

Malformazioni vascolari epatiche: Ruolo degli ultrasuoni



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PATOLOGIE VASCOLARI EPATICHE

- Trombosi portale (PVT)
- Trombosi arteria epatica o sue diramazioni
- Ostruzione Extraepatica della Vena Porta (EHPVO)
- Sindrome di Budd-Chiari (BCS)
- Sindrome da ostruzione sinusoidale (SOS) o veno-occlusiva (VOD)
- Ipertensione portale post-sinusoidale da cirrosi
- Morbo di Rendu-Osler
- Fistole arteroportali (intraepatiche o intraspleniche) con ipertensione portale
- Aneurisma / pseudoaneurisma arteria epatica / splenica



Il “laboratorio” epatico

Condizioni che producono ipossia

- **GOT** ↑↑
- **GPT** ↑
- **GammaGT** (GlutamilTransferasi) ↑↑
- **Fosfatasi alcalina** ←
- **Bilirubina totale** ↑
- **Bilirubina diretta** ↑
- **INR** ← se grave ↑
- **Protidemia totale + elettroforesi proteine** ←
(cambia solo se malnutrizione)
- **Emocromo** (leucociti, emazie, piastrine) ←
(variabile secondo eziologia)



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PVT e EHPVO non alterazioni enzimi e funzione epatica

BCS alterazioni funzioni epatica in relazione a grado di ostruzione e rapidità insorgenza

Nel lungo termine biliopatia ipertensiva = gGT e ALP e Bilirubina

Piastrine basse e anemia normocromica con reticolociti alti associati a splenomegalia (talora leucopenia)

NB: piastrine alte o HCT >45% e splenomegalia pensare a Trombocitemia / Policitemia



PATOLOGIE VASCOLARI EPATICHE

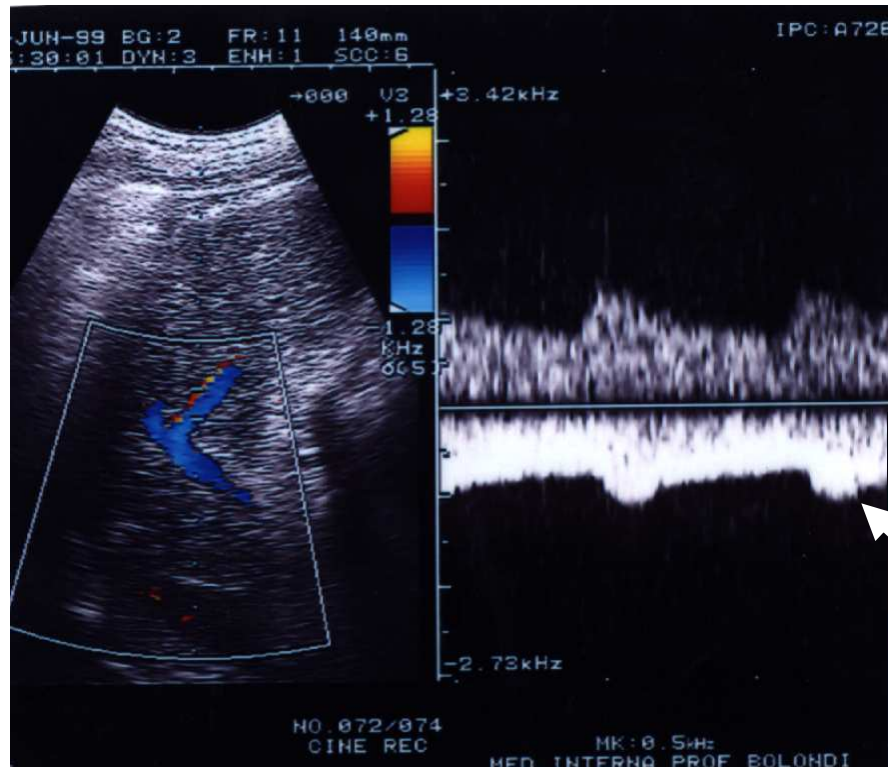
- Trombosi portale (PVT)
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↓
Laboratorio di solito "muto"..
Rischio anemizzazione acuta e
shock in caso di rottura

↓
Ipersplenismo, modesta
splenomegalia, varici esofagee



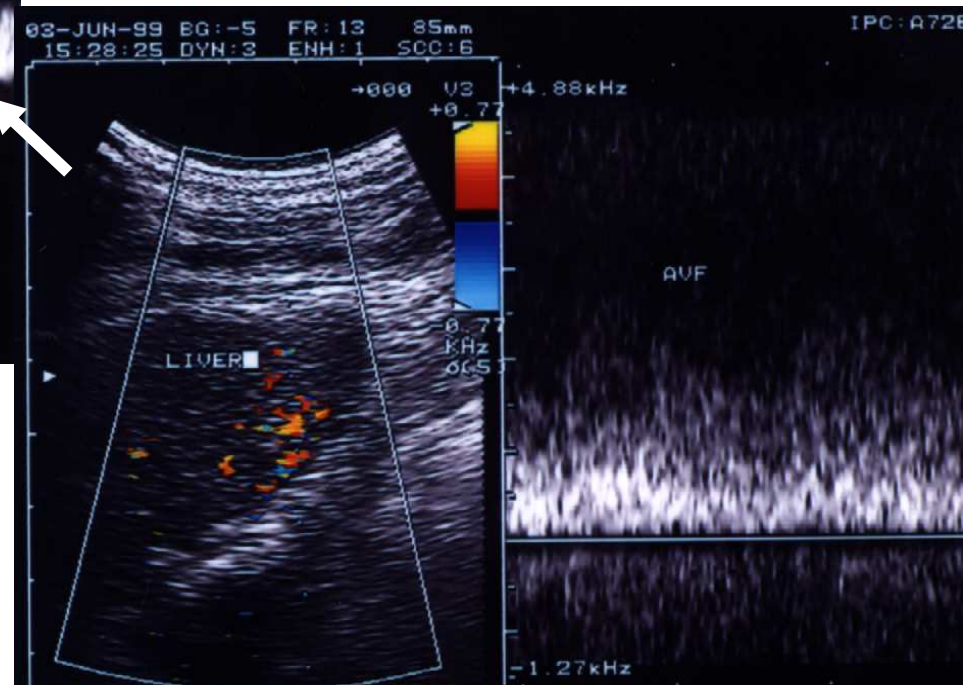
IPERTENSIONE PORTALE DA FISTOLA Artero Portale intraepatica

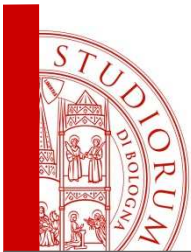


Arterializzazione flusso portale
invertito

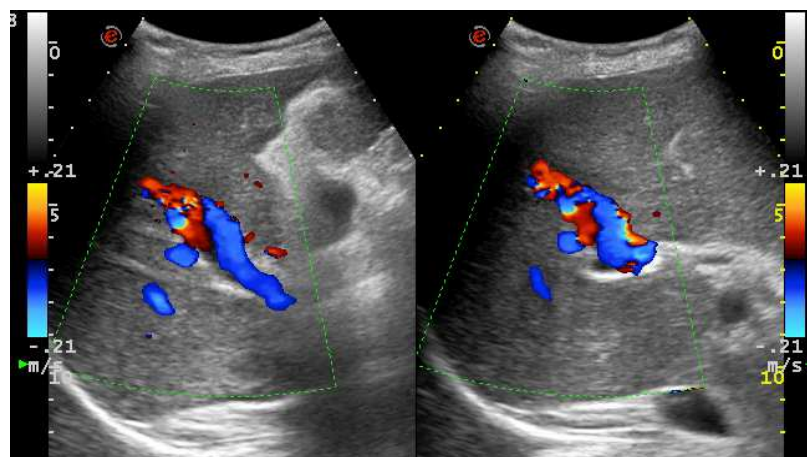
Segni laboratoristici di
ipertensione portale

Sede fistola arteroportale
intraepatico





IPERTENSIONE PORTALE DA FISTOLA Artero Portale intraepatica

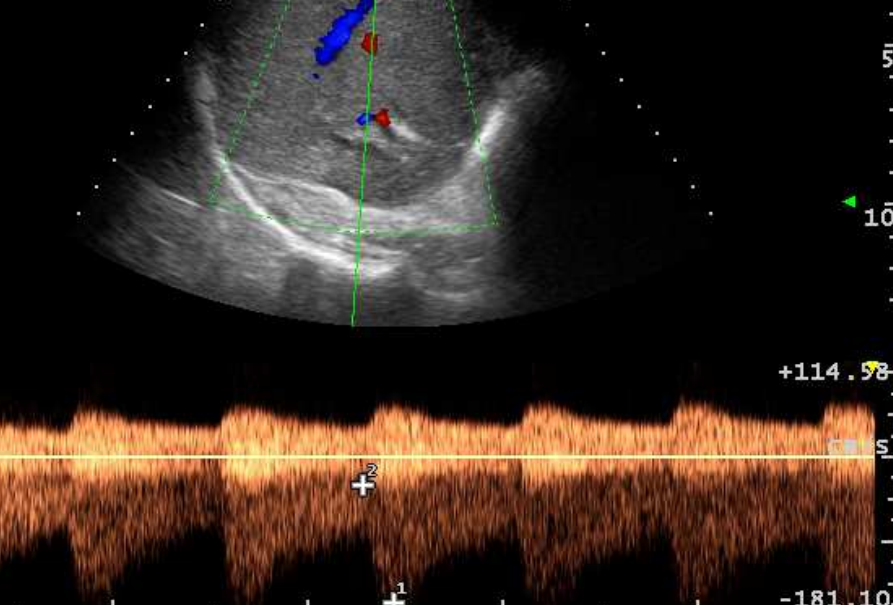
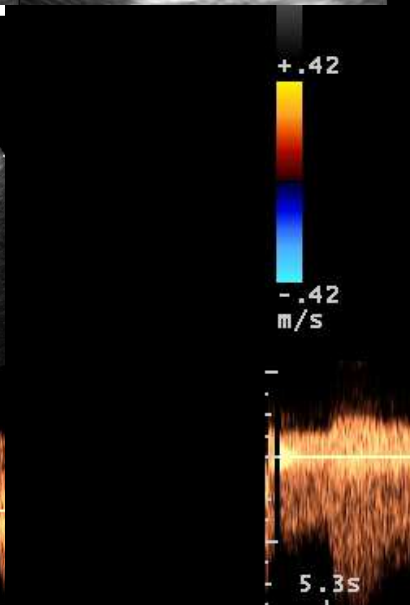
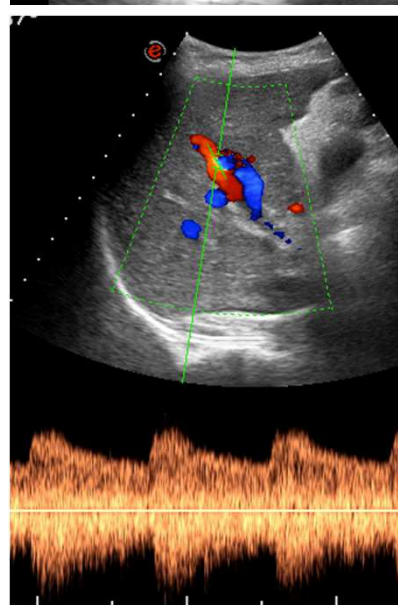


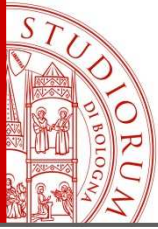
Pancitopenia con splenomegalia,
varici a rischio, lieve alterazione gGT

INTERNA-UNIV BOLOGNA

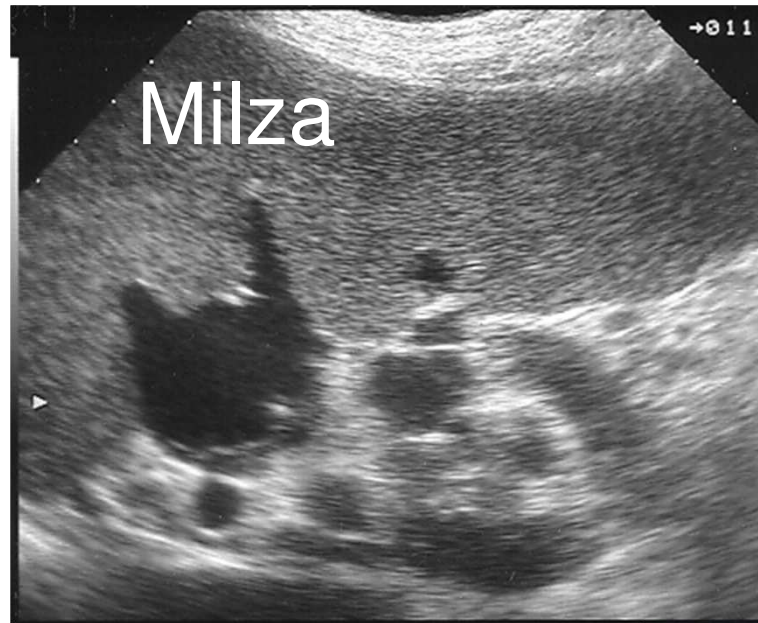
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G 40%	CFM F	2.3 MHz	G 38%	PW F	2.1 MHz	G 40%
XV +3	PRF	2.5 kHz		PRF	8.0 kHz	
PRS 4-3	PRC M/ 1		PRS 4	PRC	4	
C 0	FP 3			FP 65 Hz		
110 0°						





IPERTENSIONE PORTALE DA FISTOLA Artero Portale intrasplenica

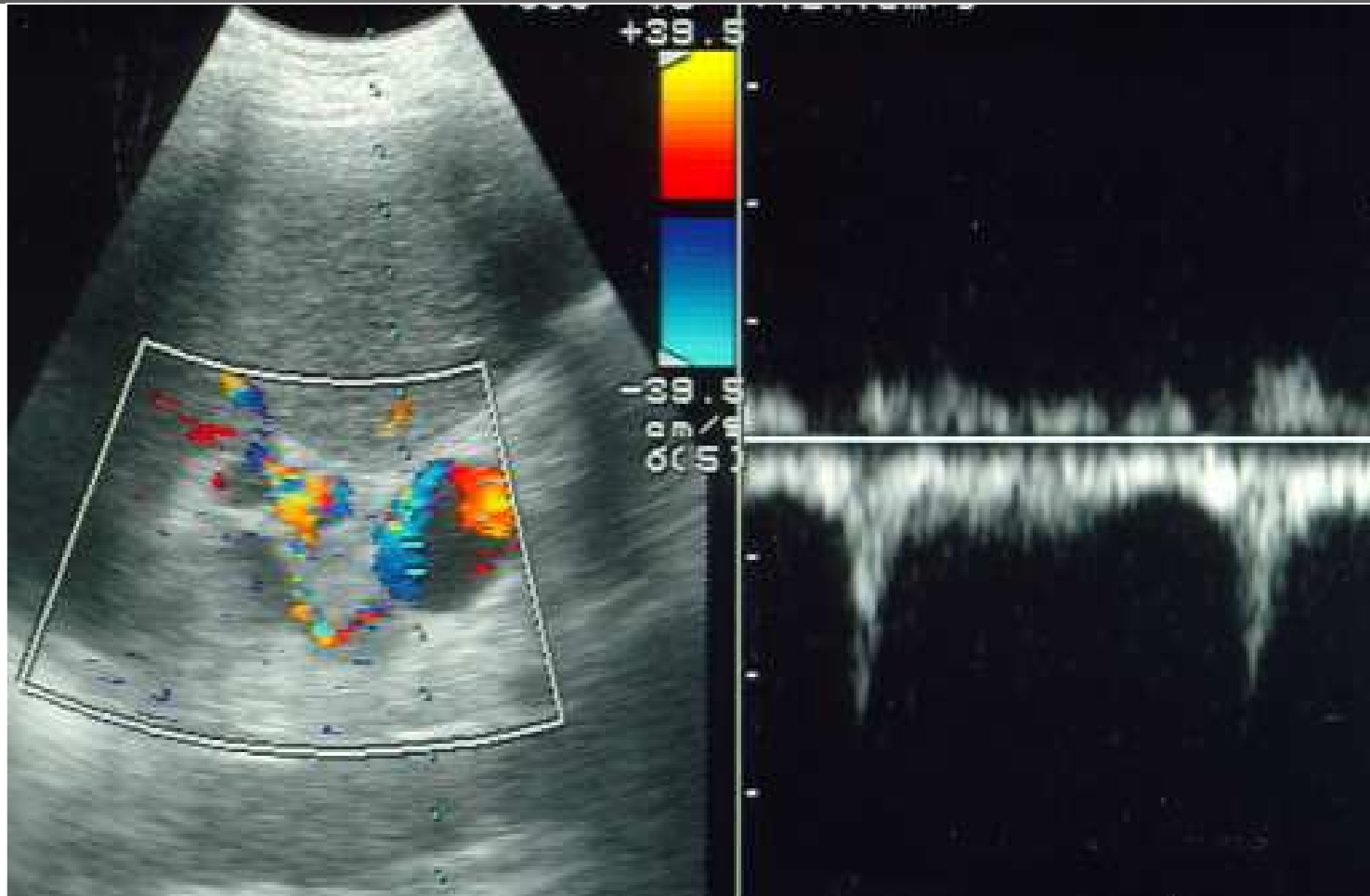


Fistola AV all'ilo splenico.
Il "segno del gatto"

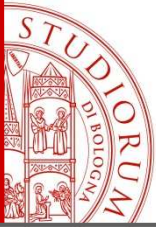


[Piscaglia, J Clin Ultrasound, 1998;26:103-5]

ANEURISMA ARTERIA SPLENICA



Aneurisma arteria splenica. Laboratorio “muto” fino a rottura (shock ed anemizzazione acuta). Talora dolenzia (se grande)



SINDROME DI BUDD-CHIARI

Descritta per la prima volta da George Budd nel 1845 e successivamente da Hans Chiari che espose 13 casi nel 1899

G. Budd:

On diseases of the liver.

London, J. Churchill, 1845.

Page 135.

H. Chiari:

Erfahrungen ber Infarktbildungen in der Leber des Menschen.

Zeitschrift Heilkunde, Prague, 1898, 19: 475-512.



Hans von Chiari

Budd–Chiari syndrome: a review by an expert panel

Harry L.A. Janssen^{1,*}, Juan-Carlos Garcia-Pagan², Elwyn Elias³, Gilles Mentha⁴, Antoine Hadengue⁴, Dominique-Charles Valla⁵, for the European Group for the Study of Vascular Disorders of the Liver

¹Department of Gastroenterology and Hepatology, University Hospital Rotterdam, P.O. Box 2040, 3000 CA, Rotterdam, The Netherlands

²Hepatic Hemodynamic Laboratory, Liver Unit, Hospital Clinic, Barcelona, Spain

³Liver Unit, Queen Elizabeth Hospital, Birmingham, UK

⁴Division of Hepatology and Transplantation Unit, University Hospital of Geneva, Geneva, Switzerland

⁵Service d'Hépatologie, Hôpital Beaujon, Clichy, France

of MEDICINE

2004

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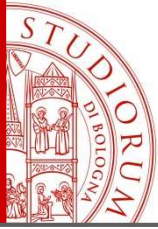
CURRENT CONCEPTS

The Budd–Chiari Syndrome

K.V. Narayanan Menon, M.D., Vijay Shah, M.D., and Patrick S. Kamath, M.D.

DEFINIZIONE

Si definisce **Sindrome di Budd–Chiari** qualsiasi ostruzione al deflusso venoso epatico a qualsiasi livello, dalle piccole vene epatiche fino alla giunzione della vena cava inferiore con l'atrio destro, indipendentemente dalla causa. Le ostruzioni causate dalla malattia veno occlusiva epatica e le patologie cardiache vengono escluse da questa definizione.



CAUSE COMUNI

Stati discoagulativi

Ereditari:

Deficit di anti-trombina III

Deficit di Proteina C

Deficit di Proteina S

Acquisiti

Disordini mieloproliferativi

Emoglobinuria Parossistica Notturna

Sindrome da Anticorpi Antifosfolipidi

Neoplasie solide

Gravidanza

Uso di contraccettivi

**75% DELLE
FORME
PRIMITIVE**

CAUSE NON COMUNI

Invasione neoplastica

HCC

Carcinoma renale

Carcinoma surrenalico

Miscellanea

Aspergillosi

Traumi

IBD

Sindrome di Behcet

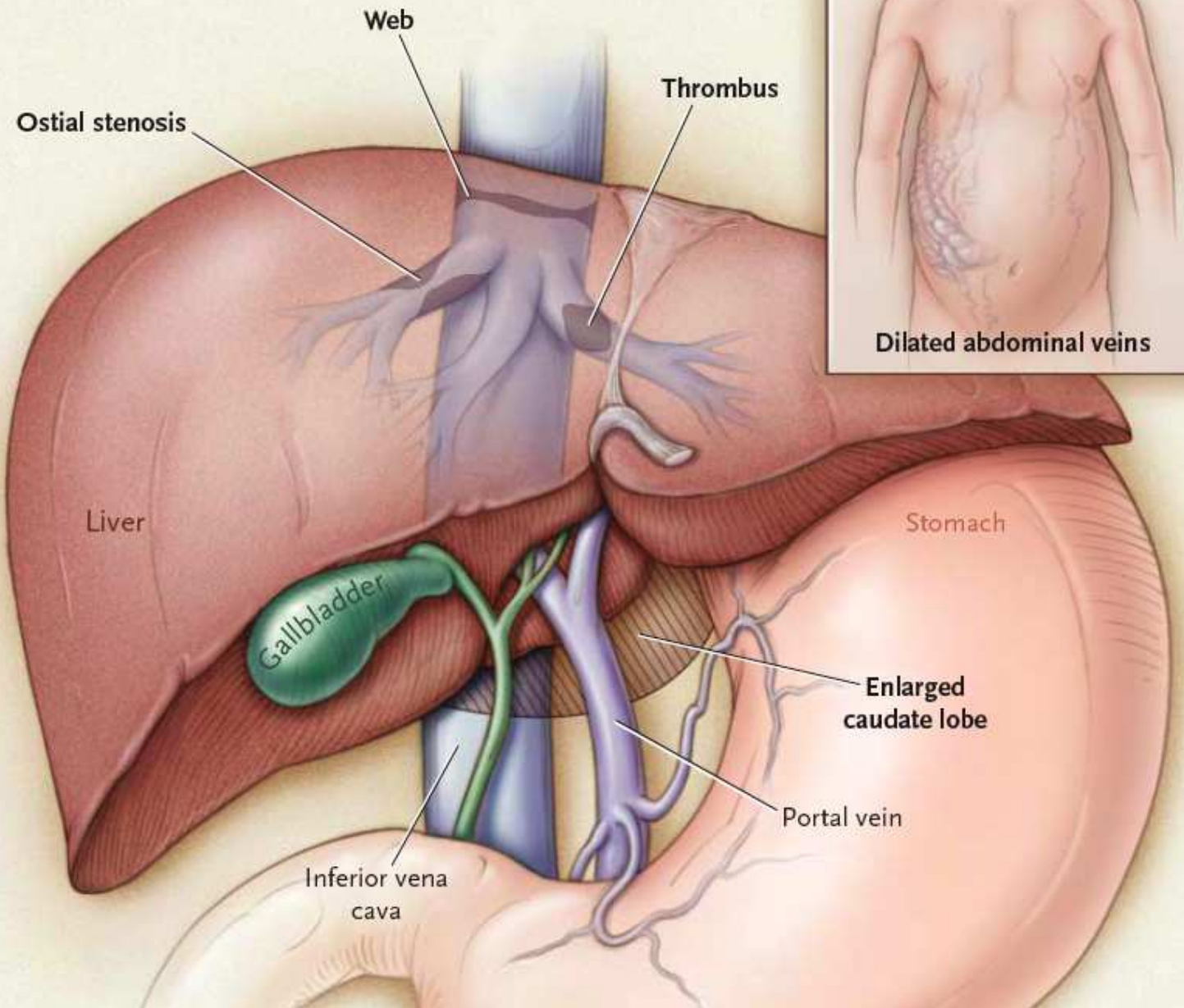


La policitemia vera è responsabile del 10-40 % dei casi di malattia, mentre la trombocitemia essenziale e la mielofibrosi appaiono meno frequenti. Cloni eritroidi sono presenti in circa l' 87% dei casi di sindrome di Budd-Chiari idiopatica, suggerendo che una patologia mieloproliferativa sia all' origine delle situazioni in cui l' eziologia non è apparente.

Emoglobinuria Parossistica Nottura: circa il 5-10% dei casi di sindrome di Budd-Chiari. E' molto importante riconoscerla, data la scarsa prognosi del trapianto di fegato in questi pazienti, dovuta all' elevato rischio di recidiva di trombosi

A

2-Sede dell' ostruzione





Sindrome di BUDD CHIARI: Manifestazioni cliniche e durata

Il quadro clinico dipende dalla modalità di insorgenza e dall'estensione dell'ostruzione.

Dolore addominale, epatomegalia e ascite sono presenti in quasi in tutti i pazienti. Sono state descritte anche forme asintomatiche. Nausea, vomito ed ittero sono più frequenti nelle forme fulminanti, mentre le varici esofagee e la splenomegalia sono tipiche della forma cronica.

Possiamo distinguere quattro quadri clinici:

Fulminante: encefalopatia epatica entro otto settimane dalla comparsa dell'ittero

Acuta: sintomi di breve durata, ascite intrattabile, necrosi epatica senza formazione di circoli collaterali

Sub-acuta: è la più comune ed ha un esordio subdolo, l'ascite e la necrosi epatica possono essere minime per la formazione di circoli collaterali

Cronica: solitamente si manifesta come complicanza della cirrosi epatica



DIAGNOSI

Quando sospettare una sindrome di Budd-Chiari?

- Ascite, epatomegalia e dolore addominale
- Ascite intrattabile in contrasto con test della funzione epatica minimamente alterati, in pazienti con patologia epatica cronica
- Patologia epatica documentata in un paziente con disordini coagulativi noti
- Epatite fulminante associata ad epatomegalia e ascite
- Epatopatia cronica ad eziologia ignota



DIAGNOSI BCS

- ***Clinica***
- ***Laboratorio:*** GOT e GPT fino a cinque volte superiori il valore normale nelle forme fulminanti, rialzo di fosfatasi alcalina e bilirubina di grado variabile, riduzione dell'albuminemia e della protrombinemia
- ***Esami strumentali***
 - 1) Ecografia e color- Doppler
 - 2) TAC
 - 3) MRI
 - 4) ANGIOGRAFIA



DIAGNOSI BCS

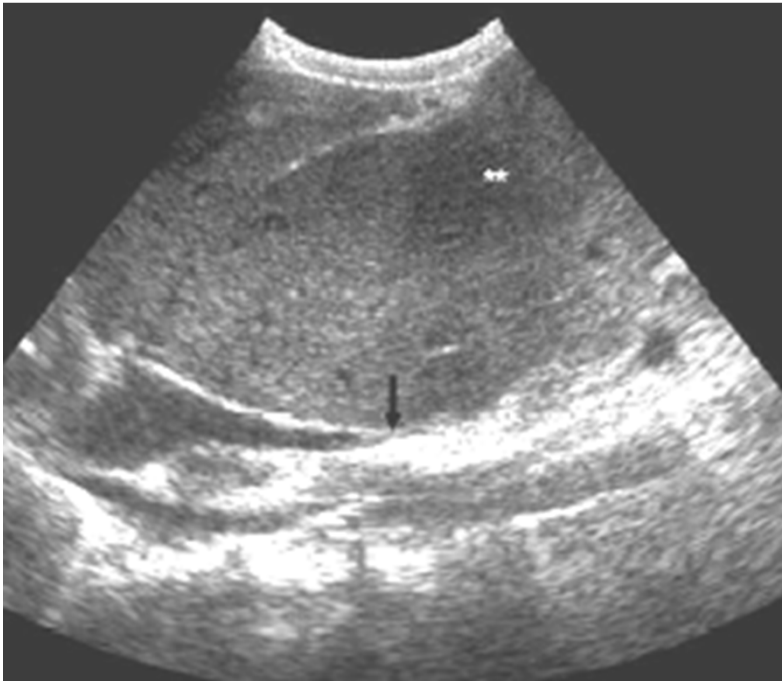
Ecografia convenzionale

- EPATOMEGALIA
- **Spiccata IPERTROFIA DEL LOBO CAUDATO**
- ZONE DI ALTERATA ECOGENICITA' DA INFARCIMENTO EMORRAGICO
- **INCAPACITA' A VISUALIZZARE PARZIALMENTE O TOTALMENTE LE VENE SOVRAEPATICHE**
- STENOSI CON DILATAZIONI PROSSIMALI, ECOGENICITA' INTRALUMINALE, PARETI SOTTILI E TROMBOSI DELLE VENE SOVRAEPATICHE
- LA VENA CAVA PUO' ESSERE SOTTILE A CAUSA DELLA COMPRESSIONE ESERCITATA DAL LOBO CAUDATO
- PRESENZA DI CIRCOLI COLLETERALI INTRAEPATICI, ECCETTO LA VENA PARAOMBELICALE
- Dilatazione delle vene epatiche del lobo caudato (accessorie, sbocco diretto in vena cava)



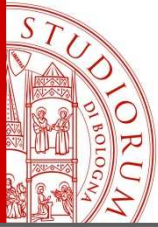
DIAGNOSI BCS

Ecografia convenzionale



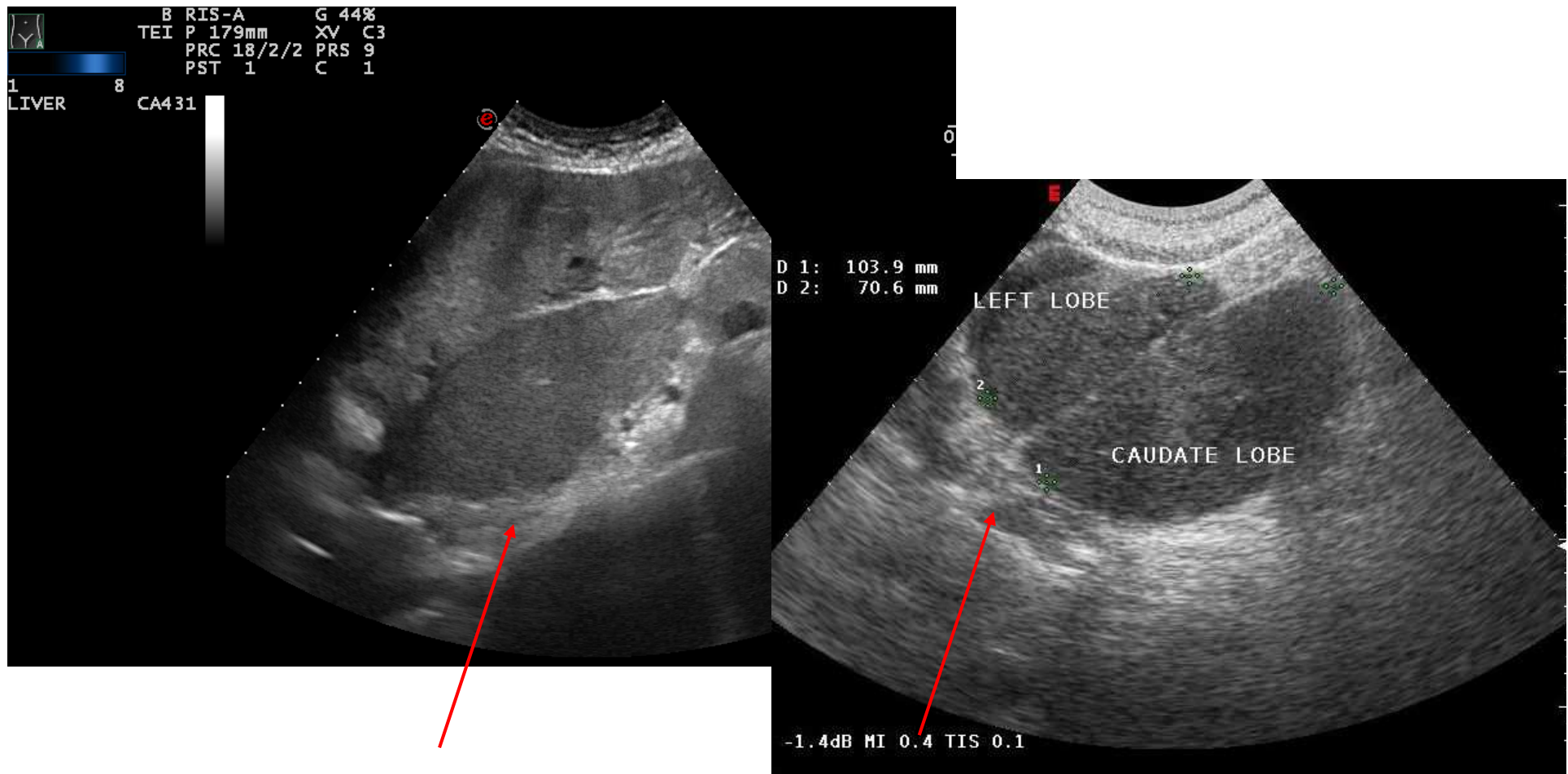
Le vene che drenano il lobo caudato si immettono nella vena cava ad un livello inferiore rispetto alle vene sovraepatiche, determinando un ipertrofia del lobo in caso di occlusione delle altre vene sovraepatiche.

