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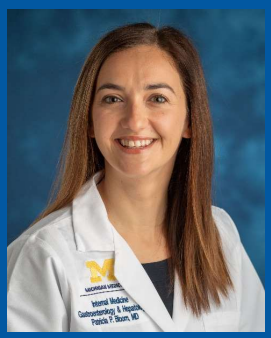

Moderator
Patricia L. Bloom, MD

All attendees will be muted and will remain in "Listen Only Mode"

Type your questions here so that the moderator can see them.
Not all questions will be answered but we will get to as many as possible.

A handout with the slides and room to take notes can be downloaded from your control panel.

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ACG Virtual Grand Rounds

Join us for upcoming Virtual Grand Rounds!


Week 31 – Thursday, August 3, 2023
American College of Gastroenterology Guidelines Update:
Diagnosis and Management of Celiac Disease
Faculty: Benjamin Lebwohl, MD, MS
Moderator: Carol E. Semrad, MD, FACG
At Noon and 8pm Eastern


Week 32 – Thursday, August 10, 2023
Unleashing the Power of AI in Gastroenterology: Going Beyond Lesion Detection to Transform Clinical Tasks and Everyday Practice
Faculty: Sravanthi Parasa, MD
Moderator: Vladimir Kushnir, MD, FACG
At Noon and 8pm Eastern

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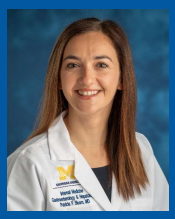
Disclosures



Patricia L. Bloom, MD
Vedanta: Grant/Research Support

Uchenna A. Agbim, MD
Dr. Agbim has no relevant financial relationships with ineligible companies.

Arnab Mitra, MD
Dr. Mitra has no relevant financial relationships with ineligible companies.

**All of the relevant financial relationships listed for these individuals have been mitigated*

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Recent Updates in the Management of Varices

Uchenna Agbim, MD
Assistant Professor
Saint Louis University

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Objectives

- Understand the development of portal hypertension and development of varices
- Highlight changes from the Baveno VII consensus conference regarding variceal and portal hypertensive bleeding
- Identify appropriate treatment for patients with variceal bleeding

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Outline

- Pathophysiology of Varices
- Prevention of Variceal Bleed
- Active/Acute Variceal Bleed
- Preventing Subsequent Variceal Bleeds
- Take-Home Points

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Pathophysiology

- Intrahepatic resistance
- Portal venous blood flow
- HVPG > 5 portal hypertension
- HVPG > 10mmHg = Clinically significant portal hypertension

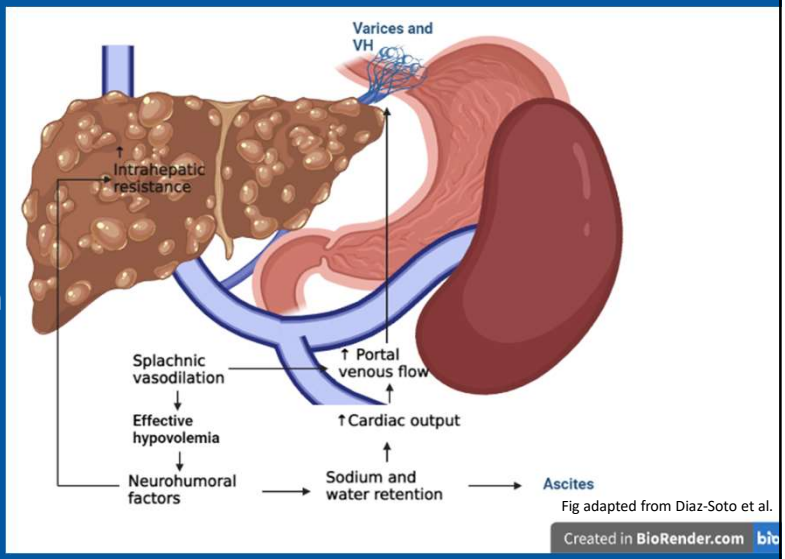

Fig adapted from Diaz-Soto et al.

Diaz-Soto M & Garci-Tsao G. Therap Adv Gastroenterol. 2022 Jun 20;15: 17562848221101712

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Pathophysiology

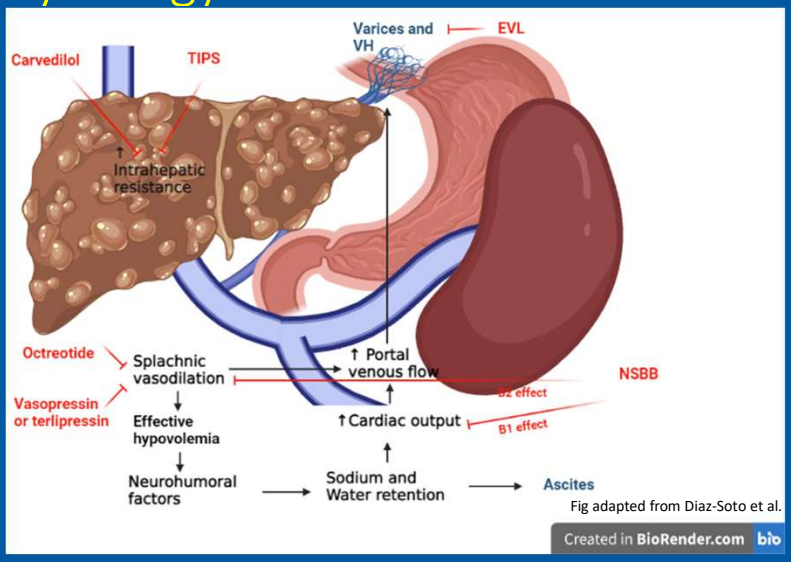

Fig adapted from Diaz-Soto et al.

Diaz-Soto M & Garci-Tsao G. Therap Adv Gastroenterol. 2022 Jun 20;15: 17562848221101712

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Pathophysiology and Natural History

	Compensated		Decompensated		
	No clinically significant portal HTN	Clinically significant portal HTN (CSPH)	First decompensating event	Acute VH	Secondary prophylaxis
Goals	Prevent CSPH	Prevent decompensating events	Prevent further decompensating events	Stabilize/control bleeding, prevent early rebleeding and death	Prevent further bleeding, other decompensations, death

Garcia-Tsao G et al. Hepatology. 2017 Jan;65(1):310-335.

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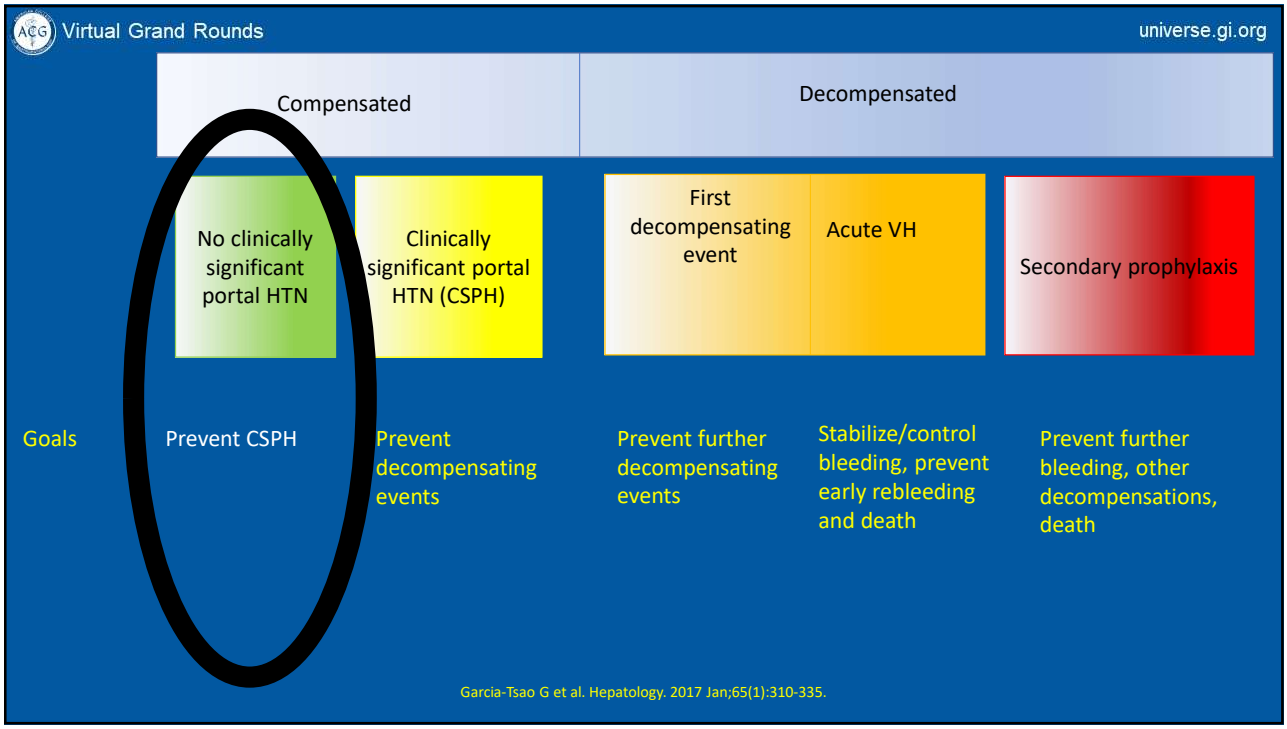
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Garcia-Tsao G et al. Hepatology. 2017 Jan;65(1):310-335.

