

Transient Left Ventricular Dysfunction due to Stress-Induced Cardiomyopathy

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The case presented here is of a 71-yr-old female patient who met the diagnostic criteria for stress-induced cardiomyopathy, which was triggered by intense emotional stress after being hit by a bicycle. The clinical picture mimicked that of an acute myocardial infarction, manifesting as precordial pain, ST-segment depression followed by deep negative T waves and prolonging of the QT interval, slight increase in cardiac enzymes and coursing with transient apical ballooning of the left ventricle and hyperkinesis of the basal walls (conferring the aspect of “apical ballooning”), although in the absence of subepicardial coronary obstruction. Ventricular function normalized after the second week of clinical evolution.

Introduction

A new cardiological syndrome called stress-induced cardiomyopathy was recently characterized based on cases initially described in Japanese patients¹ and subsequent reports of its occurrence in several Western countries^{2,3}. Also called “the broken heart syndrome” and transient apical ballooning of the left ventricle, it is characterized by a clinical picture that mimics the acute myocardial infarction (AMI), presenting as precordial pain, ST segment displacement and increase of the biological markers of myocardial necrosis, coursing with transient apical ballooning of the left ventricle, but in the absence of significant sub-epicardial coronary obstruction. Characteristically, the clinical picture is preceded by intense emotional and/or physical stress^{4,5}.

We present herein the case of a 71-year-old patient in whom the acute transient ventricular dysfunction was triggered by intense emotional stress, fulfilling the current criteria for the diagnosis of this syndrome.

Case Report

A 71-year-old female Caucasian patient presented a sudden picture of intense pain in the anterior portion of the thorax, irradiating to the upper left limb and pyrosis, associated to

Key words

Cardiomyopathies; ventricular dysfunction, left; myocardial stunning.

sudoresis, vomiting and dyspnea. The clinical picture started after intense physical and emotional stress, when she was hit by a bicycle, without evidence of thoracic trauma. The symptom persisted for 6 hours, when she was brought to specialized cardiological care. The patient had a history of systemic arterial hypertension for 30 years. She denied a history of diabetes, dyslipidemia, alcohol consumption or smoking.

At the physical examination she was alert, tachydyspneic, presented rosy and hydrated mucosae, was afebrile, acyanotic, anicteric, with no turgescence of the jugular vein.

Teleinspiratory crackles were present in the lower third of both pulmonary fields. No signs of cardiomegaly, regular heart rhythm with normal heart sounds and presence of protodiastolic gallop. BP was 130/70 mmHg, HR=112 bpm. The pulses were wide and symmetric, the extremities were warm and there was good peripheral perfusion.

The initial ECG (Figure 1) showed tachycardic sinus rhythm, wide QRS due to conduction disorder in the left bundle branch and secondary alterations of ventricular repolarization with ST-segment depression in the anteroseptal wall. Within the first hours of clinical evolution, the ECG showed reversion of the left branch blockage and the appearance of deep negative T waves and prolonging of the QT interval (640 ms), (Figure 1).

Radiological assessment of the thorax showed normal cardiothoracic index, signs of pulmonary venocapillary congestion with remodeled pulmonary vasculature (Figure 2). Biochemical assessment showed that the CK-MB levels were not significantly increased (maximum value of 31 UI/l, 3 consecutive measurements) and slight troponin increase (1.06 ng/ml).

The initial diagnostic hypothesis was acute coronary syndrome associated to left ventricular dysfunction. Treatment with non-fractionated heparin, aspirin, intravenous clopidogrel, nitroglycerin and furosemide was initiated.

On the fourth day of evolution, a transthoracic echocardiographic study showed a dilated left atrium (47 mm), left ventricle with preserved diastolic diameter at the upper range of normality (55 mm), slightly reduced global systolic performance (ejection fraction = 42%) and multiple defects of segmental parietal mobility involving the apical and middle portions of the left ventricle (Figure 3).

