GI Alliance

Advancing GI Patient Care 2021

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Management of Complications of Cirrhosis

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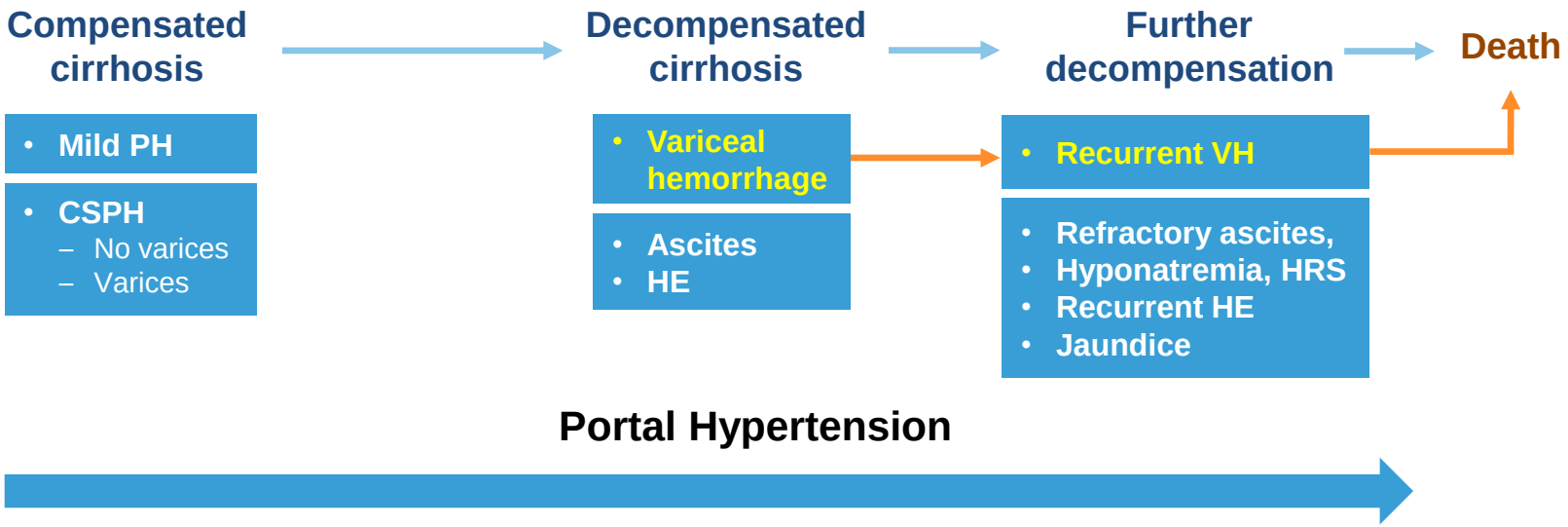
GI Alliance Arizona Digestive Health

Faculty Disclosures

Nayan Patel, DO

- There are no conflicts of interest to disclose

Cirrhosis Natural History



VH = variceal hemorrhage; HE = hepatic encephalopathy; HRS = hepatorenal syndrome
AASLD Guidance. Garcia-Tsao et al. *Hepatology*. 2017; 65: 310-335.

Complications of Portal Hypertension

- Gastroesophageal Varices
- Ascites
- Hepatic Hydrothorax
- Hepatic Encephalopathy
- AKI/HRS
- Hyponatremia
- HPS
- PoPH
- Cirrhotic cardiomyopathy

HEPATOLOGY

AASLD
AMERICAN ASSOCIATION FOR THE STUDY OF LIVER DISEASES

PRACTICE GUIDANCE | Free Access

Diagnosis, evaluation, and management of ascites and hepatorenal syndrome

Scott W. Biggins, Paulo Angeli, Guadalupe Garcia-Tsao, Pere Ginès, Simon Ling, Mitra K. Nadim, Florence Wong, W. Ray Kim

