β-Receptor Antagonist Cessation Resulting in Tako-Tsubo Cardiomyopathy in a Man With Quadriplegia

To the Editor: A 48-year-old man presented with sudden onset of shortness of breath and retrosternal chest pain. He had a 16-year history of quadriplegia due to a C3-4 spinal fracture, which occurred in a motor vehicle crash. The patient had been taking metoprolol (12.5 mg twice daily) to control hypertension; however, use of this drug had been discontinued 48 hours earlier because of low blood pressure. Elicitation of a detailed history showed no unusual physical or emotional stress.

Physical examination revealed a blood pressure of 80/50 mm Hg, a regular heart rate of 98 beats/min, audible bibasilar crackles, and an audible S_3 but no murmurs or rubs. Electrocardiography showed a 1-mm ST-segment elevation in leads V_2 through V_5. The troponin T level was 1.20 ng/dL (to convert to μg/L, multiply by 1). Two-dimensional echocardiography revealed akinesis of the left ventricular anterior/anteroapical walls, with an ejection fraction of 15%. No obstructive coronary artery disease was evident on angiography. However, left ventriculography showed hyperkinesis of the basal walls and akinesis of the rest of the walls (Figure).

Use of metoprolol was resumed, and 48 hours later the patient was asymptomatic. Crackles in both bases of the lungs had resolved, and the S_3 had normalized. Findings on electrocardiography were normal. The patient was discharged. Six weeks later, two-dimensional echocardiography revealed normal left ventricular function, with an ejection fraction of 65%.

Tako-tsubo cardiomyopathy is characterized by acute and reversible cardiac dysfunction without obstructive coronary artery disease.[1] It is predominantly seen in elderly women and is typically precipitated by acute emotional or physical stress.[2] Recently, excessive sympathetic stimulation has been suggested to play an essential role in the pathogenesis of tako-tsubo cardiomyopathy.[3] However, even though studies have showed elevated plasma catecholamine levels in individuals with tako-tsubo cardiomyopathy, reflecting increased synthesis and reuptake or decreased removal of adrenergic hormones,[4] no basic research has proved that the catecholamine signal pathway plays an important role in the development of tako-tsubo cardiomyopathy. Also, β-receptor antagonists have not consistently prevented onset of tako-tsubo cardiomyopathy.

Long-term effects of β-receptor blockade could result in an up-regulated postreceptor signal transduction cascade, which increases either cardiac β-adrenergic receptor density or its sensitivity (threshold) to stress-induced cardiomyopathy. Abrupt discontinuation of β-blockade therapy can result in "hyperadrenergic syndrome" and cardiomyopathy. Rapid resolution of cardiac abnormalities in response to resumption of β-receptor antagonists also supports the causal link between the β-adrenergic receptor pathway and development of tako-tsubo cardiomyopathy.

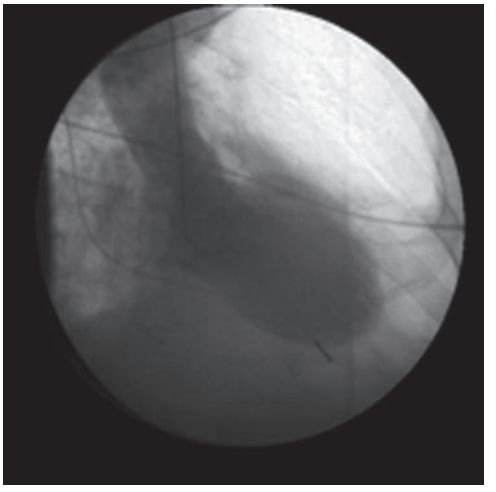

FIGURE. Ventriculogram revealing apical ballooning during systole.

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CORRECTION

Incorrect spelling of an author's name in the reference list: In the editorial entitled "Khat Chewing: A Smoking Gun?" published in the November 2010 issue of *Mayo Clinic Proceedings* (*Mayo Clin Proc*. 2010;85(11):971-973), an author in reference No. 13 was spelled incorrectly. It should read as follows: **El-Menyar** A, Zubaid M, Sulaiman K, et al. In-hospital major clinical outcomes in patients with chronic renal insufficiency presenting with acute coronary syndrome: data from a registry of 8176 patients. *Mayo Clin Proc*. 2010; 85(4):332-340.

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