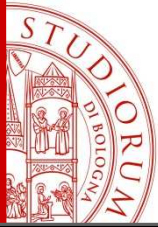
Le vene che drenano il lobo caudato sono visibili nel 50% dei pazienti con sindrome di Budd-Chiari.



DIAGNOSI BCS

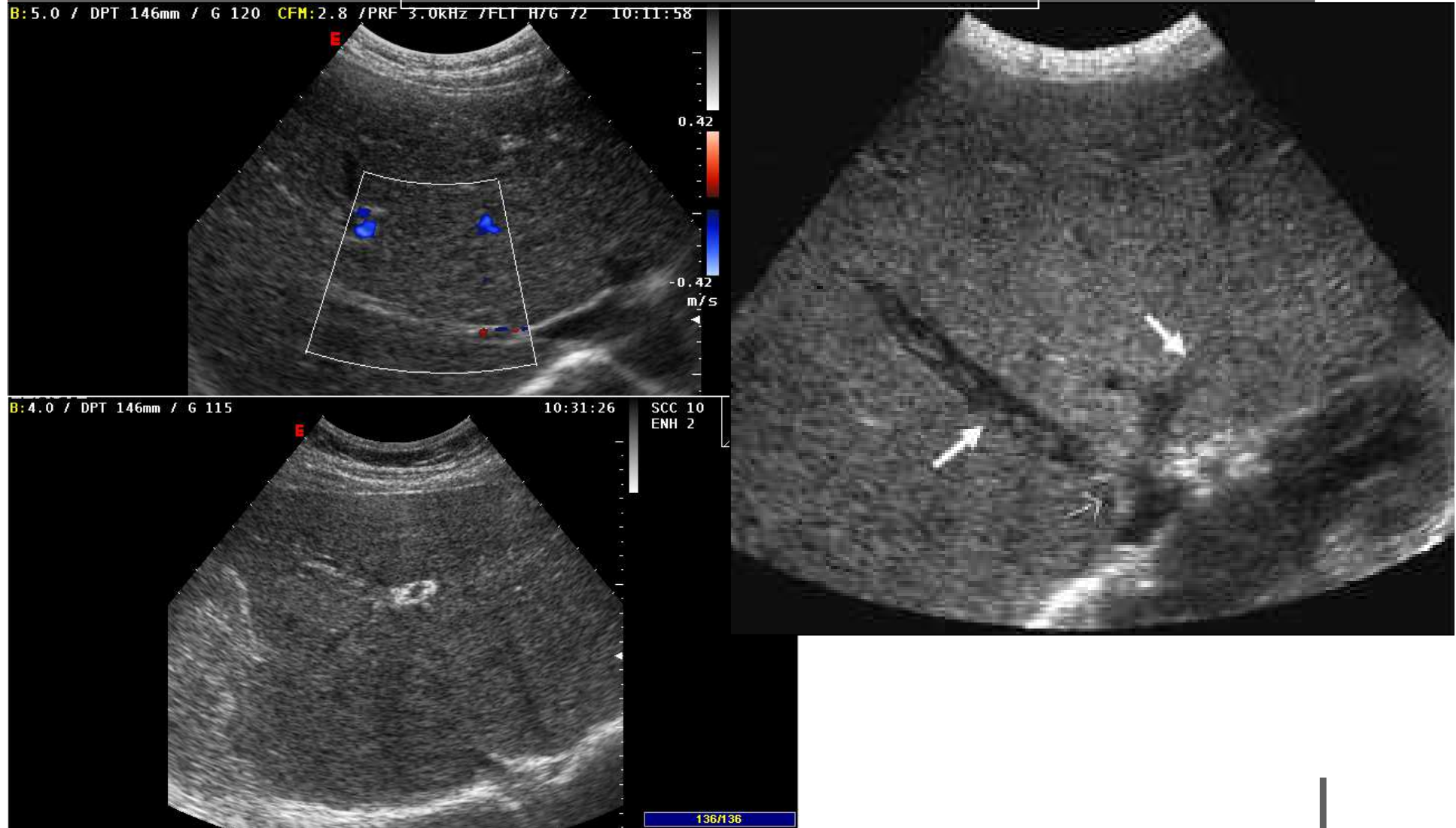
Ecografia convenzionale



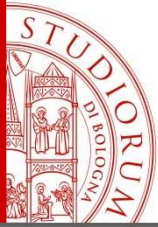


DIAGNOSI BCS

Ecografia convenzionale







DIAGNOSI BCS

Ecografia convenzionale





DIAGNOSI BCS

EcoDoppler

Color Doppler e analisi spettrale

L'eco Doppler, con una sensibilità e specificità fra l'85 ed il 90%, è la tecnica di scelta per una valutazione iniziale quando si sospetta una Sindrome di Budd-Chiari

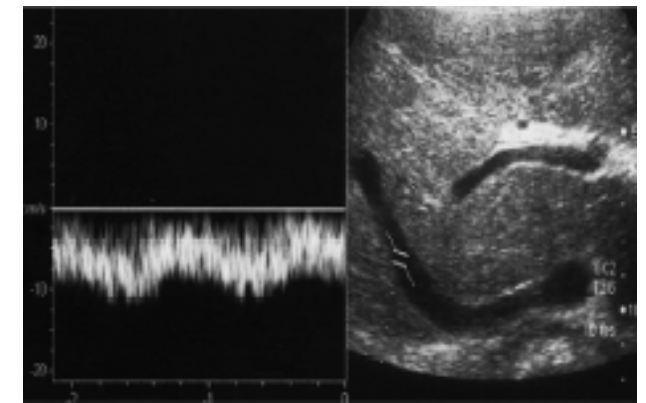
Bolondi L, Gaiani S, Li Bassi S, Zironi G, Bonino F, Brunetto M, Barbara L. Diagnosis of Budd-Chiari syndrome by pulsed Doppler ultrasound. Gastroenterology. 1991

DIAGNOSI BCS

EcoDoppler

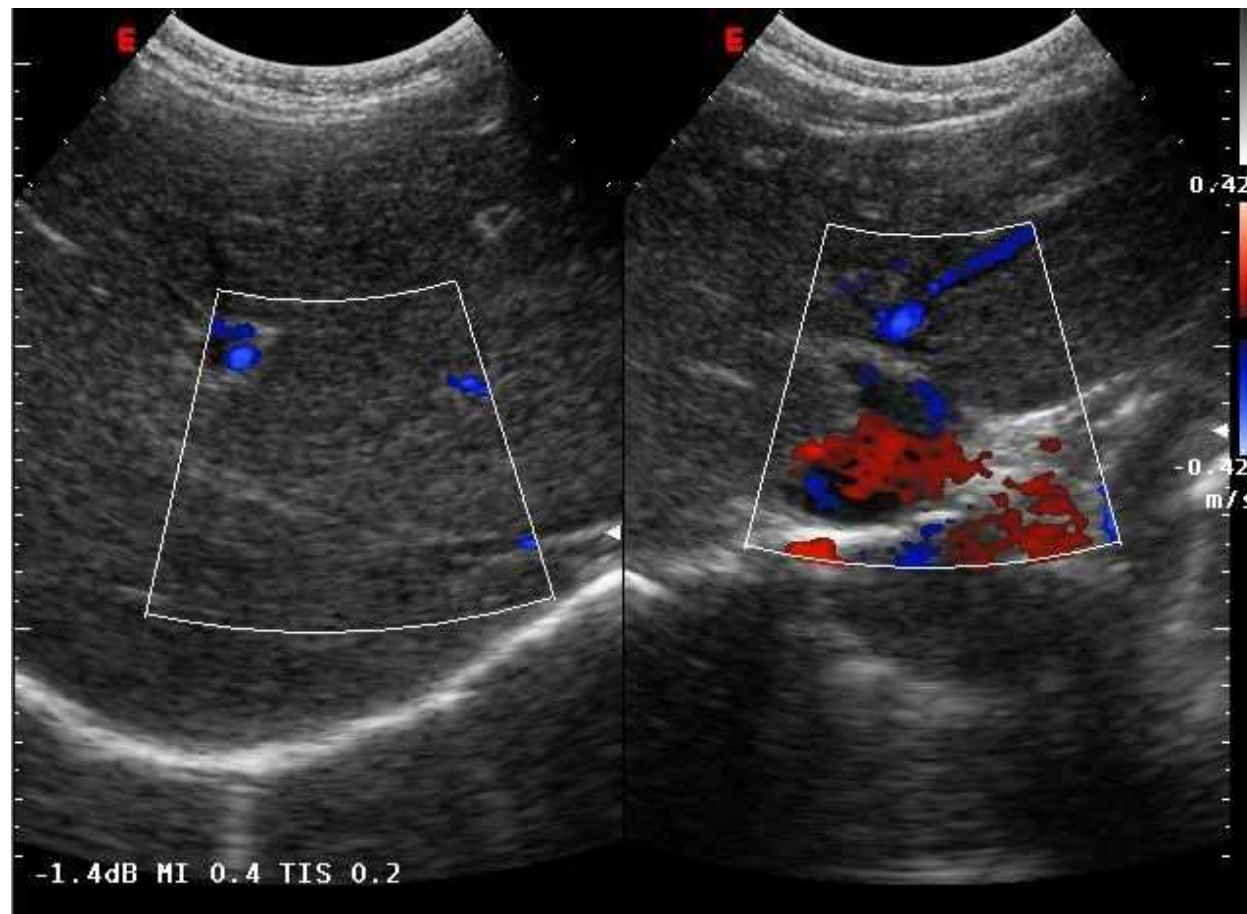
Color Doppler e analisi spettrale

- ASSENZA O INVERSIONE DI FLUSSO NELLE VENE EPATICHE
- FLUSSO AD ALTA VELOCITA' NELLE SEDI DELLE STENOSI
- FLUSSO NELLE VENE EPATICHE E NELLA VENA CAVA INFERIORE DA FASICO AD ASSENTE, INVERTITO, TURBOLENTO O CONTINUO
- FLUSSO PORTALE RALLENTATO O INVERTITO



DIAGNOSI BCS

EcoDoppler





DIAGNOSI BCS

Ecografia convenzionale

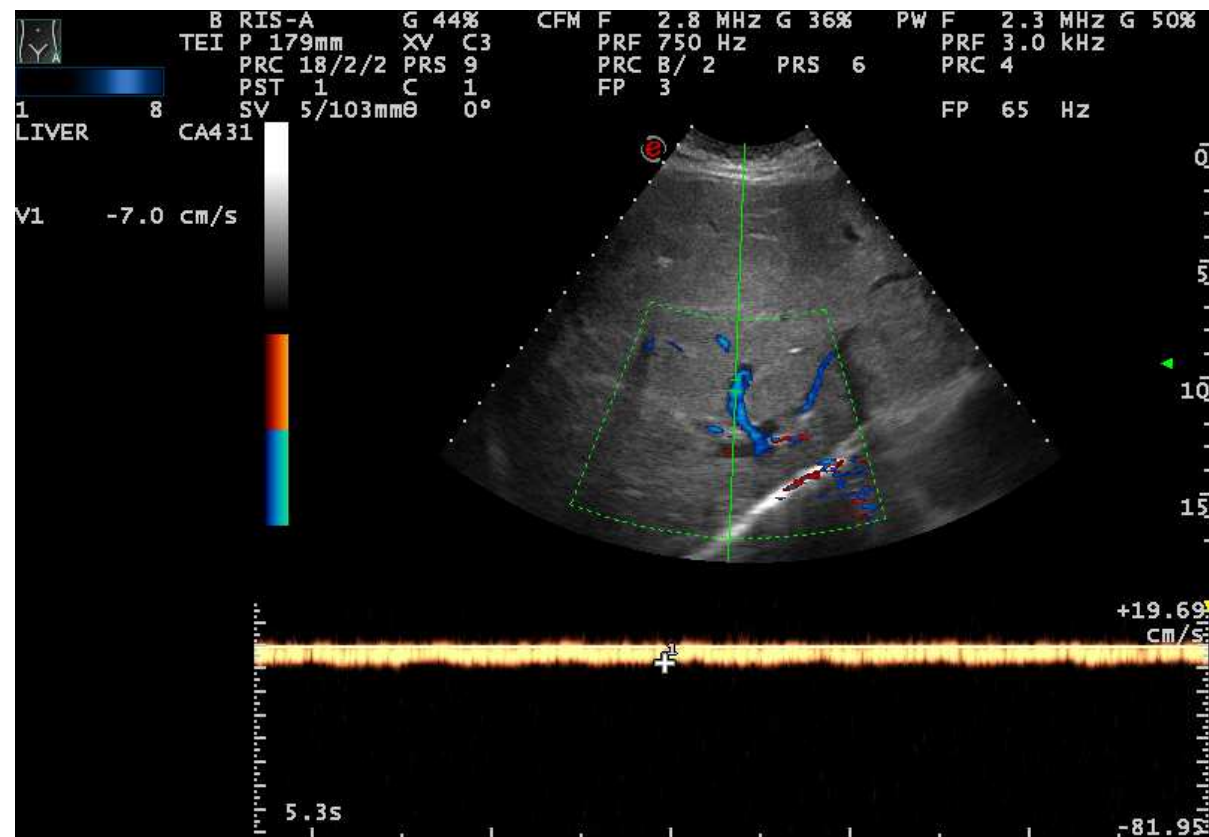
SEGNİ ECOGRAFICI NON SPECIFICI

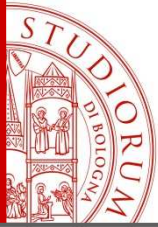
- Noduli di rigenerazione
- Ecostruttura disomogenea da congestione fino alla cirrosi (forme croniche)
- Onda di analisi spettrale monofasica o bifasica (stenosi a valle)
- Trombosi portale (20%)
- Ricanalizzazione della vena ombelicale
- Ascite
- Splenomegalia

**IPERTENSIONE PORTALE
POST-SINUSOIDALE**

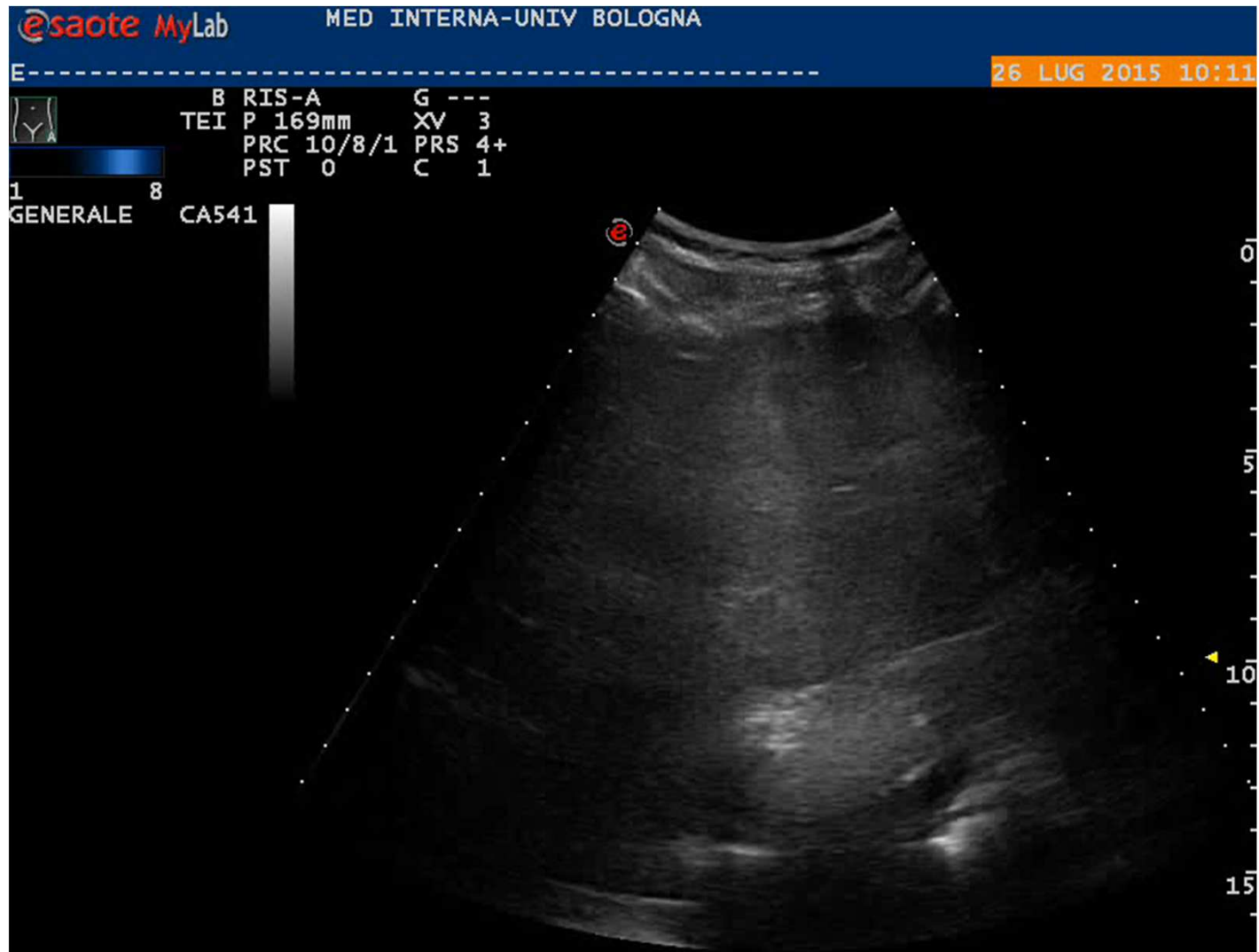


BCS





BCS

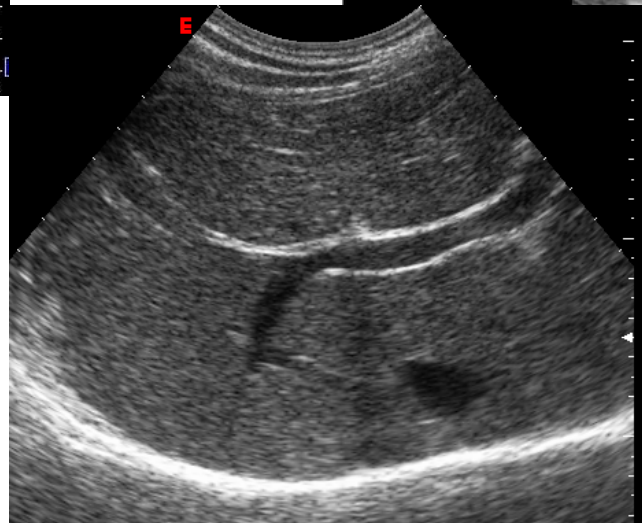
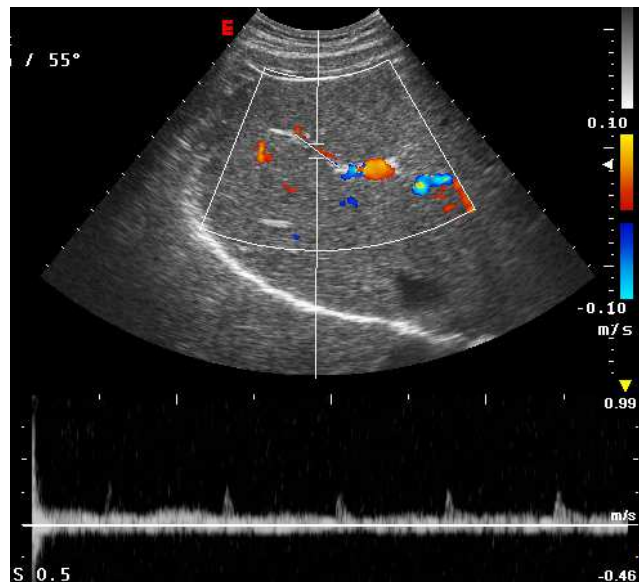




Caso

Eco convenzionale

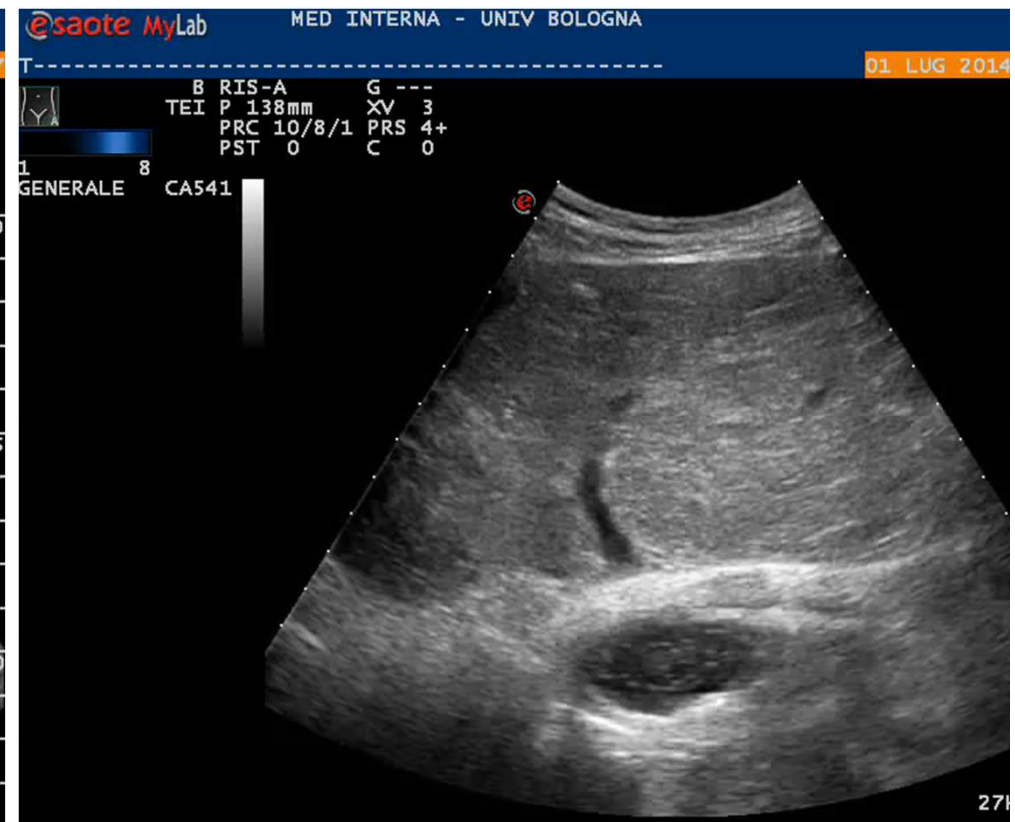
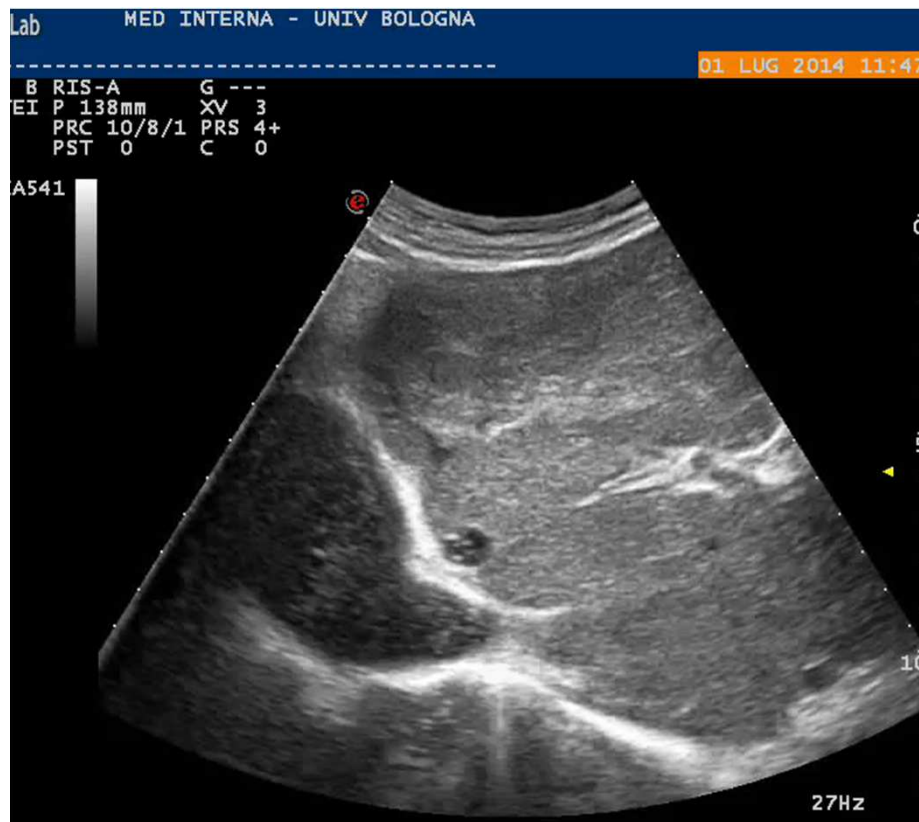
Paziente di 45 aa. Splenomegalia inspiegata. PLT 317.000, Hb 15.9, WBC 72000.

