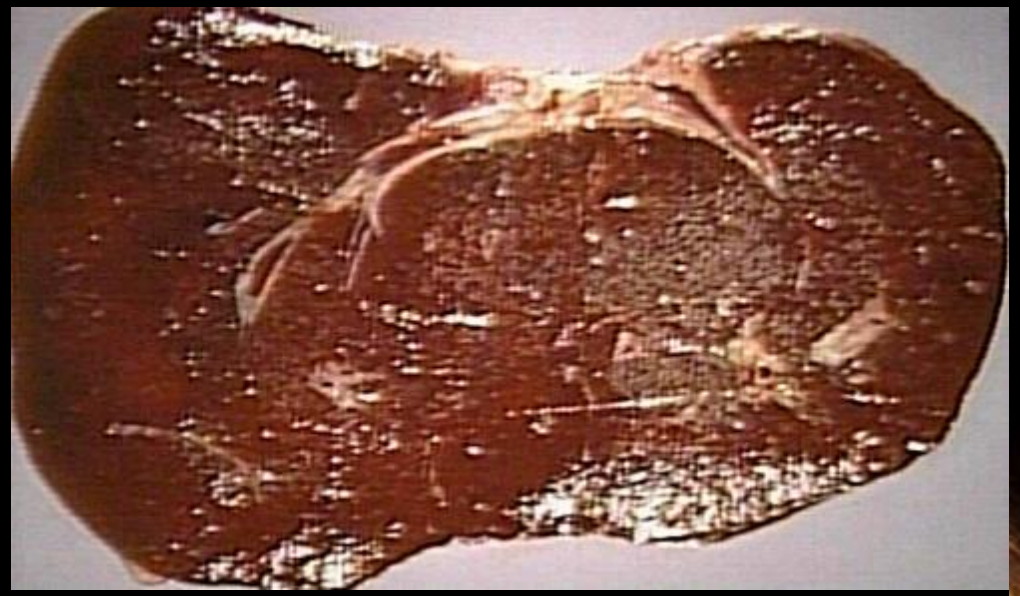


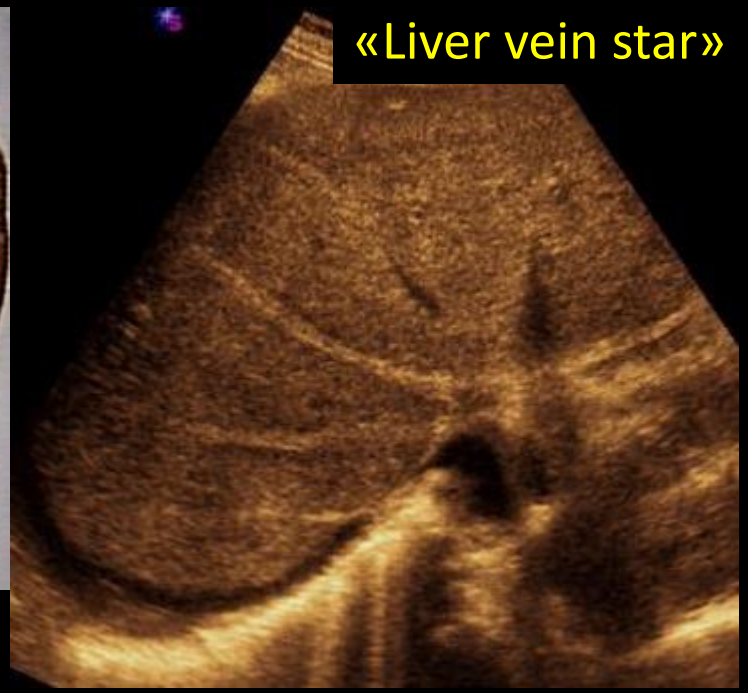
«membrane» at the origin of the liver veins

Very difficulto to detect without CEUS

Bansal, Brit J Radiol, 2018

Chronic BCS: Hyperechoic cords (chronic fibrotic changes)





«Liver vein star»

Hepatic veins: indirect signs of BCS

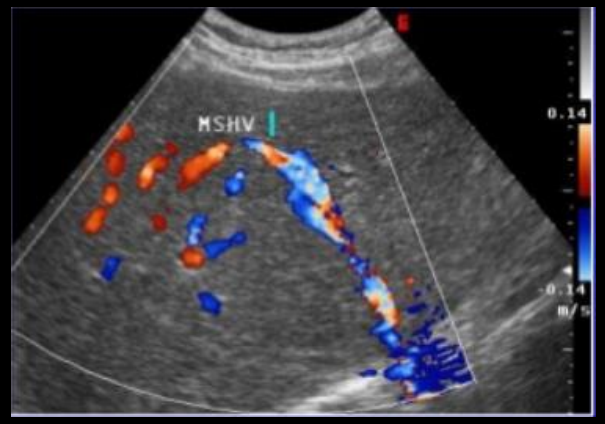
- Reversed flow in one of the HVs or a tract of it

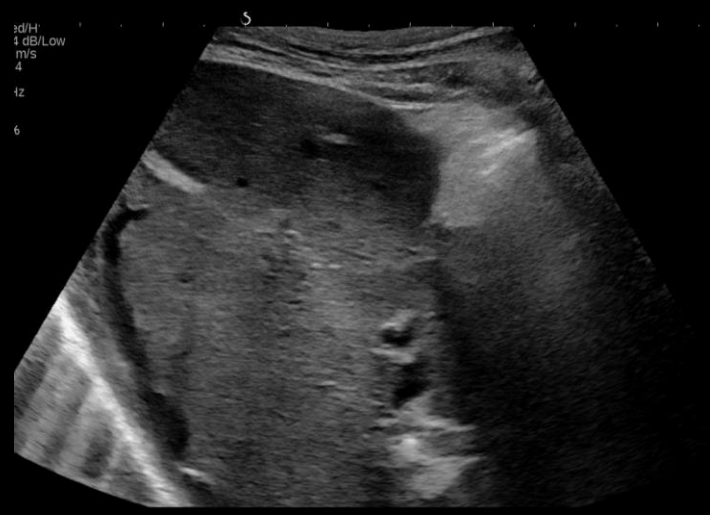
- Large communication between one vein and another one

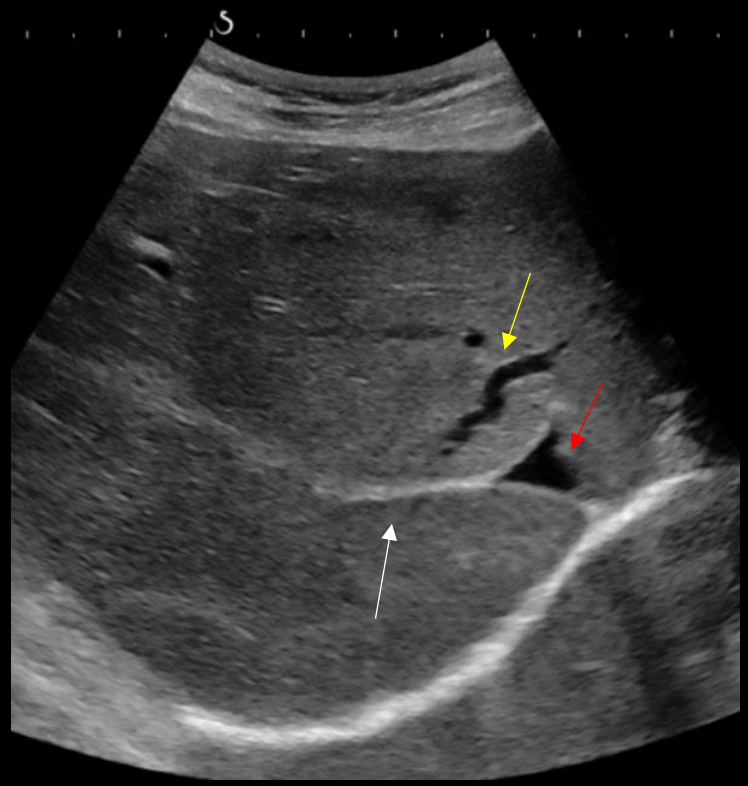
«Bicolored hepatic vein»

- Large subcapsular veins draining in the IVC (compensatory)

Bargalló X et al. Am J Roent 2006





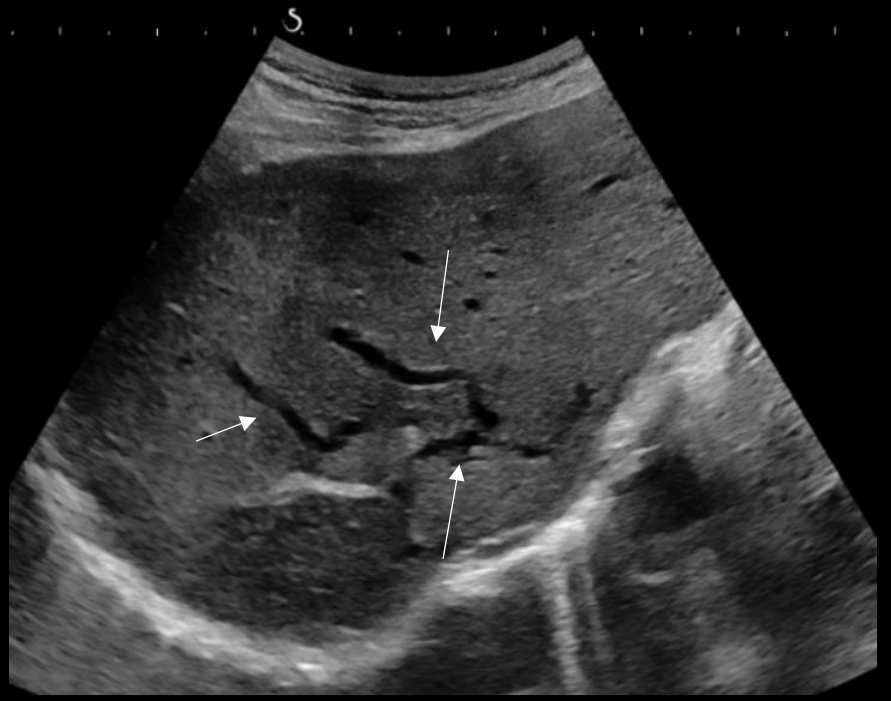


Chronic BCS

White arrow: right liver vein substituted by a fibrous cord

Yellow arrow: veno-venous collaterals

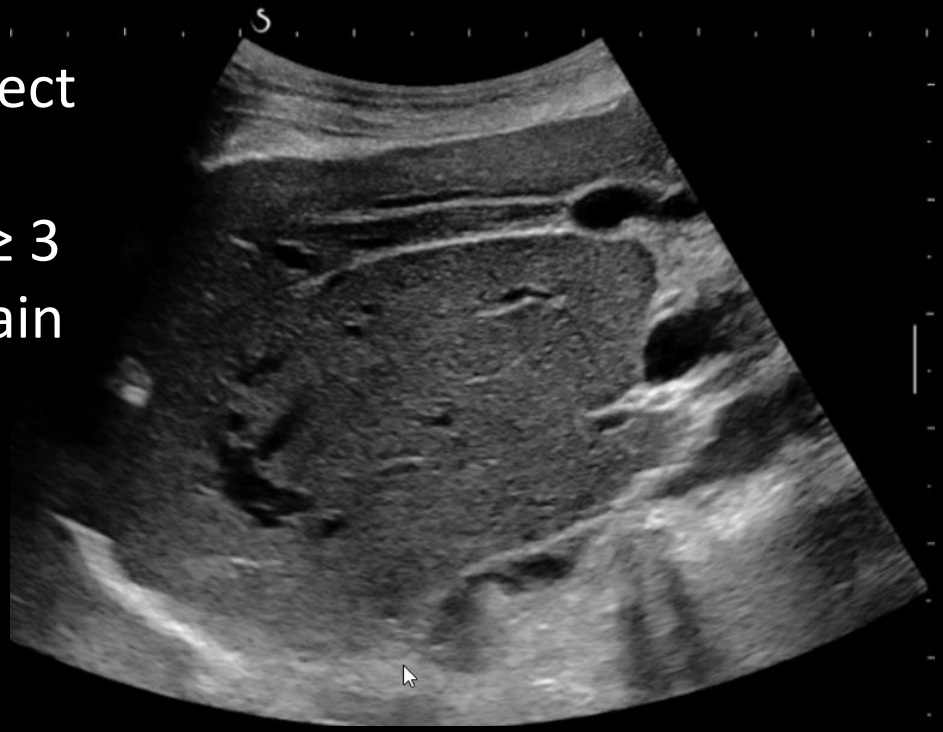
Red arrow: intrahepatic part of the IVC compressed by the liver parenchyma



