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**Participating in the Webinar**

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.
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
Recent Updates in the Management of Varices
Uchenna Agbim, MD
Assistant Professor
Saint Louis University

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
Outline

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

Pathophysiology

• Intrahepatic resistance
• Portal venous blood flow
• HVPG > 5 portal hypertension
• HVPG > 10mmHg = Clinically significant portal hypertension
Pathophysiology and Natural History

<table>
<thead>
<tr>
<th>Compensated</th>
<th>Decomposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>No clinically significant portal HTN</td>
<td>Clinically significant portal HTN (CSPH)</td>
</tr>
<tr>
<td>First decompensating event</td>
<td>Acute VH</td>
</tr>
<tr>
<td>Prevent CPH</td>
<td>Prevent further decompensating events</td>
</tr>
<tr>
<td>Prevent early rebleeding and death</td>
<td>Prevent further decompensating events</td>
</tr>
<tr>
<td>Stabilize/control bleeding, other decompensations, death</td>
<td></td>
</tr>
</tbody>
</table>

Goals

Prevent CSPH

Prevent decompensating events

Prevent further decompensating events
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 ≤ 150×10^9 and cannot initiate NSBB → Screening EGD
Compensated Decompensated

No clinically significant portal HTN (CSPH) 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

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
Prevention of Variceal Bleed in Compensated Patients with CSPH

Why use NSBB to prevent any Decompensation?

<table>
<thead>
<tr>
<th>Compensated</th>
<th>Decompensated</th>
</tr>
</thead>
<tbody>
<tr>
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<td>Secondary prophylaxis</td>
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</tr>
<tr>
<td>Goals</td>
<td>Prevent CSPH</td>
</tr>
<tr>
<td>Prevent further decompensating events</td>
<td>Stabilize/control bleeding, prevent early rebleeding and death</td>
</tr>
<tr>
<td>Prevent further bleeding, other decompensations, death</td>
<td></td>
</tr>
</tbody>
</table>


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

Compensated
- No clinically significant portal HTN
- Clinically significant portal HTN (CSPH)

Goals
- Prevent CSPH
- Prevent decompensating events

Decompensated
- First decompensating event
- Acute VH
- Secondary prophylaxis
- Prevent further bleeding, other decompensations, death
- Prevent CSPH
- Prevent further decompensating events
- Stabilize/control bleeding, prevent early rebleeding and death

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

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
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

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

Other Interventions

• Interventional Radiology
  • TIPS
  • DIPS
  • BRTO
  • PARTO
  • CARTO
  • BATO

• Advanced Endoscopy
  • EUS-guided injection of coils and cyanoacrylate for gastric varices


Bleeding from Other Varices

• Proximal esophageal varices
  • Relieve SVC obstruction

• Gastric varices due to splenic vein thrombosis
  • Splenectomy

• Rectal Varices
  • TIPS

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

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
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

LO- “Recognize diagnostic, surveillance, and treatment strategies for portal and hepatic vein thrombosis...”
Why?

Evolving Understanding of Liver & Coagulation

PVT- Challenging Clinical Conundrum

BCS- ALF, Easy to Miss

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
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

Cirrhosis = Risk for VTE based on Child-Pugh Stage

![Graph showing incidence of VTE](image)

- **A** (N=24) 4.2%
- **B** (N=66) 4.6%
- **C** (N=100) 8.0%

Dabbagh, Chest 2010;137:1145
### Precarious Coagulation Balance in Liver Disease

<table>
<thead>
<tr>
<th>Platelets</th>
<th>Fibrinogen</th>
<th>Vit K</th>
<th>Protein C</th>
<th>Plasminogen</th>
<th>vWF/FVIII</th>
<th>Antithrombin</th>
<th>D-Dimer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child B</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Child C</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Coagulation Testing - PT/INR + PLT vs. TEG**

