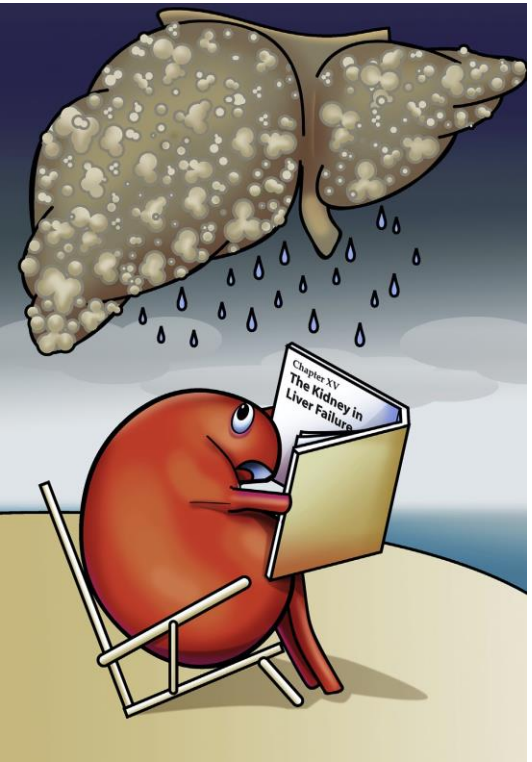
Update in AKI in Patients with Cirrhosis

ADQI

INTERNATIONAL CLUB OF ASCITES

Kidney Dysfunction in Patients with Cirrhosis: ADQI-ICA Consensus Conference



Mitra K. Nadim, MD, FASN
Professor of Clinical Medicine
Division of Nephrology and Hypertension
Keck School of Medicine
University of Southern California

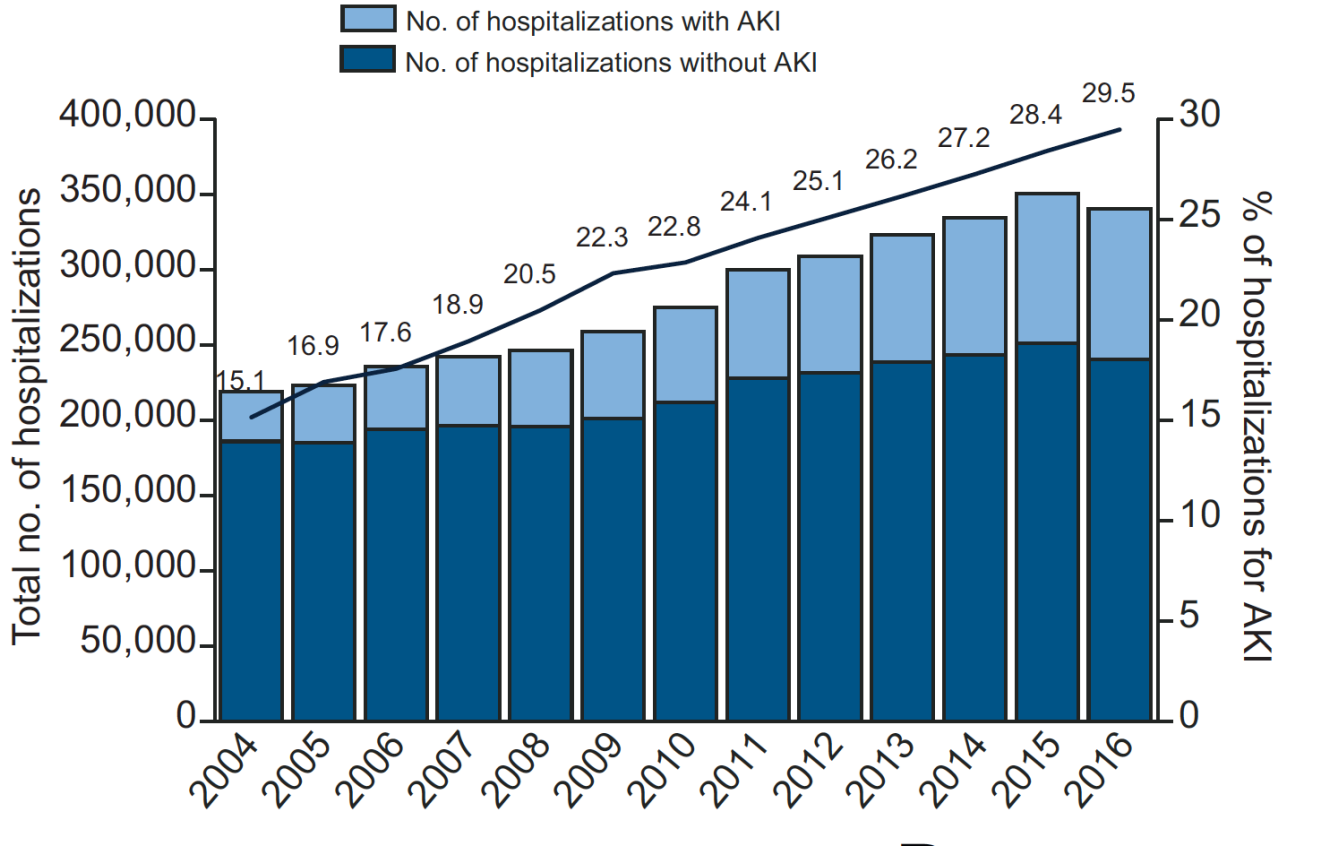
Research Article
Cirrhosis and Liver Failure

JOURNAL OF HEPATOLOGY

2020

Changing epidemiology and outcomes of acute kidney injury in hospitalized patients with cirrhosis – a US population-based study

Archita P. Desai[1,*], Shannon M. Knapp[2], Eric S. Orman[1], Marwan S. Ghabril[1], Lauren D. Nephew[1], Melissa Anderson[3], Pere Ginès[4], Naga P. Chalasani[1], Kavish R. Patidar[1,*]



Evolution of AKI / HRS Definition

1979

Sassari Criteria (HRS)

Piccin Medical Books

1996

International Club of Ascites (ICA) Criteria (HRS)

Arroyo V et al., Hepatology

2007

ICA Criteria (HRS)

Salerno F et al., Gut

2012

Acute Disease Quality Initiative (ADQI) (AKI)

Nadim MK et al., Critical Care

2015

ICA Criteria (AKI + HRS)

Angeli P et al., J Hepatology

2023

ADQI + ICA

(AKI + HRS-AKI)

Earley LE. Presentation of diagnostic criteria of the hepatorenal syndrome. In: Bartoli E, Chiandussi L, eds. Hepatorenal Syndrome. Padova: Piccin Medical Books, 1979:495-504.

Arroyo et al.,
Hepatology 1996

Salerno et al.,
Gut 2007

TABLE 1. The Sassari's Diagnostic Criteria of Hepatorenal Syndrome

Major criteria

Renal insufficiency (plasma creatinine >1.5 mg/dL) that progresses over days or weeks in the presence of severe liver disease and in the absence of recognized nephrotoxic agents.
Tubular function initially intact as measured by:
- U/P Osm >1.0
- U/P creatinine >30
- UNa remarkably low: <10 mEq/L, often <5 mEq/L

The above findings undergo no sustained improvement with expansion of the intravascular space to achieve a central venous pressure up to 10 cm H_2O.

Additional Minor Criteria

The urine may or may not contain trace amounts of protein and the sediment may or may not contain hyaline and/or granular casts.
Urine volume usually small (<800 mL/d) but not invariably so.
The onset of renal failure may occur spontaneously in the course of liver disease or may be associated with infection or bleeding, paracentesis, diuretic therapy or other forms of volume loss.
The initial characteristics of the renal failure may be followed in a few to several days by tubular dysfunction characterized by isotonic urine, increased UNa, and a fall in U/P creatinine. These changes may be accompanied by an accelerated increase in plasma creatinine concentration.
Post-mortem renal histology is variable, nonspecific and may be normal.

TABLE 2. International Ascites Club's Diagnostic Criteria of Hepatorenal Syndrome

Major Criteria

Chronic or acute liver disease with advanced hepatic failure and portal hypertension.
Low glomerular filtration rate, as indicated by serum creatinine of >1.5 mg/dL or 24-h creatinine clearance <40 mL/min.
Absence of shock, ongoing bacterial infection, and current or recent treatment with nephrotoxic drugs. Absence of gastrointestinal fluid losses (repeated vomiting or intense diarrhea) or renal fluid losses (weight loss >500 g/d for several days in patients with ascites without peripheral edema or 1,000 g/d in patients with peripheral edema).
No sustained improvement in renal function (decrease in serum creatinine to 1.5 mg/dL or less or increase in creatinine clearance to 40 mL/min or more) following diuretic withdrawal and expansion of plasma volume with 1.5 L of isotonic saline.
Proteinuria <500 mg/dL and no ultrasonographic evidence of obstructive uropathy or parenchymal renal disease.

Additional Criteria

Urine volume <500 mL/d.
Urine sodium <10 mEq/L.
Urine osmolality greater than plasma osmolality.
Urine red blood cells <50 per high power field.
Serum sodium concentration <130 mEq/L.

New diagnostic hepatorenal syndrome criteria in cirrhosis

- Cirrhosis with ascites.
- Serum creatinine >133 μmol/l (1.5 mg/dl).
- No improvement of serum creatinine (decrease to a level of ⩽133 μmol/l) after at least 2 days with diuretic withdrawal and volume expansion with albumin. The recommended dose of albumin is 1 g/kg of body weight per day up to a maximum of 100 g/day.
- Absence of shock.
- No current or recent treatment with nephrotoxic drugs.
- Absence of parenchymal kidney disease as indicated by proteinuria >500 mg/day, microhaematuria (>50 red blood cells per high power field) and/or abnormal renal ultrasonography.

Nadim et al. Critical Care 2012, 16:R23
http://ccforum.com/content/16/1/R23

CRITICAL CARE

RESEARCH Open Access

Hepatorenal syndrome: the 8th international consensus conference of the Acute Dialysis Quality Initiative (ADQI) Group

Mitra K Nadim[1*], John A Kellum[2], Andrew Davenport[3], Florence Wong[4], Connie Davis[5], Neesh Pannu[6], Ashita Tolwani[7], Rinaldo Bellomo[8] and Yuri S Genyk[9], for The ADQI Workgroup

Diagnosis	Definition
AKI	• ↑ Scr ≥ 50% from baseline or ↑ Scr ≥ 0.3mg/dL < 48 hours • HRS Type I is a specific form of AKI
CKD	• GFR < 60ml/min for > 3 month, calculated by MDRD-6 • HRS Type II is a specific form of CKD

Stages of AKI based on AKIN Scr Criteria

Position Paper

CrossMark

EASL JOURNAL OF HEPATOLOGY

Diagnosis and management of acute kidney injury in patients with cirrhosis: Revised consensus recommendations of the International Club of Ascites☆

Paolo Angeli[1,*], Pere Ginès[2,3,4,5], Florence Wong[6], Mauro Bernardi[7], Thomas D. Boyer[8], Alexander Gerbes[9], Richard Moreau[10,11,12], Rajiv Jalan[13], Shiv K. Sarin[14], Salvatore Piano[1], Kevin Moore[15], Samuel S. Lee[16], Francois Durand[17,18], Francesco Salerno[19], Paolo Caraceni[7], W. Ray Kim[20], Vicente Arroyo[2,3,4], Guadalupe Garcia-Tsao[21]

Baseline SCr
- Stable SCr ≤3 months
- If not available, a stable SCr closest to the current one
- If no previous SCr at all, use admission SCr

Definition of AKI
- Increase in SCr ≥26.5 μmol/l (0.3 mg/dl) <48 h, or
- Increase 50% from baseline

Stages of AKI based on KDIGO Scr Criteria

UO criteria were not included because (a) these patients are frequently oliguric with avid sodium retention (b) may have an increased UO because of diuretics, and (c) on a regular ward, urine collection is often inaccurate

HRS-AKI

- Diagnosis of cirrhosis and ascites
- Diagnosis of AKI according to ICA-AKI criteria
- No response after 2 consecutive days of diuretic withdrawal and plasma volume expansion with albumin 1 g per kg of body weight
- Absence of shock
- No current or recent use of nephrotoxic drugs (NSAIDs, aminoglycosides, iodinated contrast media, etc.)
- No macroscopic signs of structural kidney injury*, defined as:
  - absence of proteinuria (>500 mg/day)
  - absence of microhaematuria (>50 RBCs per high power field),
  - normal findings on renal ultrasonography

*Patients who fulfil these criteria may still have structural damage such as tubular damage. Urine biomarkers will become an important element in making a more accurate differential diagnosis between HRS and acute tubular necrosis.