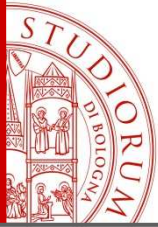


Caso - 1

Eco convenzionale

Paziente di 52 aa. Già ricoverato in altra sede. Diagnosi ecografica di cirrosi, clinicamente definita di eziologia criptogenica. Trattato con RFA per nodulo di 3 cm diagnosticato come HCC sulla base di imaging
CI viene riferito come centro di III livello per HCC

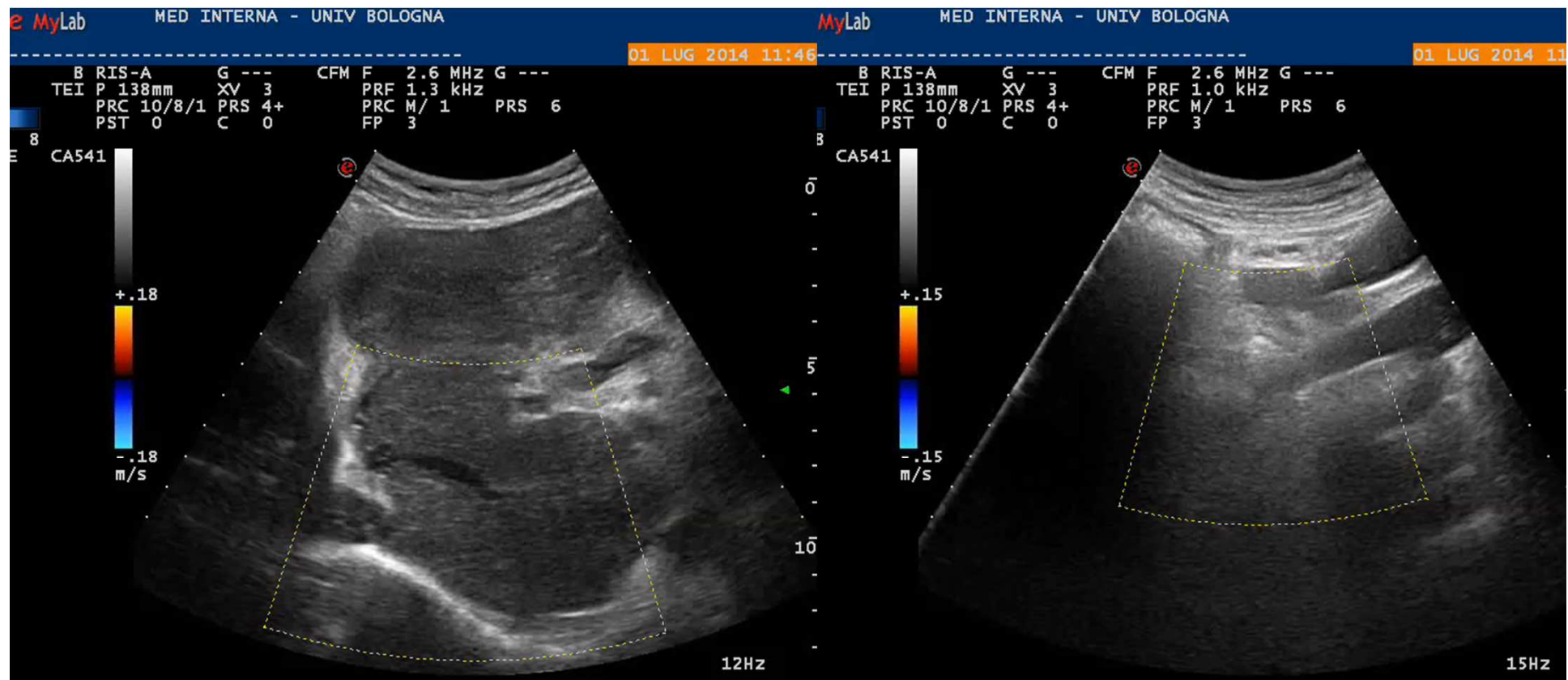




Caso - 1

EcoColorDoppler

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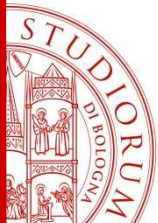


Caso - 2

Eco convenzionale

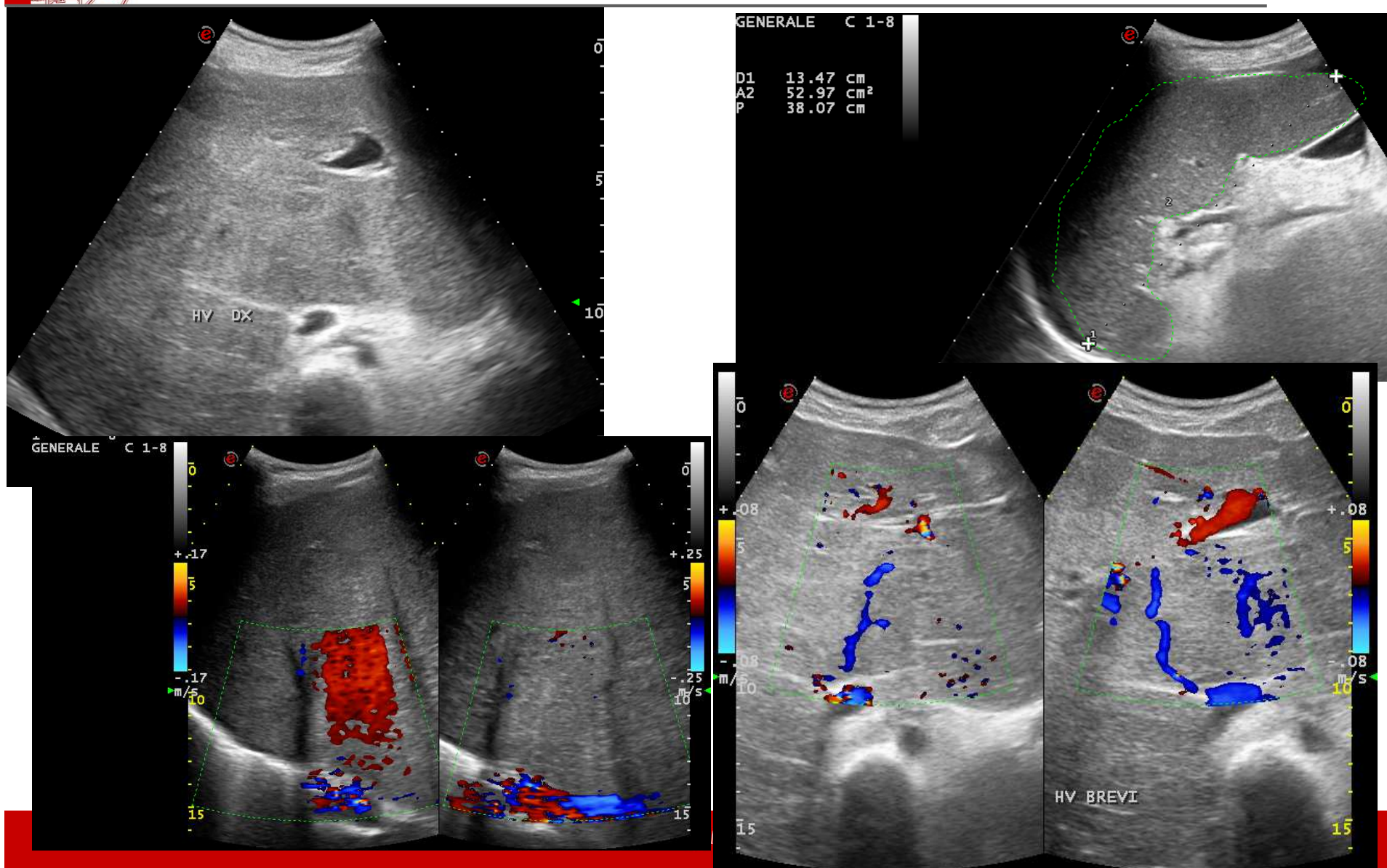
Paziente di 28 aa. Incremento volume addome. Esegue esami: GOT x3, GPT2, gGTx4. PLT 88.000, Hb 17.1gr/dl. WBC 3200. Ascite. Viene ricoverata in altra sede. Fegato disomogeneo. Almeno un nodulo. Sospetta infiltrazione neoplastica diffusa. Esegue biopsia=>negativa per cellulare maligne. Sofferenza pericentrale con sinusoidi dilatati.

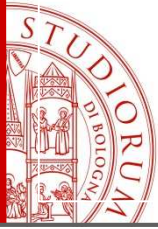




Caso - 2

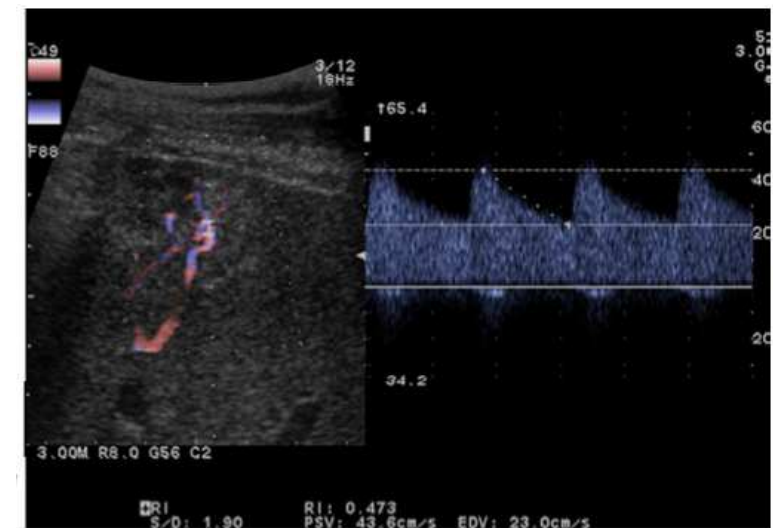
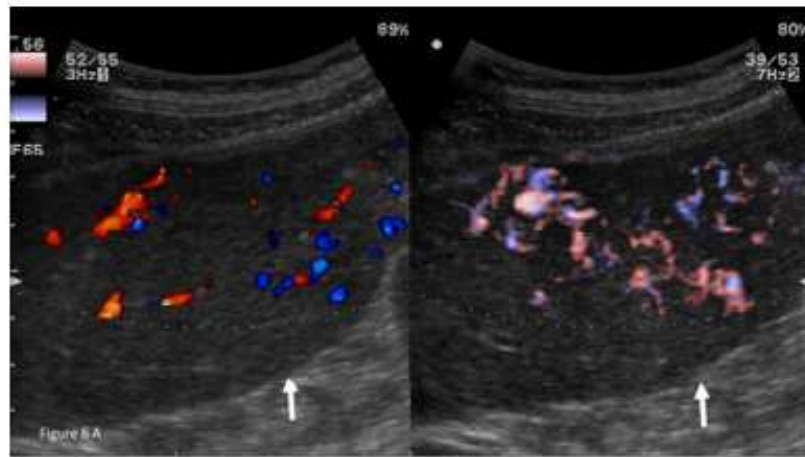
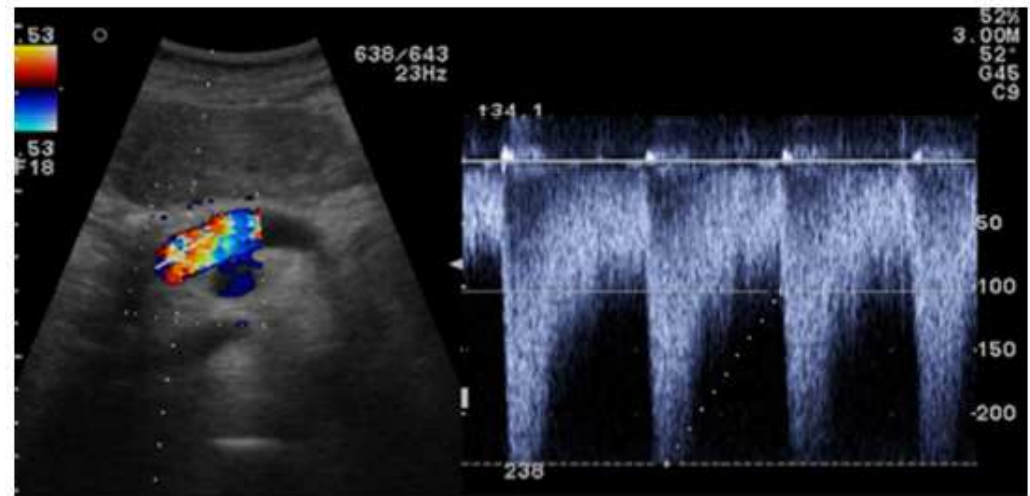
Eco convenzionale





Teleangectasia Emorragica Ereditaria HHT

Rendu Osler Disease

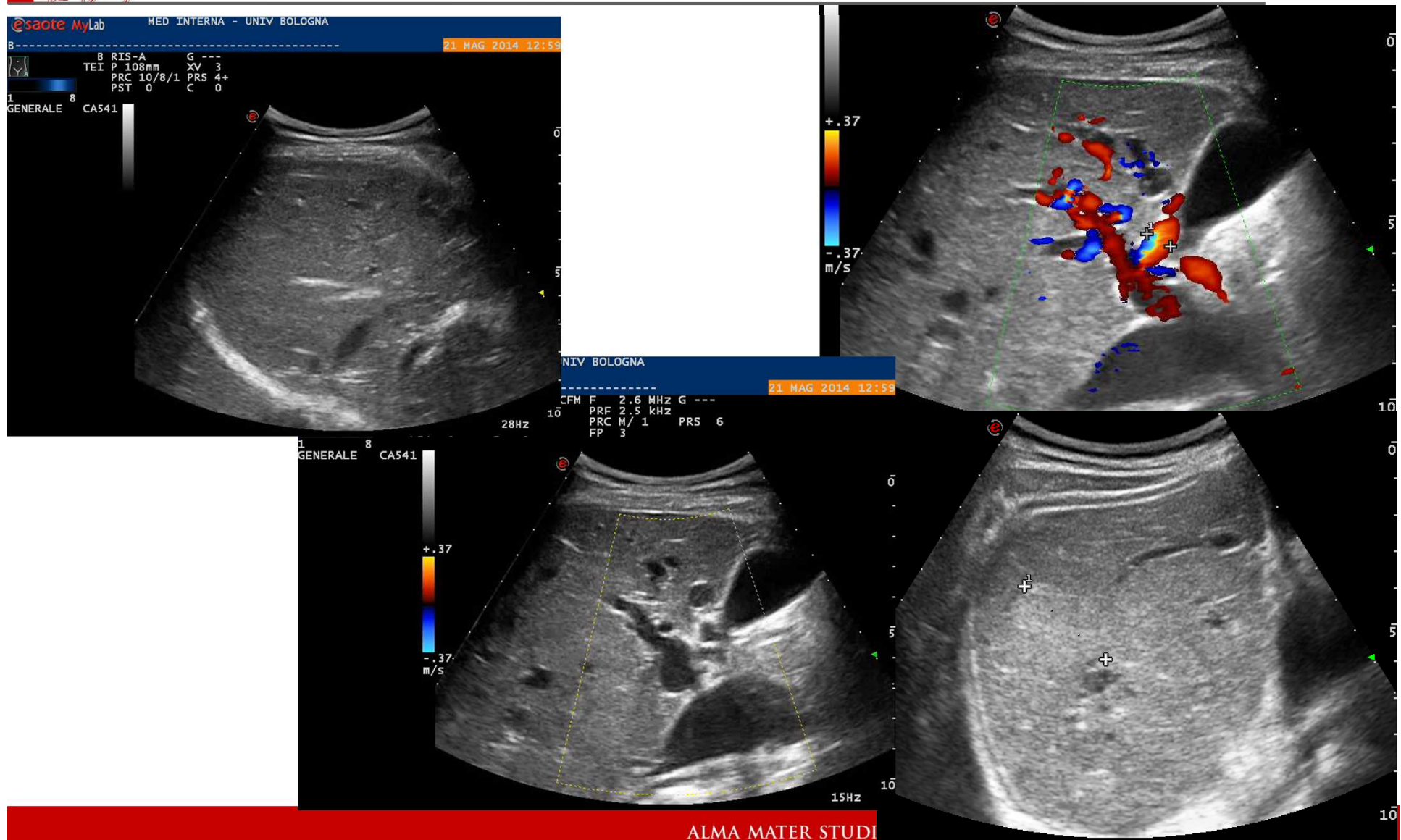


From De Gottardi A et al. *Ultraschall in Med*, 2018 in press



Teleangectasia Emorragica Ereditaria HHT

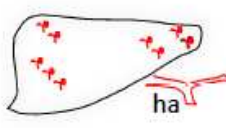
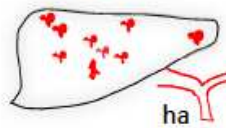
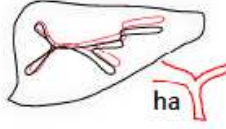
Rendu Osler Disease

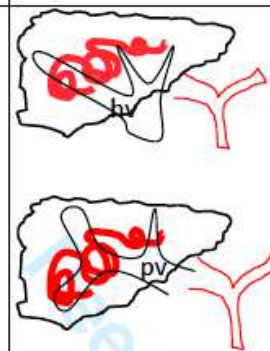


Teleangectasia Emorragica Ereditaria HHT

Rendu Osler Disease

Fig. 9. Doppler US grading of severity of hepatic VMs in HHT (37).

VMs GRADE	SCHEMA	Doppler US findings
0+		- HA diameter $>5 < 6$ mm, and/or - PFV > 80 cm/sec, and/or - RI < 0.55, and/or - Peripheral hepatic hypervascularization
1		- HA dilatation, only extrahepatic > 6 mm, and - PFV > 80 cm/sec, and/or - RI < 0.55
2		- HA dilatation, extra- and intrahepatic ("double channel" aspect) and - PFV > 80 cm/sec - possibly associated with moderate flow abnormality of hepatic and/or portal veins
3		- complex changes in hepatic artery and its branches (tortuous and tangled) with marked flow abnormalities - abnormality of hepatic

		and/or portal vein flow
4		decompensation of arteriovenous shunt with: - dilatation of hepatic and/or portal vein - marked flow abnormalities in both arteries and vein/s

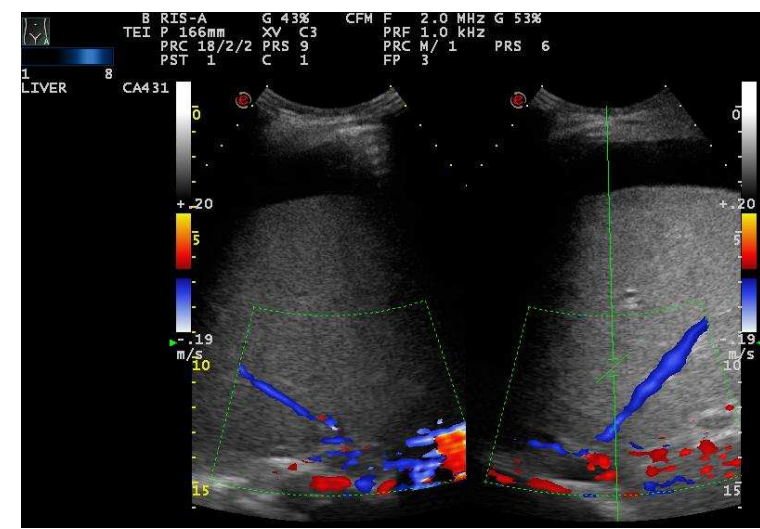
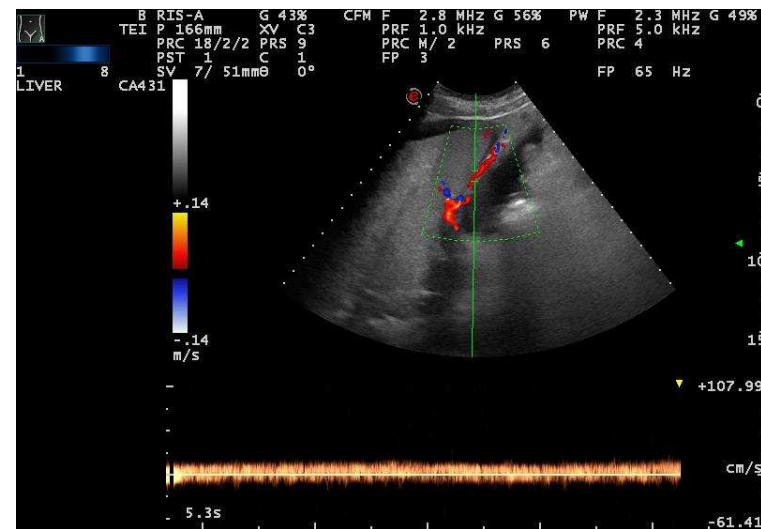
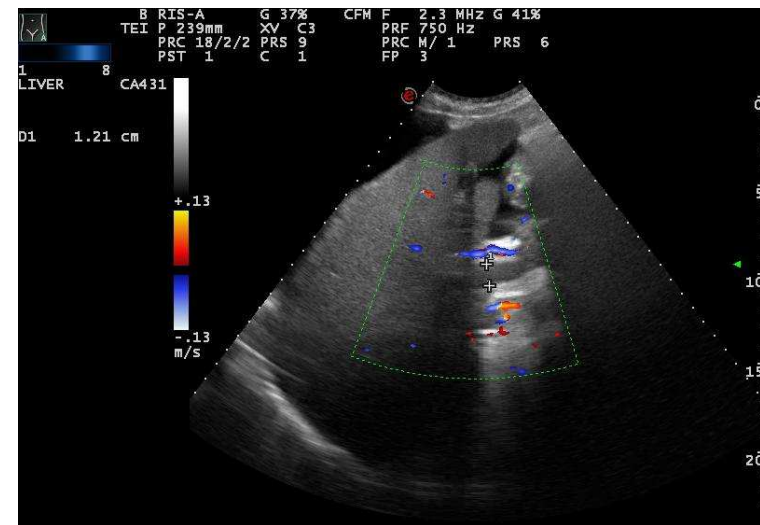
Veno-venous shunts may be found as well and do not necessarily imply a VM up-grading. Nodular transformation of hepatic parenchyma progresses along with liver VMs severity, and it is typically found in grade 4.

HA : hepatic artery, PV: portal vein, HV: hepatic vein

From De Gottardi A et al. *Ultraschall in Med*, 2019 in press

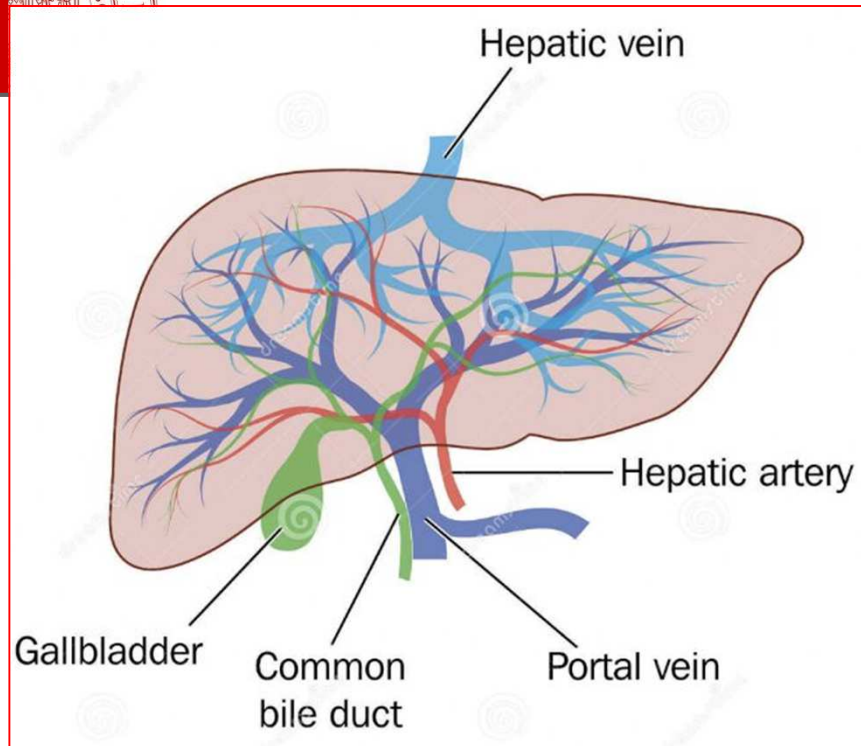


Venocclusive Disease VOD

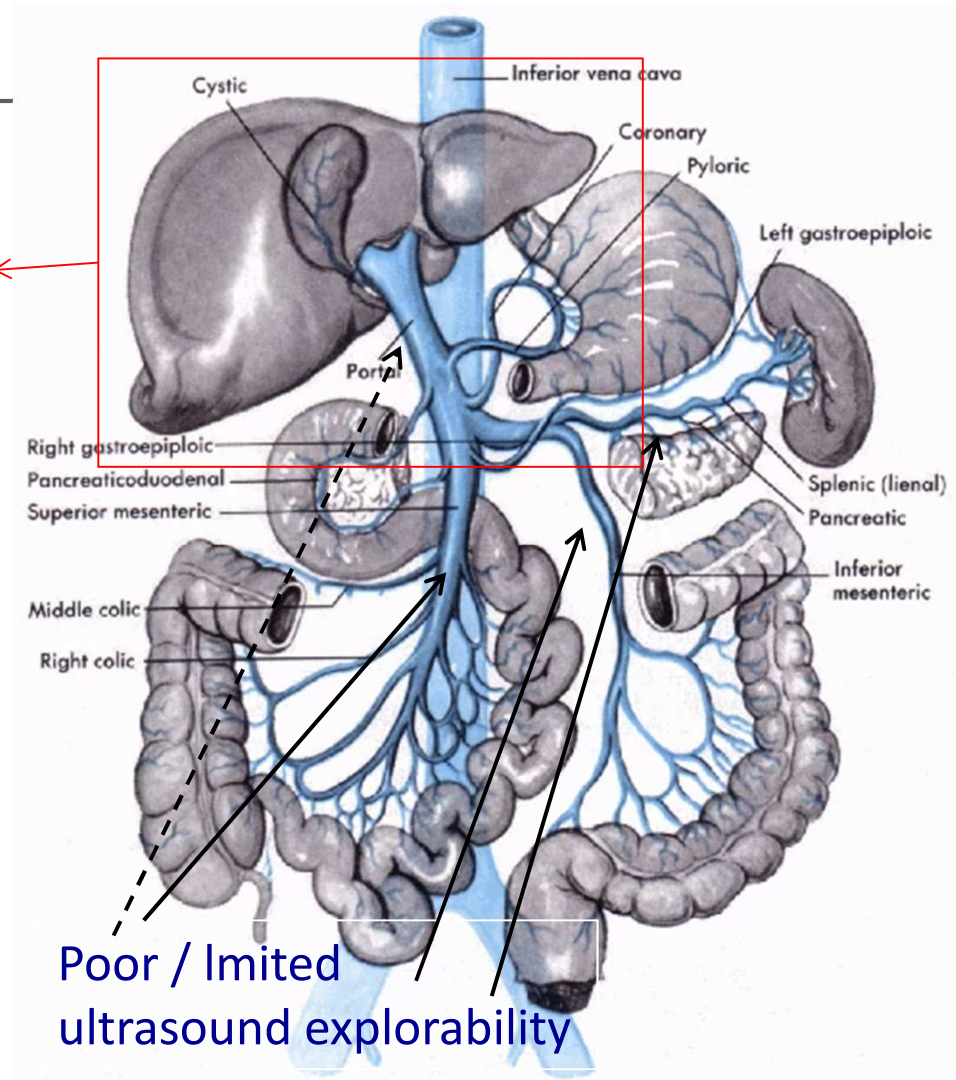




TROMBOSI PORTALE: Splanchnic portal venous system



Portal tracts most commonly well explorable by ultrasound



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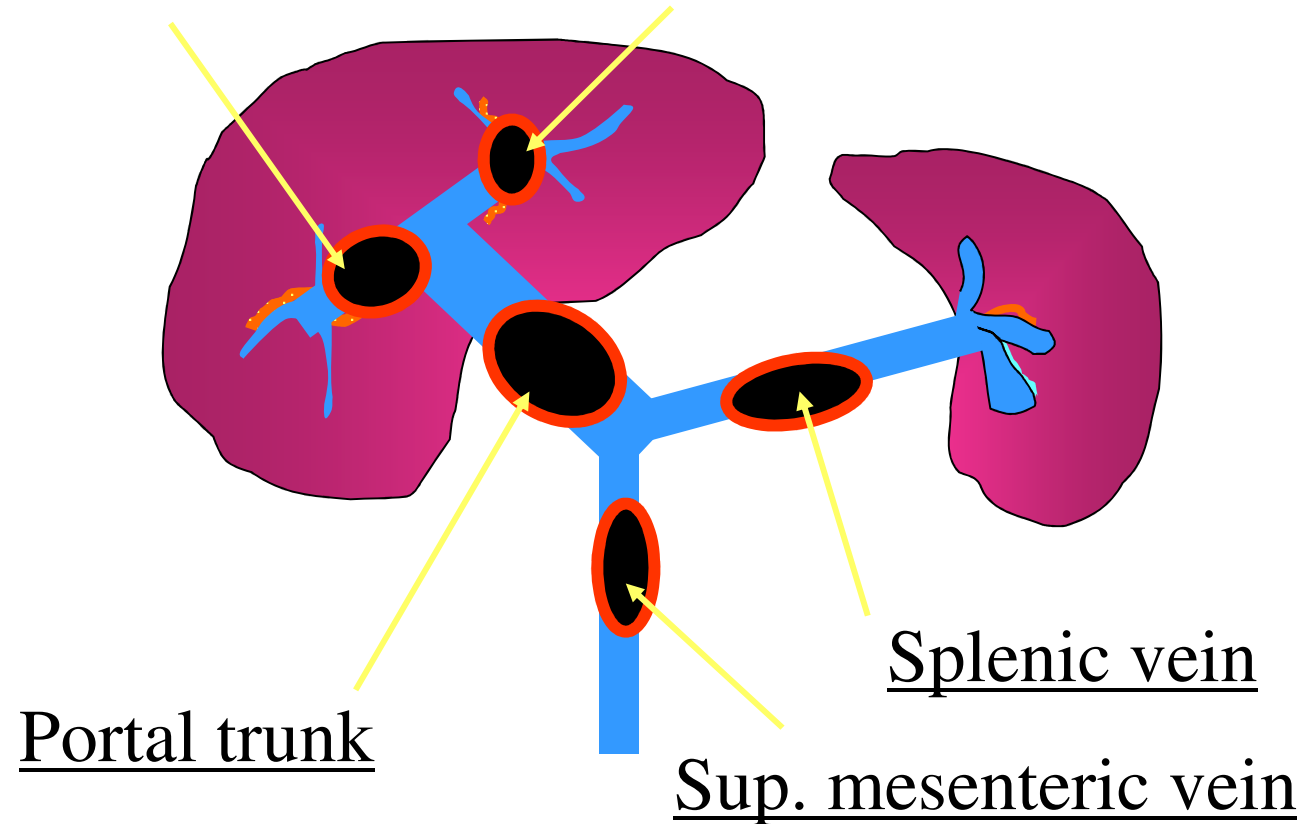
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PRE-HEPATIC PORTAL HYPERTENSION POSSIBLE SITES OF PORTAL THROMBOSIS

Main intrahepatic portal branches





DETECTION OF PORTAL VEIN THROMBOSIS

- Ultrasound (gray-scale and Doppler imaging) is accurate to diagnose and exclude PVT (at least in the intrahepatic branches, splenic vein and distal portal trunk) and should be used as the first-line imaging method (sensitivity and specificity >90%) apart from rapid onset of symptoms.





