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# Care of the patient with cirrhosis:
# management of acute alcohol-associated hepatitis and acute on chronic liver failure


**Rebecca G. Kim, M.D., M.A.S.**
**Assistant Professor of Medicine**
**Division of Gastroenterology and Hepatology**
**University of Utah Health**

# Outline

- Common scenarios
  - Acute alcohol-associated hepatitis
  - Ascites
  - Spontaneous bacterial peritonitis
  - Acute on chronic liver failure
- Transitions of care
- Questions

# Common Scenarios: Case Presentation

31 yo man presents to the ED with **3 weeks of jaundice, difficulty sleeping, and worsening fatigue**. He is well-known to the ED as he has presented in the past for **alcohol intoxication and withdrawal**.

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31 yo man presents to the ED with **3 weeks of jaundice, difficulty sleeping, and worsening fatigue**. He is well-known to the ED as he has presented in the past for **alcohol intoxication and withdrawal**.

Patient reports he has **not had any alcohol for the last 3 weeks** since noticing that his skin was turning yellow and after he developed loss of appetite, nausea, worsening fatigue and difficulty sleeping.

# Common Scenarios: Case Presentation

Vitals: within normal limits

Exam
Mildly jaundiced, otherwise normal exam with spider angioma on his chest, no fluid overload, no asterixis

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Exam
Mildly jaundiced, otherwise normal exam with spider angioma on his chest, no fluid overload, no asterixis

Labs
WBC 11, Hgb 13, platelets 147
Na 136, Cr 1.1, albumin 3.2
AST 97, ALT 49, alk phos 121, total bilirubin 4.8
PT 16, INR 1.3

Imaging: abdominal ultrasound shows liver with nodular contour without focal masses, mild splenomegaly, patent hepatic vasculature, no ascites, no biliary dilatation

# Common Scenarios: Case Presentation

**Most likely diagnosis?**

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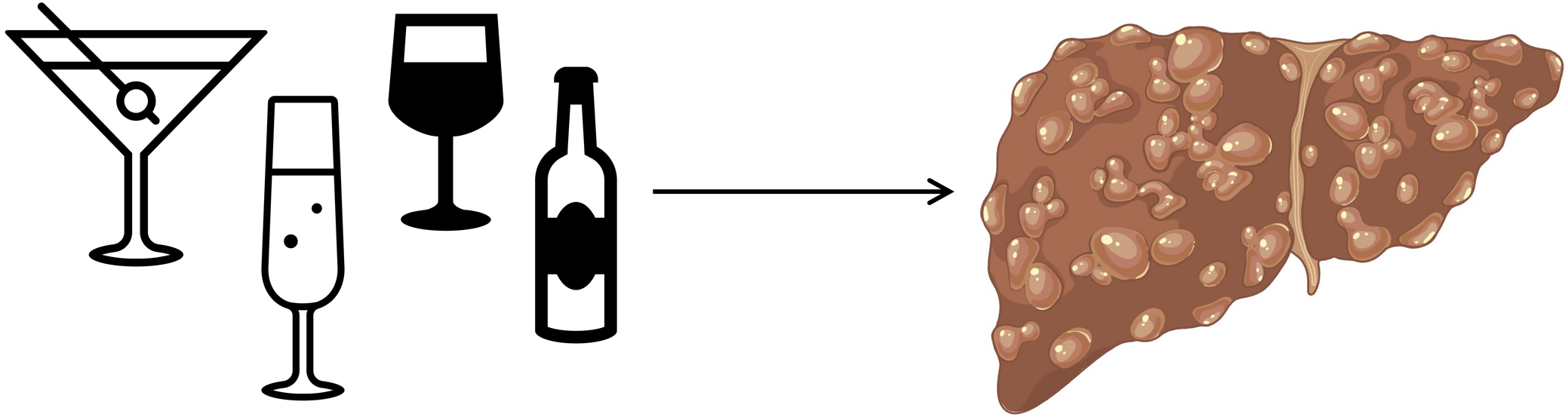
Acute alcohol-associated hepatitis in setting of alcohol-associated cirrhosis



# Diagnosis of alcohol-associated hepatitis (AH)

Note: “alcoholic” --> “alcohol-associated” or “alcohol-related”

**Definition of AH:**

| Clinical Diagnosis of AH |
|---|
| Onset of jaundice within prior 8 weeks |
| Ongoing consumption of >30g (F) or 60g (M) alcohol/day for ≥6 months, with <60d of abstinence before onset of jaundice |
| AST >50<br>AST/ALT >1.5<br>Both AST and ALT <400 IU/L |
| Total Bilirubin >3 mg/dL |

| Potential Confounding Factors |
|---|
| Possible ischemic hepatitis (severe UGIB, hypotension, recent cocaine) |
| Metabolic liver disease (Wilson, a1AT def) |
| Possible DILI |
| Uncertain alcohol use (pt denies excess use) |
| Atypical lab tests<br>AST <50 or >400<br>AST/ALT <1.5<br>ANA >1:160 or SMA >1:80 |

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*slide adapted from Courtney Sherman, MD

Crabb et al. AASLD Practice Guidelines. 2019.
Crabb et al. Gastroenterology. 2016.

# Common Scenarios: Case Presentation

**Most likely diagnosis?**

Acute alcohol-associated hepatitis in setting of alcohol-associated cirrhosis

**Next steps to determine management?**

# Assessing AH Prognosis

| Score & Components | Stratification | Clinical Application | Pros | Cons |
|---|---|---|---|---|
| **Maddrey Discriminant Function**<br>- TB, PT | Severe ≥32<br><br>Predicts high risk of short-term mortality | Start steroids if severe | Extensive experience in AH<br><br>Inclusion criteria for most AH clinical trials | False positive → excess steroid use<br><br>Less accurate in intermediate and long term |

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Maddrey et al. Gastroenterology. 1978.

# Assessing AH Prognosis

| Score & Components | Stratification | Clinical Application | Pros | Cons |
|---|---|---|---|---|
| **MELD 3.0**<br>- DOB, sex, albumin, Na, Cr, INR, TB | Severe > 20 | Prognosis | Extensive experience<br><br>ΔMELD over time may add additional prognostic info | Unclear threshold to start steroids, MELD >20 proposed |



Crabb et al. AASLD Practice Guidelines. 2019.
Maddrey et al. Gastroenterology. 1978.

# Common Scenarios: Case Presentation

**Most likely diagnosis?**

Acute alcohol-associated hepatitis in setting of alcohol-associated cirrhosis

**Next steps to determine management?**

**Maddrey's Discriminant Function: 23.2**
**MELD 3.0: 18**

# Common Scenarios: Case Presentation

**Most likely diagnosis?**

Acute alcohol-associated hepatitis in setting of alcohol-associated cirrhosis

**Next steps to determine management?**

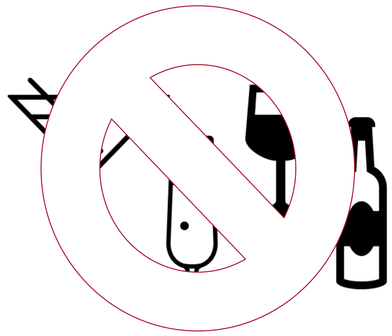
Maddrey's Discriminant Function: 23.2
MELD 3.0: 18

**Management?**

# Common Scenarios: Case Presentation

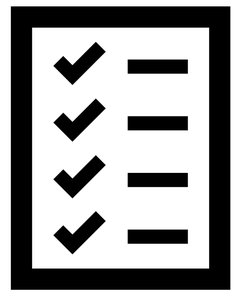
**<u>Management?</u>**

**Alcohol cessation**

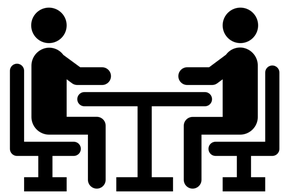


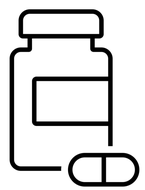
**Hepatology referral**

**PCP follow up**



**Addiction medicine referral or resources for AUD treatment**





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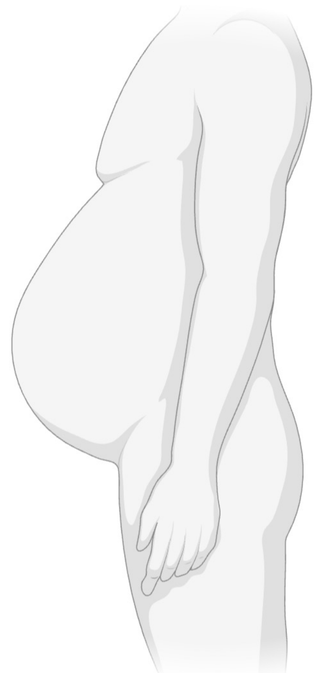
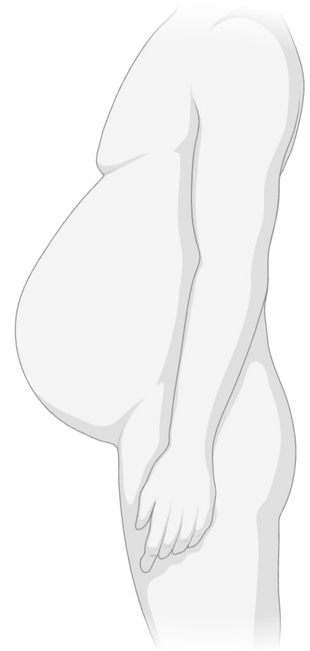


