Inpatient Evaluation and Management of Patients with Cirrhosis

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I have no disclosures to make relative to my presentation
In 2017, the presidents of all 4 American GI Societies were female

Karen Woods
ASGE

Sheila Crowe
AGA

Carol Burke
ACG

Anna Lok
AASLD

In 2017, four females had been AASLD presidents

Terry Wright (2005)
Guadalupe Garcia-Tsao (2012)
Gyongyi Szabo (2015)
Anna Lok (2017)
Laurie DeLeve (2022)
Norah Terrault (2023)
Grace Su (2026)
Decompensation is the main determinant of death in cirrhosis and the main driver of decompensation is CSPH.

- Chronic liver disease
- Compensated cirrhosis
  - Median survival >12 yrs
  - Long asymptomatic stage
- Clinically significant portal hypertension (CSPH)
  - Defined as a hepatic venous pressure gradient (HVPG) ≥ 10 mmHg
- Decompensated cirrhosis
  - Median survival ~2 yrs
  - Ascites
  - Variceal hemorrhage
  - Encephalopathy
- Death

Ripoll et al. Gastroenterology 2007; 133:481.
CSPH results from increased intrahepatic resistance and increased portal venous inflow.

CSPH = clinically significant portal hypertension, HVPG = hepatic venous pressure gradient.
Variceal hemorrhage is an episodic but deadly complication of cirrhosis.
Mechanism of action of different strategies used to treat variceal hemorrhage

CSPH = clinically significant portal hypertension; EVL = endoscopic variceal ligation;
Management of variceal hemorrhage – Standard of Care (SOC)

• Cautious PRBC transfusion: start at 7 g/dL, maintain at 7-9 g/dL
• Short term (maximum 7 days) antibiotic prophylaxis (ceftriaxone 1 g/d)
• Safe IV vasoactive drug (octreotide, somatostatin or terlipressin)

Variceal bleeding is due to portal hypertension, and the aim of the treatment should be focused on lowering portal pressure rather than correcting coagulation abnormalities.

FFP transfusion is not recommended as it will not correct coagulopathy and may lead to volume overload and worsening of portal hypertension.

Garcia-Tsao et al. AASLD guidance. Hepatology 2017;65:310-335
Management of variceal hemorrhage – Standard of Care (SOC)

- Cautious PRBC transfusion: start at 7 g/dL, maintain at 7-9 g/dL
- Short term (maximum 7 days) antibiotic prophylaxis (ceftriaxone 1 g/d)
- Safe IV vasoactive drug (octreotide, somatostatin or terlipressin)

Endoscopy (within 12 hours): VH confirmed

Stop PPI

PPIs, when started before endoscopy, should be stopped immediately after endoscopy confirms variceal hemorrhage unless there is a strict indication to continue them

*Baveno VII, 2021*

Garcia-Tsao et al. AASLD guidance. Hepatology 2017;65:310-335
TIPS in acute variceal hemorrhage

- Cautious PRBC transfusion: start at 7 g/dL, maintain at 7-9 g/dL
- Short term (maximum 7 days) antibiotic prophylaxis (ceftriaxone 1 g/d)
- Safe IV vasoactive drug (octreotide, somatostatin or terlipressin)

Garcia-Tsao et al. AASLD guidance. Hepatology 2017;65:310-335

Endoscopy (within 12 hours): VH confirmed

Perform endoscopic therapy (EVL)

Continue IV vasoactive drug (2-5 days)

Uncontrolled bleeding:
- bleeding not controlled by EVL
- EVL cannot be performed because of intense bleeding

Salvage TIPS

Contraindications are not relevant

Rescue TIPS

Contraindications are not relevant

D/C IV drug, start NSBB

No bleed

Rebleed

Patient at high risk of failure of standard therapy

Pre-emptive TIPS (pTIPS)
- TIPS is placed before patient fails and needs a rescue TIPS

Contraindications are relevant

Child C patients are most likely to need rescue TIPS with high mortality
Patients excluded from pTIPS studies

- Child-Pugh score 14 and 15
- Age >70–75 years
- Recurrent overt encephalopathy without precipitating factors
- Serum creatinine above 2.5-3 g/dl
- Sepsis/active infection
- Heart failure
- Pulmonary hypertension
- HCC beyond Milan
- Complete PV thrombosis

Cardiac echo

Doppler US or cross-sectional imaging
Pre-emptive TIPS (pTIPS) placed within 72 hours of admission improves survival in Child C (10-13 points) and in selected Child B patients.