DETECTION OF PORTAL VEIN THROMBOSIS

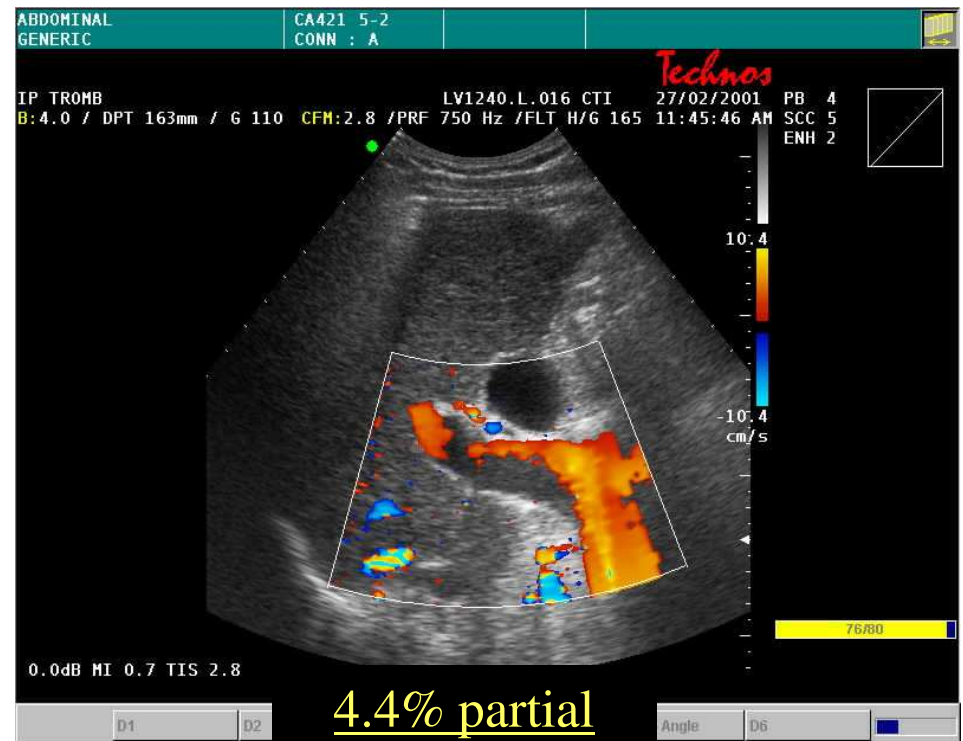
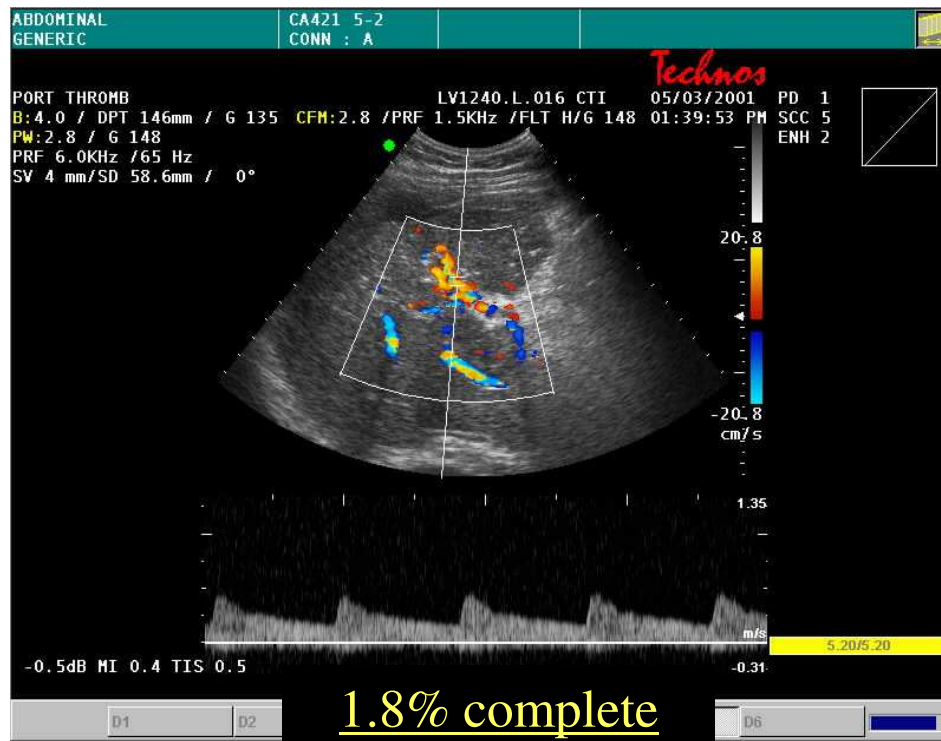
- PVT appears as echogenic material within the PV lumen, occupying it either partially or completely (distinction suboptimal by baseline US)
- The involved portal tract can appear dilated at the site of a complete and recent occlusion (mainly in the case of tumor thrombus).
- The echogenicity of the clot is variable: hypoechoic (can be missed!), iso- or hyperechoic. A hypoechoic pattern is more commonly observed in recent PVT, but a precise dating of the thrombus is unreliable.
- Color Doppler and spectral Doppler US improve sensitivity and accuracy of baseline grayscale US as they differentiate complete occlusive thrombosis (complete lack of flow signals) from parietal incomplete thrombosis. These techniques are usually used in combination



PORTAL THROMBOSIS

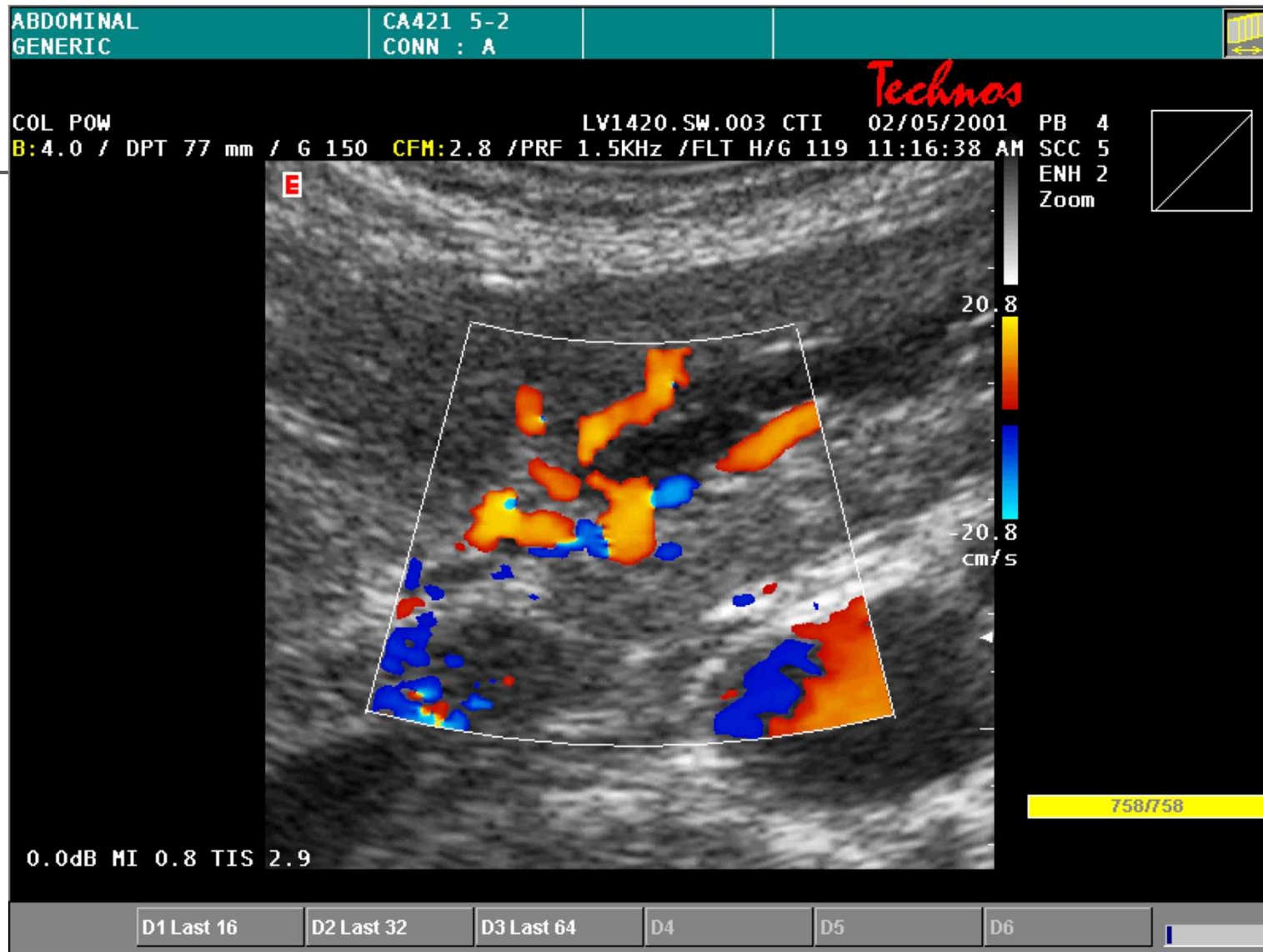
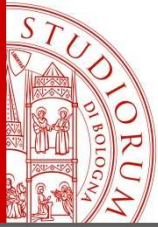
patognomonic for portal hypertension

- echoic material within the lumen, dilated in early stages, but thinned in late phase + possible cavernomatous transformation



Prevalence of portal trunk thrombosis
in cirrhosis without HCC

Gaiani, Gastroenterology 1991



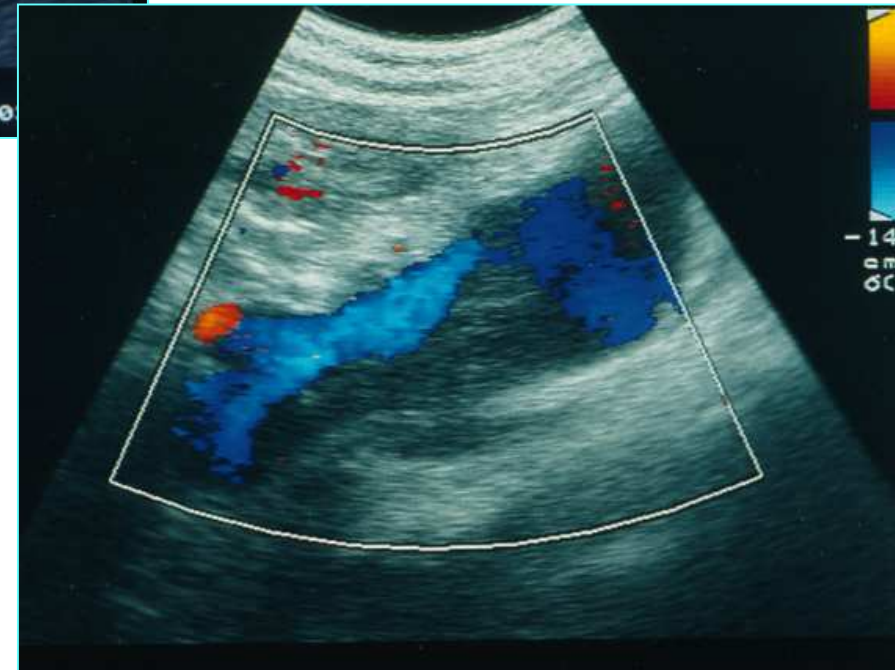
SEGMENTARY THROMBOSIS: LEFT PORTAL BRANCH



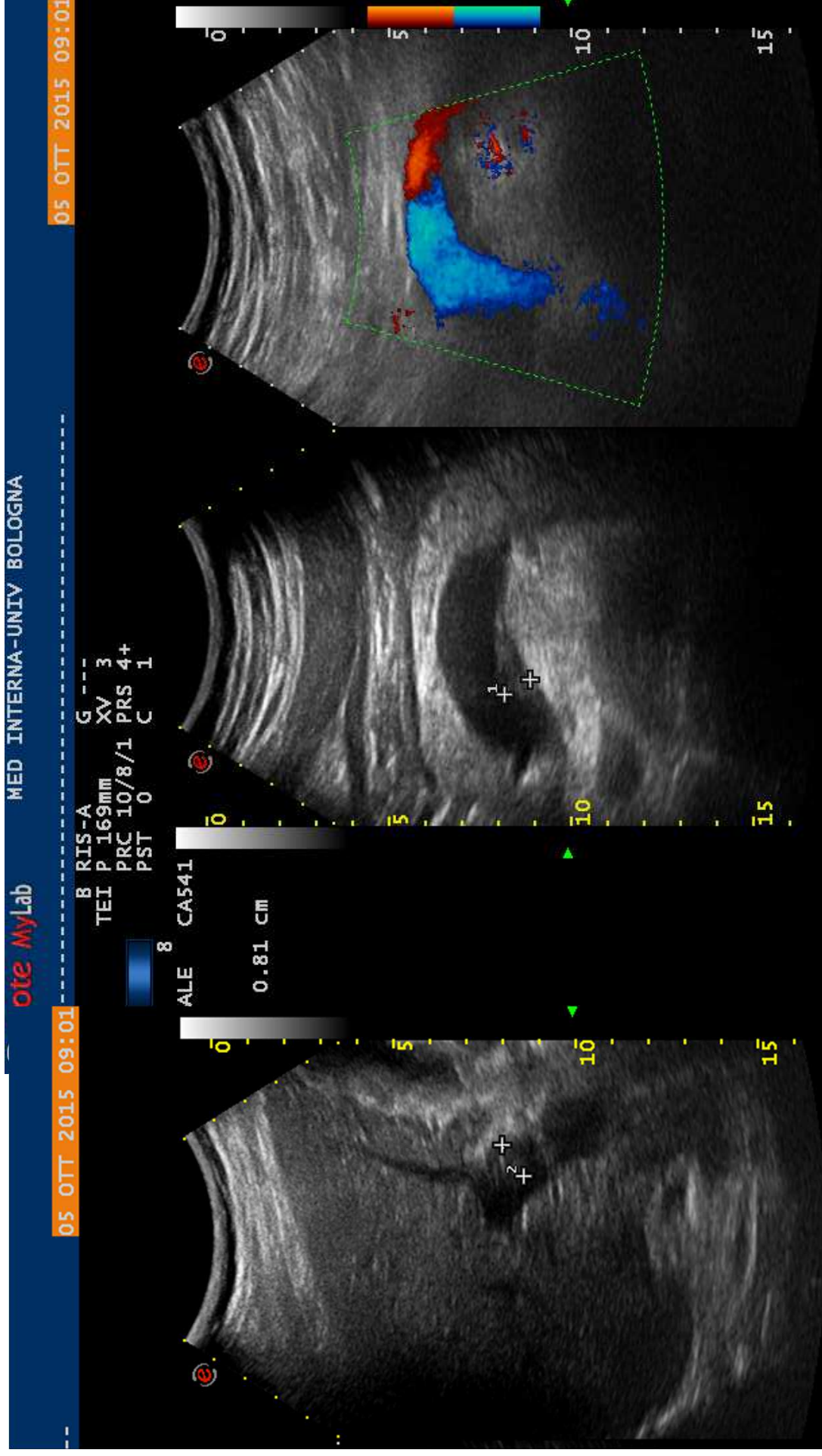
WALL THROMBOSIS



Right branch



Portal trunk

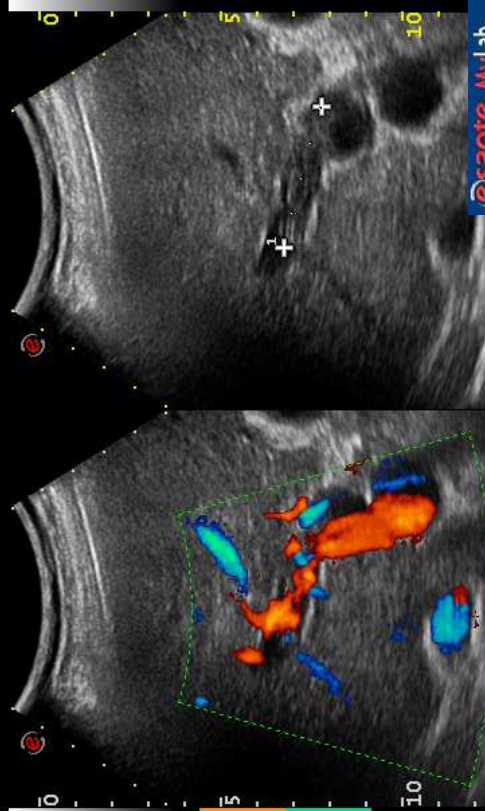


05 OTT 2015 09:03

B RIS-A G 448
TEI P 169mm XV 3
PRC 10/8/1 PRS 4+
PST 0 C 1

1 GENERALE 8 CA541

D1 4.02 cm



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esaoite MyLab

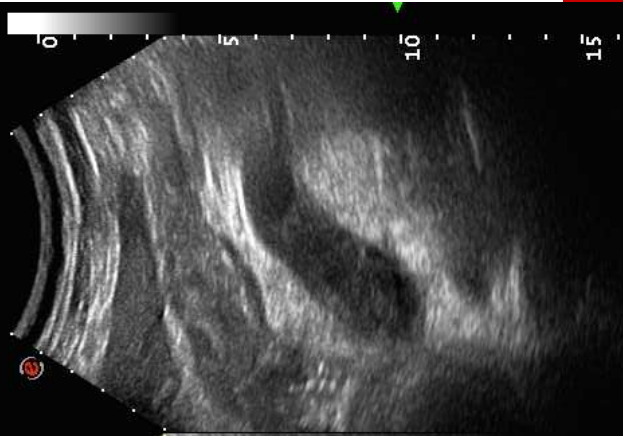
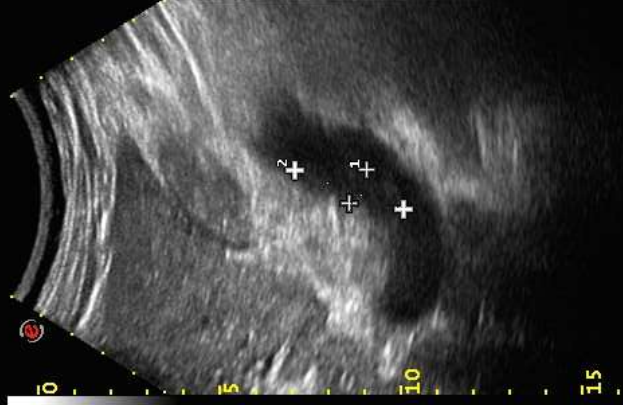
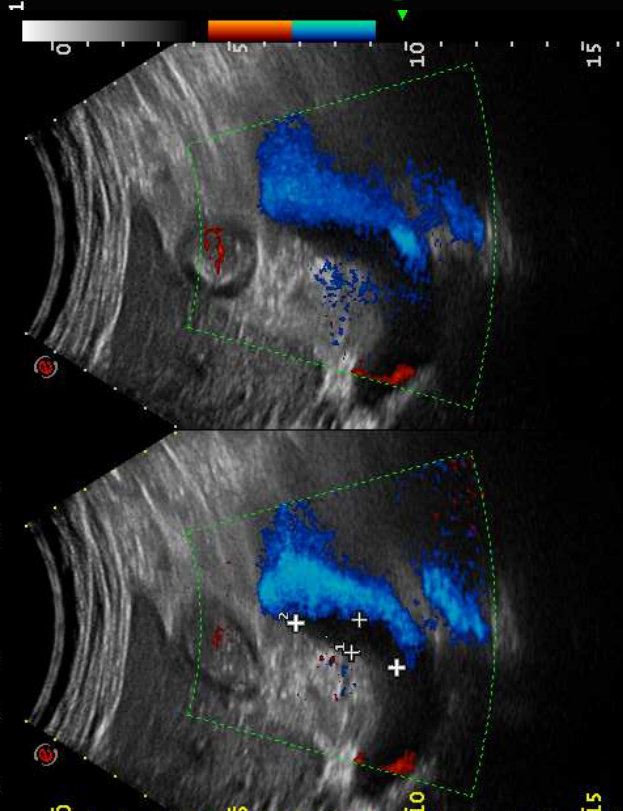
05 OTT 2015 09:04

B RIS-A G ---
TEI P 169mm XV 3
PRC 10/8/1 PRS 4+
PST 0 C 1

1 GENERALE 8 CA541

D1 0.95 cm
D2 3.11 cm

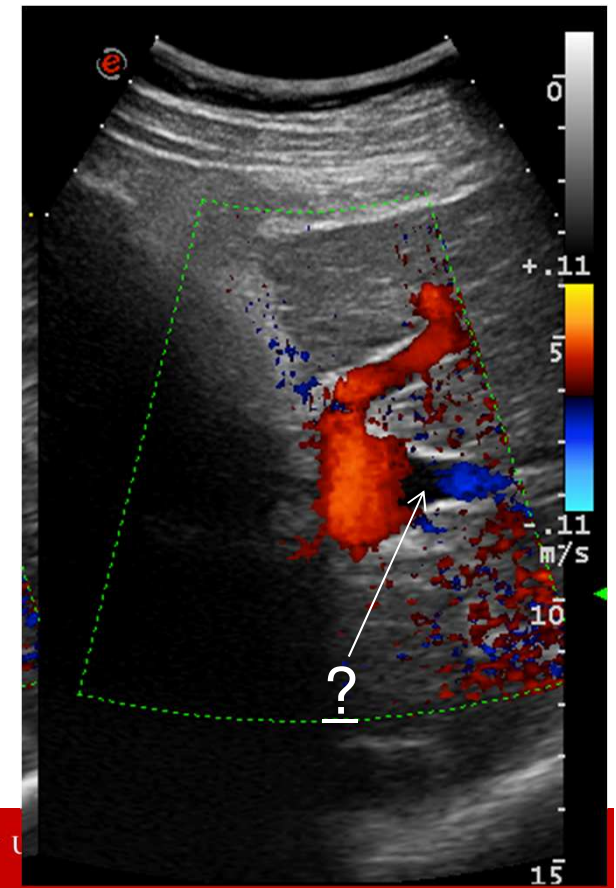
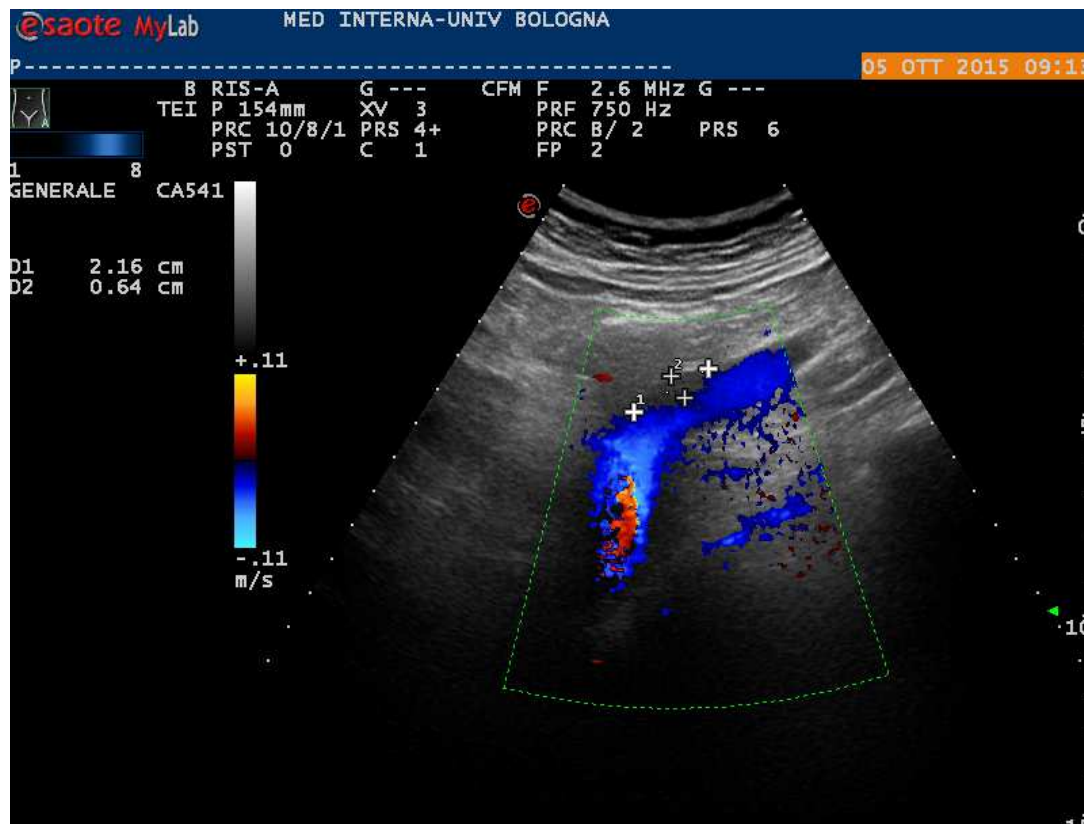
B RIS-A G --- CFM F 2.6 MHz G ---
TEI P 169mm XV 3 PRF 1.0 kHz
PRC 10/8/1 PRS 4+ PRC B/2 PRS 6
PST 0 C 1 FP 3

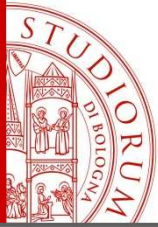




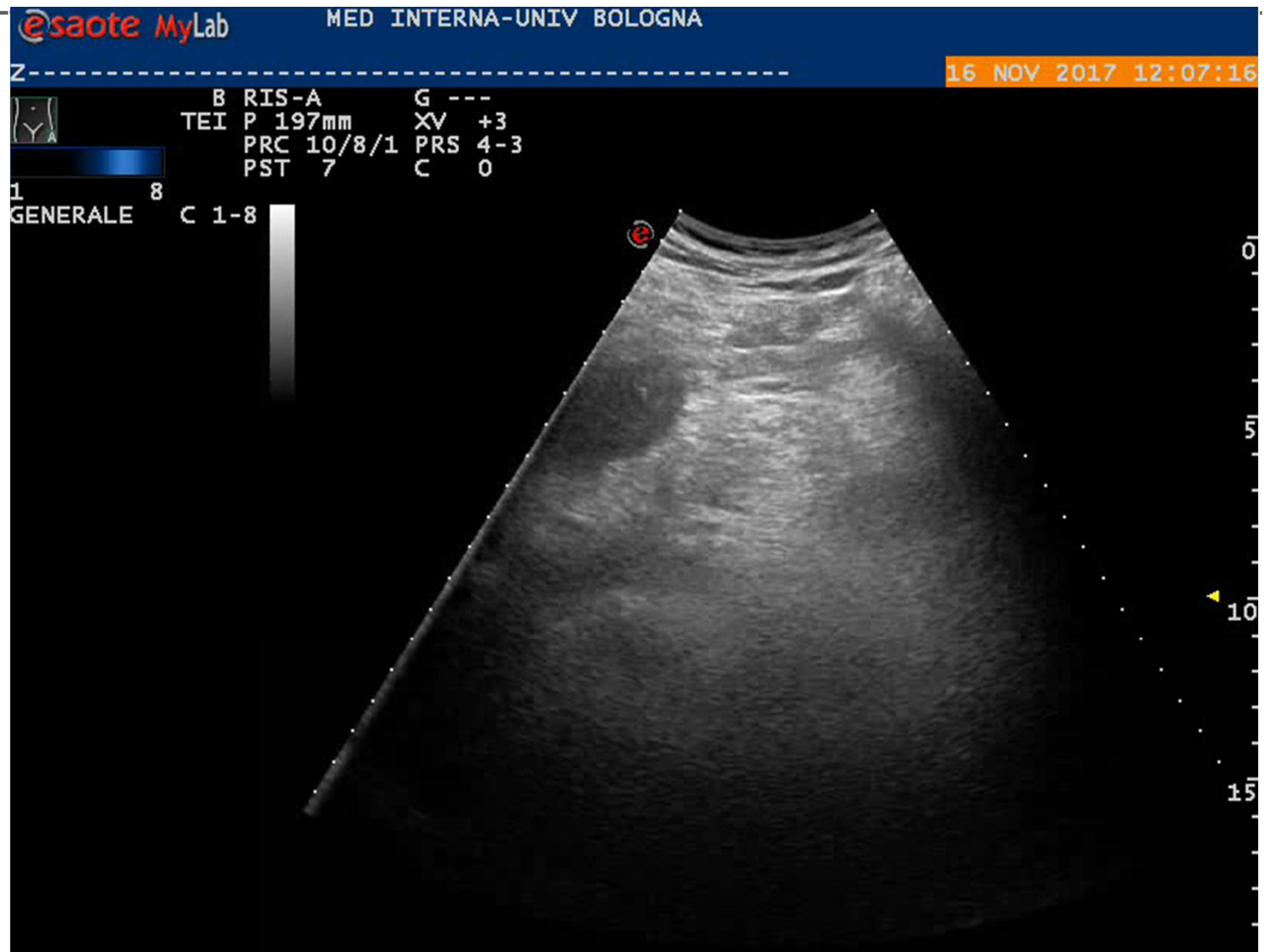
How to increase the sensitivity of Doppler to low flows for the diagnosis of PVT

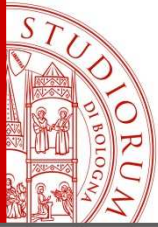
- Lower the Pulse Repetition Frequency PRF (up to aliasing)
- Increase gain (up to disturbing noise)
- Narrowing insonation angle
- Try with Power Doppler
- Inject contrast agents during colorDoppler (slow continuous infusion, not bolus injection to avoid blooming effect)