# Common Scenarios: Case Presentation

Found to have **abdominal distention with ascites**



**Labs:** AST 193, ALT 78, INR 2, total bilirubin 13, PT 21, albumin 2.9, Na 130, Cr 1.3

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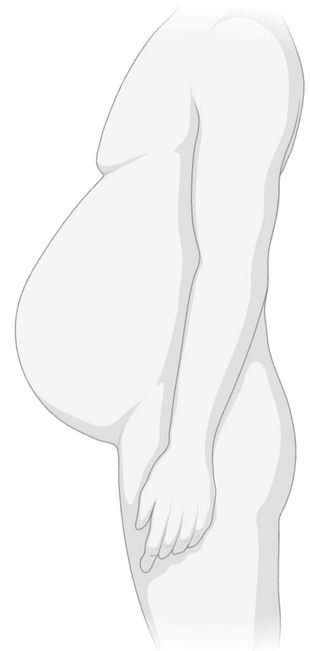


**Labs:** AST 193, ALT 78, INR 2, total bilirubin 13, PT 21, albumin 2.9, Na 130, Cr 1.3

**Maddrey's Discriminant Function 54.4**
**MELD 3.0 29**

# Common Scenarios: Case Presentation

**Does this patient have cirrhosis?**

# Common Scenarios: Case Presentation

**Management?**

# Severe AH Management: Corticosteroids

- **STOPAH** – Steroids or Pentoxifylline for AH
  - Largest RCT in severe AH → multicenter, randomized, double-blind, enrolled 1103 pts with clinically severe AH in UK
  - *Post hoc* multivariable analysis → steroids associated with improved 28d survival (OR 0.61, p=0.02), but not 90d or 1y
  - Pentoxifylline did not improve survival
  - Serious infections occurred in 13% tx with steroids vs 7% who did not receive steroids (p=0.002)



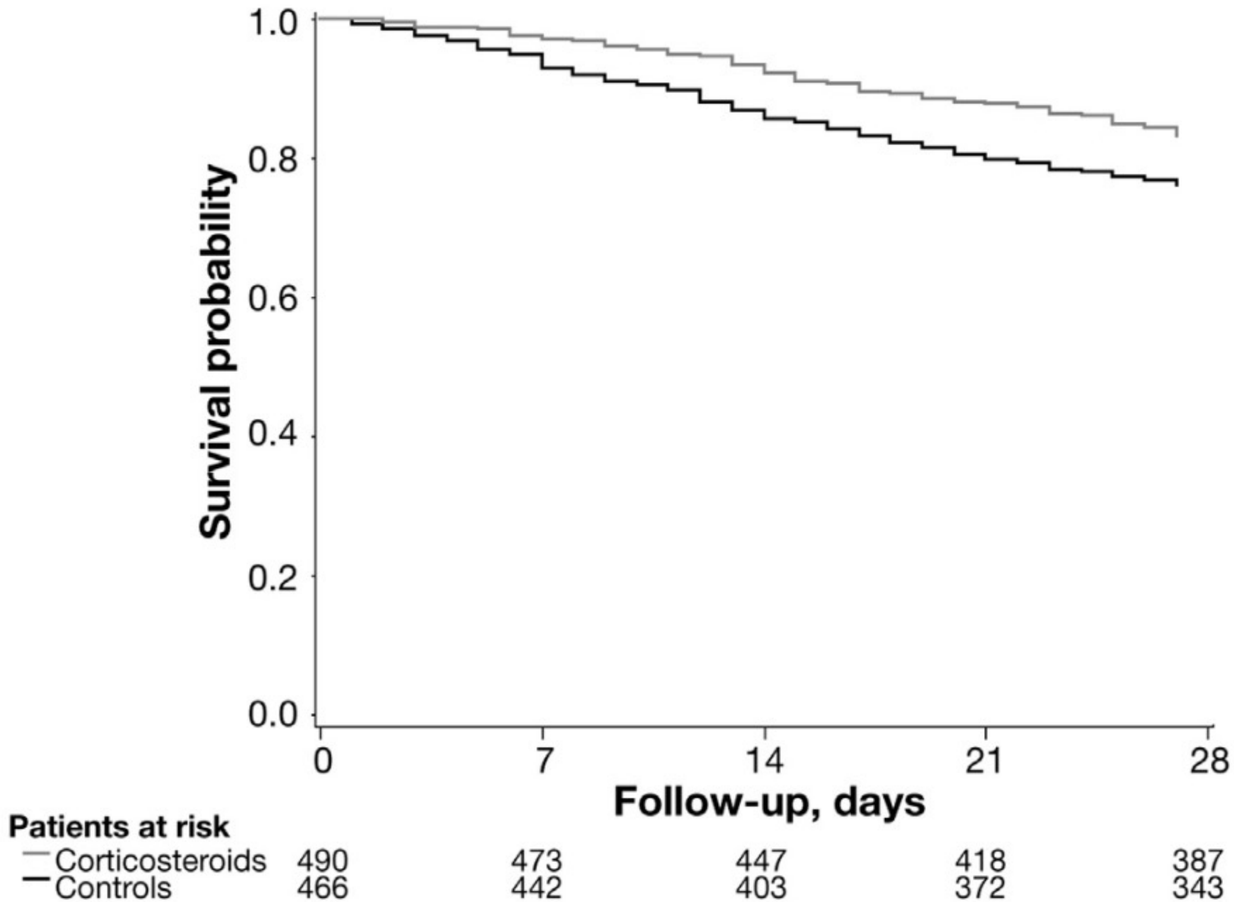
Thursz et al. NEJM. 2015.

# Severe AH Management: Corticosteroids

Meta-analysis of individual pt data from 11 RCTs (2111 pts) comparing steroids, pentoxifylline or combo in severe AH

**Steroids significantly reduced 28d mortality** vs placebo (36% risk reduction) or pentoxifylline

No significant differences in 6-month mortality with any tx or controls






Louvet et al. Gastroenterology. 2018.

# Common Scenarios: Case Presentation

**Management?**

Admission to medicine

Willing to be admitted → start assessment for contraindications for steroids

# Severe AH Management: Corticosteroids

- **Relative contraindications to steroids**

| | **Considerations** |
|---|---|
| Uncontrolled infection | -Need thorough evaluation and time to r/o infection prior to starting steroids |
| Acute kidney injury<br>Cr > 2.5 | -AKI excluded in major AH trials so data lacking<br>-If AKI resolves, then steroids can be considered |
| GI bleeding | -GIB excluded from many trials<br>-After control of GIB, can consider steroids |
| Other: multisystem organ failure, shock, active HBV, active TB | |



Crabb et al. AASLD Practice Guidelines. 2019.

