Caring for the patient with cirrhosis Role of the hospitalist

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Disclosure

Grant/research support: Grifols

Topics to be covered

Evaluation and management of hepatic decompensation
Hepatic encephalopathy
Gastrointestinal bleeding
Ascites
Hepatorenal syndrome
Acute on chronic liver failure
Liver transplant evaluation basics

What will not be covered

Acute liver failure
Management of alcoholic hepatitis
Hepatocellular carcinoma diagnosis and management

 57yo man with alcoholic cirrhosis presents with altered mental status

His family brought him in because he was staring blankly at them when they asked him questions and seemed unable to feed himself

His family brought him in because he was staring blankly at them when they asked him questions and seemed unable to feed himself

T 37 HR 75 BP 112/73 RR 12 SpO2 97%

 Slow to respond but awake, oriented to first name only and keeps repeating that despite other questions asked.
 +asterixis

Icteric sclerae

Nontender abdomen with bulging flanks

WBC 4, hct 29, plts 85, INR 1.8, Na 136, Cr 0.8, tbili 6.3

Hepatic encephalopathy (HE)

Presents with a spectrum of symptoms

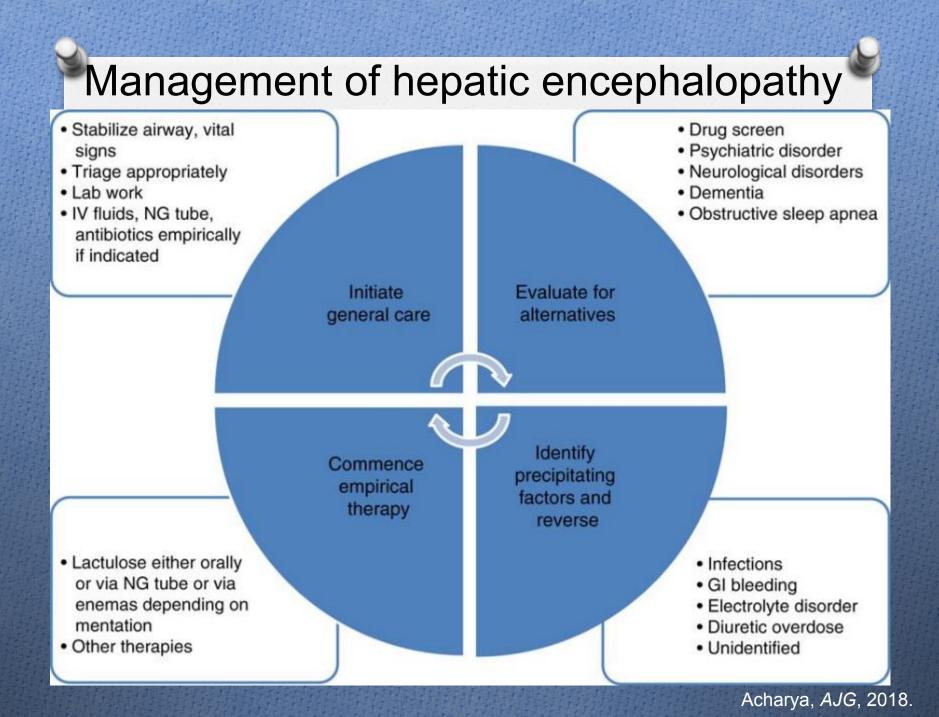
Covert/minimal

Overt: change in attention, sleep→disorientation, asterixis, lethargy→coma

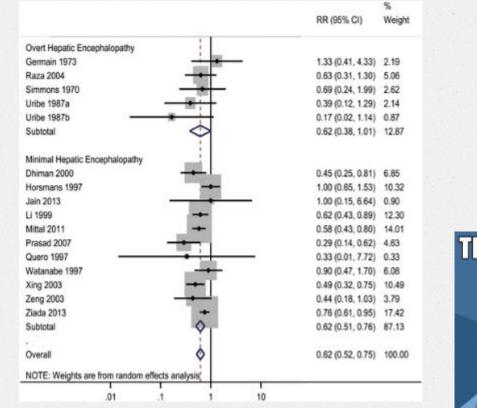
Overt hepatic encephalopathy (OHE) will occur in 30-40% of all patients with cirrhosis

Recurrent OHE risk is 40% at 1 year

Subsequent recurrence is 40% at 6 months



Nonabsorbable disaccharides





Gluud, Hepatology, 2016.

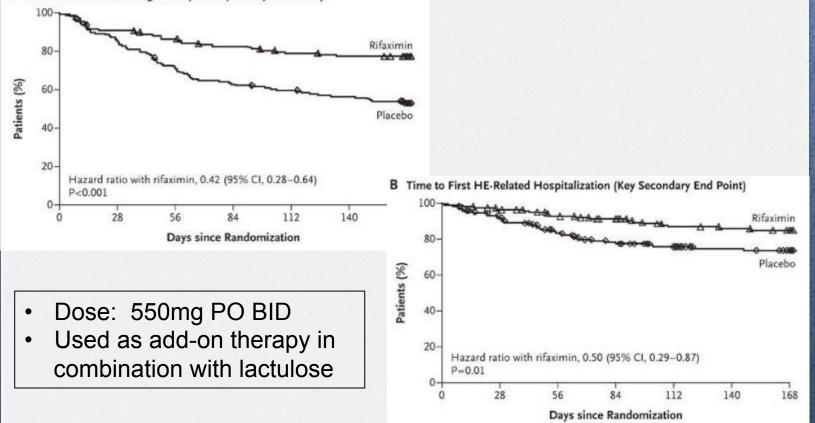
LACTULOSE SOLUTION, USP 10 g/15 mL

A second second

Qualitest*

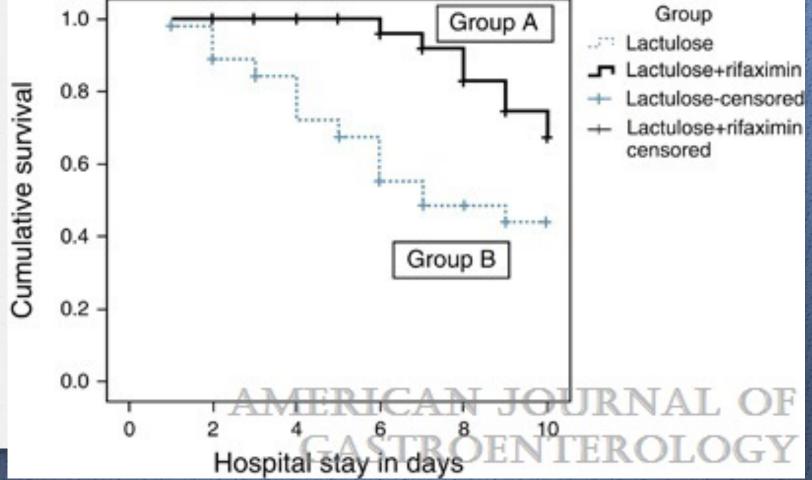
Rifaximin reduces HE recurrence and need for hospitalization

A Time to First Breakthrough HE Episode (Primary End Point)

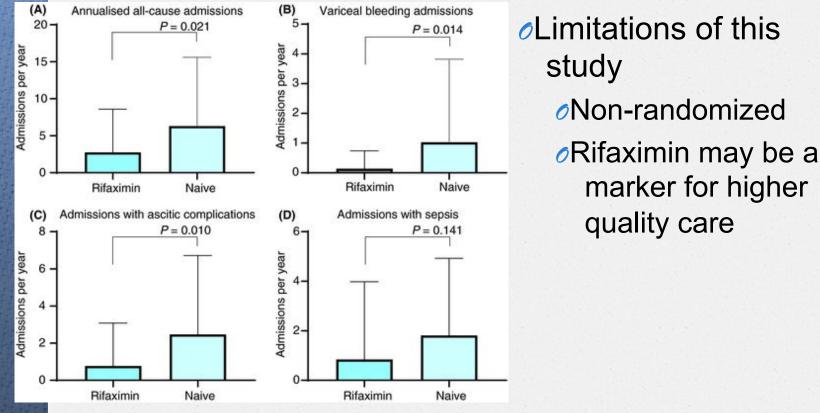


Bass, NEJM, 2010.