Veno-venous collateral circulation

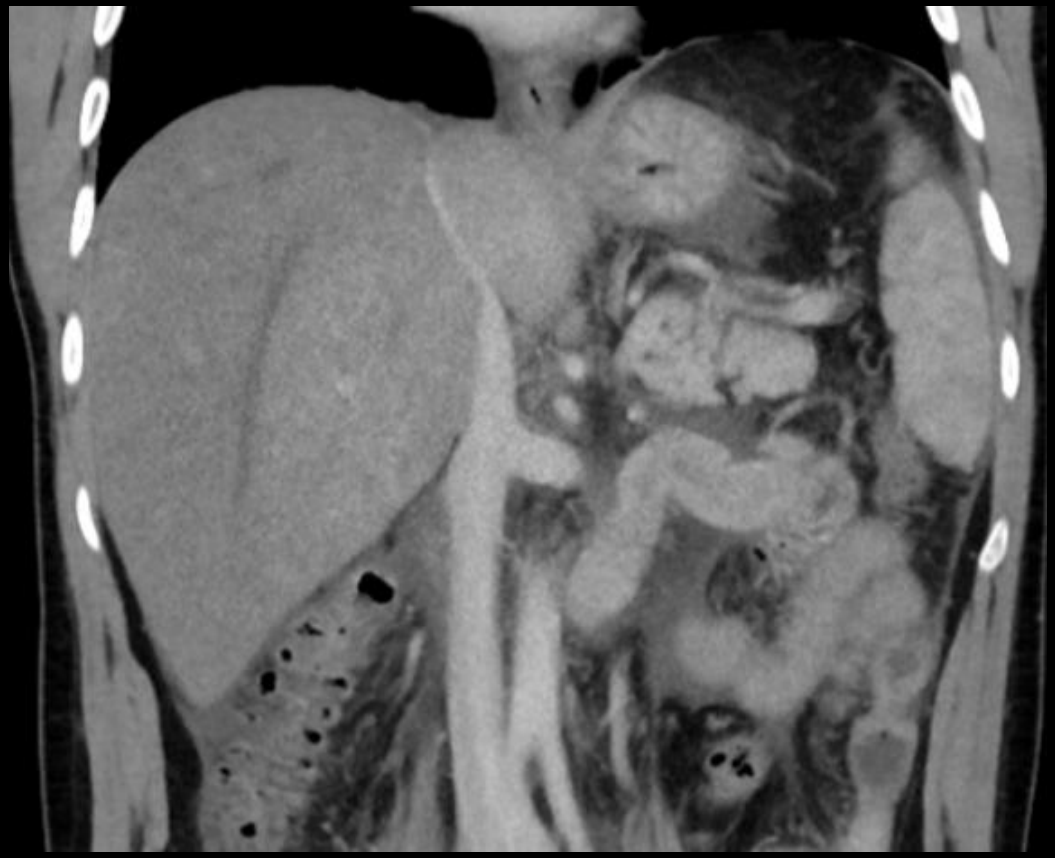
Caudate lobe hypertrophy

- Sometimes grotesque aspect
- Caudate vein ≥ 3 mm (direct drain into IVC)

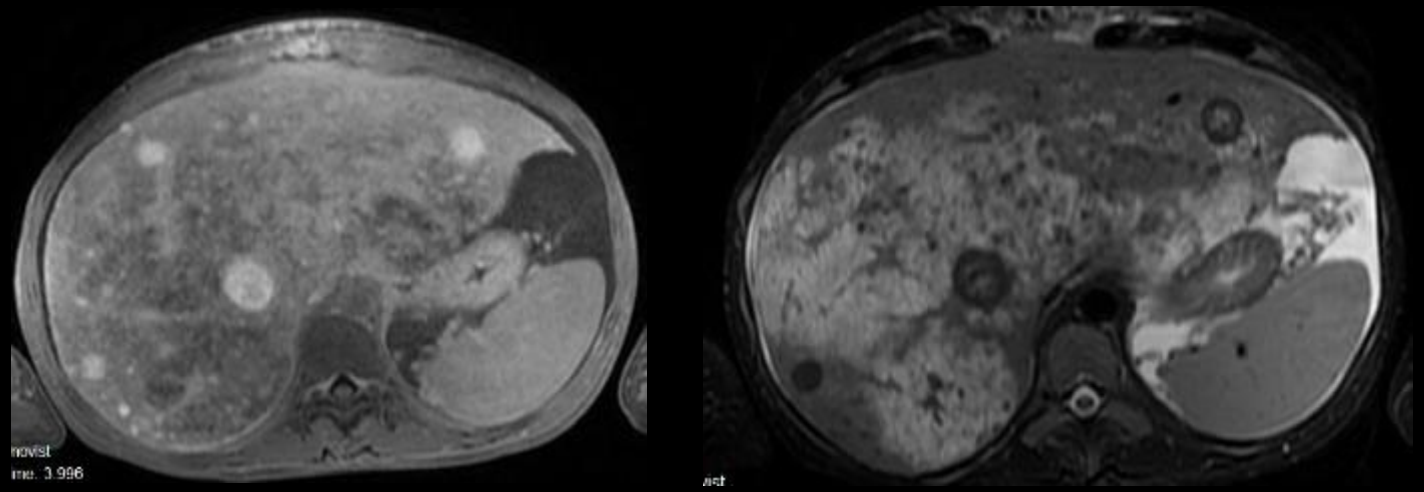


Massive increase in size of the caudate lobe and hypotrophy of the anterior segments of the left liver lobe

CECT: IVC compressed in its intrahepatic part



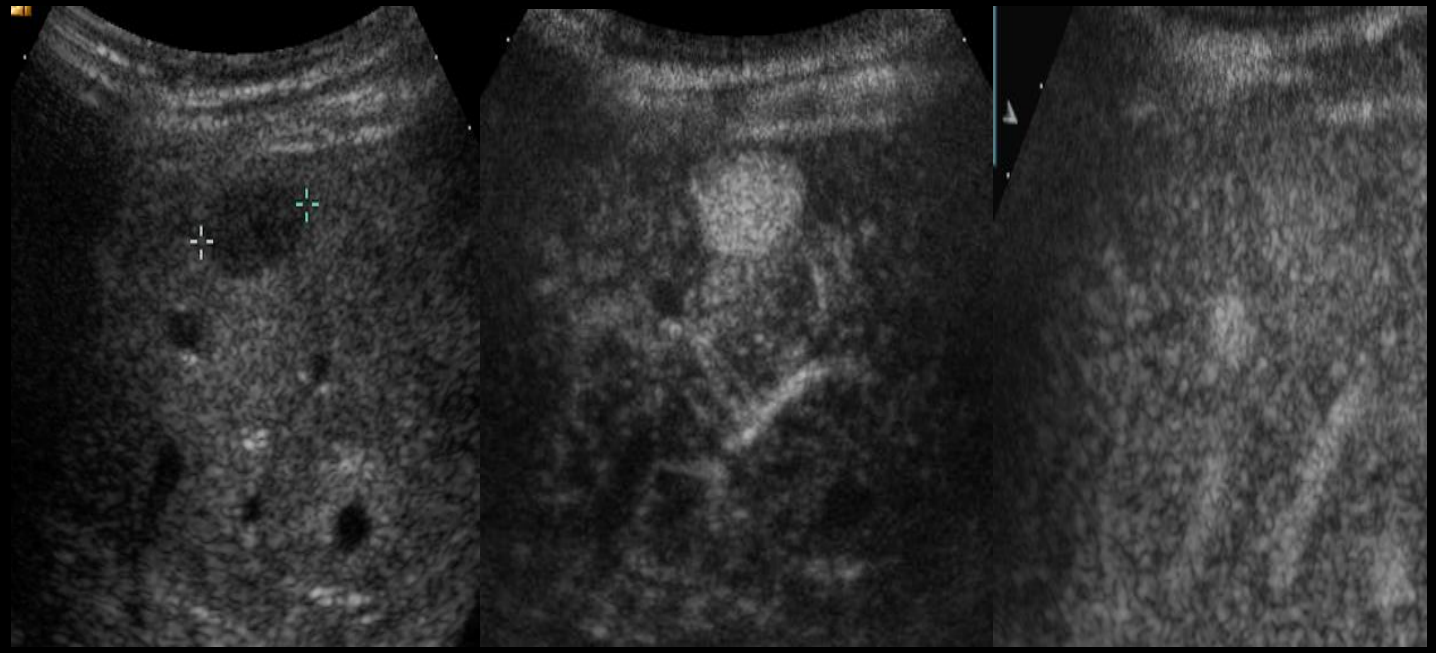
Liver nodules in Budd-Chiari



- **Very frequent** 60–80% of patients on pathology
- **Often large; often increase in size**
- **Mostly <u>benign</u> (FNH-like and large regenerative nodules)**
- **But HCC can appear: f-up and biopsy if increase in size**

Moucari et al. Gut 2008

CEUS to assess nodules in BCS



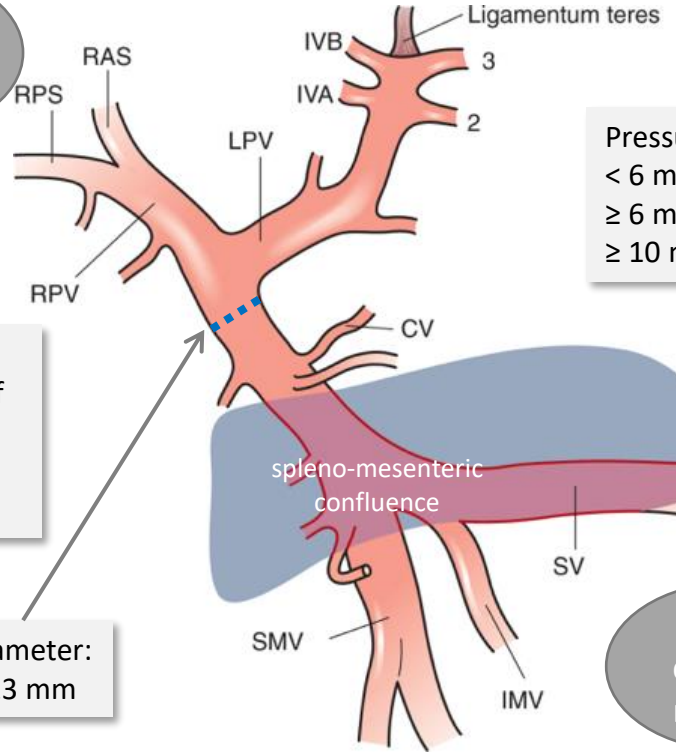
Basal US

Arterial phase

Venous phase

Portal circulation: normal conditions

Hepatic sinusoids



Pressure gradient to hepatic veins:
< 6 mmHg = normal
≥ 6 mmHg = portal hypertension
≥ 10 mmHg = clinically relevant PHT

Blood flow:
750 – 1500 mL/min (2/3 of the hepatic blood inflow)
Velocity:
15 cm/s

Diameter:
< 13 mm

GI capillary network

Portal vein thrombosis: definition and prevalence

Presence of a clot in any of the vessels of the PV axis:

- main portal vein trunk
- left and/or right intrahepatic PV branches
- splenic vein
- superior and/or inferior mesenteric veins

How frequent is it?