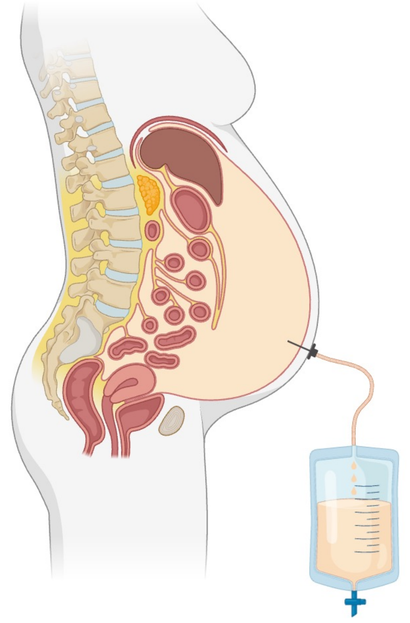
# Common Scenarios: Case Presentation

**Infectious work up:**

Diagnostic paracentesis to rule out spontaneous bacterial peritonitis

Should always be done prior to initiation of antibiotics in any patient with ascites

Studies: cell count, albumin, total protein, gram stain, culture



# Ascites: Prognosis and Management

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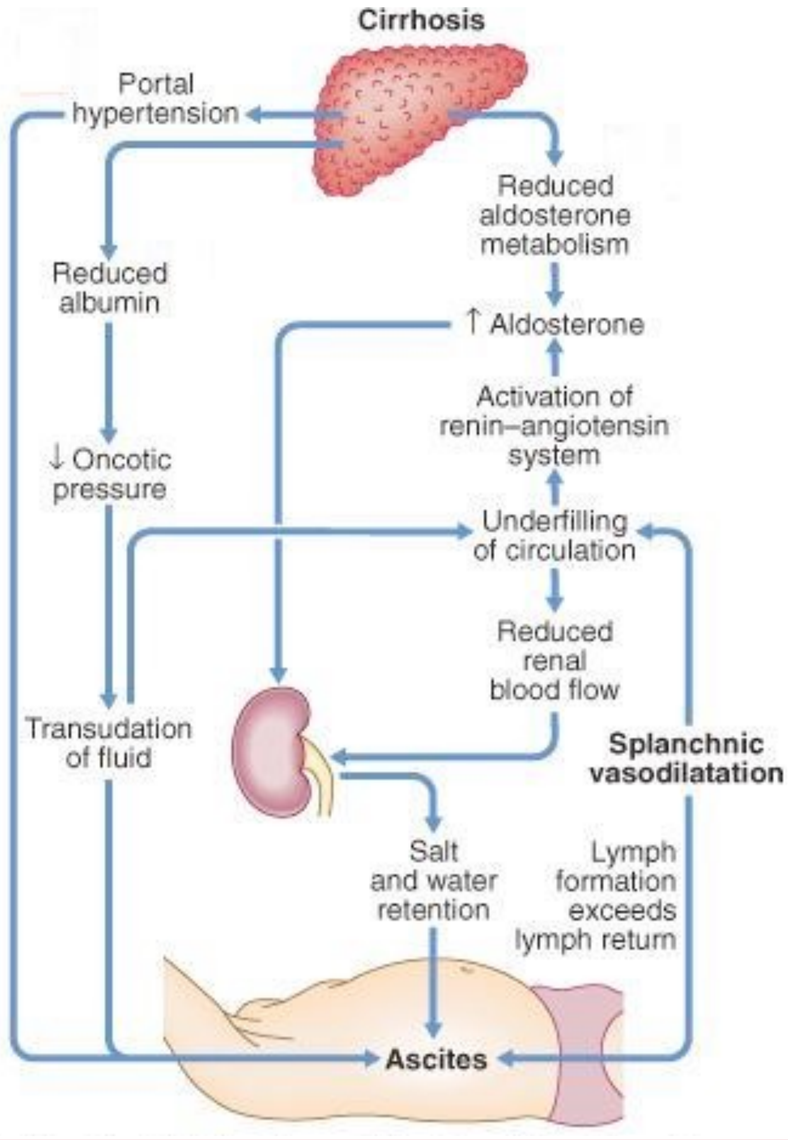
- Most common complication of cirrhosis – 60% of patients with compensated cirrhosis develop ascites within 10 years

- Occurs with **excessive formation or impaired absorption of peritoneal fluid**



# Ascites: Prognosis and Management

Development of ascites in cirrhosis indicates poor prognosis –
**mortality is ~40% at 1 year and ~50% at 2 years**

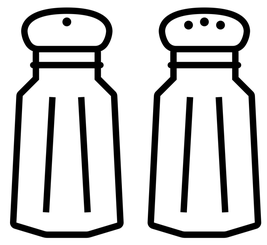
Predictors of poor prognosis:

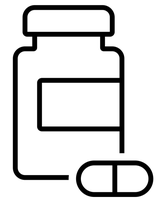
**hypoNa**
**increased Cr**
**low arterial pressure**
**low urine sodium**

# Ascites: Prognosis and Management

Achieve a negative sodium balance with:

**1) dietary salt restriction (<2g/day)**
**2) diuretics**





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Ratio of 5:2 is to maintain potassium balance

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Start spironolactone (50-100mg) + furosemide (20-40mg) daily
Ratio of 5:2 is to maintain potassium balance

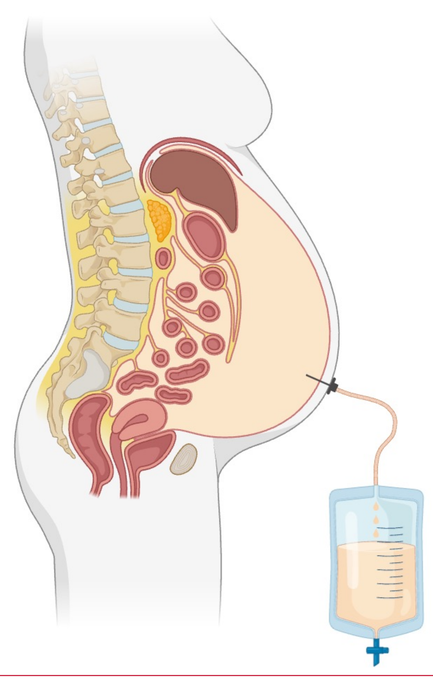
Diuretic-induced complications are common, so close monitoring of weight, serum electrolytes and renal function is essential

Uptitrate at intervals of 4-7 days based on exam and labs

# Ascites: Prognosis and Management

Therapeutic paracenteses are appropriate for patients with tense ascites

To minimize **hypotension, renal impairment** and **fluid and electrolyte imbalance**, give IV infusion of 25% albumin (6-8g albumin for each liter of fluid removed)



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**Infectious work up:**

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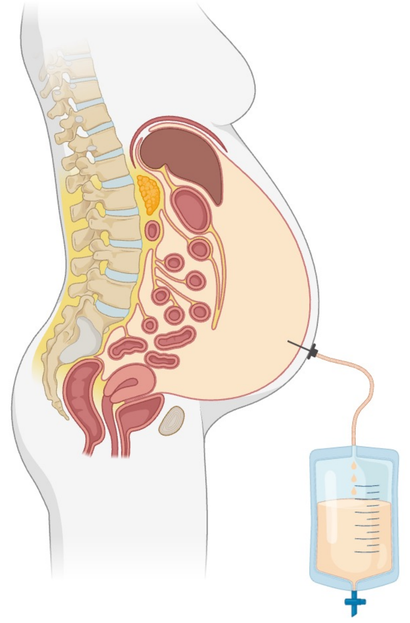
Studies: cell count, albumin, total protein, gram stain, culture

**>250 neutrophils = spontaneous bacterial peritonitis**

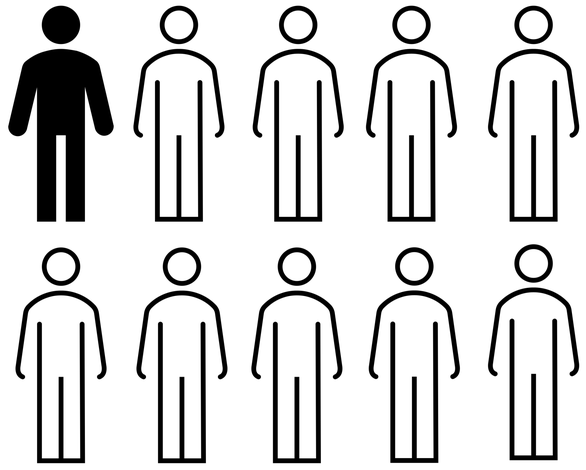


# Spontaneous Bacterial Peritonitis: Prognosis and Management

Prevalence of SBP:

Outpatient 1.5-3.5%

Hospitalized patients, ~10%



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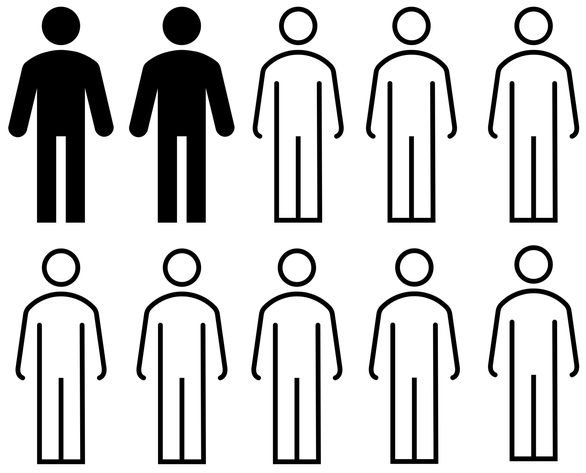

**<u>Mortality</u>** previously >90%, but is now down to **<u>20%</u>** with **early diagnosis and treatment**

# Spontaneous Bacterial Peritonitis: Prognosis and Management

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- Hospitalized patients, ~10%