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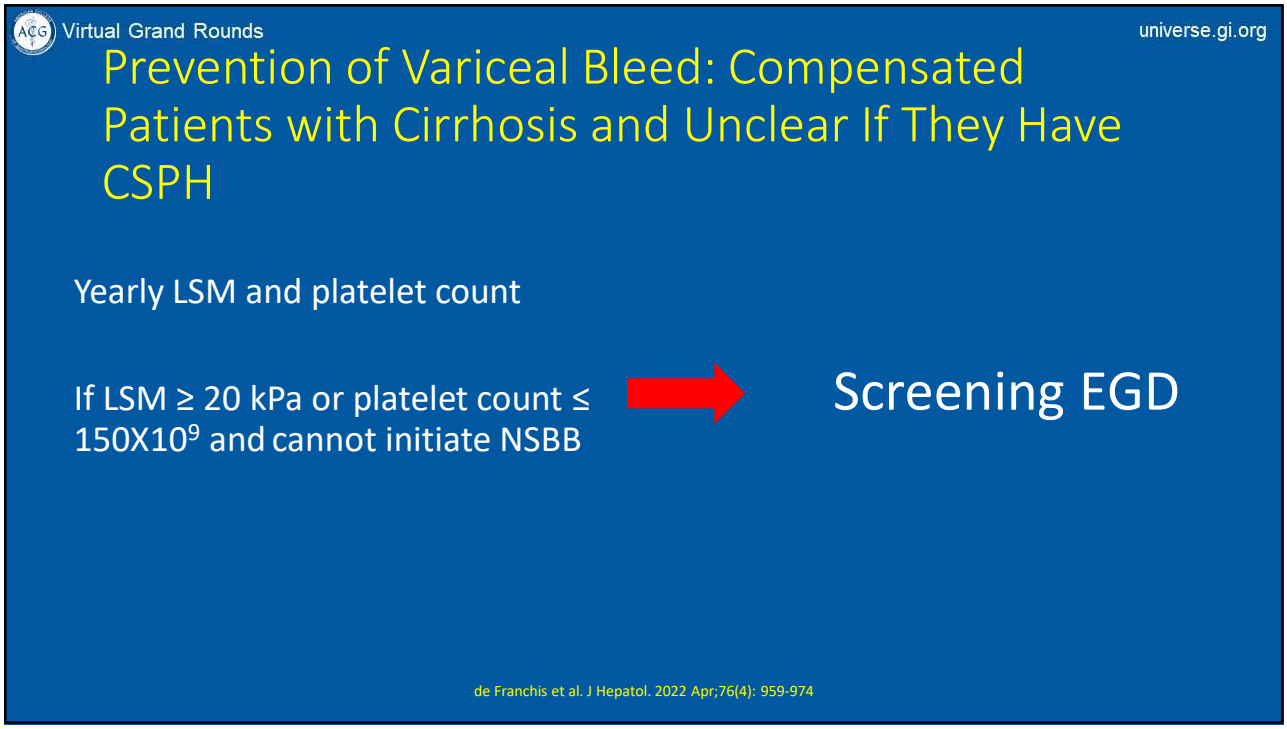
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Prevention of Variceal Bleed: Compensated Patients with Cirrhosis and Unclear If They Have CSPH
Yearly LSM and platelet count
If LSM ≥ 20 kPa or platelet count ≤ 150X10^9 and cannot initiate NSBB
Screening EGD
de Franchis et al. J Hepatol. 2022 Apr;76(4): 959-974

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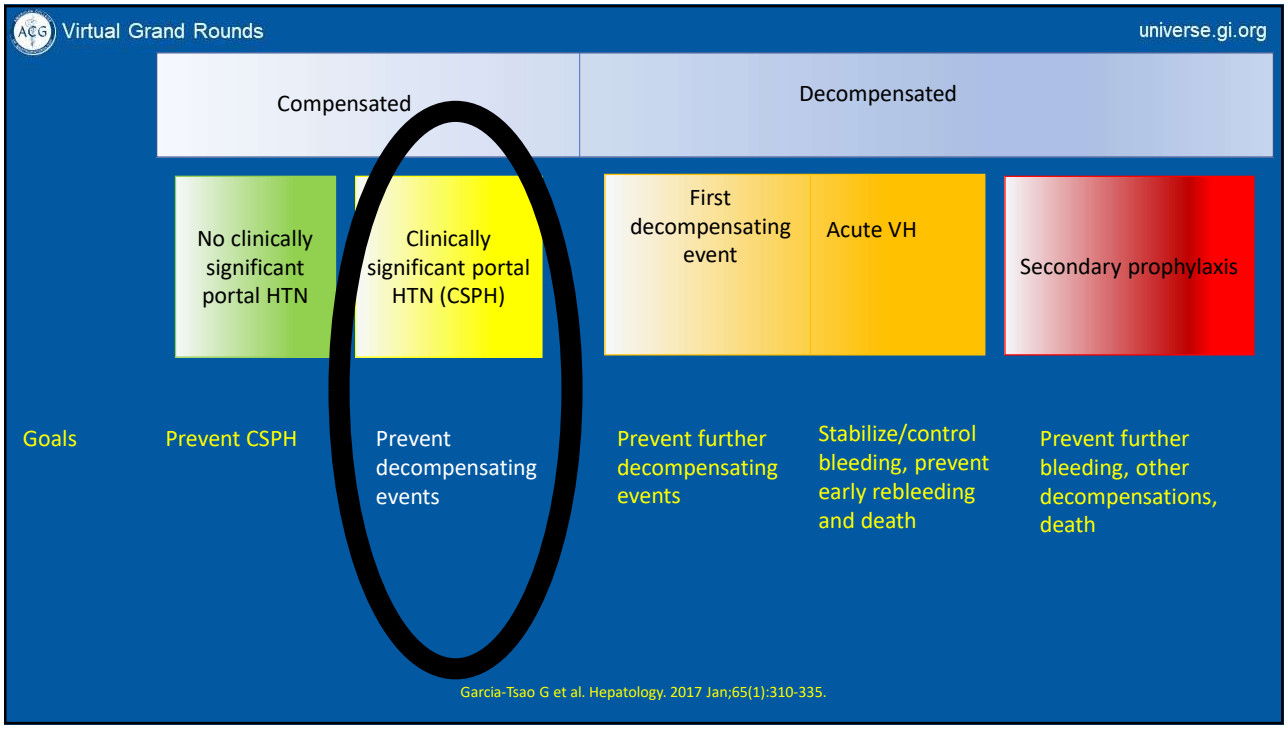

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Prevention of Variceal Bleed in Compensated Patients with CSPH

NSBB

- Consider using NSBB to prevent decompensation
  - **Any liver decompensation**
- Large type 2 gastroesophageal or isolated type 2 gastroesophageal varices
- Carvedilol
  - Preferred NSBB in compensated cirrhosis
  - Anti α adrenergic vasodilatory effects → greater portal pressure reducing effect
- Nadolol and propranolol

OR

Endoscopic Interventions

- Endoscopic Variceal Ligation
  - Varices and intolerance to NSBB
  - EVL in compensated patients with high risk-stigmata
- Cyanoacrylate
  - Large type 2 gastroesophageal or isolated type 2 gastroesophageal varices
    - Use NSBB
    - Cyanoacrylate vs NSBB: No difference in survival, but cyanoacrylate may be more effective

1. de Franchis. J Hepatol. 2022 Apr;76(4): 959-974
2. Diaz-Soto M & Garci-Tsao G. *Therap Adv Gastroenterol.* 2022 Jun 20;15: 17562848221101712

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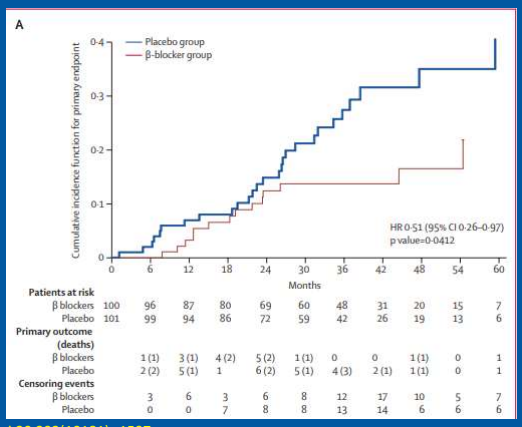
Prevention of Variceal Bleed in Compensated Patients with CSPH

Why use NSBB to prevent any Decompensation?

β blockers to prevent decompensation of cirrhosis in patients with clinically significant portal hypertension (PREDESCI): a randomised, double-blind, placebo-controlled, multicentre trial

Càndid Villanueva, Agustín Albillos, Joan Genescà, Joan C Garcia-Pagan, José L Calleja, Carles Aracil, Rafael Bañares, Rosa M Morillas, María Poca, Beatriz Peñas, Salvador Augustin, Juan G Abraldes, Edilmar Alvarado, Ferran Torres, Jaume Bosch*†*

Decompensation or death
17% in BB group vs 27% in placebo



Villanueva C et al. Lancet. 2019. Apri 20;393(10181): 1597-1608.