First published: 03 May 2021 | https://doi.org/10.1002/hep.31884

Classification of Ascites

According to Amount of Ascites		According to the Response to Treatment	
Grade 1. Mild ascites	Only detected by ultrasound	**Responsive ascites**	Ascites that can be fully mobilized or limited to grade 1 with diuretic therapy associated or not to moderate dietary sodium restriction
Grade 2. Moderate ascites	Moderate symmetric distension of abdomen	**Recurrent ascites**	Ascites that recurs on at least 3 occasions within a 12-month period despite dietary sodium restriction and adequate diuretic dosage
Grade 3. Large or gross ascites	Marked distension of the abdomen	**Refractory ascites**	Ascites that cannot be mobilized or the early recurrence of which (i.e., after LVP) cannot be satisfactorily prevented by medical therapy

Refractory Ascites

- Ascites that cannot be mobilized or recurs after LVP despite dietary sodium restriction and diuretic therapy
- 5%-10% of all cirrhotic patients with ascites
  - P**oor survival of 50% at 6 months**
- **Diuretic resistant** – lack of response to sodium restriction and diuretics
- **Diuretic intractable** – development of diuretic induced complications
  - Renal impairment: increase in serum creatinine by >100% to a value >2.0 mg/dL
  - Hyponatremia with a decrease of >10 mmol/L or an absolute value of <125 mmol/L
  - Hypo- or hyperkalemia of <3 mmol/L or >6 mmol/L
  - Hepatic encephalopathy

Refractory Ascites – Guidance Statements

- LVP is the first-line treatment for RA.
- Albumin infusion at the time of LVP of >5 L is recommended to mitigate the risk of PPCD (post paracentesis circulatory dysfunction).
- The recommended dose of albumin replacement, based on expert opinion, is 6-8 g for every liter of ascites removed.
- Fluid restriction is ineffective for the management of refractory ascites (RA).
- Based on currently available data, NSBBs are not necessarily contraindicated in patients with RA.
- However, caution is recommended in patients with hypotension, hyponatremia, or AKI.

Hyponatremia

- Hypervolemic hyponatremia
  - Mild hyponatremia (Na 126-135 mEq/L) in cirrhosis without symptoms does not require specific management apart from monitoring and water restriction.
  - Moderate hyponatremia (120-125 mEq/L) - Water restriction to 1,000 mL/day and cessation of diuretics is recommended.
  - Severe hyponatremia (<120mEq/L) - more severe restriction of water intake with albumin infusion is recommended.
  - Correction of hypokalemia aids correction of hyponatremia via improved cellular Na-K exchange
  - The use of vasopressin receptor antagonists in cirrhosis can raise serum sodium during treatment. However, they should be used with caution only for a short term (≤30 days).

Hyponatremia

- Hypovolemic hyponatremia – overdiuresis, poor oral intake, GI losses
  - D/C diuretics, laxative
  - Provide fluid resuscitation 5% IV albumin or crystalloid (lactated Ringers preferred)
- Euvolemic hyponatremia – SIADH, meds (sertraline, carbamazepine), adrenal ↓, hypothyroid
  - Address specific cause

SBP

- NSBBs should be temporarily held in patients with SBP who develop hypotension (mean arterial pressure <65 mmHg) or AKI.
- Patients who have recovered from an episode of SBP should receive long-term prophylaxis with daily norfloxacin.
  - Oral ciprofloxacin is acceptable
- In patients with cirrhosis and low protein (<1.5 g/L) ascites, primary SBP prophylaxis can be considered in selected patients with renal dysfunction (serum creatinine level >1.2 mg/dL, blood urea nitrogen level >25 mg/dL, or serum sodium level <130 mEq/L) or liver failure (Child-Pugh-Turcotte score >9 and bilirubin >3 mg/dL).