Lactulose + rifaximin is more effective than lactulose alone

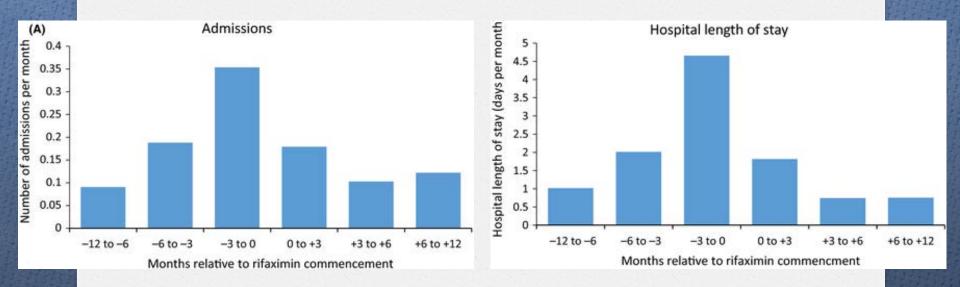


Impact of rifaximin may extend beyond HE



Rifaximin reduces cost

Several studies have demonstrated potentially favorable cost effectiveness



Neff, *PharmacoEconomics*, 2018. Orr, *Liver International*, 2016.

Other HE treatments of interest

Polyethylene glycol (GoLytely)
L-ornithine-I-aspartate (LOLA)
Glyceryl phenylbutyrate
Fecal microbiota transplant
Probiotics

 Transvenous obliteration of portosystemic shunts

(Neomycin, metronidazole)

Nutritional status and HE

It is important to do a nutritional assessment on patients with HE

Subjective global assessment (lacks sensitivty)Grip strength

Protein restriction should be avoided

1.2-1.5g/kg ideal body weight recommended
 Avoid fasting >3-6 hours during the day
 Small, frequent meals

Late evening snack



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Hepatic encephalopathy Summary

Precipitants of overt hepatic encephalopathy should be investigated
Lactulose is the cornerstone of HE management
Rifaximin should be used as add on therapy and reduces cost of care
Protein restriction should be avoided

63M with cirrhosis due to autoimmune hepatitis presents with complaints of several episodes of melena x 1 day

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Recent onset ascites and jaundice

63M with cirrhosis due to autoimmune hepatitis presents with complaints of several episodes of melena x 1 day

Recent onset ascites and jaundice

VS: HR 120 BP 95/63 RR 20 SpO2 95%

Gen: uncomfortable, lethargic

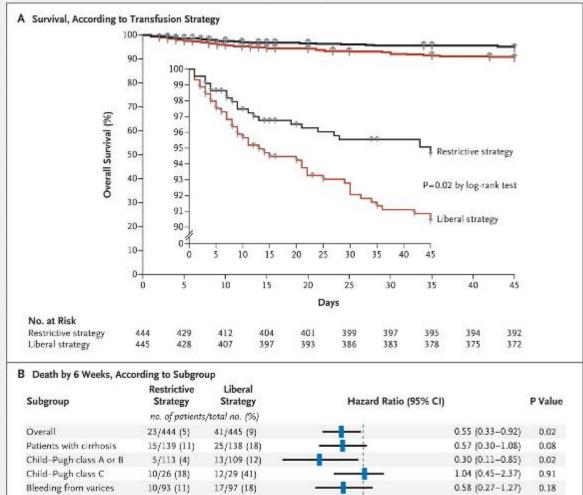
Abd: distended, bulging flanks, mildly uncomfortable to palpation but no peritoneal signs. +melenic stool
Labs: WBC 4, Hb 5.7, plts 80, INR 1.6, Na 136, Cr 0.9, total bili 4.3

Management of GI bleeding in cirrhosis

OABCs

- Type and cross pRBCs +/- FFP and platelets
- Octreotide
- ⊘PPI IV

Transfuse to a goal Hb 7-9g/dL



1.0

Restrictive Strategy Liberal Strategy

Better

Better

11/209 (5)

0.1

7/228 (3)

Bleeding from peptic ulcer

Villanueva, NEJM, 2013.

0.70 (0.26-1.25)

10.0

0.26

No definitive data on INR or platelet goals

- INR is a poor predictor of bleeding (or clotting) risk in cirrhosis
- Recombinant factor VIIa not clearly beneficial
- No guidance available on platelet goal

Octreotide reduces mortality and need for transfusion

Octreotide dosing

 Initial bolus of 50 µg (repeat in first hour if ongoing bleeding)

- Continuous IV infusion of 50 µg/hr for up to 5 days
- Ouse of vasoactive agents reduces 7-day mortality by 36%

32% decreased risk of rebleeding

Blood transfusion requirement 0.7 units lower n patients receiving vasoactive agents

Wells, *Alim Pharm Ther*, 2012. Garcia-Tsao, *Hepatology*, 2016.

Antibiotics improve outcomes in GI bleeding in cirrhosis

Risk of infection after GI bleeding may be as high as 35-66% within 2 weeks

Meta-analysis demonstrated reduced risk of infection compared with placebo

Any infection: 14% vs 45%

First line antibiotic choice: ceftriaxone

Bernard, *Hepatology*, 2003. Garcia-Tsao, *Hepatology*, 2016.

Predictors of poor outcome after variceal bleeding

Child-Pugh class

⊘AST

Shock on admission

Portal vein thrombosis

OHCC

10-15% of patients with have persistent and/or early rebleeding

Active bleeding at endoscopy
Hepatic venous pressure gradient >20
MELD

Avgerinos, *Hepatoloogy*, 2004. Reverter, *Gastroenterology*, 2013. Ripoll, *Hepatology*, 2005. Thomopoulos, *Dig Liver Dis*, 2006. Bambha, *Gut*, 2008. Lecleire, *J Clin Gastro*, 2005.

Endoscopic therapy in variceal bleeding

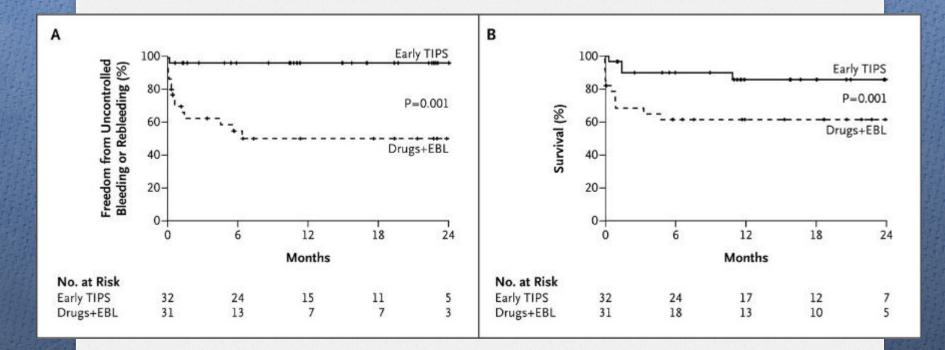
Band ligation within 12 hours considered standard of care for esophageal varices Other modalities (Hemostatic powder/spray) Esophgeal stent (Sclerosants) Gastro-esophageal balloon tamponade Treatment for gastric varices: cyanoacrylate injection +/- coil



Ibrahim, Gastro, 2018.