SC Only	UO Only				
	No AKI	Stage 1	Stage 2	Stage 3	Total
No AKI	-	-	-	-	-
Patients, no.	604[a]	235[b]	563[b]	54[b]	1456
Dead (%)	4.97	6.38	8.17	22.22	7.07
Stage 1	-	-	-	-	
Patients, no.	212[c]	131[d]	496[d]	168[d]	1007
Dead (%)	8.96	12.21	14.31	34.52	16.29
Stage 2	-	-	-	-	
Patients, no.	59[c]	41[e]	230[e]	162[e]	492
Dead (%)	15.25	24.39	23.48	40.74	28.25
Stage 3	-	-	-	-	
Patients, no.	30[c]	43[f]	130[f]	300[g]	503
Dead (%)	13.33	30.47	40.77	48.33	45.33

HEPATOLOGY

AASLD AMERICAN ASSOCIATION FOR THE STUDY OF LIVER DISEASES

HEPATOLOGY, VOL. 66, NO. 5, 2017

Significance of Oliguria in Critically Ill Patients With Chronic Liver Disease

Roland Amathieu,[1–3] Ali Al-Khafaji,[1,2] Florentina E. Sileanu,[1,2] Emily Foldes,[1,2] Rebecca DeSensi,[1,2] Ibtesam Hilmi,[1,4] and John A. Kellum[1,2]

2018

Clinical Practice Guidelines

JOURNAL OF HEPATOLOGY

EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis☆

European Association for the Study of the Liver*

It should be highlighted that the KDIGO criteria also include criteria based on urinary output in the diagnosis of AKI…. these criteria may also be applied whenever a patient with cirrhosis requires a bladder catheter.

HEPATOLOGY

PRACTICE GUIDANCE | HEPATOLOGY, VOL. 74, NO. 2, 2021

Diagnosis, Evaluation, and Management of Ascites, Spontaneous Bacterial Peritonitis and Hepatorenal Syndrome: 2021 Practice Guidance by the American Association for the Study of Liver Diseases

Scott W. Biggins,[1] Paulo Angeli,[2] Guadalupe Garcia-Tsao,[3,4] Pere Ginès,[5,6] Simon C. Ling,[7] Mitra K. Nadim,[8] Florence Wong,[9] and W. Ray Kim[10]

Measurement of urine volume, a component in the diagnosis of AKI, is important because oliguria is associated with poor prognosis.

Review
2019

JOURNAL
OF HEPATOLOGY

News in pathophysiology, definition and classification of hepatorenal syndrome: A step beyond the International Club of Ascites (ICA) consensus document☆

Paolo Angeli[1,*,†], Guadalupe Garcia-Tsao[2,3,†], Mitra K. Nadim[4], Chirag R. Parikh[5]

Table 1. New classification of HRS subtypes.

Old classification	New classification		Criteria
HRS-1[#]	HRS-AKI		a) Absolute increase in sCr ≥0.3 mg/dl within 48 h *and/or* b) Urinary output ≤ 0.5 ml/kg B.W. ≥6 h[*] *or* c) Percent increase in sCr ≥50% using the last available value of outpatient sCr within 3 months as the baseline value
HRS-2[#]	HRS-NAKI	HRS-AKD	a) eGFR <60 ml/min per 1.73 m^2 for <3 months in the absence of other (structural) causes b) Percent increase in sCr <50% using the last available value of outpatient sCr within 3 months as the baseline value
		HRS-CKD	a) eGFR <60 ml/min per 1.73 m^2 for ≥3 months in the absence of other (structural) causes

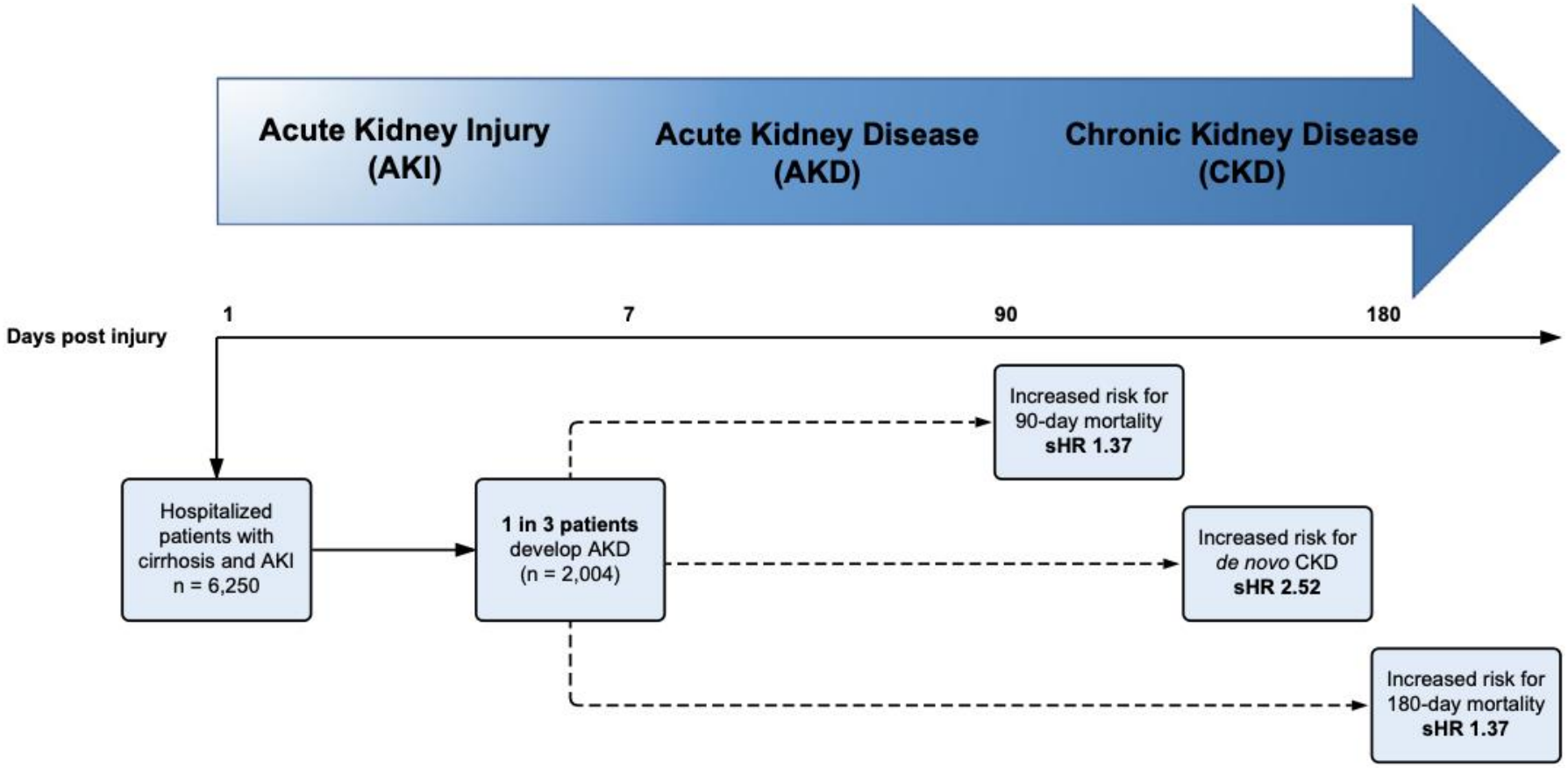
Research Article
Cirrhosis and Liver Failure

JOURNAL
OF HEPATOLOGY

2022

Acute kidney disease is common and associated with poor outcomes in patients with cirrhosis and acute kidney injury

Kavish R. Patidar[1,*], Mobasshir A. Naved[2], Ananth Grama[2], Mohammad Adibuzzaman[3], Arzina Aziz Ali[4], James E. Slaven[5], Archita P. Desai[1], Marwan S. Ghabril[1], Lauren Nephew[1], Naga Chalasani[1], Eric S. Orman[1]



Group 1: Epidemiology, and definition of kidney dysfunction

Chair / Co-Chair: Claire Francoz – CC/Hep (France), Lui Forni – CC/Neph (UK)

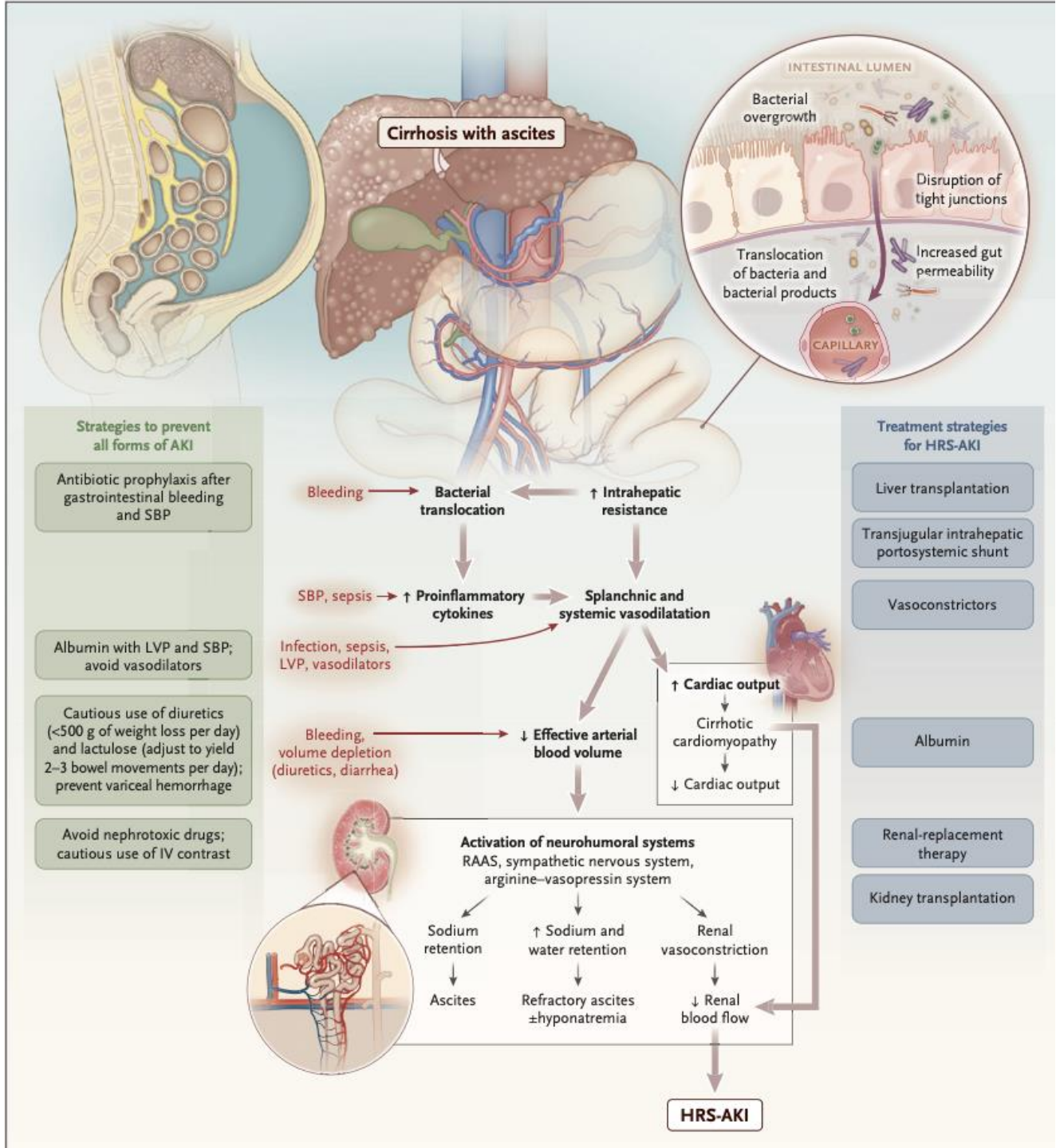
Members: Paolo Angeli – Hepatology (Italy), Lupe Garcia-Tsao- Hep (USA), John Kellum – CC (USA), Ayse Akcan-Arikan – Peds CC/Neph (USA)

1. What is the epidemiology and outcomes of kidney dysfunction in patients with Compensated, Decompensated Cirrhosis, and acute on chronic liver failure (ACLF)?