<table>
<thead>
<tr>
<th>Component</th>
<th>Definition</th>
<th>Normal Values</th>
<th>Problem with...</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTT</td>
<td>Time to start forming clot</td>
<td>5 – 10 minutes</td>
<td>Coagulation</td>
<td>FFP</td>
</tr>
<tr>
<td>PT</td>
<td>Time until clot reaches a fixed</td>
<td>1 – 3 minutes</td>
<td>Factors</td>
<td></td>
</tr>
<tr>
<td></td>
<td>strength</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alpha angle</td>
<td>Speed of fibrin accumulation</td>
<td>50 – 70 degrees</td>
<td>Fibrinogen</td>
<td>Cryoprecipitate</td>
</tr>
<tr>
<td>Maximum Amplitude</td>
<td>Highest vertical amplitude of the</td>
<td>50 – 70 mm</td>
<td>Platelets</td>
<td></td>
</tr>
<tr>
<td>(MA)</td>
<td>TEG</td>
<td></td>
<td>Platelets and/or</td>
<td></td>
</tr>
<tr>
<td>Lytic at 30 minutes</td>
<td>Percentage of amplitude</td>
<td>0 – 8%</td>
<td>Excess fibrinolyse</td>
<td></td>
</tr>
<tr>
<td>(LY30)</td>
<td>reduction 30 minutes after</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Thromboelastography (TEG)**

- MA: Maximum Amplitude
- LY30: Lytic at 30 minutes
- FFP: Fresh Frozen Plasma
- Cryoprecipitate
- Excess fibrinolysis
- Transeserin-Acid and/or Aminocaproic Acid
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

Anticoagulation Options

- **Antiplatelet**
  - To be avoided
  - Raised risk of GIB

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

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

- **DOAC**
  - Stable Dosing
  - No monitoring
  - Few DDI
  - Can Reverse
  - Many have liver metabolism
Growing Safety, Efficacy Data

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

Hematology Consultation Helpful

CPT C
- Case by Case

Anatomy
Portal Vein Thrombosis (PVT)

- Virchow’s Triad
- Increased severity of PHTN
- Variable prevalence reported
  - 5% of LTx candidates at listing

Life Threatening:
Mesenteric Vein Thrombosis ~ Ischemia

Poor prognosis:
HCC extension into PV

Impact on Transplant Candidacy
Prothrombotic State

Nery, Hep 2015;61:660
Saidi, Int J Org Tx 2012;3:105

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

Chronic = Cavernoma

Zhang, WJG 2011;17:4334

American College of Gastroenterology
PVT + SMVT

- Thrombus in main portal vein
- Thrombus in SMV
- Ascites
- Multiple collateral vessels

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
**PVT- Management Goals**

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

**Prevent Complications:**
- Variceal bleeding
- Portal Cholangiopathy
- Recurrent thrombosis

---

**PVT- Who to Anticoagulate?**

**Yes**
- Noncirrhotic
- Acute
- Symptomatic
- Occlusive, Main
- SMVT
- Thrombophilia
- LTx Candidate

**No**
- 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
Portal Vein Thrombosis (PVT)—**Cirrhosis**

- **Prothrombotic State Evaluation**
  - **SMV Thrombosis**
  - **Recurrent symptoms, pain**

  - **Low bleeding risk**
    - **LT candidate**
    - **Anti-coagulate**
      - **Until LT**

  - **High bleeding risk**
    - **Not LT candidate**
    - **Anti-coagulate**
      - **6-12 months**
      - **stop if PV recanalization**

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

**Prothrombotic State**

**PROGNOSIS, NATURAL HISTORY**

**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**

*Courtesy of Dr. Scott Naugler*
Hepatic Vein Thrombosis (HVT) ~ Budd Chiari Syndrome (BCS)

Menon, NEJM 2004, 350:578

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

Menon, NEJM 2004

Janssen, J Hep 2003

American College of Gastroenterology
BCS- Variable Presentation

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

BCS- Risk Factors

- Underlying thrombophilia disorder in > 75%
- Multiple Risk Factors in > 25%
- OCPs, Pregnancy

Table 1. Causes of the Budd-Chiari Syndrome.