**Mortality** previously >90%, but is now down to **20%** with **early diagnosis and treatment**

Possible symptoms: abdominal pain/tenderness, vomiting, diarrhea, hyper or hypothermia, chills, altered mental status, renal dysfunction, or gastrointestinal bleeding

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**Mortality** previously

**treatment**

**rly diagnosis and**

10% of infected patients present completely asymptomatic

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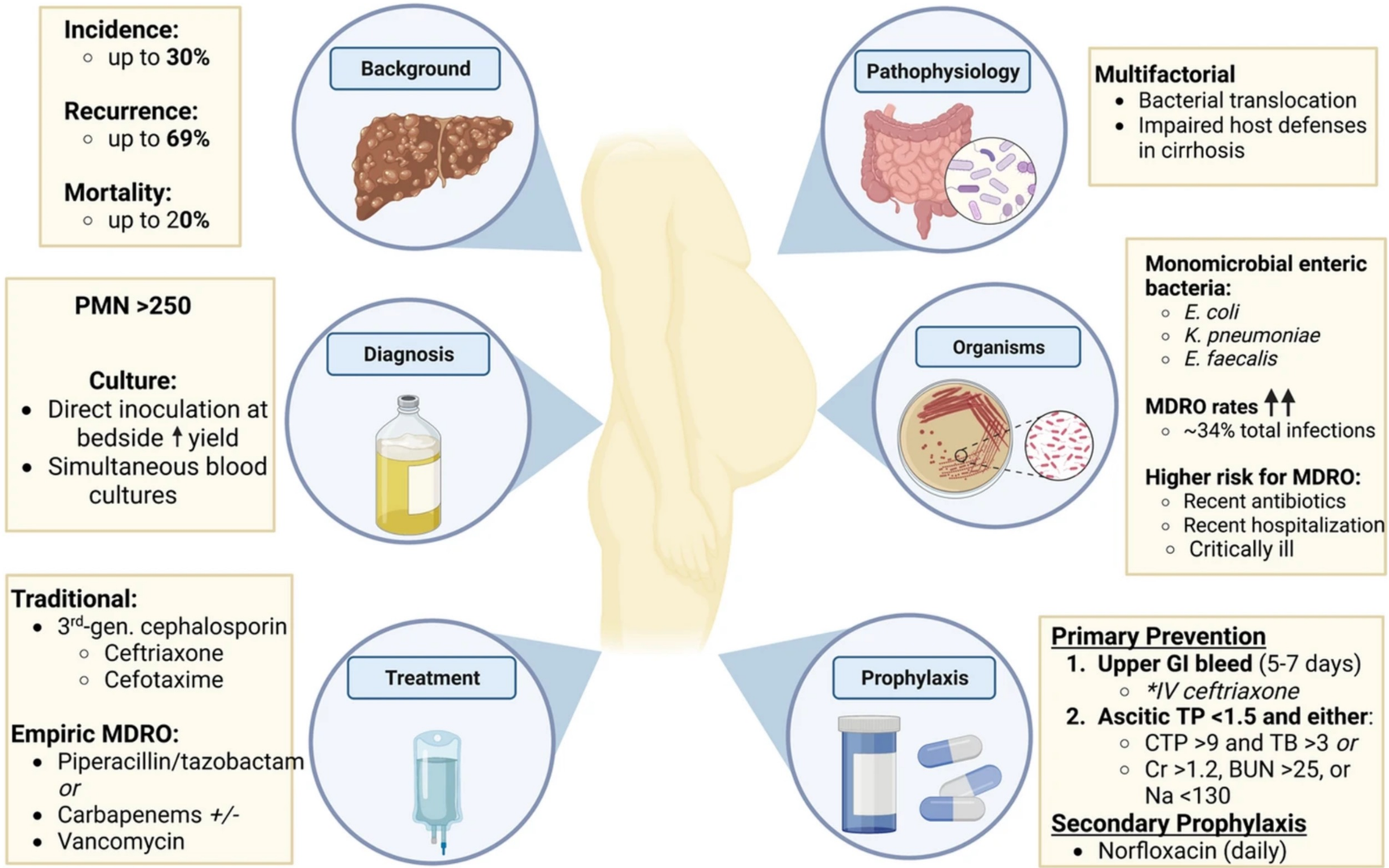
# Spontaneous Bacterial Peritonitis: Prognosis and Management





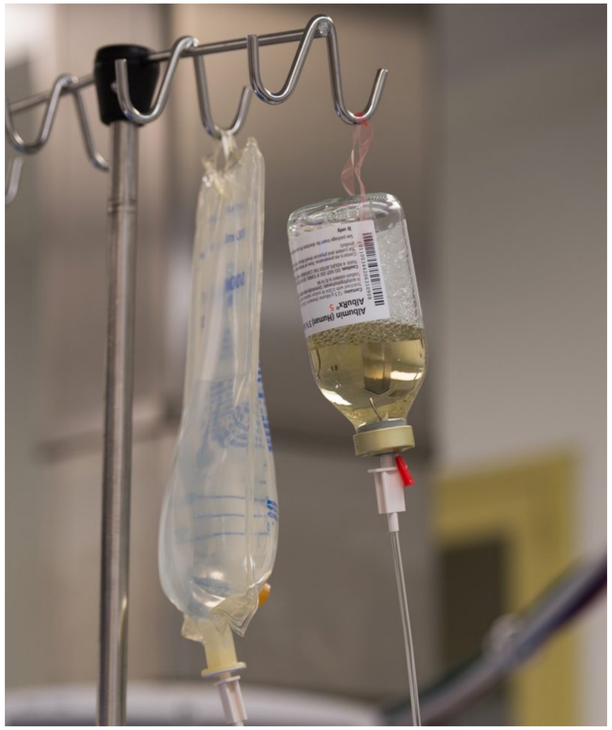

Sandhu, S., John, B.V. Spontaneous Bacterial Peritonitis: The Bug Matters. *Dig Dis Sci* **68**, 1667–1669 (2023)

# Spontaneous Bacterial Peritonitis: Prognosis and Management

**<u>In addition to antibiotics (5-day course):</u>**

Albumin infusions on day 1 of diagnosis and day 3

- Day 1: albumin 25% 1.5g/kg
- Day 3: albumin 25% 1g/kg



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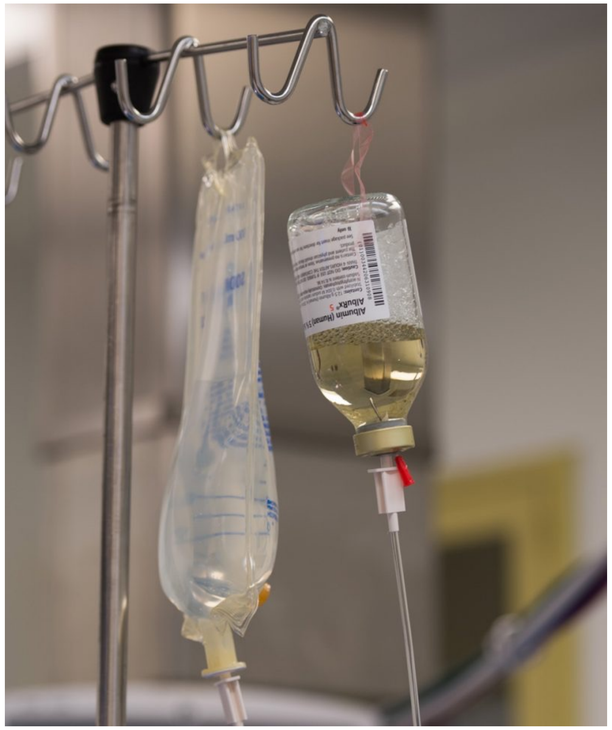

**<u>Secondary prophylaxis:</u>** Prophylactic quinolones or TMP/SMX have been shown to decrease rate of primary and recurrent SBP by **70-90%**

# Common Scenarios: Case Presentation

**Back to the case…severe acute alcohol-associated hepatitis**

**Management?**

Admission to medicine

Declines admission and requests to leave from ED → ???

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**Management?**

Admission to medicine

Declines admission and requests to leave from ED → ???