2. How should the definitions AKI / AKD / CKD / AKI on CKD, and recovery be harmonized with current KDIGO and ICA definitions?

3. What baseline Scr should be used to define AKI?

4. How should HRS be defined?



The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

C. Corey Hardin, M.D., Ph.D., *Editor*

Acute Kidney Injury in Patients with Cirrhosis

Mitra K. Nadim, M.D., and Guadalupe Garcia-Tsao, M.D.

Group 2: Pathophysiology and Risk Factors

Chair / Co-Chair: Lisa VanWagner – Tx Hep (USA), Salvatore Piano – Hep (Italy)

Members: Juan-Carlos Velez – Neph (USA), Akash Deep- Peds CC (UK), Claudio Ronco – Neph (Italy), Patrick Kamath- Hep (USA)

1. What is the pathophysiology of kidney dysfunction in patients with Compensated, Decompensated Cirrhosis, and acute on chronic liver failure (ACLF)?

2. What are the risk factors for developing AKI / HRS-AKI in compensated, decompensate and ACLF?

Protecting the Kidney in Liver Transplant Candidates: *Practice-Based Recommendations From the American Society of Transplantation Liver and Intestine Community of Practice*

J. G. O'Leary[1,†,*], J. Levitsky[2,†], F. Wong[3], M. K. Nadim[4], M. Charlton[5] and W. R. Kim[6]

American Journal of Transplantation 2016; 16: 2516–2531
Wiley Periodicals Inc.

Risk factors	Preventive strategies
Hepatorenal syndrome development	• Antibiotic prophylaxis following gastrointestinal bleeding for 7 days (grade 1A) • Albumin infusion during large-volume paracentesis (>5 L, 6–8 g/L of ascitic fluid removed) (grade 1A) • Secondary and primary SBP prophylaxis with daily antibiotics, preferably norfloxacin (grade 1B) • Early recognition and treatment of SBP with antibiotics and IV albumin at the dose of 1.5 g/kg of body weight at the time of diagnosis of SBP and 1 g/kg of body weight on the third day of treatment (grade 1B) • Judicious use of diuretics • Avoid dehydration with lactulose use
Exposure to nephrotoxic medications (e.g. NSAIDs, aminogyclosides, amphotericin, vancomycin)	• Close monitoring of drug toxicity and early recognition of drug-induced AKI and discontinuation of offending agent if possible (grade 1A) • Use lipid formulations of amphotericin B rather than conventional formulations of amphotericin B (grade 2A) • Use azole antifungal agents and/or the echinocandins rather than conventional amphotericin B, if equal therapeutic efficacy can be assumed (grade 1A) • Avoid nephrotoxic medications whenever possible
Radiocontrast exposure	• Consider alternative imaging methods or avoidance of IV contrast if possible • Use low or iso-osmolar agents with lowest volume possible (grade 1B) • Optimize fluid status prior to administration of IV contrast with IV normal saline or IV bicarbonate (grade 1A) • Consider N-acetylcysteine use in combination with IV hydration (grade 2D)
Hemodynamic instability	• Increase mean arterial pressure in setting of shock to >65 mmHg (grade 1C) • Use of protocol-based management of hemodynamic and oxygenation parameters (grade 2C) • Optimal fluid resuscitation with crystalloids or colloids (grade 2B) • Vasopressors in patients with persistent hypotension (grade 1C), consider norepinephrine as first line (grade 2D)

NEJM 1999

EFFECT OF INTRAVENOUS ALBUMIN ON RENAL IMPAIRMENT AND MORTALITY IN PATIENTS WITH CIRRHOSIS AND SPONTANEOUS BACTERIAL PERITONITIS

PAU SORT, M.D., MIQUEL NAVASA, M.D., VICENTE ARROYO, M.D., XAVIER ALDEGUER, M.D., RAMON PLANAS, M.D., LUIS RUIZ-DEL-ARBOL, M.D., LLUIS CASTELLS, M.D., VICTOR VARGAS, M.D., GERMÁN SORIANO, M.D., MÓNICA GUEVARA, M.D., PERE GINÈS, M.D., AND JOAN RODÉS, M.D.

- RCT, unblinded, Abx vs Abx+ albumin (1.5 g/kg/day day 1; 1g/kg day 3); Labs checked every 3 days
- Renal failure defined as BUN > 30 mg/dL & Scr > 1.5 mg/dL.
- Renal impairment defined as a nonreversible deterioration of renal function , diagnosed when BUN and Cr increased >50 % of the pretreatment value, to levels higher than 30 mg/ dL or 1.5 mg/dL.

TABLE 1. BASE-LINE CHARACTERISTICS OF THE 126 PATIENTS ACCORDING TO THE ASSIGNED TREATMENT.*

CHARACTERISTIC	CEFOTAXIME (N=63)	CEFOTAXIME PLUS ALBUMIN (N=63)
Age — yr	62±1	60±1
Sex — M/F	38/25	43/20
Alcoholic cirrhosis — no. (%)	19 (30)	18 (29)
Hepatocellular carcinoma — no. (%)	7 (11)	10 (16)
Hepatic encephalopathy — no. (%)	15 (24)	13 (21)
White-cell count — per mm³	9221±814	7883±560
Ascitic-fluid polymorphonuclear cells — per mm³	4228±750	5223±1541
Serum bilirubin — mg/dl	6±1	4±1
Serum albumin — g/dl	2.5±0.1	2.7±0.1
Prothrombin time — % of control	58±2	55±2
Child–Pugh score‡	10±0.2	10±0.2
Renal failure — no. (%)	28 (44)	25 (40)
Diuretic treatment — no. (%)	41 (65)	45 (71)
Spironolactone — mg/day	73±5	81±6
Furosemide — mg/day	19±1	18±2
Previous prophylactic treatment with norfloxacin — no. (%)	5 (8)	6 (10)
Isolated organisms — no. (%)‡	36 (57)	32 (51)
Escherichia coli	22	20
Other gram-negative bacilli	6	7
Other bacteria	8	5

NEJM 1999

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TABLE 2. CLINICAL OUTCOME ACCORDING TO THE ASSIGNED TREATMENT.*

OUTCOME VARIABLE	CEFOTAXIME (N=63)	CEFOTAXIME PLUS ALBUMIN (N=63)	P VALUE
Resolution of infection — no. (%)†	59 (94)	62 (98)	0.36
Duration of antibiotic therapy — days	6±1	5±1	0.48
Paracentesis for ascites after resolution of infection — no. (%)‡	16 (25)	14 (22)	0.83
Hospital stay — days	13±1	14±1	0.48
Renal impairment — no. (%)	21 (33)	6 (10)	0.002
Death — no. (%)			
In hospital§	18 (29)	6 (10)	0.01
At three months¶	26 (41)	14 (22)	0.03

TABLE 3. RENAL FUNCTION, SERUM SODIUM LEVELS, AND MEAN ARTERIAL PRESSURE AT ENROLLMENT AND DURING THE FIRST NINE DAYS OF HOSPITALIZATION IN THE 126 PATIENTS.*

VARIABLE	CEFOTAXIME (N=63)	CEFOTAXIME PLUS ALBUMIN (N=63)	P VALUE
Blood urea nitrogen — mg/dl (no. of patients)			
Day 0	31±3 (63)	28±3 (63)	0.48
Day 3	34±3 (59)	25±3 (58)	0.03
Day 6	36±3 (58)	22±3 (57)	0.003
Day 9	36±3 (48)	22±3 (53)	0.01
Serum creatinine — mg/dl (no. of patients)			
Day 0	1.1±0.1 (63)	1.2±0.1 (63)	0.66
Day 3	1.3±0.1 (61)	1±0.1 (60)	0.16
Day 6	1.3±0.1 (59)	1±0.1 (59)	0.03
Day 9	1.4±0.1 (48)	1±0.1 (55)	0.04
Serum sodium — mmol/liter (no. of patients)			
Day 0	133±1 (63)	134±1 (63)	0.24
Day 3	130±1 (59)	134±1 (61)	0.001
Day 6	130±1 (56)	134±1 (57)	<0.001
Day 9	130±1 (51)	134±1 (53)	0.002
Mean arterial pressure — mm Hg (no. of patients)			
Day 0	86±2 (63)	86±2 (63)	0.91
Day 3	81±2 (59)	81±2 (59)	0.91
Day 6	79±2 (55)	80±1 (57)	0.71
Day 9	81±2 (51)	81±2 (55)	0.72

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

A Randomized Trial of Albumin Infusions in Hospitalized Patients with Cirrhosis

Louise China, Ph.D., Nick Freemantle, Ph.D., Ewan Forrest, M.D., Yiannis Kallis, Ph.D., Stephen D. Ryder, D.M., Gavin Wright, Ph.D., Andrew J. Portal, M.D., Natalia Becares Salles, Ph.D., Derek W. Gilroy, Ph.D., and Alastair O'Brien, Ph.D., for the ATTIRE Trial Investigators*

- A randomized, multicenter, open-label, parallel-group trial involving hospitalized patients with decompensated cirrhosis who had a serum albumin level of less than 30 g / L
- Patients were randomly assigned to receive either targeted 20% human albumin solution for up to 14 days or until discharge, whichever came first, vs standard of care.
- Treatment started within 3 days after admission
- Composite primary end-point was new infection, kidney dysfunction, or death between day 3 and 15 after initiation of treatment.

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

A Randomized Trial of Albumin Infusions in Hospitalized Patients with Cirrhosis

Louise China, Ph.D., Nick Freemantle, Ph.D., Ewan Forrest, M.D., Yiannis Kallis, Ph.D., Stephen D. Ryder, D.M., Gavin Wright, Ph.D., Andrew J. Portal, M.D., Natalia Becares Salles, Ph.D., Derek W. Gilroy, Ph.D., and Alastair O'Brien, Ph.D., for the ATTIRE Trial Investigators*