<table>
<thead>
<tr>
<th>Common causes</th>
<th>Uncommon causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypocoagulable states</td>
<td>Tumoral invasion</td>
</tr>
<tr>
<td>Inherited</td>
<td>Hepatocellular carcinoma</td>
</tr>
<tr>
<td>Antithrombin III deficiency</td>
<td>Renal-cell carcinoma</td>
</tr>
<tr>
<td>Protein C deficiency</td>
<td>Adrenal carcinoma</td>
</tr>
<tr>
<td>Protein S deficiency</td>
<td>Miscellaneous</td>
</tr>
<tr>
<td>Factor V Leiden mutation</td>
<td>Aspergillosis</td>
</tr>
<tr>
<td>Prothrombin mutation</td>
<td>Behçet's syndrome</td>
</tr>
<tr>
<td>Acquired</td>
<td>Inferior vena cava thrombosis</td>
</tr>
<tr>
<td>Myofibroblastic disorders</td>
<td>Trauma</td>
</tr>
<tr>
<td>Paroxysmal nocturnal hemoglobinuria</td>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td>Antiphospholipid syndrome</td>
<td>Decarboxylase deficiency</td>
</tr>
<tr>
<td>Cancer</td>
<td>Idiopathic</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Use of oral contraceptives</td>
</tr>
</tbody>
</table>
**BCS- Diagnosis**

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

- **Confirmatory**
  - Evaluate for features of cirrhosis, masses

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

---

**BCS- Management Goals**

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

**Treatment Duration- Indefinite**

**Prevent Complications:**
- Variceal bleeding
- Recurrent thrombosis
BCS- Management

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

Medical treatment

ACG PG 2020
EASL PG 2016

BCS- Management

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

Medical treatment

Angioplasty/stenting/thrombolysis

ACG PG 2020
EASL PG 2016
BCS- Management

- Lower morbidity, mortality than surgical shunts
- Bridge to Transplant

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
Cirrhosis is a Thrombotic Risk Factor
PVT- Don’t Miss SMVT, HCC
BCS- Variable Presentation- Easy to Miss

Stepwise BCS Rx
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?
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

**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
HRS Physiology

Angeli et al. J Hepatology, 2019
**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
- Urinary output ≤0.5 ml/kg B.W. ≥6 h
- No full or partial response, according to the ICA consensus document, 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

**HRS Can Overlap with Other Etiologies**

Velez, Kidney360, 2022
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
    - Pre-renal
    - Hepatorenal Syndrome
    - Acute Tubular Necrosis
  - Physiology
    - No tubular injury
    - ↑ renal vasoconstriction
    - Ischemic injury
  - NGAL level
    - Low NGAL
    - Intermediate NGAL
    - 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

- 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
International Ascites Club Approach to AKI in Cirrhosis

Paracentesis

Stage 1 AKI
- Close monitoring
- Remove risk factors (withdrawal of nephrotoxic drugs, vasodilators and NSAIDs, decrease/withdrawal of diuretics, treatment of infections when diagnosed), plasma volume expansion in case of hypovolemia

Stage 2 and 3 AKI
- Withdrawal of diuretics (if not withdrawn already) and volume expansion with albumin (1 g/kg) for 2 days
- Response
  - YES
    - Meets criteria of HRS
  - NO
    - Specific treatment for other AKI phenotypes
    - Vasocontractors and albumin

Further treatment of AKI decided on a case-by-case basis

Resolution
- Stable
- Progression
- Close follow up

Angeli et al, J Hepatology, 2015

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
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
HRS Treatment: Alternative Approach

Velez, Kidney360, 2022

HRS Treatment

Adapted from Velez et al, Nat Reviews, 2020

American College of Gastroenterology
Questions

Patricia L. Bloom, MD

Uchenna A. Agbim, MD

Arnab Mitra, MD

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