American Journal of Emergency Medicine (2013) 31, 447.e1–447.e3

The American Journal of Emergency Medicine

www.elsevier.com/locate/ajem

Case Report

Stress-induced transient midventricular ballooning: a new variant of broken heart syndrome☆

Abstract

A 72-year-old female patient admitted to the emergency department with substernal chest pain due to vigorous emotional stress. Her electrocardiogram revealed negative T waves in DI, aVL, and V_1 to V_3 derivations, and she had an elevated troponin level. Cardiac catheterization was performed and showed angiographically normal epicardial coronary arteries. The left ventriculogram demonstrated midventricular dilatation and akinesis with a hypercontractile apex and base. We report a case of new variant of broken heart syndrome in which only the midventricle is affected.

Transient midventricular ballooning syndrome is a clinical entity that is probably a new variant of left ventricular (LV) ballooning syndrome, which is pathophysiologic; mechanisms are not well understood. The systolic dysfunction and the regional wall motion abnormalities are transient and resolve completely within a matter of days to a few weeks [1]. In this report, we present a case of transient midventricular ballooning syndrome due to vigorous emotional stress.

A 72-year-old woman with a history of hypertension but no clinical history of coronary artery disease was admitted to the emergency department with substernal chest pain. Her symptoms began after vigorous emotional stress. On admission, her arterial blood pressure and heart rate were 165/95 mm Hg and 96 beats per minute, respectively. Electrocardiography (ECG) revealed deep negative T waves in leads DI, aVL, and V_1 to V_3 (Fig. 1). Serum biomarkers of myocardial injury were also elevated, and measured troponin I was 0.99 ng/dL. Therefore, she was immediately taken to catheterization laboratory for emergent angiography with the diagnosis of possible acute coronary syndrome. Coronary angiography demonstrated normal coronary arteries with no vasospasm or coronary dissection, and left ventriculogram showed midventricular dilatation and akinesis with a hypercontractile apex and base in systole (Figs. 2 and 3, Movie 1). Ejection fraction (EF) was 45%. The patient was initiated on acetylsalicylic acid, angiotensin-converting enzyme inhibitor, and β-blocker therapy. No additional symptom had occurred during hospitalization, and she was discharged from the hospital after 7 days without any complaints. A left ventriculography obtained 4 weeks later showed normal LV size with recovery of function and marked improvement of the midventricular wall motion abnormalities (Fig. 4, Movie 2).

Transient LV ballooning syndrome (TLVBS) was first described in Japan in 1991. When this clinical syndrome was first described, it was referred to as transient LV apical ballooning because all the patients had characteristic akinesia and ballooning of the apex and a normal or hypercontractile basal segments as evident by the left venticulogram [1]. Hurst et al [2] reported a new variant of this clinical syndrome in 4 female patients who presented with symptoms suggestive of acute coronary syndrome. Left side of the heart catheterization revealed normal epicardial coronary arteries, whereas left ventriculogram showed midventricular akinesis and dilatation with hypercontractile apex and base, which is consistent with transient midventricular ballooning syndrome similar to the patient we presented above. Transient midventricular ballooning is a recently described variant of stress-associated transient ventricular dysfunction that shares many of the clinical features seen in the more common transient apical ventricular ballooning or Takotsubo cardiomyopathy [3]. Most of the TLVBS (apical or midventricular) were observed in postmenopausal women, and the most common clinical presentations are chest pain or dyspnea [4].

However, the underlying mechanism of TLVBS is still poorly understood. One of the postulated mechanism is microvascular dysfunction, which has been identified in two-third of the patients presented with apical ballooning syndrome; impaired myocardial perfusion despite normal epicardial flow has been documented based on coronary angiography [5,6]. Catecholamine levels were found to be high in patients with LV apical or midventricular ballooning compared with patients with acute myocardial infarction. The levels remain elevated even after 1 week after the initial presentation. The postulated mechanisms of catecholamine-induced cardiomyopathy include mutivessel epicardial spasm, micovascular dysfunction, or direct myocytes injury [7].

The optimal management of apical or midventricular ballooning syndrome has not been established, but supportive therapy invariably leads to spontaneous recovery. Because

☆ The authors declare that they have no commercial associations or sources of support that might pose a conflict of interest.