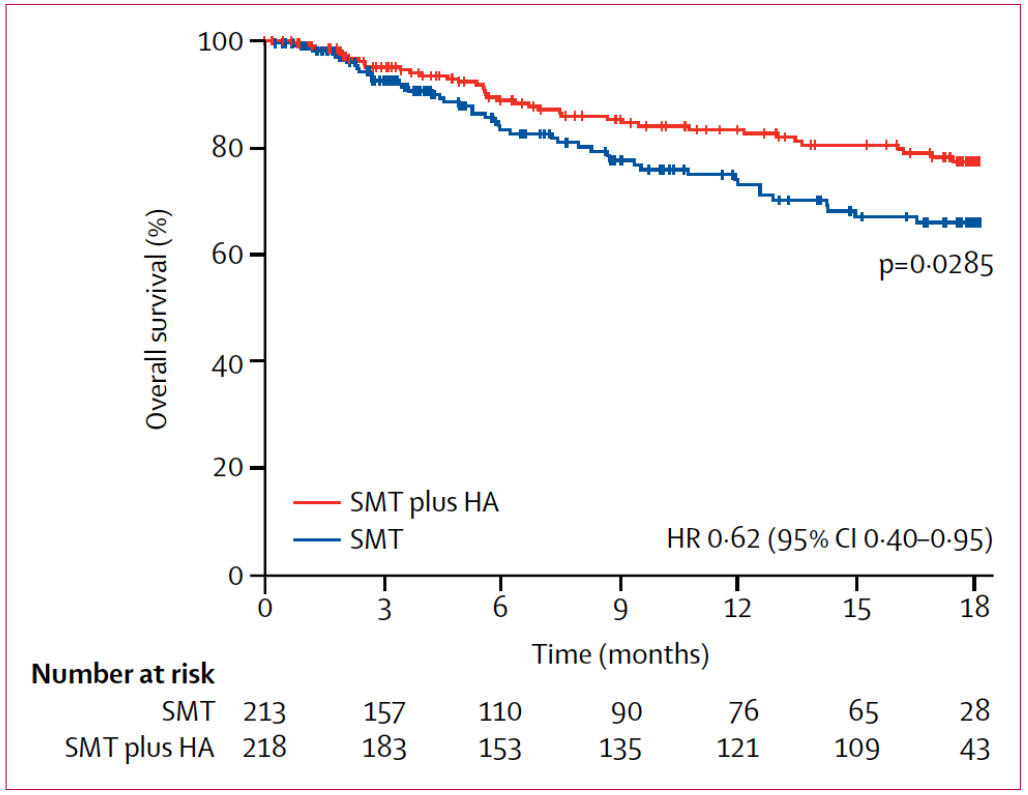
Table 2. End Points.*

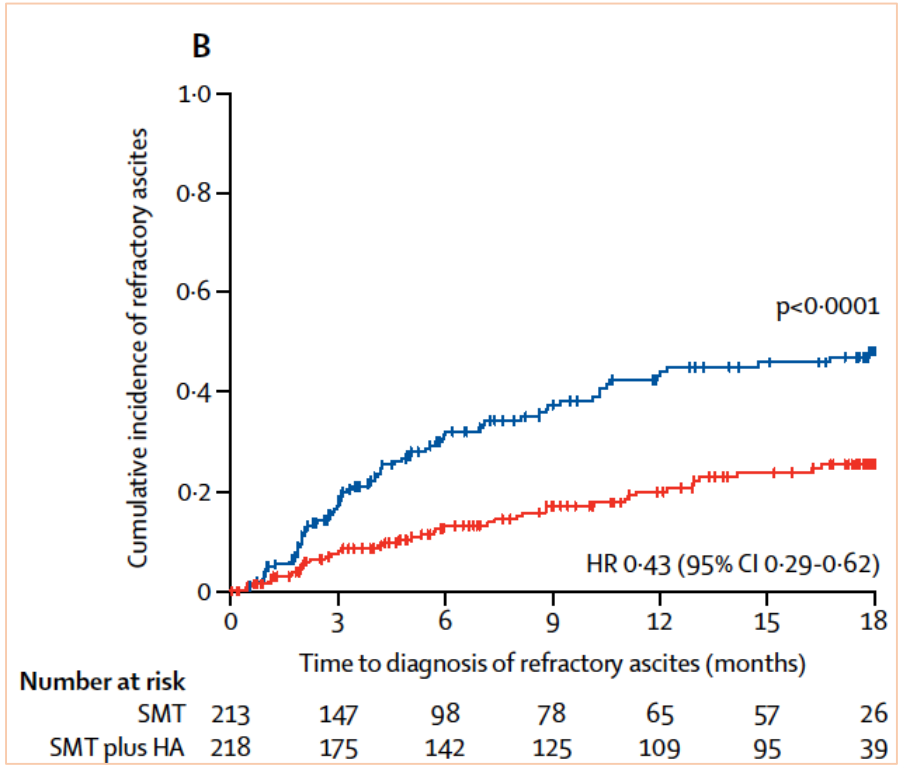
Variable	Albumin Group (N=380)	Standard-Care Group (N=397)	Adjusted Odds Ratio (95% CI)†	P Value
Composite primary end point — no. (%)	113 (29.7)	120 (30.2)	0.98 (0.71–1.33)	0.87
Components of composite primary end point — no. (%)‡				
Incidence of new infection	79 (20.8)	71 (17.9)	1.22 (0.85–1.75)	
Incidence of kidney dysfunction	40 (10.5)	57 (14.4)	0.68 (0.44–1.11)	
Incidence of death	30 (7.9)	33 (8.3)	0.95 (0.56–1.59)	
Death at 28 days	53 (14.0)	62 (15.6)	0.86 (0.57–1.30)	
Death at 3 mo	92 (24.2)	93 (23.4)	1.05 (0.74–1.48)	
Death at 6 mo	132 (34.7)	119 (30.0)	1.27 (0.93–1.73)	
Total median albumin infused per patient (IQR) — g	200 (140–280)	20 (0–120)	143 (127–158)§	

There were more severe or life-threatening serious adverse events, especially pulmonary edema or fluid overload, in the albumin group than in the standard-care group

Long-term albumin administration in decompensated cirrhosis (ANSWER): an open-label randomised trial

- Multi-center, randomized trial in 33 academic and non-academic Italian hospitals
- Patients with cirrhosis and uncomplicated ascites on diuretics (≥ 200 mg/d aldacone + ≥ 25 mg/day lasix) to receive SMT or SMT + Albumin (40 g twice weekly x 2 weeks, and then 40 g weekly) x 18 months





Caraceni P et al., *LANCET* 2018

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

C. Corey Hardin, M.D., Ph.D., *Editor*

Acute Kidney Injury in Patients with Cirrhosis

Mitra K. Nadim, M.D., and Guadalupe Garcia-Tsao, M.D.

Acute Kidney Injury in a Patient with Cirrhosis

Conduct history and physical examination: consider risk factors that may suggest cause of AKI (e.g. recent shock, fever, recent initiation of diuretics, diarrhea)
- Review medications list for active or recent nephrotoxin or IV contrast exposure
- Discontinue diuretics, NSAIDs, ACEI/ARB, NSBB
- Perform diagnostic paracentesis to rule out SBP
- Perform urinary tests: microscopical assessment of urinary sediment, FeNa, FeUrea, albumin, NGAL (if available)

Renal Hypoperfusion (most common)
- Hypovolemia
  - diuretics, diarrhea, GIB, LVP
- Hepatorenal Syndrome (HRS-AKI)*

Intrinsic / Parenchymal
- ATN (most common): sepsis, IV contrast, medications, bile-cast nephropathy
- AIN: antibiotics, NSAIDs, PPI
- Glomerular Diseases: e.g. IgA, MPGN, Cryoglobulinemia, membranous

- ATN: discontinue nephrotoxic agents
- AIN: discontinue offending agent
- Consider kidney biopsy
- Glomerular disease: specific treatment

Urinary Obstruction (least common)
- Urethral / ureteral obstruction

- Determine post-void residual
- Renal ultrasonography

Diuretics may need to be initiated or continued if there is evidence of volume overload. Volume overload (presence of anasarca, JVD , CXR with pulmonary congestion, or elevated RVSP.

Renal Hypoperfusion (most common)
- Hypovolemia
  - diuretics, diarrhea, GIB, LVP
- Hepatorenal Syndrome (HRS-AKI)

Assess volume status
(PE, POCUS, CXR, echocardiogram)

Intravascular volume depletion

Crystalloid solutions

Equivocal volume status

25% albumin 1g/kg
(≤100 g / day divided doses)

SCr after 24-48 hours?

Improving

Continue monitoring for renal recovery

Unchanged or Worsening

•Reassess causes of intrinsic AKI
•Repeat urine studies

HRS-AKI

Vasoconstrictor+ albumin

ATN

•Diuretics if evidence of significant volume overload
•RRT in select patients

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

C. Corey Hardin, M.D., Ph.D., *Editor*

Acute Kidney Injury in Patients with Cirrhosis

Mitra K. Nadim, M.D., and Guadalupe Garcia-Tsao, M.D.

A sine qua non for the development of HRS-AKI is the presence of ascites, and is often associated with hyponatremia, a low MAP, and oliguria.

Findings suggestive of HRS-AKI: normal urinary sediment, FeNa < 01%and FeUrea *<21%. Urine NGAL > 220- 244 µg / Cr is suggestive of ATN.*

Poor correlation between kidney function, UA & kidney biopsy !

Author	Year	N	Results
Jouet et al	1996	70	No correlation between degree of renal dysfunction and severity of renal lesion: • 28% pts with severe renal dysfunction had mild or no renal lesions • 55% pts without overt renal failure had moderate /severe renal lesions • 51% : no clinical or laboratory element predictive of presence or absence and the type of renal lesions
Arase et al	1998	188	Autopsy of 188 patients with hepatitis C • 45% with normal glomeruli. Abnormal UA observed in 12% of cases
McGuire et al	2006	25	60% with immune-complex GN had normal urinary findings
Wadei et al	2008	44	87% with ATN on biopsy (n=7) and 90% patients with normal biopsy (presumed HRS) (n=10) with FeNa < 1%
Trawale et al	2010	65	55% with renal impairment but proteinuria < 500 mg and no hematuria • 55% with glomerular lesion, 72% with chronic interstitial , 67% with acute tubular interstitial

Wadei H et al., *Am J Transpl* 2008
Trawale JM et al., Liver Int 2010

Jouet P et al., *Hepatology* 1996
Arase Y et al., I*ntern Med* 1998
McGuire BM et al., *Ann Intern Med* 2006

Kidney Biomarkers and Differential Diagnosis of Patients With Cirrhosis and Acute Kidney Injury

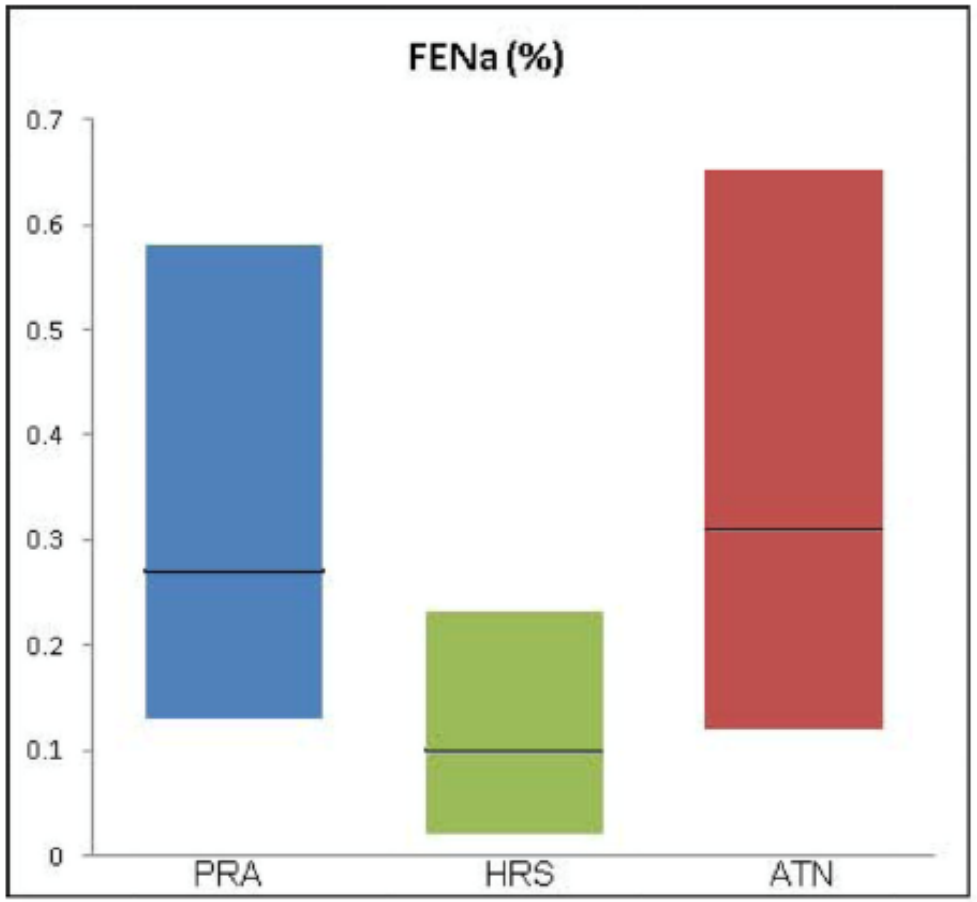
Justin M. Belcher, MD[1,2,3], Arun J. Sanyal, MD[4], Aldo J. Peixoto, MD[2,5], Mark A. Perazella, MD[2,5], Joseph Lim, MD[6], Heather Thiessen-Philbrook, MMath[7], Naheed Ansari, MD[8], Steven G. Coca, DO, MS[1,2,3], Guadalupe Garcia-Tsao, MD[3,5,6], Chirag R. Parikh, MD, PhD[1,2,3], and for the TRIBE-AKI Consortium