**Average** time from presentation to steroids in trials was **6 days**

**Alcohol-associated hepatitis**

**Mild to moderate**
Alcohol cessation, PCP and hepatology outpatient

**Severe (MDF ≥ 32, MELD > 20)**
Infectious work up, alcohol cessation, consider prednisolone, adequate nutrition

**Ascites**

**Altered mental status**

**Gastrointestinal bleeding**

**SBP**

**Hepatorenal syndrome**

# Acute on Chronic Liver Failure (ACLF)

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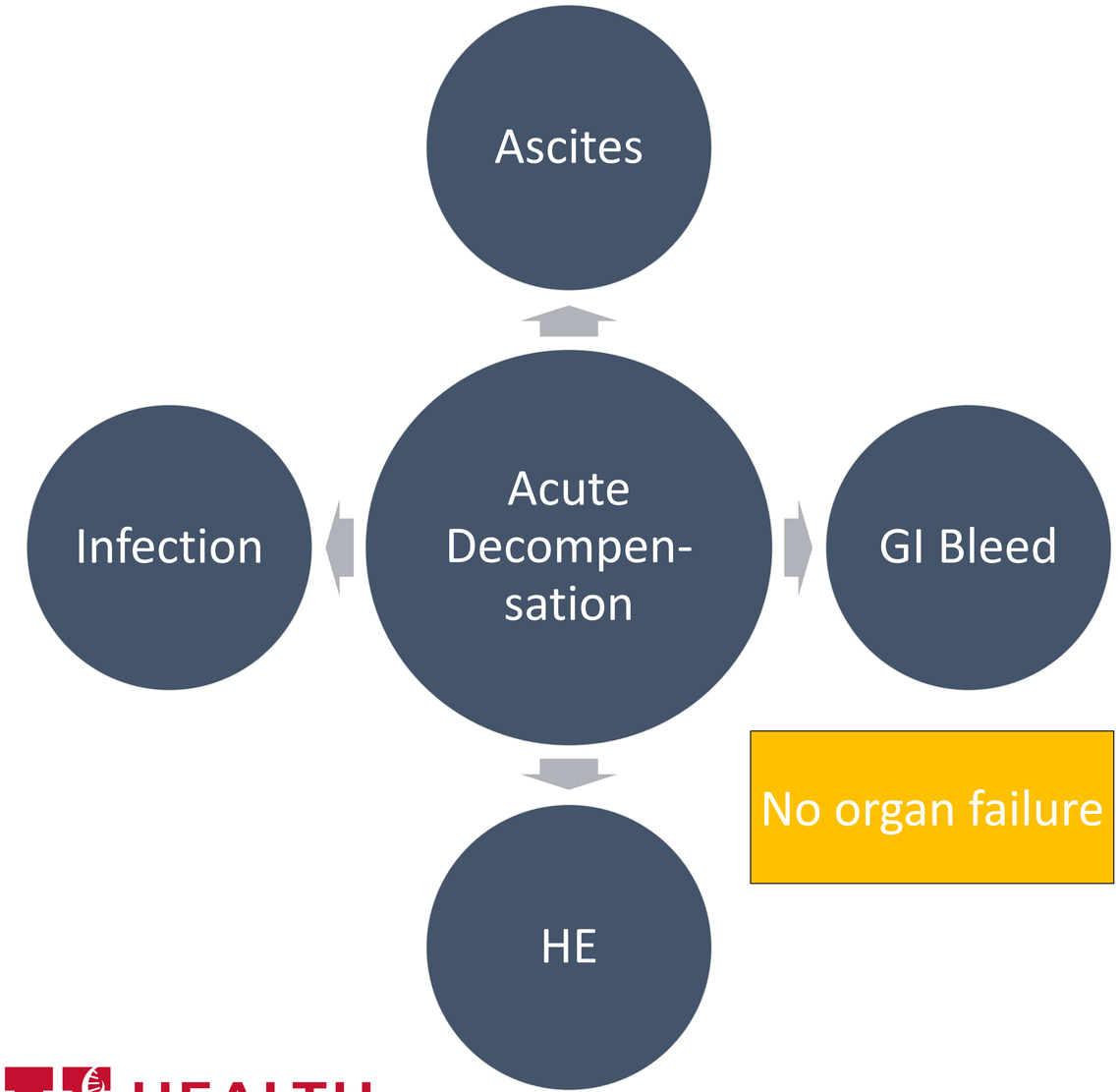


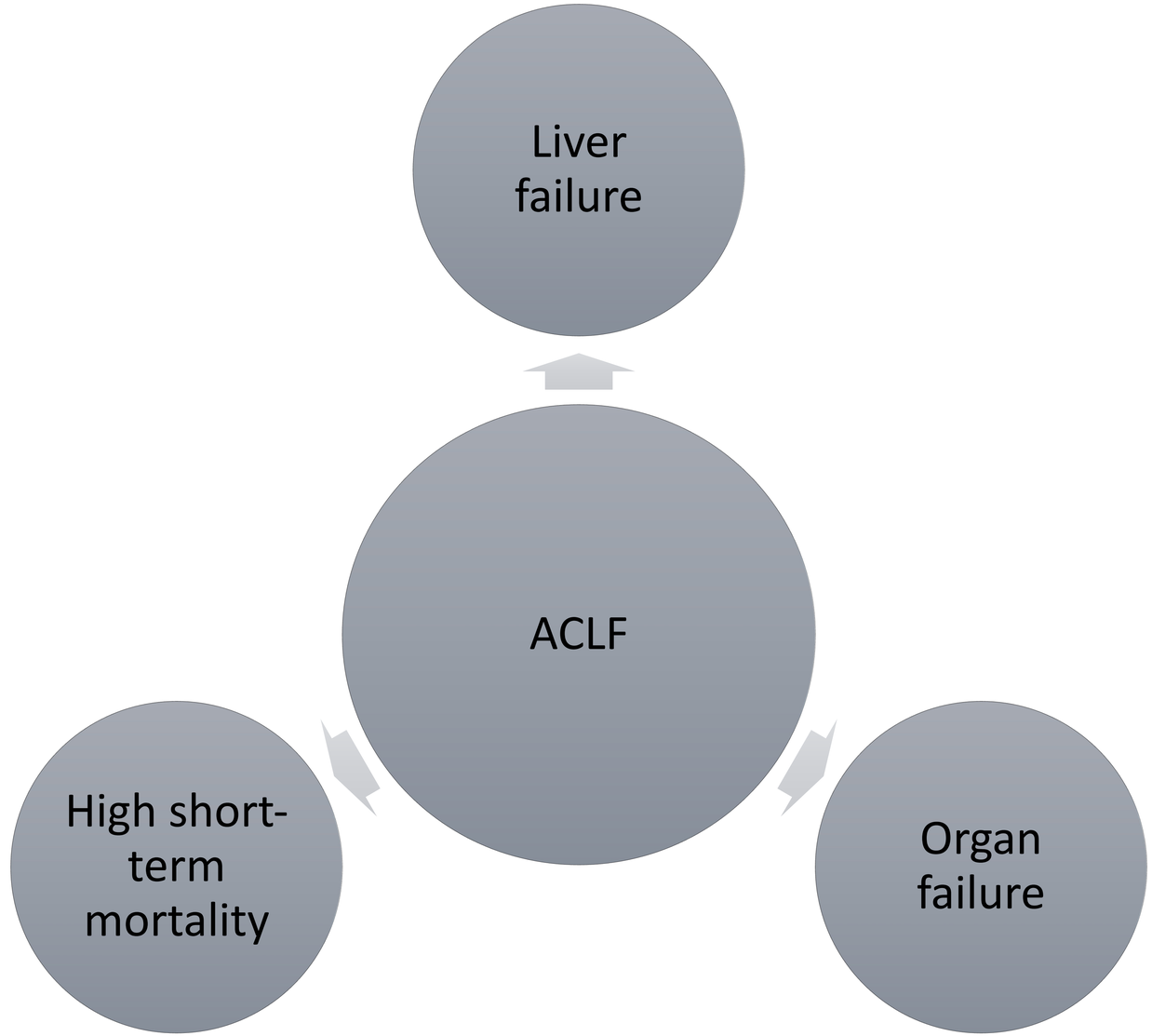


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Jalan et al. J Hep 2015
Arroyo et al. Nat Rev Dis Primers 2016

# Acute on Chronic Liver Failure (ACLF)

| ACLF Grade (CANONIC) | |
|---|---|
| No ACLF | 1.No OF<br>2.Single OF (liver, coagulation, circulatory, respiratory) with creatinine < 1.5 mg/dL and no HE<br>3.Single cerebral failure and creatinine< 1.5 mg/dL |
| ACLF-1 | One of the following:<br>1.Single renal failure<br>2.Single non-renal OF plus creatinine 1.5-1.9 and/or HE 1-2<br>3.Single brain failure plus creatinine 1.5-1.9 |
| ACLF-2 | Two OFs of any combination |
| ACLF-3 | Three or more OFs of any combination |

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Moreau et al. Gastroenterology. 2013.
Bernal et al. Lancet. 2015.

