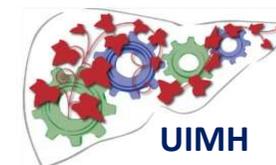


Management of AKI in Cirrhosis

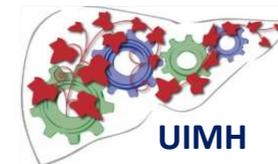
Salvatore Piano, MD, PhD
Unit of Internal Medicine and Hepatology -UIMH
Department of Medicine - DIMED
University of Padova, Italy

*Inedsys Hepatology Club
October 18th, 2025*

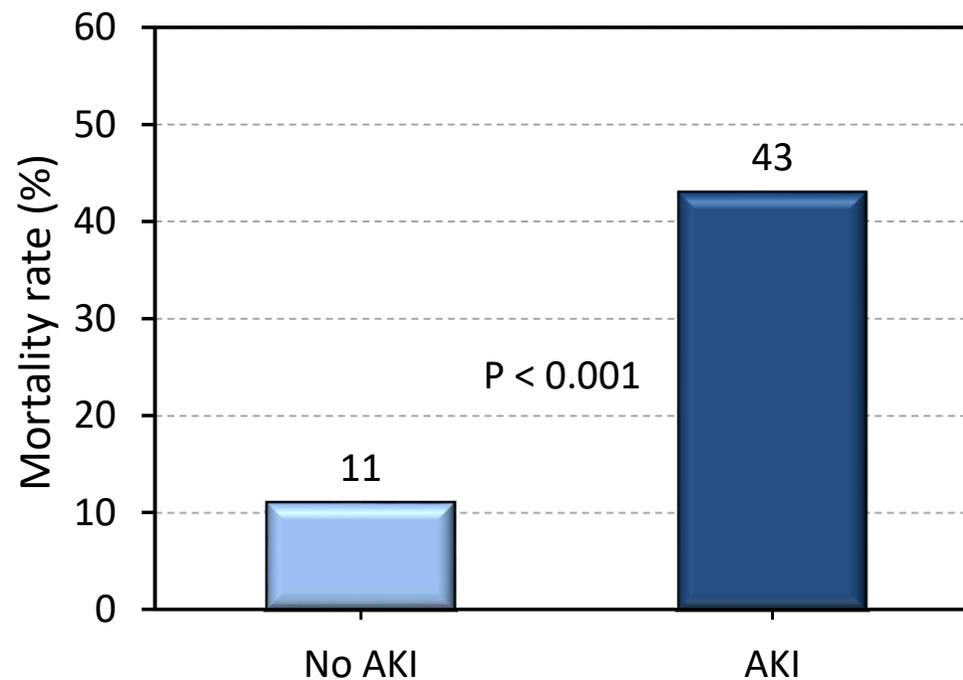
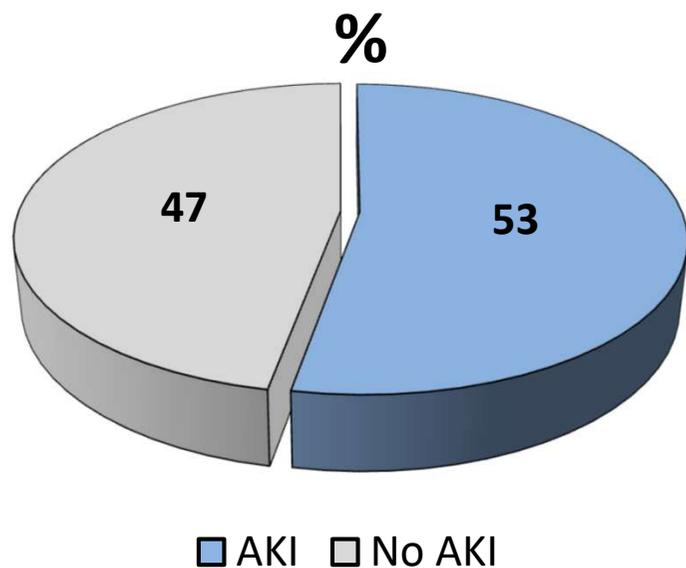


Outline

- Diagnosis and treatment of AKI
- Controversial issues and future strategies



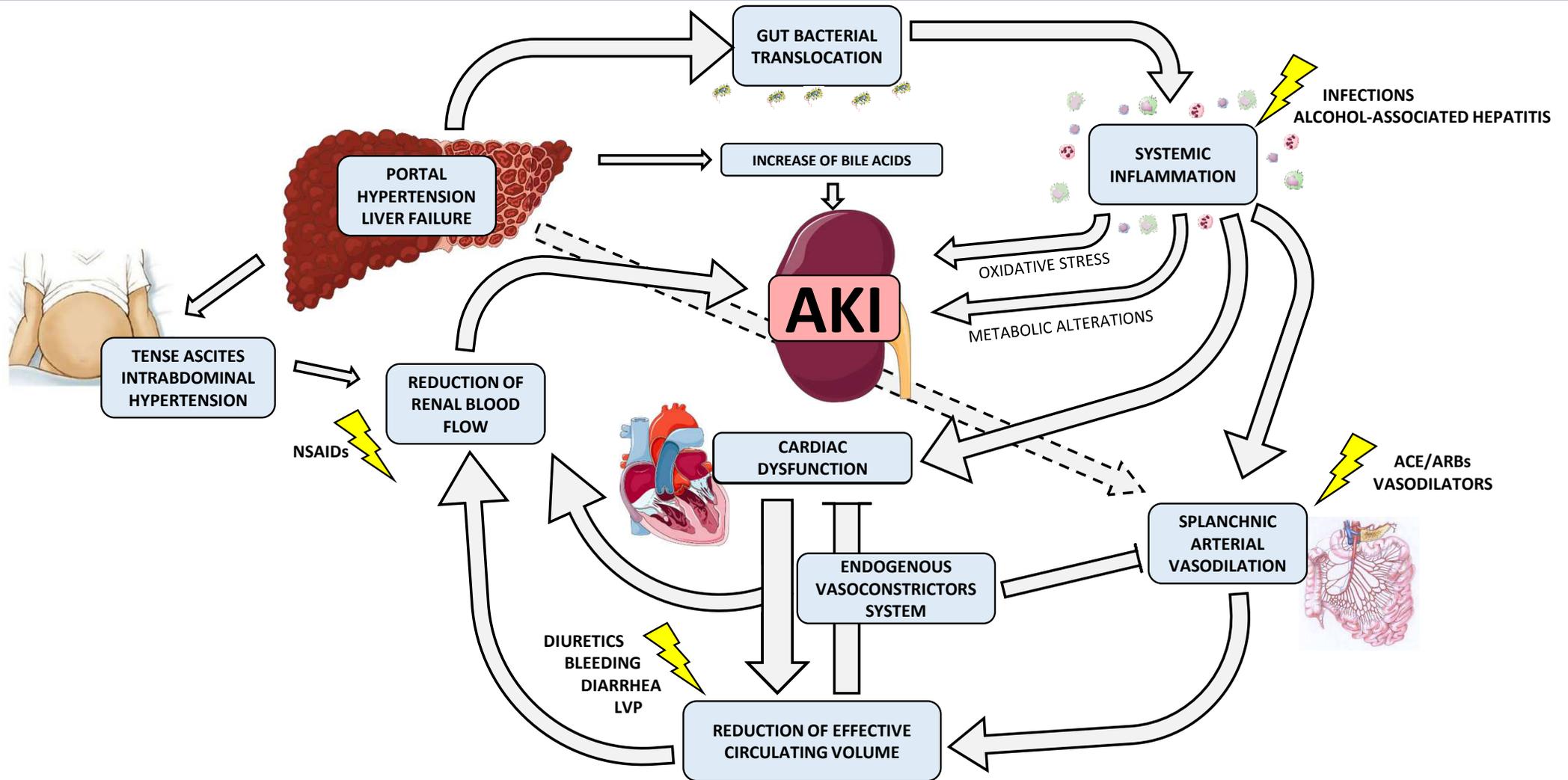
Prevalence and of acute kidney injury (AKI) and 90-day mortality in hospitalized patients with cirrhosis

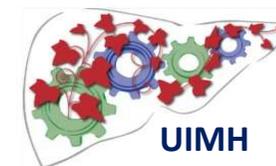


Huelin P, Piano S et al. Clin Gastroenterol Hepatol. 2017 ; 15 : 438-445



Pathophysiology of acute kidney injury in cirrhosis





Hepatorenal syndrome (HRS-AKI)

- HRS-AKI is a predominantly functional renal failure that often occurs in patients with cirrhosis and ascites
- HRS-AKI has features of prerenal AKI, but without any improvement after volume expansion
- HRS develops as a consequence of a severe reduction of effective circulating volume due to both extreme splanchnic arterial vasodilatation and relative reduction in cardiac output
- The hyperactivation of endogenous vasoactive systems cause a severe renal vasoconstriction

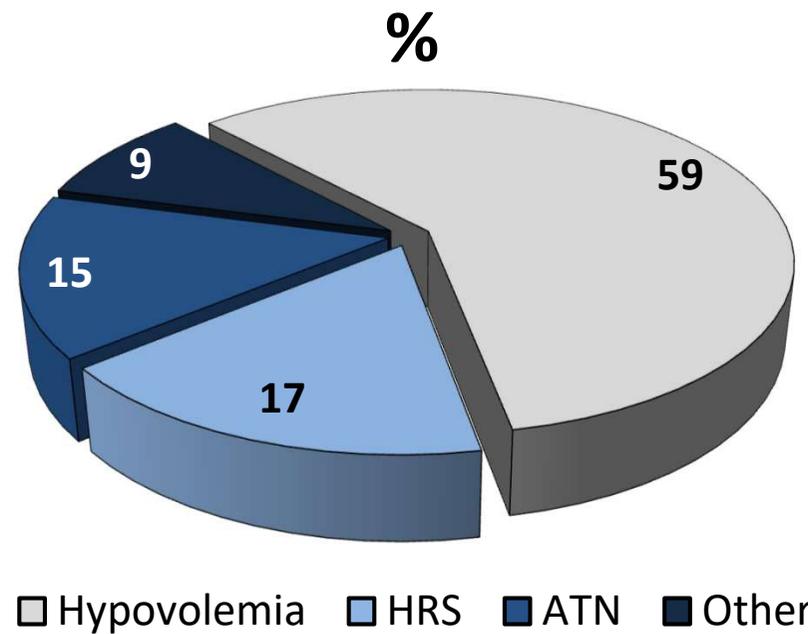
Arroyo V et al. Hepatology. Hepatology 1996 ; 23 : 164-176

Salerno F et al. Gut 2007 ; 56 : 1310-1318.

Angeli P et al. J Hepatol. 2015; 62 : 968-974

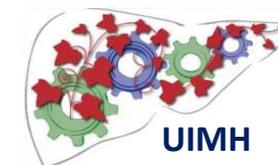


Phenotype of AKI in patients with cirrhosis (n=1,456)

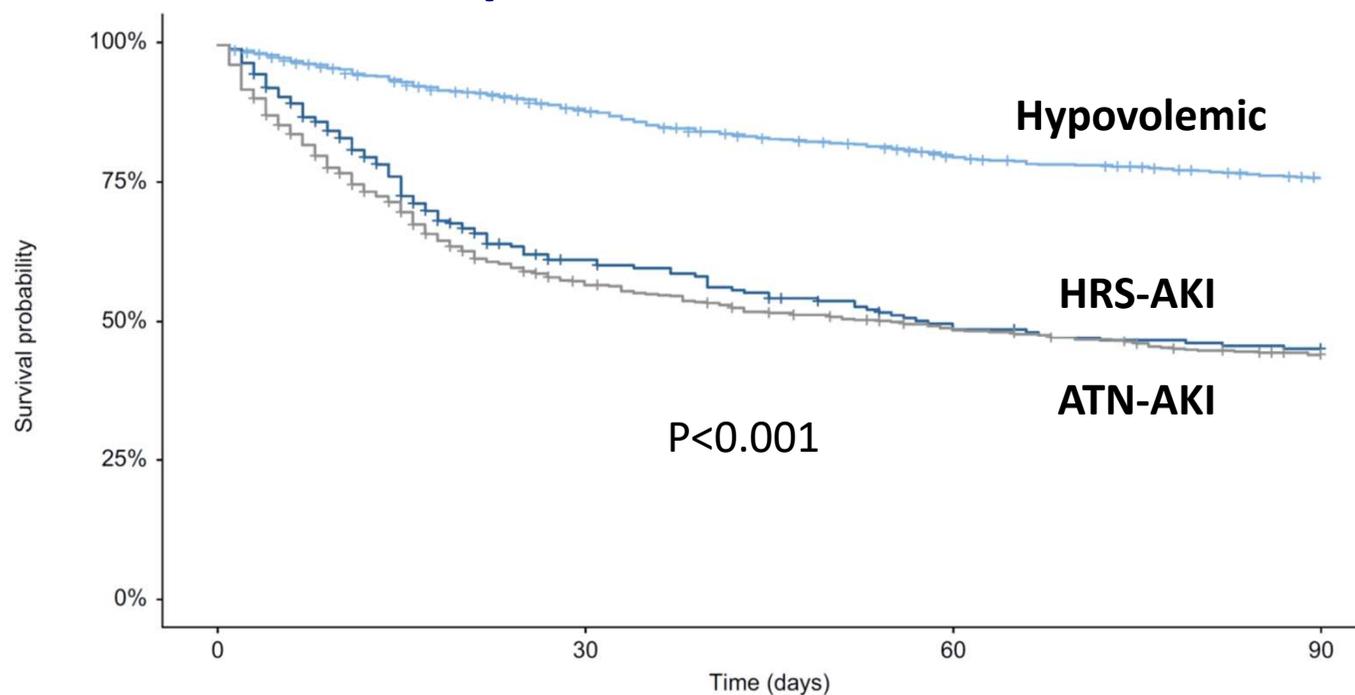


Patidar KR, [...] Piano S. *Lancet Gastroenterol Hepatol* 2025 ; 10 : 418-430.





3-month survival according to type of AKI in patients with cirrhosis



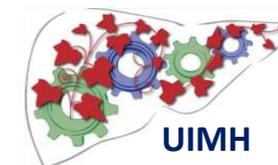
Patidar KR et al. *J Hepatol.* 2023 ; 79 : 1408-1417



Definition of acute kidney injury (AKI)

- Increase in SCr ≥ 0.3 mg/dl (26.5 $\mu\text{mol/L}$) within 48 hours
- Increase in SCr $\geq 50\%$ from baseline within one week
- Urine output < 0.5 ml/kg/h for > 6 hours

*KDIGO. Kidney Int Suppl. 2012;2:1–138.
Nadim M et al. J Hepatol. 2024;81:163–183*



Staging of acute kidney injury (AKI)

Staging	Serum creatinine criteria
Stage 1	Increase in serum creatinine of more than or equal to 0.3 mg/dl ($\geq 26.4 \mu\text{mol/l}$) within 48 hours or a percentage increase in serum creatinine of more or equal to 50 % (< 2-fold from baseline) within one week.
Stage 2	Increase in serum creatinine to more than 200% to 300% (> 2- to 3-fold) from baseline
Stage 3	Increase in serum creatinine to more than 300 % (> 3-fold) from baseline or serum creatinine of more or equal to 4.0 mg/dl ($\geq 354 \mu\text{mol/l}$) with an acute increase of at least 0.3 mg/dl (44 $\mu\text{mol/l}$) or need for renal replacement therapy

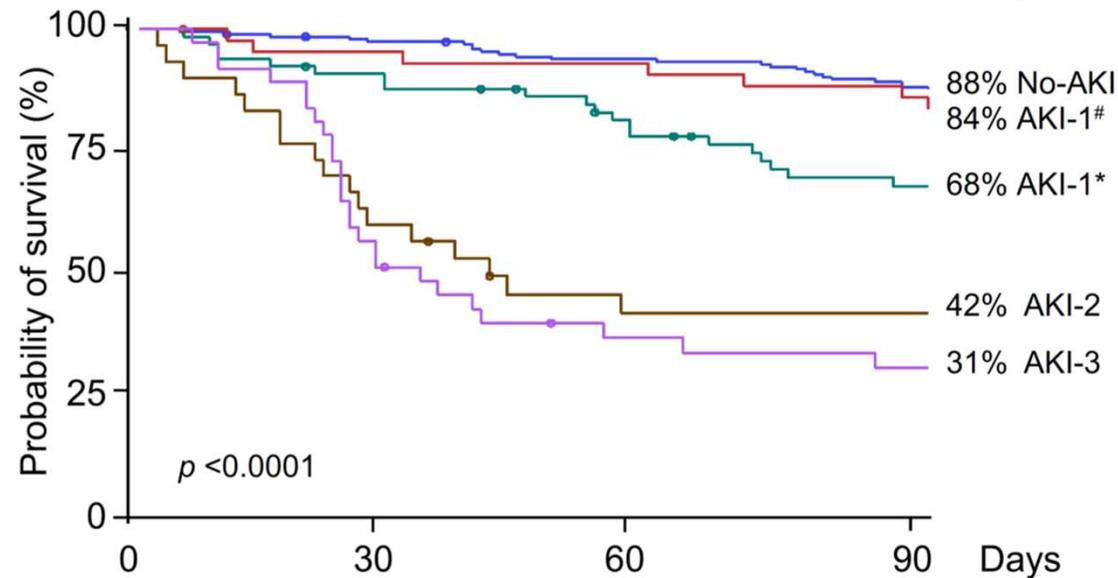


Acute Kidney Injury



AKI stage 1 subclassification according to serum creatinine

- AKI stage 1A: AKI and serum creatinine < 1.5 mg/dl
- AKI stage 1B: AKI and serum creatinine \geq 1.5 mg/dl

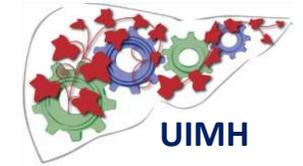


Fagundes C et al. *J Hepatol.* 2013; 59 : 482-489

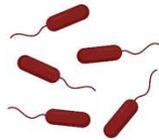
Piano S et al. *J Hepatol.* 2013; 59 : 482-489

Huelin P, Piano S et al. *Clin Gastroenterol Hepatol.* 2017 ;15 : 438-445

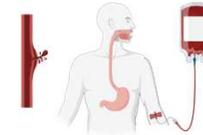
Patidar KR et al. *Liver Transpl.* 2024 ; 30 : 244-253



Initial management of AKI in decompensated cirrhosis



Identify and treat precipitating factors





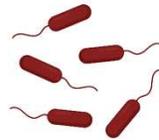
Prevalence of precipitating factors in 1,456 patients with cirrhosis and AKI

Variables	N (%)
Volume loss	683 (46.9)
Infections	613 (42.1)
GI bleeding	251 (17.2)
Nephrotoxic drugs	82 (5.6)
Others	42 (2.9)
No identifiable precipitants	142 (9.8)

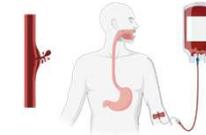
Patidar KR. [...] Piano S. *Lancet Gastroenterol Hepatol* 2025 ; 10 : 418-430.