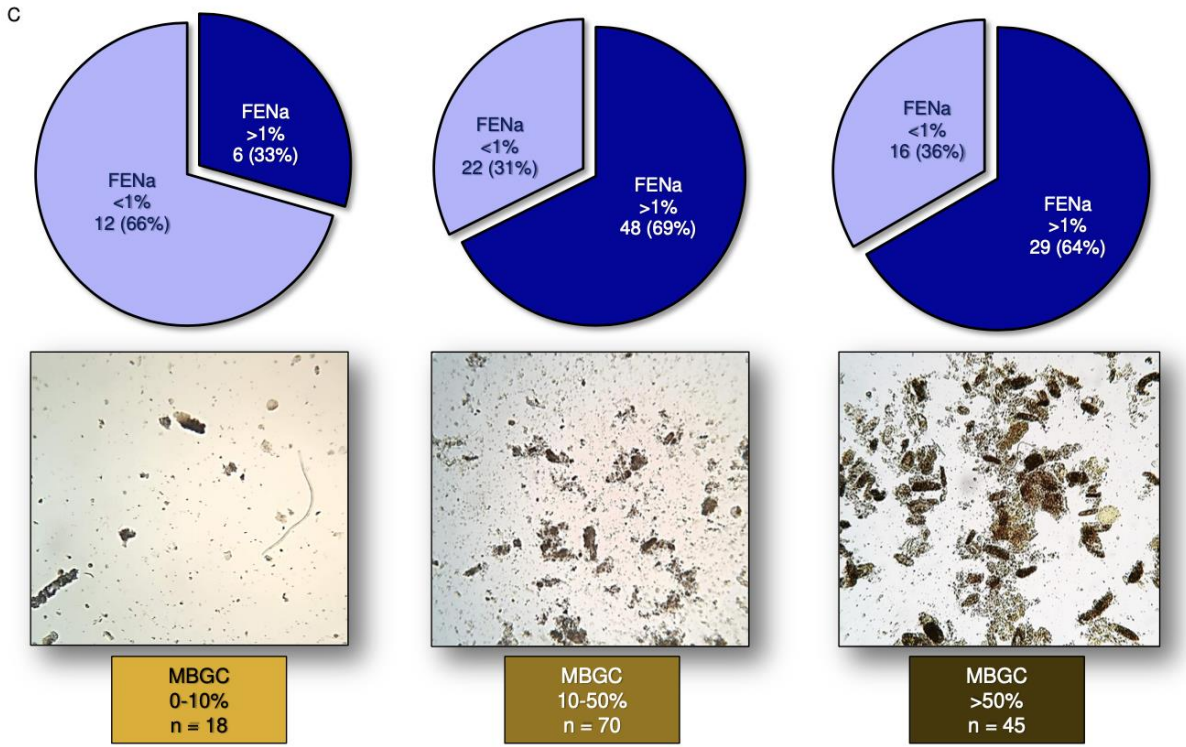
Hepatology. 2014 August ; 60(2): 622–632

Kidney360

American Society of Nephrology
1401 H St NW, Suite 900
Washington, DC 20005
Phone: 202-640-4660 | Fax 202-637-9793
vramsey@kidney360.org

How to Cite this article: Vipin Varghese, Maria Soledad Rivera, Ali Alalwan, Ayman Alghamdi, Akanksh Ramanand, Sumayyah Khan, Jose Najul-Seda, and Juan Carlos Velez, Concomitant Identification of Muddy Brown Granular Casts and Low FENa in Acute Kidney Injury, *Kidney360*, Publish Ahead of Print, 10.34067/KID.0005692021





Group 3: Workup and Prevention of AKI

Chair / Co-Chair: Marlies Ostermann – CC/Neph (UK), Sumeet Asrani – Tx Hep (USA)

Members: Sandra Kane-Gill – Pharm (USA), Justin Belcher – Neph (USA), Scott Biggins – Tx Hep (USA)
Manish Kaushik – CC/Neph (Singapore)

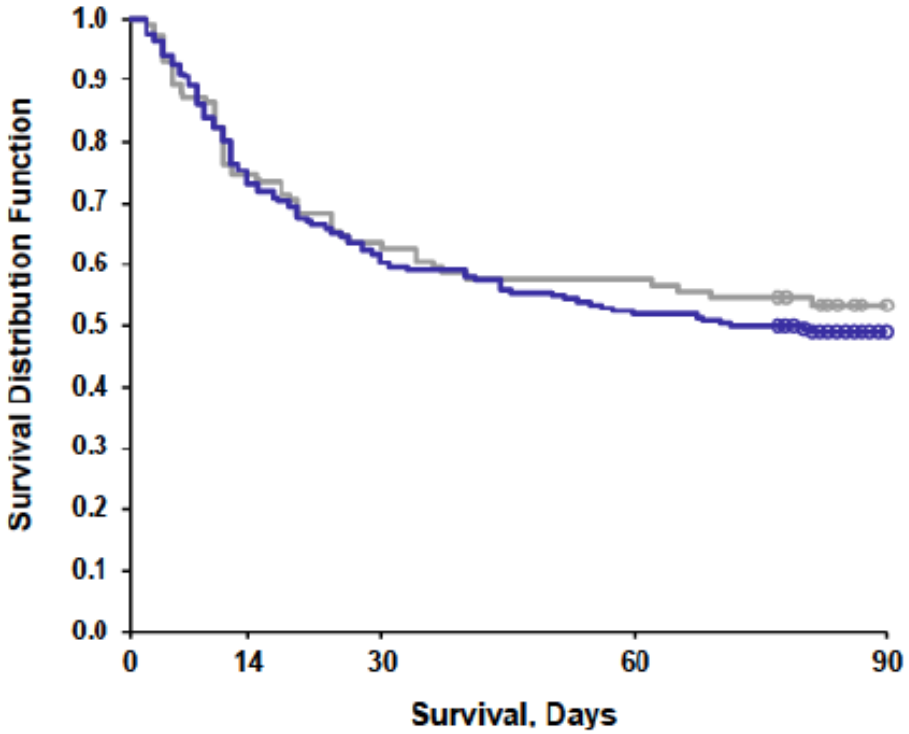
1. What diagnostic tools should be included in the workup of patients with kidney dysfunction? (ex: POCUS, biomarkers)
2. Can biomarkers be used to guide different treatment protocols?
3. What strategies exist for prevention of kidney dysfunction – SBP prophylaxis, GIB, LVP etc.?
4. What fluids should be used for prevention of kidney dysfunction?

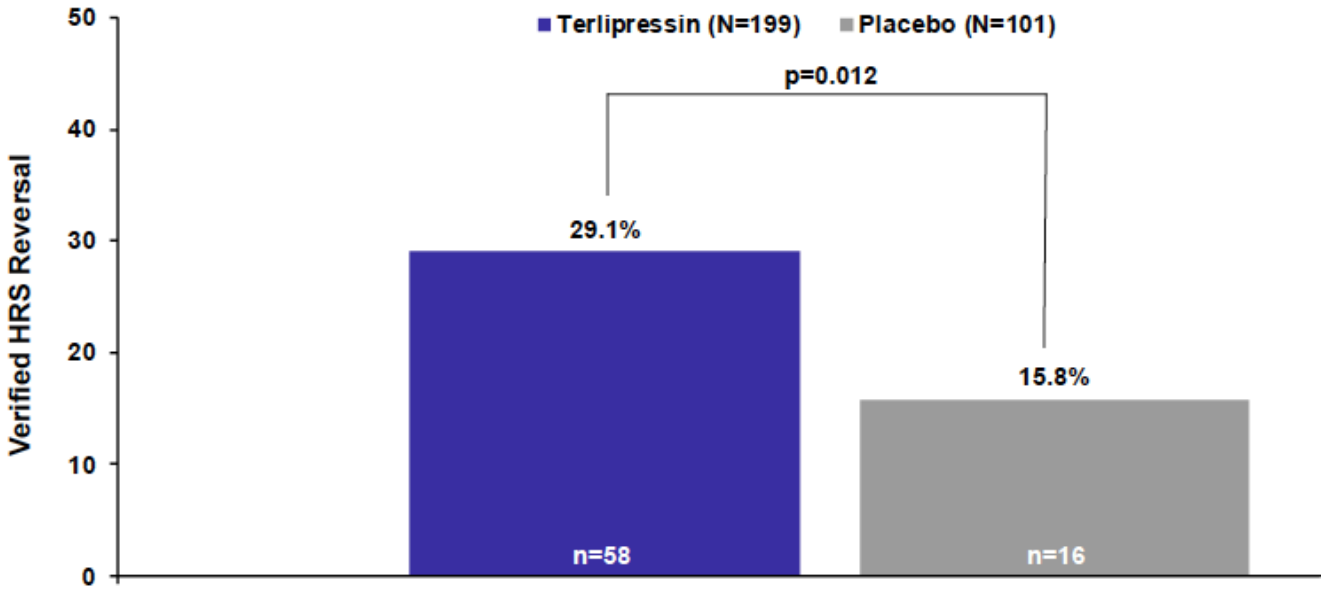
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Terlipressin plus Albumin for the Treatment of Type 1 Hepatorenal Syndrome

F. Wong, S.C. Pappas, M.P. Curry, K.R. Reddy, R.A. Rubin, M.K. Porayko, S.A. Gonzalez, K. Mumtaz, N. Lim, D.A. Simonetto, P. Sharma, A.J. Sanyal, M.J. Mayo, R.T. Frederick, S. Escalante, and K. Jamil, for the CONFIRM Study Investigators*





The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Terlipressin plus Albumin for the Treatment of Type 1 Hepatorenal Syndrome

F. Wong, S.C. Pappas, M.P. Curry, K.R. Reddy, R.A. Rubin, M.K. Porayko, S.A. Gonzalez, K. Mumtaz, N. Lim, D.A. Simonetto, P. Sharma, A.J. Sanyal, M.J. Mayo, R.T. Frederick, S. Escalante, and K. Jamil, for the CONFIRM Study Investigators*

	Integrated Phase 3 Studies	
Preferred Term[a,b]	Terlipressin N=349 n (%)	Placebo N=249 n (%)
Any SAE	227 (65.0)	149 (59.8)
Respiratory failure	29 (8.3)	6 (2.4)
MODS	26 (7.4)	8 (3.2)
Chronic hepatic failure	21 (6.0)	15 (6.0)
Hepatic failure	21 (6.0)	23 (9.2)
Sepsis	18 (5.2)	4 (1.6)
Abdominal pain	15 (4.3)	2 (0.8)
Acute respiratory failure	11 (3.2)	5 (2.0)
HRS	11 (3.2)	12 (4.8)
GI hemorrhage	10 (2.9)	1 (0.4)
Hepatic encephalopathy	10 (2.9)	9 (3.6)
Renal failure	10 (2.9)	6 (2.4)
Pneumonia	9 (2.6)	8 (3.2)
Septic shock	9 (2.6)	2 (0.8)
Acute kidney injury	8 (2.3)	5 (2.0)
Esophageal varices hemorrhage	7 (2.0)	4 (1.6)

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

- Risk for respiratory failure with terlipressin:
  - MELD ≥37
  - Baseline Grade 3 HE
  - ACLF Grade 3
  - Significant history of prior or treatment-emergent cardiorespiratory events (eg, dyspnea, wheezing, cardiomegaly, pneumonia/aspiration pneumonia, atelectasis)
  - Recent upper GI hemorrhage