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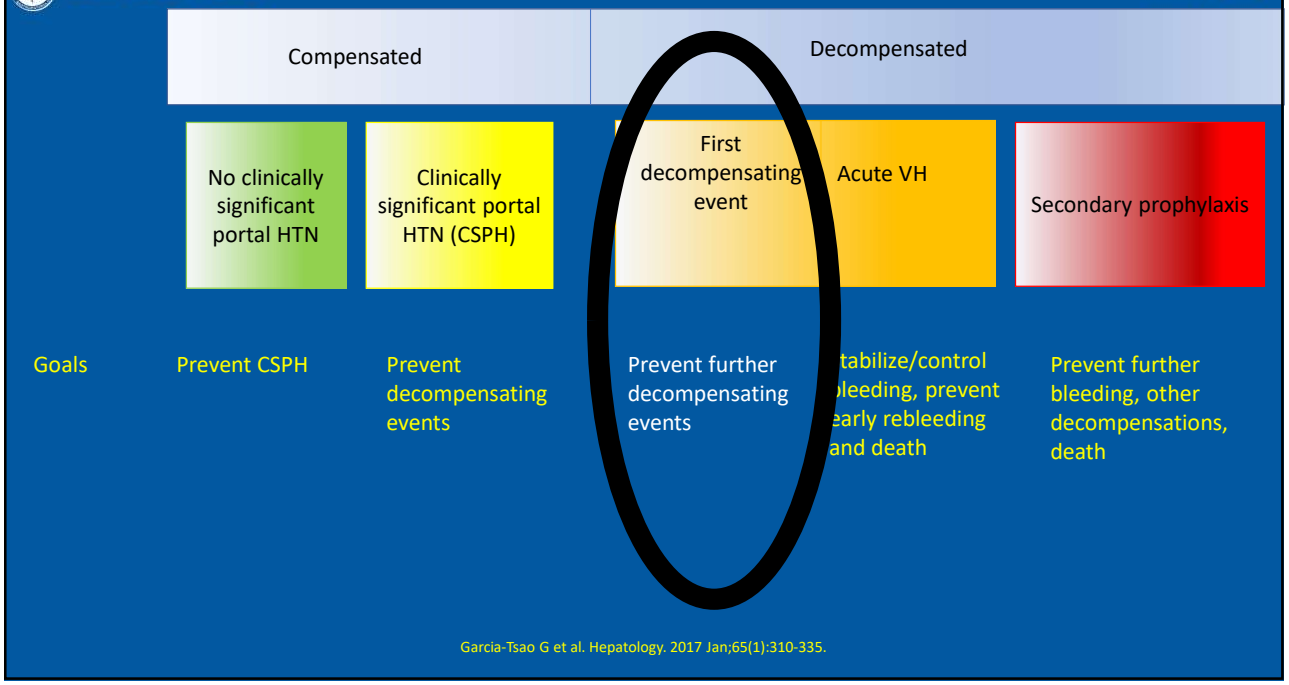

Garcia-Tsao G et al. Hepatology. 2017 Jan;65(1):310-335.

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Prevention of Variceal Bleed in Decompensated Patients (who haven't bled)

- Need screening EGD if not on NSBB
- Ascites + low-risk varices (<5mm, no red wale signs) NSBB can be utilized for prevention
- Ascites + high-risk varices (≥ 5mm, red wale signs) NEED prevention
  - NSBB preferred over EVL
- Dose reduce or d/c NSBB
  - in those with ascites and hypotension (SBP <90 mmHg)
  - ascites + AKI/HRS

de Franchis et al. J Hepatol. 2022 Apr;76(4): 959-974

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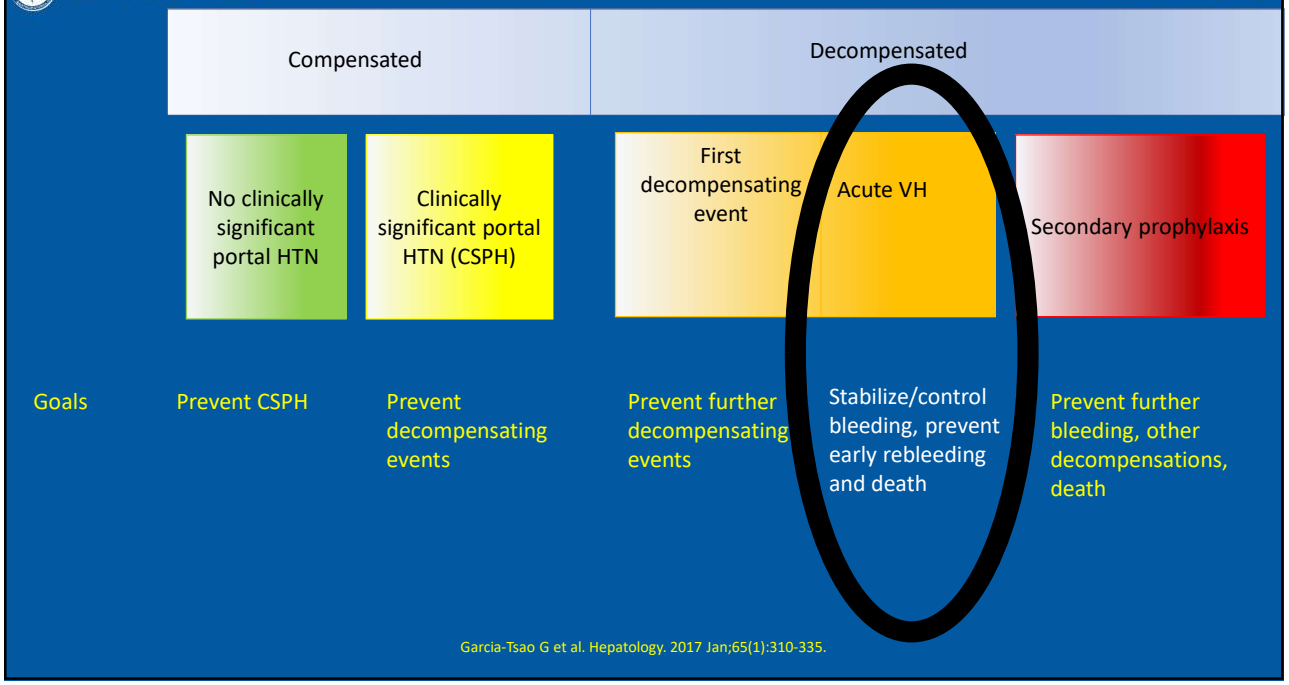

Garcia-Tsao G et al. Hepatology. 2017 Jan;65(1):310-335.

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Active Variceal Bleeding

- Resuscitation
  - Target hemoglobin between **7-8** g/dl
  - Transfusion of FFP not recommended ★
- Aim of treatment = lowering portal pressure not correcting coagulation abnormalities
- Recombinant Factor VIIa and TXA not recommended ★
- Ceftriaxone 1 g/24 hour
- Vasoactive drugs for 2-5 days, then NSBB titrated to HR 55-60

de Franchis et al. J Hepatol. 2022 Apr;76(4): 959-974

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Active Variceal Bleeding

- EGD within 12 hours of presentation
  - EVL preferred for esophageal varices
  - EVL or cyanoacrylate for GOV1
  - Cyanoacrylate for GOV2
  - **NO** hemostatic powder ★
- Start oral nutrition ASAP
- Caution with airway manipulation (NG Tubes)
- PHG and GAVE can be treated with APC, RFA, or band ligation
- All patients should undergo imaging ★

de Franchis et al. J Hepatol. 2022 Apr;76(4): 959-974

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Acute Variceal Bleeding

- Refractory bleeding? SEMS or balloon tamponade as bridge to TIPS
- PFTE-covered TIPS goal
  - Target portal pressure gradient < 12 mmHg **OR**
  - A reduction of pre-TIPS gradient by 50%
- GOV2/IGV1/IGV2 can consider BRTO
- TIPS + embolization
- Preemptive TIPS for EV and GOV1/GOV2 AND
  - CPT C
  - CPT B with active bleeding at initial EGD
  - HVPG ≥20 mmHg at time of bleeding

de Franchis et al. J Hepatol. 2022 Apr;76(4): 959-974

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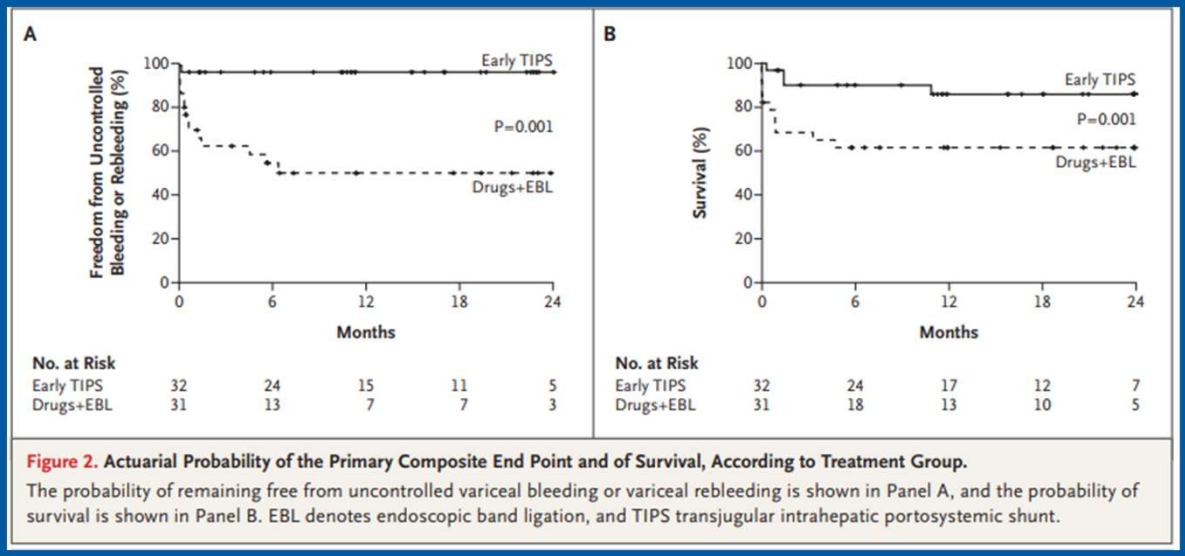

Figure 2. Actuarial Probability of the Primary Composite End Point and of Survival, According to Treatment Group.
The probability of remaining free from uncontrolled variceal bleeding or variceal rebleeding is shown in Panel A, and the probability of survival is shown in Panel B. EBL denotes endoscopic band ligation, and TIPS transjugular intrahepatic portosystemic shunt.