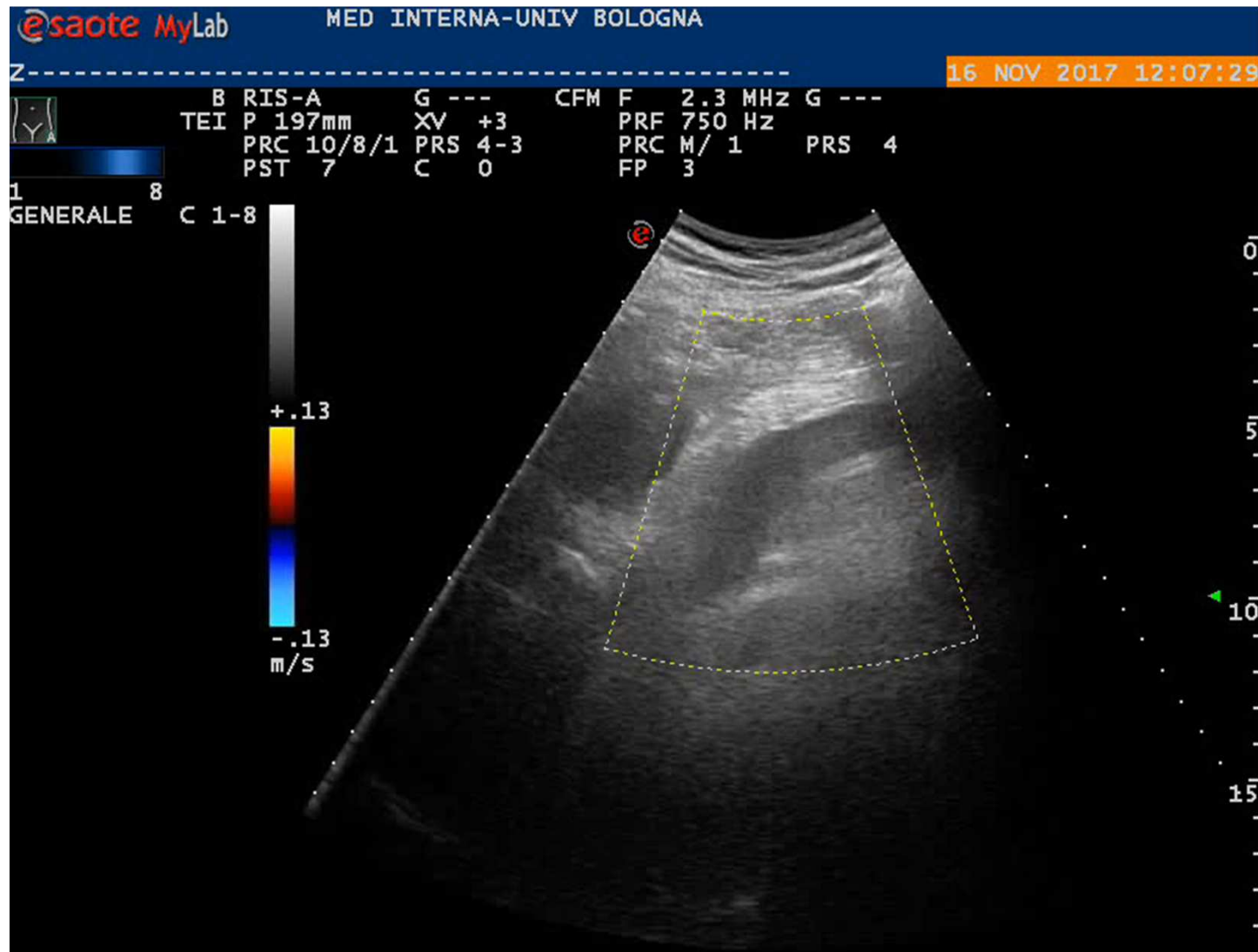


PARTIAL PORTAL VEIN THROMBOSIS: BASELINE GRAY SCALE US

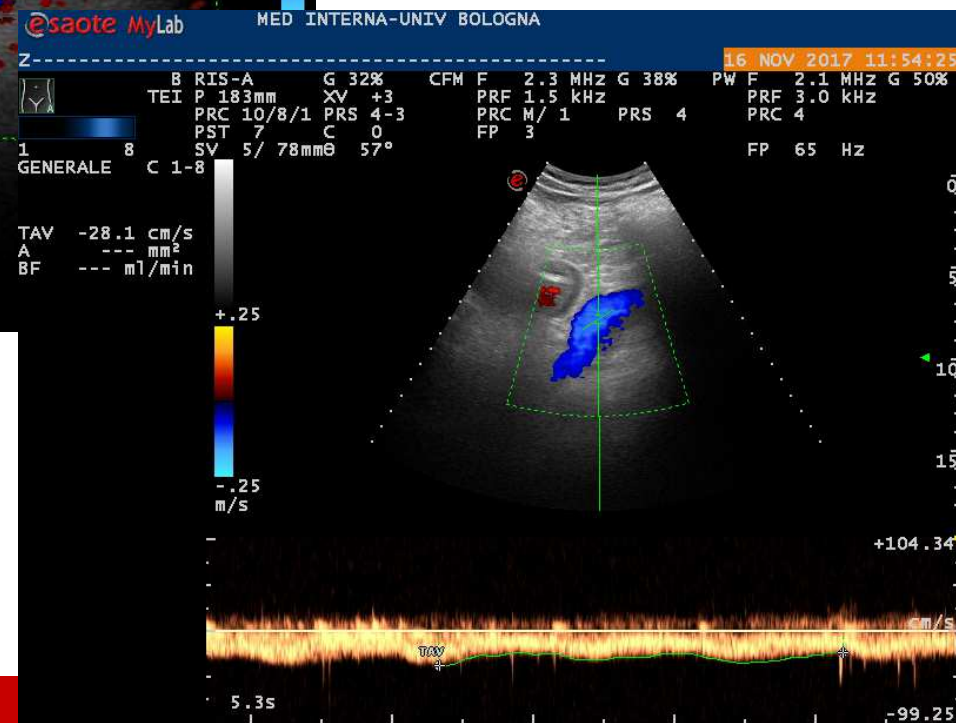
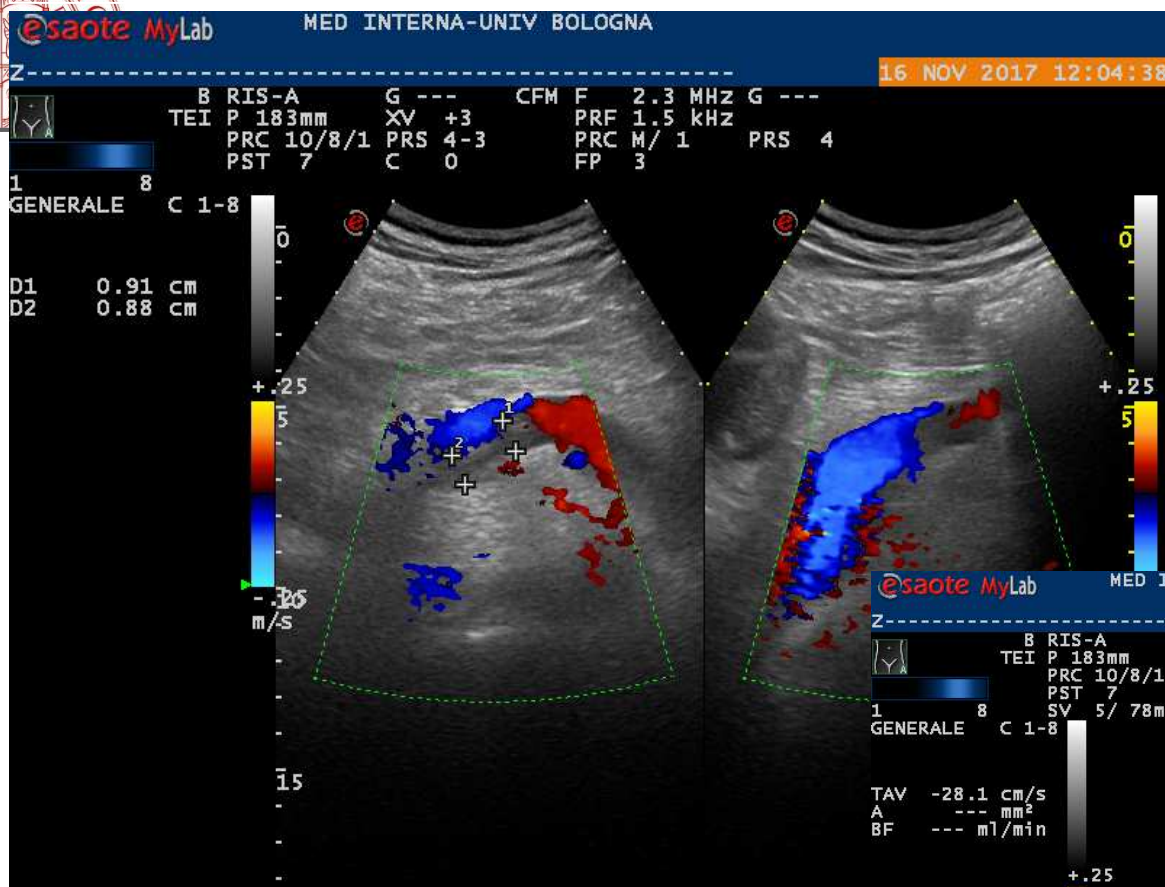




PARTIAL PORTAL VEIN THROMBOSIS: COLOR DOPPLER

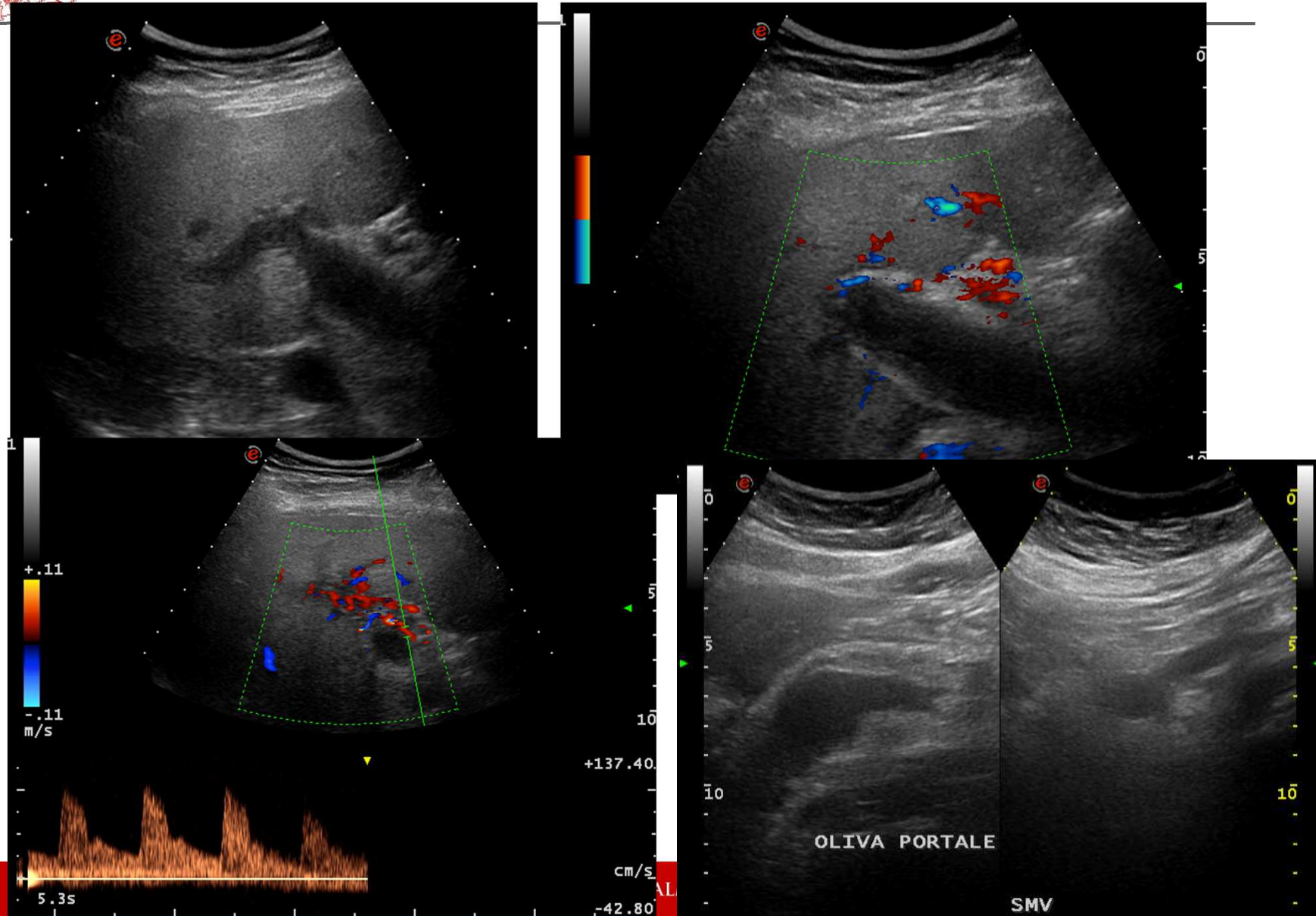


PARTIAL PORTAL VEIN THROMBOSIS: COLOR



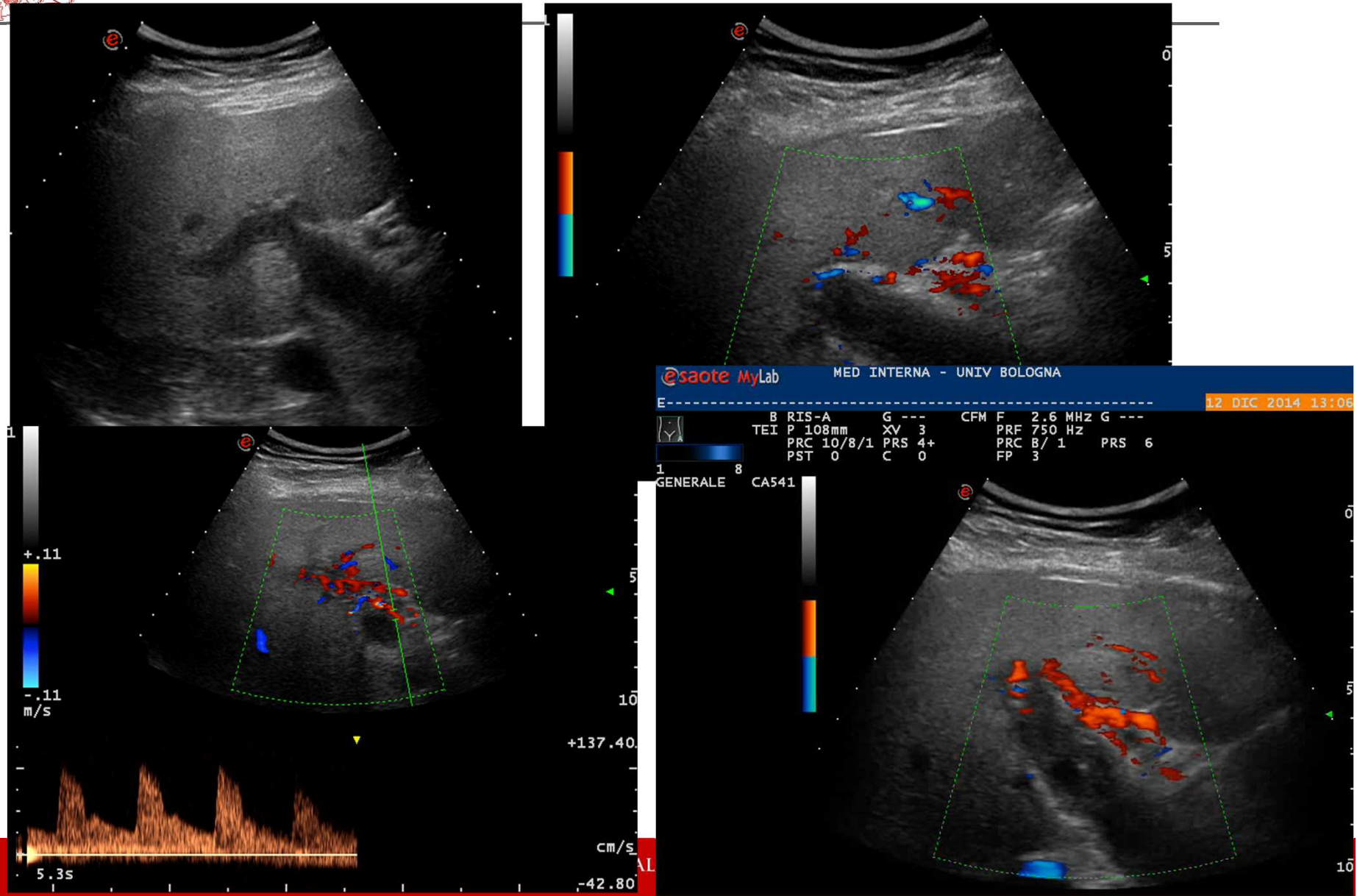


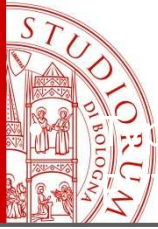
SYMPTOMATIC RECENT PORTAL VEIN THROMBOSIS CAN BE POORLY ECHOIC AT BASELINE GRAY SCALE US





SYMPTOMATIC RECENT PORTAL VEIN THROMBOSIS CAN BE POORLY ECHOIC AT BASELINE GRAY SCALE US => higher gain





Bland PVT appearance at CT/MRI

Before contrast



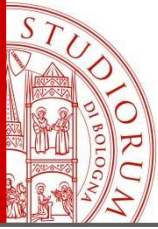
Arterial phase



Portal phase



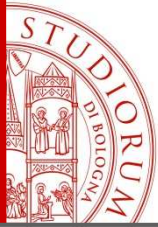
CT/MRI required to accurately assess extension of PVT



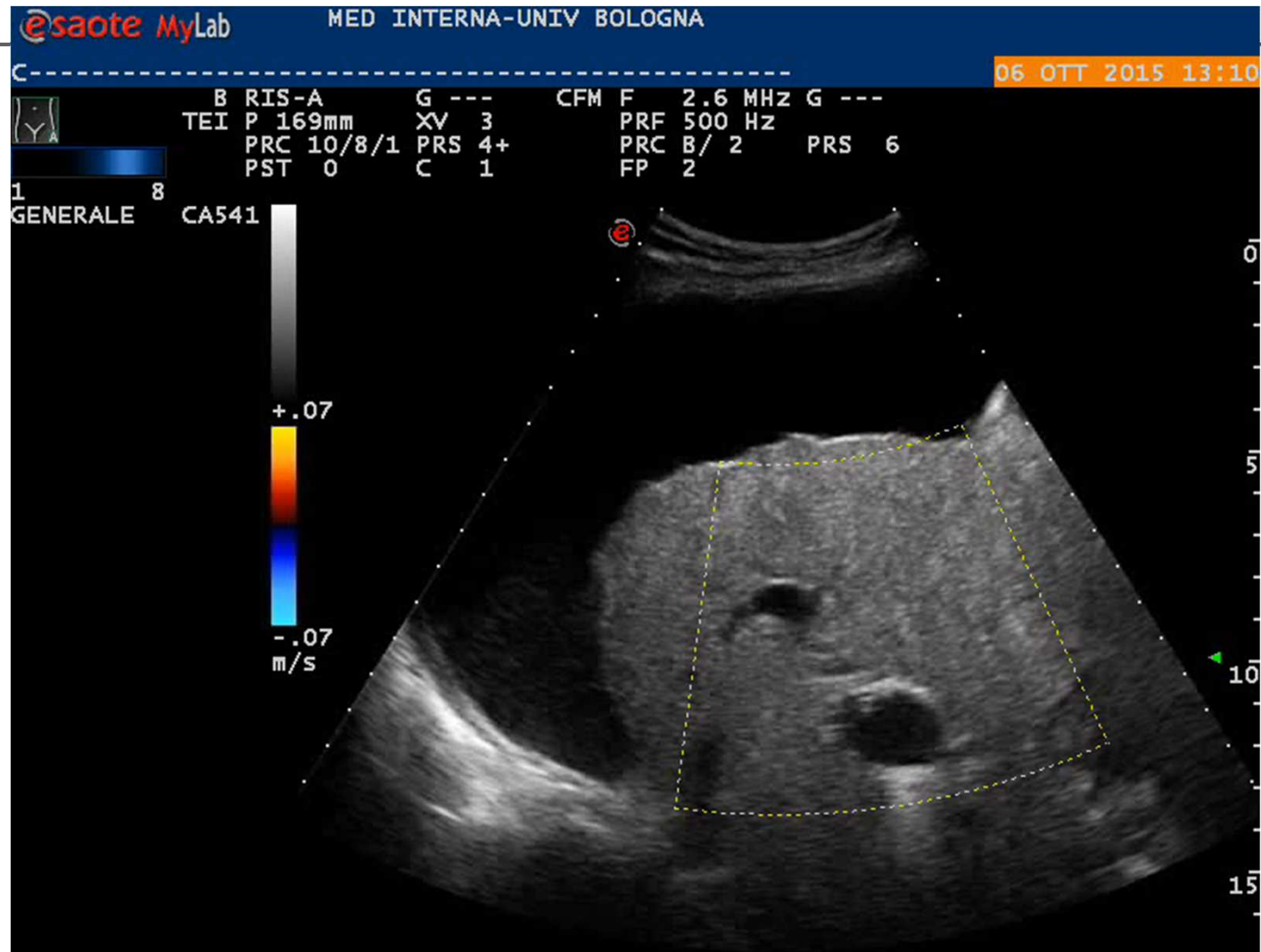
DETECTION OF PORTAL VEIN THROMBOSIS

- CEUS maximize the diagnostic accuracy of ultrasound to finely detect the presence and extension of the thrombus (US contrast agents can also enhance Doppler US)



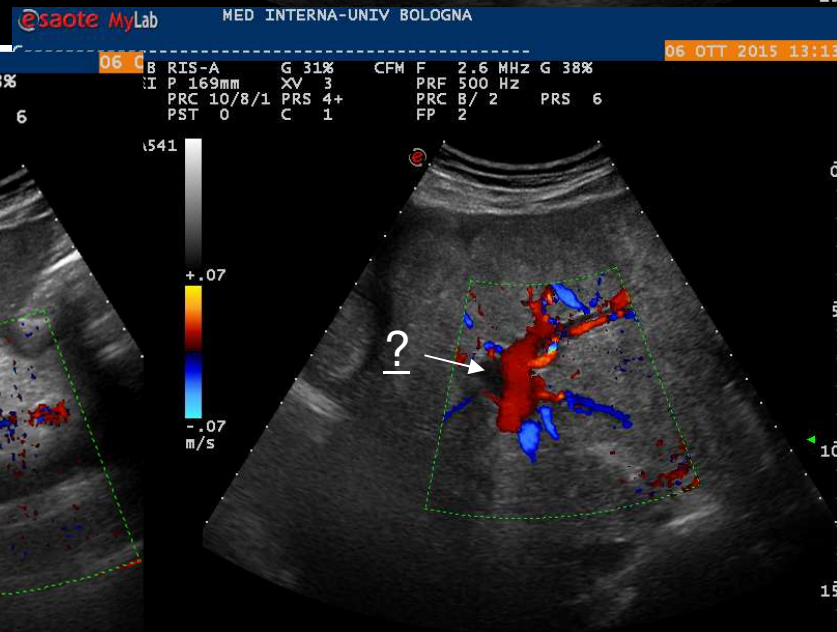
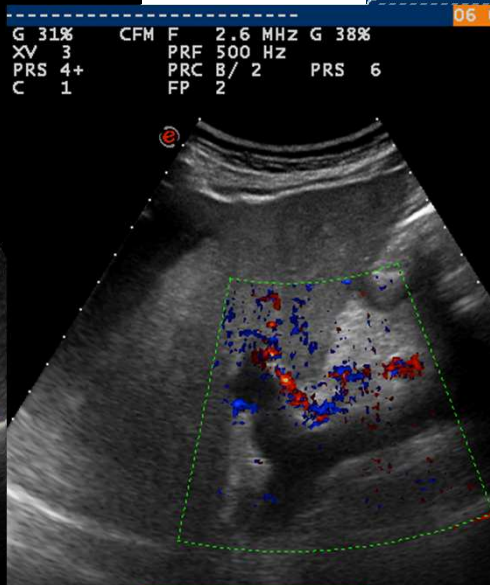
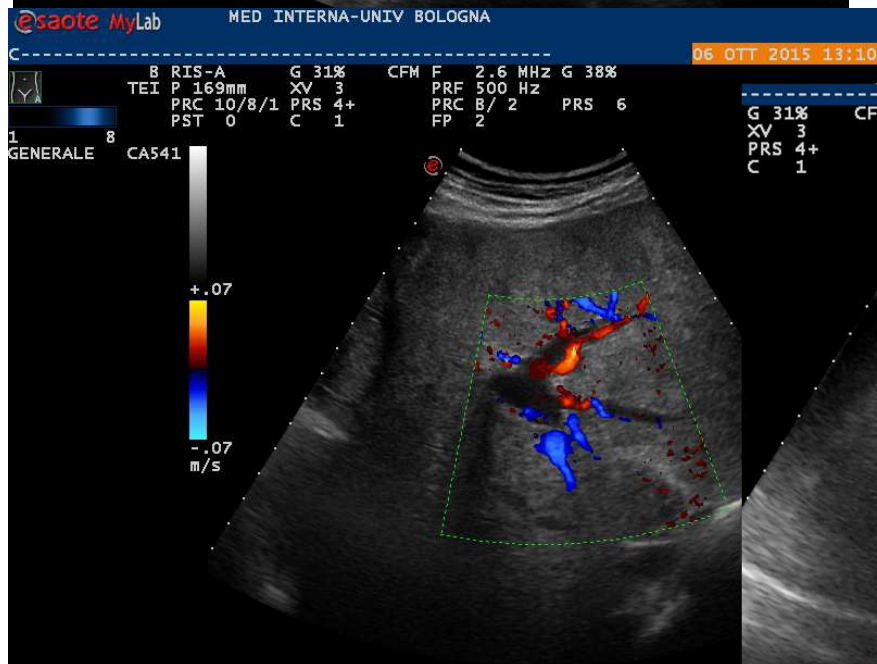
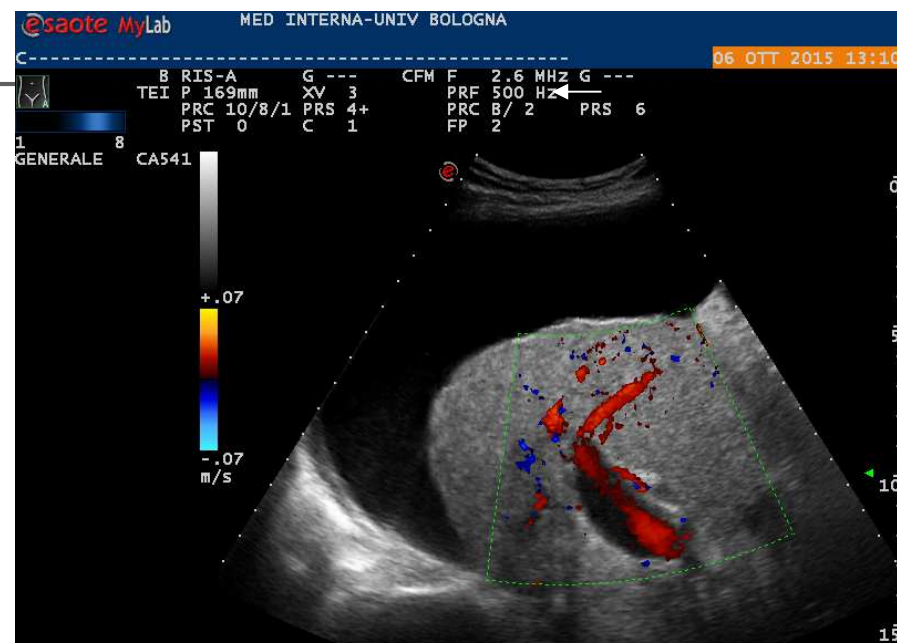
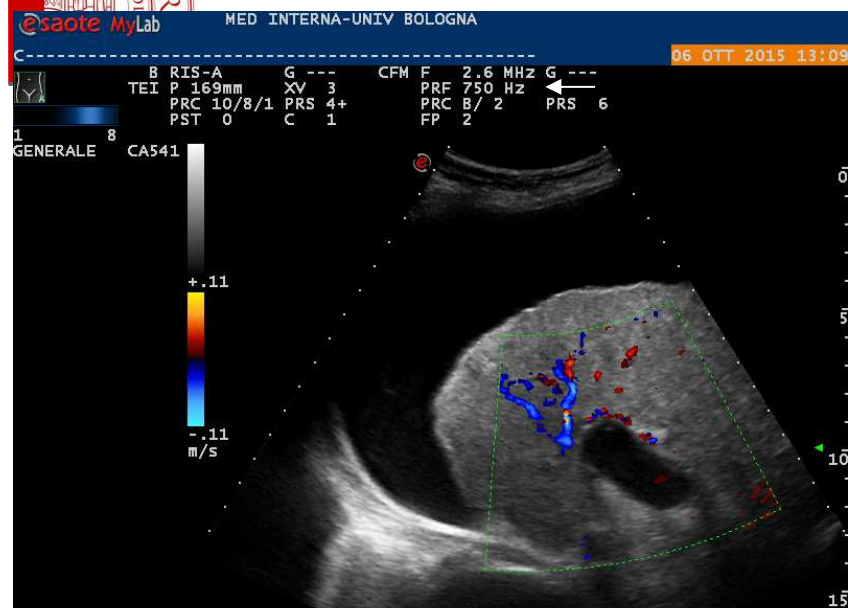


Is it there partial PVT or only slow flow?

