CRITICAL CARE

LETTER

Hepatorenal syndrome: one size does not fit all

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See related research by Nadim *et al.*, http://ccforum.com/content/16/1/R23

We read with interest the Acute Dialysis Quality Initiative (ADQI) VIII consensus statements on the treatment of patients with hepatorenal syndrome (HRS) and acute kidney injury [1]. While we appreciate the authors' discussion, we question the hemodynamic recommendations and suggest further areas of study (Section III in [1]).

Renal perfusion relies upon cardiac output, renal blood flow, and autoregulation. HRS influences cardiac output and systemic vascular resistance, and establishing a pressure gradient across the glomerulus ensures renal blood flow and glomerular filtration rate [2,3]. In fluid responsive patients, volume resuscitation is a key component of HRS management.

The traditional target mean arterial pressure (MAP) of 65 mmHg to ensure renal perfusion assumes that 'one size fits all' in HRS. The kidneys are in the abdominal compartment, and intraabdominal pressure varies among individuals. The pressure of the compartment during disease states that cause ascites decreases renal perfusion pressure and should be overcome, especially when autoregulation is impaired [4]. In other words, the arbitrary suggestion of increasing the MAP by 10 mmHg (Table 6 in [1]) may not be enough (or may be too much).

Titrating norepinephrine to a baseline MAP of 65 mmHg plus the intraabdominal pressure [4] and administering terlipressin or vasopressin (which may constrict the efferent glomerular arteriole [5]) may be an effective hemodynamic strategy to ensure renal perfusion pressure. Although this recommendation may not be based on grade A evidence, it is physiologically sound (establishes a pressure gradient) and may inspire further studies of hemodynamic management in patients with HRS.

Authors' response

Andrew Davenport, Mitra K Nadim and John A Kellum, for the authors

We thank Drs Parikh and Moitra for their letter concerning our paper reviewing the medical management of HRS [1]. We agree that the hemodynamic alterations of advanced liver disease are complex. Early in the course of cirrhosis the effects of increased splanchnic vasodilatation, primarily due to local nitric oxide synthesis, have limited systemic manifestations. As liver disease progresses, however, systemic vasodilatation develops despite increased visceral sympathetic tone, renin-angiotensin-aldosterone activation, endothelin and vasopressin release, leading to a loss of renal autoregulation [6], increasing the risk of 'pre-renal' acute kidney injury [7].

Terlipressin, a potent vasoconstrictor, particularly for the mesenteric circulation, increases renal perfusion pressure. However, the optimum renal perfusion pressure for patients with cirrhosis is unknown [1]. Following coronary artery surgery, renal auto-regulation is impaired and glomerular filtration rates are higher, with a mean arterial pressure of 70 mmHg [8]. Patients with cirrhosis differ in that they may have ascites and right atrial dilatation. Studies in patients with heart failure with elevated right atrial pressures have shown that intra-abdominal pressures even as low as 8 mmHg adversely affect renal function [9]. In patients with cirrhosis, ascitic drainage can be shown to have an almost immediate dynamic effect on renal perfusion, with changes in intra-renal pressure demonstrated with color Doppler assessment of intra-renal blood flow. Further prospective studies are thus required to determine whether there is an optimal target renal perfusion pressure for patients with HRS treated with terlipressin, but these will also need to include assessment of intraabdominal pressure.

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Abbreviations
ADQI, Acute Dialysis Quality Initiative; HRS, hepatorenal syndrome; MAP, mean arterial pressure.

Competing interests
The authors declare that they have no competing interests.

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Published: 30 March 2012

References

1. Nadim MK, Kellum JA, Davenport A, Wong F, Davis C, Pannu N, Tolwani A, Bellomo R, Genyk YS; the ADQI Workgroup: **Hepatorenal syndrome: the 8th international consensus conference of the Acute Dialysis Quality Initiative (ADQI) group.** *Crit Care* 2012, **16:**R23.
2. Jalan R, Forrest EH, Redhead DN, Dillon JF, Hayes PC: **Reduction in renal blood flow following acute increase in the portal pressure: evidence for the existence of a hepatorenal reflex in man?** *Gut* 1997, **40:**664-670.
3. Stadlbauer V, Wright GA, Banaji M, Mukhopadhya A, Mookerjee RP, Moore K, Jalan R: **Relationship between activation of the sympathetic nervous system and renal blood flow autoregulation in cirrhosis.** *Gastroenterology* 2008, **134:**111-119.
4. Wauters J, Claus P, Brosens N, McLaughlin M, Malbrain M, Wilmer A: **Pathophysiology of renal hemodynamics and renal cortical microcirculation in a porcine model of elevated intra-abdominal pressure.** *J Trauma* 2009, **66:**713-719.
5. Gest AL, Moise AA, Hansen TN, Kaplan S: **Effects of arginine vasopressin on hemodynamics and lung fluid balance in lambs.** *Am J Physiol* 1989, **256:**H641-647.
6. Davenport A, Ahmad J, Al-Khafaji A, Kellum JA, Genyk YS, Nadim MK: **Medical management of hepatorenal syndrome.** *Nephrol Dial Transplant* 2012, **27:**34-41.
7. Wong F, Nadim MK, Kellum JA, Salerno F, Bellomo R, Gerbes A, Angeli P, Moreau R, Davenport A, Jalan R, Ronco C, Genyk Y, Arroyo V: **Working Party proposal for a revised classification system of renal dysfunction in patients with cirrhosis.** *Gut* 2011, **60:**702-709.
8. Redfors B, Bragadottir G, Sellgren J, Swärd K, Ricksten SE: **Effects of norepinephrine on renal perfusion, filtration and oxygenation in vasodilatory shock and acute kidney injury.** *Intensive Care Med* 2011, **37:**60-67.
9. Mullens W, Abrahams Z, Skouri HN, Francis GS, Taylor DO, Starling RC, Paganini E, Tang WH: **Elevated intra-abdominal pressure in acute decompensated heart failure: a potential contributor to worsening renal function?** *J Am Coll Cardiol* 2008, **51:**300-306.

doi:10.1186/cc11246
Cite this article as: Parikh A, Moitra VK: **Hepatorenal syndrome: one size does not fit all.** *Critical Care* 2012, **16:**421.