García-Pagán et al. N Engl J Med. 2010 Jun 24;362(25):2370-9

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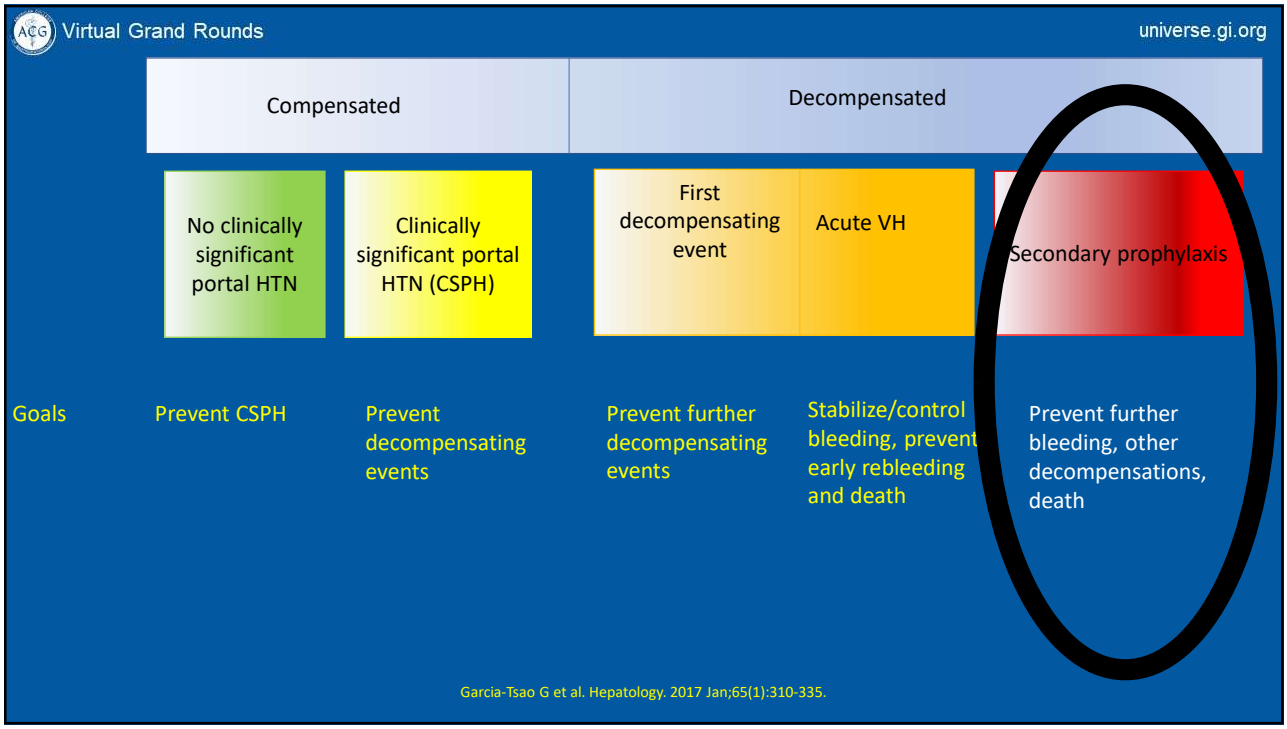
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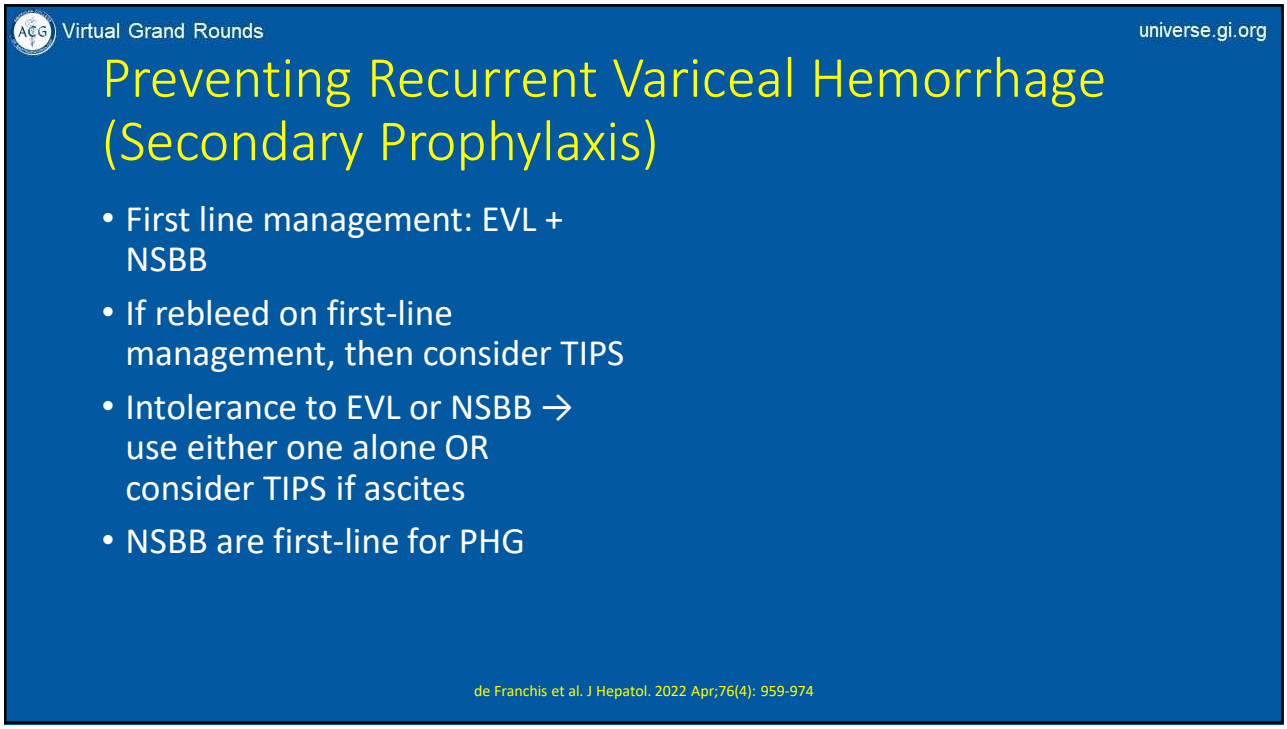
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Preventing Recurrent Variceal Hemorrhage (Secondary Prophylaxis)
• First line management: EVL + NSBB
• If rebleed on first-line management, then consider TIPS
• Intolerance to EVL or NSBB → use either one alone OR consider TIPS if ascites
• NSBB are first-line for PHG
de Franchis et al. J Hepatol. 2022 Apr;76(4): 959-974

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Other Interventions

- Interventional Radiology
  - TIPS
  - DIPS
  - BRTO
  - PARTO
  - CARTO
  - BATO
- Advanced Endoscopy
  - EUS-guided injection of coils and cyanoacrylate for gastric varices

Bhat Y et al. Gastrointest Endosc. 2016 Jun;83(6):1164-72

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Bleeding from Other Varices

- Proximal esophageal varices
  - Relieve SVC obstruction
- Gastric varices due to splenic vein thrombosis
  - Splenectomy
- Rectal Varices
  - TIPS

Rhoades DP et al. Clin Gastroenterol Hepatol. 2016. Sep;14(9):e105-6

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Take-Home Points

- Goal for patients with cirrhosis is to prevent any decompensation
- Use TE and/or platelet count to rule out CSPH
- For patients with cirrhosis and signs of CSPH use of a NSBB is recommended to prevent ANY decompensation
- No need to perform EGD in compensated patient with cirrhosis on NSBB
- Goal hemoglobin for variceal bleed is between 7-8 g/dL.
- Perform EGD within 12 hours

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Take-Home Points

- Consider preemptive TIPS in patients at high-risk for failure of endoscopic therapy
  - CPT C
  - CPT B with active bleeding at initial EGD
  - HVPG ≥20 mmHg at time of bleeding

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Portal Vein Thrombosis & Hepatic Vein Thrombosis

Arnab Mitra, MD
Assistant Professor of Medicine
Division of Gastroenterology and Hepatology, School of Medicine
Oregon Health & Science University

OREGON HEALTH & SCIENCE UNIVERSITY

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*LO- "*Recognize **diagnostic**, **surveillance**, and **treatment** strategies for portal and hepatic vein thrombosis…*"*

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Why?