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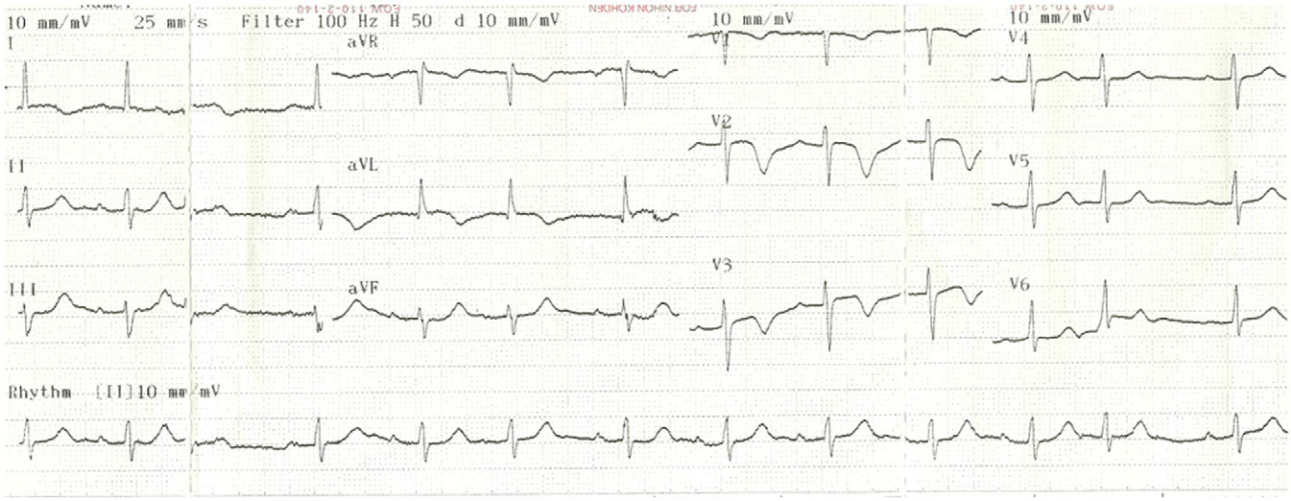

Fig. 1 Electrocardiogram obtained at admission showed deep negative T waves in leads DI, aVL, and V_1 to V_3.

the presentation in these patients is similar to acute coronary syndrome, initial management should be directed toward the treatment of myocardial ischemia, with continuous ECG monitoring and administration of acetylsalicylic acid, intravenous heparin, angiotensin-converting inhibitors, and *β*-blockers. Once the diagnosis of transient LV dysfunction has been made, aspirin can be discontinued unless there is coexisting coronary atherosclerosis. *β*-Blockers are the cornerstone of treatment if no contraindications exist because an excess of catecholamines may play a role in precipitating the conditions. On rare occasions, when there is significant hypotension, it is important to exclude dynamic LV outflow tract obstruction with echocardiography. If present, a cautious trial of *β*-blockers may help by reducing the hypercontractility of the base of the LV. Diuretics are effective for the treatment of congestive heart failure [8]. The right ventricle is also involved in approximately one-third of cases, and these patients are sicker and more likely to develop congestive heart failure.

Patients with TLVBS generally have a good prognosis in the absence of significant underlying comorbid conditions. In-hospital mortality from transient LV dysfunction is very low and unlikely to be greater than 1% to 2%. The recurrence rate of transient LV dysfunction is no more than 10%. In the absence of contraindications, long-term *β*-blocker therapy is recommended with the aim of reducing the likelihood of a recurrent episode [9].

In conclusion, a few recent studies revealed that there are no differences in demographic, clinical, angiographic, and laboratory parameters or outcome between apical or midventricular forms of transient LV dysfunction. Based on these data, transient cardiomyopathy, also termed *Takotsubo cardiomyopathy*, therefore, should no longer be regarded as an exclusively apical ballooning syndrome but rather a transient LV dysfunction syndrome with an apical or midventricular pattern of wall motion abnormality.

Supplementary materials related to this article can be found online at http://dx.doi.org/10.1016/j.ajem.2012.05.038.