- 2.5 cases per 100'000 per year (without cirrhosis): EHPVO
- 5 cases per 100 per year (in patients with cirrhosis)
- 9 cases per 100 per year in PSVD

Northup et al. AASLD Guidance 2020

Clinical presentation:
recent obstructive portal vein thrombosis

No liver disease

↓

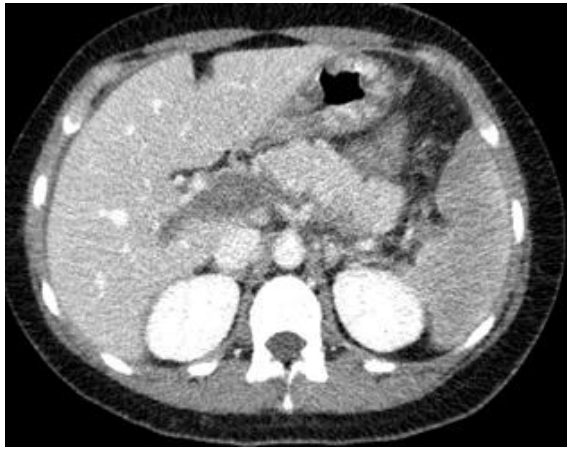
Absence of porto-systemic collaterals

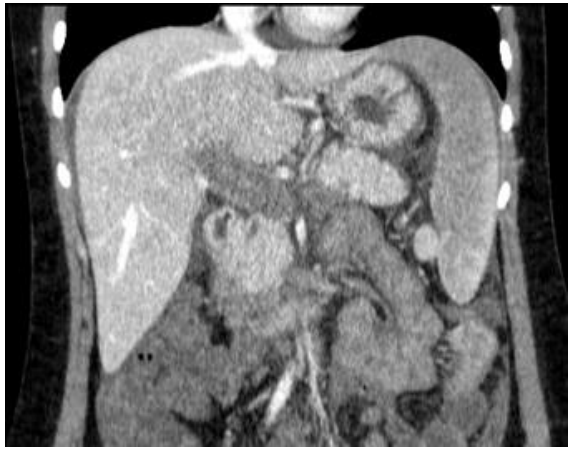
↓

Rapid and clinically significant increase in portal pressure

↓

Development of relevant complications → Intestinal infarction | Ascites | Infections





Clinical presentation in cirrhosis: obstructive portal vein thrombosis

Advanced chronic liver disease

↓

Presence of porto-systemic collaterals

↓

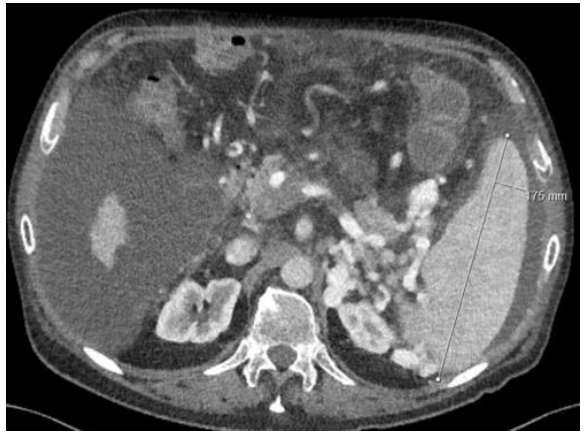
Attenuated increase in portal pressure

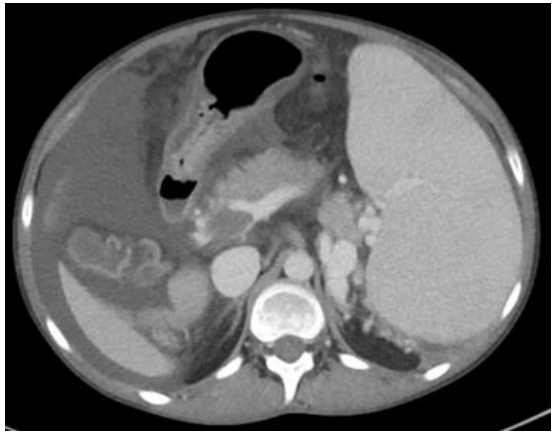
↓

Possible worsening of clinically relevant complications of PHT

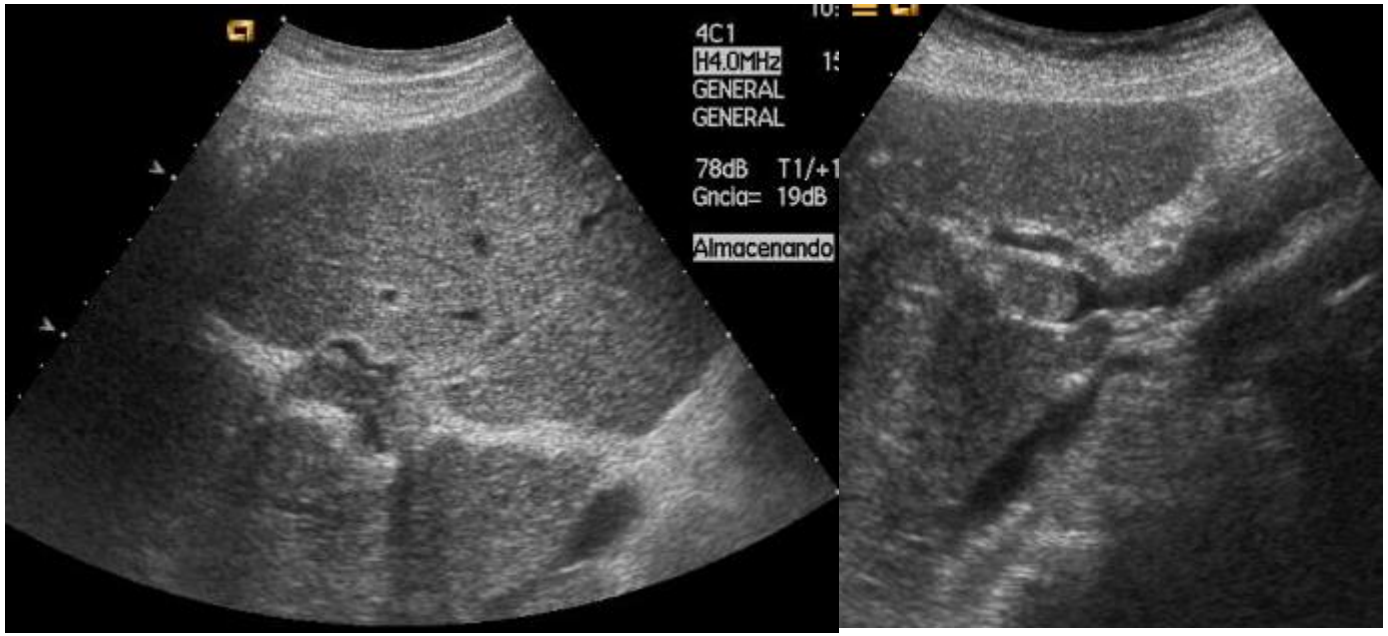
→ Oesophageal and/or gastric varices

- Encephalopathy
- Ascites
- Hypersplenism





How does it appear on US?

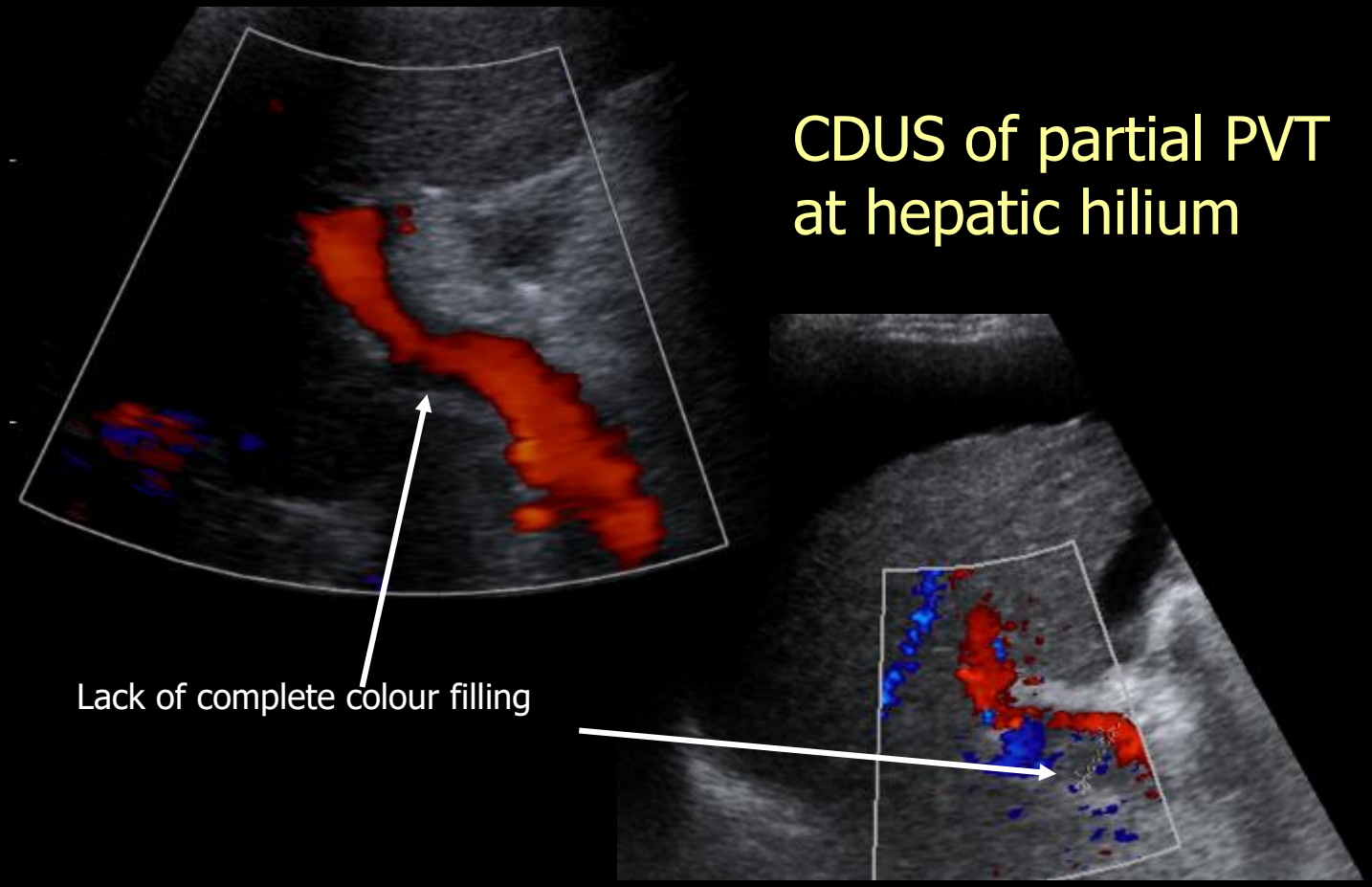


Echogenic material (hypo-, iso- or slightly hyperechogenic) within the vessel (better visualized in B-mode)