Ibrahim, Gut, 2018. Pfisterer, *Liver Int*, 2018.

Early TIPS in variceal bleeding



Careful patient selection is critical

Garcia-Pagan, NEJM, 2010.

Recurrent variceal bleeding risk is 60% in the first year, and up to 33% mortality

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- Nonselective beta blockers (NSBB) should be initiated
 - Combination of NSBB + band ligation is superior to either alone

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Consider PPI for 10 days post-banding

Garcia-Tsap, *Hepatology*, 2016. Shaheen, *Hepatology*, 2005.

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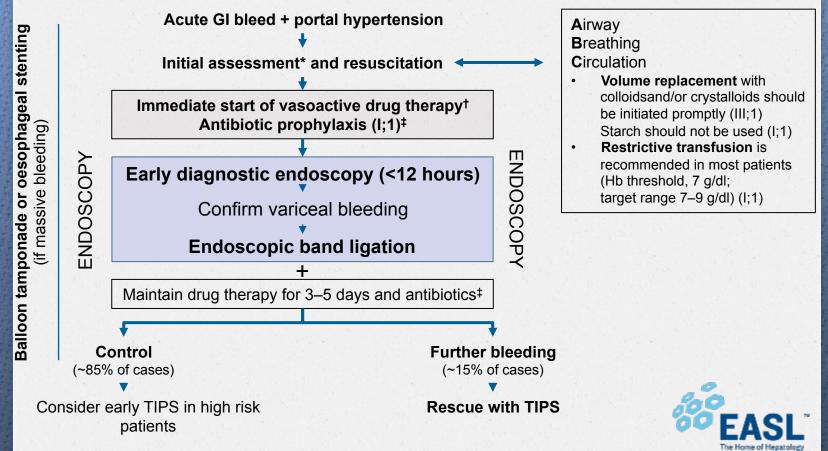
Endoscopy should be repeated every 1-4 weeks until varices eradicated

- Consider PPI for 10 days post-banding
- TIPS for recurrent bleeding

Acute variceal bleeding Summary

Medical emergency: high rate of complications and mortality in DC

Requires immediate treatment and close monitoring



VS: T37 HR 65 BP 110/70 RR 20 SpO2 98%
Gen: chronically ill, slightly uncomfortable due to abdominal distension

 Resp: normal other than decreased BS at bases
 GI: tensely distended abdomen with dullness to percussion, nontender

Neuro: AAOx3, no asterixis

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 Gen: chronically ill, slightly uncomfortable due to abdominal distension

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Neuro: AAOx3, no asterixis

Labs: WBC 5, hct 30, plt 70, INR 1.5, Na 130, Cr 0.7, total bili 5, albumin 3.0

Ascites: Diagnostic tests

Abdominal ultrasound: confirm ascites, eval portal and hepatic vein patency, r/o HCC

Diagnostic paracentesis

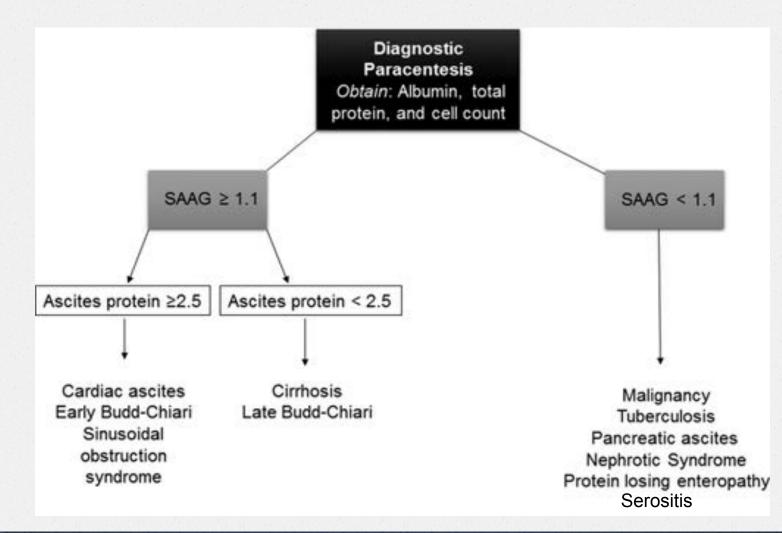
- Complication rate: 1%; <0.1% risk of hemoperitoneum or bowel entry)</p>
- Routine fluid analysis: cell count with differential, albumin, total protein, culture
 - Serum to ascites albumin gradient (SAAG)
 - Ouse of blood culture bottles with higher culture yield
- Additional fluid analysis: LDH, glucose, CEA, alkaline phosphatase, cytology, AFB culture, triglycerides, bilirubin, creatinine

Paracentesis

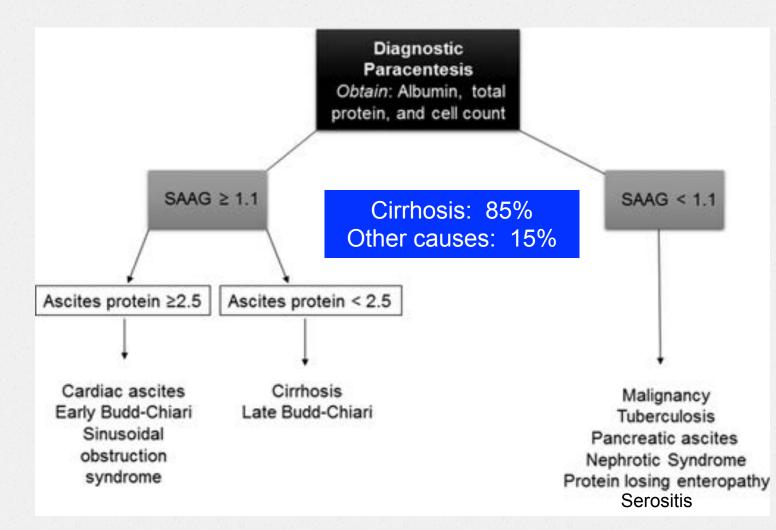
Diagnostic paracentesis 1st paracentesis Cell count w/ diff *o*Culture **Albumin** Total protein Additional studies as guided by clinical presentation Subsequent paracentesis: cell count w/ diff and culture



Ascites: diagnosis by SAAG



Ascites: diagnosis by SAAG



Hernaez, Clin Liver Dis, 2016.

Case 3 (cont'd)

- OUS: Coarse, nodular liver without focal mass. Splenomegaly. Patent portal and hepatic veins. Large ascites
- Paracentesis with removal of 5L amber fluid
 WBC 893 (75% PMNs), RBC 100
 Albumin 1.0, total protein 1.2
 Cultures pending

Spontaneous bacterial peritonitis (SBP)

 o~30% of patients with SBP may lack typical signs/symptoms of fever, abdominal pain, and/or leukocytosis

Diagnosis: 250 PMNs/mm³

Prognosis

In-hospital death: 10-20%

Median survival: 9 months

Recurrent SBP: 40-70% at 1 year

Garcia-Tsao, Sherlock's Dis of the Liver and Biliary System, 2011.