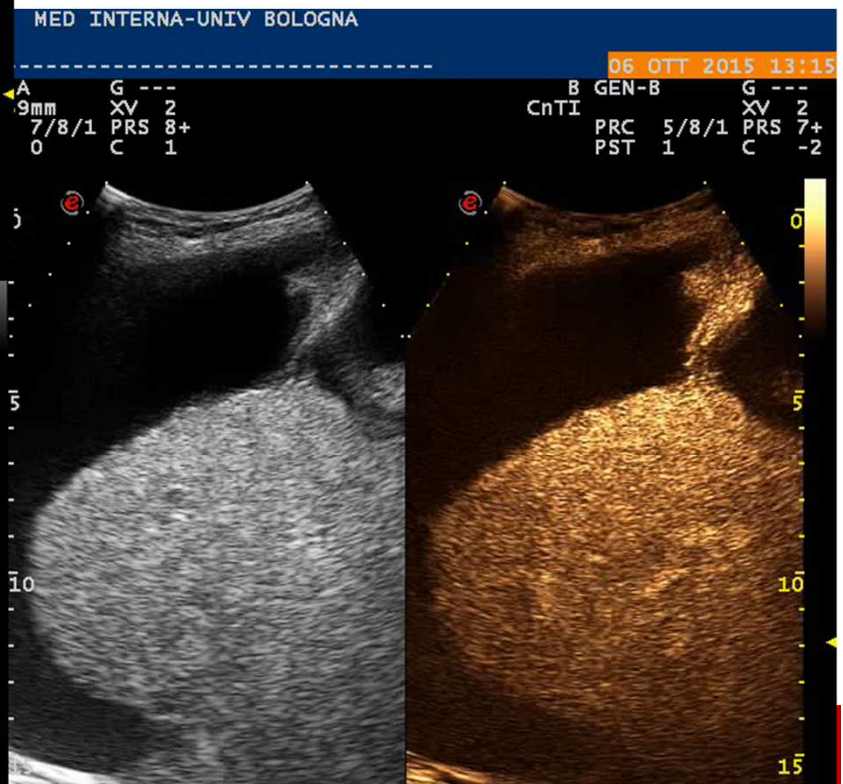
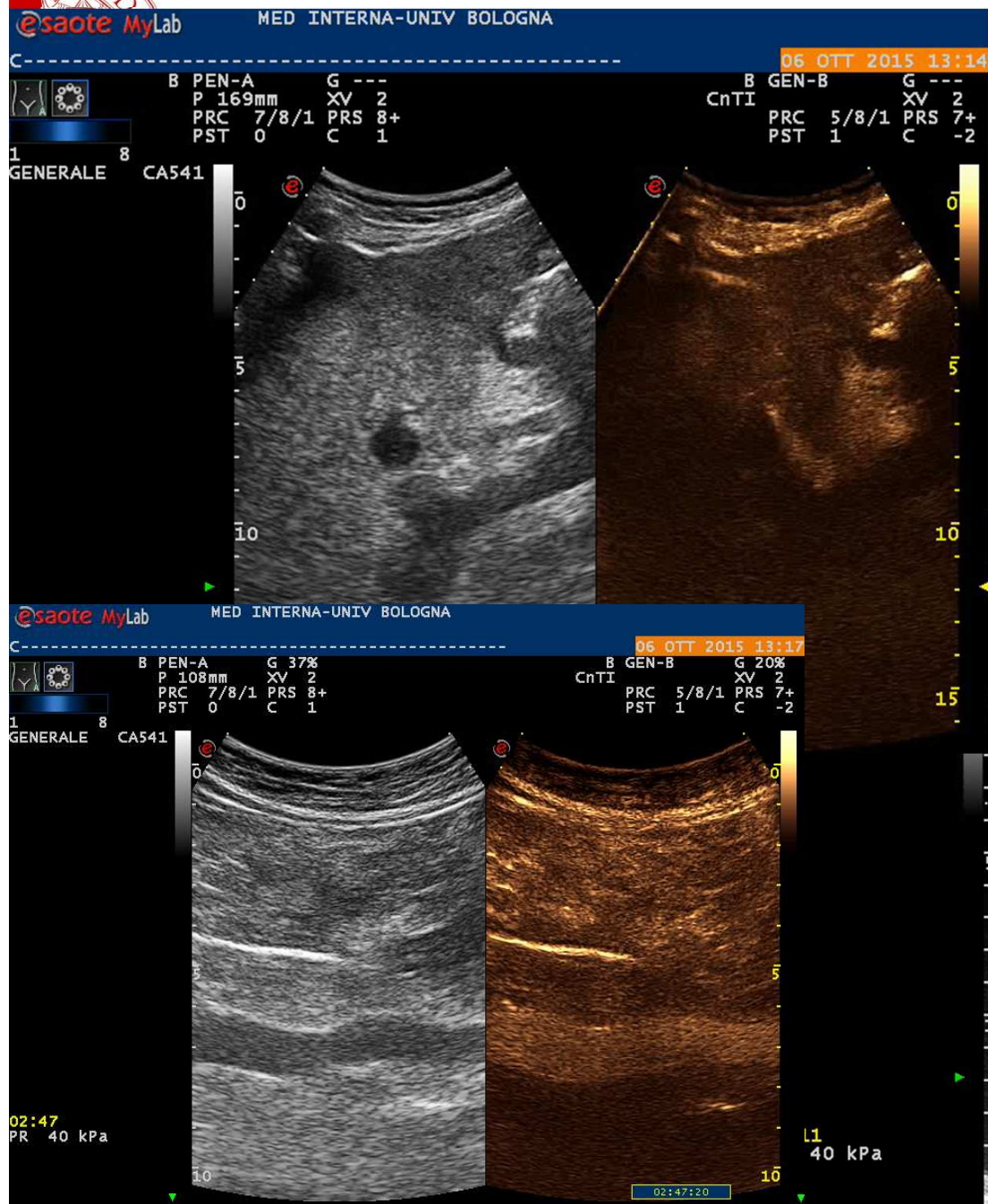


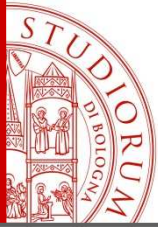


Is it there partial PVT or only slow flow?

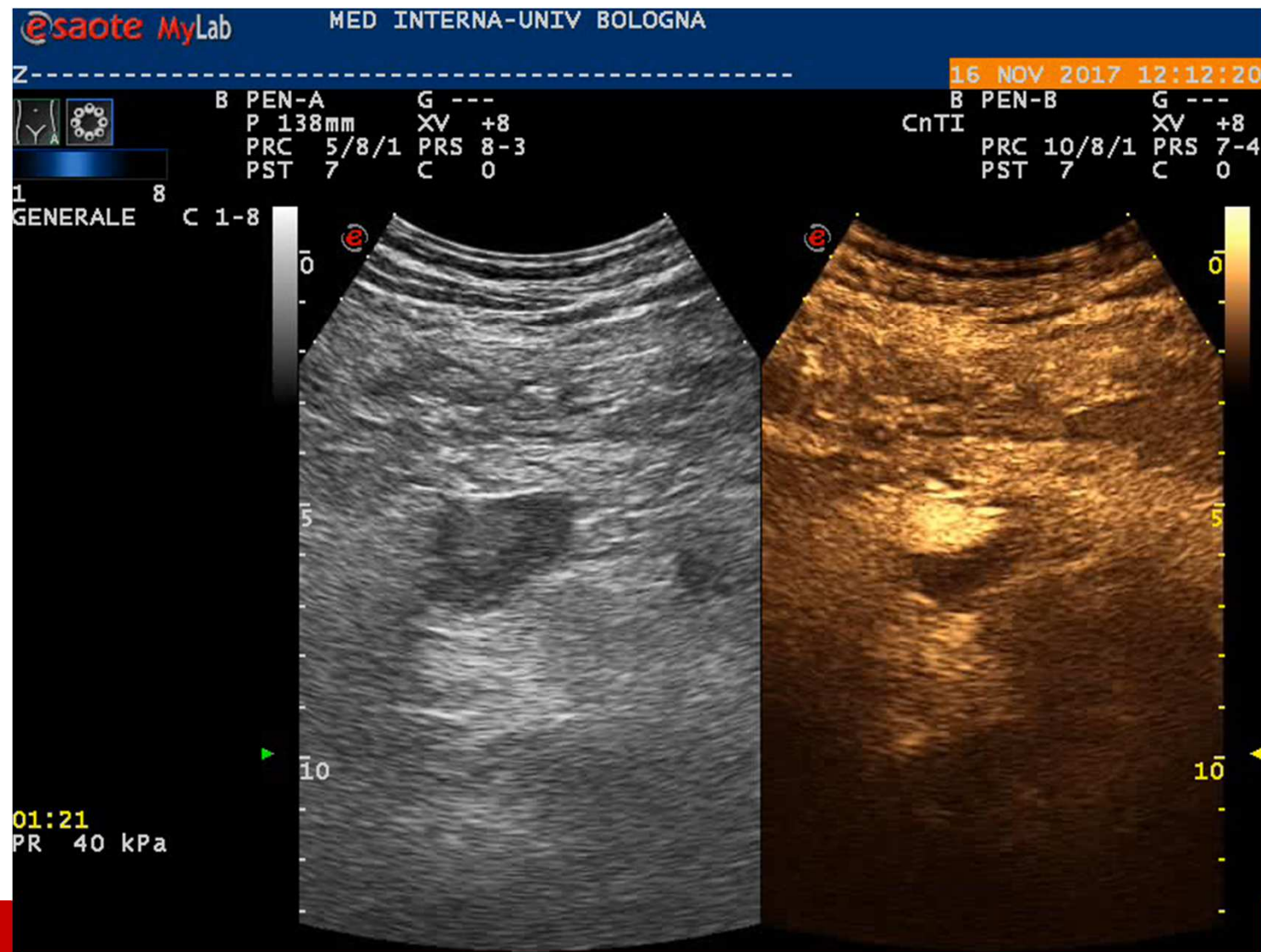


Is it there partial PVT or only slow flow? Role of CEUS



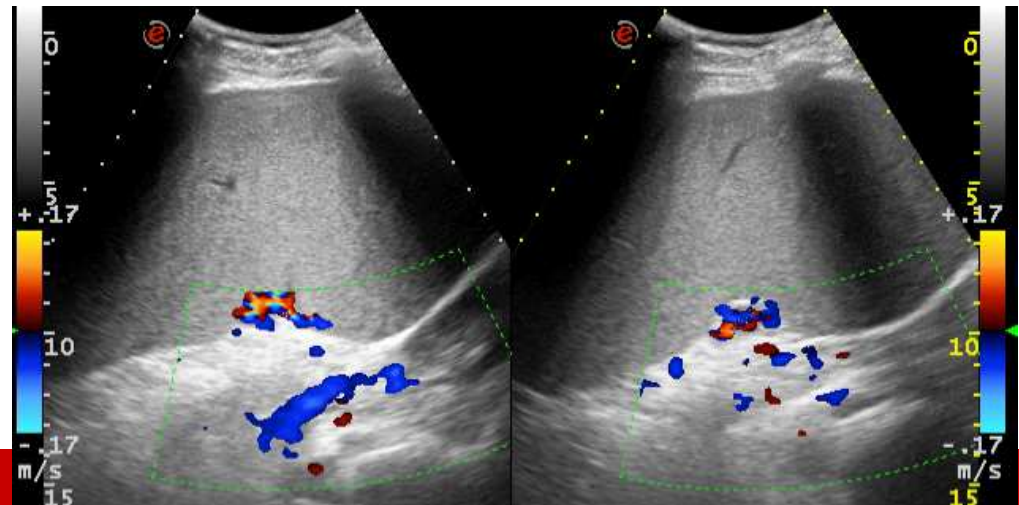
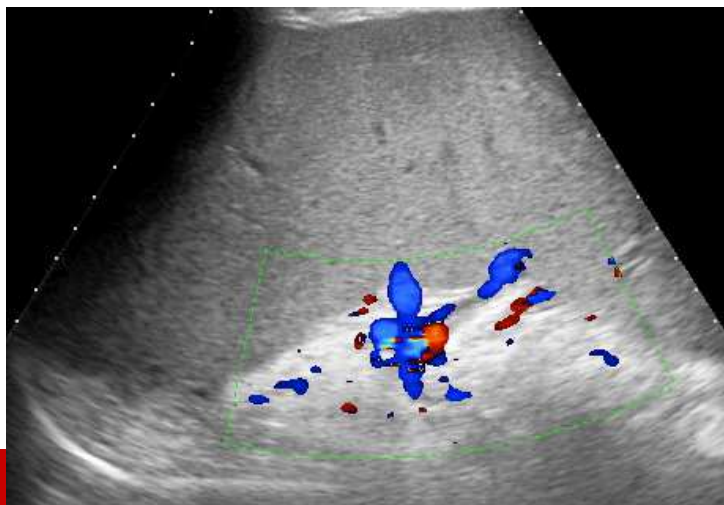
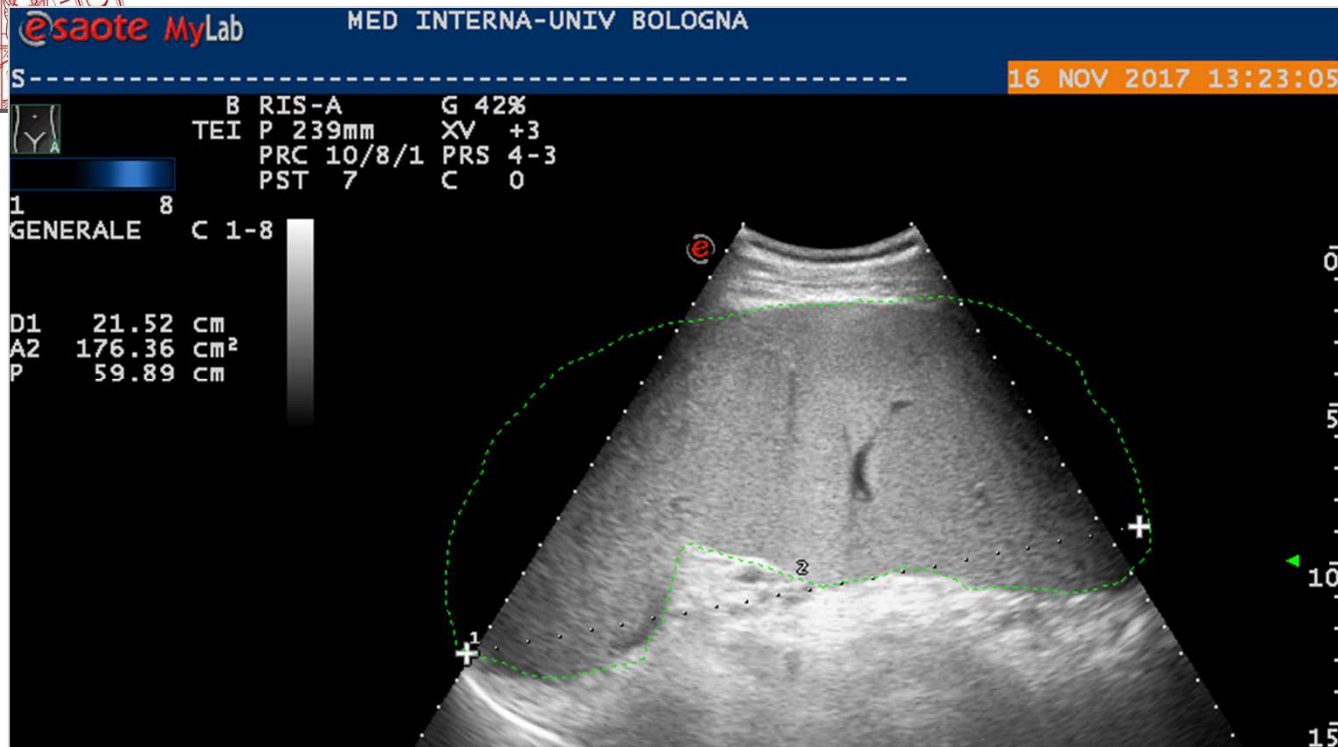


CEUS ACCURATELY SHOW PRESENCE AND EXTENT OF PARTIAL THROMBOSIS OF THE PORTAL TRUNK HARDLY VISIBLE ON COLOR



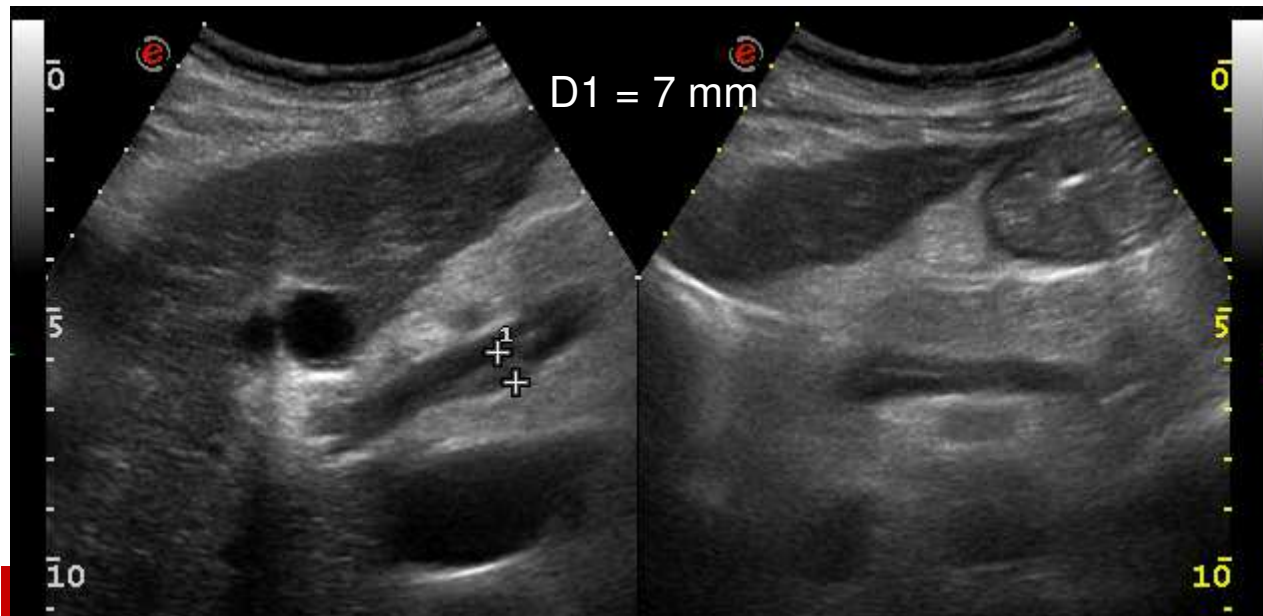
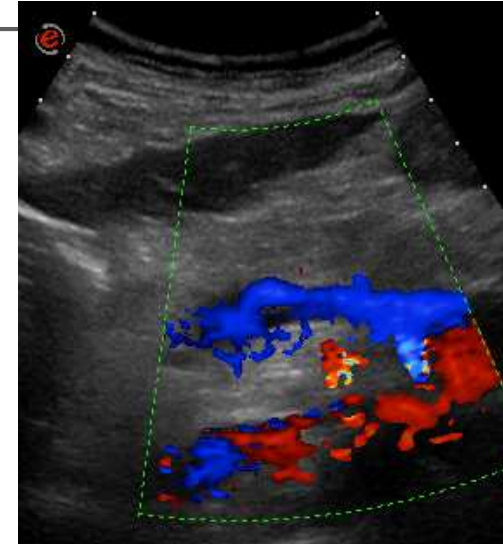
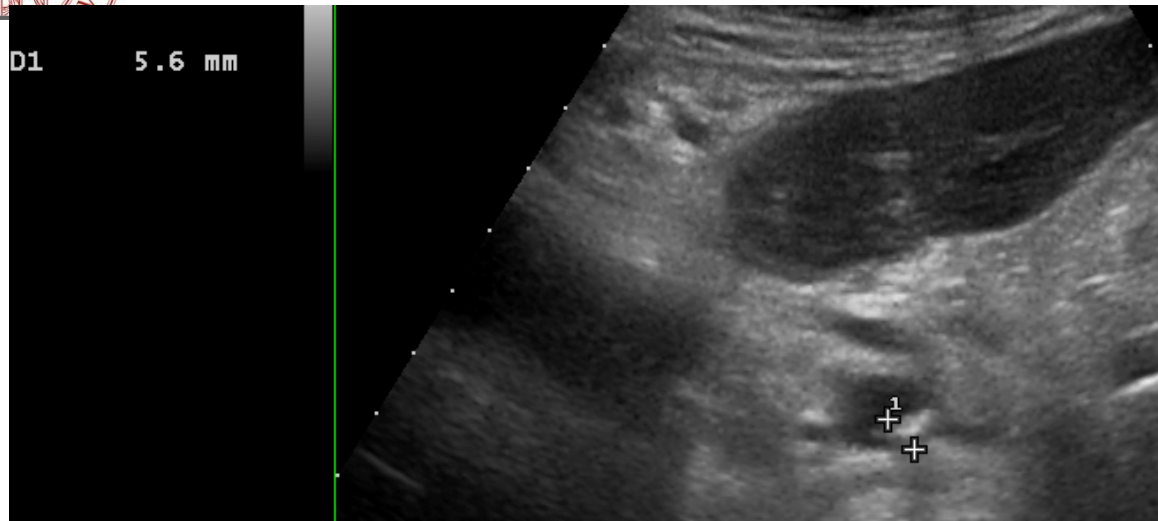


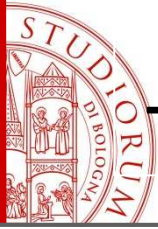
CHRONIC SPLENIC VEIN THROMBOSIS



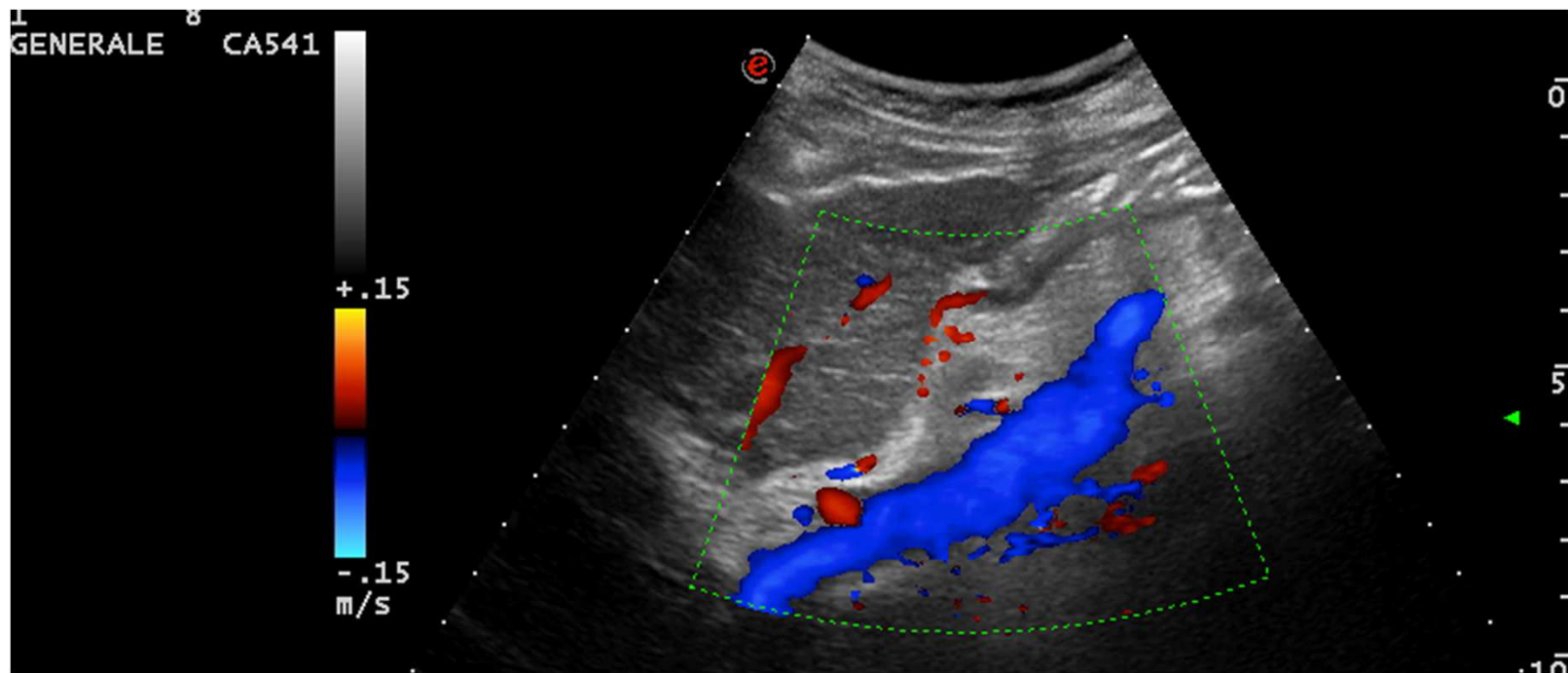


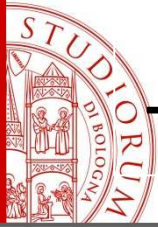
MURAL PARTIAL RECANALIZED THROMBOSIS OF THE PORTAL SYSTEM CAN BE OVERLOOKED BY COLOR



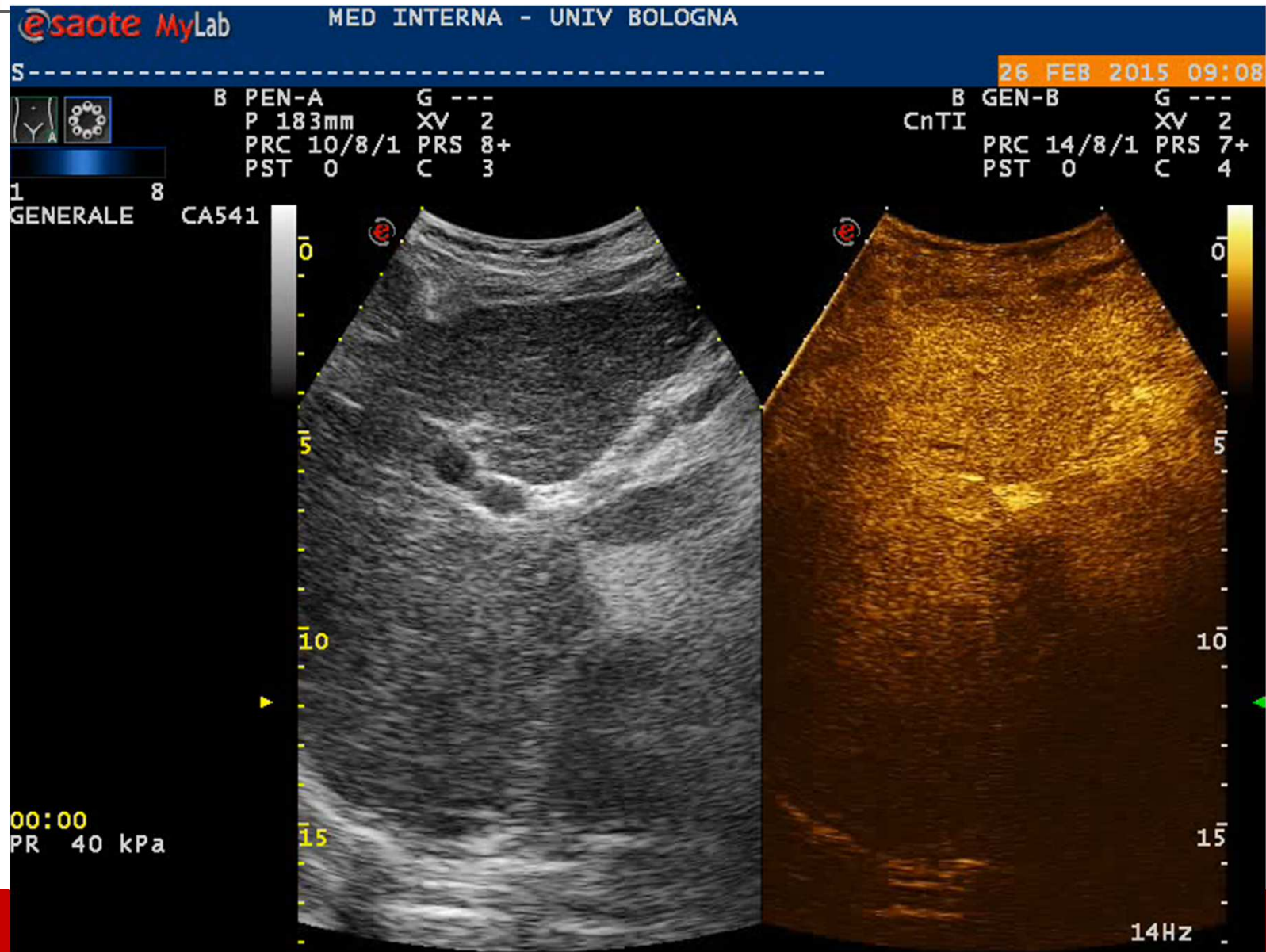


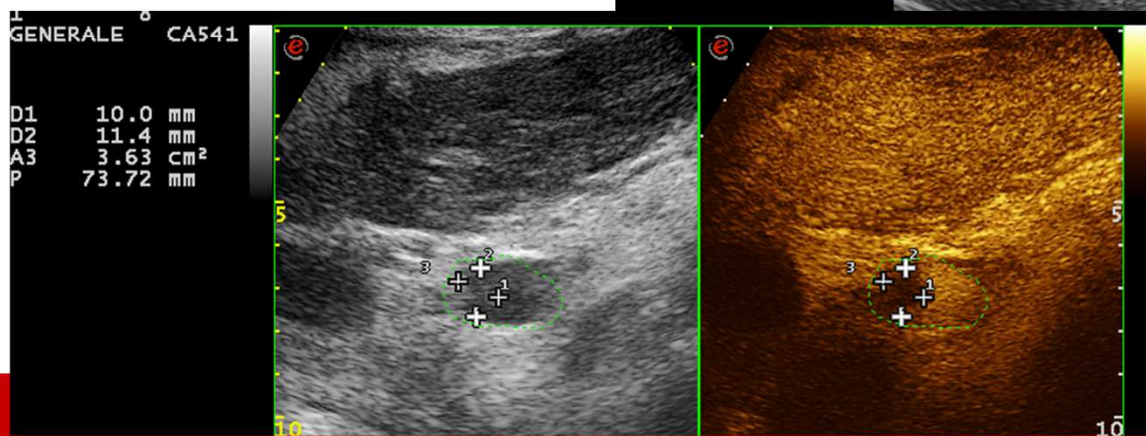
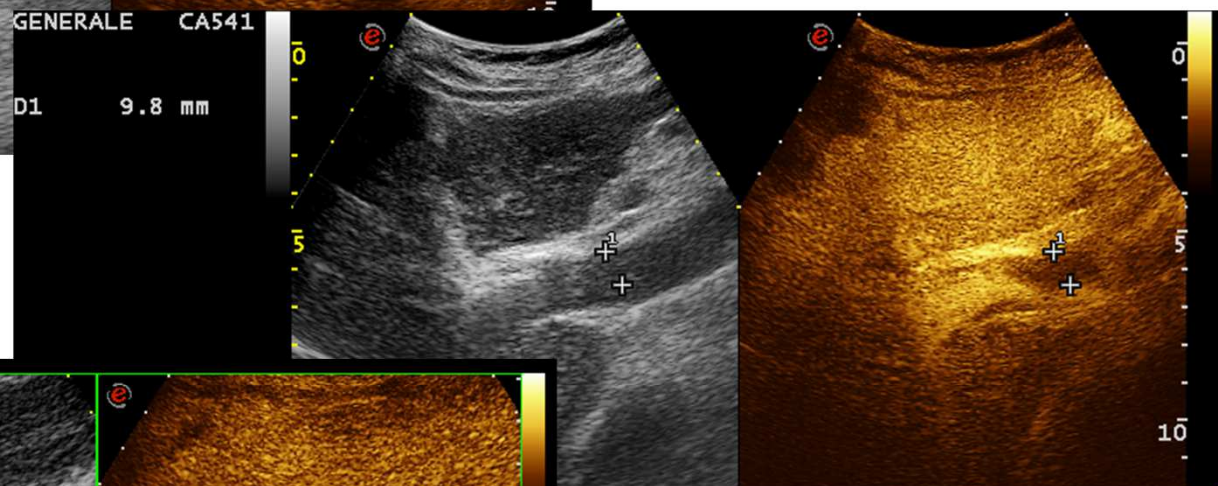
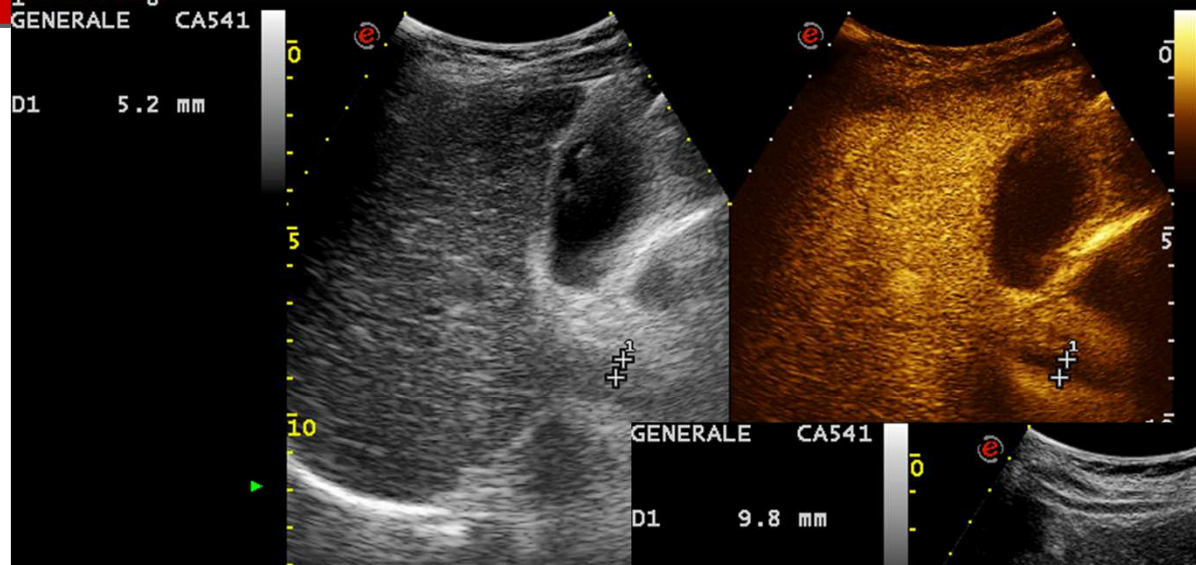
CEUS ACCURATELY SHOW PRESENCE AND EXTENT OF PARTIAL THROMBOSIS OF THE PORTAL TRUNK HARDLY VISIBLE ON COLOR