Initial management of AKI in decompensated cirrhosis



Identify and treat precipitating factors



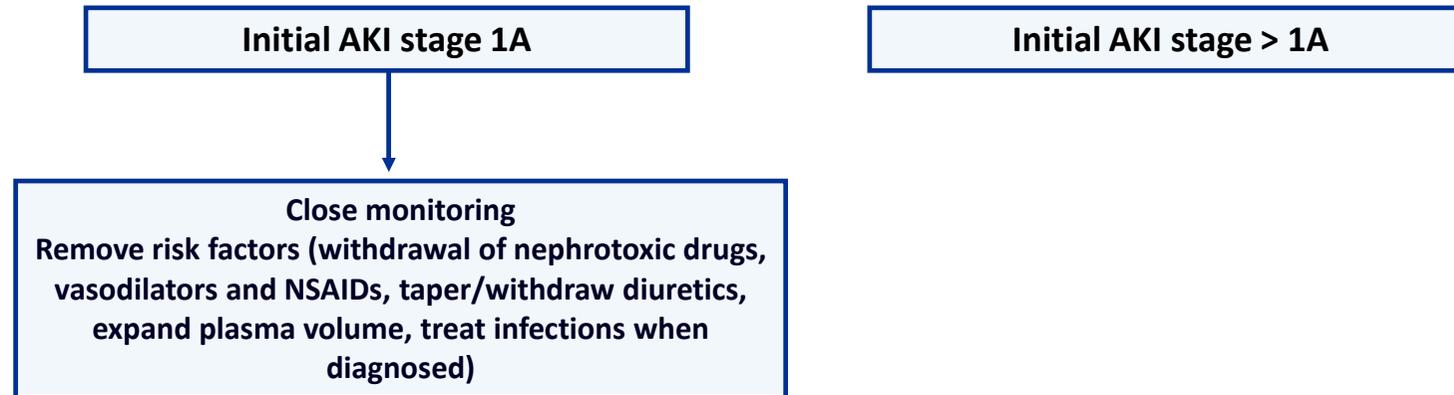
Volume resuscitation



Differential diagnosis and specific AKI treatment



Algorithm for AKI management in patients with cirrhosis

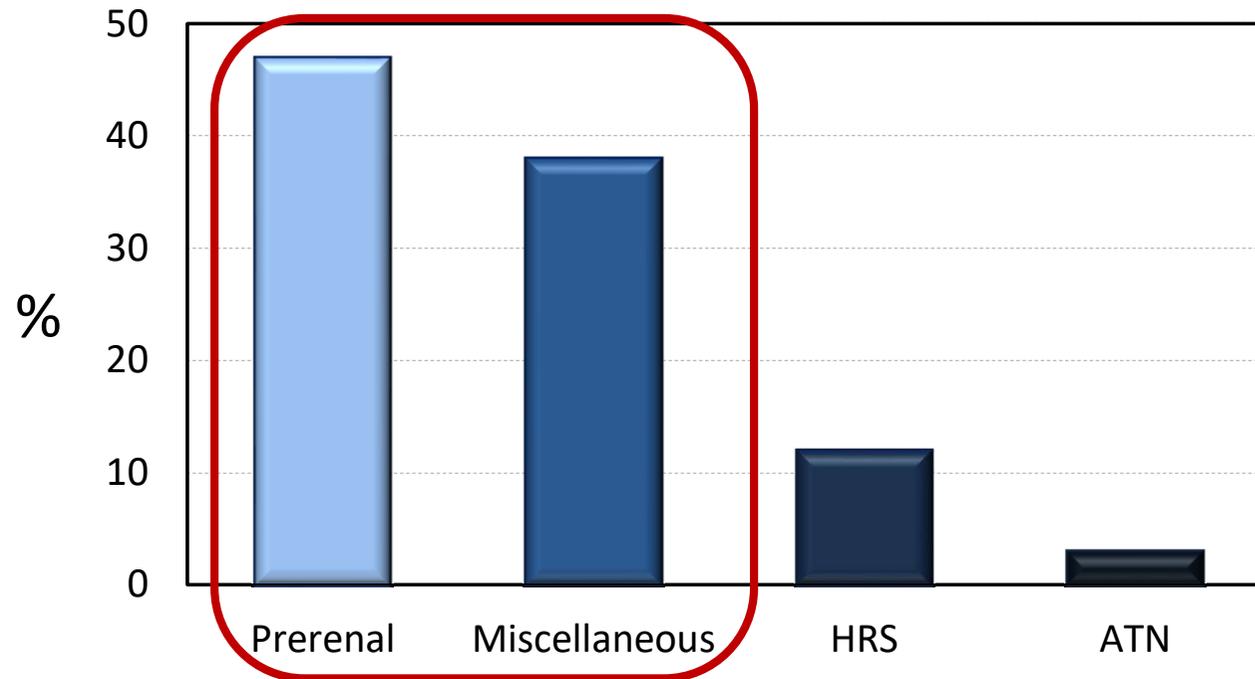




Acute Kidney Injury

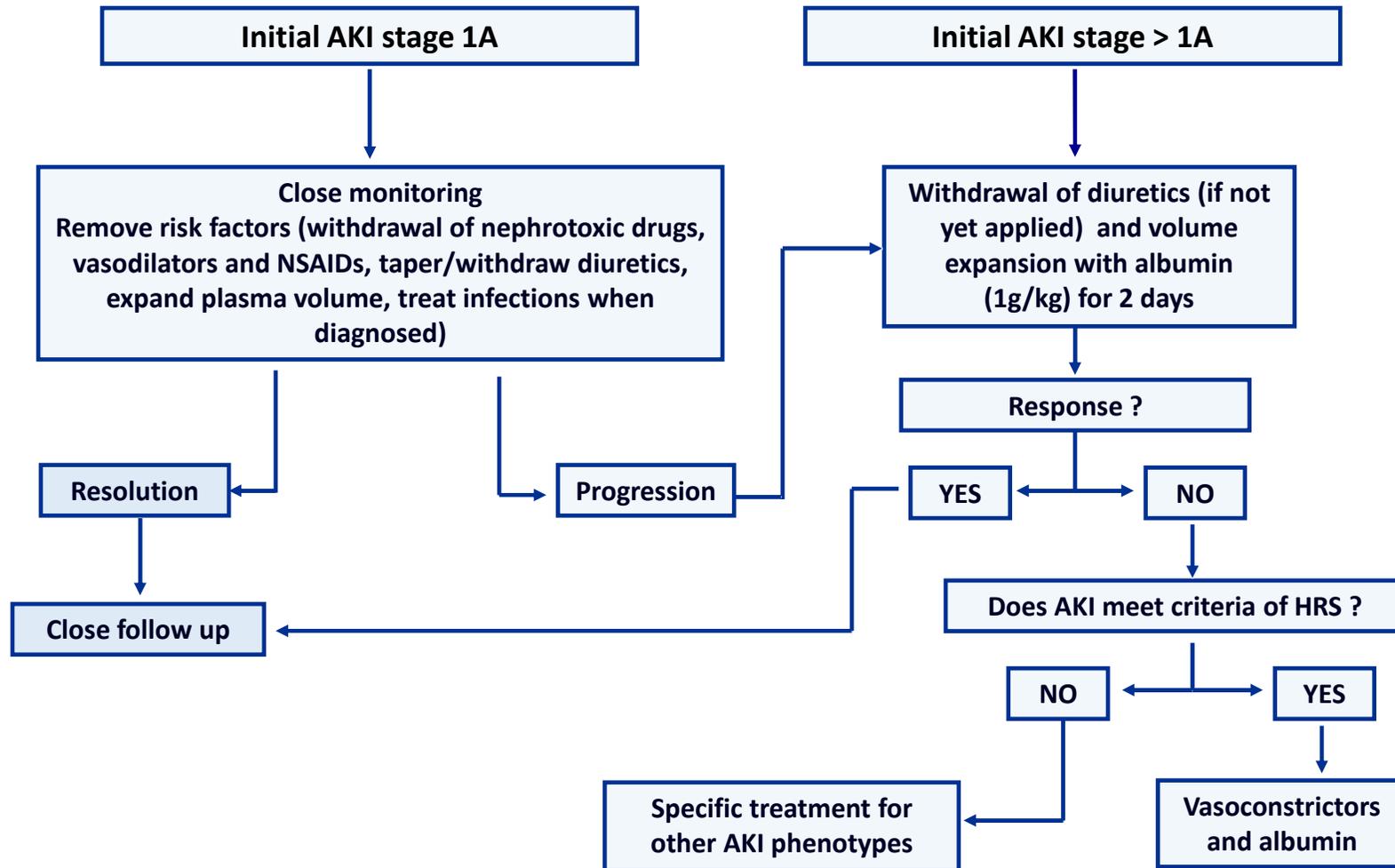


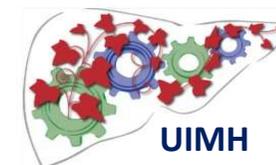
Causes of renal failure in patients with AKI stage 1A



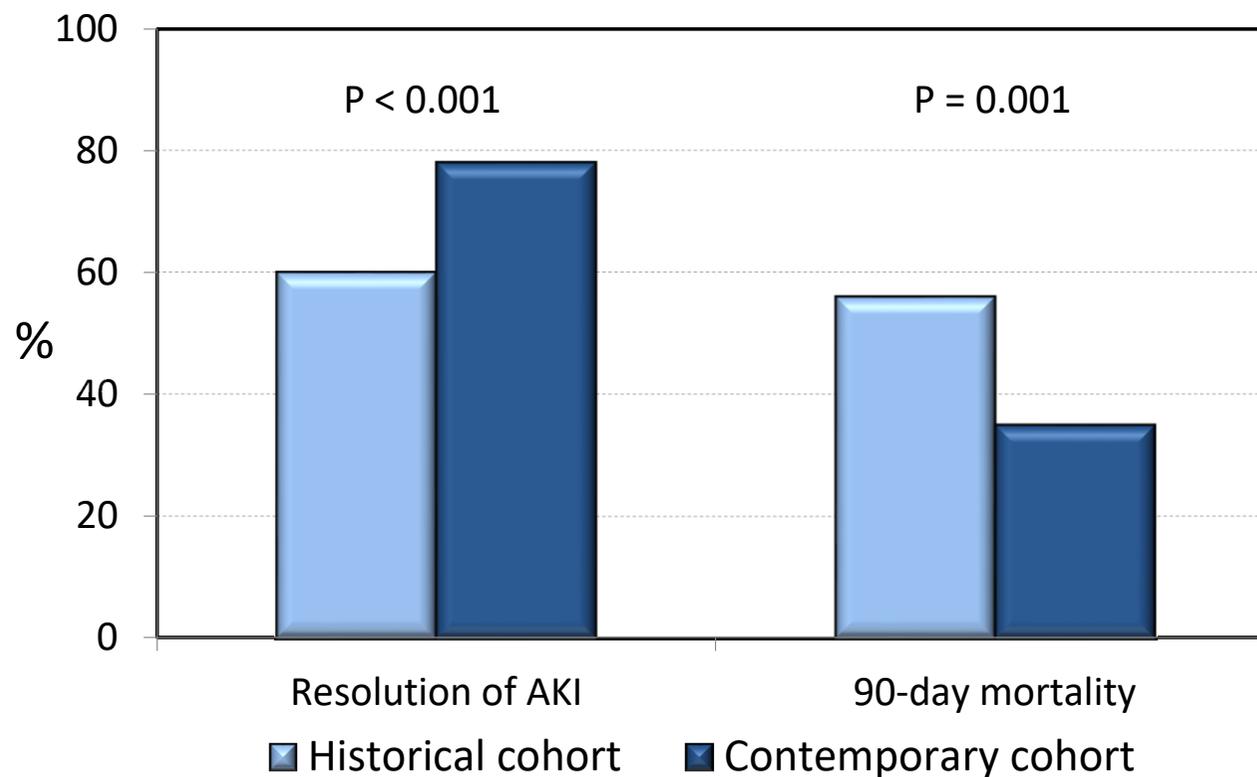
Huelin P, Piano S et al. Clin Gastroenterol Hepatol. 2017 ;15 : 438-445

Algorithm for AKI management in patients with cirrhosis



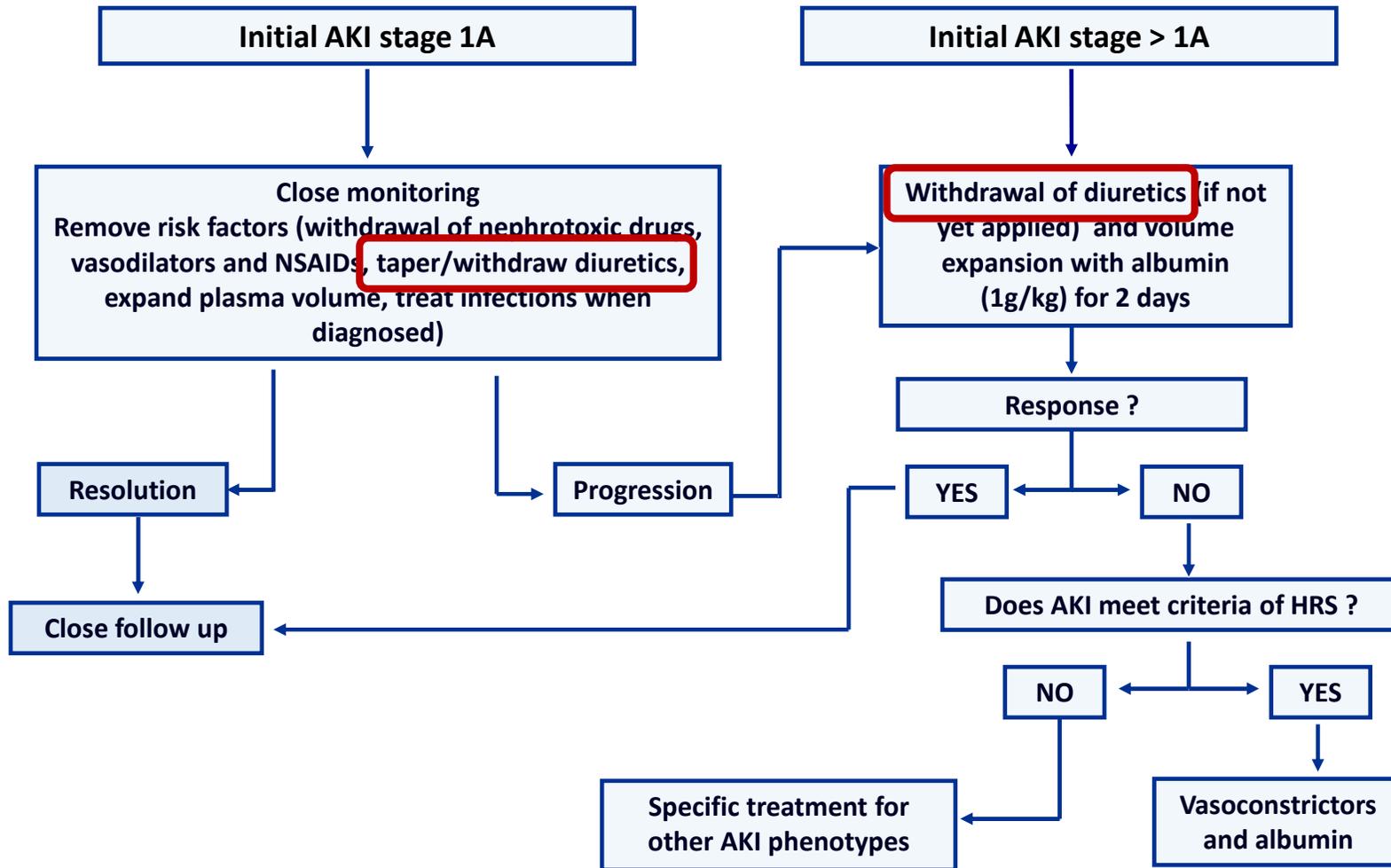


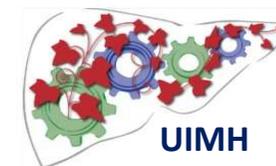
Resolution of AKI and 90-day mortality in patients with AKI managed according to the EASL/ICA algorithm vs an historical cohort



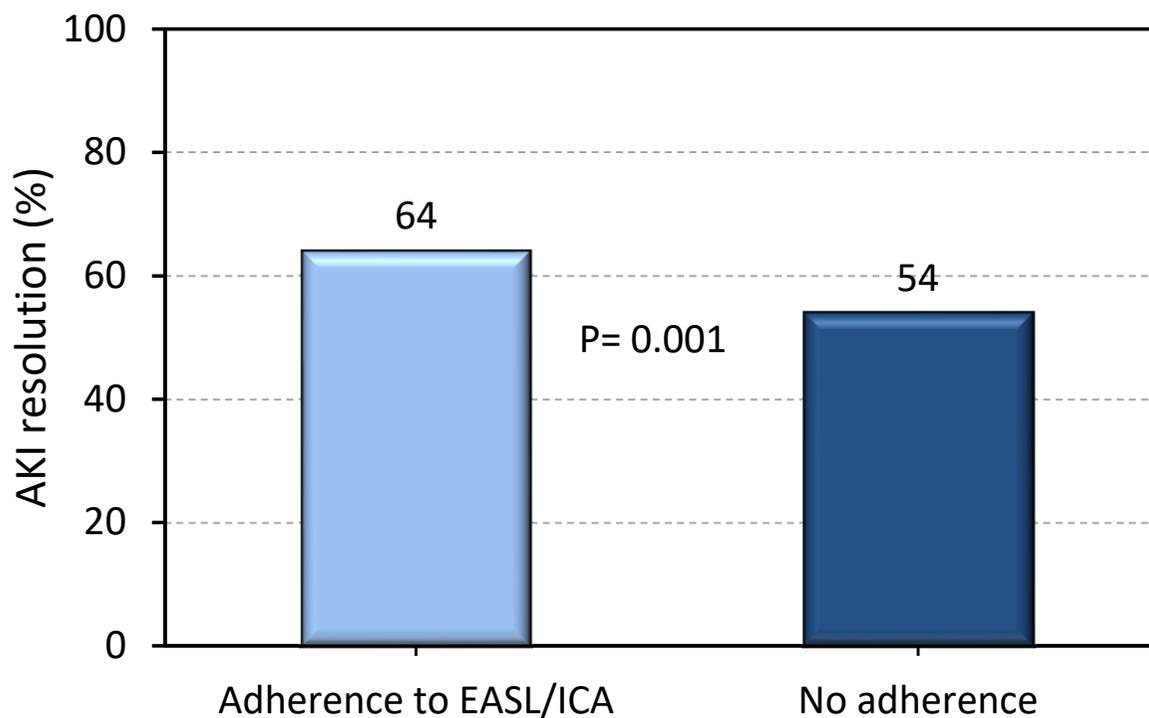
Ma AT, et. al. J Hepatol. 2024 ; 81 : 441-450

Algorithm for AKI management in patients with cirrhosis



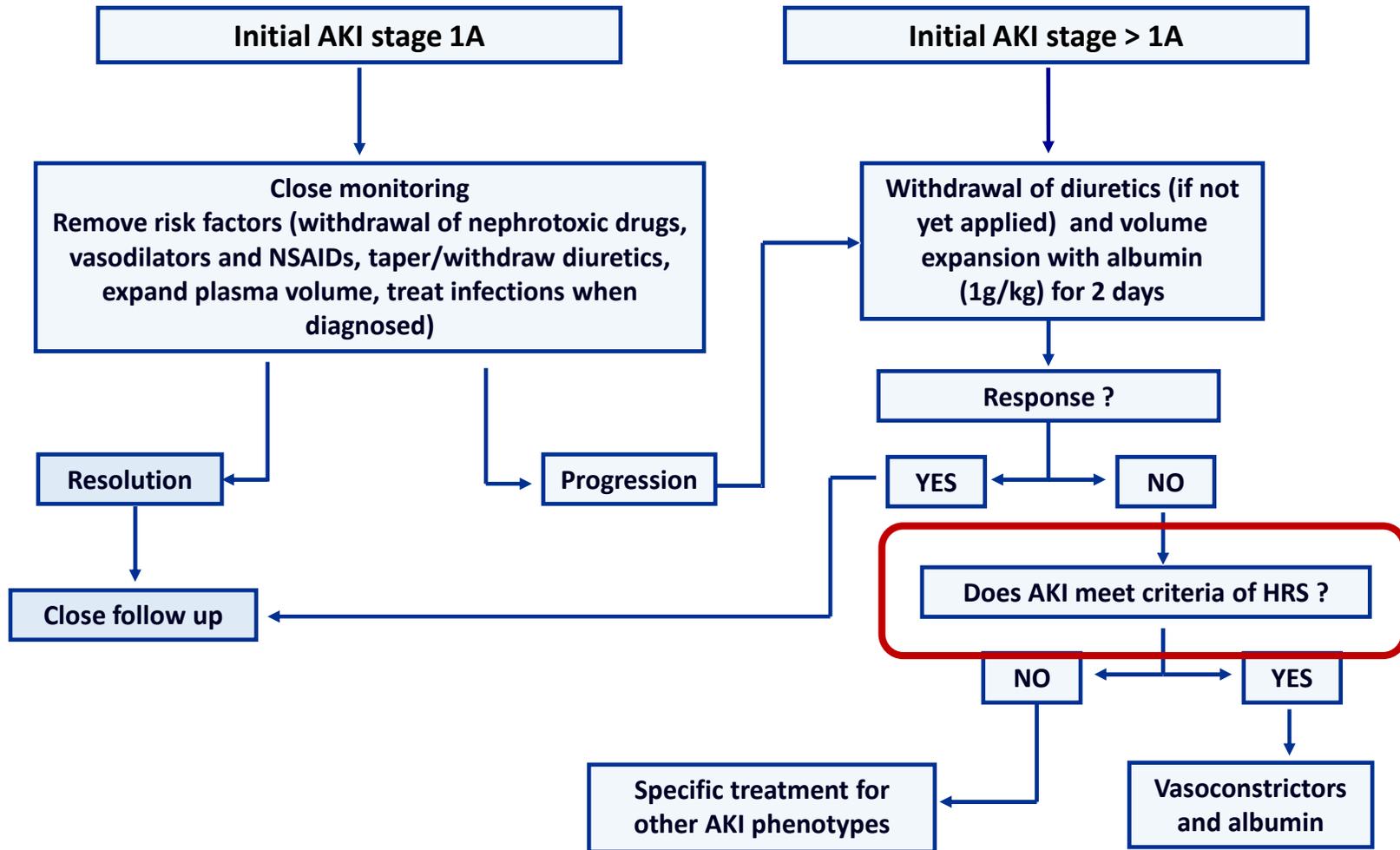


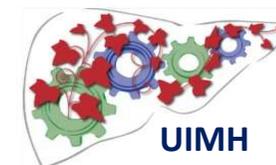
Diuretic tapering/withdrawal according to EASL/ICA recommendation is associated with AKI resolution (n=1,088)