Management of SBP Key principles

Treatment of infection
Prevention of hepatorenal syndrome/AKI
Assessment of response to treatment
Prevention of recurrent infection

Antibiotic therapy for SBP

Community acquired"

•Typical bacteria: E. coli, K. pneumoniae,

streptococcus

 ^o3rd gen cephalosporin or fluoroquinolone for 5-7d

"Nosocomial"

 Specific choice of antimicrobial should be guided by local flora and resistance patterns

RCT: Meropenem+dapto vs ceftazidime had higher rates of response, but no substantial impact on survival

> Runyon, *Hepatology*, 2012. Piano, *Hepatology*, 2016.

Infections with antibiotic resistant organisms

Risk factors

Prior exposure to antibiotics within 30 days of diagnosis of AR infection

Nosocomial infection

Prior infection with AR organisms within 6 months

Impact on outcome

Lower rate of infection resolution

Increased risk of in hospital mortality

Tandon P, *Clin Gastro Hep*, 2016. Fernandez J, *Hepatology*, 2011.

Prevention of HRS in SBP

RCT of 126 patients with SBP treated with cefotaxime, albumin vs no albumin

1.5g/kg on day 1, 1g/kg on day 3

an)	Albumin (Hum 25%, USP
-	Plasbumin®-25 20 mL Ingle Dose Vial
e cisk	Smy Meris Biotherapeutics, Inc. Research Triangle Park, NC 2710 U.S. Ucense No. 1716
	Pesearch Triangle Park, NC 2770

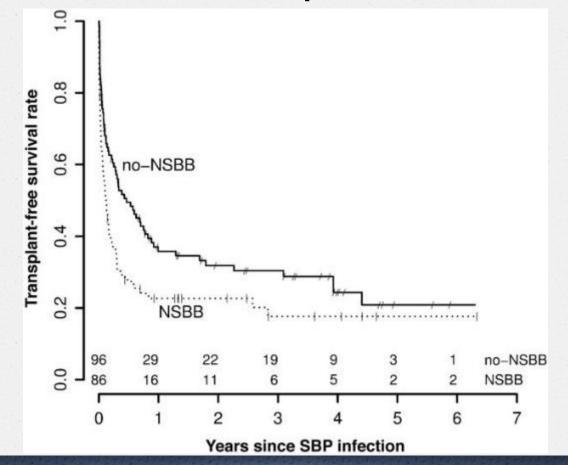
	Albumin	Control	p value
Renal impairment	10%	33%	0.002
Death In hospital 3 months	10% 22%	29% 41%	0.01 0.03

Impact may be greatest in patients with Cr>1, BUN>30, and/or tbili >4

> Sort P et al, *NEJM*, 1999. Sigal et al, *Gut*, 2007.

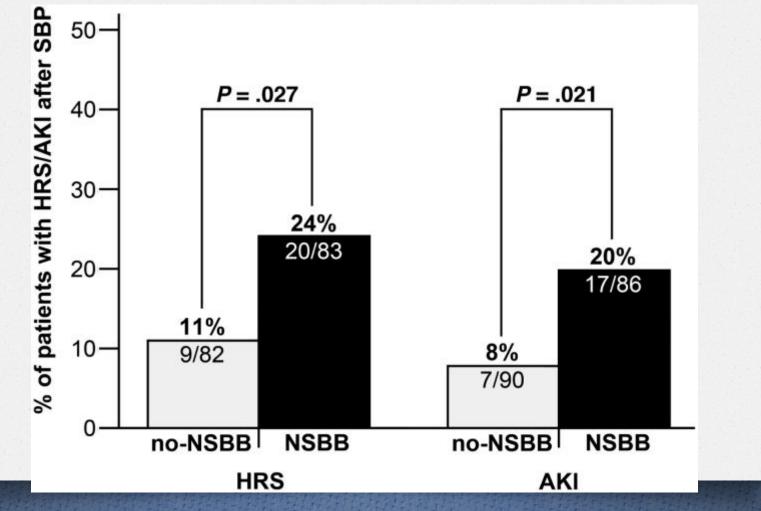
What about beta blockers?

Beta blockers increase risk of death after first episode of SBP



Mandorfer, Gastro, 2014.

Beta blockers increase risk of HRS/ AKI after first episode of SBP



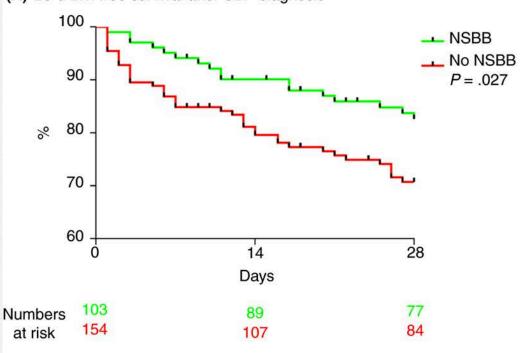
Mandorfer, Gastro, 2014.

Beta blockers increase risk of HRS/AKI after first episode of SBP

Patients treated with beta blockers
 More often Child's C cirrhosis (67 vs 53%)
 Higher bilirubin (5 vs 3)
 MELD similar between groups

Or do they?

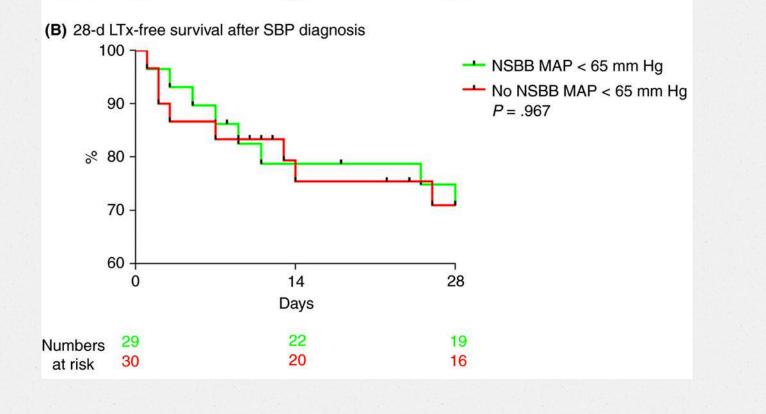
NSBB may be associated with improved survival