Child C 10-13 + Child B with active bleeding at endoscopy

50% Survival

P=0.001

1-year ARR = 25%

Child C (10-13) + Child B with or without active bleeding at endoscopy

22% Survival

1-year ARR = 13%

Mostly EtOH/HCV cirrhosis

No. at Risk

<table>
<thead>
<tr>
<th>Early TIPS</th>
<th>Drugs+EBL</th>
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<tbody>
<tr>
<td>32</td>
<td>31</td>
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</table>

Mostly HBV cirrhosis

ARR = absolute risk reduction

A recent additional RCT including 58 patients (29/group) did not find differences in survival and encountered problems regarding feasibility of pTIPS within the timeframe.


In an individual data meta-analysis, the groups that seem to benefit from preemptive TIPS (pTIPS) are Child C (10-13 pts) and Child B (8-9 pts).
Management of variceal hemorrhage

- Cautious PRBC transfusion: start at 7 g/dL, maintain at 7-9 g/dL
- Short term (maximum 7 days) antibiotic prophylaxis (ceftriaxone 1 g/d)
- Safe IV vasoactive drug (octreotide, somatostatin, terlipressin)

Endoscopy (within 12 hours): VH confirmed

Perform endoscopic therapy (EVL)

Not pTIPS candidate
- Child A
- Child B7 ± active bleed
- Child C 14-15

Continue IV vasoactive drug (2-5 days)

- Bleed
  - Rescue TIPS
- No bleed

Consider pTIPS

pTIPS (placed within 72 hours, i.e. “early”)
- Child C (10-13 points)
- Child B (8-9 points) + active bleed at endoscopy

pTIPS = preemptive TIPS; NSBB = nonselective beta-blockers; EVL = endoscopic variceal ligation
Ascites is the most common complication of ascites but it is a chronic event that, unless complicated, does not require hospitalization.

- Clinically significant portal hypertension (CSPH)
- Compensated cirrhosis
  - Ascites
  - VH
  - HE
- Decompensated cirrhosis
  - Ascites is not an emergency
  - Start diuretics once other complications (GI bleed, infection, acute kidney injury, encephalopathy) are absent or have resolved
  - If patient uncomfortable because of tense ascites → large-volume paracentesis
  - Main goal in a hospitalized patient is to rule out spontaneous bacterial peritonitis
  - In a non-elective admission → hold diuretics


CSPH= clinically significant portal hypertension; VH= variceal hemorrhage; HE= hepatic encephalopathy
Ascites, refractory ascites, hyponatremia and HRS represent a continuum in decompensated cirrhosis with progressive hemodynamic alterations.

Portal (sinusoidal) hypertension

↑ Systemic Inflammation

↑ Splanchnic / systemic vasodilatation

↓ ↓ Mean arterial pressure

↑ Activation of neurohumoral systems

 Fluid leaks into the peritoneal cavity

 Ascites

 Sodium retention

 Water retention

 Hyponatremia

 Refractory Ascites

 Renal vasoconstriction

 Hepatorenal syndrome

 Spontaneous bacterial peritonitis
Recurrent/refractory ascites, spontaneous bacterial peritonitis and hepatorenal syndrome define a stage of “further” decompensation.