18% risk reduction of AKI non resolution

Algorithm for AKI management in patients with cirrhosis



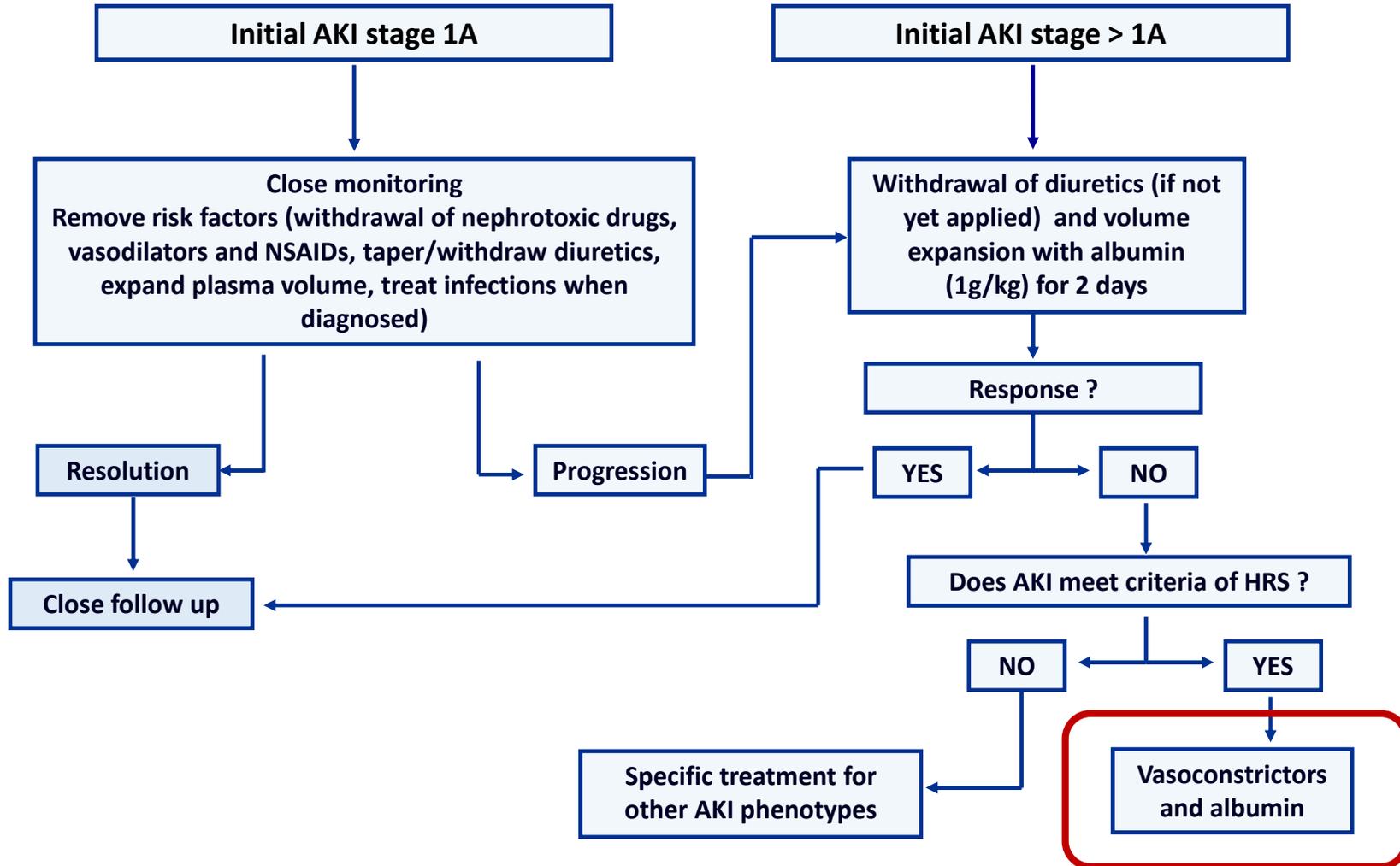


Definition of HRS-AKI

1. Cirrhosis with ascites;
2. Acute kidney injury
3. No sustained improvement of serum creatinine after at least two days of diuretic withdrawal and volume expansion with albumin;
4. Absence of shock
5. No current or recent treatment with nephrotoxic drugs;
6. Absence of parenchymal disease as indicated by proteinuria >500 mg/day, microhematuria (>50 red blood cells per high power field) and/or abnormal renal ultrasonography.

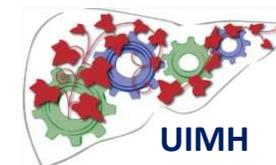
Angeli P et al. J Hepatol. 2015 ; 62 : 968-974

Algorithm for AKI management in patients with cirrhosis





Acute Kidney Injury

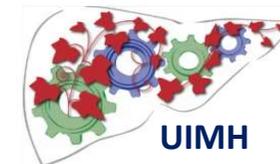


Pharmacological therapy for HRS

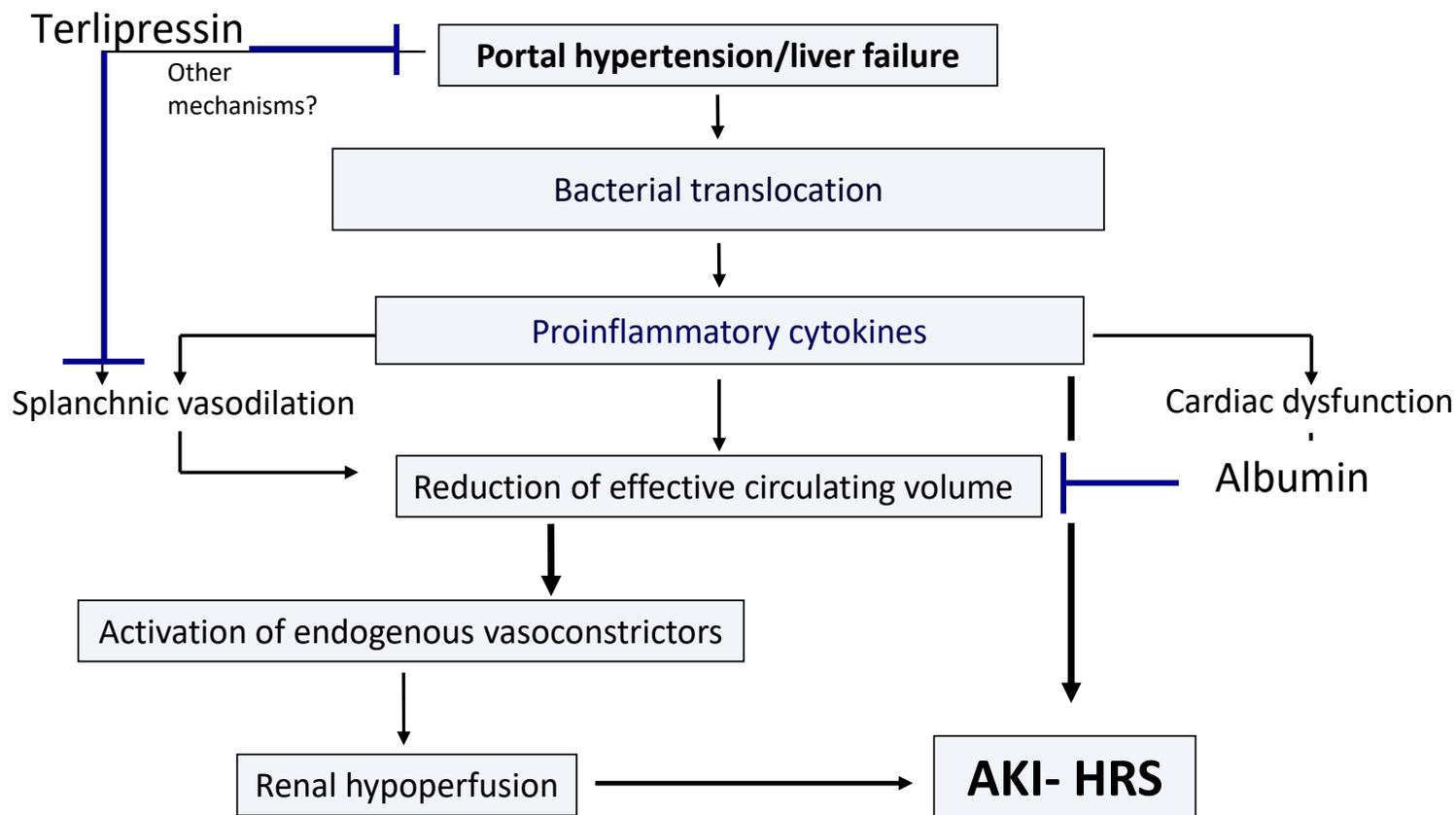
- Albumin (20-40 g/day, intravenously)
- Terlipressin (2-12 mg/day intravenously)

- Albumin (20-40 g/day, intravenously)
- Noradrenalin (0.5-3 mg/hr, intravenously)

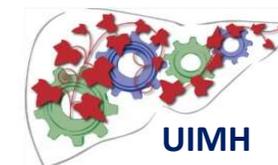
- Albumin (20-40 g/day, intravenously)
- Midodrine (7.5-12.5 mg *t.i.d.*, orally)
- Octreotide (100-200 µg *t.i.d.*, subcutaneously)



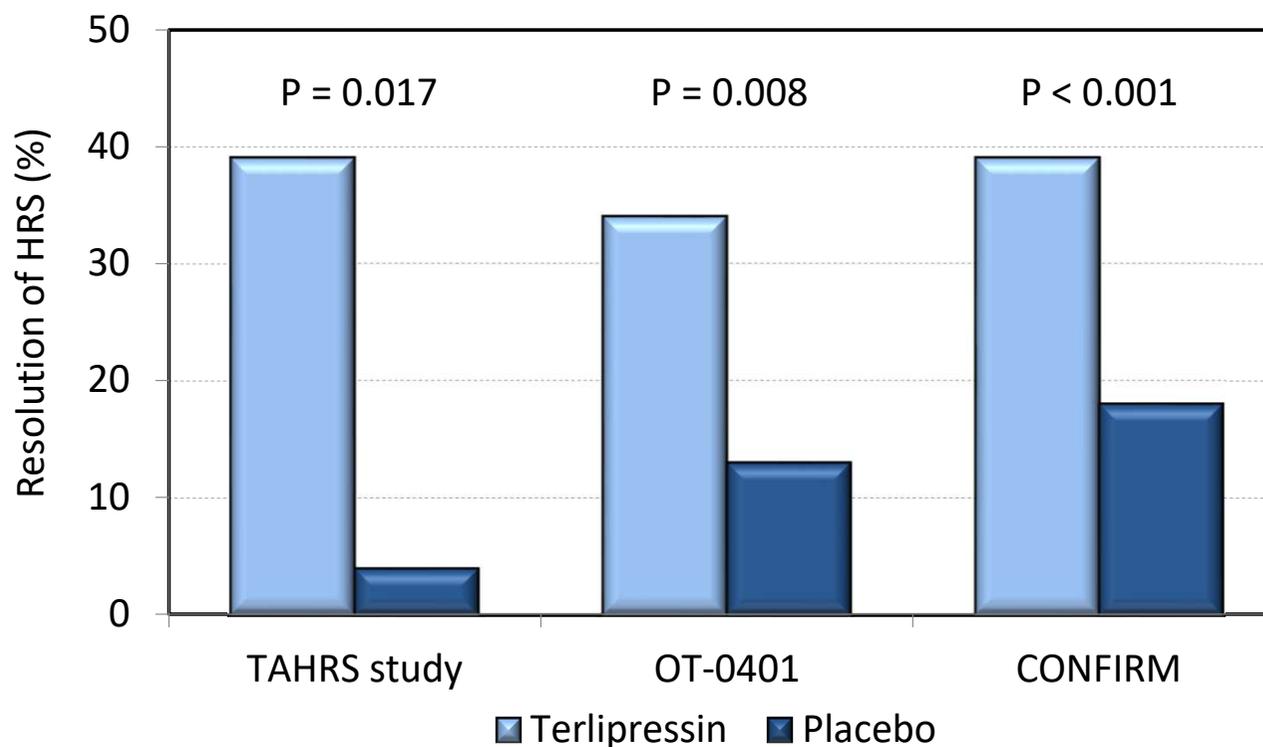
Pathophysiological hypothesis of HRS-AKI and target of treatment



Adapted from Bernardi M et al. *J. Hepatol.* 2015 and Piano S et al. *Semin Liver Dis.* 2018



Terlipressin plus albumin vs placebo plus albumin in the treatment of HRS-AKI



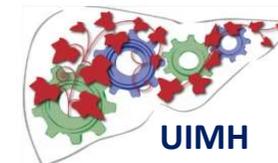
Martin-Llahi M et al. Gastroenterology. 2008 ; 134 : 1352-1359

Sanyal AJ et al Gastroenterology. 2008 ; 134 : 1360-1368

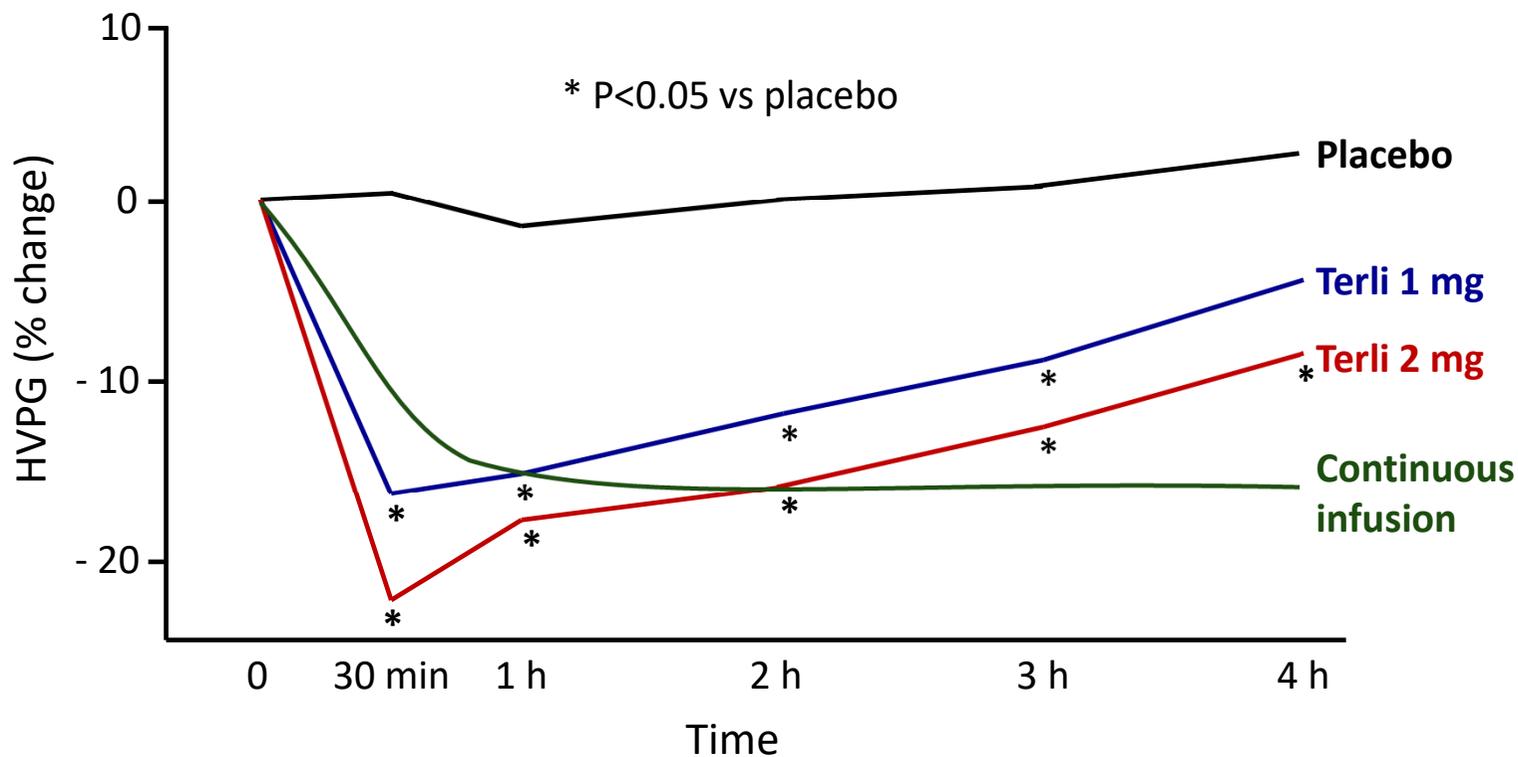
Wong F et al. NEJM. 2021 ; 384 : 818-828



Acute Kidney Injury



Effects of terlipressin on hepatic venous pressure gradient



Modified from:

Escorsell A et al *J Hepatol* 1997 ; 26 : 621-627

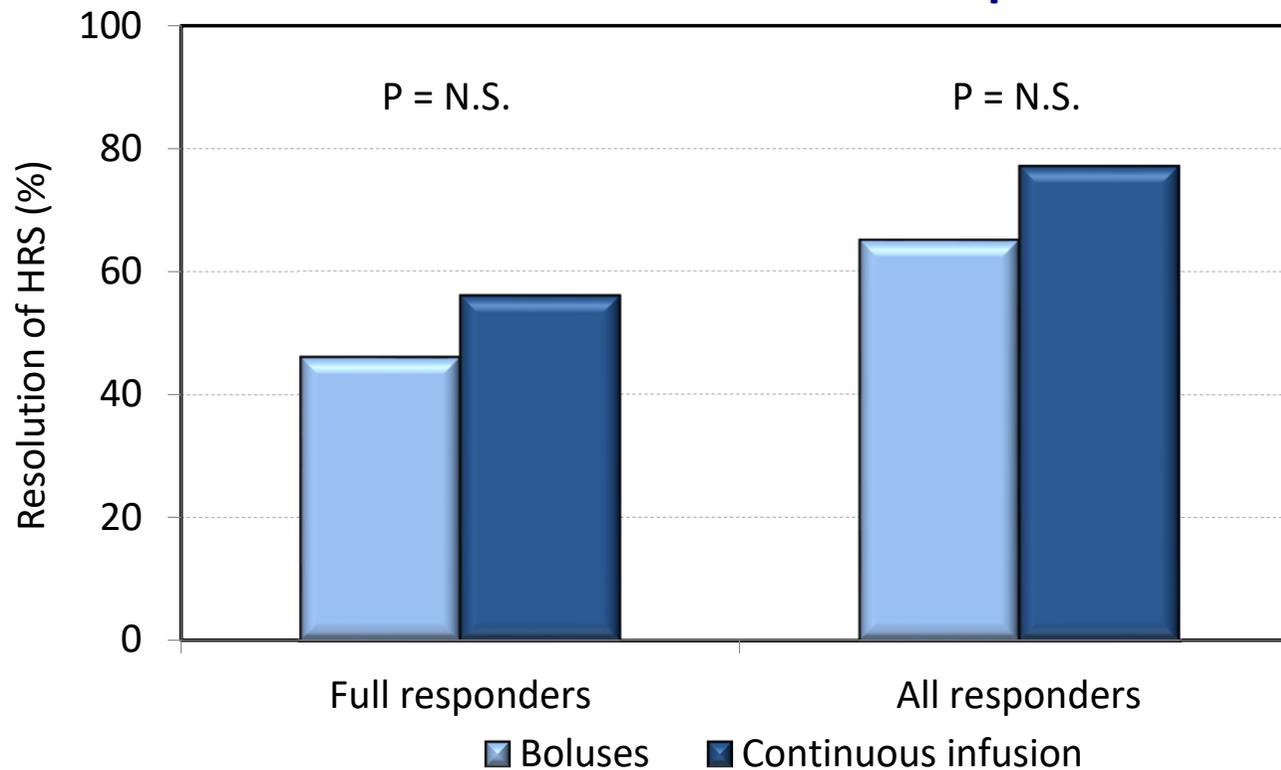
Ding C et al *J Gastroenterol Hepatol.* 2013 ; 28 : 1242-1246



Acute Kidney Injury



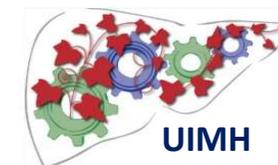
Rate of response in patients with type 1 HRS according to administration route of terlipressin