Evolving Understanding of Liver & Coagulation

PVT- Challenging Clinical Conundrum

BCS- ALF, Easy to Miss

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Evolving Appreciations of Liver & Coagulation

Increasing Data that Liver Disease is a Thrombotic Risk Factor

Cirrhosis= RF for Venous Thrombosis

Liver Disease Patients are NOT "Auto-Anticoagulated"

Rebalanced Hemostasis: More Vulnerable to External Factors, esp. with Decompensation

Garbuzenko- WJH 2017;9:1197

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Liver Disease associated with VTE

- Meta-Analysis
  - 11 studies
  - N= 695,000 vs. 1,494,660 controls
  - RR= 1.7
    - DVT 2.0
    - PE 1.65

- Danish Population Case-Control study
  - RR of DVT doubled with cirrhosis and with non-cirrhotic liver disease

Throm Hae 2017;117:139

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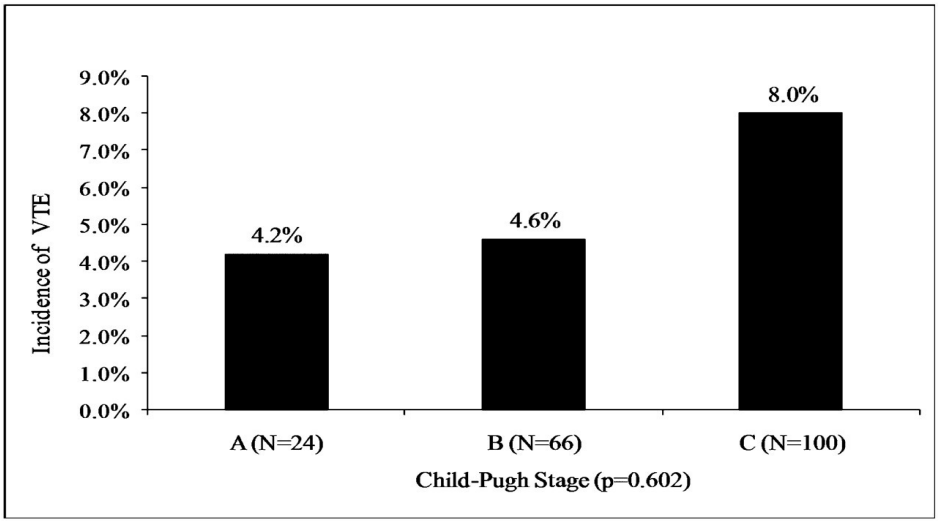
Cirrhosis = Risk for VTE based on Child-Pugh Stage



Dabbagh, Chest 2010;137:1145

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Precarious Coagulation Balance in Liver Disease

	ANTICOAGULANTS				PROCOAGULANTS			
	Platelets	Fibrinogen	Vit K	Protein C	Plasminogen	vWF/FVIII	Antithrombin	D-Dimer
Child A	↓	▬	▬	↓	↓	↑	↓	↑
Child B	↓↓↓	↓	↓	↓↓	↓↓	↑↑↑	↓↓	↑↑
Child C	↓↓	↓↓↓	↓↓	↓↓↓	↓↓↓	↑↑↑↑	↓↓↓	↑↑↑↑

DIC
Shock

Infection
AKI/Uremia

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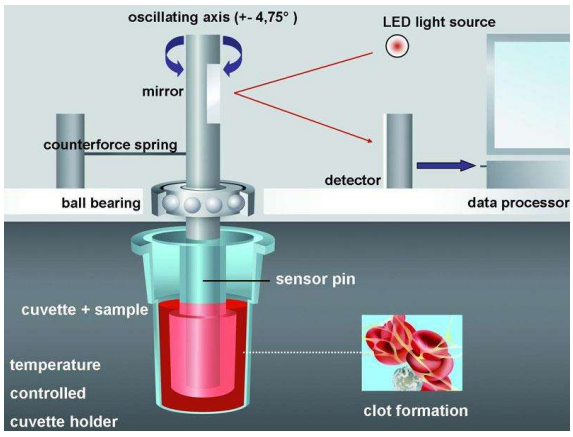
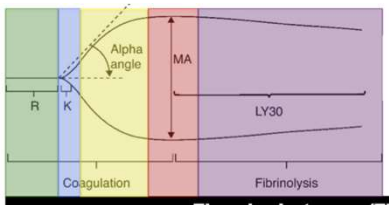
Coagulation Testing- PT/INR + PLT vs. TEG





Thromboelastography (TEG)

Thromboelastogram (TEG)

Components	Definition	Normal Values	Problem with...	Treatment
R Time	Time to start forming clot	5 – 10 minutes	Coagulation Factors	FFP
K Time	Time until clot reaches a fixed strength	1 – 3 minutes	Fibrinogen	Cryoprecipitate
Alpha angle	Speed of fibrin accumulation	53 – 72 degrees	Fibrinogen	Cryoprecipitate
Maximum Amplitude (MA)	Highest vertical amplitude of the TEG	50 – 70 mm	Platelets	Platelets and/or DDAVP
Lysis at 30 Minutes (LY30)	Percentage of amplitude reduction 30 minutes after maximum amplitude	0 – 8%	Excess Fibrinolysis	Tranexemic Acid and/or Aminocaproic Acid

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Observations

Bleeding- more often due to mechanical injury and portal HTN than coagulation status

Systemic Bleeding is Rare (ie. CNS)

Limited Data → Expert Opinion PG

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Anticoagulation Options

Antiplatelet
- To be avoided
- Raised risk of GIB

Warfarin
- Old favorite
- Difficult to control
- Slow onset/offset
- Monitoring challenges with INR

LMWH
- No monitoring
- Stable dosing
- Quick on/off

DOAC
- Stable Dosing
- No monitoring
- Few DDI
- Can Reverse
- Many have liver metabolism

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DOAC & Liver Disease

Growing Safety, Efficacy Data

Dabigatran- CPT A
Apixaban- CPT A, B
Rivaroxaban- CPT A

CPT C
- Case by Case

Hematology Consultation Helpful

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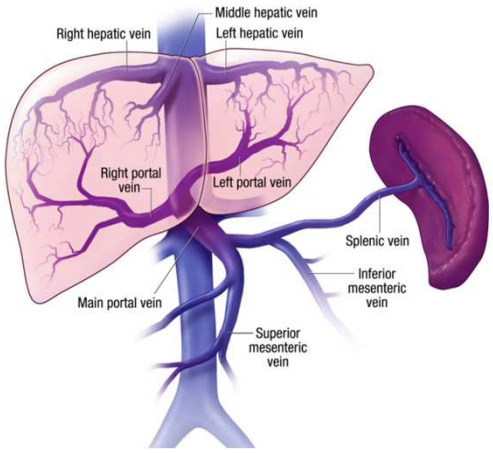
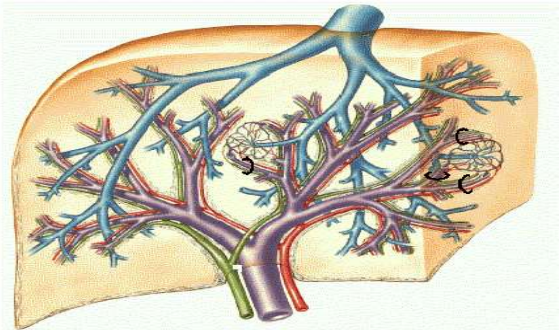
Anatomy





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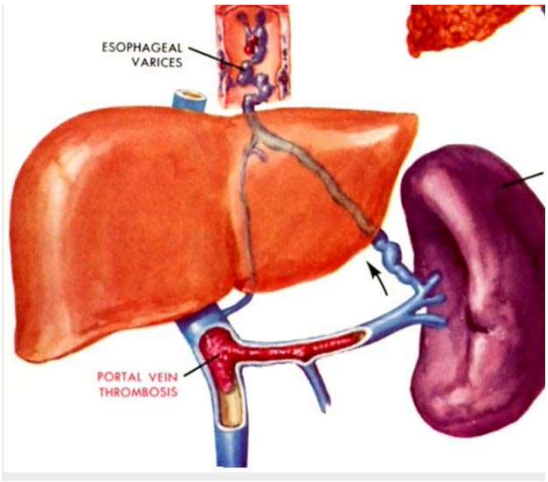
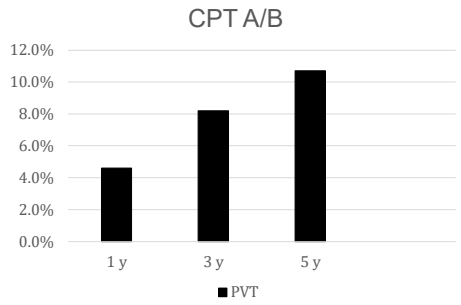
Portal Vein Thrombosis (PVT)





- Virchow's Triad
  - Inc c severity of PHTN
- Variable prevalence reported
  - 5% of LTx candidates at listing

Nery, Hep 2015;61:660
Violi, Int Emerg Med 2016;11:1059
Saidi, Int J Org Tx 2012;3:105

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PVT- What Not to Miss

Life Threatening:
Mesenteric Vein Thrombosis ~ Ischemia

Poor prognosis:
HCC extension into PV

Impact on Transplant Candidacy

Prothrombotic State

ACG PG 2020
EASL PG 2016
Intagliata, Thromb Haemost 2018;118:1491

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PVT- Making the Diagnosis

Often the Dx Comes Unexpectedly, Incidentally

CT/MRI Needed to Characterize PVT

PVT

Acute vs. Chronic

Complete (Occlusive) vs. Partial (Incomplete)

Extension into SMV

LTx Candidate?

HCC?

Infection?

Thrombophilia?

