Letter to the Editor

Ascites in Tropical Endomyocardial Fibrosis

Kenneth B. Desser, M.D.: *Clin Cardiol* 2005;29:375

To the Editor:

Marden and Ou describe a patient with endomyocardial fibrosis who had massive ascites and little or no peripheral edema.¹ The authors state that this disproportionate ascites is one of the mysterious aspects of the disease.

In 1967, Snapper and Kahn² described this peculiar phenomenon in the setting of pericardial entrapment and compression of the caval veins. In such cases, the hepatic veins join the inferior vena cava above the diaphragm within the pericardial sac. They also describe ascites preceding peripheral edema and pericardial constriction associated with endomyocarditis in the same chapter.

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Author's reply:

We read with great interest Dr. Desser's comment regarding the potential hypothesis which might explain such a peculiar clinical aspect of patients with endomyocardial fibrosis (EMF): ascites out of proportion with minimal or absent pedal edema, even in patients with pure left ventricular dysfunction.^{1,2} Several hypotheses may be postulated: (1) Inflammatory cause of ascites related to EMF, in favor of a systemic disease,^{3,4} (2) Other causes of ascites associated with EMF, in particular, related to chronic hepatopathy,⁵ (3) Anomaly of

the hepato-cave junction through the diaphragm muscle and pericardium with partial obstruction to hepatic venous flow as noted by Desser's comment in reference to Snapper, and (4) Different consequences of right cardiac pressure on both hepatic and systemic venous systems.

Indeed, Freers and colleagues' findings substantiate speculations that EMF is a cardiac and systemic disease: peritoneal inflammation with high protein content as well as fibrosis, both leading to an increased sequestration of ascitic fluid.⁵ This first hypothesis is currently debated. Concerning the second point, we have no argument for an associated chronic hepatopathy to EMF,⁵ or other local causes of ascites. Third, we would like to specify that in none of our patients (more than 100 patients a year) with EMF and ascites, hepato-cave junction anomaly through the diaphragm muscle and pericardium has been reported on echography. Finally, high venous pressure levels might have different hemodynamic and vascular consequences on both hepatic and systemic venous systems, favoring peritoneal protein extravasation.

Nevertheless, it seems that the typical clinical picture of disproportionate gross ascites with little or absent edema is not a constant worldwide characteristic of EMF and may differ in certain areas.⁶ The hypothesis of systemic EMF renamed "endomyocardial fibrosis syndrome" by Freers and colleagues³ is still controversial. Most authors only incline toward the severe hemodynamic status of these patients.

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