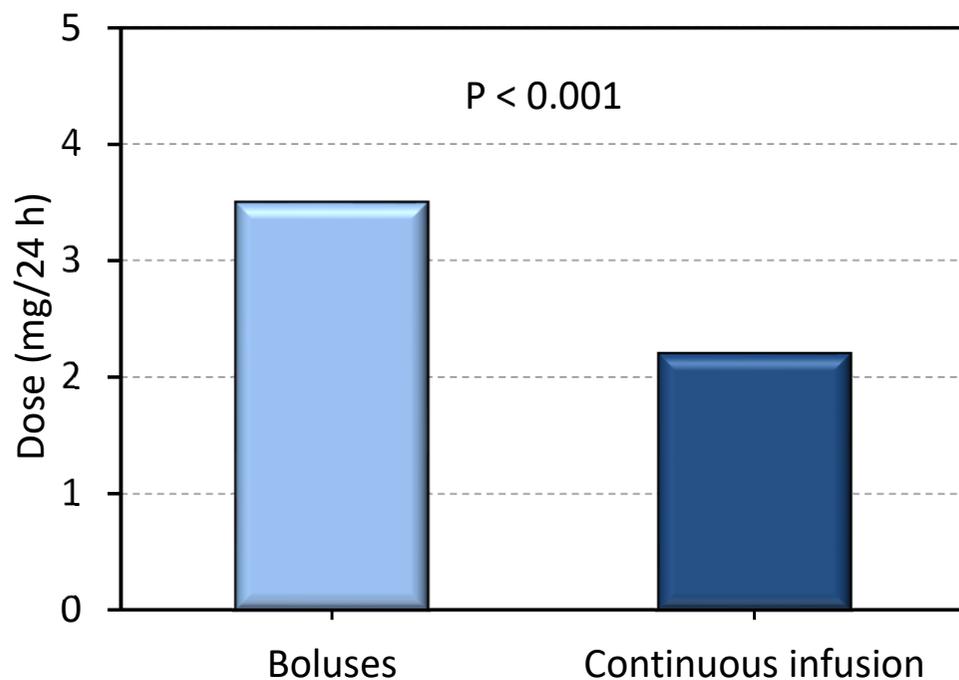
Cavallin M, Piano S et. al. Hepatology. 2016 ; 63 : 983-992



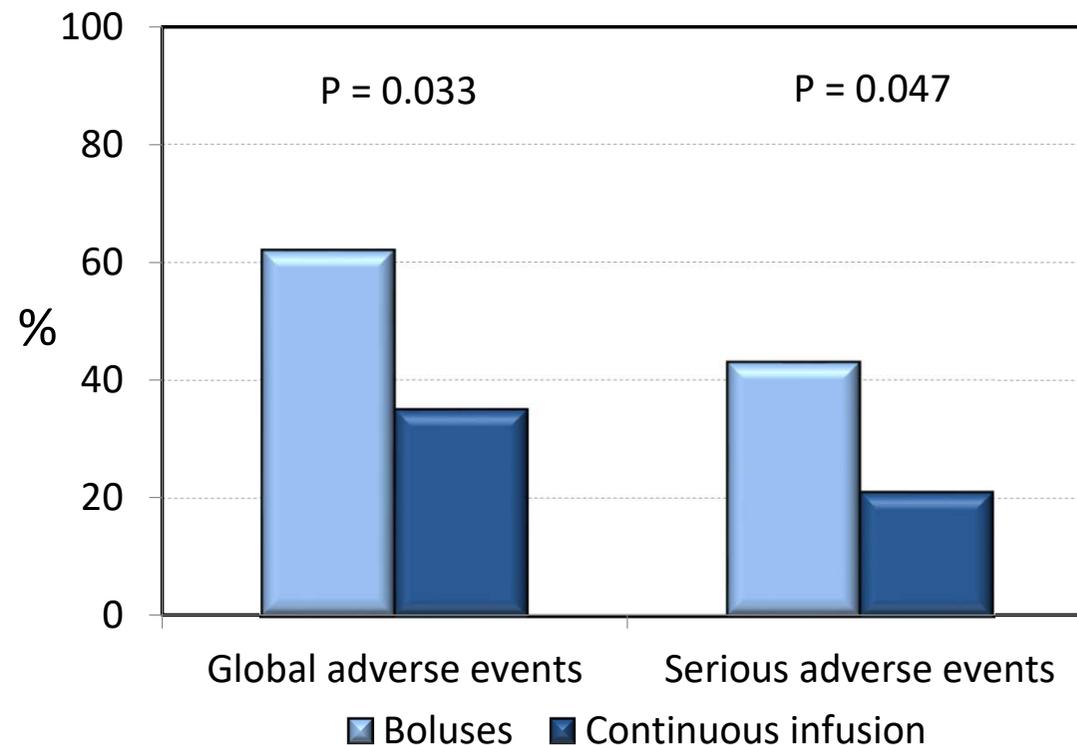
Acute Kidney Injury



Mean effective daily dose of terlipressin according to administration route



Incidence of side effects according to the administration route of terlipressin



Cavallin M, Piano S et. al. Hepatology. 2016 ; 63 : 983-992



Adverse events with terlipressin use in HRS-AKI

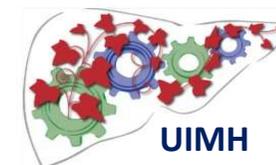
- Acrocyanosis/Peripheral ischemia
- Arrhythmias, myocardial ischemia
- Diarrhea, abdominal pain
- **Respiratory failure**/pulmonary edema

Martin-Llahi M et al. Gastroenterology. 2008 ; 134 : 1352-1359

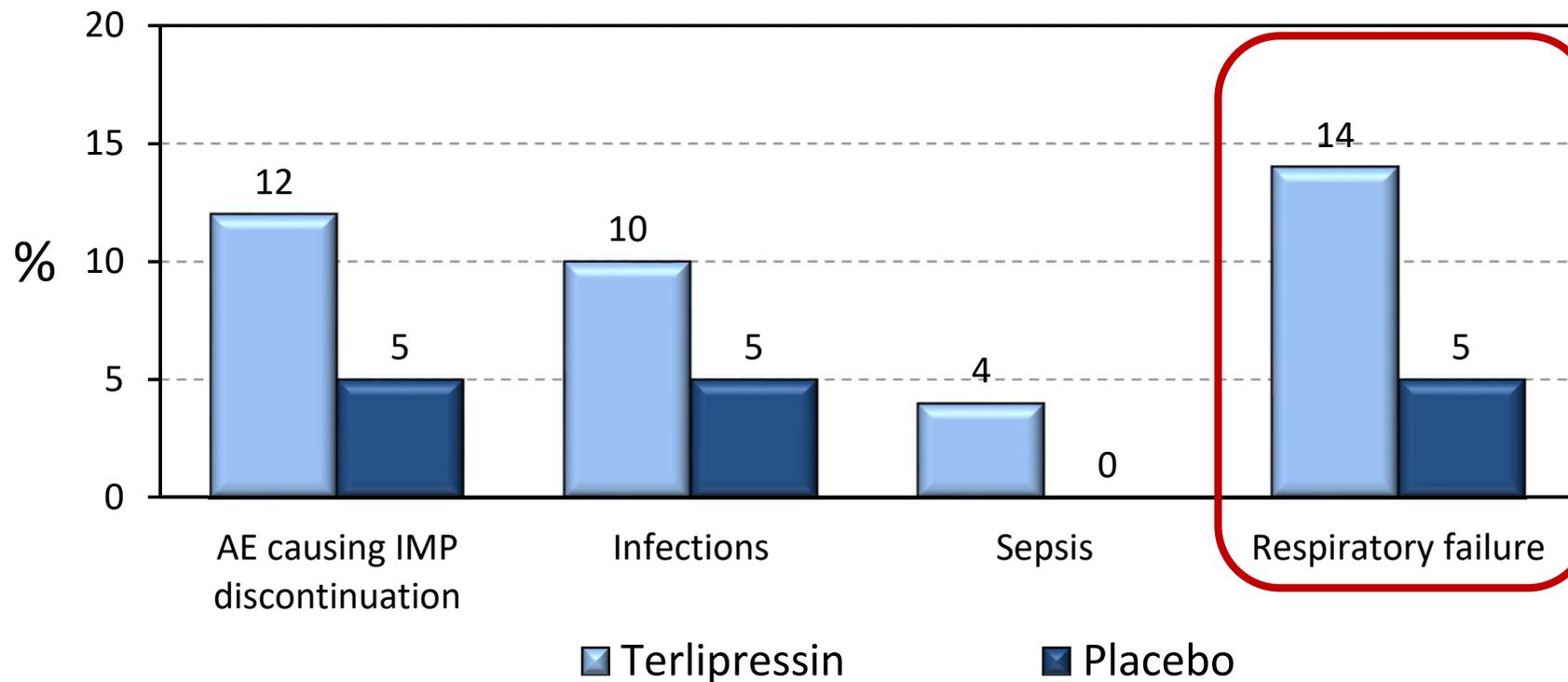
Sanyal AJ et al Gastroenterology. 2008 ; 134 : 1360-1368

Cavallin M et. al. Hepatology. 2016 ; 63 : 983-992

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



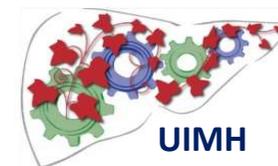
Terlipressin vs placebo in the treatment of HRS Serious adverse events in the CONFIRM trial



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



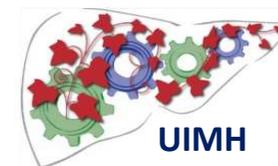
Acute Kidney Injury



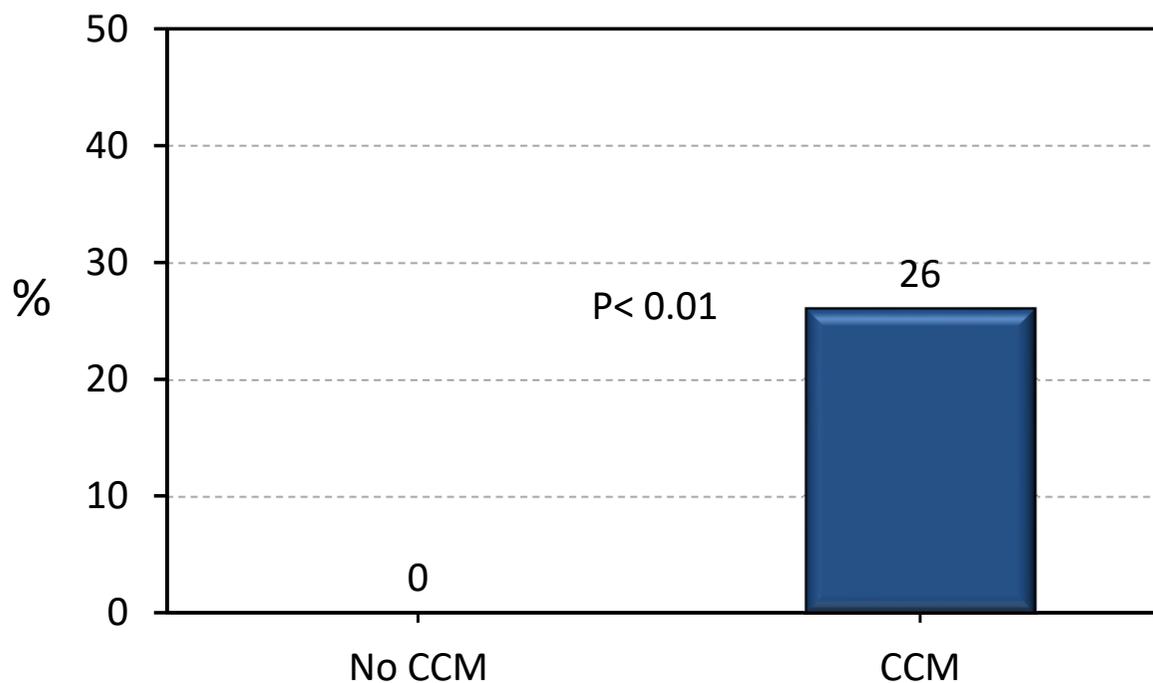
Mechanisms of terlipressin and pulmonary edema

**In CONFIRM trial albumin dose
prior terlipressin was higher than
maximum dose recommended**

Albumin concentration normalized!



Pulmonary edema with respiratory failure in patients treated with terlipressin and albumin according to the presence of cirrhotic cardiomyopathy



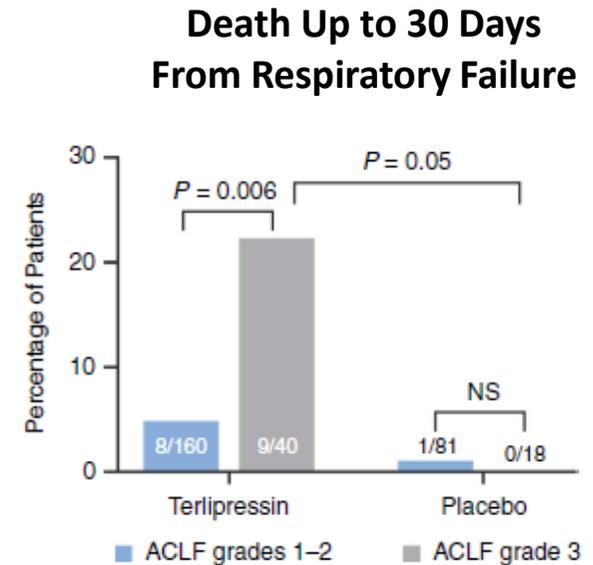
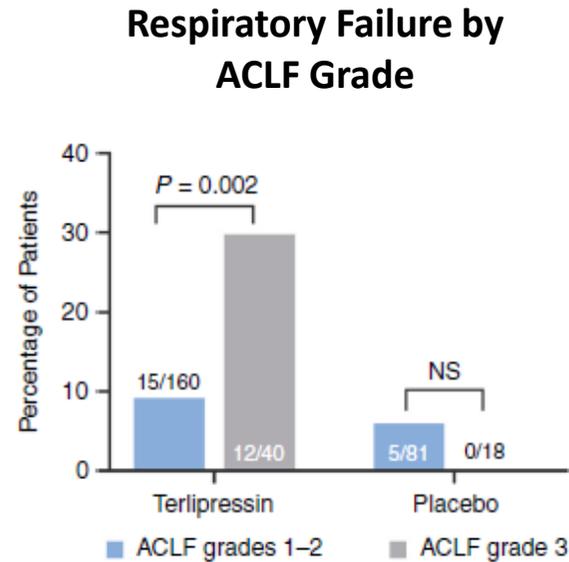
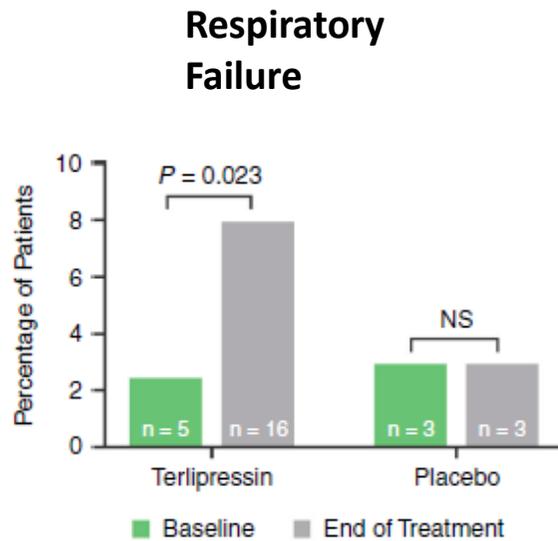
Premkumar M et al. Hepatology. 2024 ; 79 : 1048–1064



Hepatorenal syndrome



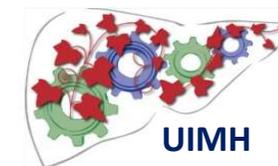
Risk of respiratory failure according to ACLF grade



Wong F et al. Aliment Pharmacol Ther. 2022 ; 56 : 1284-1293.

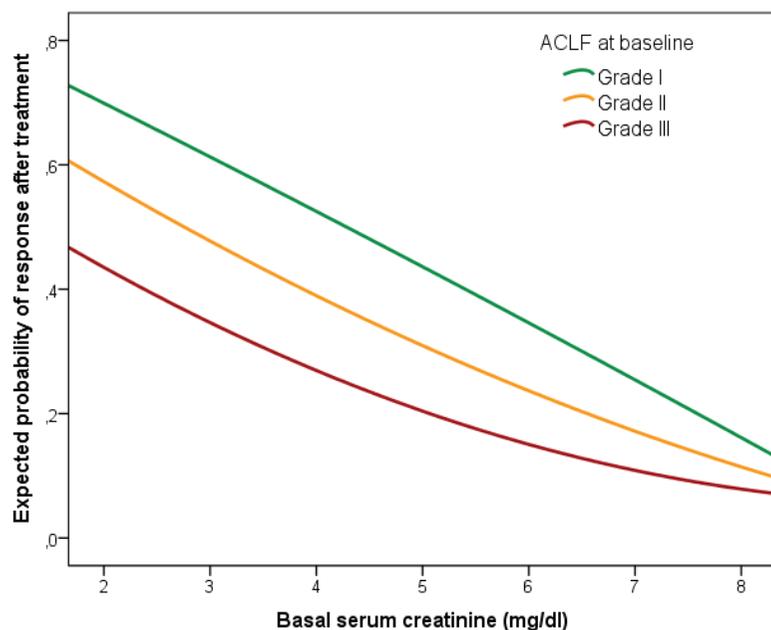


Hepatorenal syndrome

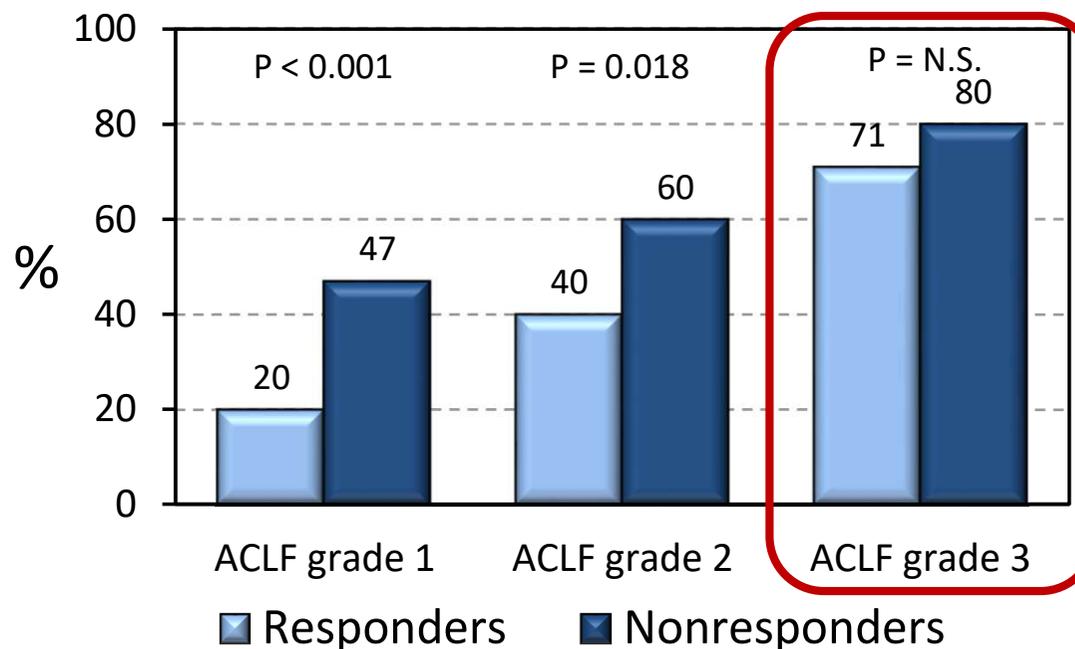


Patients with HRS-AKI and ACLF grade 3 are less likely to respond to terlipressin albumin and have no survival benefit from response