US + Doppler

CT/MRI

ACG PG 2020

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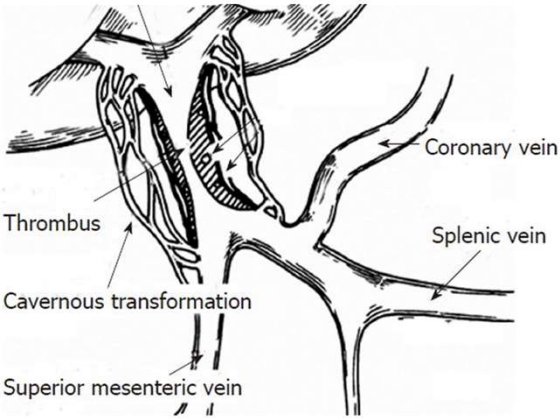
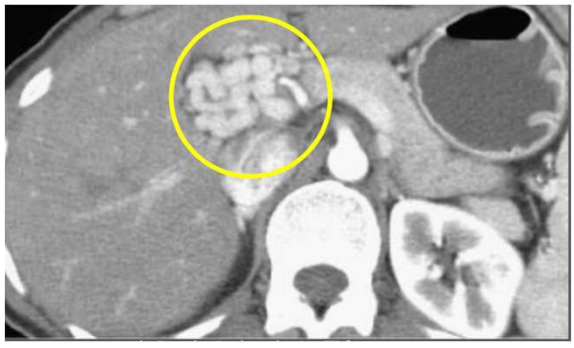
Chronic = Cavernoma





Zhang, WJG 2011;17:4334

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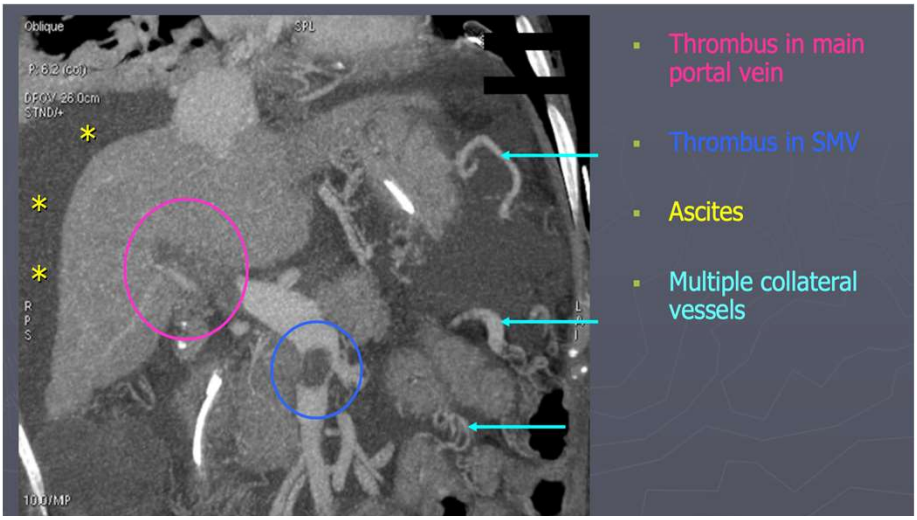
PVT + SMVT



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PVT Challenge

Limited Studies
Multiple Guidelines- ACG, AASLD, EASL

Determining Acute vs. Chronic

Perceived Risk of Anticoagulation

ACG PG 2020
EASL PG 2016
Intagliata, Thromb Haemost 2018;118:1491

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PVT- Management Goals

Reverse Thrombosis

- Prevent progression to SMVT, ischemia
- Achieve PV recanalization
- Maintain Tx candidacy

Prevent Complications:

- Variceal bleeding
- Portal Cholangiopathy
- Recurrent thrombosis

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PVT- Who to Anticoagulate?

Yes	No
• Noncirrhotic • Acute • Symptomatic • Occlusive, Main • SMVT • Thrombophilia • LTx Candidate	• High risk of bleeding~ Large varices that have not been Rx • Nonadherence • Underlying poor prognosis • Poor functional status, comorbidities

? Chronic PVT with cavernous transformation- Case by Case

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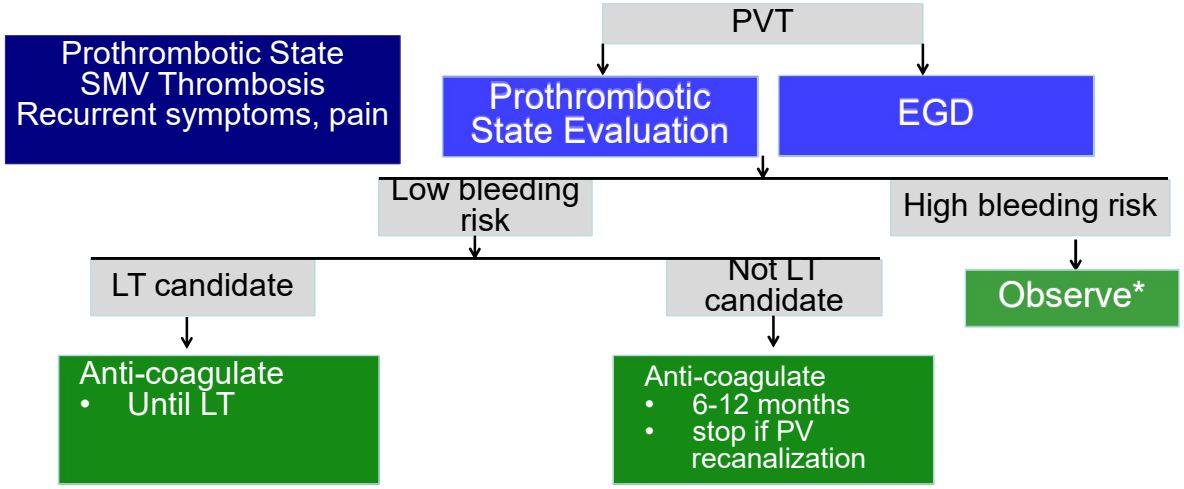
Portal Vein Thrombosis (PVT)—Cirrhosis



*Unless you consult a hematologist (anticoagulation will be recommended)

Courtesy of Dr. Scott Naugler

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Anticoagulation

Duration- 3-6 mo (until recanalization or LT)
Indefinite- thrombophilia, SMVT, recurrent thrombosis, ischemia

No INCREASE risk of bleeding on AC
- Meta-analysis
- ACG PG 2020

Prognosis, Natural History Unclear

AC → TIPS- rescue option

Loffredo, Gastro 2017;153:480

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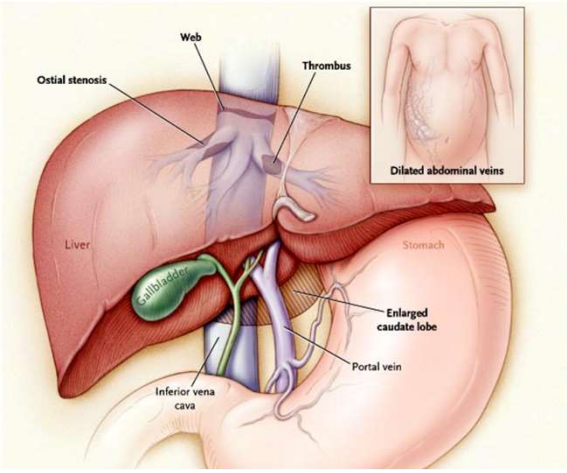
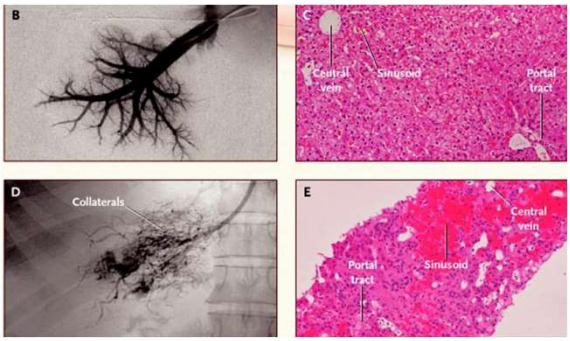
Hepatic Vein Thrombosis (HVT) ~ Budd Chiari Syndrome (BCS)





Menon, NEJM 2004, 350:578

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BCS

Hepatic Vein outflow obstruction @ HV, IVC or RA excluding SOS/VOD, cardiac disorders

- Primary
  Originates from endoluminal venous lesion
  - Thrombosis
  - Webs

- Secondary
  Originates from lesion outside the venous system
  - HCC, tumors, Cyst
  - Abscess
  - NRH- large nodules
  - Trauma → hematoma

Janssen, J Hep 2003
Menon, NEJM 2004

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BCS- Variable Presentation

1	Acute Liver Failure
2	Abnormal Liver Enzymes
3	Acute on Chronic Liver Failure
4	ESLD

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BCS- Risk Factors

Underlying thrombophilia disorder in > 75%

Multiple Risk Factors in > 25%

OCPs, Pregnancy

Table 1. Causes of the Budd–Chiari Syndrome.