CEUS ACCURATELY SHOW PRESENCE AND EXTENT OF PARTIAL THROMBOSIS OF THE PORTAL TRUNK HARDLY VISIBLE ON COLOR







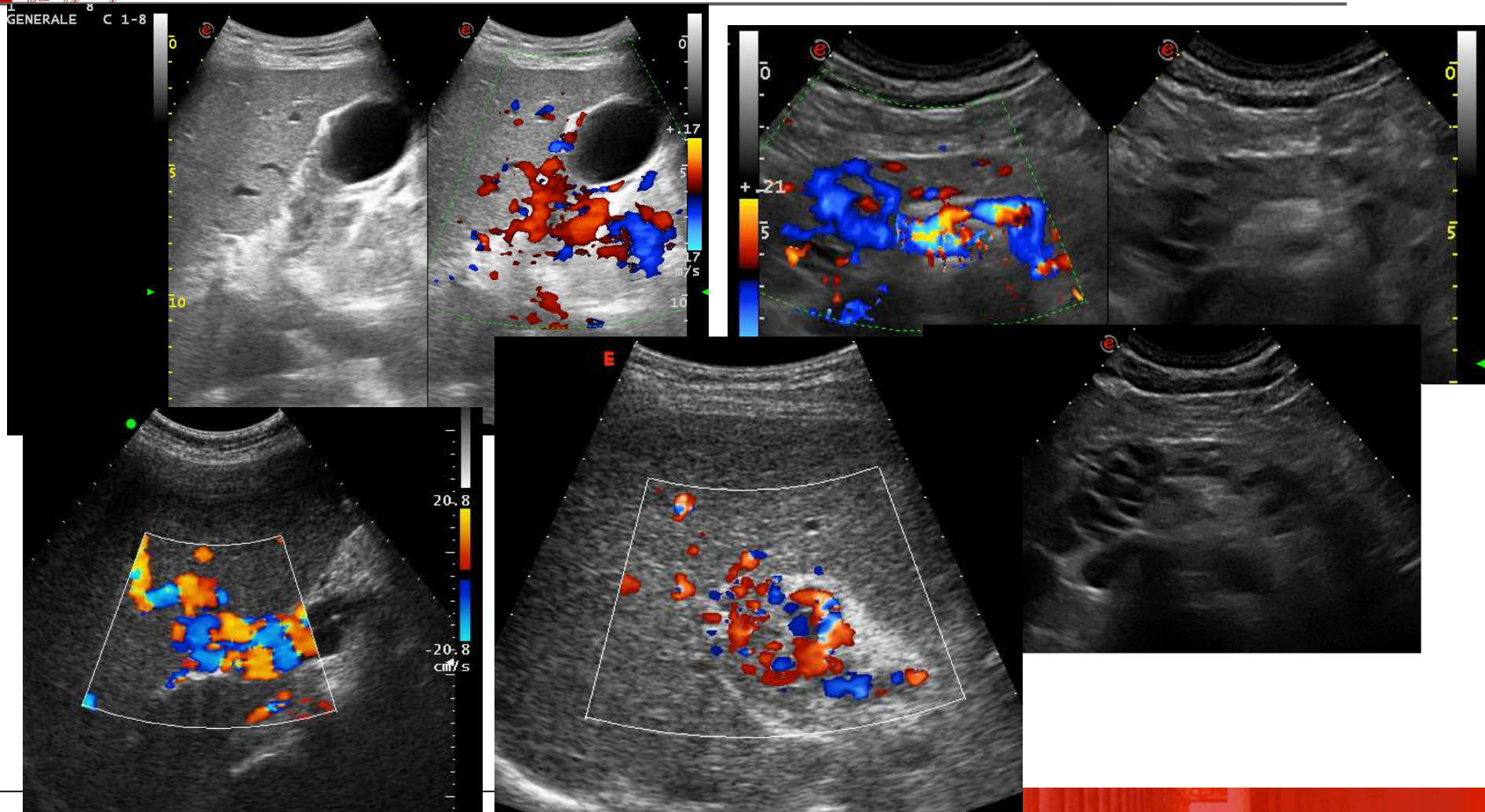
Cavernous transformation and collaterals

- Cavernous transformation of PVT is characterized by the occurrence of small tortuous vessels with hepatopetal flow in the site or around the original portal tract affected by the thrombus
- It starts between 6 and 20 days after an acute onset and once cavernous transformation of the PV is detected, patients are regarded as having entered the chronic stage
- In patients with cavernous transformation of the portal vein or long-lasting extrahepatic portal vein obstruction (EHPVO) morphological changes of the liver, compensatory hypertrophy of the hepatic artery and signs of portal hypertension are frequent.
- Porto-portal collaterals (e.g. in the gallbladder walls) can be seen in non recanalized portal trunk thrombosis





Cavernous transformation and collaterals

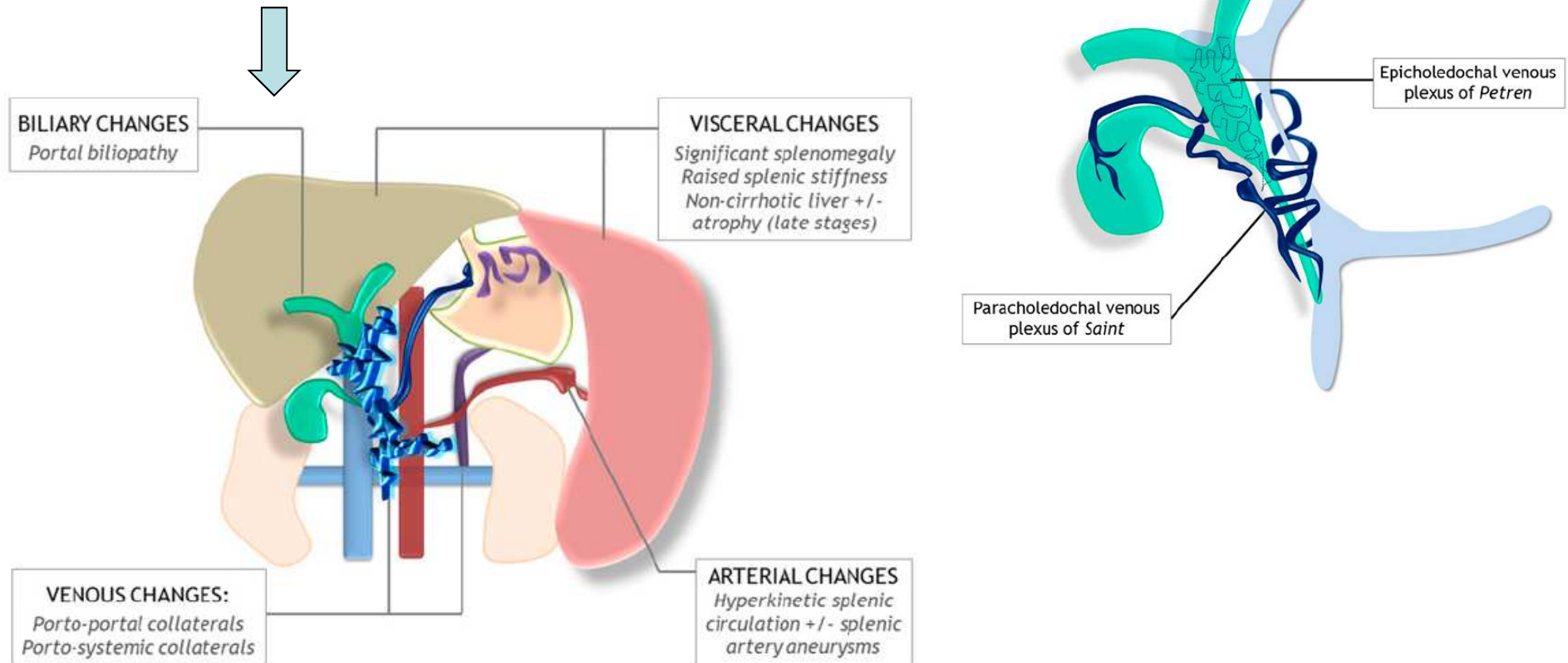


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Extrahepatic Portal Vein Obstruction (EHPVO)

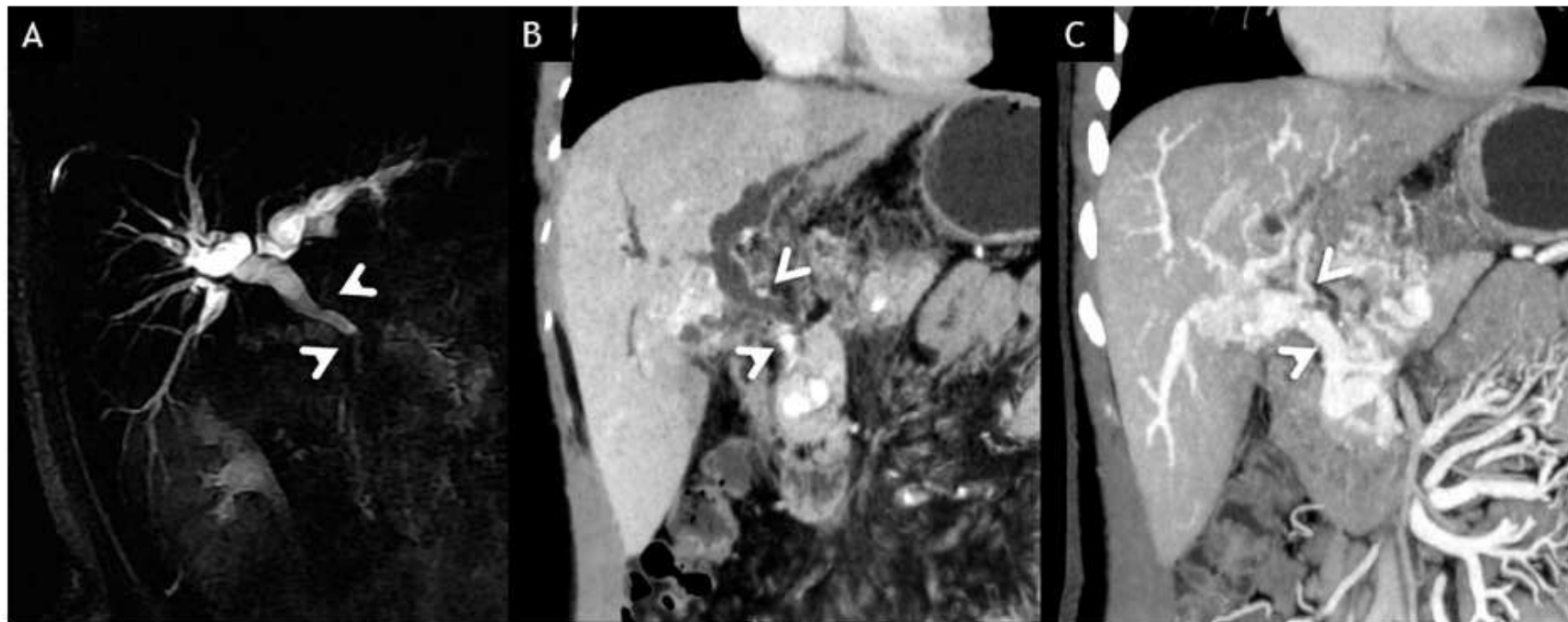
- EHPVO is the chronic evolution of portal vein thrombosis with cavernomatous transformation. ←



IMAGING FOR SUSPECTED PORTAL HYPERTENSIVE CHOLANGIOPATHY

Magnetic resonance cholangiopancreatography (MRCP) is the technique of choice to be applied upon suspicion of bile ducts abnormalities which can occur as a result of long-lasting cavernous transformation of the

Figure 8. Portal cavernoma cholangiopathy. (a) Thick-slab two-dimensional MR cholangiopancreatography showing an ectatic common duct with extrinsic indentations (arrowheads), luminal compromise and upstream biliary dilatation. (b) Coronal CT minimum intensity projection showing paracholedochal collaterals indenting the biliary tree (arrowheads). (c) Coronal CT maximum intensity projection showing cavernomatous transformation of the portal vein (arrowheads).



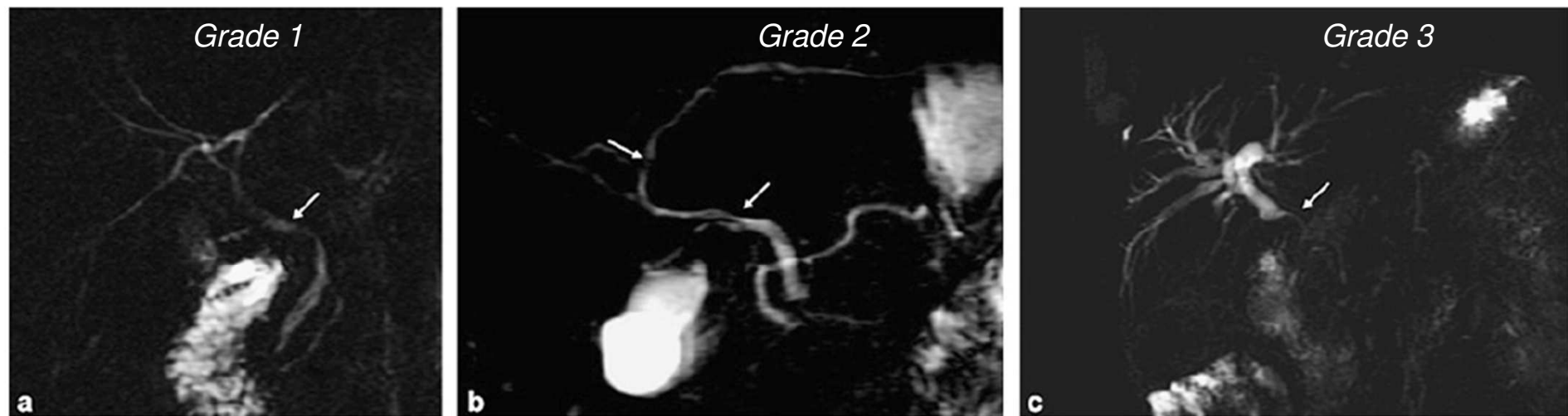


Figure 1 Thick-slab magnetic resonance cholangiography images. (A) Parietal irregularities affecting the intrahepatic and extrahepatic ducts (arrow) consistent with grade I portal cholangiopathy (PC). (B) Grade II PC: strictures of the biliary ducts (arrows) without dilation. (C) Grade III PC: presence of strictures (arrow) with dilation affecting the intrahepatic and extrahepatic ducts.

Llop. Gut 2011;60:853-860

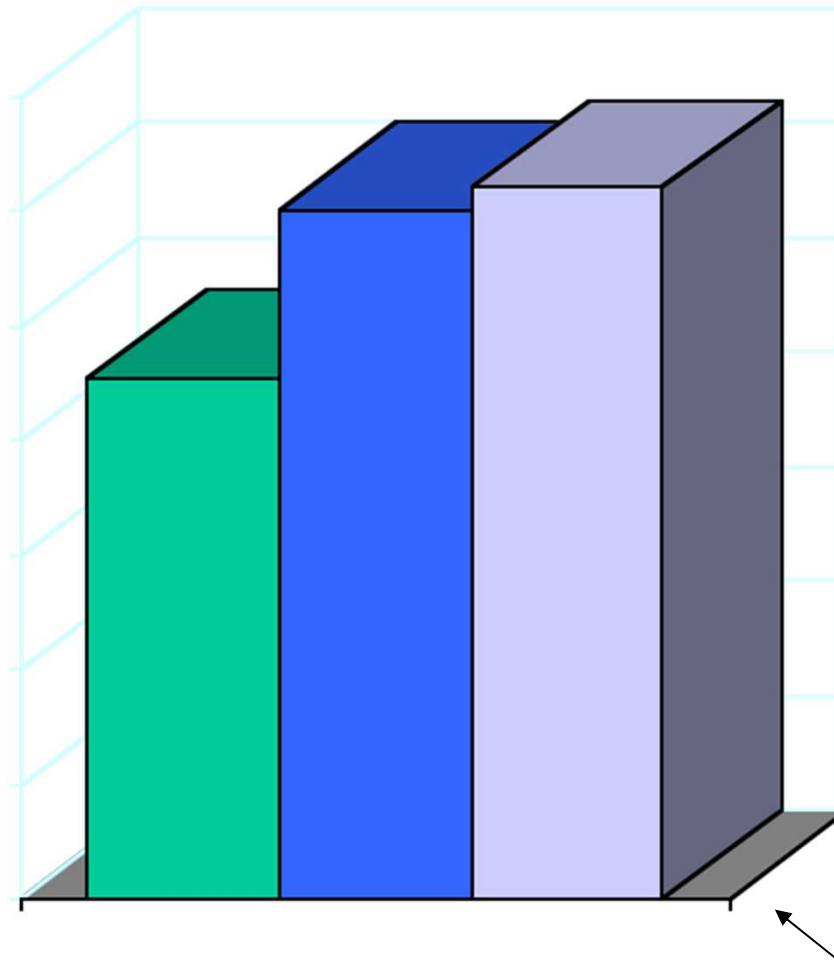


US AND DOPPLER DIAGNOSIS OF PORTAL HYPERTENSION



Enlargement of the portal system

Suggestive but not specific for portal hypertension



Venous Dilation:

Portal vein

- normal <12 mm
- mild enlarg 12-13 mm
- significant enlarg >13 mm
- severe enlarg. >15mm

Splenic vein

normal >10 mm

[Piscaglia, Bolondi
Eur J Gastro Hepatol, 1997]

AScites



Non Invasive detection of portal hypertension

- PT < 70%
- PLT < 100.000/L
- Portal vein > 13 mm



Predict presence of esophageal varices

[Schepis, Hepatology 2001]

Portal vein > 15 mm



Predict presence of large esophageal varices (F2-F3)

[Cottone, Am J Med 1985]

N.B. conditions at risk for false negative results: portal flow inversion and/or large porto-systemic collaterals

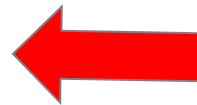


Stiffness of the portal system at forced breathing

Specific sign for portal hypertension

Normal

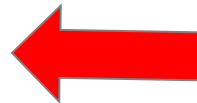
Collapse or marked thinning
of portal system (sup. mesent
and splenic veins)



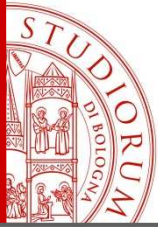
*Expiration after deep
inspiration*

Portal hypertension

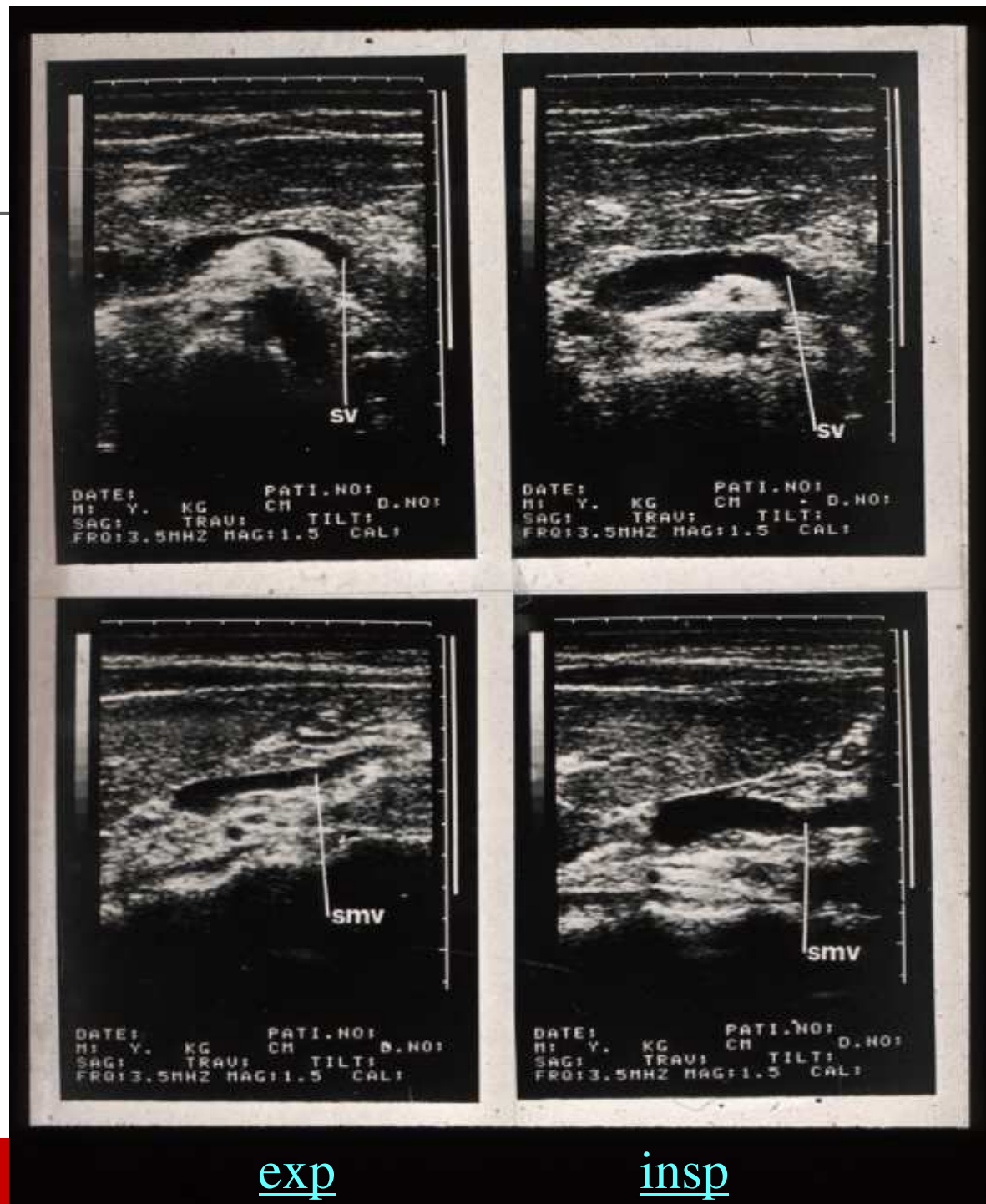
Caliber reduction <40%

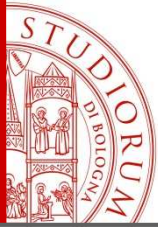


[Bolondi, Surgery 1984]