Probability of reversal of type-1 HRS



90-day mortality according to response

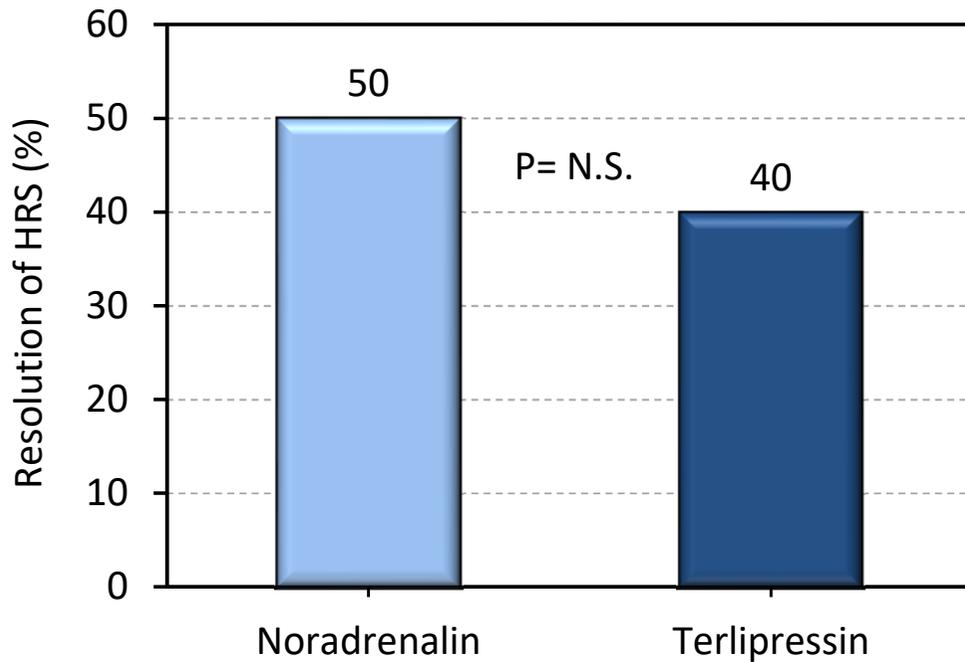


Piano S et al. Clin Gastroenterol Hepatol. 2018 ; 16 : 1792-1800



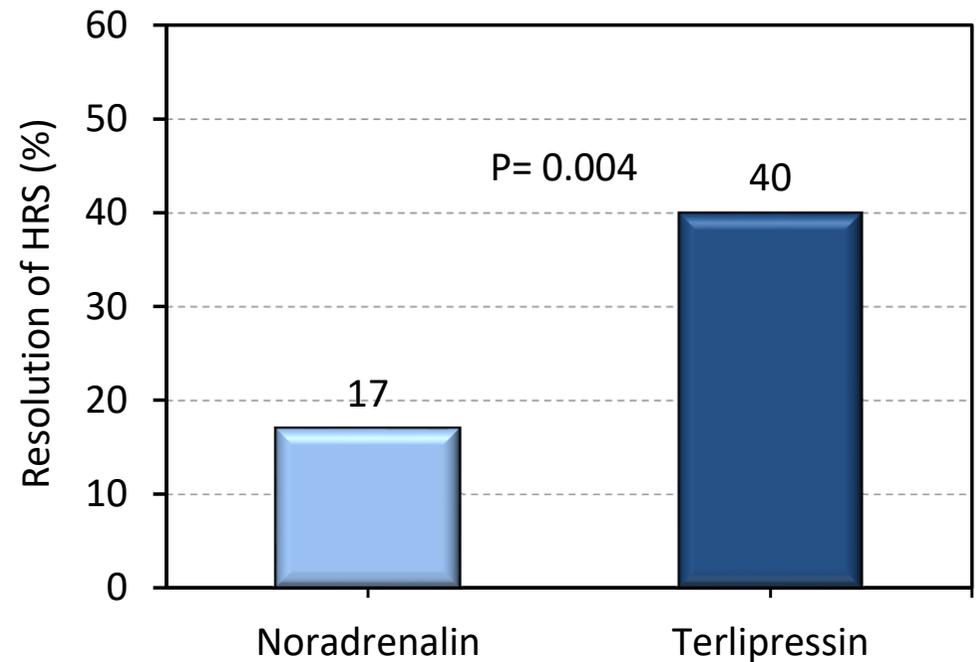
Noradrenalin vs terlipressin in the treatment of HRS

Type 1 HRS

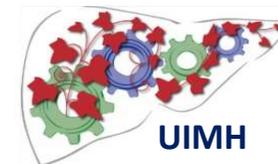


Sharma P et al. *Am. J. Gastroenterol.* 2008 ; 103 : 1689–1697.

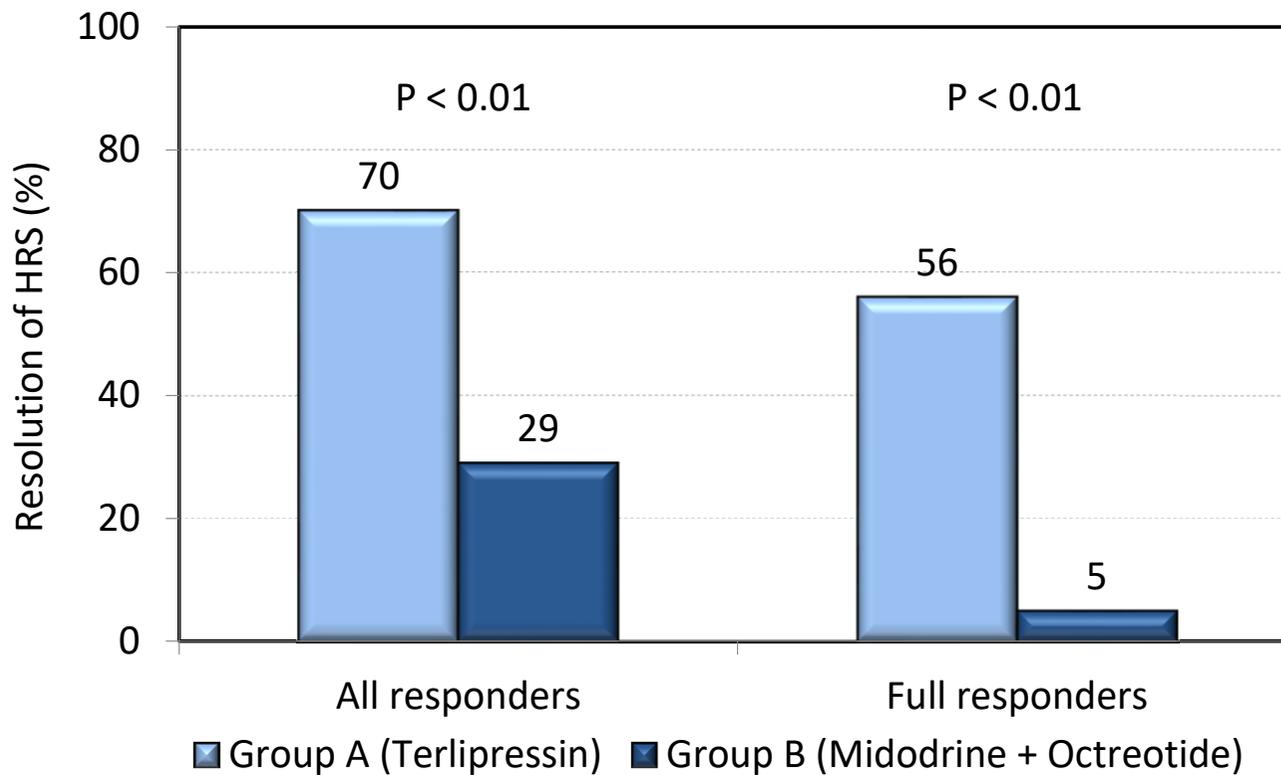
HRS-AKI and ACLF (APASL criteria)



Arora V et al. *Hepatology.* 2020 ; 71 : 600-610.



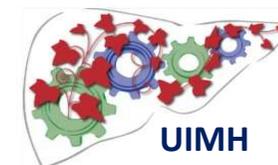
Terlipressin vs midodrine + octreotide in the treatment of HRS



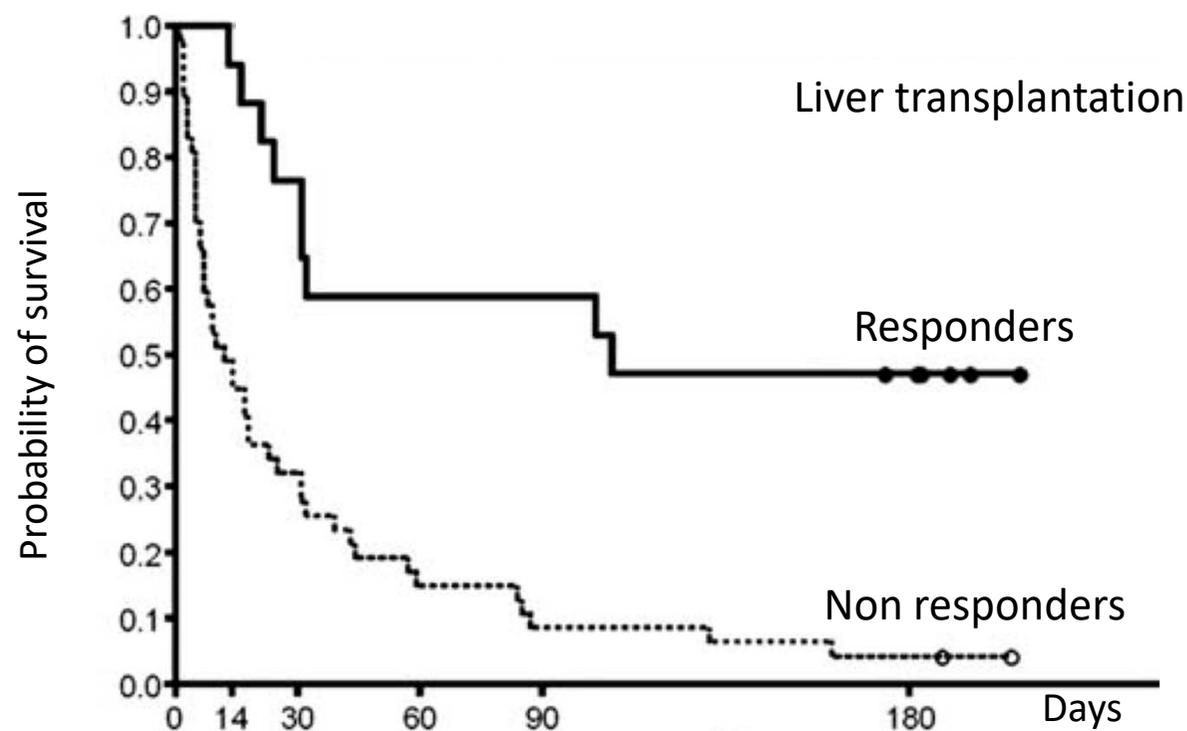
Cavallin M, et. al. Hepatology. 2015 ; 62 : 567-574



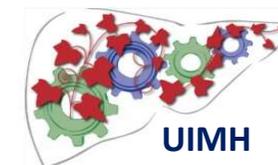
Hepatorenal syndrome



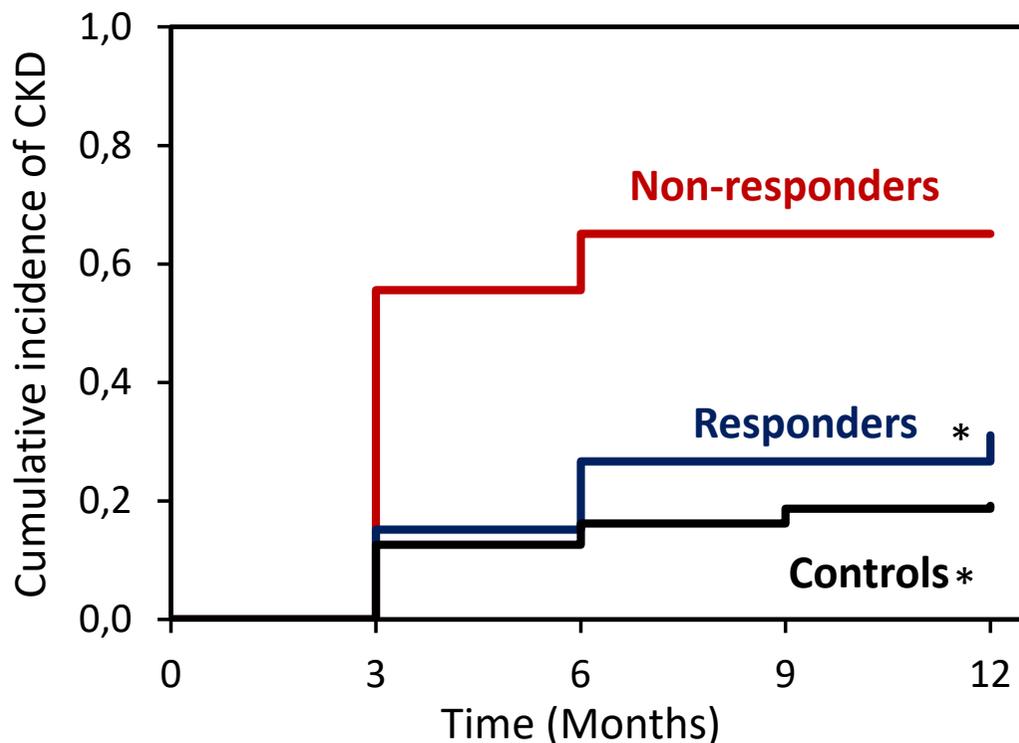
Probability of survival according to response to treatment with terlipressin and albumin



Boyer TD et al. *Liver Transpl.* 2011 ; 17 : 1328-1332



Incidence of chronic kidney disease (CKD) at 12 months after LT according to response to treatment with terlipressin and albumin



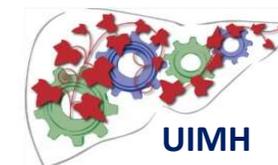
Variable	sHR	95% CI	p-value
Age	1.04	1.01 – 1.09	0.024
Diabetes	1.64	1.00 – 2.67	0.048
AKI post transplant	1.81	1.14 – 2.88	0.012
MELD at transplant	1.03	1.00 – 1.06	0.028
Response to terlipressin and albumin[#]			
Responders	1.69	0.89 – 3.21	0.110
Non-responders	2.57	1.37 – 4.82	0.003

[#], Patients without HRS were considered as reference group

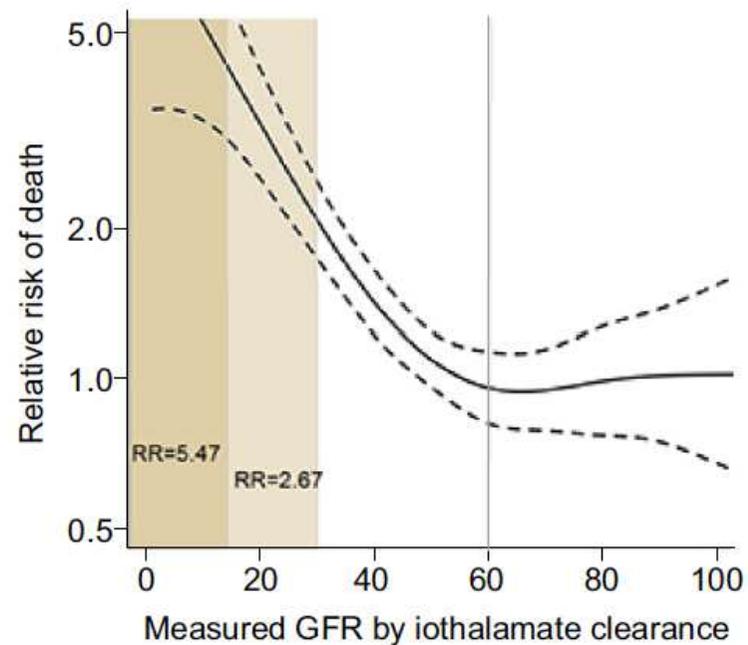
*, P < 0.025 vs non-responders



Hepatorenal syndrome



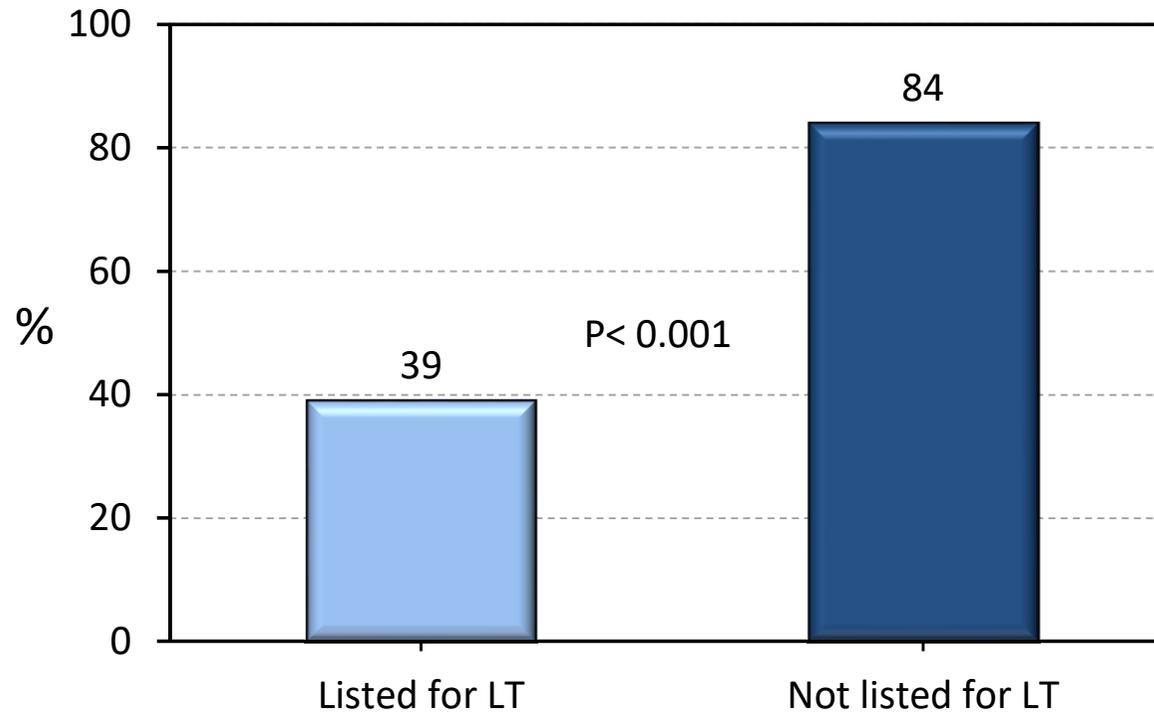
Consequences associated with CKD in LT: risk of mortality according to measured GFR



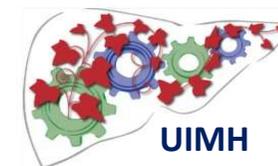
Allen AM et al. *J. Hepatol.* 2014 ; 61 : 286-292



Mortality rate at 6-months in patients with HRS treated with renal replacement therapy listed or not for liver transplantation (LT)



Allegretti AS et al. Clin J Am Soc Nephrol. 2018 ; 13: 16–25



Agenda

- Diagnosis and treatment of AKI
- **Controversial issues and future strategies**



Definition of HRS-AKI

1. Cirrhosis with ascites;
2. Acute kidney injury
3. No sustained improvement of serum creatinine after at least two days of diuretic withdrawal and volume expansion with albumin;
4. Absence of shock
5. No current or recent treatment with nephrotoxic drugs;
6. Absence of parenchymal disease as indicated by proteinuria >500 mg/day, microhematuria (>50 red blood cells per high power field) and/or abnormal renal ultrasonography.

Angeli P et al. J Hepatol. 2015 ; 62 : 968-974



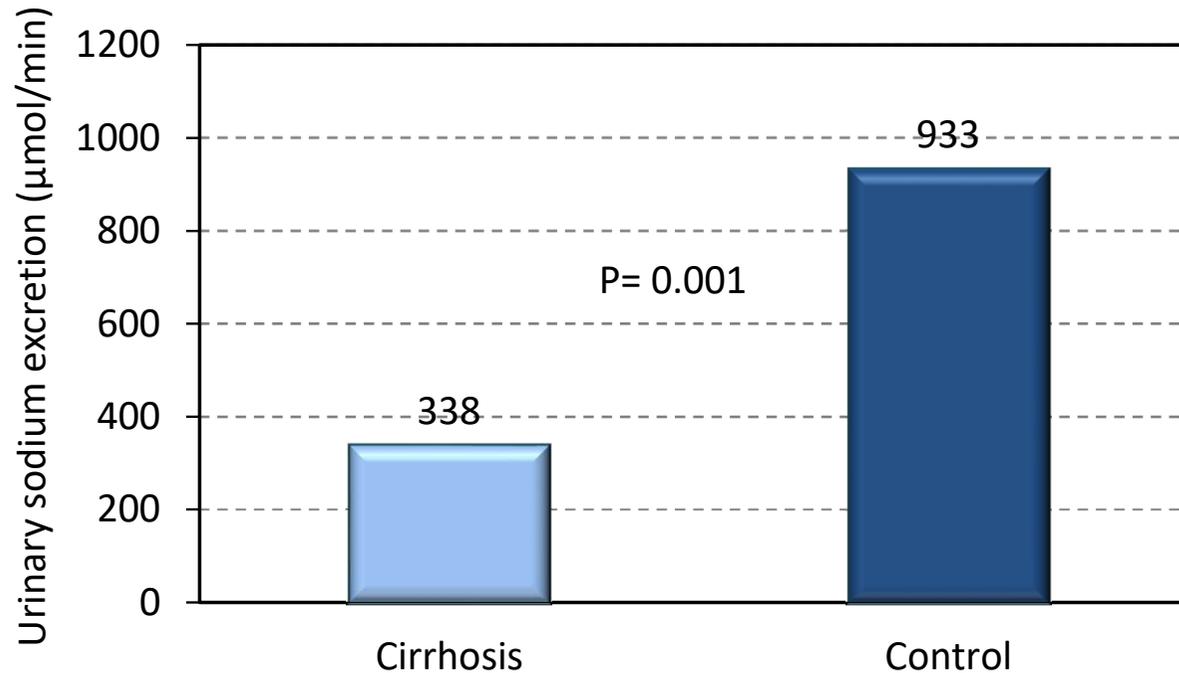
Updated diagnostic criteria of HRS-AKI

1. Cirrhosis with ascites;
2. Acute kidney injury;
3. Absence of improvement in SCr and/or UO within 24 h following adequate volume resuscitation*
4. Absence of strong evidence for an alternative explanation as the primary cause of AKI

*, “We recommend against systematic administration of albumin for 48 h as a requisite for the diagnosis of HRS-AKI”



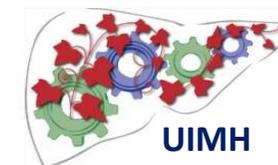
Impaired natriuresis in patients with compensated cirrhosis after saline loading



Caregaro L et al. *Eur J Clin Invest.* 1985 ; 15 : 360-364.