- **Common causes**
  - Hypercoagulable states
    - Inherited
      - Antithrombin III deficiency
      - Protein C deficiency
      - Protein S deficiency
      - Factor V Leiden mutation
      - Prothrombin mutation
    - Acquired
      - Myeloproliferative disorders
      - Paroxysmal nocturnal hemoglobinuria
      - Antiphospholipid syndrome
      - Cancer
      - Pregnancy
      - Use of oral contraceptives
- **Uncommon causes**
  - Tumoral invasion
    - Hepatocellular carcinoma
    - Renal-cell carcinoma
    - Adrenal carcinoma
  - Miscellaneous
    - Aspergillosis
    - Behçet's syndrome
    - Inferior vena caval webs
    - Trauma
    - Inflammatory bowel disease
    - Dacarbazine therapy
  - Idiopathic

ACG PG 2020
EASL PG 2016

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BCS- Diagnosis

US
- First Line
- Caudate lobe enlargement, narrowing/lack of visualization of HV, Collaterals

CT/MRI
- Confirmatory
- Evaluate for features of cirrhosis, masses

Venogram
- Pursue if clinical suspicion remains
- Concomitant liver biopsy can be considered but rarely needed

ACG PG 2020
EASL PG 2016

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BCS- Management Goals

Reverse Thrombosis
- Prevent progression
- Decompress liver
- Achieve recanalization
- Maintain Tx candidacy

Treatment Duration- Indefinite

Prevent Complications:
- Variceal bleeding
- Recurrent thrombosis

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BCS- Management

Medical treatment

- Anticoagulate ASAP
  - Bleeding risk stratification: EGD
- Rx underlying condition
- Rx complications of portal HTN
- Assess for potential IR candidacy

ACG PG 2020
EASL PG 2016

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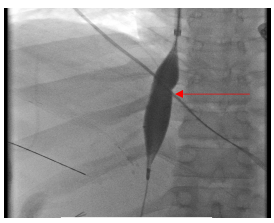
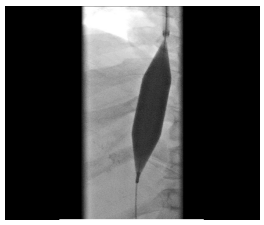
BCS- Management

Medical treatment

Angioplasty/stenting/thrombolysis





- Thrombolysis experience limited
- Angioplasty/stents for discrete, focal lesions or short-length stenosis
  ~ < 10% in US are candidates

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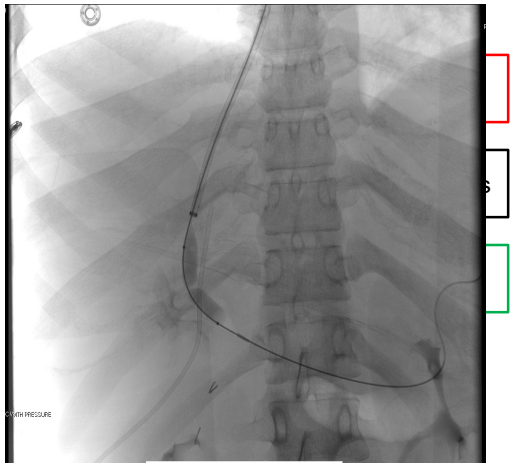
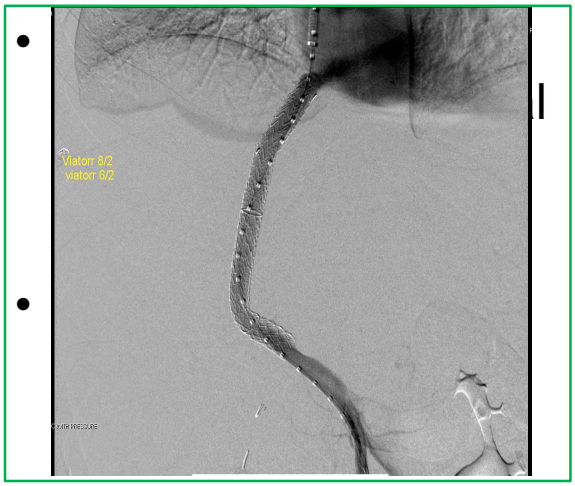
BCS- Management





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BCS- Management

Medical treatment

Angioplasty/stenting/thrombolysis

TIPS

Liver transplant

- Similar overall survival to non-BCS candidates
- BCS may recur → continue anticoagulation

Segev, Liv Tx 2007;13:1285
Mentha, J Hep 2006;44:520

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Take Aways

Cirrhosis is a Thrombotic Risk Factor

PVT- Don't Miss SMVT, HCC

BCS- Variable Presentation- Easy to Miss

Stepwise BCS Rx

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Give thanks. Give life.

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Acute Kidney Injury in Cirrhosis: Everything is not Hepatorenal Syndrome

Patricia Bloom MD
Assistant Professor
University of Michigan

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- **Objective**: Identify and differentiate types of acute kidney injury in patients with cirrhosis

Agenda

- Why does type of acute kidney injury matter?
- What is hepatorenal syndrome?
- Can hepatorenal syndrome overlap with other etiologies?
- How to differentiate types of acute kidney injury in cirrhosis?
- How to manage acute kidney injury in cirrhosis?

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Acute Kidney Injury Carries a Poor Prognosis

- 20-50% of inpatients have acute kidney injury
- Kidney injury in cirrhosis → 7-fold increase in mortality
- Prevalence of acute kidney injury in cirrhosis is increasing
- Type of acute kidney injury has a **BIG** impact on management

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Definition of Acute Kidney Injury

- **Modified KDIGO Definition of AKI:**
  - ↑ serum creatinine ≥ 0.3 mg/dL within 48 hours
  - **OR** ↑ serum creatinine ≥ 1.5 times baseline (from prior 3 months)
- **Stages**:
  - **1**: ↑ creatinine ≥ 0.3 mg/dL **OR** ↑ creatinine 1.5-2 times baseline
  - **2**: ↑ creatinine 2-3 times baseline
  - **3**: ↑ creatinine >3 times baseline **OR** creatinine ≥ 4 mg/dL **OR** renal replacement therapy

Angeli et al, J Hepatology, 2015

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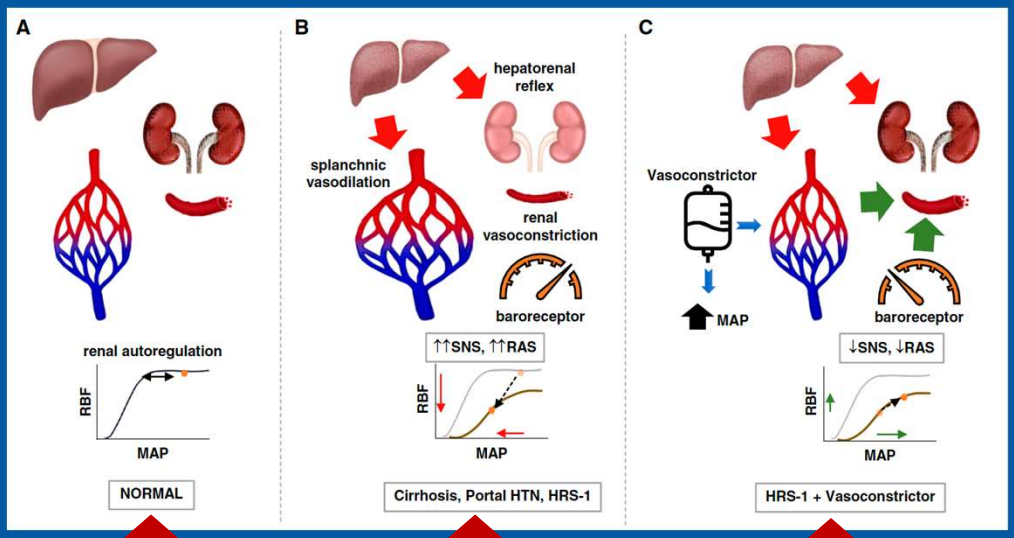
HRS Physiology



Velez, [illegible]ney360, 2022

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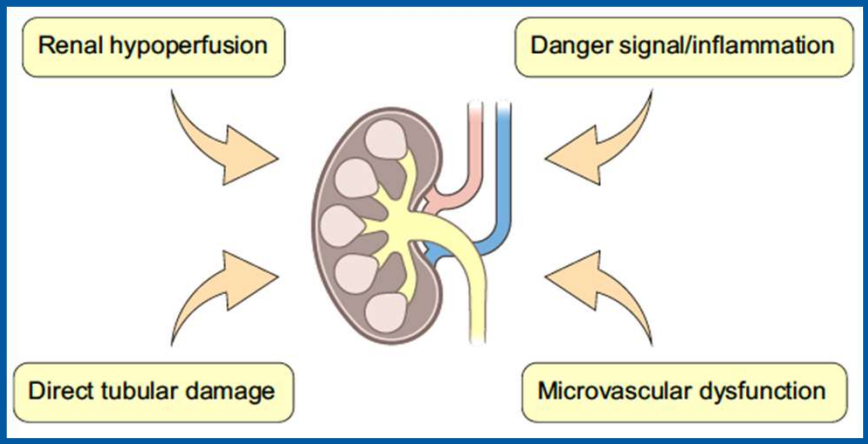
HRS Physiology



Angeli et al. J Hepatology, 2019

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Definition of Hepatorenal Syndrome

Diagnostic criteria

- Cirrhosis; acute liver failure; acute-on-chronic liver failure
- Increase in serum creatinine ≥0.3 mg/dl within 48 h or ≥50% from baseline value according to ICA consensus document
  and/or
  Urinary output ≤0.5 ml/kg B.W. ≥6 h*
- No full or partial response, according to the ICA consensus document[20], after at least 2 days of diuretic withdrawal and volume expansion with albumin. The recommended dose of albumin is 1 g/kg of body weight per day to a maximum of 100 g/day
- Absence of shock
- No current or recent treatment with nephrotoxic drugs
- Absence of parenchymal disease as indicated by proteinuria >500 mg/day, microhaematuria (>50 red blood cells per high power field), urinary injury biomarkers (if available) and/or abnormal renal ultrasonography**.
  Suggestion of renal vasoconstriction with FENa of <0.2% (with levels <0.1% being highly predictive)

Issues

Risk of pulmonary edema
Delays HRS treatment

RBC > 50 not highly sensitive or specific
Most have low MAP
Without biopsy, hard to tell if antibiotic is cause

Chronic proteinuria doesn't necessarily exclude HRS

Renal imaging challenging with ascites

No urine microscopy!