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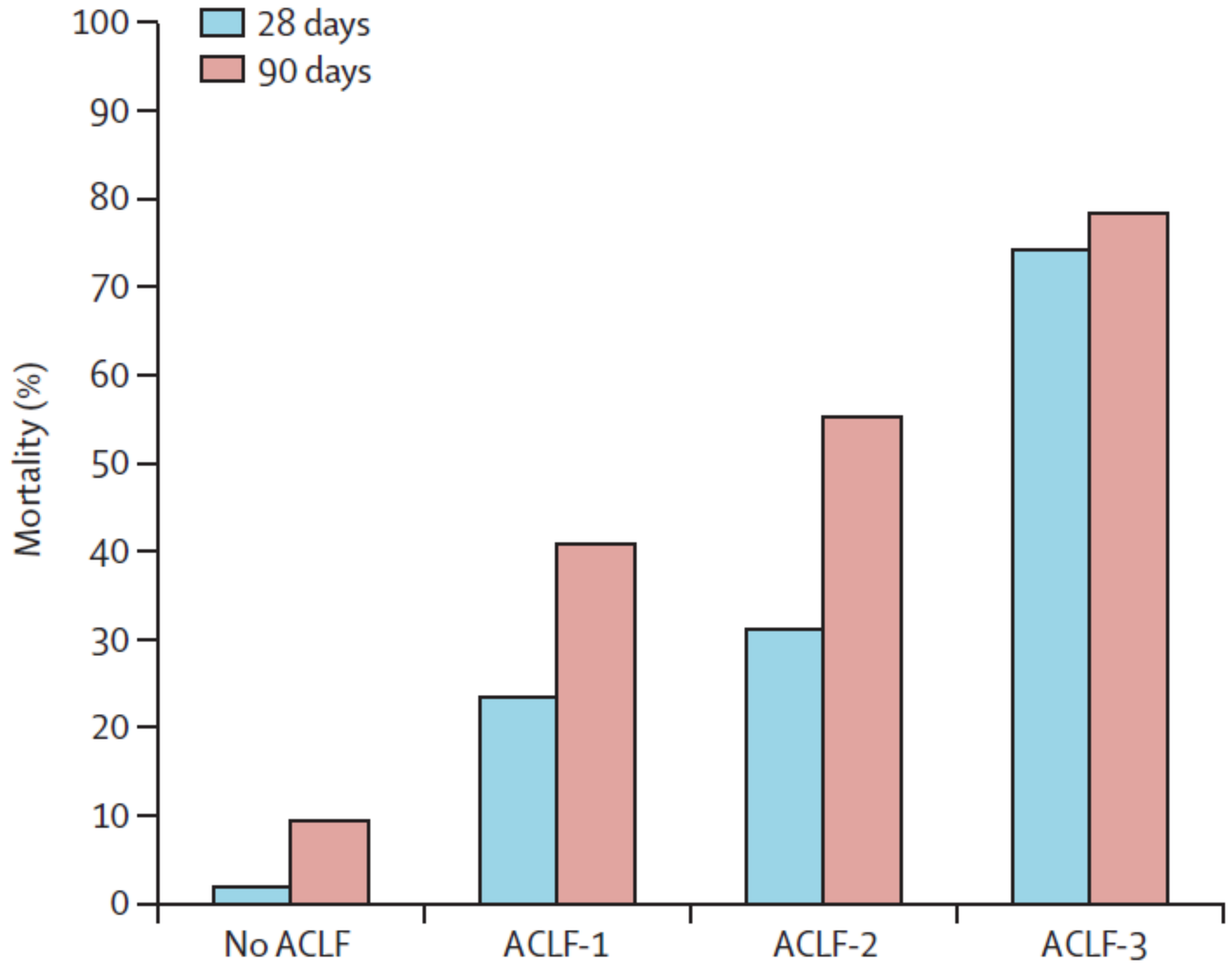


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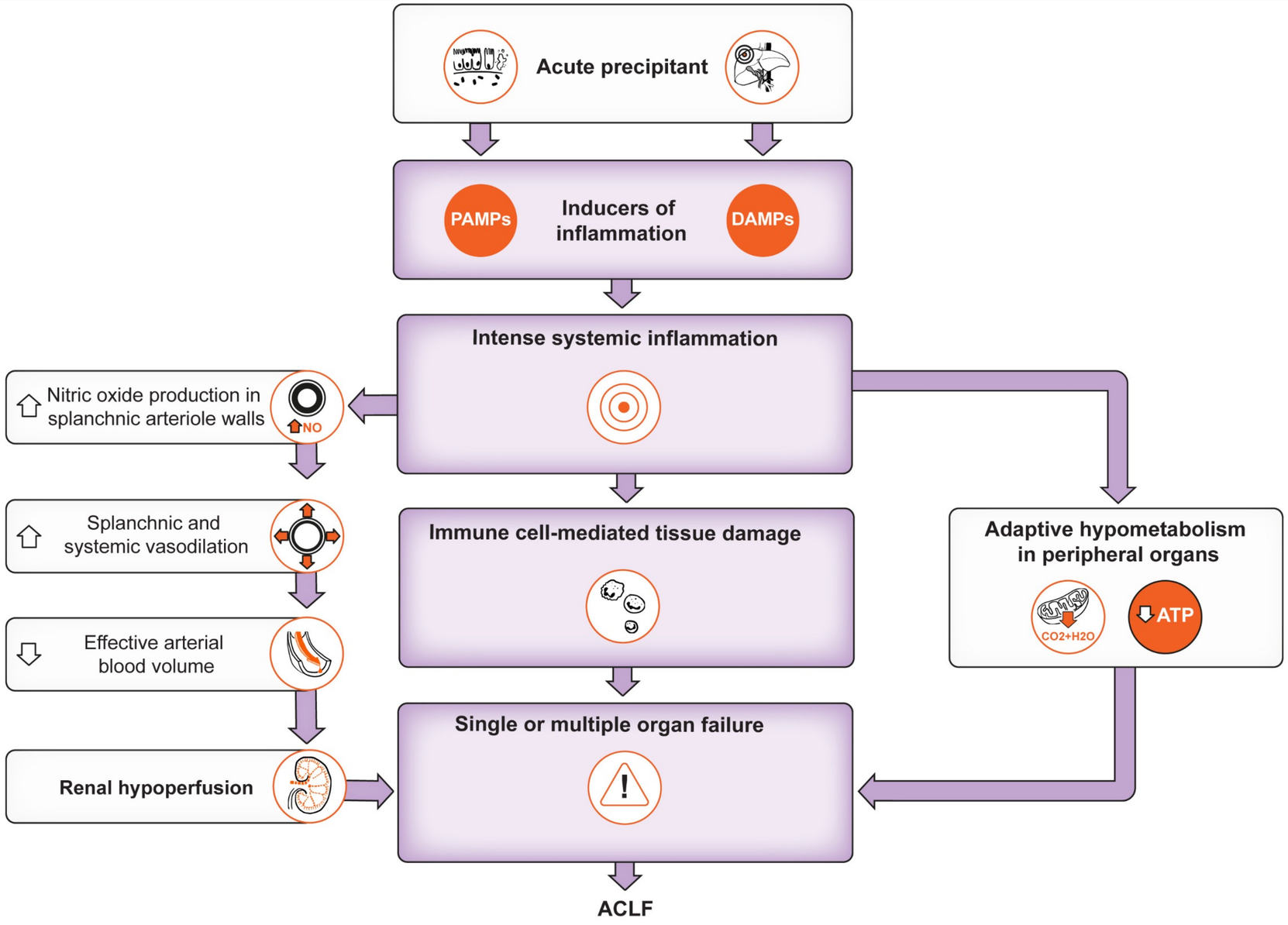


Clària J, et al, 2023,.