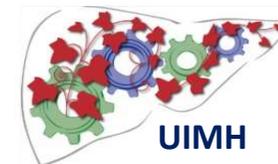
Acute Kidney Injury



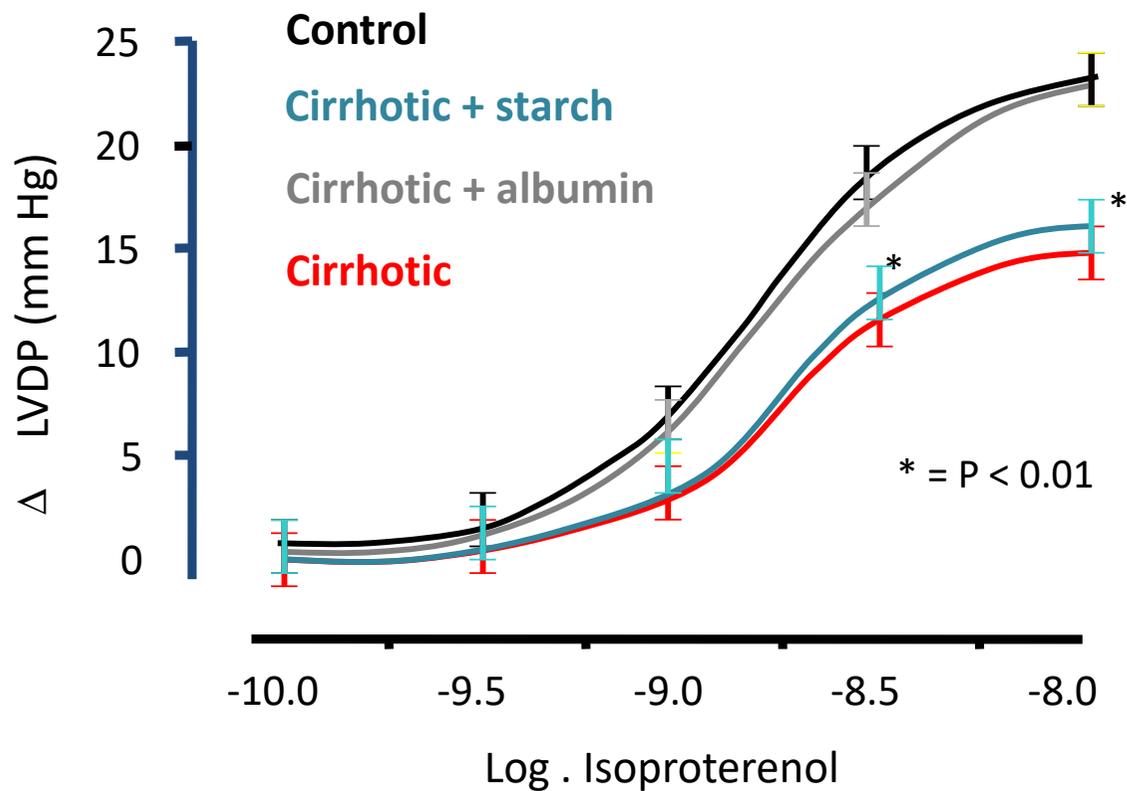
Effects of albumin or hydroxyethyl starch on systemic hemodynamic in patients with SBP

	Group 1 (Albumin)			Group 2 (Hydroxyethyl Starch)		
	At Diagnosis	At Resolution	P	At Diagnosis	At Resolution	P
Systemic hemodynamics						
Mean arterial pressure (mmHg)	76 ± 9	85 ± 13	.01	80 ± 15	81 ± 8	.36
Cardiac index (L/min/m ²)	4.9 ± 0.7	4.6 ± 0.9	.21	4.8 ± 1.0	4.9 ± 1.4	.44
Systemic vascular resistance (dyn-sec/cm ⁵)	668 ± 134	803 ± 197	.03	777 ± 239	778 ± 290	.96
Systolic volume (mL)	97 ± 20	104 ± 17	.05	83 ± 15	92 ± 23	.12
Heart rate (beats/min)	91 ± 14	80 ± 13	.01	90 ± 10	84 ± 9	.01
Stroke work index (g·m/m ²)	51 ± 17	59 ± 17	.01	48 ± 13	53 ± 13	.24
Right atrial pressure (mmHg)	8 ± 2	9 ± 2	.03	7 ± 3	7 ± 3	1.0
Pulmonary artery pressure (mmHg)	17 ± 4	21 ± 5	.01	18 ± 6	18 ± 7	.88
Pulmonary capillary pressure (mmHg)	11 ± 3	14 ± 4	.03	10 ± 4	11 ± 5	.51
Plasma hormonal systems						
Plasma renin activity (ng·mL/h)	5.7 ± 4.7	3.1 ± 3.4	.04	8.5 ± 7.3	16.8 ± 24.6	.65
Atrial natriuretic factor (fmol/mL)	55 ± 21	85 ± 37	.05	45 ± 32	28 ± 18†	.51
Splanchnic and renal hemodynamics						
Wedge hepatic venous pressure (mmHg)	30.6 ± 2.9	31.9 ± 4.0	.31	30.6 ± 5.6	31.6 ± 5.2	.29
Free hepatic venous pressure (mmHg)	12.8 ± 3.0	13.4 ± 2.1	.47	10.5 ± 3.5	11.0 ± 2.7	.57
HVPG (mmHg)	17.8 ± 2.5	18.5 ± 3.1	.31	20.1 ± 4.3	20.6 ± 4.6	.59
Hepatic blood flow (L·min ⁻¹)*	0.8 ± 0.2	1.0 ± 0.3	.14	0.9 ± 0.4	1.2 ± 0.5	.71
Renal resistive index	0.80 ± 0.04	0.78 ± 0.04	.11	0.80 ± 0.02	0.81 ± 0.04	.44

Fernandez J, et al. *Hepatology* 2005 ; 42 : 627-34.



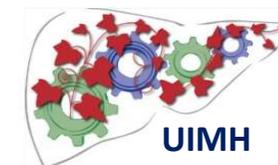
Impairment of cardiac contractility in cirrhotic rats



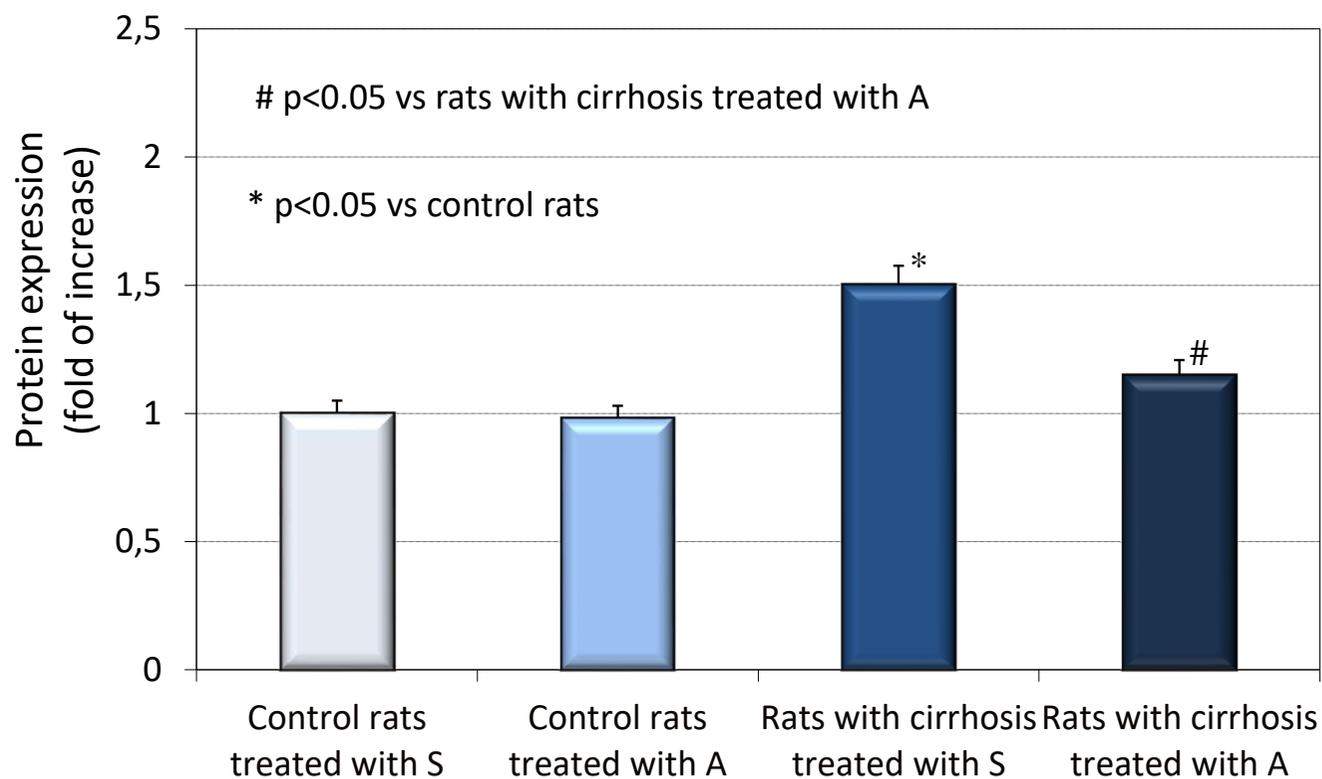
A. Bortoluzzi et al. *Hepatology* 2013 ; 57 : 266-276



Acute Kidney Injury



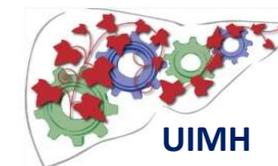
Effects of albumin on TNF-alpha protein expression in the cardiac tissue according to treatment with saline (S) or albumin (A)



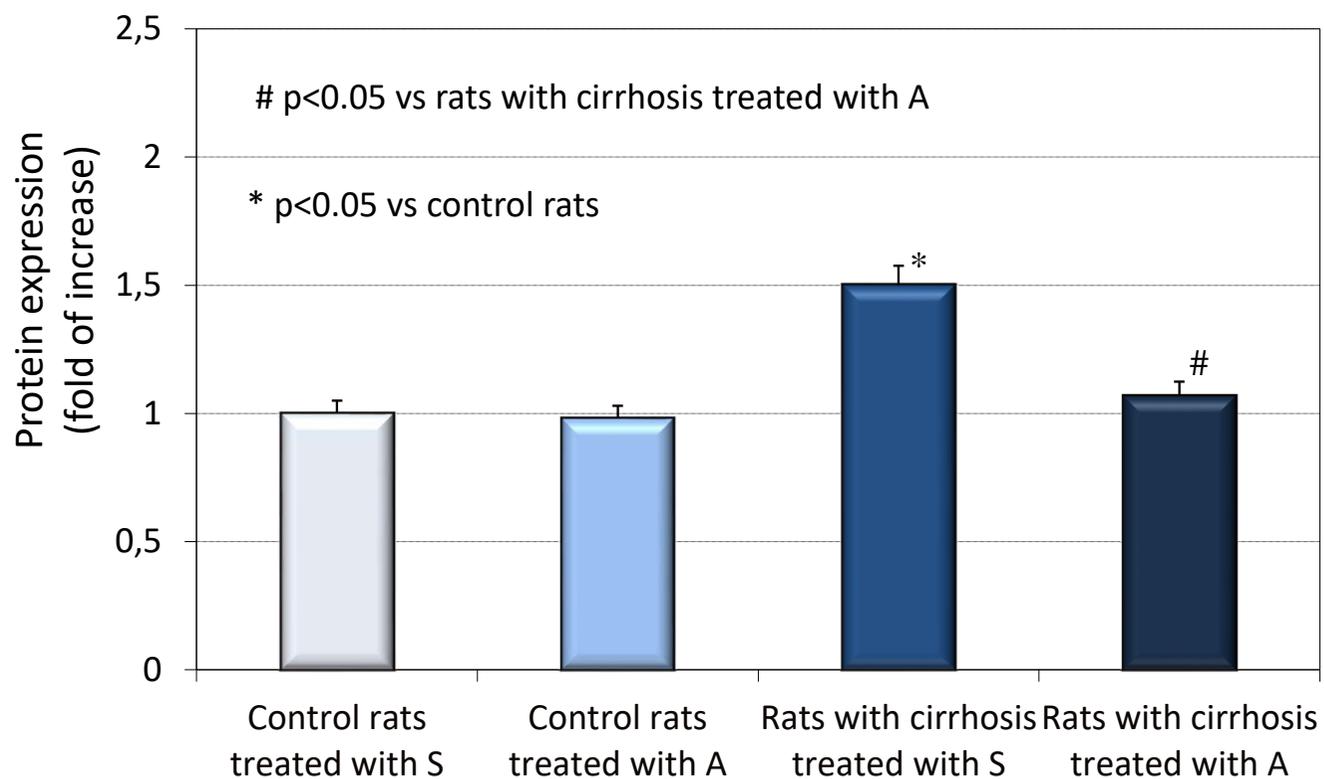
Bortoluzzi A et al. Hepatology 2013 ; 57 : 266-276



Acute Kidney Injury



Effects of albumin on iNOs protein expression in the cardiac tissue according to treatment with saline (S) or albumin (A)



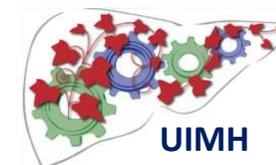
Bortoluzzi A et al. Hepatology 2013 ; 57 : 266-276



Updated diagnostic criteria of HRS-AKI

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2. Acute kidney injury;
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4. Absence of strong evidence for an alternative explanation as the primary cause of AKI

*, “We recommend against systematic administration of albumin for 48 h as a requisite for the diagnosis of HRS-AKI”



Shortening the albumin challenge from 48 to 24 hours may lead to overdiagnosis of HRS-AKI

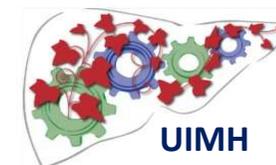
- Population: 139 patients with AKI \geq 1B
 - Response to albumin challenge at 48 hours: 47 patients (34%)
 - Response to albumin challenge within 24 hours: 28 (61% or responders)
- Population: 127 patients with AKI \geq 1B
 - Response to albumin challenge at 48 hours: 58 patients (46%)
 - Response to albumin challenge within 24 hours: 36 (62% or responders)

Ma AT et al. J Hepatol. 2024 (in press)

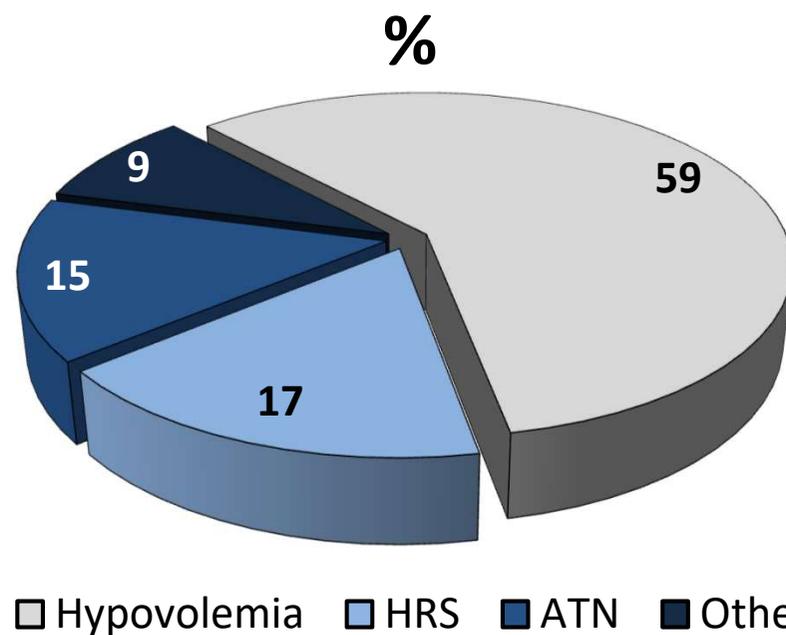
Schleicher EA et al. J Hepatol. 2025 (in press)



Acute Kidney Injury



Phenotype of AKI in patients with cirrhosis (n=1,456)

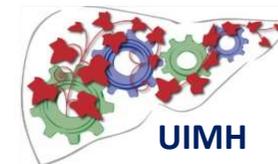


Patidar KR. [...] Piano S. *Lancet Gastroenterol Hepatol* 2025 (in press)

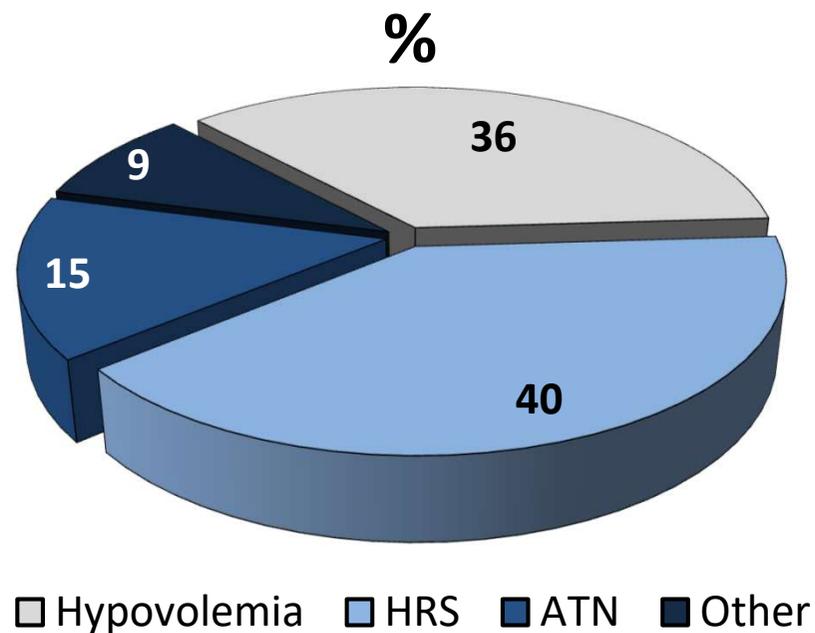




Acute Kidney Injury

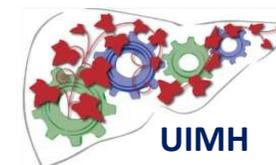


Simulation of the impact of shortening albumin challenge on AKI phenotype





Acute Kidney Injury



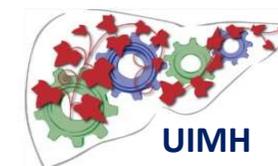
Expert Opinion

JOURNAL
OF HEPATOLOGY

Albumin infusion in hepatorenal syndrome-acute kidney injury: New evidence challenges recent consensus

Paolo Angeli^{1,*}, Christian Labenz², Salvatore Piano³, Adrià Juanola⁴, Aleksander Krag⁵, Paolo Caraceni⁶, Jonel Trebicka⁷, Rakhi Maiwall⁸, Virendra Singh⁹, Elisa Pose⁴, Carmine Gambino³, Sebastian Marciano¹⁰, Peter R. Galle², Shiv K. Sarin⁷, Pere Ginès^{4,‡}, Patrick S. Kamath^{11,‡}

“[...]until further data become available, we recommend following the EASL algorithm, particularly regarding the use of 48-hour albumin infusion and established diagnostic criteria.”