AKI/HRS

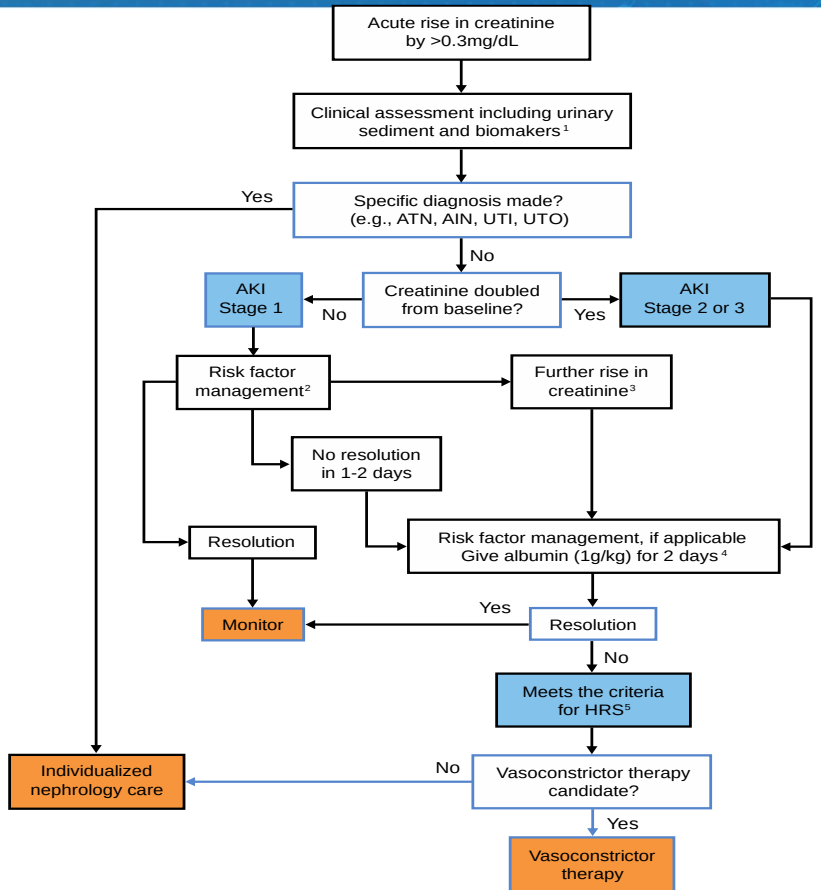
AKI Stage	Description
Stage 1	Increase of creatinine ≥0.3 mg/dL up to 2-fold of baseline
Stage 2	Increase in creatinine between 2-fold and 3-fold of baseline
Stage 3	Increase in creatinine >3-fold of baseline or creatinine >4 mg/dL (353.6 µmol/L) with an acute increase ≥0.3 mg/dL (26.5 µmol/L) or initiation of renal replacement therapy

- HRS 1 is now HRS-AKI
- HRS 2 now is part of CKD.

AKI/HRS

- Once AKI is diagnosed, an investigation to uncover and treat precipitating factors must be conducted swiftly.
  - Relevant risk factors include fluid losses, bacterial infections, hemodynamic instability, and potentially nephrotoxic agents (e.g., particularly nonsteroidal anti-inflammatory drugs).
- Hypovolemia-induced AKI should be managed with fluid replacement therapy, correction of the cause that led to volume depletion, and diuretic withdrawal.

AKI/HRS


Acute rise in creatinine by >0.3mg/dL
Clinical assessment including urinary sediment and biomakers[1]
Specific diagnosis made? (e.g., ATN, AIN, UTI, UTO)
Yes
No
Creatinine doubled from baseline?
No
Yes
AKI Stage 1
AKI Stage 2 or 3
Risk factor management[2]
Further rise in creatinine[3]
No resolution in 1-2 days
Resolution
Risk factor management, if applicable
Give albumin (1g/kg) for 2 days[4]
Yes
Monitor
Resolution
No
Meets the criteria for HRS[5]
No
Individualized nephrology care
Vasoconstrictor therapy candidate?
Yes
Vasoconstrictor therapy

HRS

- The treatment of choice for HRS-AKI is vasoconstrictor drugs in combination with albumin. The preferred drug is terlipressin, administered either as IV bolus or continuous IV infusion.
- In settings where terlipressin is not available, norepinephrine should be given. If neither can be administered, a trial of oral midodrine (5 to 15 mg po every 8 hours) in combination with octreotide (100 to 200 µg every 8 hours or 50 µg/hour IV) may be considered, yet the efficacy is low.