which can be partially (better seen on B mode) or completely occluded

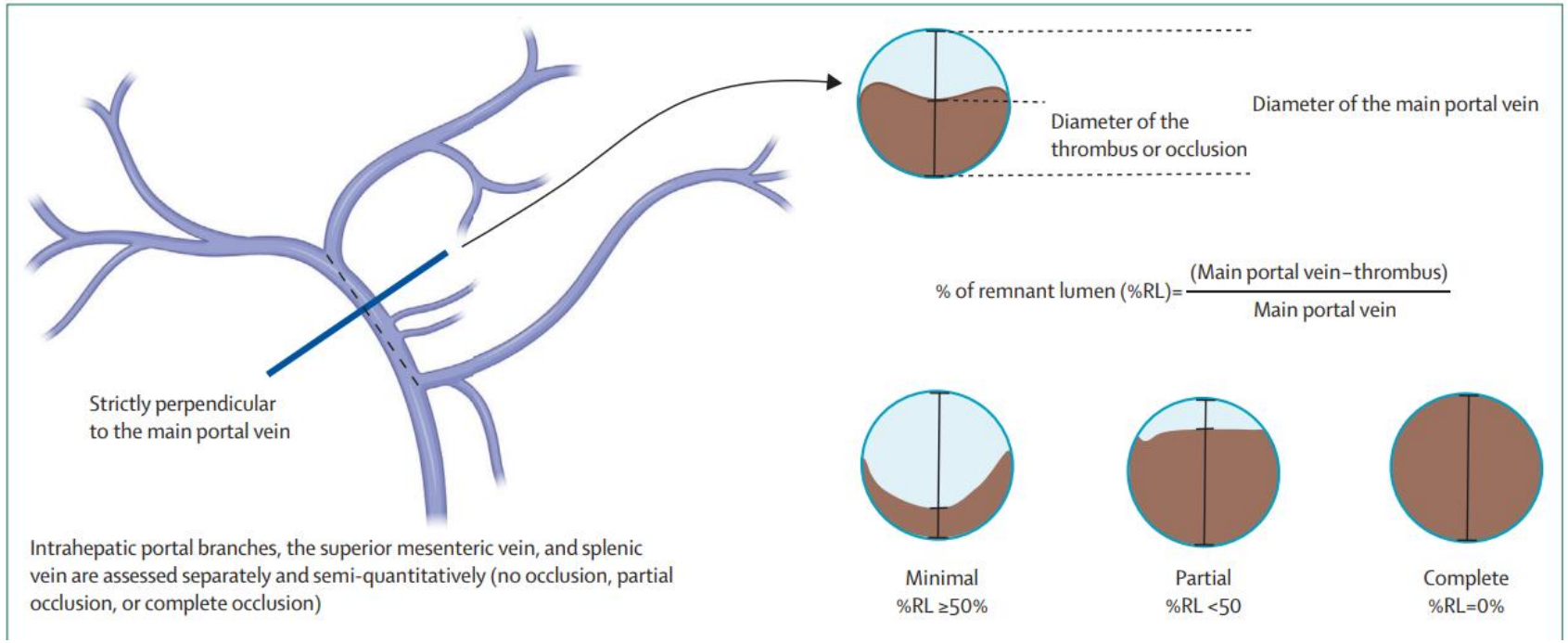
(flow better visualized by CDUS and PWUS)

CDUS of partial PVT
at hepatic hilium

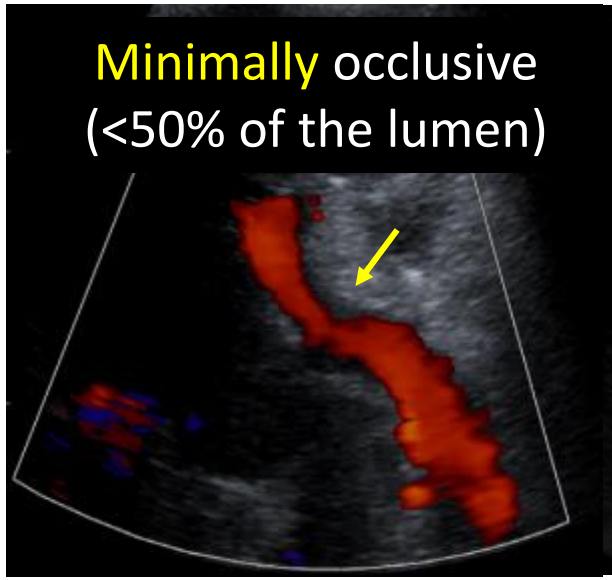


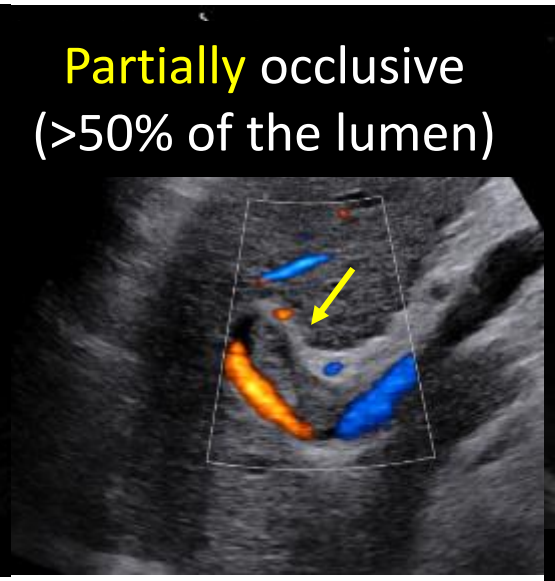
Grey scale first; false negative if CDUS is used on first place!