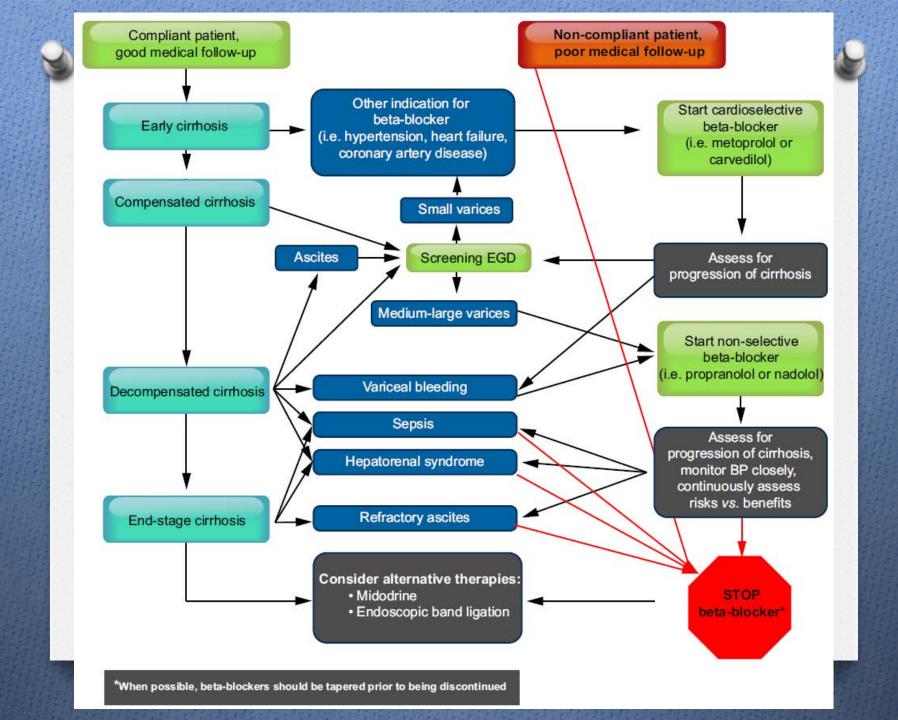
(A) 28-d LTx-free survival after SBP diagnosis

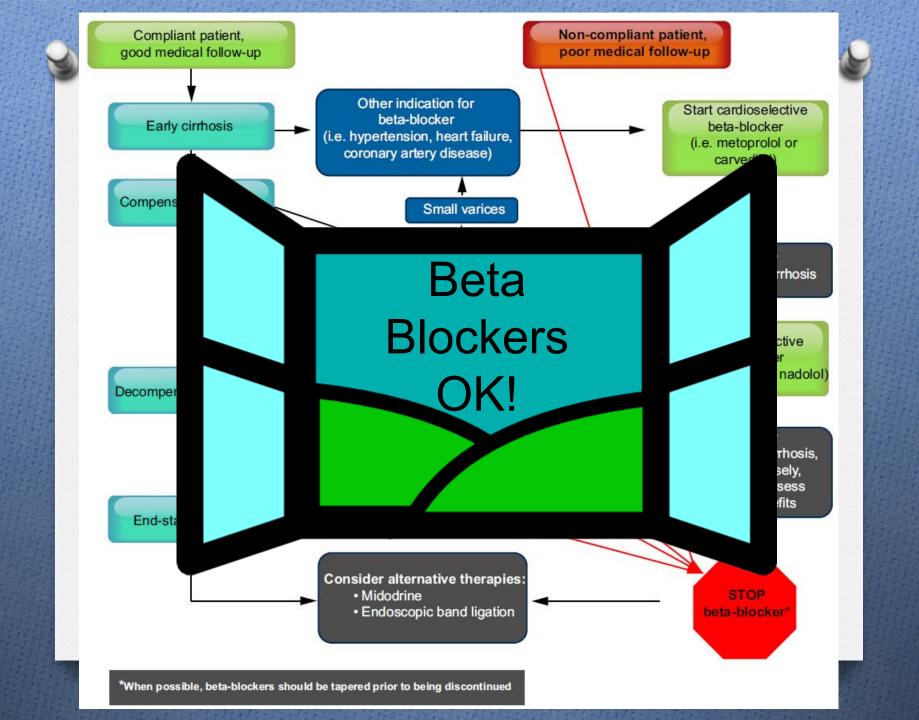
Tergast, AP&T, 2019.

Benefit of NSBB lost if MAP<65



Tergast, AP&T, 2019.







SBP prophylaxis

Indications

 ⊘Primary prophylaxis: low-protein ascites (<1.5) + impaired renal function, Child's C cirrhosis/bilirubin ≥3
 ⊘Secondary prophylaxis

	Antibiotics	Control	RR (95% CI)	ARR/NNT
Overall mortality	16%	25%	0.65 (0.48-0.88)	9%/11
3-month mortality	6.2%	22.3%	0.28 (0.12-0.68)	16.1%/6
Long-term mortality	19.9%	28.5%	0.71 (0.49-1.04)	8.5%/12
SBP	12.7%	25%	0.49 (0.35-0.69)	12%/8

Ascites Summary

- Ascites in a hospitalized patient should be evaluated
 - Diagnostic paracentesis to establish etiology (1st paracentesis) and rule out infection (all paracentesis)
- SBP should be treated with antibiotics and IV albumin
- SBP prophylaxis should be prescribed for primary and secondary prophylaxis

 54yo woman with NASH cirrhosis is advised by her hepatologist to go to the ED due to abnormal labs

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She has refractory ascites, and requires therapeutic paracentesis every 2 weeks

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She has refractory ascites, and requires therapeutic paracentesis every 2 weeks
VS: T 37 HR 80 BP 109/65 RR 12 SpO2 98%
Abd: Distended with dullness to percussion
CBC at baseline, Na 131, Cr 1.8, tbili 6, INR 2
Baseline Cr 0.6

AKI in cirrhosis International Ascites Club criteria

Subject	Definition			
Baseline sCr	 sCr obtained within 3 months prior to admission If >1 value within the previous 3 months, the value closest to the admission If no previous sCr, the sCr on admission should be used 			
Definition of AKI	 Increase in sCr ≥0.3 mg/dl (≥26.5 µmol/L) within 48 hours or Increase sCr ≥50% within the prior 7 days 			
	Stage 1A (sCr <1.5mg/dl)* Stage 1B (sCr ≥1.5mg/dl)*			
Staging of AKI	 Stage 2: increase in sCr >2-fold to 3-fold from baseline Stage 3: increase of sCr >3-fold from baseline or sCr ≥4.0 mg/dl (353.6 µmol/L) w acute increase ≥0.3 mg/dl (≥26.5 µmol/L) or initiation of renal replacement therapy 			
Progression of AKI	Progression Progression of AKI to a higher stage and/or need for RRT		Regression Regression	n of AKI to a lower stage
Response to treatment	No response No regression of AKI	egression of Regression of AKI stage with a reduction of sCr to ≥0.3 mg/dl (≥26.5 µmol/L) above baseline		Full response Return of sCr to a value within 0.3 mg/dl (≥26.5 µmol/L) of baseline

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Management of AKI

Investigate non-HRS causes:

 Review medication history: diuretic dose change or initiation, NSAIDs or other nephrotoxic drugs, iodinated contrast
 Urinalysis with microscopy

Renal ultrasound

Evaluate for infection

Administer volume expansion: IV albumin 1g/kg x 2 days

Hepatorenal syndrome (HRS) International Ascites Club Criteria

Cirrhosis with ascites AKI as defined by ICA-AKI criteria No response after 2 consecutive days of diuretic withdrawal and volume expansion Absence of shock No nephrotoxins No signs of structural kidney injury Ourine protein <500mg/day</p> No microscopic hematuria Normal renal ultrasound

Hepatorenal syndrome (HRS) International Ascites Club Criteria Type 1 HRS: HRS-AKI