NAFLD and HCC

CLINICAL PRACTICE UPDATE

AGA Clinical Practice Update on Screening and Surveillance for Hepatocellular Carcinoma in Patients With Nonalcoholic Fatty Liver Disease: Expert Review

Rohit Loomba,[1,2] Joseph K. Lim,[3] Heather Patton,[4,5] and Hashem B. El-Serag[6]

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Gastroenterology. 2020; 158: 1822-1830.

Best Practices Takeaways

1. HCC screening in all cirrhosis from NAFLD
2. HCC screening in NAFLD patients with non-invasive markers of advanced fibrosis (on at least 2 non-invasive imaging modalities)
   - Point of care tests (FIB-4)
   - Specialized blood tests (Liver Fibrosis Panels)
   - Non-invasive imaging tests (elastography based tests)
3. When the quality of US is suboptimal for screening of HCC (due to obesity), future screening should be performed with either CT or MRI +/- AFP

Sarcopenia

Sarcopenia

- Disproportionate loss of muscle mass
- Main indicator of adverse outcomes
  - Hepatic decompensation,
  - Mortality in patients with cirrhosis evaluated for LT,
  - Longer hospital and intensive care unit stay,
  - Higher incidence of infection following LT,
  - Independent risk factor for HE
- Interventions for clinical practice
  - **Daily protein intake of 1.2-1.5grams/kg protein**
  - Late night snack 10-20 grams protein
  - Moderate intensity exercise
  - Screen for Vit D deficiency

Carey et al. A North American Expert Opinion on Sarcopenia in Liver Transplantation. *Hepatology*. 2019.

CLINICAL PRACTICE UPDATE

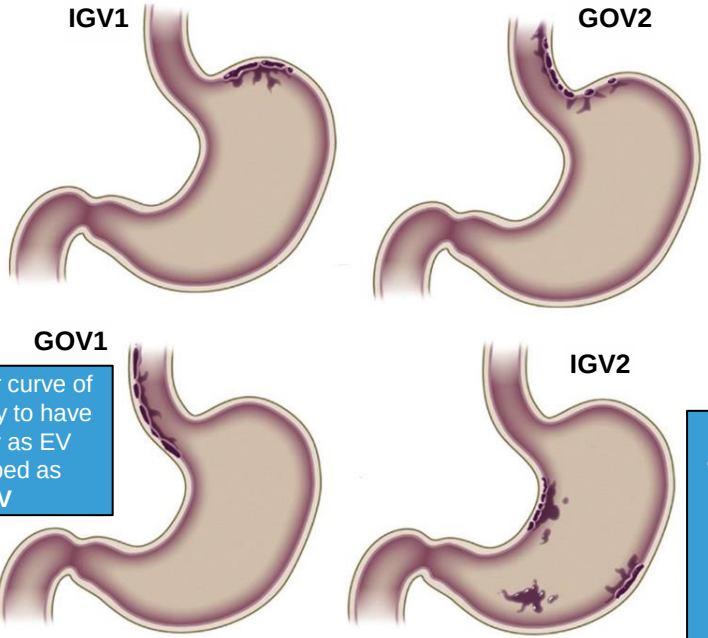
AGA Clinical Practice Update on Management of Bleeding Gastric Varices: Expert Review

Zachary Henry,[*] Kalpesh Patel,[‡] Heather Patton,[§] and Wael Saad[‖]

[*]Division of Gastroenterology and Hepatology, Department of Medicine, University of Virginia, Charlottesville, Virginia; [‡]Division of Gastroenterology and Hepatology, Department of Medicine, Baylor College of Medicine, Houston, Texas; [§]Gastroenterology Section, VA San Diego Healthcare System, San Diego, California; and [‖]Interventional Radiology, Radiology and Imaging Sciences, National Institutes of Health, Washington, DC

Clinical Gastroenterology and Hepatology. 2021; 19: 1098-1107.

GV located on the posterior and/or greater curvature side of the cardia are likely to have a distinct vascular supply from EV and are better described as **Cardiofundal GV**



GV located on the lesser curve of the cardia are more likely to have similar vascular supply as EV and are better described as **Lesser Curve GV**

GV in the gastric body and antrum are better described as **Distal GV**. These are rare, often associated with splenic vein thrombosis and are managed differently compared to GV in other sites.[13]

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Thank you!