Extent of PV Thrombosis: terminology according to the Baveno VII consensus



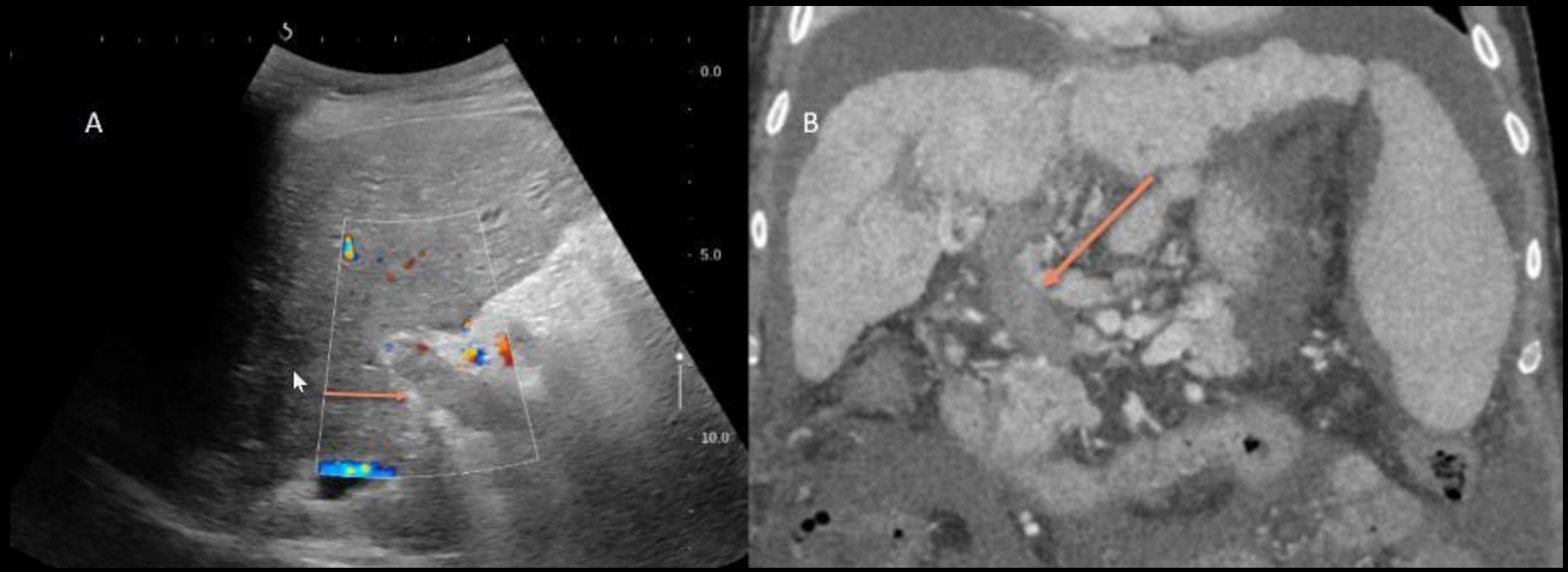
Elkrief et al. Lancet GH 2024



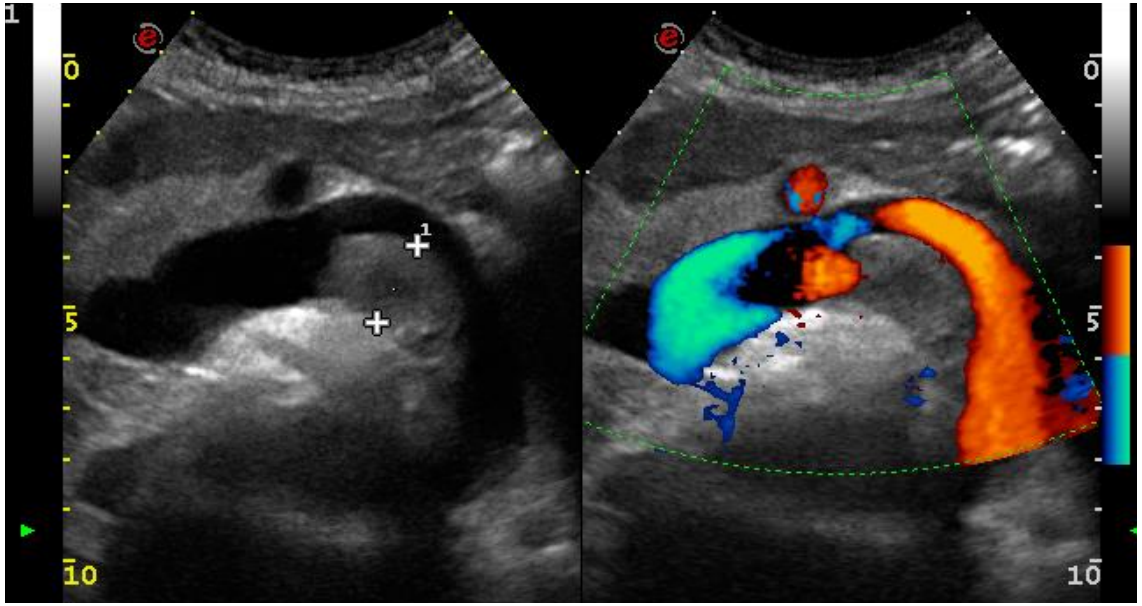


Extent of PV Thrombosis: terminology according to the Baveno VII consensus

Complete PVT



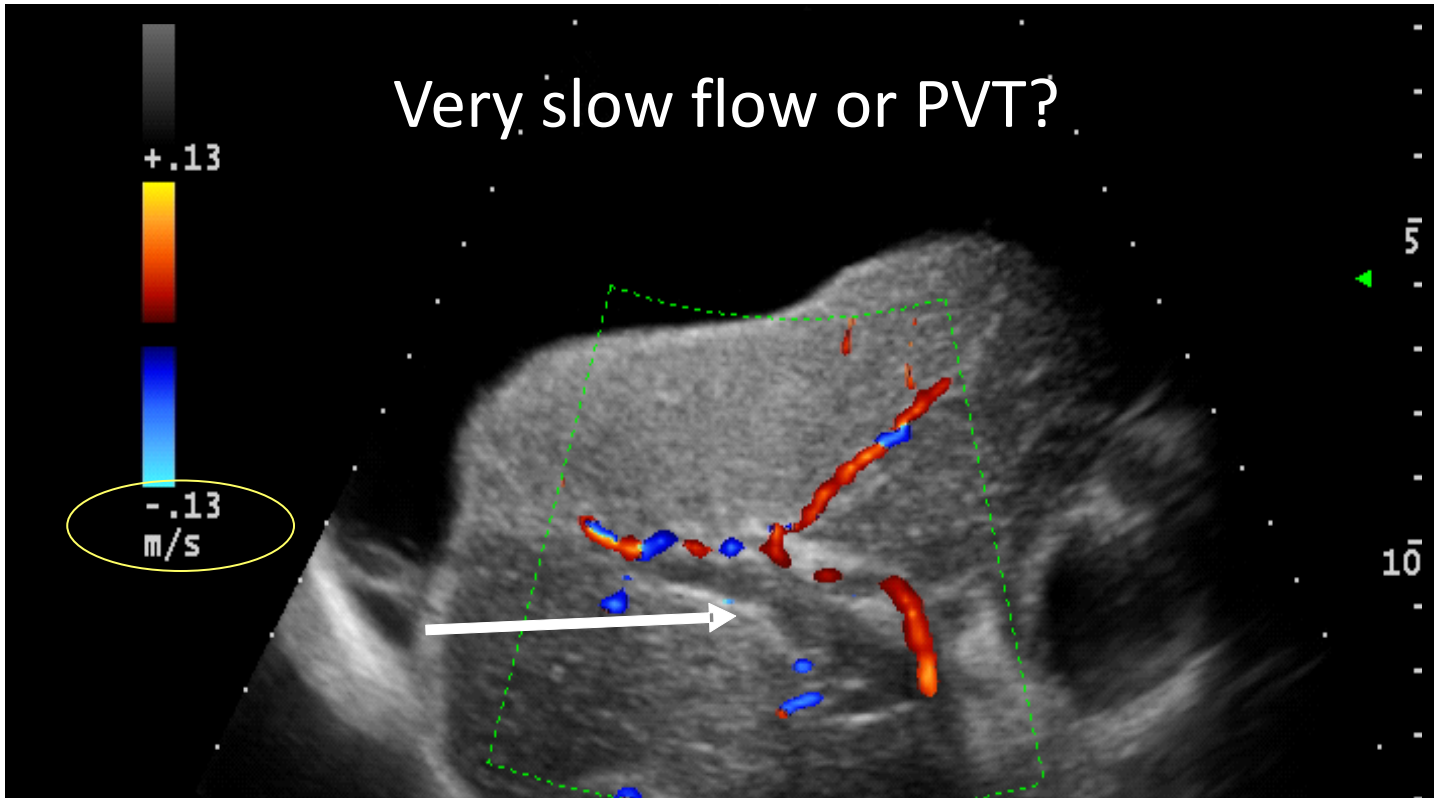
Thrombosis of the splenic vein



US/CDUS is > 90% accurate in diagnosing PVT and SVT, but accuracy is lower for SMVT

Bach et al. Radiology 1996
Kuszyk et al. Radiology 1998

Once thrombosis is diagnosed, study of extension should be done with other imaging techniques


Very slow flow or PVT?
+.13
-.13
m/s
5
10

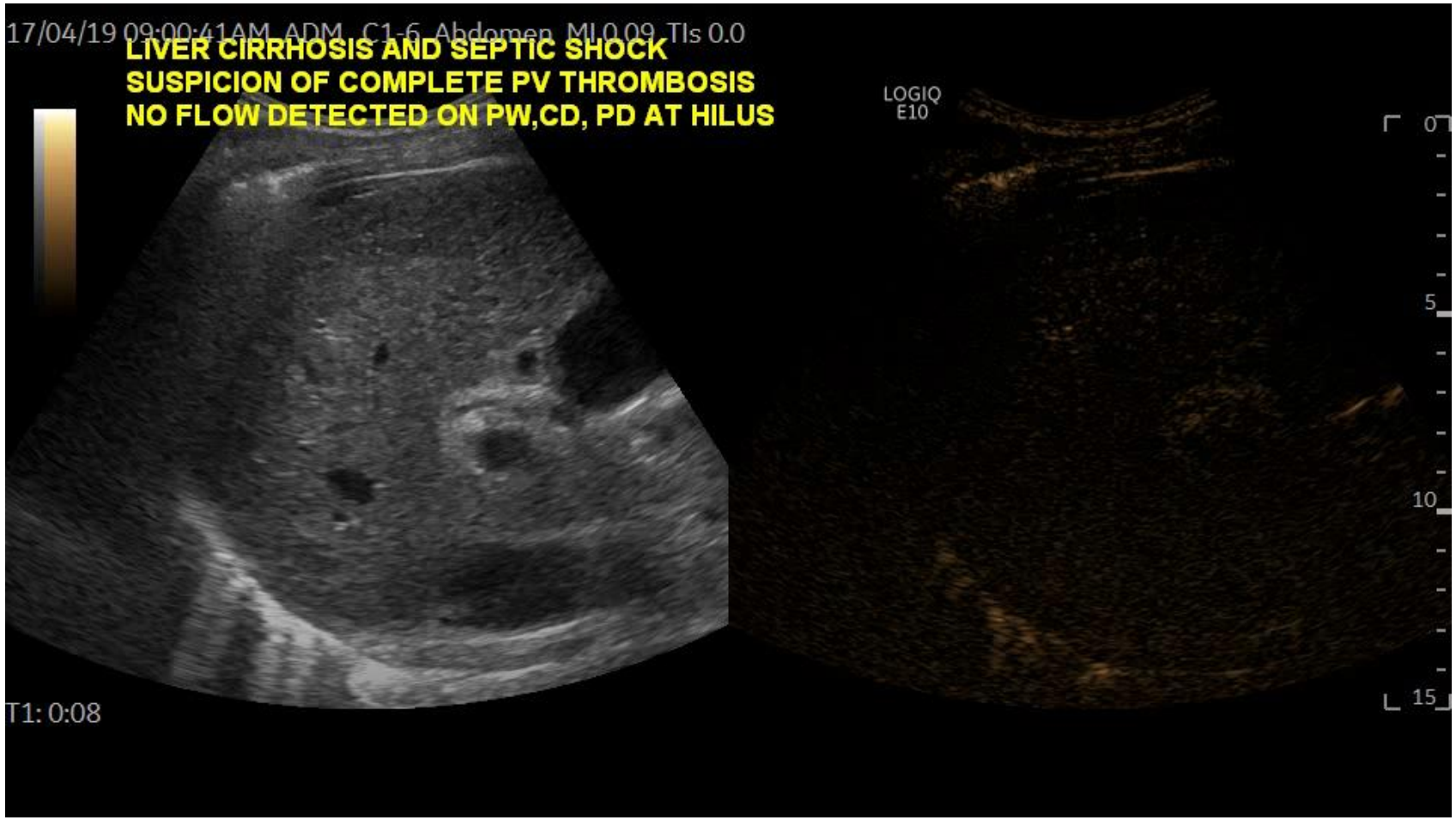
Avoiding US false positives (pseudothrombosis)

1- CDUS: reduce pulse repetition frequency (PRF) to minimum → slow flows

2- Power Doppler US or angle-independent (not Doppler) flow imaging

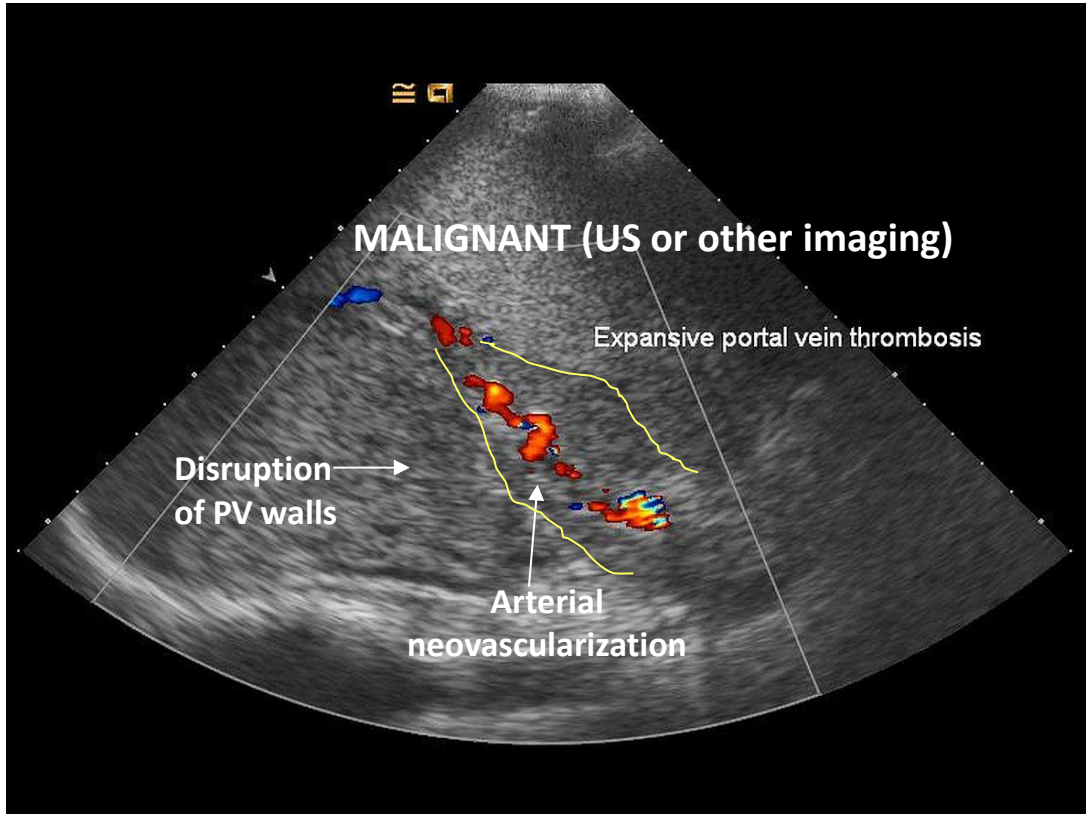
3- CEUS if CDUS and other modes are not conclusive