 Clinically significant portal hypertension (CSPH)

Compensated cirrhosis → Decompensated cirrhosis → Further decompensation

- Ascites
- VH
- HE

Inflammation → Vasodilatation

Further decompensation:
- Development of a second event
- Recurrent/refractory ascites
- Hepatorenal syndrome (HRS-AKI)
- Spontaneous bacterial peritonitis
- Jaundice (liver insufficiency and/or superimposed injury)

CSPH: clinically significant portal hypertension; VH: variceal hemorrhage; HE: hepatic encephalopathy

Large-volume paracentesis (LVP) + albumin is the mainstay of therapy for recurrent / tense ascites

Cirrhosis → Portal (sinusoidal) hypertension → Systemic Inflammation → \(\downarrow\) Splanchnic / systemic vasodilatation \((\text{PCD})\) → \(\downarrow\) Effective arterial blood volume → \(\uparrow\) Activation of neurohumoral systems

- Fluid leaks into the peritoneal cavity → Ascites
- Sodium retention → Refractory Ascites
- Water retention → Hypo-natremia
- Renal vasoconstriction → \(\downarrow\) Renal blood flow → Acute kidney injury \((\text{HRS})\)

- Albumin infusion (6-8 g/L ascites removed) is recommended with LVP of >5L
- In the presence of AKI, remove smaller amounts + albumin

AASLD guidance 2021

PCD=post-paracentesis circulatory dysfunction

Is albumin alone useful??

• Albumin alone useful??

• Albumin infusion (6-8 g/L ascites removed) is recommended with LVP of >5L
• In the presence of AKI, remove smaller amounts + albumin

AASLD guidance 2021
Chronic intravenous albumin administration in outpatients with cirrhosis and ascites have yielded contradictory results.

**Overall survival**

- **Median MELD 12 (10-15)**

- **Mean MELD 17 ± 6**

- **No recommendation can be made regarding the outpatient use of albumin in routine clinical practice**

**Survival**

- **Double-placebo**

- **Albumin (40 g q 2 wks) + midodrine**

- **p = 0.298**

Also associated with lower rates of need for LVP, hyponatremia, SBP and hepatorenal syndrome.

**Caraceni et al (ANSWER trial). Lancet 2018;391:2417-2429**

**No differences in need for LVP, renal failure, hyponatremia, bacterial infections, encephalopathy or GI bleeding**

**Solà et al (MACHT trial) . J Hepatol. 2018;69:1250-1259**
The transjugular intrahepatic portosystemic shunt (TIPS) acts upstream of the ascites pathogenic cascade.

Cirrhosis

- Portal (sinusoidal) hypertension
  - Systemic Inflammation
    - Splanchnic/systemic vasodilation
    - Effective arterial blood volume
      - Activation of neurohumoral systems

Fluid leaks into the peritoneal cavity

- Sodium retention
  - Ascites
  - Refractory Ascites

TIPS
TIPS with PTFE-covered stent improves survival in patients with cirrhosis and recurrent ascites


TIPS= transjugular intrahepatic portosystemic shunt; LVP + A = large-volume paracentesis + albumin

All pts had “recurrent” tense ascites defined as requiring 2 LVP in a minimum period of 3 weeks

TIPS should be considered in selected patients who require at least three LVPs in a year despite optimal medical therapy

ALTA consensus, 2020; Baveno consensus, 2022

Probability of survival
Bacterial infections can lead to liver and extrahepatic organ failures in patients at any stage of cirrhosis.

Compensated cirrhosis → Bacterial Infections → Decompensated cirrhosis → Further decompensation → Death

- VH
- Ascites
- HE
- Recurrent VH/HE
- Refractory ascites
- Renal failure (HRS)
- Liver failure
  - Jaundice
  - Coagulopathy
  - Encephalopathy

Multiorgan failure (ACLF)

38% are “spontaneous” (SBP, SBE, SB)

Piano et al. Gastro 2019

PH= portal hypertension; VH=variceal hemorrhage; HE= hepatic encephalopathy; HRS= hepatorenal syndrome; ACLF= acute on chronic liver failure
Diagnosis of SBP (or SBE) is based on fluid (ascites, pleural) PMN

- Although patient may present with abdominal pain, tenderness, ileus, the patient with SBP is often asymptomatic and may present only with encephalopathy or AKI

- In a prospective study, 6/17 (35%) patients with SBP were deemed not to have SBP based on clinical evaluation in the emergency department


- Diagnosis is based on ascites (or hydrothorax fluid) PMNs, independent of culture results

  \[ \text{PMN} > 250/\text{mm}^3 = \text{SBP (or SBE)} \]

  In grossly hemorrhagic ascites: subtract 1 PMN per 250 RBC

*Biggins et al. AASLD guidelines. Hepatology 2021;74:1014-1048*
On admission (independent of symptoms)

Dx paracentesis

Delays in the performance of diagnostic paracentesis in SBP result in a higher mortality

SBP = spontaneous bacterial peritonitis

Start antibiotic on admission (independent of symptoms) for patients with SIRS and/or acute development of kidney injury, jaundice and/or encephalopathy.