2014

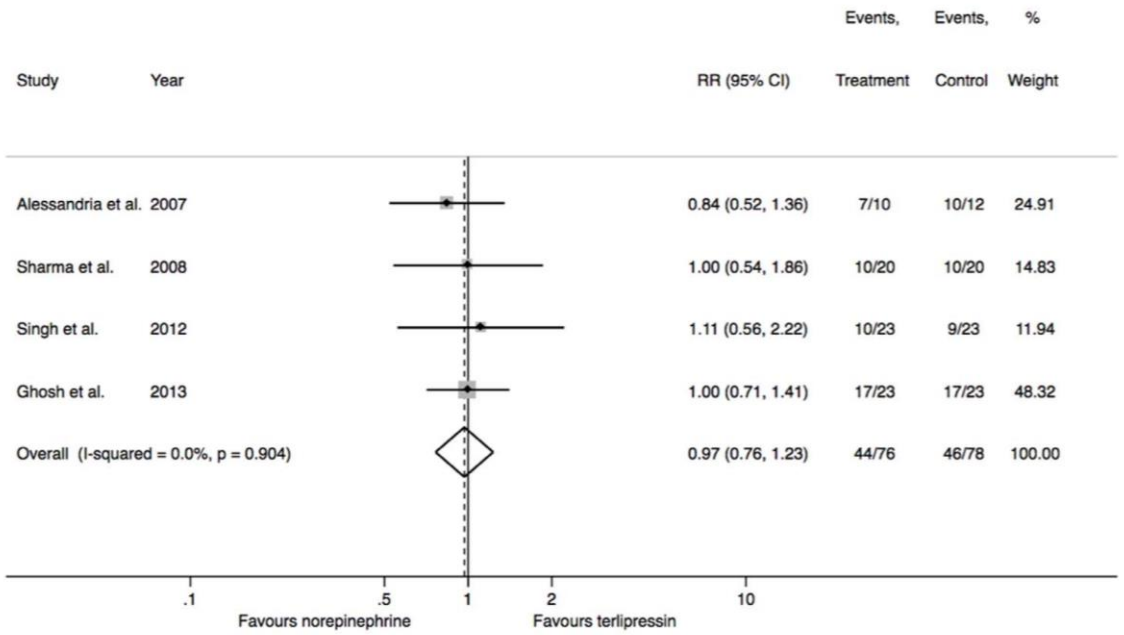
OPEN ACCESS Freely available online

PLOS ONE

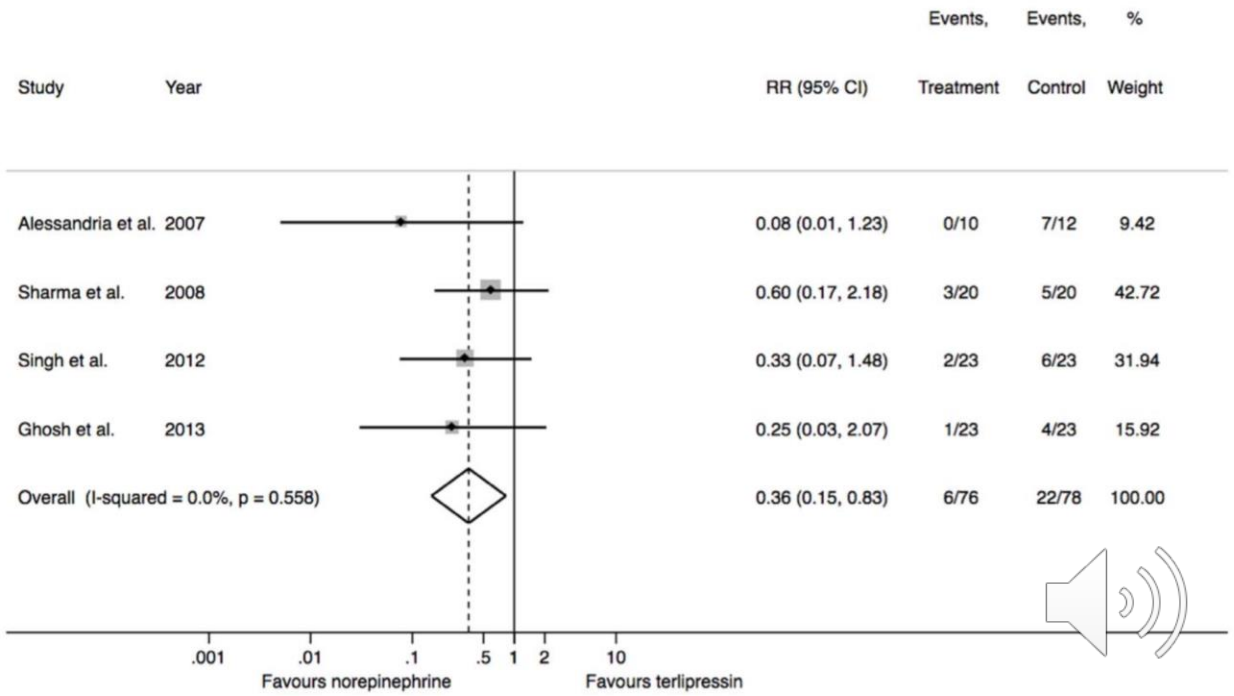
Terlipressin versus Norepinephrine in the Treatment of Hepatorenal Syndrome: A Systematic Review and Meta-Analysis

Antonio Paulo Nassar Junior[1]*, Alberto Queiroz Farias[2], Luiz Augusto Carneiro d' Albuquerque[3], Flair José Carrilho[2], Luiz Marcelo Sá Malbouisson[1]

HRS Reversal



Adverse Events



Terlipressin Given by Continuous Intravenous Infusion Versus Intravenous Boluses in the Treatment of Hepatorenal Syndrome: A Randomized Controlled Study

Marta Cavallin,[1] Salvatore Piano,[1,2] Antonietta Romano,[1] Silvano Fasolato,[1,2] Anna Chiara Frigo,[3] Gianpiero Benetti,[4] Elisabetta Gola,[1] Filippo Morando,[1] Marialuisa Stanco,[1] Silvia Rosi,[1] Antonietta Sticca,[1] Umberto Cillo,[5] and Paolo Angeli[1,2]

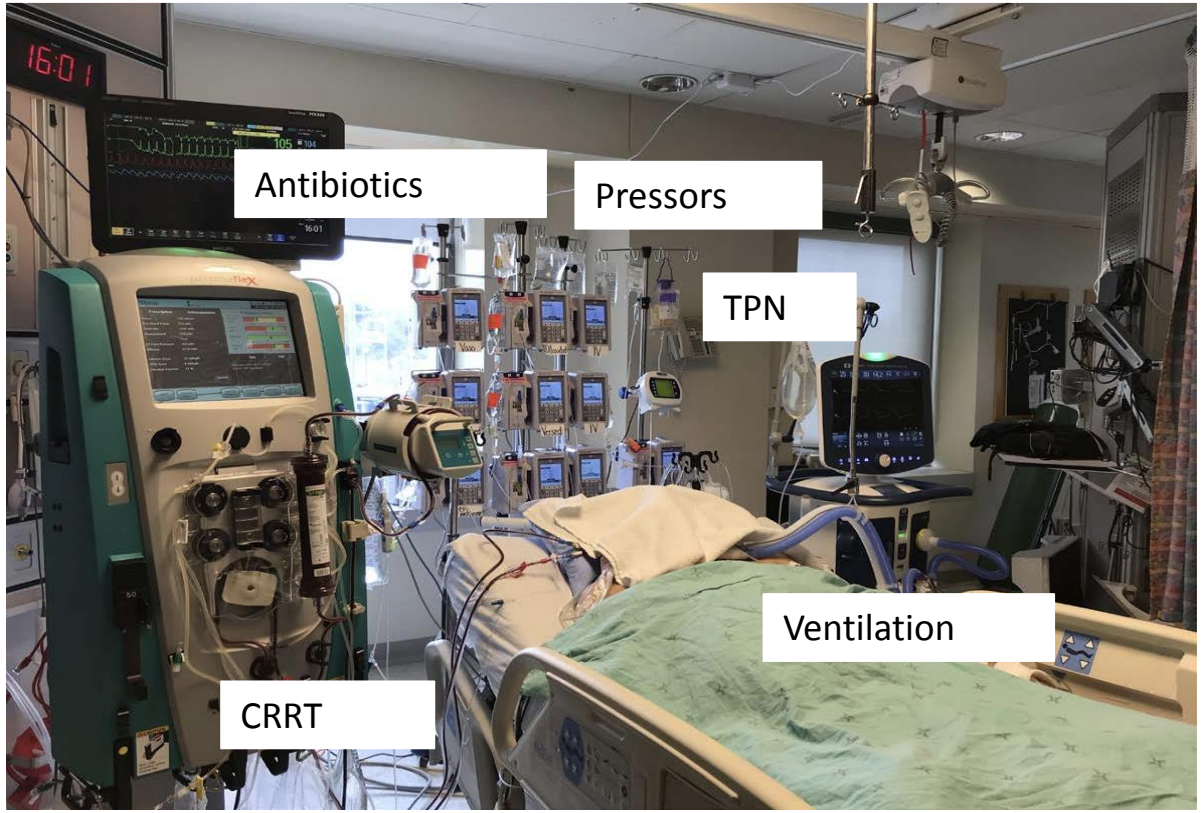
	TERLI-INF Group (n = 34)	TERLI-BOL Group (n = 37)	*P*
Patients, no. (%)	7 (20.59)	16 (43.24)	<0.05
Suspected intestinal ischemia	—	3	—
Peripheral ischemia	1	—	—
Circulatory overload	2	5	—
Angina pectoris	3	3	—
Arrhythmia	—	1	—
Arterial hypertension	1	—	—
Persistent diarrhea	—	2	—

- N=78 patients receiving terlipressin: comparison of boluses versus continuous IV
- Maximum dose 12 mg/day in both groups
- No difference in the rate of response to treatment
- Lower rate of adverse events in the continuous IV group 35% vs 62%)
- Mean daily dose of terlipressin lower in the continuous infusion group

Clinical scenario in the ICU in high MELD regions

To Dialyze or Not to Dialyze: That is the Question!

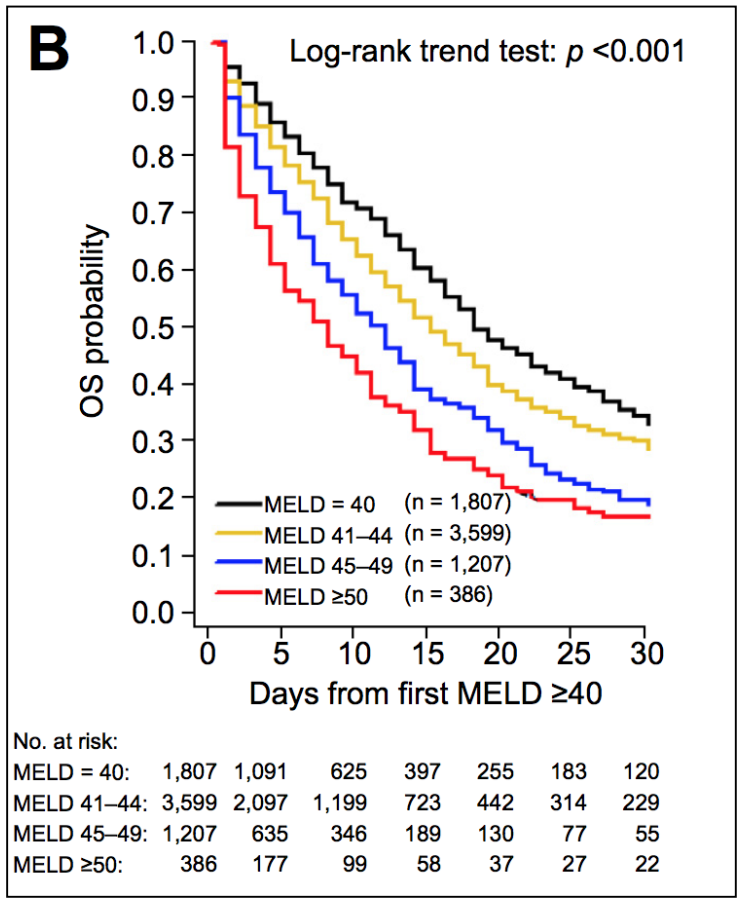
Whether or not to offer dialysis and when to withdraw dialysis is one of the many choices we face on a daily basis when caring for critically ill cirrhotic patients

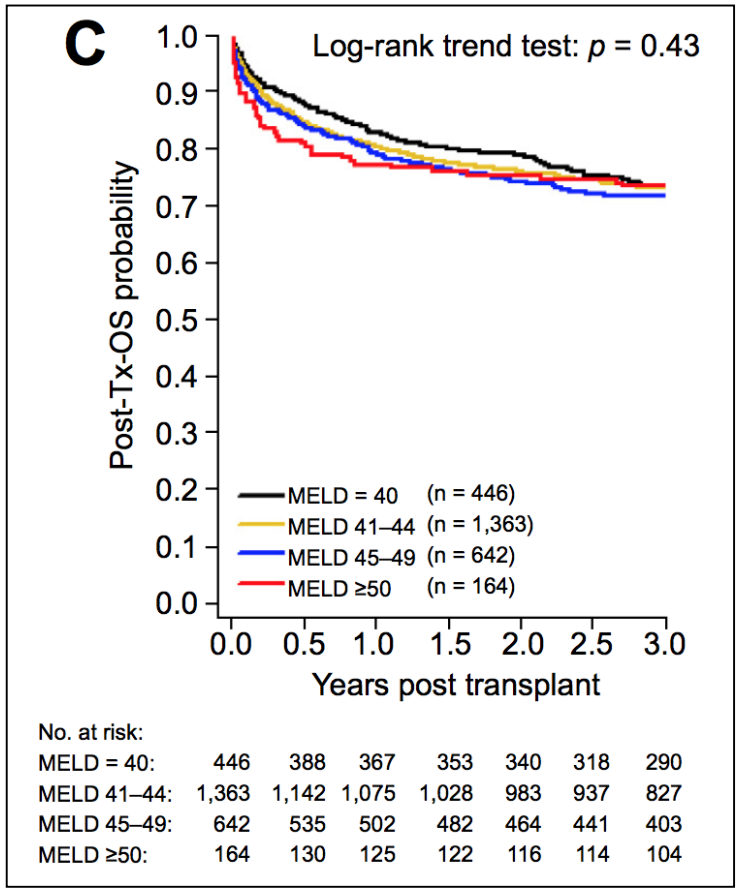


J Hepatol. 2017 September ; 67(3): 517–525.

