

ALBUMIN IN HEPATORENAL SYNDROME

Optimizing outcomes in the era of vasoconstrictors - HRS-AKI

A focused clinical brief on integrating albumin with vasoconstrictor therapy:

from circulatory physiology to diagnostic criteria and predictors of response.



LEARNING OBJECTIVES

Role of Albumin

Understand the role of albumin in circulatory dysfunction and renal perfusion.³

Vasoconstrictor Therapy

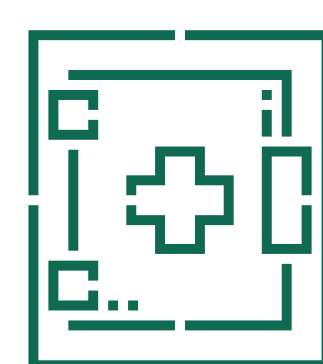
Integrate albumin with terlipressin/norepinephrine protocols.^{2,3}

Diagnosis

Apply ICA criteria for HRS-AKI diagnosis.^{1,3}

Treatment Response

Recognize predictors of response and treatment failure.^{4,5}



DIAGNOSTIC FRAME - WORK-ICA CRITERIA, 2019 UPDATE

HRS-AKI is diagnosed within the broader ICA-AKI staging framework, after excluding shock, nephrotoxic exposure, and structural renal injury.¹

The diagnosis also requires no improvement following diuretic withdrawal and volume expansion with albumin.^{1,3}

The evolving definition and classification of HRS help distinguish HRS-AKI from other causes of acute kidney injury in patients with cirrhosis.¹



COMBINATION THERAPY IN PRACTICE

Management of HRS-AKI focuses on addressing the underlying circulatory dysfunction through vasoconstrictor therapy combined with albumin.^{2,3}

Terlipressin OR Norepinephrine + Albumin

Terlipressin provides Splanchnic Vasoconstriction and, when combined with albumin, forms an established therapeutic approach for HRS-AKI.^{2,3}

Albumin provides plasma expansion to help restore effective arterial volume and support renal perfusion.^{2,3}

The efficacy of terlipressin plus albumin has been evaluated in a randomized controlled trial, demonstrating improved HRS reversal compared with placebo plus albumin.²

Norepinephrine provides an alternative vasoconstrictor approach within ICU-based protocols.³



PREDICTORS WORTH WATCHING

Treatment response can vary according to the severity of underlying liver dysfunction and the patient's clinical status.^{4,5}

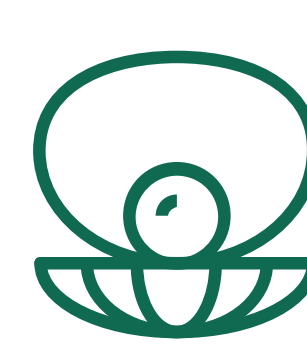
FAVOUR RESPONSE

- Early treatment initiation^{4,5}
- Lower baseline creatinine^{4,5}
- A rise in mean arterial pressure on therapy²

PREDICT FAILURE

- Advanced ACLF grade^{4,5}
- High baseline creatinine^{4,5}
- Lack of early hemodynamic response²

The presence and severity of ACLF are important considerations when assessing prognosis and treatment response.^{4,5}



CLINICAL PEARL

Not all AKI is caused by hypoperfusion. Early recognition of congestion and prevention of fluid overload may help protect renal function in critically ill patients.

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*The cases presented are hypothetical in nature and intended for illustrative purposes only. The management strategies discussed are evidence-based and aligned with established clinical guidelines and scientific literature.

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1. Malbrain MLNG, Marik PE, Witters I, et al. Fluid overload, de-resuscitation, and outcomes in critically ill patients. *Annals of Intensive Care*. 2014;4:16. 2. Divatia JV, Amin PR, Ramakrishnan N, et al. Intensive care in India: The INDICAPS study. *Indian Journal of Critical Care Medicine*. 2016;20(4):216-225. 3. Evans L, Rhodes A, Alhazzani W, et al. Surviving Sepsis Campaign: International guidelines for management of sepsis and septic shock 2021. *Intensive Care Medicine*. 2021;47:1181-1247. 4. Vincent JL, Dubois MJ, Navickis RJ, Wilkes MM. Hypoalbuminemia in acute illness: Is there a rationale for intervention? *Annals of Surgery*. 2003;237(3):319-334. 5. Caironi P, Tognoni G, Masson S, et al. Albumin replacement in patients with severe sepsis or septic shock (ALBIOS trial). *New England Journal of Medicine*. 2014;370:1412-1421.