- Paracentesis > 250 PMN
- Blood, urine cultures, chest X-ray, diagnostic paracentesis
- Creatinine > 1.0
- BUN > 30
- Bilirubin > 4.0
- IV albumin

**Community-acquired SBP**

**Nosocomial SBP**

- Piperacillin/tazobactam AND
- Daptomycin (if VRE in past or GI colonization) OR
- Meropenem if known to harbor MDR gram-negative organisms

- AASLD: 1.5 g/kg at day 1 and 1 g/kg at day 3; pts with AKI and/or jaundice are more likely to benefit from albumin

- BSG: In patients with SBP and an increased/rising serum creatinine

**SBP** = spontaneous bacterial peritonitis

*Biggins et al. AASLD guidance. Hepatology 2021:74:1014-1048*

*Aithal et al. Gut 2021:70:9–29*
A repeat ascites PMN count 48 hours after antibiotic initiation is of therapeutic and prognostic significance.


Failure of therapy is defined as decrease in PMN <25% from baseline

Biggins, AASLD guidance 2021

- Check on culture results
- Investigate secondary peritonitis (abdominal film, CT scan)
- Broaden antibiotic spectrum

Ascites PMN decreases by at least 50% at day 2 after starting antibiotics.

A decline in PMN cell count > 80% predicted improved in-hospital survival (aOR 0.32, 95% CI 0.17-0.59) independent of cirrhosis severity.

Saffo et al. CGH 2021 [ePub ahead of print]
SBP is a frequent precipitant of acute kidney injury (AKI) and hepatorenal syndrome (HRS)

Cirrhosis → Portal (sinusoidal) hypertension → ↑ Systemic Inflammation → ↑ Splanchnic / systemic vasodilatation → ↓ Effective arterial blood volume → ↑ Activation of neurohumoral systems

- Sodium and water retention → Ascites
- Renal vasoconstriction → AKI / HRS

Prompt and appropriate antibiotics

Albumin
How should albumin be administered in patients with SBP/non-SBP infections?

- Dose of albumin used in patients with SBP (Sort et al. NEJM 1999) was empirical (1.5 g/Kg day at day 1 → 1 g/Kg day at day 3) and this dose is recommended in recent AASLD guidelines (2021).

- Albumin did not improve renal function or survival in non-SBP infections and led to pulmonary edema (Thevenot et al. J Hep 2015).

- Main predictor of death in SBP and non-SBP infections is the presence of AKI.

- It would appear sensible to guide administration of albumin based on the presence/course of AKI (per Ascites Club criteria).
Serum creatinine (sCr) is used in the diagnosis of acute kidney injury (AKI) in cirrhosis but criteria have changed.

<table>
<thead>
<tr>
<th>The Old</th>
<th>An increase in sCr ≥ 1.5 mg/dl (133 mmol/L)</th>
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<tbody>
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</table>

**Arroyo et al (International Ascites Club). Hepatology 1996;23:164-76**

<table>
<thead>
<tr>
<th>The New (based on KDIGO criteria)</th>
<th>a) An absolute increase in sCr ≥ 0.3 mg/dl (26.5 mmol/L) within 48 hours and/or b) Urinary output ≤ 0.5ml/Kg BW ≥ 6 hours (urinary catheterization) or b) Percent increase in sCr ≥ 50% within 3 months using the last available value of sCr</th>
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**Angeli, Garcia-Tsao, Nadim, Parikh. J Hepatol 2019;71:811-822**
Main differential among causes of AKI in cirrhosis

- **Pre-renal**: renal hypoperfusion without glomerular or tubular damage
  - **Prerrenal azotemia (most common)**
  - **Hepatorenal syndrome (HRS-AKI*)** is a type of pre-renal AKI that is unique to patients with cirrhosis and has the worst prognosis
- **Intra-renal**: acute tubular necrosis, interstitial nephritis or glomerulo-nephritis
- **Post-renal**: urinary tract obstruction (least common)

*Renamed from HRS-1 per Angeli, Garcia-Tsao, Nadim, Parikh. J Hepatol 2019;71:811-822*
Once AKI is diagnosed, it should be worked-up and treated as soon as possible.