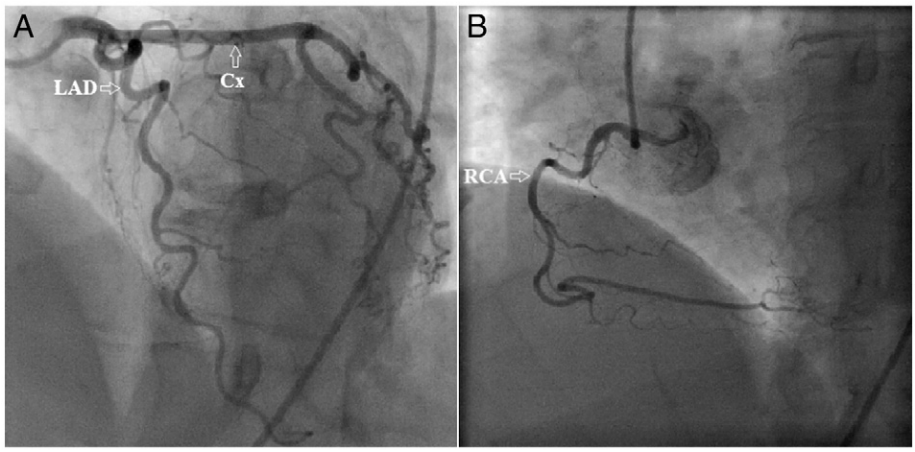

Fig. 2 A, Selective left coronary angiogram obtained at left anterior oblique cranial projection showing normal left anterior descending and circumflex arteries. B. Selective right coronary angiogram obtained at left anterior oblique caudal projection showing normal right coronary artery. LAD indicates left anterior descending; Cx, circumflex; RCA, right coronary artery.

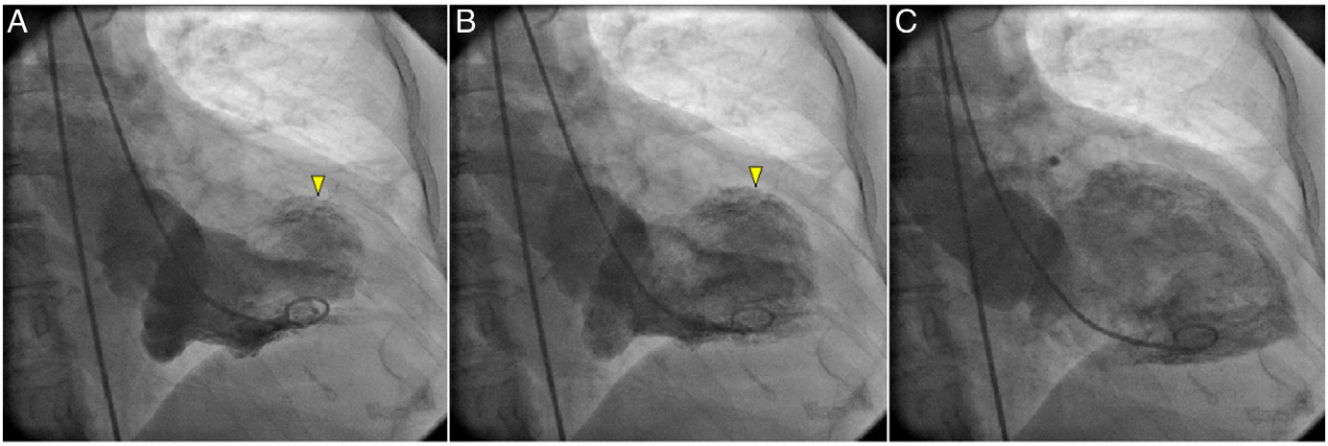

Fig. 3 A and B, A left ventriculography obtained at right anterior oblique cranial projection showing midventricular ballooning during systole (arrows). C, Ventriculographic imaging during diastole.

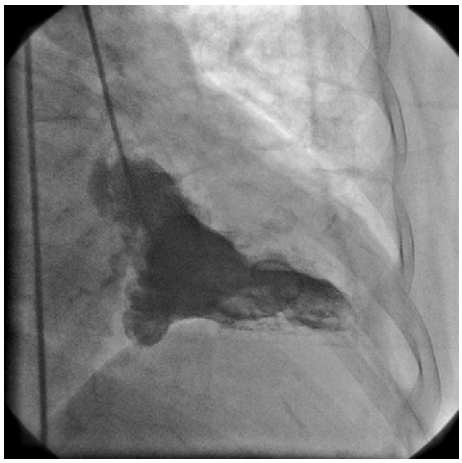

Fig. 4 A left ventriculography obtained 4 weeks later showed normal LV size and function (EF, 60%) and no regional wall motion abnormalities.

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http://dx.doi.org/10.1016/j.ajem.2012.05.038

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