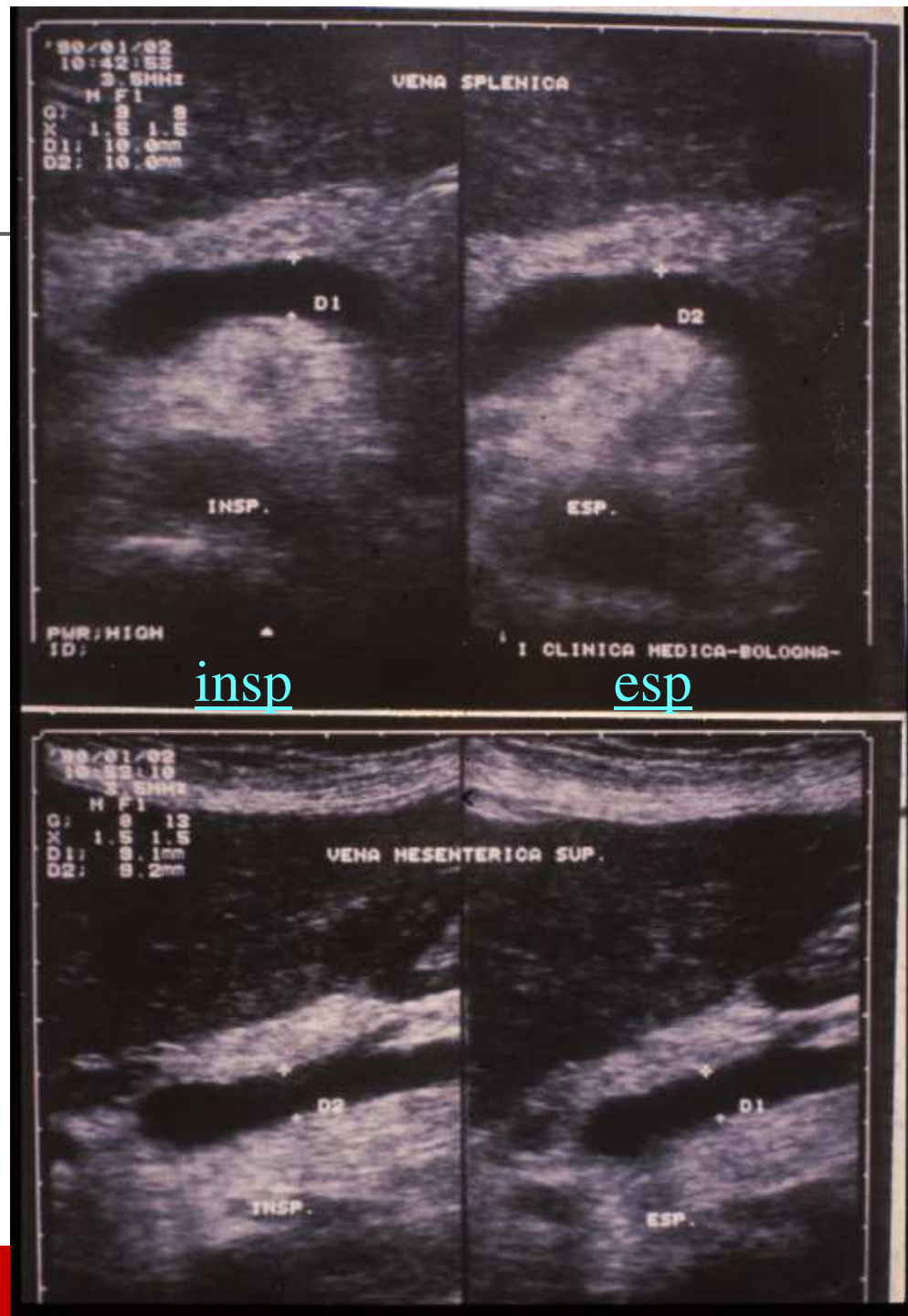


normal



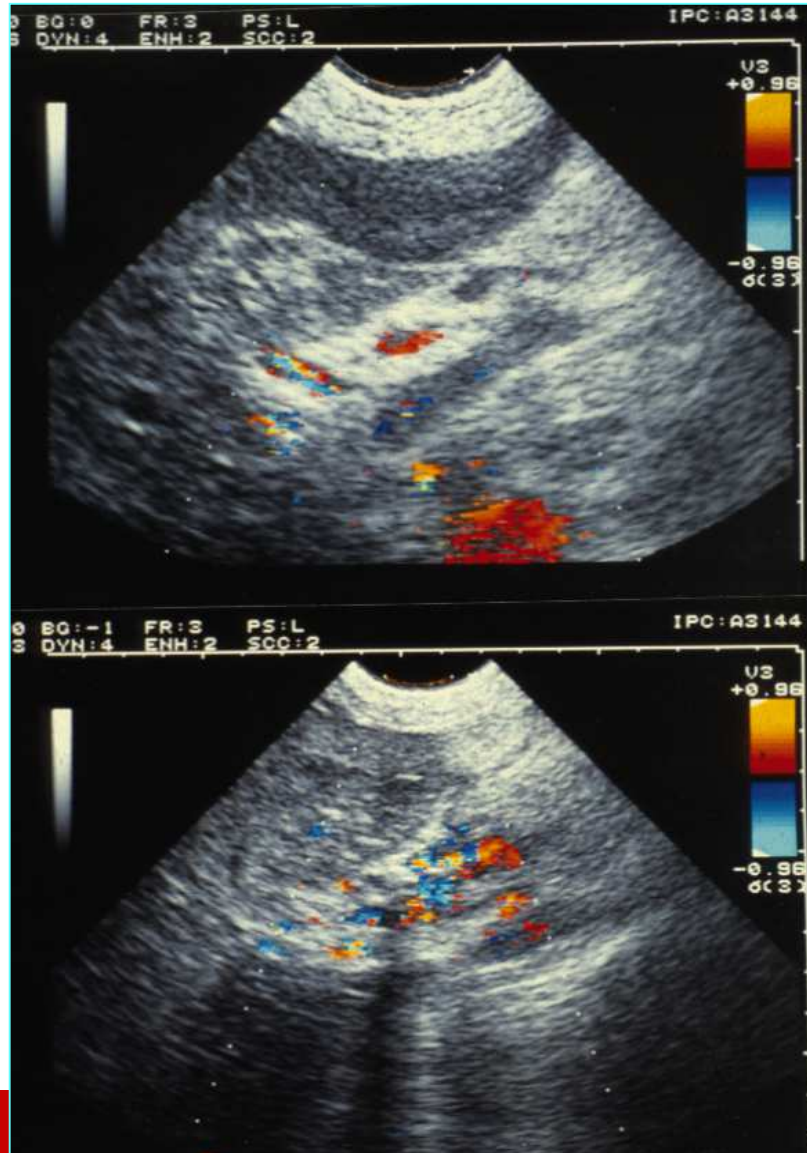


portal
hypertension

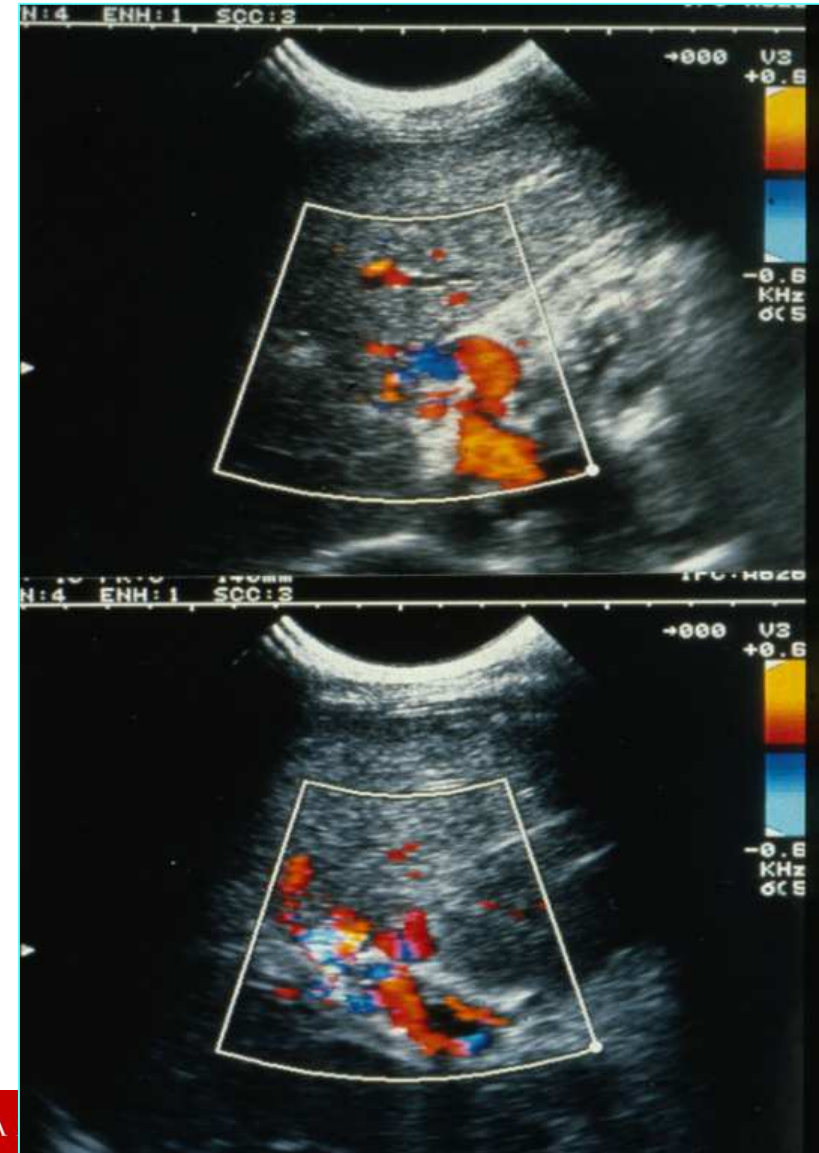




Complete thrombosis of the main portal vein + initial cavernomatous transformation

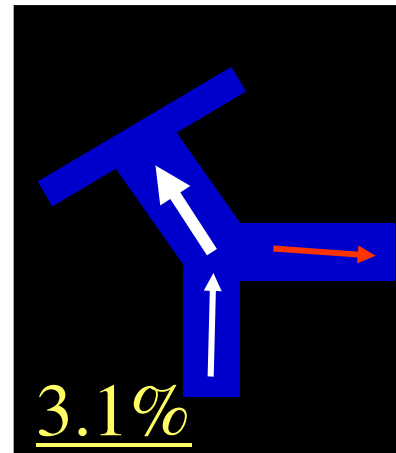
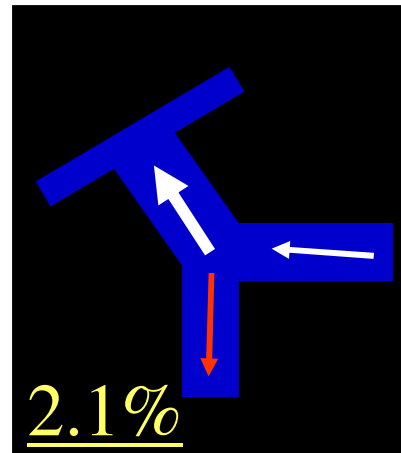
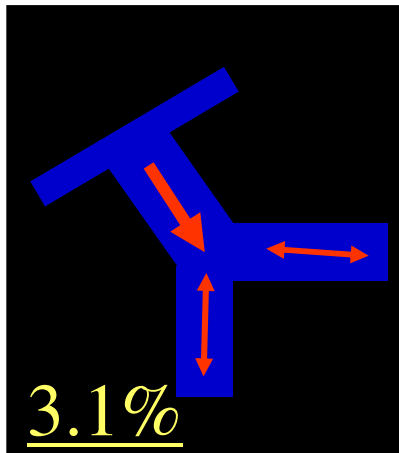


Long lasting thrombosis with cavernomatous transformation



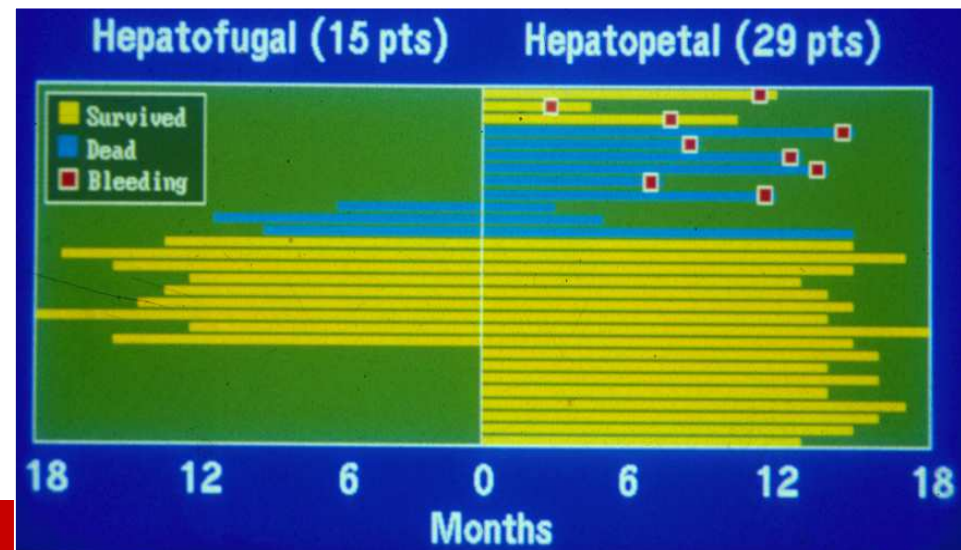
DOPPLER: PORTAL FLOW DIRECTION

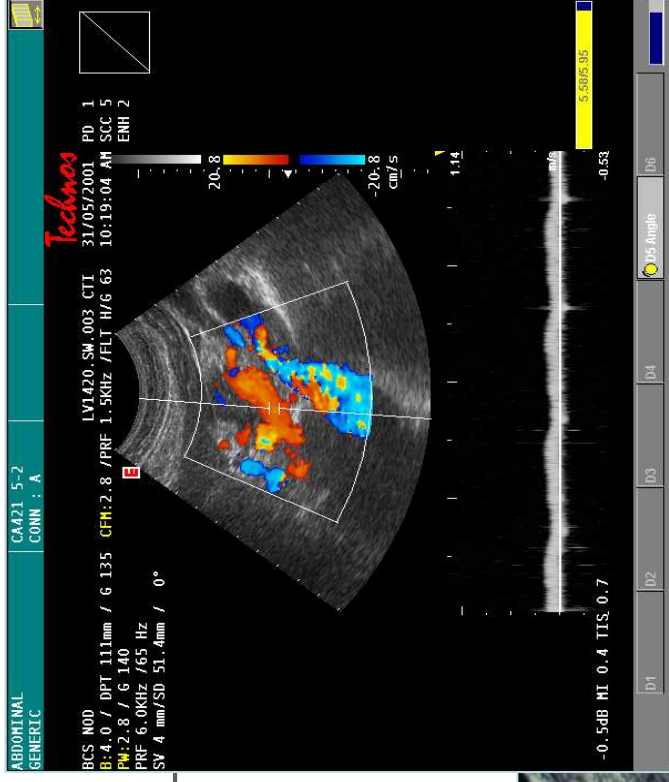
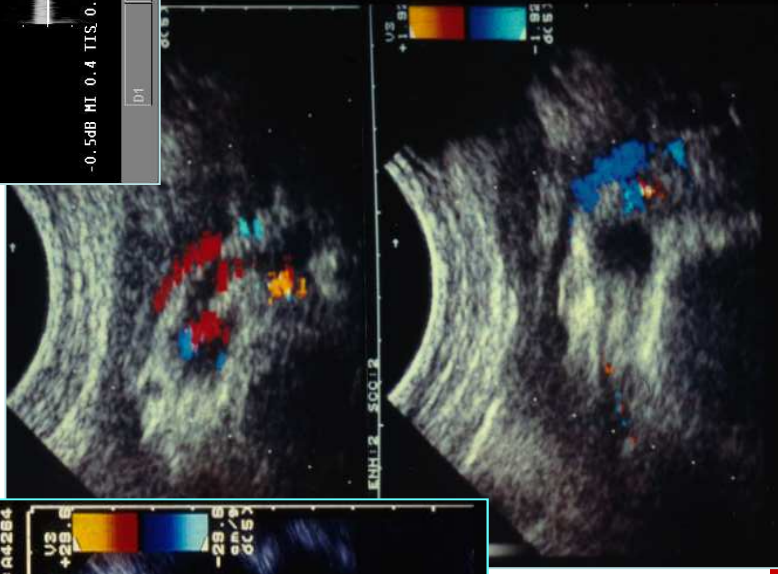
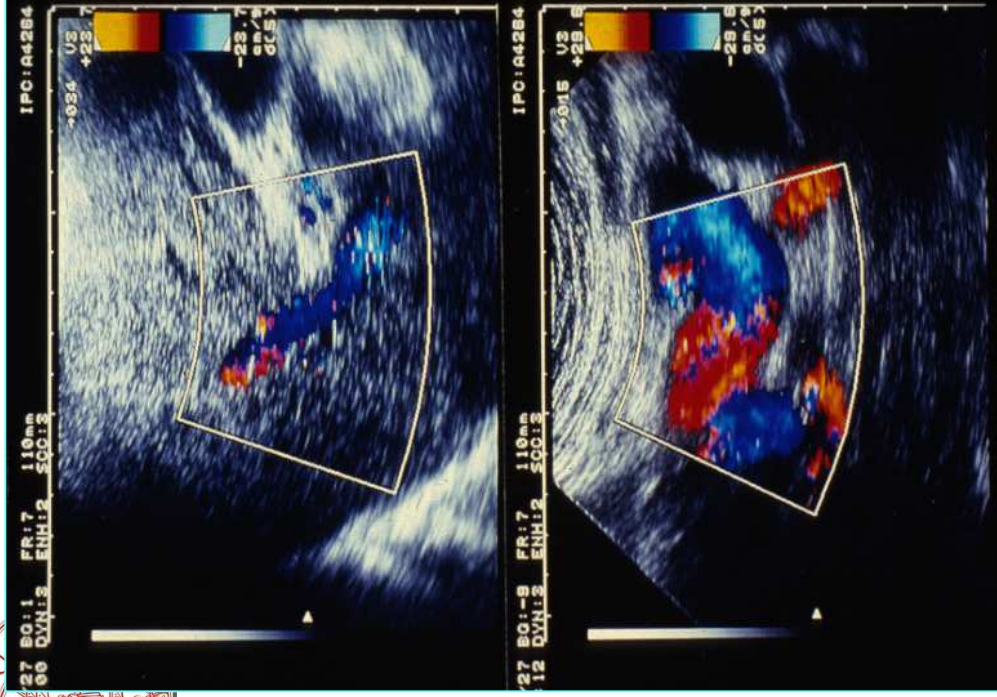
- Inversion of portal flow direction is pathognomonic of portal hypertension. This sign is present in 8.3% of cirrhotic patients without HCC



inversion of portal flow
appear associated to a
decrease of hemorrhage
risk (no change of survival)

Bolondi Gastroenterology 1991





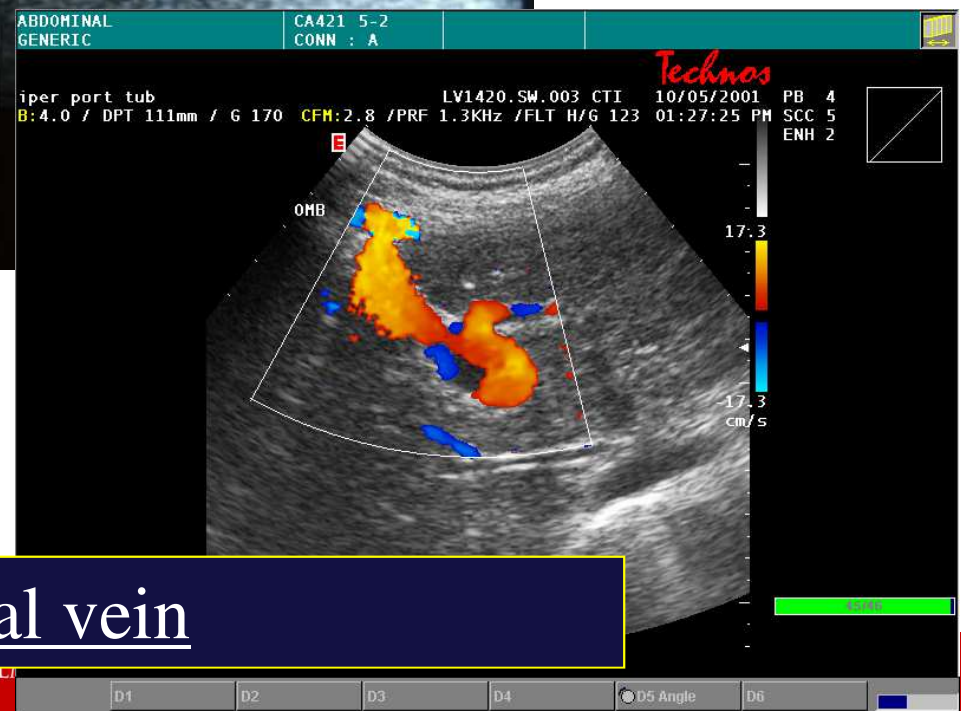


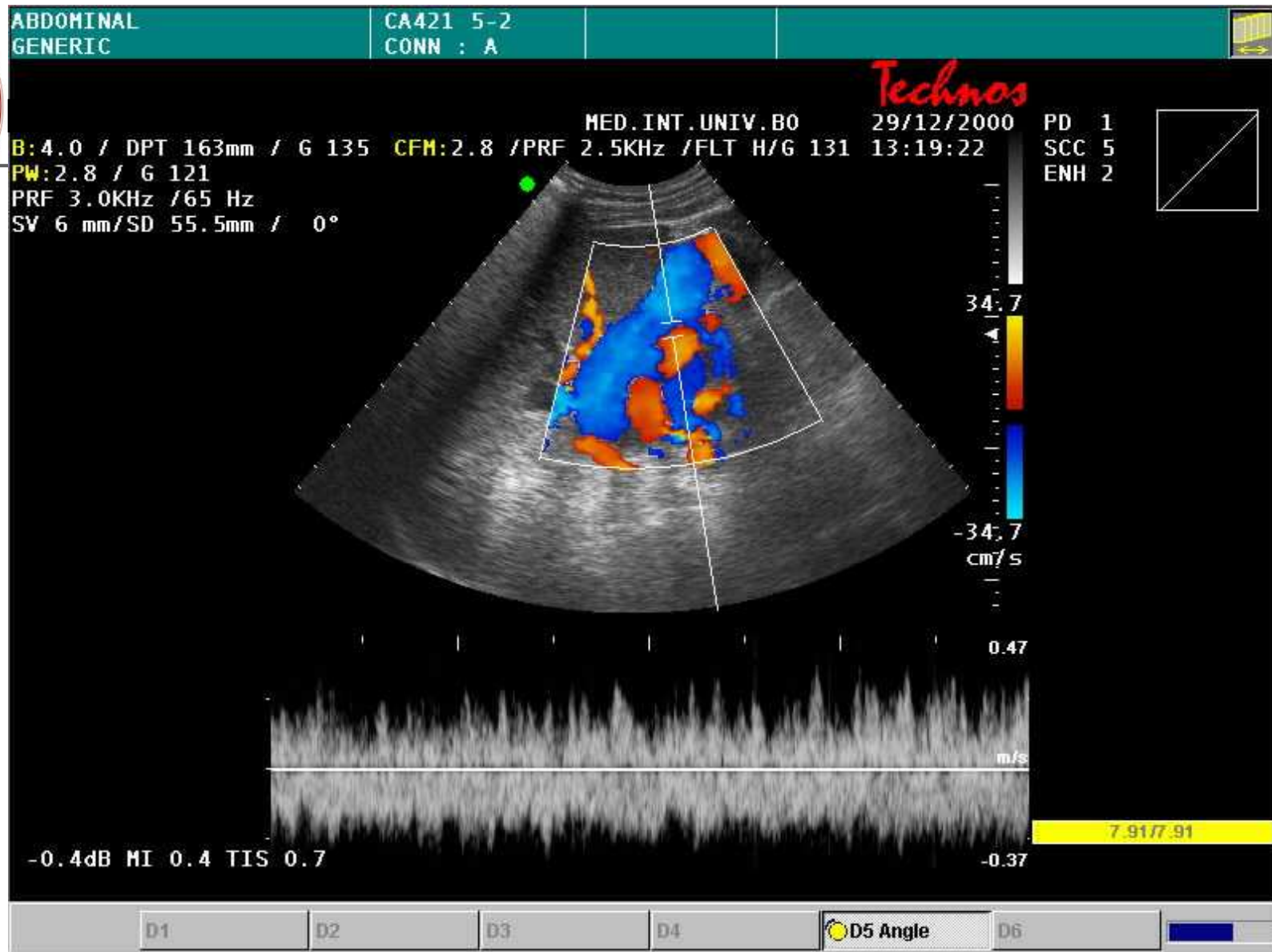
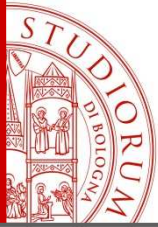
COLLATERAL CIRCULATION

The detection of porto-systemic collateral vessels is specific of portal hypertension

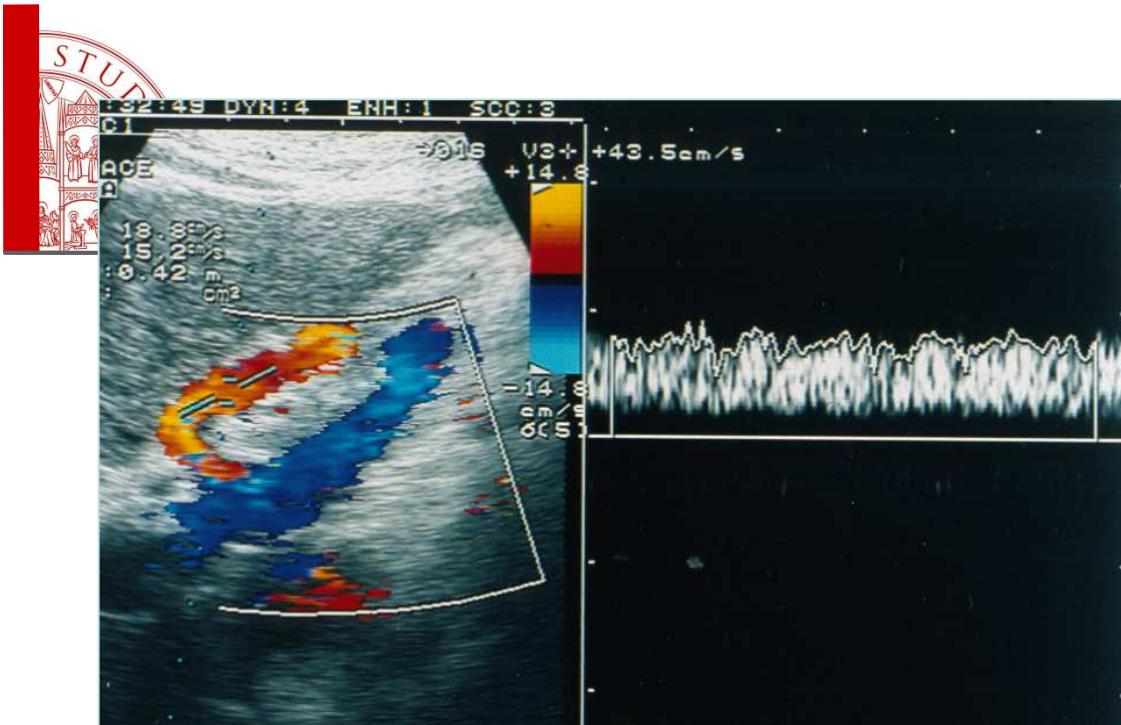
- umbilical vein
- spontaneous splenorenal shunts
- gastroesophageal vessels: left gastric vein and short gastric veins.
- retroperitoneal circulation
- gallbladder wall vessels

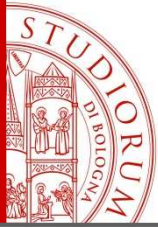
caput medusae



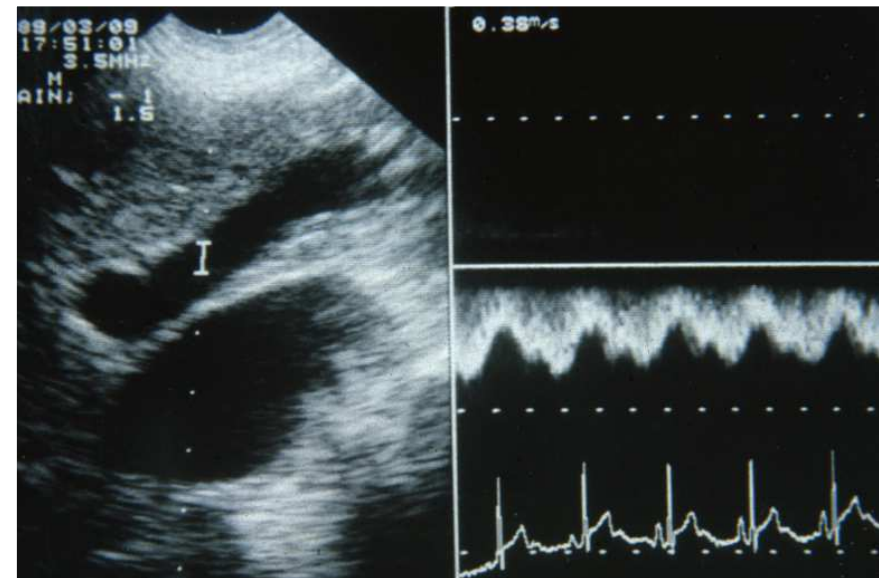
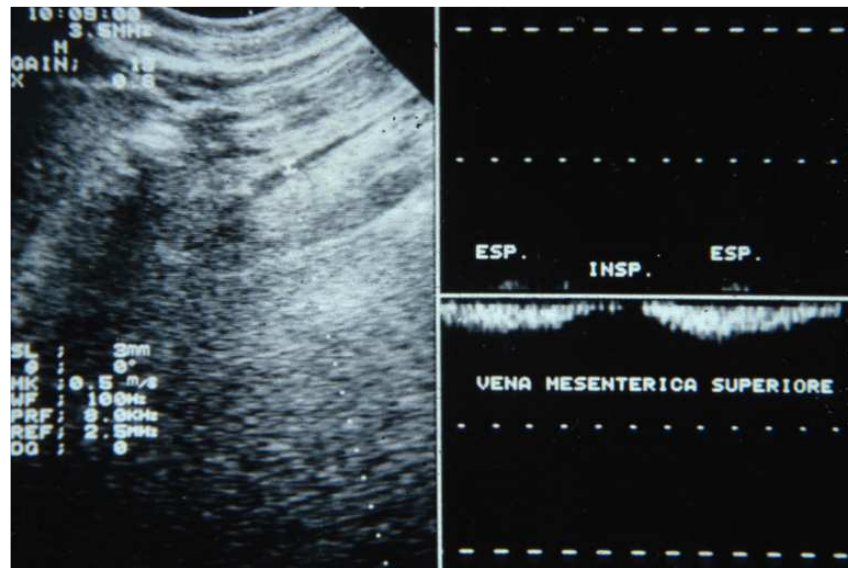


spontaneous splenorenal collaterals





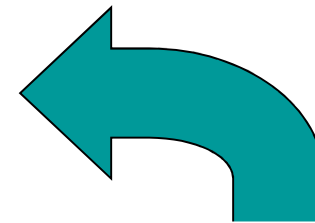
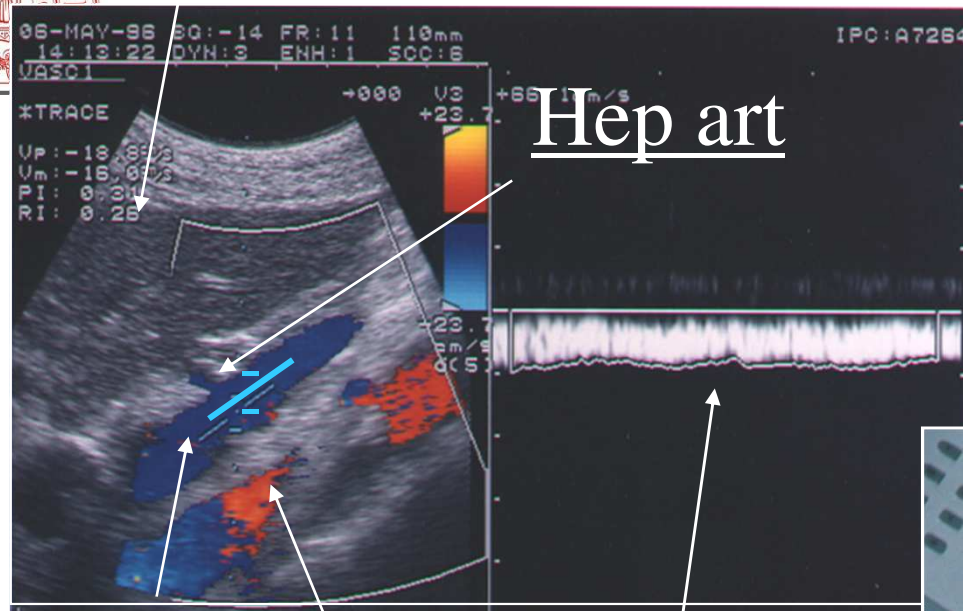
Portal system assessment



Qualitative characteristic of portal flow.
Normal physiologic variations.



Assessment of portal vein in cirrhosis



During suspended normal breathing. Site: just after the crossing with the hepatic artery while visualizing a longitudinal tract of at least 3-4 cm [Bolondi J Hepatol 1995]. Measurement over a period of at least 3-4 seconds.



DOPPLER: PORTAL FLOW VELOCITY (P-VEL)

P-VEL, together with nodular liver profile, is the best US finding to differentiate compensated cirrhosis from chronic hepatitis (cut-off < 15 cm/s).

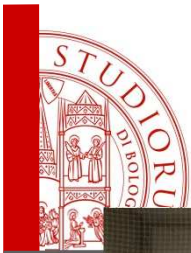
Gaiani, Bolondi *J Hepatol* 1997

P-VEL is reduced in cirrhosis and slow downs with the progression of the disease severity

Zironi, Bolondi *J Hepatol* 1992

Markedly reduced P-VEL (mean < 10 cm/s) is associated to shorter survival in compensated cirrhosis

Zoli, *J Hepatol* 1996



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