Case Report

Severe Volume Depletion Leading to Mitral Insufficiency

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We report a patient with profound hypovolemia who developed dynamic left ventricular outflow tract obstruction and severe mitral regurgitation. Both the outflow tract obstruction and mitral regurgitation resolved with volume replacement. Unlike previous reports of dynamic left ventricular outflow obstruction and mitral regurgitation, the degree of mitral regurgitation was severe. Possible mechanisms are discussed.

A hyperdynamic cardiac state secondary to severe volume depletion or catecholamine surge causes dynamic outflow obstruction even in the absence of septal hypertrophy. In previous reports of such dynamic outflow obstruction, the severity of mitral insufficiency, when present, was usually mild. We describe a patient with severe volume depletion in whom mitral insufficiency was severe and resolved with rapid intravenous saline infusion.

Case Report

A 67-year-old woman with a history of chronic hepatitis C and liver cirrhosis, who had undergone combined liver-kidney transplant 4 months prior to admission. After her liver transplant, she recovered the function of her native kidneys, which led to massive diuresis. The patient also developed a high-flow biliary fistula as a complication of her liver transplant. Despite normal oral intake, she presented with dizziness and weakness. The echocardiogram obtained prior to the liver transplant was normal, and a noninvasive ischemia workup was negative.

Physical examination revealed a blood pressure of 95/60 mmHg and a heart rate of 98 beats per minute. The apical impulse was normal. A new apical systolic murmur with an intensity of 4/6, radiating to the axilla, was heard. The electrocardiogram showed an intermittent rate related left bundle-branch block. Echocardiography revealed a normal mitral valvular apparatus, left ventricle, and left atrium. Left ventricular wall thickness was normal, and the estimated ejection fraction was 70%. No septal hypertrophy was present. On Doppler, severe mitral insufficiency was revealed with evidence of flow-reversal in the pulmonary veins. Turbulent flow occurred across the left ventricular outflow tract with a peak gradient of 57.2 mmHg. Systolic anterior motion (SAM) could not be demonstrated by M-mode echocardiography.

It was suspected that the severe mitral insufficiency was secondary to the hyperdynamic left ventricular function and severe volume depletion. During echocardiographic monitoring, 1000 mL of normal saline was rapidly infused over a period of 30 minutes. A progressive slowing of the heart rate from 96 to 64 beats per minute, a decrease in outflow gradient (from 57.2 to 10.1 mmHg), and total resolution of the mitral insufficiency were documented (Fig. 1a, 1b, 2a, and 2b).

Discussion

The effect of volume depletion on the mitral valvular apparatus has been previously reported. Volume depletion may cause left ventricular outflow tract obstruction, SAM, and mitral valve dysfunction. Our patient had a demonstrable gradient across the outflow tract and severe mitral regurgitation during the hyperdy-
Volume depletion leading to mitral insufficiency

Fig. 2 - a) Outflow tract Doppler velocity demonstrating a significant reduction in the gradient to 10 mmHg after infusion of normal saline; b) Resolution of the mitral insufficiency after the saline infusion.

References