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Diagnosis of Cirrhotic Cardiomyopathy: The Role of an Impaired Cardiac Reserve

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We read with interest the article by Izzy, VanWagner et al¹ on redefining the criteria for diagnosis of cirrhotic cardiomyopathy (CCM). The authors should be commended on this document that reflects important contemporary advances in cardiovascular imaging. However, the proposed exclusion of a blunted contractile response on stress testing from the diagnostic criteria, warrants discussion.

Mechanistic, echocardiographic and post-mortem studies have all suggested pathogenic alterations including downregulation of β -adrenergic receptors and adverse cardiac remodelling in cirrhosis.² Despite the resting hypercontractile state, it was postulated, and later proven, that these patients demonstrate an attenuated rise in cardiac output (CO) during exercise and on challenge with inotropic and vasoconstrictor analogues.² This is of clinical significance as subclinical cardiac dysfunction that characterizes cirrhosis can be unmasked during periods of physiological stress including hypovolemia, sepsis and liver transplantation.² As such, a normal baseline cardiac function is just one side of the picture. Further, a hyperdynamic resting function, often associated with more severe liver disease, may in fact represent patients encroaching on their cardiac reserve at rest.³

Development of hepatorenal syndrome (HRS) in particular, was suggested as a potential consequence of CCM.¹ However, contemporary echocardiographic studies at rest have demonstrated no differences in ejection fraction or strain in patients with progressive circulatory dysfunction and HRS.⁴ This occurred despite significant increases in neurohormonal and sympathetic activity. A recent study from our group of over 500 patients demonstrated a low cardiac reserve (defined as a Δ CO <25% with low-dose dobutamine) on dobutamine stress testing to be associated with a four-fold increased risk of developing HRS.⁵ Cardiac output in the aforementioned study was estimated using ECG-derived heart rate and a simple and reproducible pulse-wave doppler estimation of stroke volume that requires no post-processing software. Variability in study definitions of what constitutes a blunted cardiac reserve certainly limits its clinical application, as highlighted by the authors. Furthermore,

requisite use of inotropic or vasoconstrictor agents in addition to standard echocardiographic assessment can increase resource utilization and cost.

Notwithstanding, proposal of new diagnostic criteria should not just mirror advances in imaging techniques but also aim to translate present evidence to improving measurable clinical outcomes. Significant challenges remain in the standardization of what constitutes a blunted cardiac reserve in cirrhosis. However, omission of this metric with both pathophysiologic and clinical significance may be premature and could stifle future endeavors to characterize this entity.

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