Type 2 HRS: renal impairment meets HRS criteria but not AKI

Hepatorenal syndrome

Occurs in ~20% of patients with advanced liver disease
Poor prognosis

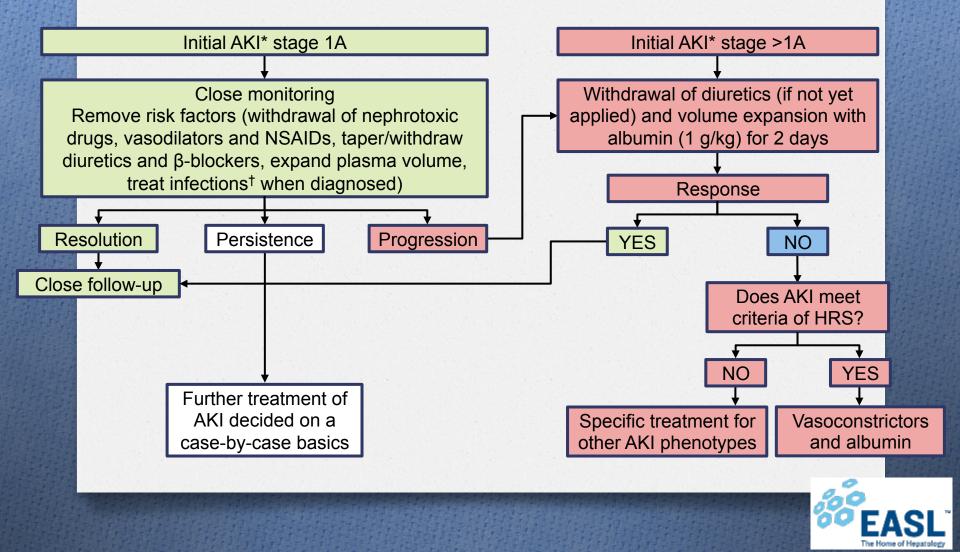
Median survival 8-10 days
3-month survival: 15%

Common precipitants: infection, GI bleeding, LVP
Can be reversible with timely liver transplantation

Treatment of hepatorenal syndrome

 Vasoconstriction of systemic and splanchnic circulation to improve effective circulating volume and renal perfusion
 Drugs studied include midodrine + octreotide, norepinephrine, or terlipressin
 Most recent meta-analyses suggest terlipressin superior to placebo with resolution of HRS in 40-50%
 Albumin dose of 20-40g/day

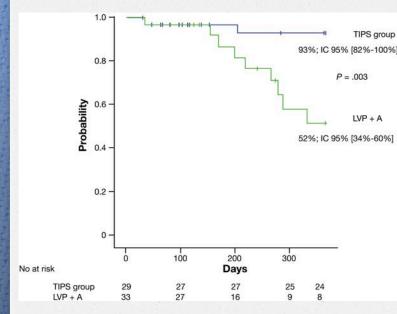
ICA management of AKI in cirrhosis



Refractory ascites

 Definition: ascites that is resistant to diuretics <u>OR</u> management with diuretics results in complications that prevent diuretic dose increase
 Median survival 6 months

TIPS vs. serial paracentesis



LT-free Survival

Predictors of mortality

	HR	95% CI
TIPS	0.61	0.41-0.91
Age	1.024	1.001-1.048
Bilirubin	1.22	1.029-1.46
Sodium	0.95	0.92-0.99

Bureau, Gastro, 2017.

Boyer, *Hepatology*, 2010. Salerno, *Gastro*, 2007.

TIPS vs. serial paracentesis

Incidence of hepatic encephalopathy is similar between TIPS vs paracentesis groups, though severe HE may be more common with TIPS

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Boyer, *Hepatology*, 2010. Salerno, *Gastro*, 2007.

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Most other portal hypertensive complications improve with TIPS

	TIPS	LVP
Total	15%	28%
GI bleeding	8%	13%
SBP	2%	3%
HRS	5%	13%

Bureau, Gastro, 2017.

Boyer, *Hepatology*, 2010. Salerno, *Gastro*, 2007.

Contraindications to TIPS

Relative	Absolute	
Hepatocellular carcinoma, especially centrally located	Primary prevention of variceal bleeding	
Obstruction of all HVs	Congestive heart failure	
PV thrombosis	Severe tricuspid regurgitation	
Moderate pulmonary hypertension	Severe pulmonary hypertension	
Severe coagulopathy (international normalized ration >5)	Multiple hepatic cysts	
Thrombocytopenia of <20,000 cells/cm ³ Hepatic encephalopathy	Uncontrolled systemic infection or sepsis Unrelieved biliary obstruction	

MELD >15-18 and/or total bilirubin >3

Patidar, Clin Liver Dis, 2014.

Case 5

60F with NASH cirrhosis presents with jaundice and worsened fluid retention

Case 5

 60F with NASH cirrhosis presents with jaundice and worsened fluid retention
 Exam:

VS: T 38, HR 110, BP 95/50, RR 20, 97%RAJaundiced

Abdominal distension with dullness to percussion

Confused, slow to respond

Case 5 (cont'd)

	6 weeks ago	Current presentation	
INR	1.3	2.5	
Na	140	134	
Cr	0.6	2.3	
Total bilirubin	1.0	5.2	
Albumin	4.0	3.3	
MELD-Na	9	32	

Acute on Chronic Failure

Acute on Chronic Failure: Consensus Definition

"A syndrome in patients with chronic liver disease with or without previously diagnosed cirrhosis which is characterized by acute hepatic decompensation resulting in:

1) liver failure (jaundice and elevated INR) and

2) one or more extrahepatic organ failures that is associated with increased mortality within a period of 28 days and up to 3 months from onset"

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Acute on Chronic Liver Failure (ACLF)

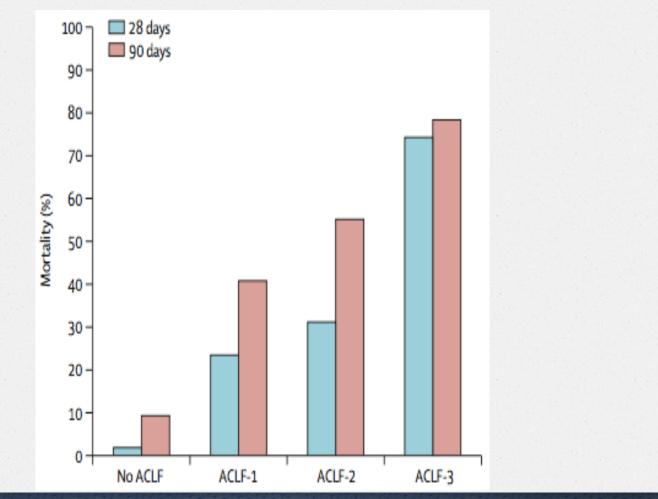
32,335 hospitalizations for ACLF per year
Mortality 50% (previously 65%)
Mean length of stay: 16 days
Indicates need for liver transplantation
Presence may increase risk of posttransplant morbidity and mortality

> Allen, *Hepatology* 2016. Huebener, *J Hepatol*, 2018.