Inequity in organ allocation for patients awaiting liver transplantation: Rationale for uncapping the model for end-stage liver disease

Mitra K. Nadim[1,*,†], Joseph DiNorcia[2,†], Lingyun Ji[3], Susan Groshen[3], Josh Levitsky[4], Randall S. Sung[5], W. Ray Kim[6], Kenneth Andreoni[7], David Mulligan[8], Yuri S. Genyk[2]





Hepatology 2015

Clinical Course of Acute-on-Chronic Liver Failure Syndrome and Effects on Prognosis

Thierry Gustot,[1*] Javier Fernandez,[2*] Elisabet Garcia,[3] Filippo Morando,[4] Paolo Caraceni,[5] Carlo Alessandria,[6] Wim Laleman,[7] Jonel Trebicka,[8] Laure Elkrief,[9] Corinna Hopf,[10] Pablo Solís-Munoz,[11] Faouzi Saliba,[12] Stefan Zeuzem,[13] Augustin Albillos,[14] Daniel Benten,[15] José Luis Montero-Alvarez,[16] Maria Teresa Chivas,[17] Mar Concepción,[18] Juan Córdoba,[19] Aiden McCormick,[20] Rudolf Stauber,[21] Wolfgang Vogel,[22] Andrea de Gottardi,[23] Tania M. Welzel,[13] Marco Domenicali,[5] Alessandro Risso,[6] Julia Wendon,[11] Carme Deulofeu,[3] Paolo Angeli,[4] François Durand,[9] Marco Pavesi,[3] Alexander Gerbes,[10] Rajiv Jalan,[24] Richard Moreau,[9] Pere Ginés,[2] Mauro Bernardi,[25] and Vicente Arroyo,[25] for the CANONIC Study Investigators of the EASL-CLIF Consortium[†]

Table 1. Clinical Course Patterns and Types in Those Patients With ACLF Studied*

Initial Grade	Final Grade			
	No ACLF (n = 165)	ACLF-1 (n = 70)	ACLF-2 (n = 59)	ACLF-3 (n = 94)
ACLF-1 (%)				
Prevalence (n = 202)	110 (54.5)	49 (24.3)	18 (8.9)	25 (12.4)
28-day tx-free mortality (n = 190)	7/104 (6.7)	10/47 (21.3)	8/15 (53.3)	21/24 (87.5)
90-day tx-free mortality (n = 172)	19/95 (20.0)	17/41 (41.5)	10/13 (76.9)	23/23 (100)
ACLF-2 (%)				
Prevalence (n = 136)	47 (34.6)	19 (14.0)	35 (25.7)	35 (25.7)
28-day tx-free mortality (n = 118)	1/42 (2.4)	2/17 (11.8)	8/27 (29.6)	29/32 (90.63)
90-day tx-free mortality (n = 110)	5/39 (12.8)	5/16 (31.3)	18/23 (78.3)	32/32 (100)
ACLF-3 (%)				
Prevalence (n = 50)	8 (16.0)	2 (4.0)	6 (12)	34 (68)
28-day tx-free mortality (n = 45)	1/8 (12.5)	0/2 (0.0)	4/6 (66.7)	28/29 (96.6)
90-day tx-free mortality (n = 45)	1/8 (12.5)	1/2 (50.0)	4/6 (66.7)	28/29 (96.6)

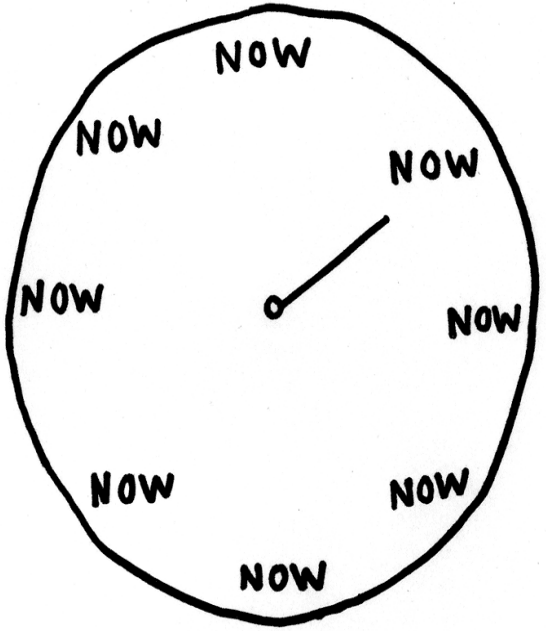
ACLF: resolution or improvement (green boxes); steady *or* fluctuating course with unchanged final ACLF grade (uncolored boxes); and worsening (red boxes).

Timing of RRT

Biochemical threshold

- **Cr, BUN, potassium, pH**

Transplant Candidacy



Onset of Symptoms

- **Encephalopathy**
- **Fluid overload**
- **Oliguria**

Relative to onset of AKI

JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PAT

Effect of Early vs Delayed Initiation of Therapy on Mortality in Critically Ill Patients With Acute Kidney Injury

The ELAIN Randomized Clinical Trial

Alexander Zarbock, MD; John A. Kellum, MD; Christoph Schmidt, MD; Hugo Van Aken, M
Hermann Pavenstädt, MD; Andreea Boanta, MD; Joachim Gerß, PhD; Melanie Meersch, M

IMPORTANCE Optimal timing of initiation of renal replacement therapy (RR
kidney injury (AKI) but without life-threatening indications is still unknown

OBJECTIVE To determine whether early initiation of RRT in patients who ar
AKI reduces 90-day all-cause mortality.

DESIGN, SETTING, AND PARTICIPANTS Single-center randomized clinical tria
critically ill patients with AKI Kidney Disease: Improving Global Outcomes (
(≥2 times baseline or urinary output <0.5 mL/kg/h for ≥12 hours) and plas
gelatinase–associated lipocalin level higher than 150 ng/mL enrolled betwe
and June 2015 from a university hospital in Germany.

INTERVENTIONS Early (within 8 hours of diagnosis of KDIGO stage 2: n = 1
(withi

MAIN
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7.0]) t
to −18.0); P < .001). Early initiation of RRT significantly reduced 90-day mo
patients [39.3%]) compared with delayed initiation of RRT (65 of 119 patien
hazard ratio [HR], 0.66 [95% CI, 0.45 to 0.97]; difference, −15.4% [95% CI
P = .03). More patients in the early group recovered renal function by day 9
patients [53.6%] in the early group vs 46 of 119 patients [38.7%] in the dela
ratio [OR], 0.55 [95% CI, 0.32 to 0. 93]; difference, 14.9% [95% CI, 2.2% to
Duration of RRT and length of hospital stay were significantly shorter in the
in the delayed group (RRT: 9 days [Q1, Q3: 4, 44] in the early group vs 25 da
in the delayed group; P = .04; HR, 0.69 [95% CI, 0.48 to 1.00]; difference,
−41 to 4]; hospital stay: 51 days [Q1, Q3: 31, 74] in the early group vs 82 days
in the delayed group; P < .001; HR, 0.34 [95% CI, 0.22 to 0.52]; difference,
−∞ to −19.5]), but there was no significant effect on requirement of RRT aft
dysfunction, and length of ICU stay.

CONCLUSIONS AND RELEVANCE Among critically ill patients with AKI, early
with delayed initiation of RRT reduced mortality over the first 90 days. Fur
trials of this intervention are warranted.

TRIAL REGISTRATION German Clinical Trial Registry Identifier: DRKS0000

JAMA. 2016;315(20):2190-2199. doi:10.1001/jama.2016.5828
Published online May 22, 2016. Corrected on August 23, 2016.

ORIGINAL ARTIC

Initiation Strategies for Re Therapy in the Intensiv

Stéphane Gaudry, M.D., David Hajage, M.D., F
Laurent Martin-Lefevre, M.D., Bertrand Pons
Alexandre Boyer, M.D., Guillaume Chevrel, M.D.,
Dorothée Carpentier, M.D., Nicolas de
Alexandre Lautrette, M.D., Anne Bretagnol, M
Saad Nseir, M.D., Ph.D., Bruno Megarbane, M.D.
Jean-Marie Forel, M.D., Julien Maizel, M.D., P
Philippe Markowicz, M.D., Guillaume Thiery, M.D.,
Jean-Damien Ricard, M.D., Ph.D., and D
for the AKIKI Study Gro

ABSTRACT

BACKGROUND
The timing of renal-replacement therapy in critically i

was met: severe hyperkalemia, metabolic acidosis, pul
gen level higher than 112 mg per deciliter, or oligu
randomization. The primary outcome was overall surv

RESULTS
A total of 620 patients underwent randomization.
mortality at day 60 did not differ significantly betwee
150 deaths occurred among 311 patients in the early-s
dence interval [CI], 42.6 to 53.8), and 153 deaths occ
delayed-strategy group (49.7%, 95% CI, 43.8 to 55.0;
(49%) in the delayed-strategy group did not receive rena
catheter-related bloodstream infections was higher in t
delayed-strategy group (10% vs. 5%, P=0.03). Diures
function, occurred earlier in the delayed-strategy grou

CONCLUSIONS
In a trial involving critically ill patients with severe
significant difference with regard to mortality betwee
for the initiation of renal-replacement therapy. A dela
renal-replacement therapy in an appreciable number o
Ministry of Health; ClinicalTrials.gov number, NCT01

ORIGINAL ARTICLE

Timing of Renal-Replacemer in Patients with Acute Kidney Inju

S.D. Barbar, R. Clere-Jehl, A. Bourredjem, R. Hernu, F.
C. Lebert, J. Bohé, J. Badie, J.-P. Eraldi, J.-P. Rigaud,
G. Louis, L. Bouadma, J.-M. Constantin, E. Mercier, K. K
G. Piton, D. Annane, S. Jaber, T. van der Linden, G.
C. Schwebel, L. Chimot, P. Guiot, M.-A. Nay, F. Meziar
B. Louart, R. Trusson, A. Dargent, C. Binquet, ar
for the IDEAL-ICU Trial Investigators and the CRICS TR

ABSTRACT

BACKGROUND
Acute kidney injury is the most frequent complication in p
and is an independent risk factor for death. Although ren

sification system is characterized by a serum creatinine le
level (or ≥4 mg per deciliter with a rapid increase of ≥0.5
output less than 0.3 ml per kilogram of body weight p
longer, or anuria for at least 12 hours. The primary outco

RESULTS
The trial was stopped early for futility after the second p
A total of 488 patients underwent randomization; there wer
group differences in the characteristics at baseline. Amo
whom follow-up data at 90 days were available, 58% of t
strategy group (138 of 239 patients) and 54% in the dela
of 238 patients) had died (P=0.38). In the delayed-strategy
did not receive renal-replacement therapy. Criteria for eme
therapy were met in 17% of the patients in the delayed-stra

CONCLUSIONS
Among patients with septic shock who had severe acute
no significant difference in overall mortality at 90 days be
assigned to an early strategy for the initiation of renal-r
those who were assigned to a delayed strategy. (Funded b
Health; IDEAL-ICU ClinicalTrials.gov number, NCT01682

ORIGINAL ARTICLE

Timing of Initiation of Renal-Replacer Therapy in Acute Kidney Injury

The STARRT-AKI Investigators, for the Canadian Critical Care Trials
the Australian and New Zealand Intensive Care Society Clinical Trials
the United Kingdom Critical Care Research Group, the Canadian Nep
Trials Network, and the Irish Critical Care Trials Group*

ABSTRACT

BACKGROUND
Acute kidney injury is common in critically ill patients, many of who
renal-replacement therapy. However, the most effective timing for the in
such therapy remains uncertain.

METHODS
We conducted a multinational, randomized, controlled trial involving c

in 903 (61.8%) in the standard-strategy group. At 90 days, death had o
643 patients (43.9%) in the accelerated-strategy group and in 639 (43.7
standard-strategy group (relative risk, 1.00; 95% confidence interval [C
1.09; P=0.92). Among survivors at 90 days, continued dependence
replacement therapy was confirmed in 85 of 814 patients (10.4%) in the a
strategy group and in 49 of 815 patients (6.0%) in the standard-strat
(relative risk, 1.74; 95% CI, 1.24 to 2.43). Adverse events occurred in 34
patients (23.0%) in the accelerated-strategy group and in 245 of 148
(16.5%) in the standard-strategy group (P<0.001).