Avoiding US false positives: use CEUS

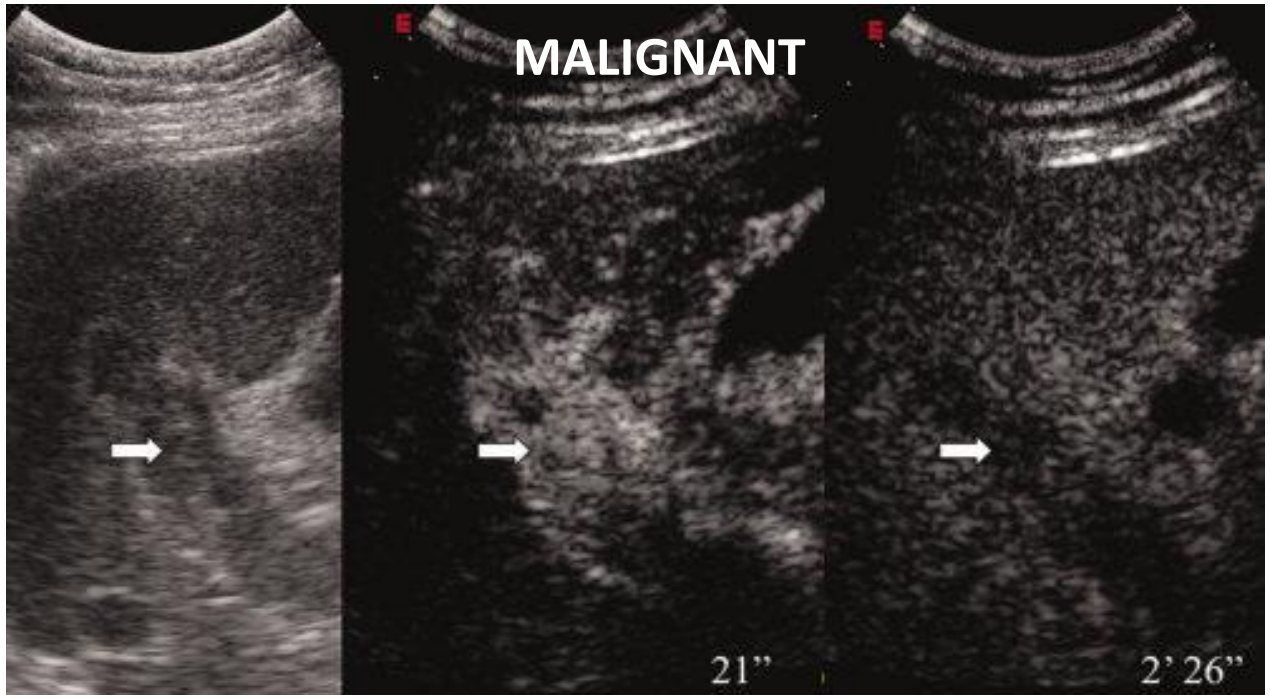


Differentiating between benign PVT and neoplastic vascular invasion (“tumor in vein”, “malignant” PVT)

US differentiation of benign vs. malignant PVT in patients with cirrhosis and HCC



CEUS for differentiating benign vs. malign portal vein thrombosis in the patients with cirrhosis and HCC



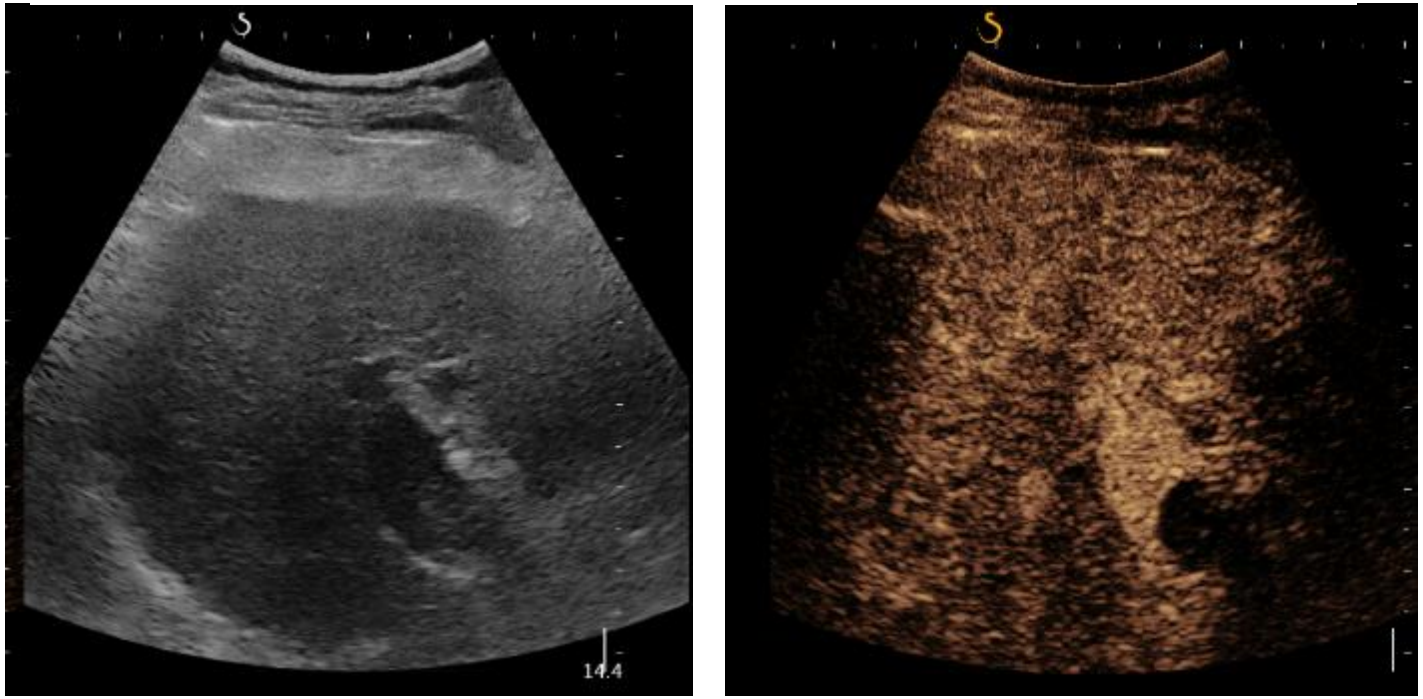
Piscaglia et al. Liver Traspl 2010

In 50 patients undergoing biopsy of the PV thrombus: better performance of CEUS vs CT scan in PVT detection ($p < 0.0001$) and thrombus characterization ($p = 0.0001$)

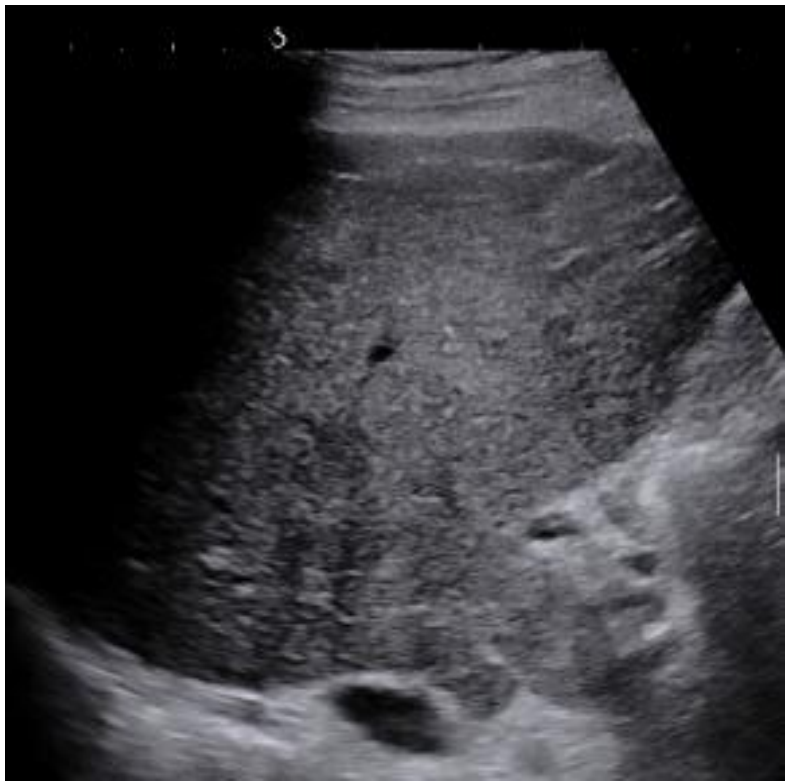
For neoplastic vascular invasion: Sens 98% CEUS vs 67.6% CT, specificity 100% CEUS, vs. 60% CT