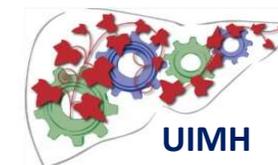


Association between cumulative fluid volume administered in the first 48 hours and the risk of 28-day mortality and respiratory failure in patients with cirrhosis and AKI.

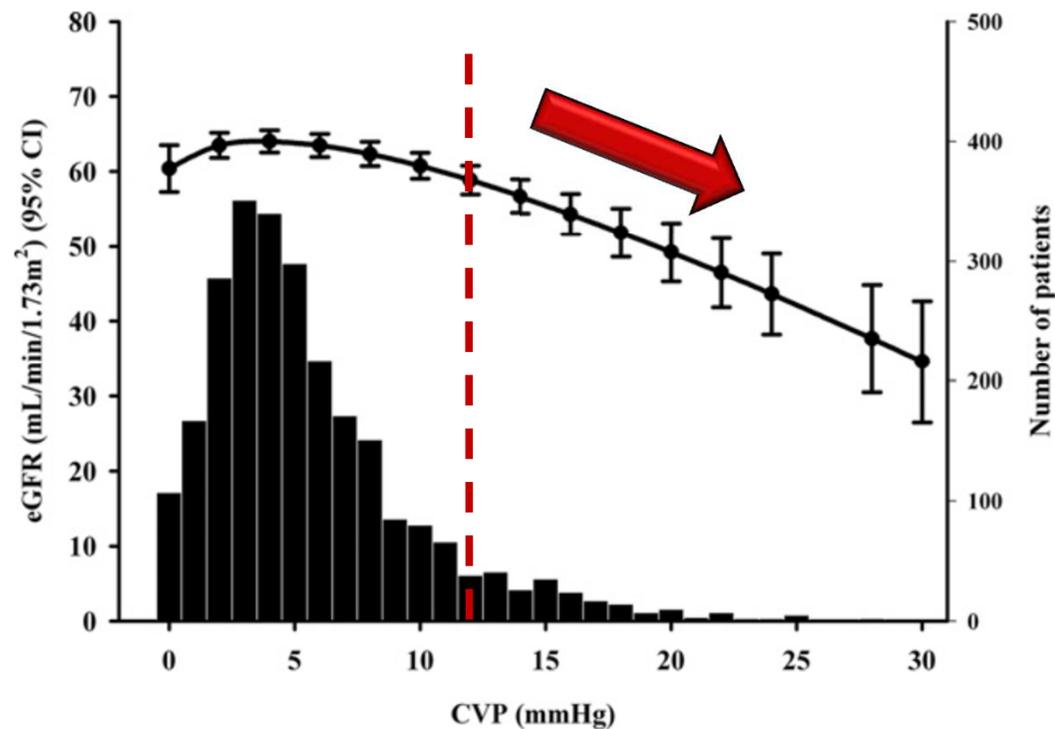
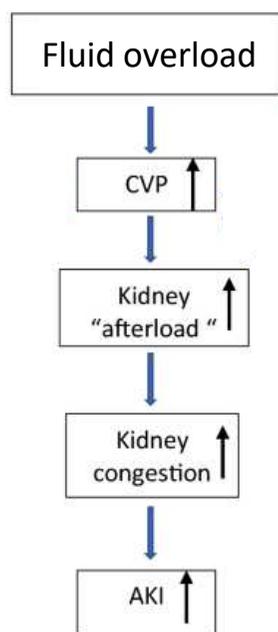
28-day mortality		
Population	sHR (95% CI)	p value
Overall	1.02 (1.00–1.03)	0.037
AKI responders	1.01 (0.98–1.04)	0.558
AKI non-responders	1.02 (1.00–1.04)	0.015

Respiratory failure		
Population	OR (95% CI)	p value
Overall	1.03 (1.00–1.05)	0.029
AKI responders	1.01 (0.97–1.05)	0.664
AKI non-responders	1.03 (1.01–1.06)	0.018

Piano S et al. J Hepatol 2025 (in press)

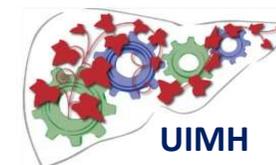


Association between central venous pressure and AKI



Chen X et al. *Ann Intensive Care*. 2018 ; 8 : 91

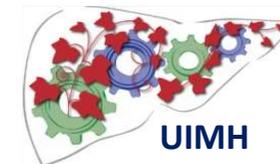
Damman K et al. *J Am Coll Cardiol*. 2009 ; 53 : 582-588.



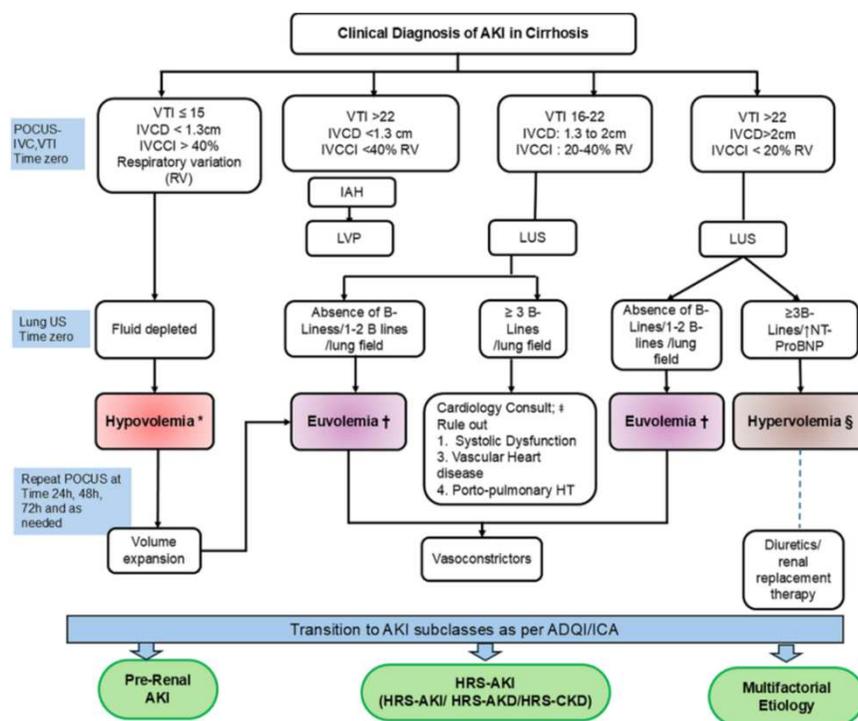
Point of care ultrasonography for assessing volume status in cirrhosis



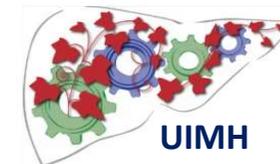
Premkumar M et al. JGH Open. 2019 ; 3 : 322-328.
Lang RM et al. J Am Soc Echocardiogr. 2015 ; 28 : 1-39



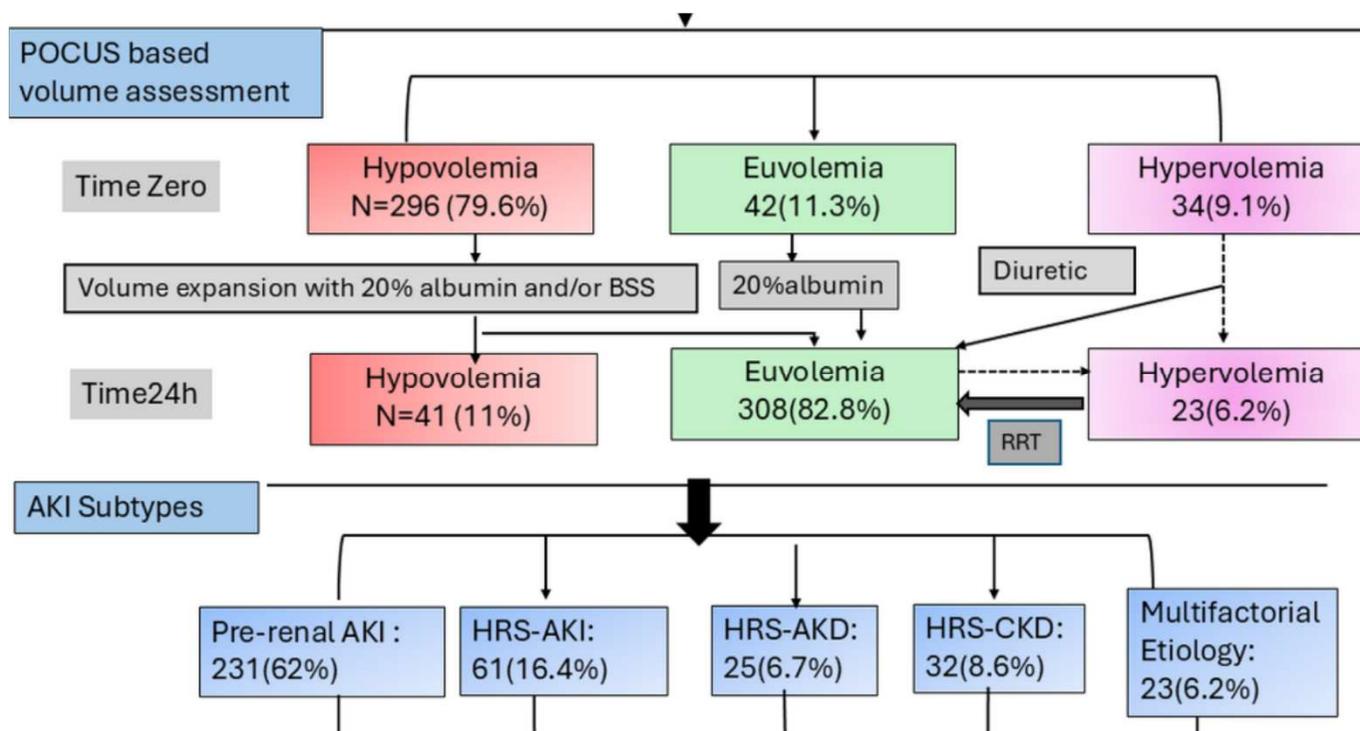
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Premkumar M et al. Hepatology 2025 (in press)



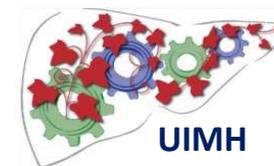
Point of care ultrasonography for assessing volume status in cirrhosis



Premkumar M et al. Hepatology 2025 (in press)

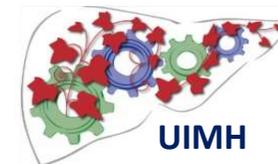


Acute Kidney Injury



Are we ready to use POCUS to guide volume expansion in patients with cirrhosis and AKI?

- Does POCUS modify clinical assessment?

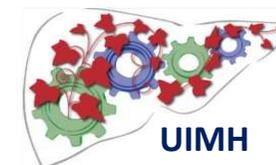


Clinical case scenario

- 75 y
- Deco
- Histo
- Adm
- stag



nd AKI

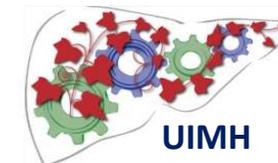


Are we ready to use POCUS to guide volume expansion in patients with cirrhosis and AKI?

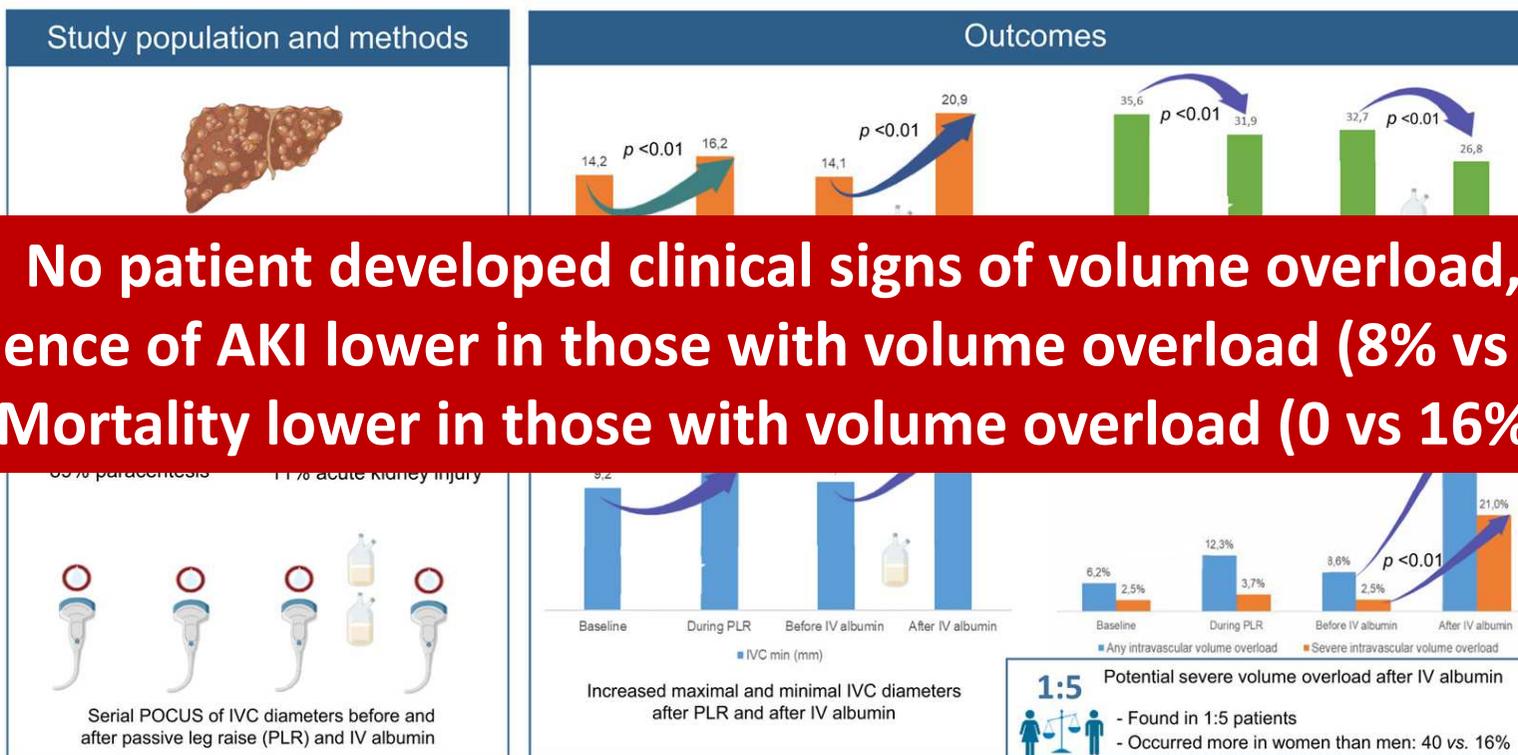
- Does POCUS modify clinical assessment?
- Does POCUS lead to improved clinical outcomes?
- Can we fully rely on POCUS when re-assessing volume status after plasma volume expansion?



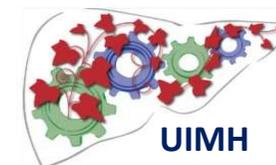
Acute Kidney Injury



POCUS of inferior vena cava following intravascular volume assessment during intravenous albumin infusion in cirrhosis



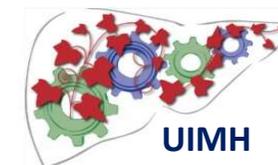
Segna D et al. JHEP Reports 2025. (in press)



Updated diagnostic criteria of HRS-AKI

1. Cirrhosis with ascites;
2. Acute kidney injury;
3. Absence of improvement in SCr and/or UO within 24 h following adequate volume resuscitation
4. Absence of strong evidence for an alternative explanation as the primary cause of AKI

HRS-AKI [...] may also occur in the presence of tubular injury, proteinuria, and/or pre-existing CKD



Comorbidities in patients with cirrhosis and AKI

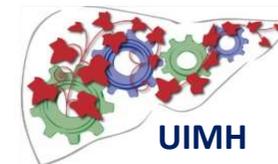
Variables	N (%)
Arterial hypertension	555 (38.1)
Diabetes	515 (35.4)
MASLD	371 (25.5)
CKD	239 (16.4)

Patidar KR, [...] Piano S. *Lancet Gastroenterol Hepatol* 2025 ; 10 : 418-430.



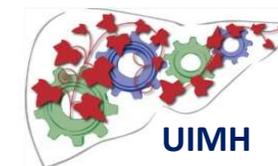


Acute Kidney Injury

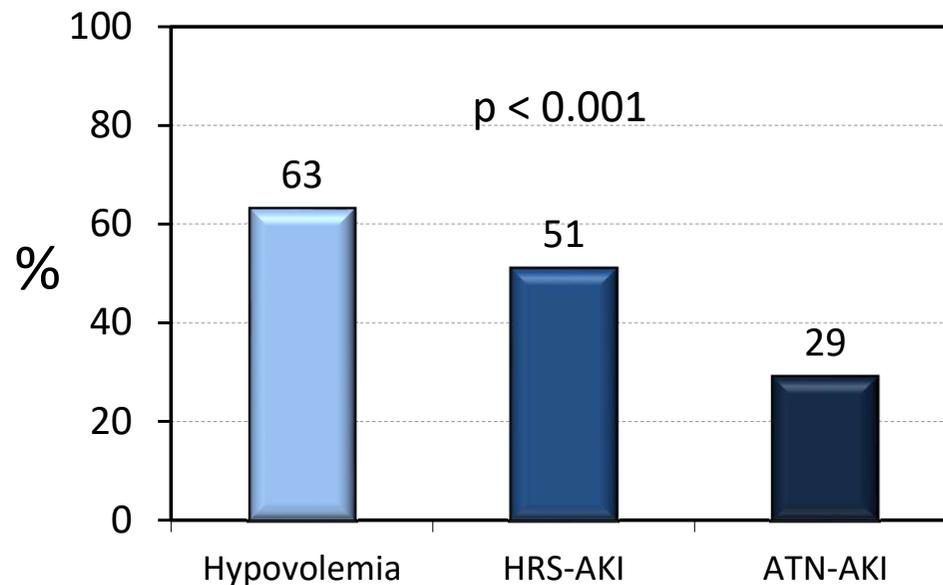
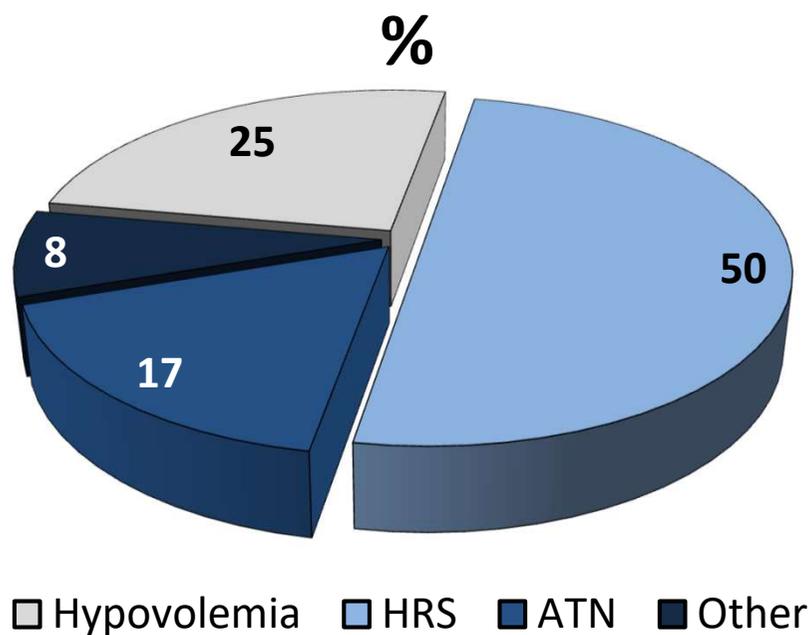


What is a strong evidence for an alternative explanation as the primary cause of AKI?