CONCLUSIONS
Among critically ill patients with acute kidney injury, an accelerated ren
ment strategy was not associated with a lower risk of death at 90 da
standard strategy. (Funded by the Canadian Institutes of Health Res
others; STARRT-AKI ClinicalTrials.gov number, NCT02568722.)

Comparison of two delayed strategies for renal replacement therapy initiation for severe acute kidney injury (AKIKI 2): a multicentre, open-label, randomised, controlled trial

Stéphane Gaudry, David Hajage, Laurent Martin-Lefevre, Saïd Lebbah, Guillaume Louis, Sébastien Moschietto, Dimitri Titeca-Beauport, Béatrice La Combe, Bertrand Pons, Nicolas de Prost, Sébastien Besset, Alain Combes, Adrien Robine, Marion Beuzelin, Julio Badie, Guillaume Chevrel, Julien Bohé, Elisabeth Couper, Nicolas Chudeau, Saber Barbar, Christophe Vinsonneau, Jean-Marie Forel, Didier Thevenin, Eric Boulet, Karim Lakhal, Nadia Aissaoui, Steven Grange, Marc Leone, Guillaume Lacave, Saad Nseir, Florent Poirson, Julien Mayaux, Karim Asehnoune, Guillaume Geri, Kada Klouche, Guillaume Thiery, Laurent Argaud, Bertrand Rozec, Cyril Cadoz, Pascal Andreu, Jean Reignier*, Jean-Damien Ricard*, Jean-Pierre Quenot', Didier Dreyfuss†

Summary

Background Delaying renal replacement therapy (RRT) for some time in critically ill patients with severe acute kidney injury and no severe complication is safe and allows optimisation of the use of medical devices. Major uncertainty remains concerning the duration for which RRT can be postponed without risk. Our aim was to test the hypothesis that a more-delayed initiation strategy would result in more RRT-free days, compared with a delayed strategy.

Methods This was an unmasked, multicentre, prospective, open-label, randomised, controlled trial done in 39 intensive care units in France. We monitored critically ill patients with severe acute kidney injury (defined as Kidney Disease: Improving Global Outcomes stage 3) until they had oliguria for more than 72 h or a blood urea nitrogen concentration higher than 112 mg/dL. Patients were then randomly assigned (1:1) to either a strategy (delayed strategy) in which RRT was started just after randomisation or to a more-delayed strategy. With the more-delayed strategy, RRT initiation was postponed until mandatory indication (noticeable hyperkalaemia or metabolic acidosis or pulmonary oedema) or until blood urea nitrogen concentration reached 140 mg/dL. The primary outcome was the

went randomisation;
plications potentially
of RRT-free days was
ategy (p=0·93). In a
·018) with the more-
idney injury or renal

blood urea nitrogen
concentration higher than 112 mg/dL and no severe complication that would mandate immediate RRT, longer postponing of RRT initiation did not confer additional benefit and was associated with potential harm.

Funding Programme Hospitalier de Recherche Clinique.

Introduction

Severe acute kidney injury is frequent among critically ill patients hospitalised in intensive care units (ICUs) and is associated with high morbidity and mortality.[1] Major uncertainty remains concerning the duration for which renal replacement therapy (RRT) can be postponed without risk as criteria for initiating RRT lack precision in the absence of complication. The majority of well conducted, randomised, controlled trials[2,3] including a recently issued very large one[4] as well as a large individual patient data meta-analysis[5] showed that an early RRT initiation strategy did not confer any survival advantage compared with a delayed strategy during severe acute kidney injury in critically ill patients when no severe complication is present.[6] Moreover, early institution of this technique was associated with more complications, some being very severe.[2,4]

The duration for which RRT initiation was delayed varied considerably, expanding from 25 hours to 57 h according to study.[2-4] The large variation in the criteria retained for initiating RRT in the delayed group of these studies[2-4] was responsible for this marked heterogeneity. The longer RRT is safely postponed, the more numerous are patients who do not receive this treatment. Severe

Patients with liver disease were excluded or were largely underrepresented (11% in STAART AKI)

2016

Seminar

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Management of the critically ill patient with cirrhosis: A multidisciplinary perspective

Mitra K. Nadim[1,*], Francois Durand[2], John A. Kellum[3], Josh Levitsky[4], Jacqueline G. O'Leary[5], Constantine J. Karvellas[6], Jasmohan S. Bajaj[7], Andrew Davenport[8], Rajiv Jalan[9], Paolo Angeli[10], Stephen H. Caldwell[11], Javier Fernández[12], Claire Francoz[2], Guadalupe Garcia-Tsao[13], Pere Ginès[12], Michael G. Ison[14], David J. Kramer[15], Ravindra L. Mehta[16], Richard Moreau[2], David Mulligan[17], Jody C. Olson[18], Elizabeth A. Pomfret[19], Marco Senzolo[20], Randolph H. Steadman[21], Ram M. Subramanian[22], Jean-Louis Vincent[23], Yuri S. Genyk[24]

1. The initiation of RRT should be made on clinical grounds, including worsening AKI, worsening volume overload despite diuretic therapy, worsening metabolic acidosis, hyperkalemia and hyponatremia not responding to medical management, and diuretic intolerance/resistance (1D)

2. RRT should be considered even in non-oliguric patients if the daily fluid balance cannot be maintained as even or negative (Ungraded)

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Diagnosis, Evaluation, and Management of Ascites, Spontaneous Bacterial Peritonitis and Hepatorenal Syndrome: 2021 Practice Guidance by the American Association for the Study of Liver Diseases

Scott W. Biggins,[1] Paulo Angeli,[2] Guadalupe Garcia-Tsao,[3,4] Pere Ginès,[5,6] Simon C. Ling,[7] Mitra K. Nadim,[8] Florence Wong,[9] and W. Ray Kim[10]

- RRT should be used in candidates for LT with worsening renal function or electrolyte disturbances or increasing volume overload unresponsive to vasoconstrictor therapy. Initiation of RRT in patients who are not candidates for LT must be made with a clear endpoint in mind.

Group 4: Management of Kidney Dysfunction in Patients with Cirrhosis

Chair / Co-Chair: Jody Olson – CC/Hep (USA), Andrew Allegretti – Neph (USA)

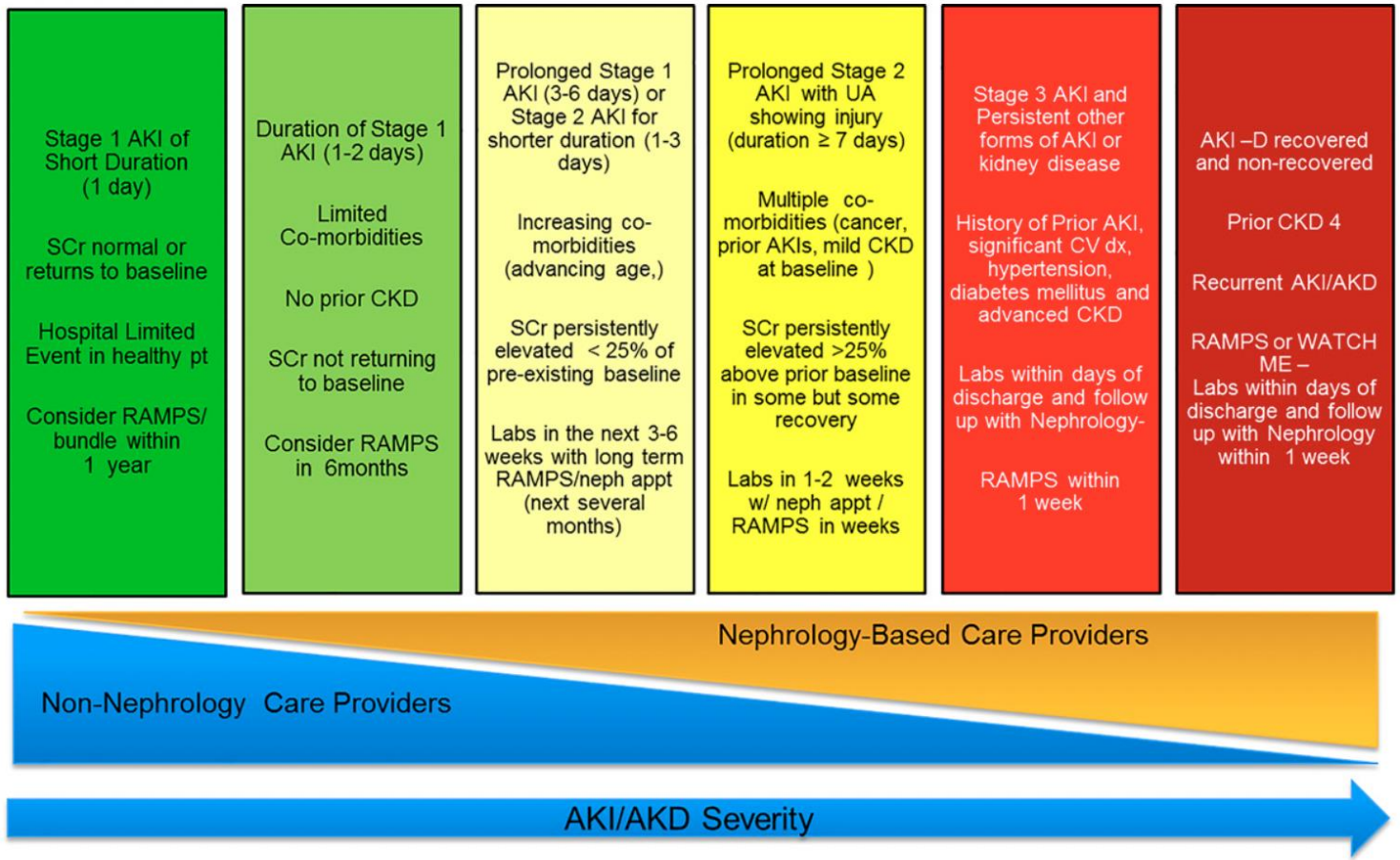
Members: Pere Gines – Hep (Spain), *Nuttha Lumertgul – CC/Neph (Thailand),* Yuri Genyk – Tx Surgery (USA), Rakhi Maiwall – Hep (India)

1. What fluids should be used for treatment of AKI?
2. What is the role of vasoconstrictors in patients and which type of vasoconstrictors should be first line therapy for patients with HRS?
3. What are the targets / endpoints for patients started on vasoconstrictors – MAP, CVP, recovery?
4. What is the indication, timing, and patient characteristic for initiation of ECOS in patients with AKI ?

Quality Improvement Goals for Acute Kidney Injury

Kianoush Kashani,[1] Mitchell Howard Rosner,[2] Michael Haase,[3,4] Andrew J.P. Lewington,[5,6] Donal J. O'Donoghue,[7] F. Perry Wilson,[8] Mitra K. Nadim,[9] Samuel A. Silver,[10] Alexander Zarbock,[11] Marlies Ostermann,[12] Ravindra L. Mehta,[13] Sandra L. Kane-Gill,[14] Xiaoqiang Ding,[15] Peter Pickkers,[16] Azra Bihorac,[17] Edward D. Siew,[18,19,20] Erin F. Barreto,[21] Etienne Macedo,[13] John A. Kellum,[22] Paul M. Palevsky,[23,24] Ashita Jiwat Tolwani,[25] Claudio Ronco,[26,27,28] Luis A. Juncos,[29] Oleksa G. Rewa,[30] Sean M. Bagshaw,[30] Theresa Ann Mottes,[31] Jay L. Koyner,[32] Kathleen D. Liu,[33] Lui G. Forni,[34] Michael Heung,[35] and Vin-Cent Wu[36]

Schematic for AKI/AKD follow-up.



CJASN 14: 941–953, 2019.

Group 5: Follow up and rehabilitation of patients with kidney dysfunction in setting of cirrhosis

Chair / Co-Chair: Betsy Verna - Hep (USA), Javier Neyra –Neph (USA)

Members: Raimund Pitchler – Neph (USA), Sebastian Marciano – Hep (Argentina), Etienne Macedo – Neph (USA), Puneeta Tandon – Hep (Canada)

1. How should patients be monitored following an episode of AKI?
2. What outcomes should be assessed following episode of AKI?

3. What is the role of palliative care in patients with kidney dysfunction who are not transplant candidates?

ADQI XXIX – Liver and Kidney