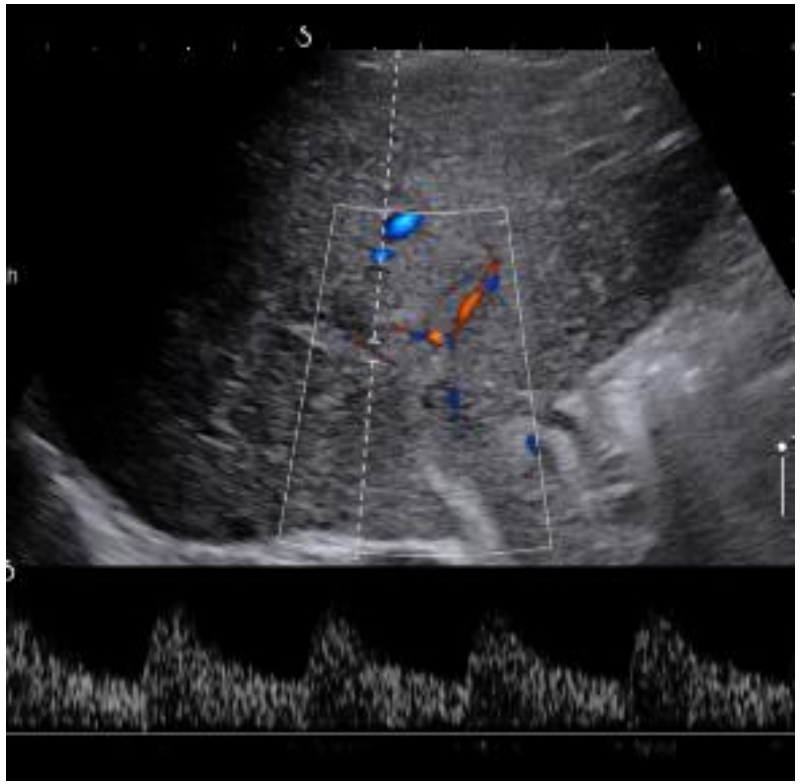
Rossi et al. Eur Radiol 2008

Partial PVT in a patient with HCC: benign

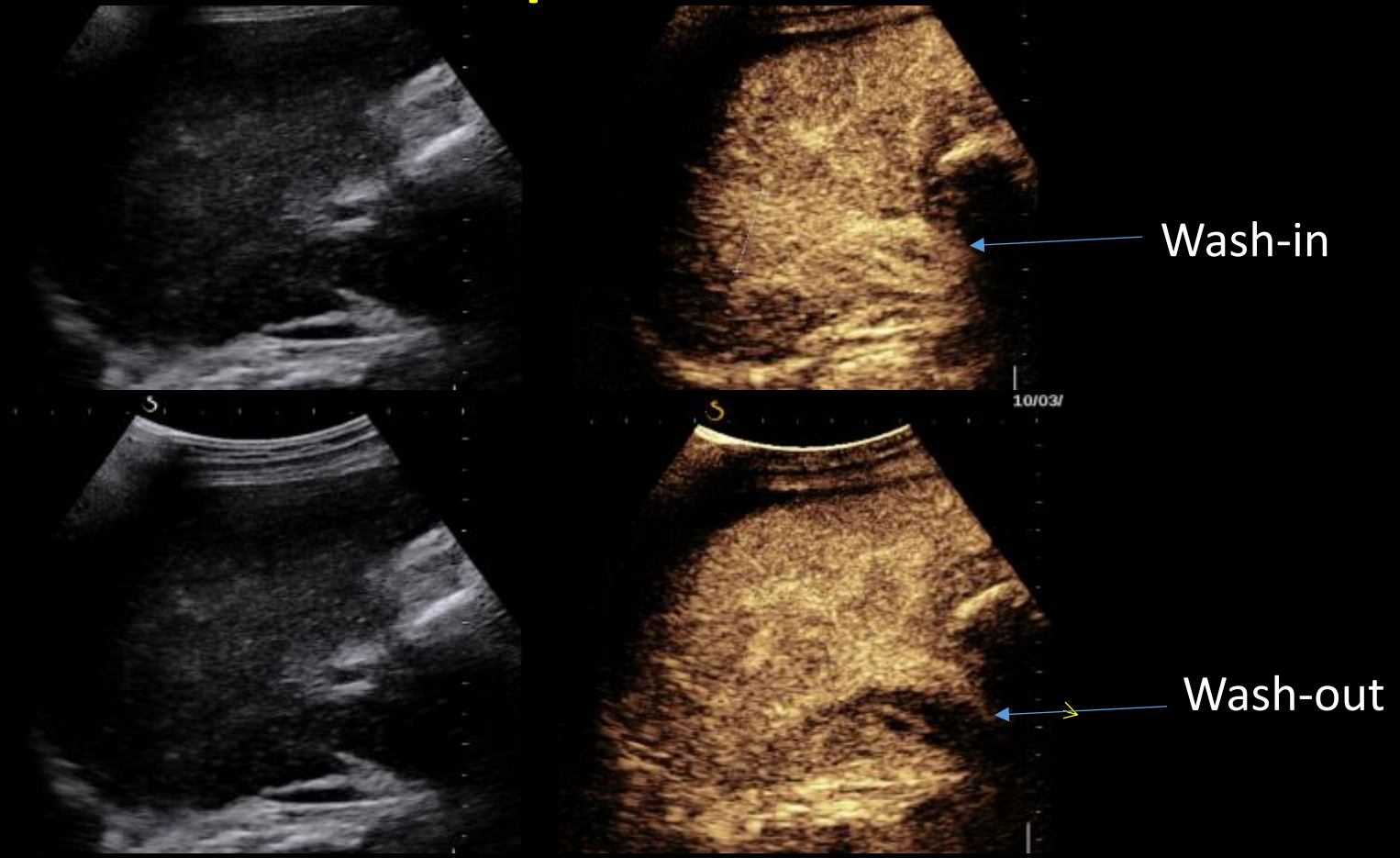


Obvious vascular invasion in a patient with HCC: MALIGNANT aspect on B mode and Doppler

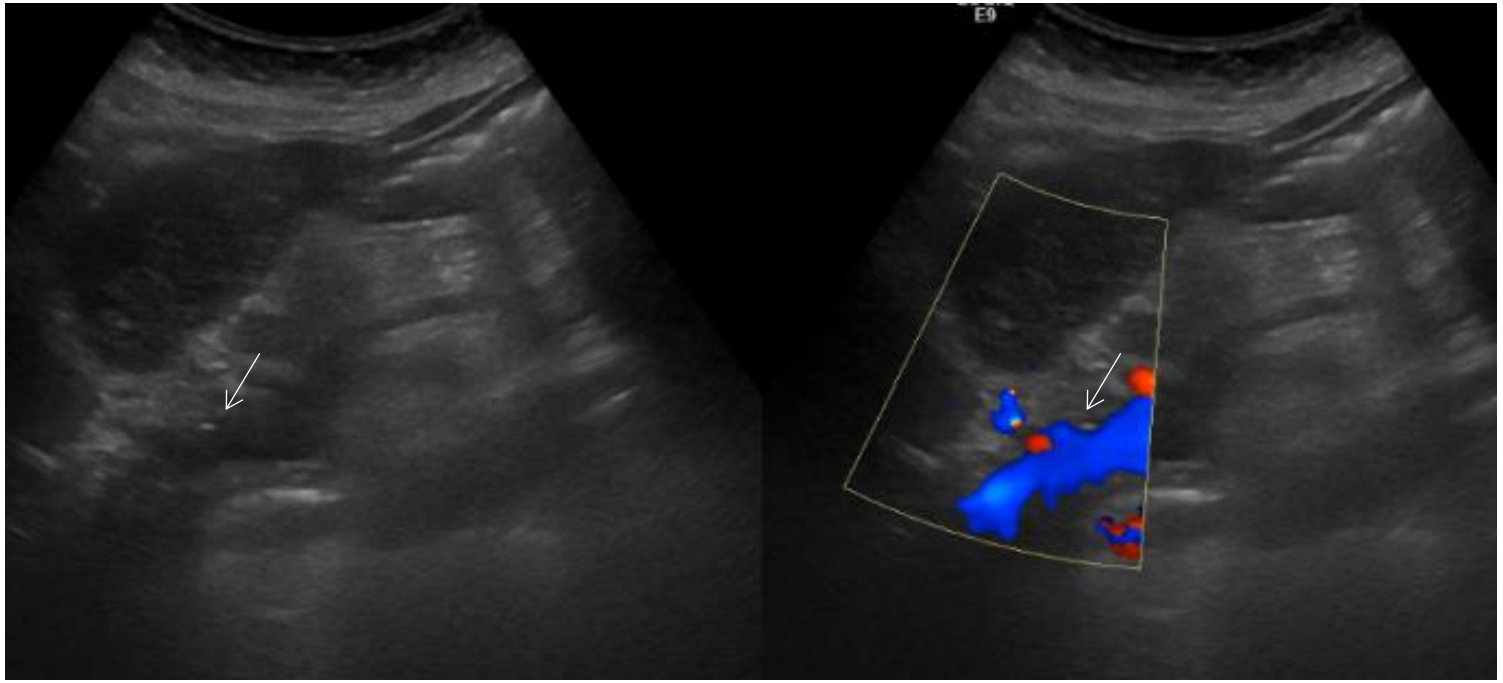




If not obvious: perform CEUS



Chronic PVT
Calcifications of the wall: specific sign

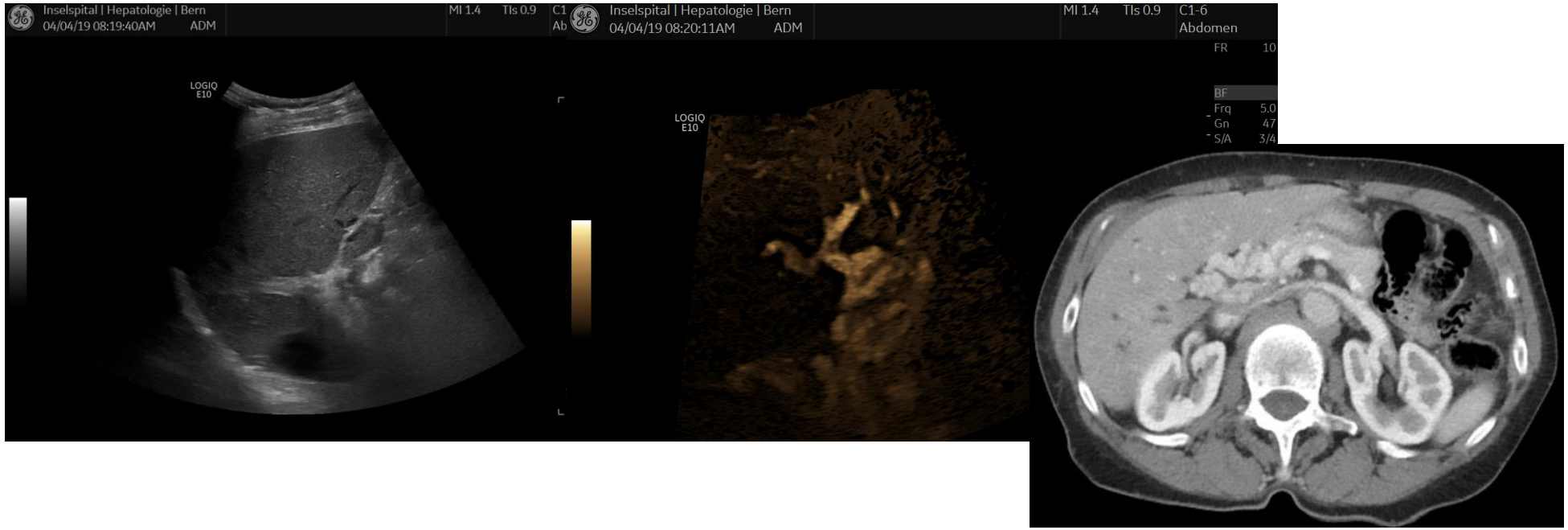


Even if the vessel is patent, calcifications indicate that thrombosis had occurred

Important in candidates to LT

(PV wall more prone to dissection)