Phenotype of AKI in patients with cirrhosis treated with terlipressin and albumin and AKI resolution (n=243)



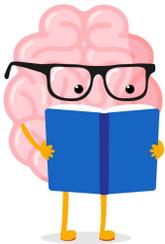
Ma AT, [...] Piano S. Clin Gastroenterol Hepatol. 2025 (in press)





Clinical scenario and laboratory characteristics of HRS-AKI versus ATN-AKI in patients with cirrhosis and ascites

	HRS-AKI	ATN-AKI
Type of precipitant	SBP, LVP	Shock, nephrotoxic drugs
MAP	↓	↓↓
Urinary sodium	<10 mmol/l	> 40 mmol/l
FeNa	< 1%	> 2%
Urine osmolarity	>500 mosm/L	<400 mosm/L
Granular casts	--	++
Renal tubular epithelial cells	--	++

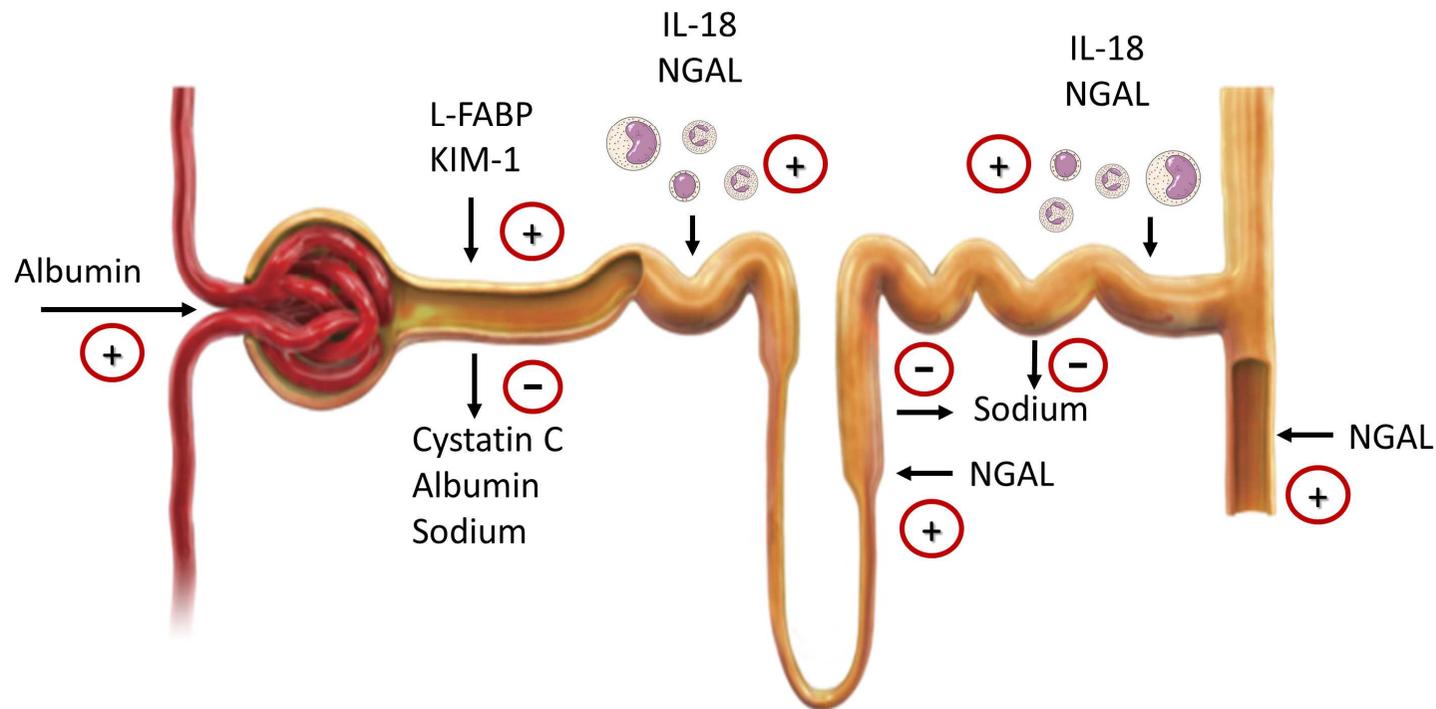




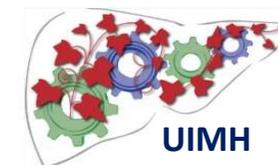
Acute Kidney Injury



Urinary biomarkers of kidney injury and changes observed during intrinsic acute kidney injury

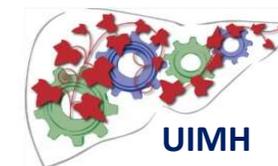


Piano S et al. *Semin Liver Dis.* 2018 ; 38 : 230-241

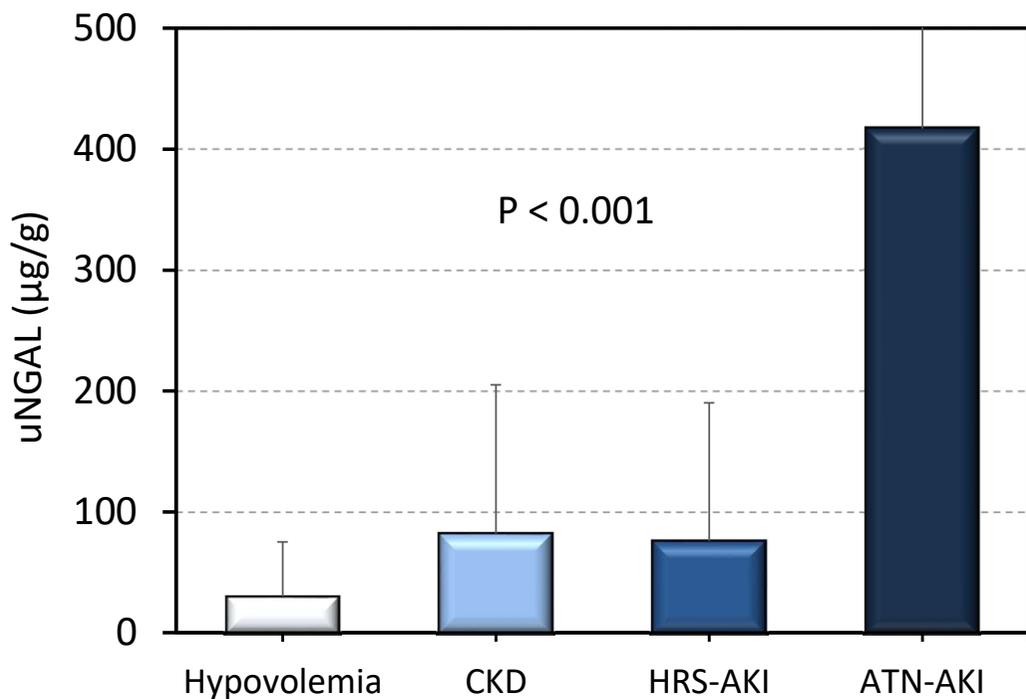


Urinary biomarkers of kidney damage and accuracy in differentiating HRS-AKI and ATN-AKI

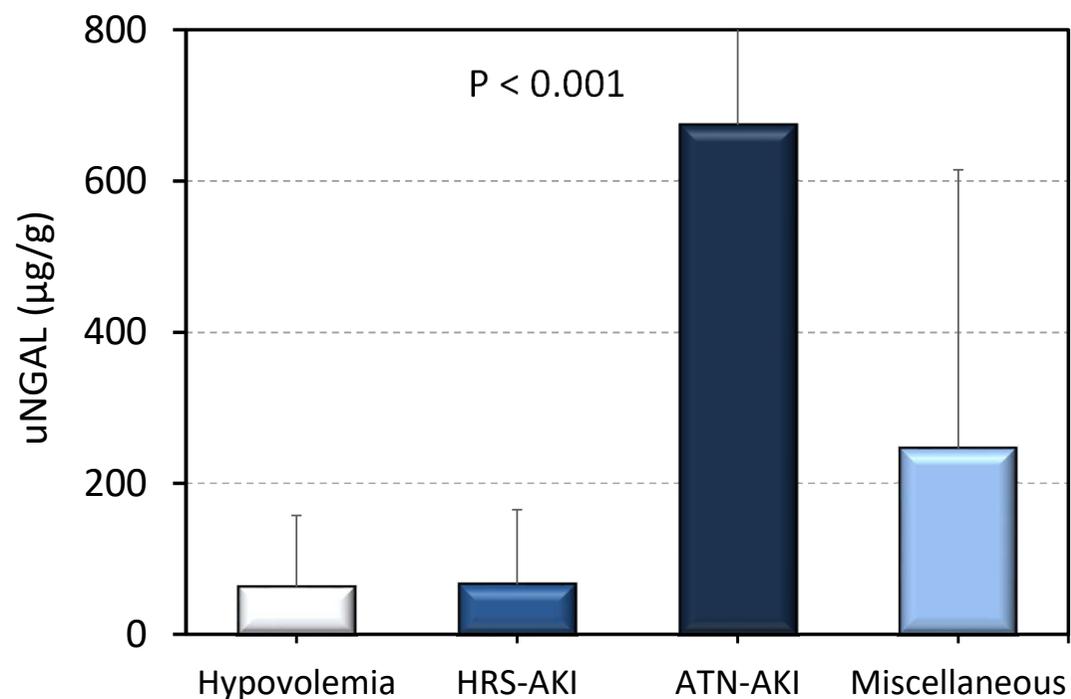
Biomarkers	Discrimination ability between HRS and ATN	Associated outcomes	Limitations
Albumin	0.73 – 0.86	ACLF, mortality	Circulating levels exogenous Tx
FE Na	0.56	Progression of AKI, mortality	Diuretic use
NGAL	0.78 – 0.96	Progression of AKI, ACLF, mortality	Increase in UTI, produced by the liver
IL-18	0.71 – 0.92	Mortality	Increase in inflammation
KIM-1	0.64 – 0.70	ACLF, mortality	--
L-FABP	0.69	Progression of AKI, mortality	Produced by the liver
Cystatin C	0.76	Mortality, ACLF	Increased in CKD



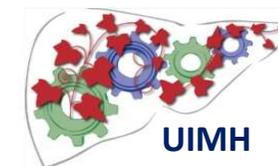
Urinary neutrophil gelatinase associated lipocalin (uNGAL) and differential diagnosis of AKI in cirrhosis



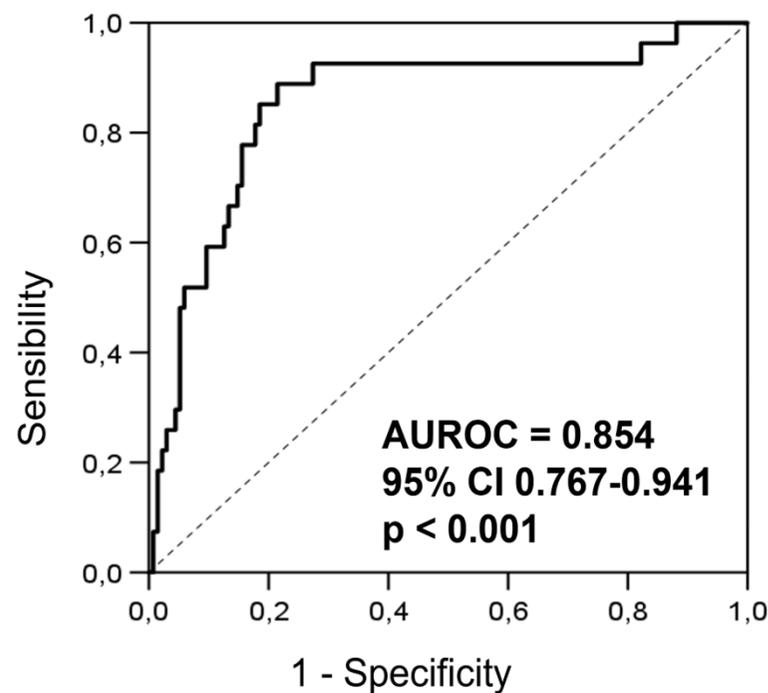
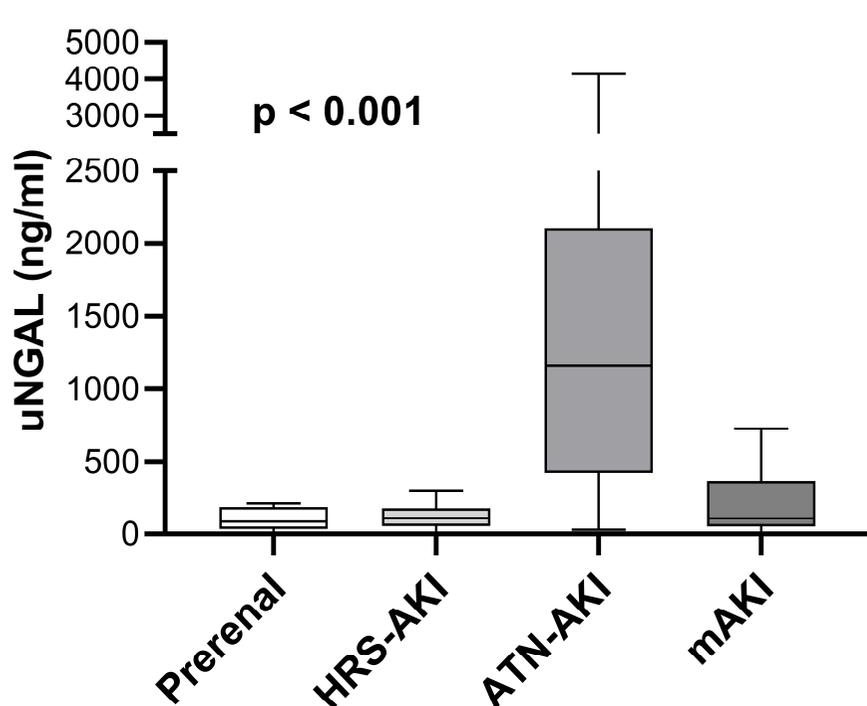
Fagundes C et al. *J Hepatol.* 2012; 57 : 267-273



Huelin P et al. *Hepatology.* 2019 ; 70 : 319-333



Urinary neutrophil gelatinase associated lipocalin (uNGAL) and differential diagnosis of AKI in cirrhosis

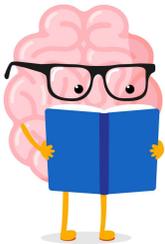


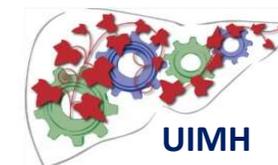
Gambino C, Piano S, et al. *Hepatology*. 2023 ; 77 : 1630-1638



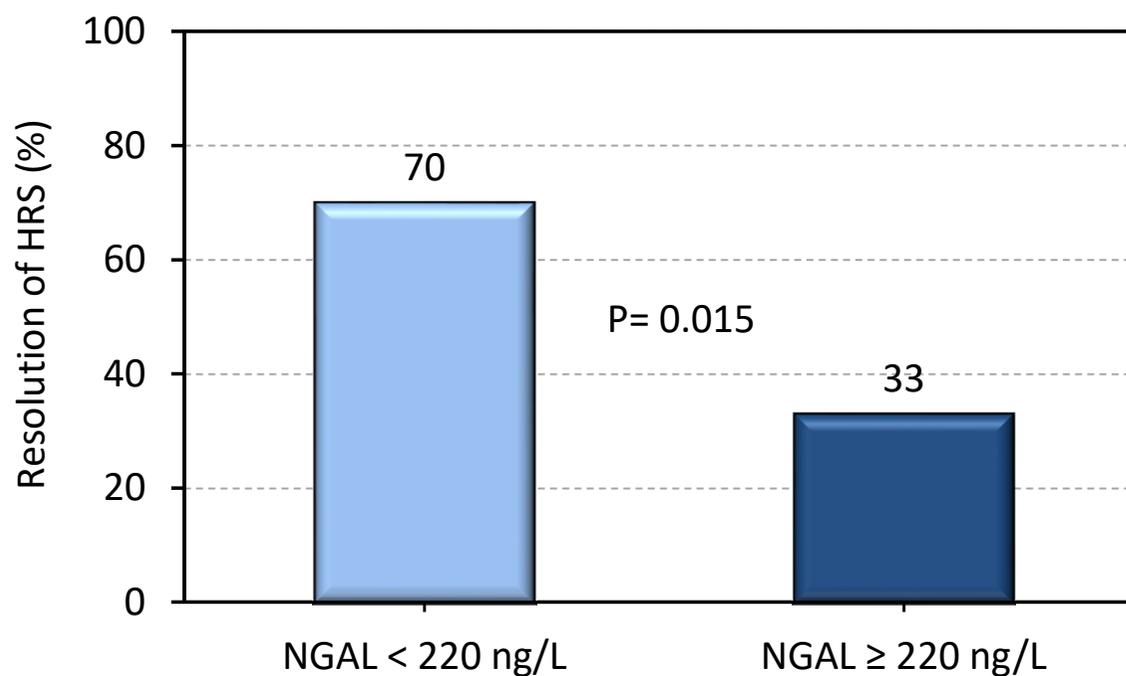
Clinical scenario and laboratory characteristics of HRS-AKI versus ATN-AKI in patients with cirrhosis and ascites

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Urine osmolarity	>500 mosm/L	<400 mosm/L
Granular casts	--	++
Renal tubular epithelial cells	--	++





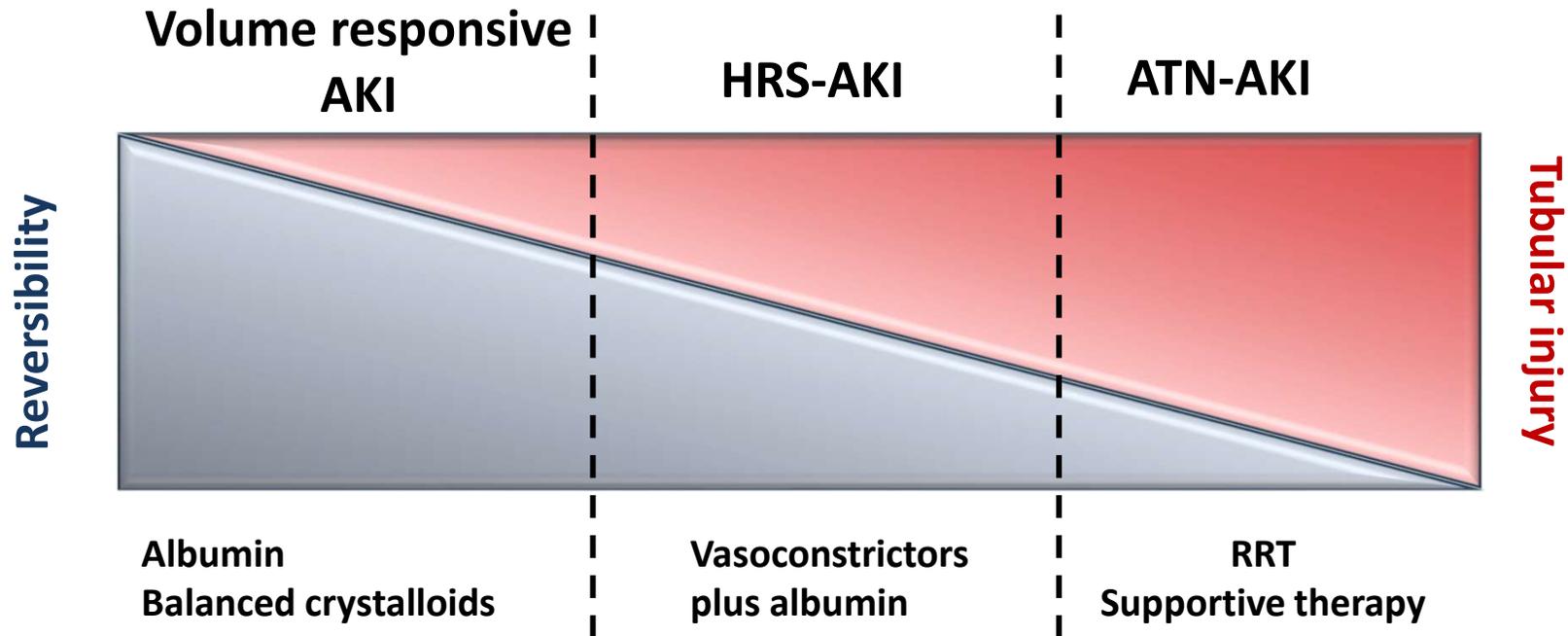
Response to terlipressin and albumin in patients with HRS according to baseline urinary NGAL values

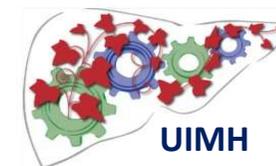


Gambino C, Piano S, et al. Hepatology. 2023 ; 77 : 1630-1638



Spectrum of AKI phenotypes in patients with cirrhosis and AKI



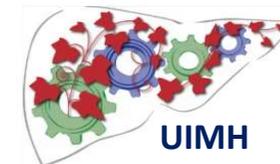


Summary

- Acute kidney injury is a common and life-threatening complication of cirrhosis
- The early management of AKI in decompensated cirrhosis include removal of risk factors, tapering/withdrawal of diuretics and restoration of volume status
- The combination of terlipressin and albumin is the first line treatment in patients with HRS-AKI, but should be used with caution in ACLF grade 3
- The use of terlipressin as continuous infusion is better tolerated
- Liver transplant is the best treatment for HRS-AKI
- Until new data become available it is reasonable to use the EASL/ICA algorithm for management of AKI in cirrhosis
- In the future, integrating POCUS into daily practice and the use of biomarkers of tubular damage, may improve the management of AKI, by enabling a more precise and individualized treatment



Acute kidney injury



Thank you for your attention

