Dear Editor,

Stress cardiomyopathy occurs mainly in females and in the typical Takotsubo pattern of left ventricular dysfunction. We report a rare case of stress cardiomyopathy of the reverse Takotsubo pattern, occurring in a male patient during dobutamine stress echocardiography.

Case Presentation

A 53-year-old Chinese male underwent dobutamine stress echocardiography for evaluation of chest pain. He had a background history of previously treated pulmonary tuberculosis and depression. Baseline left ventricular ejection fraction (LVEF) was normal. At peak intravenous dobutamine infusion of 40 mcg/kg/min, he developed retrosternal chest tightness and his systolic blood pressure rose to 235/135 mmHg. The left ventricular cavity became dilated and the LVEF dropped to 20%. Electrocardiography did not show any ST segment elevation. Urgent coronary angiography revealed minor coronary artery disease. Left ventriculogram showed apical hyperkinesis and basal akinesis, a pattern reverse that of Takotsubo cardiomyopathy (Fig. 1). There was no appreciable rise in his serial cardiac enzyme levels. The patient’s symptoms gradually resolved. He was prescribed aspirin, simvastatin, bisoprolol and enalapril. Repeat echocardiography 2 weeks later revealed normal LVEF with no segmental wall motion abnormality.

Discussion

Stress cardiomyopathy usually occurs in females. Unlike the typical Takotsubo cardiomyopathy, this case highlights a rare manifestation of stress cardiomyopathy in a male patient, with left ventriculography revealing an uncommon reverse pattern of apical hyperkinesis and basal akinesia. Shimizu et al1 have described the various ventriculographic morphologies in patients with stress cardiomyopathy, the commonest form being the Takotsubo pattern of basal hyperkinesia and apical akinesia. The other forms are rare.

The pathophysiologic mechanisms underlying the transient left ventricular dysfunction are unclear. Proposed possible explanations include focal myocarditis, catecholamine toxicity, multivessel coronary spasm, impaired coronary microcirculation and myocardial stunning.2 In our patient, the enhanced sympathetic activity from dobutamine infusion may have directly caused transient left ventricular dysfunction. Microvascular ischemia from endothelial dysfunction is another plausible cause of the wall motion abnormality.

Management of stress cardiomyopathy is supportive in nature. Treatment with beta blockers, angiotensin-converting enzyme inhibitors and aspirin is debatable.3,4

REFERENCES


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