Angeli et al, J Hepatology, 2019

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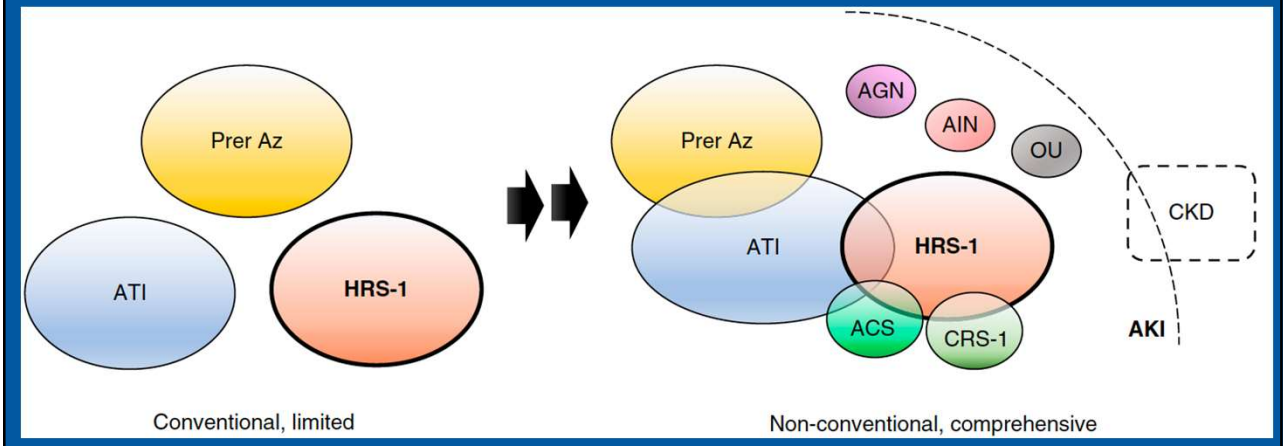
HRS Can Overlap with Other Etiologies



Velez, Kidney360, 2022

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No Excellent Biomarkers to Differentiate AKI Types

(not talking about biomarkers to identify AKI, like Cystatin C)

- Major Issue: Lack of gold standard
- Urinary neutrophil gelatinase-associated lipocalin (NGAL)

Type of AKI	Physiology	NGAL level
Pre-renal	No tubular injury	Low NGAL
Hepatorenal Syndrome	↑ renal vasoconstriction	Intermediate NGAL
Acute Tubular Necrosis	Ischemic injury	High NGAL

  - NGAL cut-off discriminated ATN vs. non-ATN (multiple studies)
  - NGAL predicts response to HRS therapy (1 abstract)
  - NGAL improves MELD accuracy of mortality prediction in decompensation

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No Excellent Biomarkers to Differentiate AKI Types

- **Fractional excretion of sodium (FENa)**
  - Most decompensated cirrhosis: FENa < 1% (even in ATN)
  - FENa cut-off of 0.1 or 0.2% better in cirrhosis (below = HRS; above = ATN)
- **Fractional excretion of urea (FEUrea)**
  - Single center: FEUrea >28.1% has positive predictive value of 89% for exclusion of HRS-1
  - Needs further research

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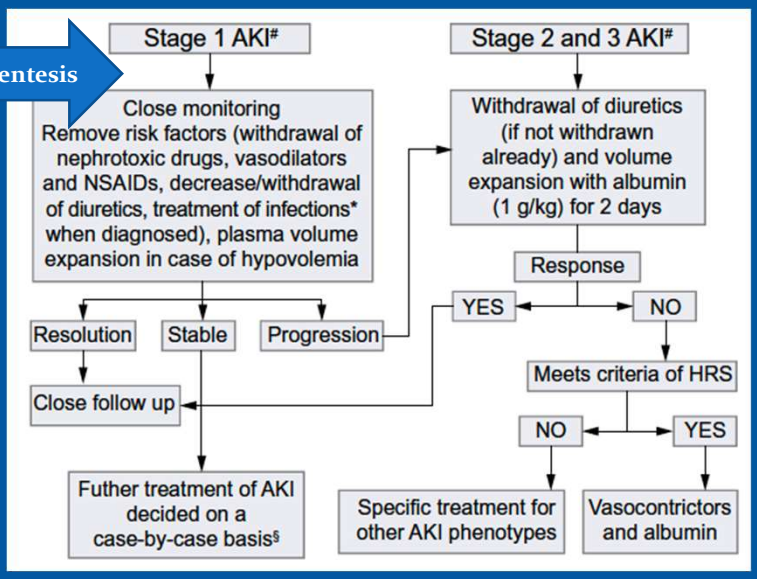
International Ascites Club Approach to AKI in Cirrhosis



Angeli et al, J Hepatology, 2015

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Large Volume Paracentesis in Acute Kidney Injury

- Paracentesis-induced circulatory dysfunction is ↓ with albumin
- High intra-abdominal pressure (≥ 20 mmHg) can compress renal vein
- Some studies have shown rise in GFR and urine output after large volume paracentesis

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Key Points

- Critical to identify and treat acute kidney injury in cirrhosis
- Important to attempt identifying etiology, as treatment varies
- However, can have more than 1 cause, and complicated to disentangle
- Hepatorenal syndrome is treated with albumin and vasoconstrictors

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EXTRA SLIDES

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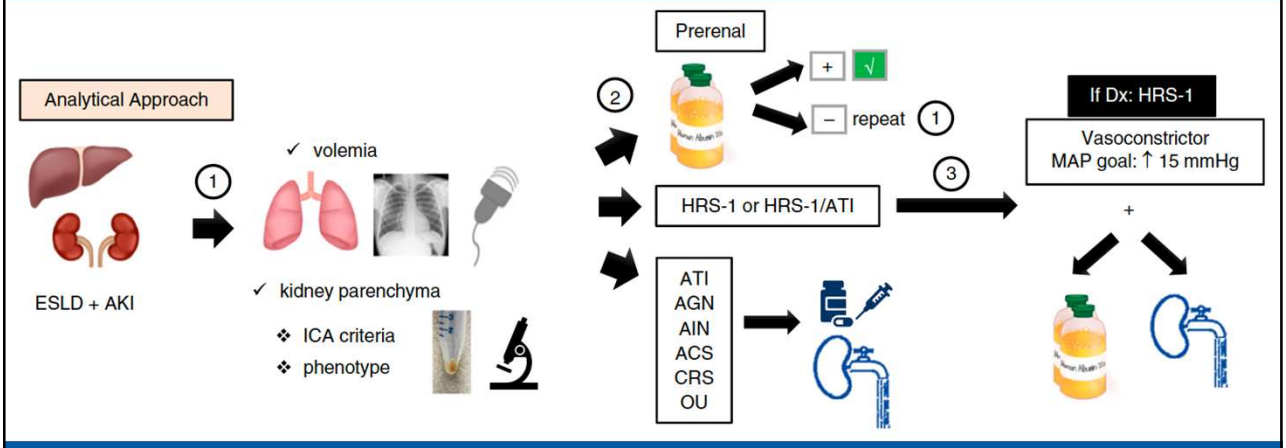
HRS Treatment: Alternative Approach



Velez, Kidney360, 2022

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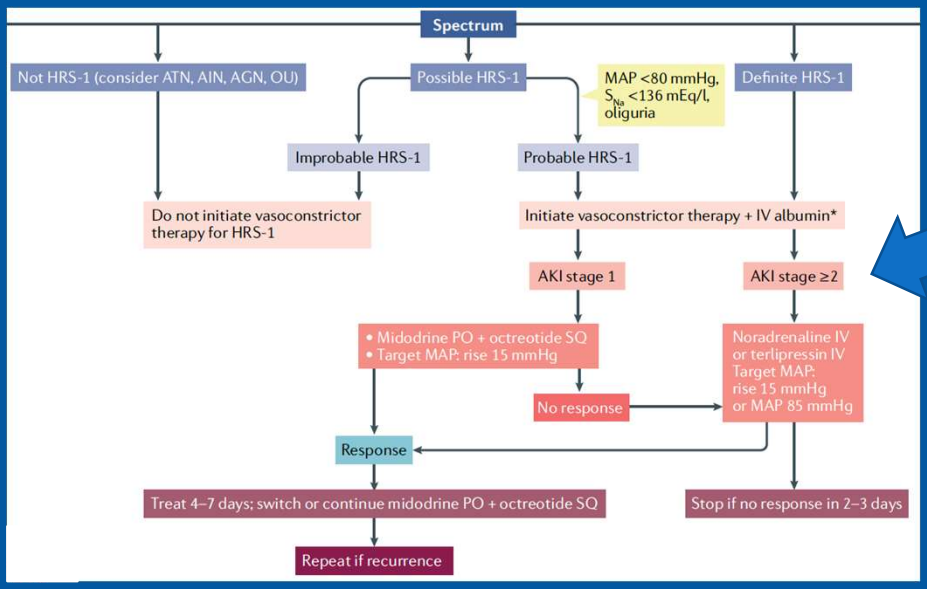
HRS Treatment



Adapted from Velez et al, Nat Reviews, 2020

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Questions

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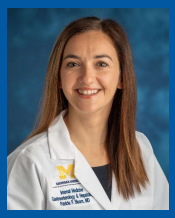

Patricia L. Bloom, MD

Uchenna A. Agbim, MD

Arnab Mitra, MD

**All of the relevant financial relationships listed for these individuals have been mitigated*

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