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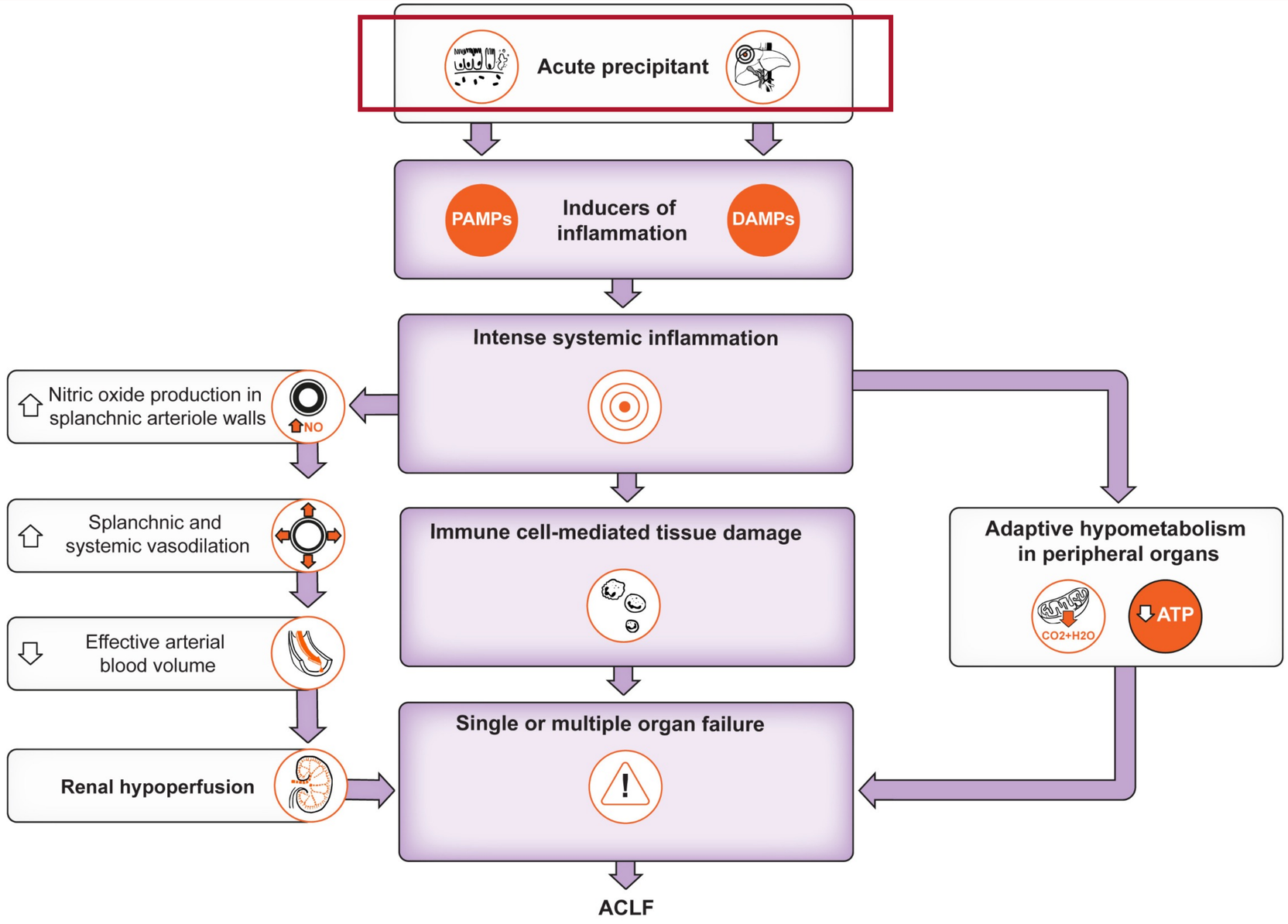


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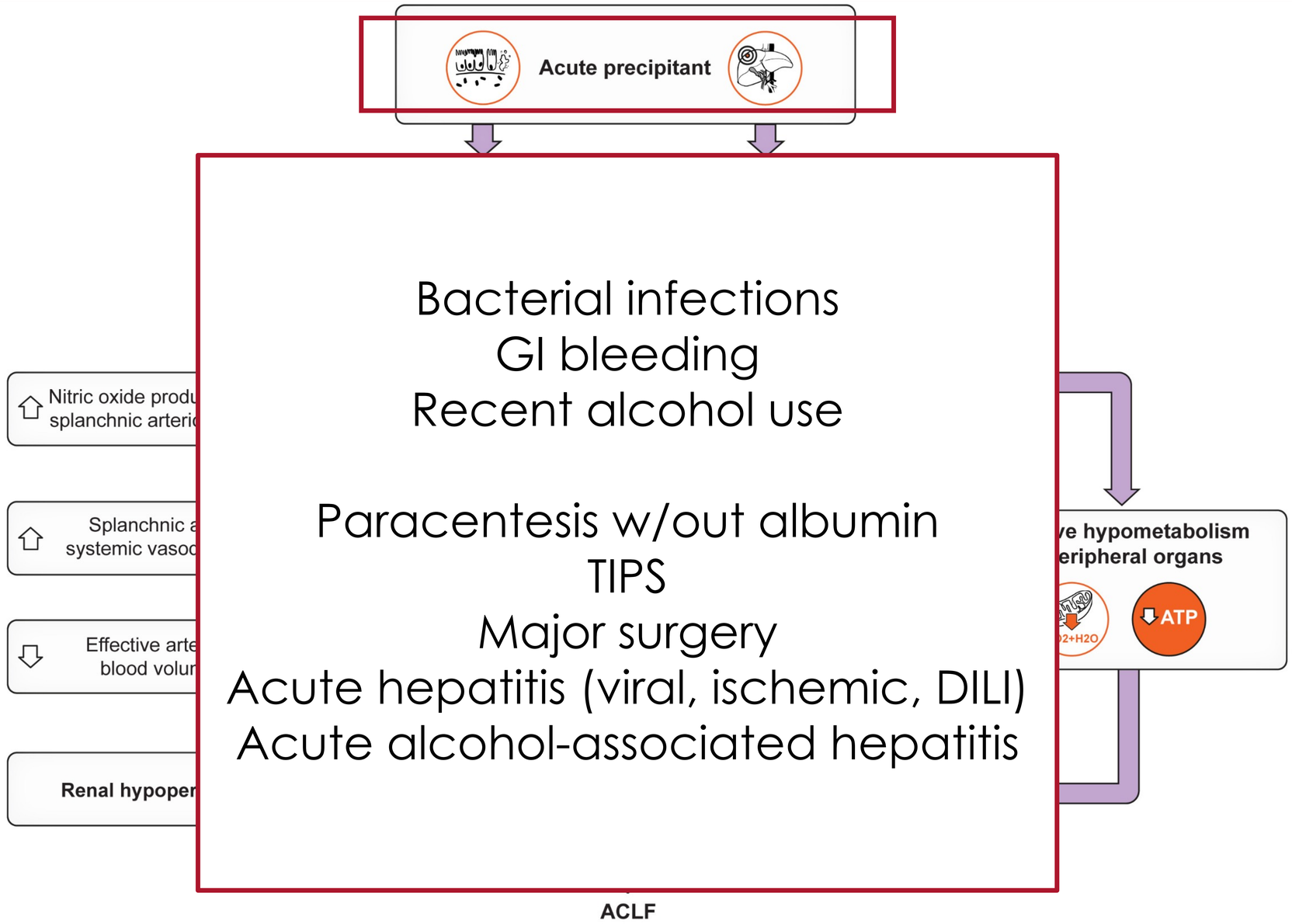


Clària J, et al, 2023,.

# Acute on Chronic Liver Failure (ACLF)

**Principles of treatment for organ failure in ACLF**

## Infections

- Complete work-up at diagnosis of ACLF to rule out infections
- High-dose broad spectrum antibiotics (tailored to local epidemiology) at ACLF diagnosis
- Daily reassesment of antibiotic therapy

- Do not delay the administration of antibiotics to the obtention of cultures
- Empirical antifungal therapy only if risk factors for invasive fungal infections

## Kidney

- Assessment of AKI severity using modified KDIGO criteria from the International Club of Ascites
- 20% albumin (1 g/kg for 48 hr) in patients with AKI stage 2-3
- In type-1 hepatorenal syndrome: 20% albumin (1 g/kg for 48 hr and then 20-40 g/day) + terlipressin (2 mg/24 hr) or norepinephrine (0.5 mg/hr, when terlipressin is not available)
- RRT - define goal: bridging to LT

- Avoid nephrotoxic drugs (NSAID)
- Avoid early initiation of RRT

## Lungs

- Endotracheal intubation for patients with West Heaven grade III or IV hepatic encephalopathy
- Lung protective ventilation strategy
- Prone positioning feasible
- Paracentesis in case of tense ascites

## Hemodynamics

- Early goal-directed therapy within the first 6 hours
- Maintain mean arterial pressure >65 mmHg
- Fluid challenges until no further hemodynamic response
- Prefer crystalloids and 5% albumin as resuscitation fluid
- Strong indications of albumin: spontaneous bacterial peritonitis, large volume parascentesis, AKI (see kidney)
- Norepinephrine as first line vasopressor; epinephrine or terlipressin when additional agent needed
- Intravenous hydrocortisone if refractory shock (norepinephrine >0.5 mg/kg.min)

- Avoid starches formulations
- Limit saline solutions in patients with ascites or anasarca

## Coagulation

- Fibrinogen and/or patelets in patients with severe hypofibrinogenemia (<1g/L) and/or thrombocytopenia (<20,000 x$10^9$/L) undergoing invasive procedures
- Prophylaxis for deep-vein thrombosis in patients without severe coagulopathy