Increase in sCr ≥ 0.3 mg/dl within 48 hours or a ≥ 50% increase in sCr within 3 months

- **Likely structural injury (ATN, GN, AIN)**
- **Clinical context**
- **Urine sediment and biomarkers**
  - FeNa, urine albumin
- **Renal ultrasound**

**Probable functional injury** (prerrenal azotemia, HRS)

- Discontinue diuretics, lactulose, vasodilators, NSBB, nephrotoxins; workup/treat infection

- **Volume depleted**
  - Crystalloid or blood

- **Not obviously dry and/or stage 2-3 AKI**
  - Albumin 1 g/Kg IV (maximum 100 mg)
  - Reassess in 24-48h
  - No resolution
  - Treat as HRS-AKI
  - Patient has ascites (refractory), likely hyponatremia and low MAP
  - FeNa <0.1%, NGAL <300 ng/mL

- **Resolution**
  - sCr= serum creatinine; ATN= acute tubular necrosis; GN=glomerulonephritis; AIN=acute interstitial nephritis

The treatment of choice for HRS-AKI is vasoconstrictor drugs in combination with albumin

The preferred vasoconstrictor is terlipressin, administered either as IV bolus or continuous IV infusion

Biggins et al. AASLD guidelines. Hepatology 2021;74:1014-1048
In a pooled analysis of trials*, an increase in MAP was strongly associated with a decrease in creatinine.

*using terlipressin as vasoconstrictor
In patients with HRS*, terlipressin was more effective than placebo in improving renal function but had more severe adverse events.

*Defined as a creatinine >2.25 mg/dL

Wong et al. CONFIRM study. NEJM 2021;384:818-828

<table>
<thead>
<tr>
<th>End Point</th>
<th>Terlipressin</th>
<th>Placebo</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary end point of verified reversal of HRS†</strong></td>
<td>63/199 (32)</td>
<td>17/101 (17)</td>
<td>0.006</td>
</tr>
<tr>
<td>Clinical success</td>
<td>121/199 (61)</td>
<td>81/101 (80)</td>
<td></td>
</tr>
<tr>
<td>Clinical failure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Competing event</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Liver transplantation</td>
<td>10/199 (5)</td>
<td>2/101 (2)</td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>5/199 (3)</td>
<td>0/101 (0)</td>
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</table>

**Verified HRS reversal**= two consecutive creatinine values ≤1.5 mg/dl at least 2 hours apart, and surviving without dialysis for at least 10 days

- Death within 90 days due to respiratory disorders occurred in 11% on terlipressin vs. 2% on placebo

- At baseline, serum albumin levels were high (3.7 g/dL terli; 4.0 g/dL placebo). While on therapy, patients received more IV albumin (199 g in terli group; 240 g in placebo group)
In a RCT, hospitalized patients with cirrhosis randomized to targeting serum albumin to a level $>=3.0$ mg/dL was not more beneficial than standard-of-care.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Albumin Group (N=380)</th>
<th>Standard-Care Group (N=397)</th>
<th>Adjusted Odds Ratio (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Composite primary end point — no. (%)</strong></td>
<td>113 (29.7)</td>
<td>120 (30.2)</td>
<td>0.98 (0.71–1.33)</td>
<td>0.87</td>
</tr>
<tr>
<td><strong>Components of composite primary end point — no. (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence of new infection</td>
<td>79 (20.8)</td>
<td>71 (17.9)</td>
<td>1.22 (0.85–1.75)</td>
<td></td>
</tr>
<tr>
<td>Incidence of kidney dysfunction</td>
<td>40 (10.5)</td>
<td>57 (14.4)</td>
<td>0.68 (0.44–1.11)</td>
<td></td>
</tr>
<tr>
<td>Incidence of death</td>
<td>30 (7.9)</td>
<td>33 (8.3)</td>
<td>0.95 (0.56–1.59)</td>
<td></td>
</tr>
<tr>
<td>Death at 28 days</td>
<td>53 (14.0)</td>
<td>62 (15.6)</td>
<td>0.86 (0.57–1.30)</td>
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</tr>
<tr>
<td>Death at 3 mo</td>
<td>92 (24.2)</td>
<td>93 (23.4)</td>
<td>1.05 (0.74–1.48)</td>
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</tr>
<tr>
<td>Death at 6 mo</td>
<td>132 (34.7)</td>
<td>119 (30.0)</td>
<td>1.27 (0.93–1.73)</td>
<td></td>
</tr>
</tbody>
</table>