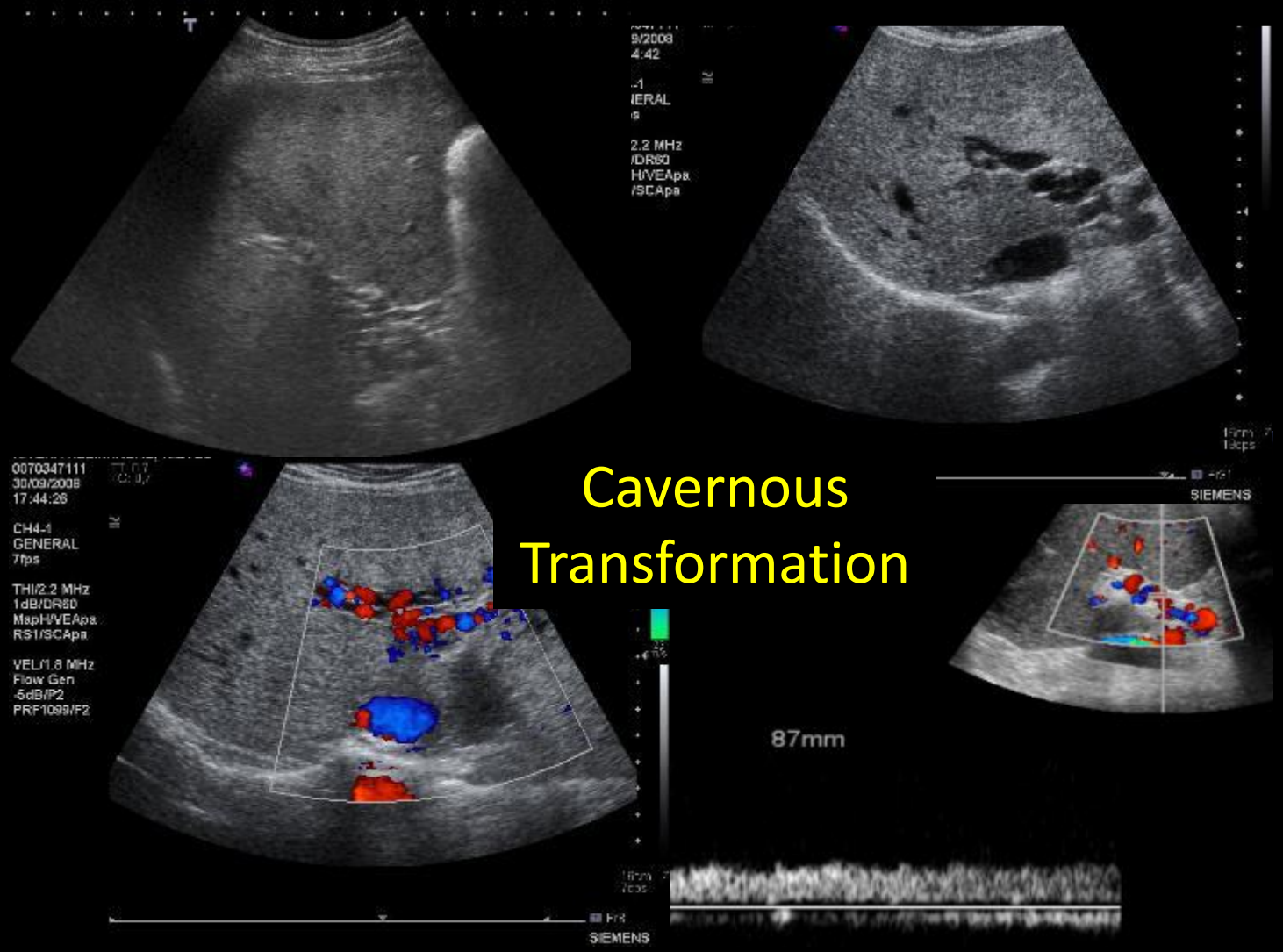
Cavernous Transformation

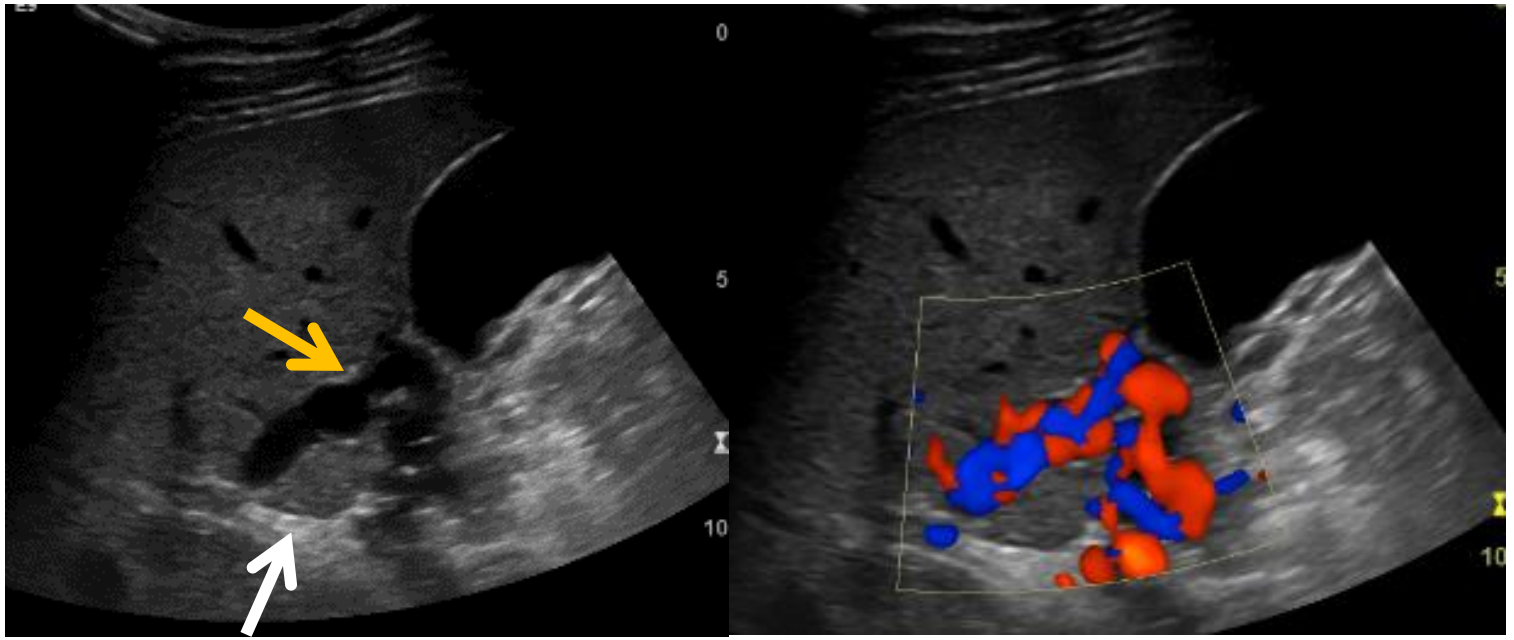


Hepatopetal collateral veins developing as soon as 15-30 days

after the onset of portal vein thrombosis.

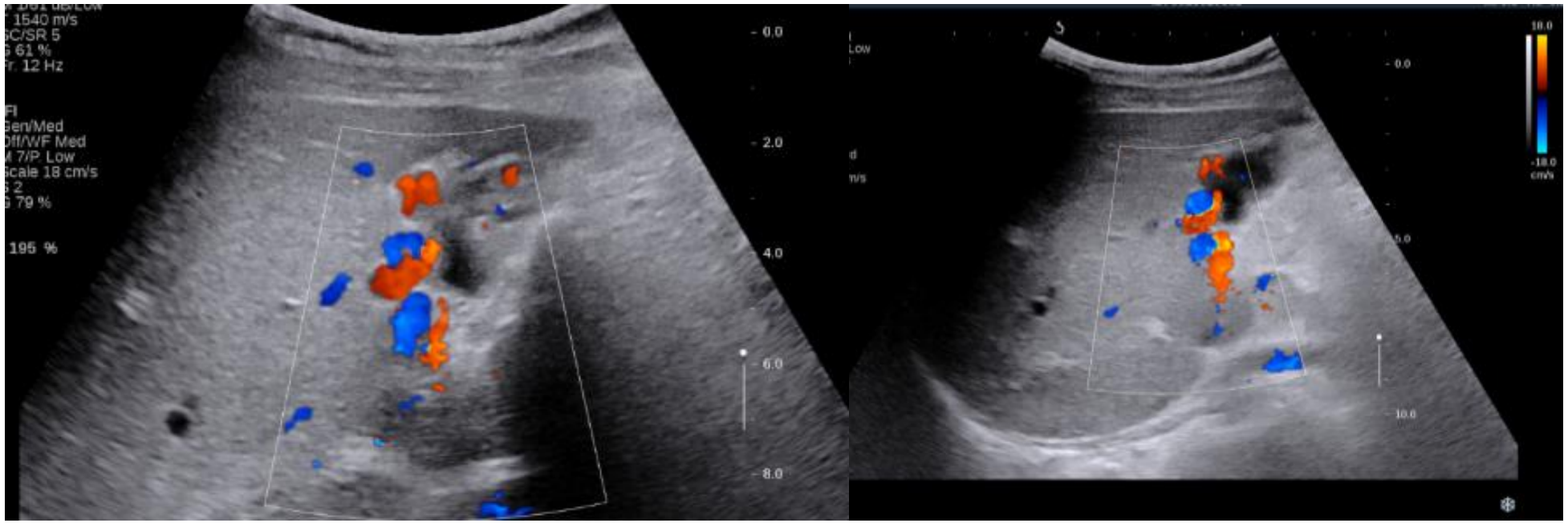
Zhang, World J Gastroenterol, 2011; reviewed in Berzigotti et al. Nature Reviews Gastroenterol Hepatol 2014; De Gottardi, Berzigotti, et al. Ultraschall in Med, 2018; Rodrigues et al. Abdom Radiol 2018


Cavernous
Transformation
87mm
SIEMENS
SIEMENS



White arrow: native portal vein shows complete old thrombosis replaced by fibrous tissue
Yellow arrow: Cavernous transformation; note that a large dominant vessel (9 mm) is observed

Collaterals at the gallbladder wall



Take home messages: BCS and PVT

- Ultrasound/CDUS is the first line technique to be performed in case of suspected BCS and PVT, allowing > 90% accuracy in expert hands
- In difficult cases the use of CEUS allows a better evaluation of PV and HVs patency
- Look for signs of chronic disease (collateral circulation; calcifications in PVT)
- Assessment of extension of PVT needs to be done (CT or MRI); mapping of collaterals can be done with these techniques
- Imaging report should describe patency/thrombosis of all vessels at first examination AND in the follow-up

Arteroportal fistulas

Can occur at any level between the HA and the PV system.
Intrahepatic APF are mostly iatrogenic/traumatic, but potentially reversible PH!

B-mode: anechoic rounded structures connecting a branch of the hepatic artery to a portal branch.

CDUS and PWUS:
- localized web of vascular channels
- hepatofugal flow in one/more portal vessels (according to size and site)
- pulsatile PV flow
- Difference ≥ 25% in PI/RI in right vs. left HA

Lafortune et al. J Ultrasound Med 1986
Bolognesi et al. Radiology 2000

Intrahepatic A-P fistula