- Avoid correction of INR alterations with fresh frozen plasma in the absence of bleeding

## Gastro-intestinal

- Consider stress-ulcer prophylaxis
- Administer early oral or enteral feedings, as tolerated, after ACLF diagnosis (goal: 10-15 kcal/kg/day by day 4)

## Nervous central system

- Treatment of the underlying cause
- Lactulose and enemas for hepatic encephalopathy
- Use sedation protocols, targeting specific endpoints
- Use short-acting sedative agents

- Avoid deep sedation, avoid benzodiazepines
- Avoid neuromuscular in patients without ARDS



Giacomo Zaccherini G, et al 2021,

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- Do not delay the administration of antibiotics to the obtention of cultures
- Empirical antifungal therapy only if risk factors for invasive fungal infections



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# Acute on Chronic Liver Failure (ACLF)

## Kidney

- Assessment of AKI severity using modified KDIGO criteria from the International Club of Ascites
- 20% albumin (1 g/kg for 48 hr) in patients with AKI stage 2-3
- In type-1 hepatorenal syndrome: 20% albumin (1 g/kg for 48 hr and then 20-40 g/day) + terlipressin (2 mg/24 hr) or norepinephrine (0.5 mg/hr, when terlipressin is not available)
- RRT - define goal: bridging to LT

- Avoid nephrotoxic drugs (NSAID)
- Avoid early initiation of RRT



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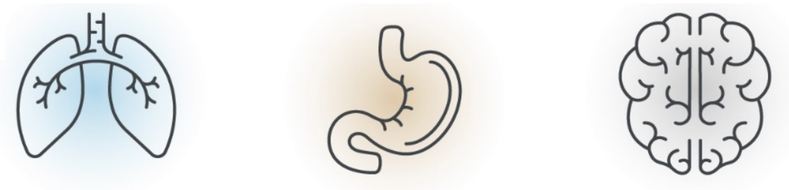
### Lungs

- Endotracheal intubation for patients with West Heaven grade III or IV hepatic encephalopathy
- Lung protective ventilation strategy
- Prone positioning feasible
- Paracentesis in case of tense ascites



### Gastro-intestinal

- Consider stress-ulcer prophylaxis
- Administer early oral or enteral feedings, as tolerated, after ACLF diagnosis (goal: 10-15 kcal/kg/day by day 4)

### Nervous central system

- Treatment of the underlying cause
- Lactulose and enemas for hepatic encephalopathy
- Use sedation protocols, targeting specific endpoints
- Use short-acting sedative agents
- Avoid deep sedation, avoid benzodiazepines
- Avoid neuromuscular in patients without ARDS

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# Acute on Chronic Liver Failure (ACLF)

## Hemodynamics

- Early goal-directed therapy within the first 6 hours
- Maintain mean arterial pressure >65 mmHg
- Fluid challenges until no further hemodynamic response
- Prefer crystalloids and 5% albumin as resuscitation fluid
- Strong indications of albumin: spontaneous bacterial peritonitis, large volume parascentesis, AKI (see kidney)
- Norepinephrine as first line vasopressor; epinephrine or terlipressin when additional agent needed
- Intravenous hydrocortisone if refractory shock (norepinephrine >0.5 mg/kg.min)

- Avoid starches formulations
- Limit saline solutions in patients with ascites or anasarca



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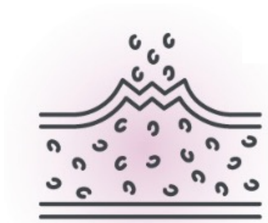

## Coagulation

- Fibrinogen and/or patelets in patients with severe hypofibrinogenemia (<1g/L) and/or thrombocytopenia (<20,000 x$10^9$/L) undergoing invasive procedures
- Prophylaxis for deep-vein thrombosis in patients without severe coagulopathy

- Avoid correction of INR alterations with fresh frozen plasma in the absence of bleeding



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# When to consider Liver Transplantation

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If severe acute alcohol-associated hepatitis is the cause of ACLF, **patients who were actively drinking at time of presentation CAN be considered liver transplant candidates**

- it has to have been their FIRST time presenting with liver disease from alcohol

- cannot be considered for transplant evaluation at that time if known cirrhosis with ongoing alcohol use or prior episodes of acute alcohol-associated hepatitis

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If patients have a known history of alcohol-associated liver disease, once they have achieved nearly 3 months of sobriety, they can be considered for liver transplant

# Liver Transplantation for Severe AH

- To succeed in LT for AH, must improve candidate selection with easily measured pre-LT variables that accurately predict post-LT relapse
- Dallas Consensus Conference 2019 suggested criteria for listing:

| Criteria Related to AH |
|---|
| 1st presentation of decompensated ALD |
| Absence of severe uncontrolled medical or psych comorbidities |
| Non-response to medical tx |

| Criteria Related to AUD | |
|---|---|
| Establish acceptable risk of relapse by multidisciplinary assessment (SW, addiction med) | Accepts ALD dx with insight |
| Coherent pt (not intubated or HE) | Pt commitment to lifelong abstinence |
| No repeated failed rehab attempts | Sober support |
| Lacks current other SUD | Close family, caregivers |



Asrani et al. Liver Transpl. 2019.

# Transitions of Care and Other Pearls

- If patient is listed or undergoing evaluation for liver transplant – ask patient to call us to FYI us OR can always page on-call hepatologist with questions, particularly for our own patients

- Mild to moderate alcohol-associated hepatitis, not requiring steroids/admission – refer to PCP, addiction medicine, hepatology

- Patients with active alcohol use CAN be referred for transplant – inpatient vs outpatient

- Compensated cirrhosis – PCPs vary in comfort with managing compensated cirrhosis – key management includes HCC and EV screening

- Diuretic management – if initiating or increasing, would have PCP repeat labs in 1 week

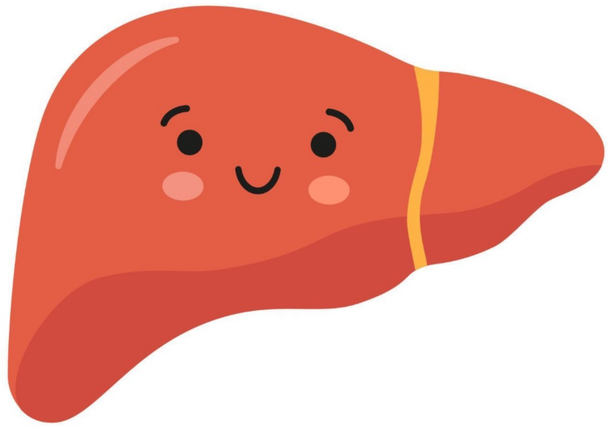
- Can always increase lactulose – ammonia is not helpful to know whether to treat

# Summary

- Common scenarios
  - Acute alcohol-associated hepatitis
  - Ascites
  - Spontaneous bacterial peritonitis
  - Acute on chronic liver failure
- Transitions of care

# Questions?

# Thank you!!



Email: Rebecca.g.kim@hsc.utah.edu

# Additional Slides

# Hepatorenal syndrome

- **Arterial vasodilation in splanchnic circulation** appears to play a central role in the hemodynamic changes and decline in renal function in HRS

| | |
|---|---|
| Progressive rise in serum creatinine | Normal urine sediment |
| No or minimal proteinuria | Urine Na < 10 |
| Oliguria | No response to albumin challenge |

- Diagnosis of exclusion!

# Hepatorenal syndrome

- Type I
- Type II

# Hepatorenal syndrome

- Type I: at least 2-fold increase in serum Cr over 2 weeks to a level of >2.5mg/dL
- Type II: slower progression; major feature is diuretic-resistant ascites

- Must have chronic or acute hepatic disease with advanced liver failure and portal hypertension
- AKI defined as increase in creatinine of ≥0.3 within 48h, or an increase from baseline of 50% or more within 7 days

# Hepatorenal syndrome

- Diagnosis of exclusion!

- Absence of any other apparent cause for AKI (ie shock, recent tx with nephrotoxic drugs including contrast, absence of e/o obstruction or parenchymal disease on ultrasound)

- Urine RBC < 50 and protein < 500
- Lack of improvement after volume expansion with IV albumin
- **Albumin challenge = ?**

# Treatment of hepatorenal syndrome

# Treatment of hepatorenal syndrome

- Albumin 25-50g BID + midodrine 15mg TID + octreotide SC max 200mg TID

- If in ICU, consider norepinephrine (goal to increase MAP by 10)

- Treat for 2 weeks but may need treatment longer if there is some incomplete improvement