*China et al. ATTIRE study. NEJM 2021;384:808-817*
Intravenous albumin targeted at a serum level $\geq 30$ g was associated with 10X the amount of albumin infused and more pulmonary edema/fluid overload.

Median albumin infused per patient:
- 200 g (140-280) in albumin group
- 20 g (0-120) in SOC group

Validation of noninvasive methods to assess blood volume will be important in the management of patients with decompensated cirrhosis receiving albumin, particularly when combined with terlipressin.
Management algorithm in patient with suspected HRS

Start a vasoconstrictor (+ albumin):
- Terlipressin (first choice)
- Norepinephrine (if terlipressin unavailable)*

Adjust dose daily by MAP (goal ↑ 10-15 mmHg) and assess response

No response
- Discontinue vasoconstrictor
- RRT (OLT candidates)

↓ SCr ≥ 25% after 3 days of therapy
- Continue vasoconstrictor
- HRS reversal (creatinine at or near baseline)
- Discontinue vasoconstrictor

Ischemic complication
- Severe hyperkalemia, acidosis or volume overload

Reassess OLT priority / status

HRS recurrence

*If neither terlipressin or norepinephrine can be administered, a trial of oral midodrine in combination with octreotide may be considered

Biggins et al. AASLD guidance 2021
ACLF is an entity that occurs in hospitalized patients with cirrhosis and that is associated with a poor 30-day survival.

ACLF = acute-on-chronic liver failure; VH = variceal hemorrhage; HE = hepatic encephalopathy; HRS = hepatorenal syndrome.
Not unexpectedly, the number of organ failures correlate directly with an increasingly higher 28-day mortality (EASL-CLIF)

Moreau et al. Gastroenterology 2013;144:1426–1437

n = 1,343
Short-term mortality in ACLF is more accurately predicted by its clinical course in the first 3 – 7 days.

Among patients with ≥4 organ failures, or CLIF-C ACLF score >64 at days 3-7, mortality was 100%.

Management of ACLF is non-specific and based on support of organ failures

Precipitating factor
- Acute inflammatory state
  - Vasodilatation and liver/cardiac dysfunction

Patient with cirrhosis
- Jaundice
- Coagulopathy
- Hepatic encephalopathy
- Kidney failure
- Hypotension
- Hypoxemia

Multiorgan failure state (ACLF)

Direct:
- Alcoholic hepatitis
- Viral hepatitis exacerbation
- Drug-induced liver injury

Indirect:
- Bacterial infections/sepsis
- GI hemorrhage
- Ischemia

Support failing organs / ICU care

Identify and treat precipitant

Assess prognosis: ACLF scores

Transplant candidate

Transplant evaluation

Palliative and end of life care

Not a transplant candidate

ACLF = acute-on-chronic liver failure; ICU = intensive care unit
Inpatient Management of Decompensated Cirrhosis

- The main decompensating events are ascites and variceal hemorrhage.
- In patients with acute variceal hemorrhage, think of pTIPS candidacy at time of admission (mainly Child C patients 10-13 points).
- A diagnostic paracentesis (to rule out SBP) should be performed with each non-elective admission and with the development of symptoms (abdominal pain, fever) or any complication (AKI, encephalopathy).
- Be cautious about excessive albumin infusion in hospitalized patients.
- ACLF is a multiorgan failure state in cirrhosis and its staging is of prognostic value and can inform future management.