Chronic Liver Failure Consortium Organ Failure Score (CLIF score)

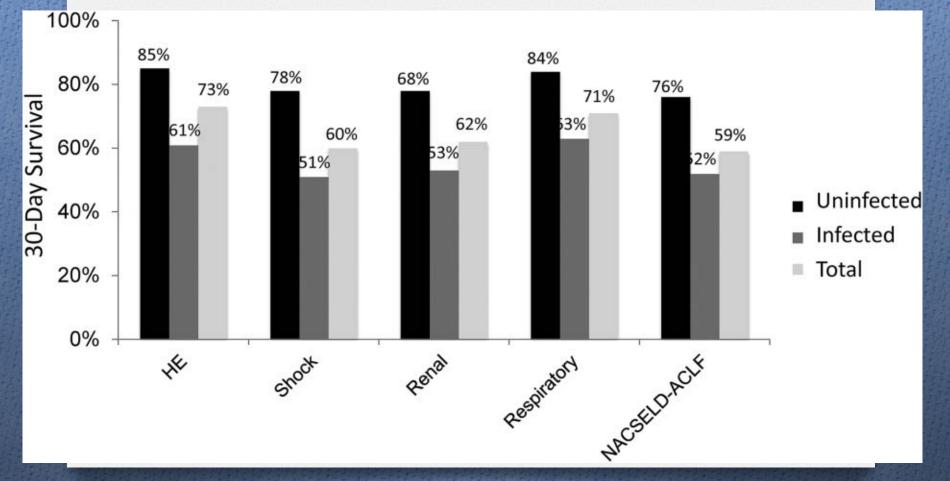
Organ Sustam	Score			
Organ System	1	2	3	
Liver	6.0 mg/dL			
Renal	Cr 2.0 mg/ dL		RRT	
Neurologic	Hepatic encephalopathy grade			
Hematologic	INR 2.0			
Circulatory	MAP <70		Vasopressors	
Respiratory	PaO2/FiO2 <	<300		

ACLF strongly predicts 28- and 90-day mortality



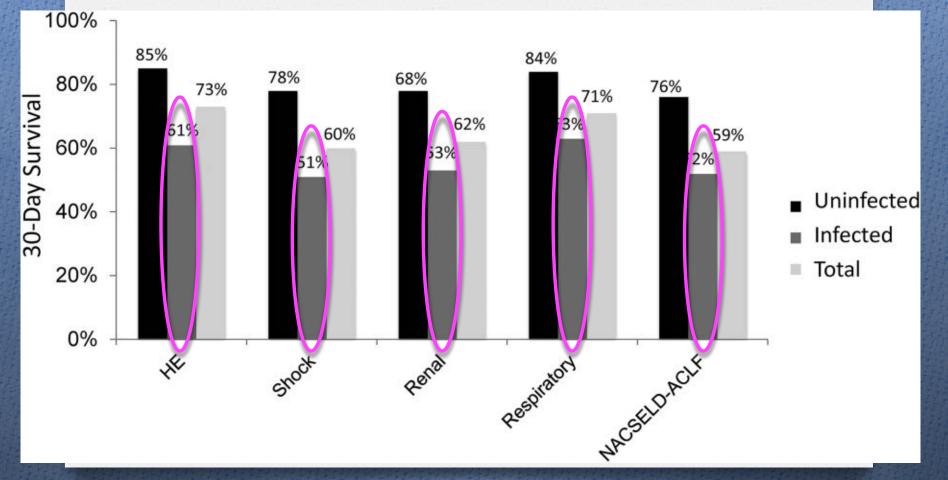
Bernal, Lancet 2015.

Infection is associated with increased risk of 30-day mortality



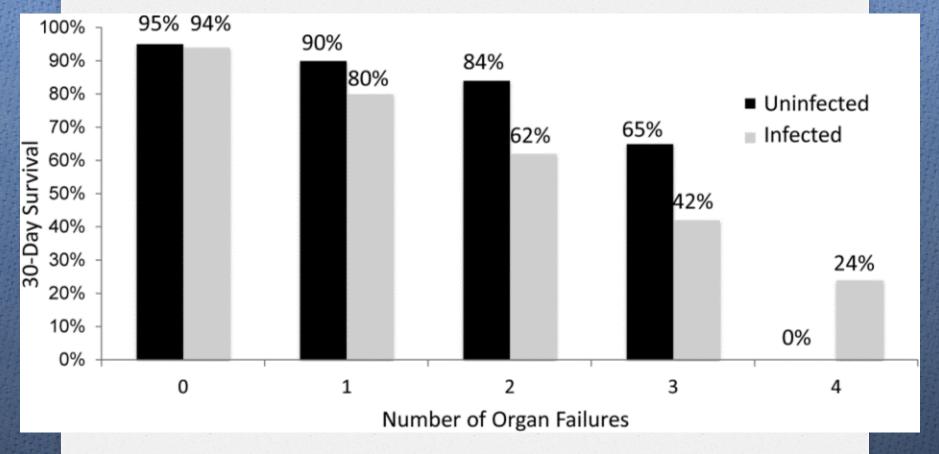
O'Leary J et al. Hepatology 2018; 2367-2374.

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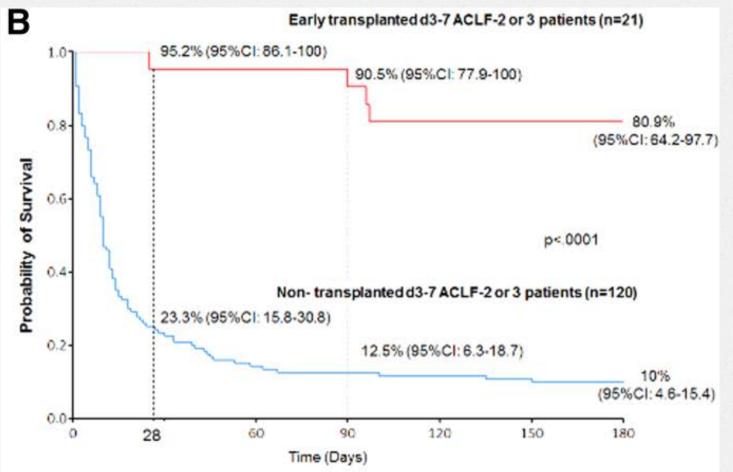
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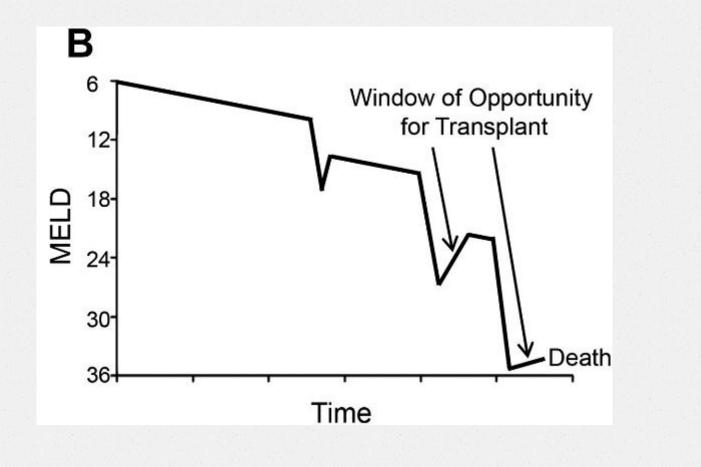
O'Leary J et al. *Hepatology* 2018; 2367-2374.

LT improves survival in ACLF



Gustot, Hepatology, 2015.

Narrow window for LT in ACLF



Asrani, Clin Liver Dis, 2014.

When should you consult hepatology for a patient with cirrhosis?

When should you consult hepatology for a patient with cirrhosis?

Decompensated cirrhosis or ACLF
Assistance in management
Liver transplant evaluation
When TIPS is being considered
Evaluation of a liver mass
Variceal bleeding (center variability)

Indications for liver transplant evaluation in patients with cirrhosis

Decompensated cirrhosis
 Child's B cirrhosis and/or
 MELD>14
 Hepatocellular carcinoma

Potential barriers to liver transplant

Medical

Severe uncontrolled extrahepatic disease
Critical illness: pressor and/or ventilator dependence
Obesity class III
Impaired functional status
Surgical
Portal and/or mesenteric vein thrombosis
Prior complex abdominal surgery

Specific selection criteria vary across transplant centers

Potential barriers to liver transplant

Psychosocial
 Active substance use/abuse
 Lack of reliable transportation or social support
 Lack of adequate insurance

Specific selection criteria vary across transplant centers

Acute on Chronic Liver Failure Summary

 Acute on chronic liver failure is associated with high risk of mortality
 Mortality risk worsened with infection and number of organ systems failing
 Liver transplant improves survival and should be considered early
 Consult your local hepatologist early

Quality measures in cirrhosis

14

Patients with ascites who are admitted to the hospital for evaluation and management of symptoms related to ascites or encephalopathy should receive a diagnostic paracentesis during the index hospitalization

1

- 2 Patients who are admitted with or develop GI bleeding should receive antibiotics within 24 hours of admission or presentation. Antibiotics should be continued for at least 5 days
- 3 Patients undergoing large-volume paracentesis (> 5 L removed) should receive intravenous albumin (6-8 g/L removed)
- 4 Hospitalized patients with ascites, with an ascitic fluid polymorphonuclear count of > 250 cells/mm³, should receive empiric antibiotics and albumin within 12 hours of the test result. The first dose of albumin should be 1.5 g/kg of body weight followed by a second infusion of 1.0 g/kg on day 3
- 5 Patients with ascites and/or hepatic hydrothorax should be managed with both sodium restriction and diuretics
- 6 Patients who undergo paracentesis should not receive fresh frozen plasma or platelets
- 7 Patients with circhosis, with platelet count < 150,000/mm³ or liver stiffness measurement > 20 kPa, and no documentation of previous GI bleeding, should receive upper endoscopy to screen for varices within 12 months of circhosis diagnosis
- 8 Patients with decompensated cirrhosis and no documented history of previous GI bleeding should receive upper endoscopy to screen for varices within 3 months of cirrhosis diagnosis
- 9 Patients with cirrhosis, no documented history of previous GI bleeding, and medium/large varices on endoscopy should receive either nonselective p-blockers or EVL within 1 month of varices diagnosis
- 10 Patients with circhosis who present with upper GI bleeding should receive upper endoscopy within 12 hours of presentation
- 11 Patients with cirrhosis who are found to have bleeding esophageal varices should receive EVL or sclerotherapy at the time of index endoscopy
- 12 Patients with cirrhosis who survive an episode of acute variceal hemorrhage should receive a combination of EVL and β-blockers
- 13 Patients with previous overt hepatic encephalopathy should be counseled regarding the risks associated with driving

- Patients with hepatic encephalopathy should have a search for evidence of precipitating factors documented in the chart
- 15 Patients who are hospitalized and have an acute episode of overt hepatic encephalopathy should receive lactulose
- 16 Patients who are discharged after an acute episode of hepatic encephalopathy should receive secondary prophylaxis with lactulose and/or rifaximin
- 17 Patients with circhosis and MELD score > 15, who do not have absolute contraindications to liver transplantation, should have documentation of evaluation for liver transplantation
- 18 Patients with circhosis, who do not have absolute contraindications to liver transplantation and have HDC meeting the transplant criteria, should be considered for liver transplantation, regardless of their MELD score
- 19 Patients with circhosis should undergo HCC screening using abdominal imaging with or without serum a-fetoprotein every 6-12 months
- 20 Patients with cirrhosis should have hepatitis B immune status and/or vaccination documented in the chart
- 21 Patients with untreated hepatifis C cirrhosis should be considered for antiviral therapy for hepatitis C
- 22 Patients with untreated hepatifis B cirrhosis should be considered for antiviral therapy for hepatitis B
- 23 Patients with cirrhosis should receive counseling or be referred to a substance abuse treatment program within 2 months of positive screening
- 24 Patients with cirrhosis who are undergoing abdominal surgery should have documentation of the risk-benefit of undergoing the surgical procedure in the medical record
- 25 Recently discharged patients with cirrhosis should have a clinic visit with a health care provider within 4 weeks of discharge
- 26 Patients with cirrhosis should be assessed for frailty using a systematic screening method

Kanwal, Hepatology, 2019.

A Quality Improvement Initiative Reduces 30-Day Rate of Readmission for Patients With Cirrhosis

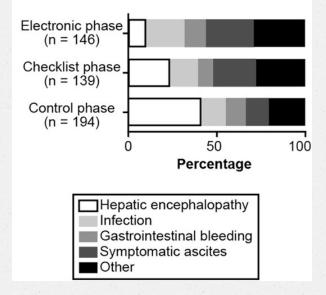
Elliot B. Tapper,* Daniel Finkelstein,[‡] Murray A. Mittleman,[§] Gail Piatkowski,^{||} Matthew Chang,[¶] and Michelle Lai*

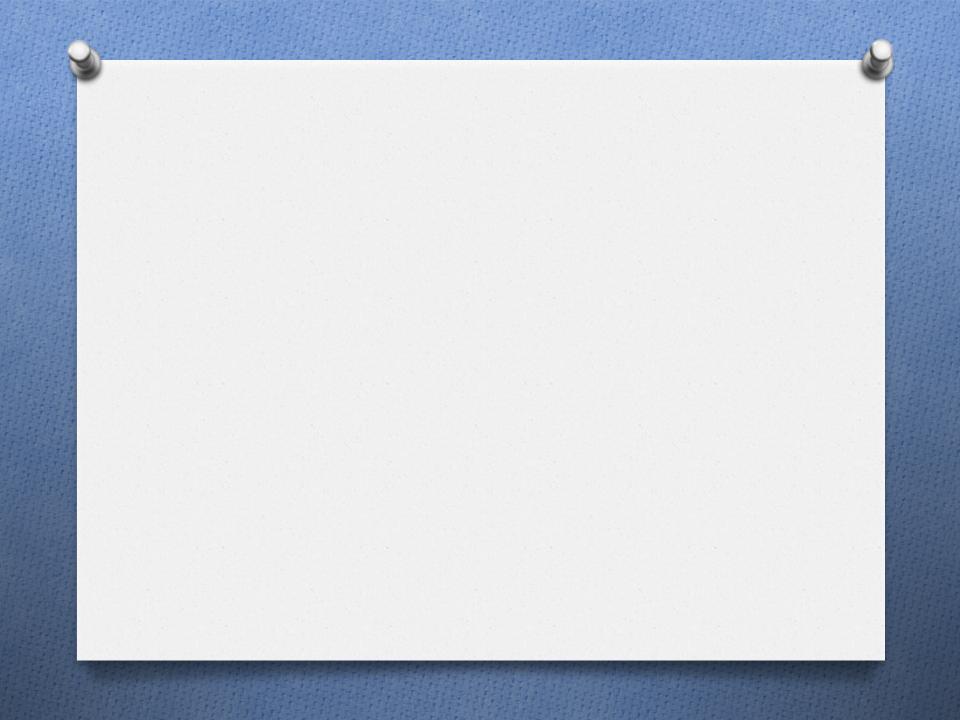
Clinical Gastroenterology and Hepatology 2016;14:753-759

 Paper checklist then electronic checklist approach to implement evidence-based care
 HE: Rifaximin for all + goal-directed lactulose dosing
 SBP treatment: timely antibiotics + IV albumin

Prophylactic measures

B Reasons for 30-day readmission by intervention phase





Thank you

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