Heart catheterism showed coronary artery circulation free of significant obstructive lesions and the cineventriculography disclosed anterolateral inferior septal akinesis and moderate inferior apical hypokinesis (Figure 4). The segmental mobility disorders extended beyond the irrigation zone of a single

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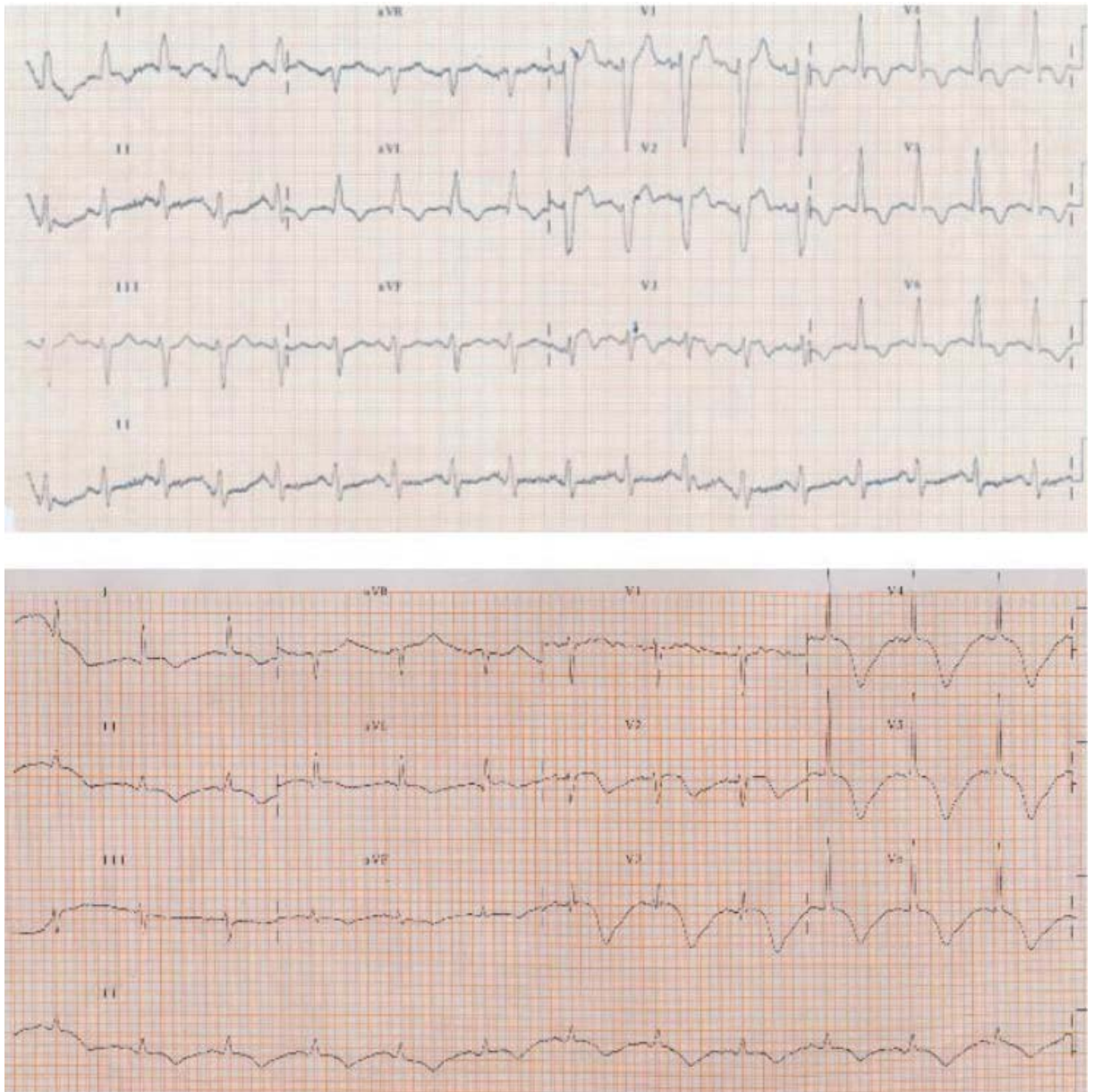


Fig. 1 - 12-derivation ECGs obtained at the hospital admission in the emergency room (upper panel) and 24 hrs later (lower panel).

coronary vessel, which gave the very typical aspect of apical ballooning to the left ventricle in the telesystolic picture of the ventriculography (Figure 4).

The clinical treatment was modified with the inclusion of beta-adrenergic blocker (metoprolol, given orally) and angiotensin II receptor blocker (losartan, given orally), maintaining the therapy with furosemide and aspirin, given orally.

The patient evolved with fast improvement of the clinical picture. A radiological study of the thorax (Figure 2) and echocardiogram (Figure 3) performed 2 weeks after the

onset of the picture showed a complete reversion of the left ventricular dysfunction.

Discussion

Although its actual prevalence has yet to be defined, retrospective studies suggest that stress-induced cardiomyopathy is not a very rare condition, affecting around 2% of the cases seen as having suspected acute coronary syndrome and being more common in older post-menopausal women^{4,6}.

In addition to the clinical presentation simulating a picture

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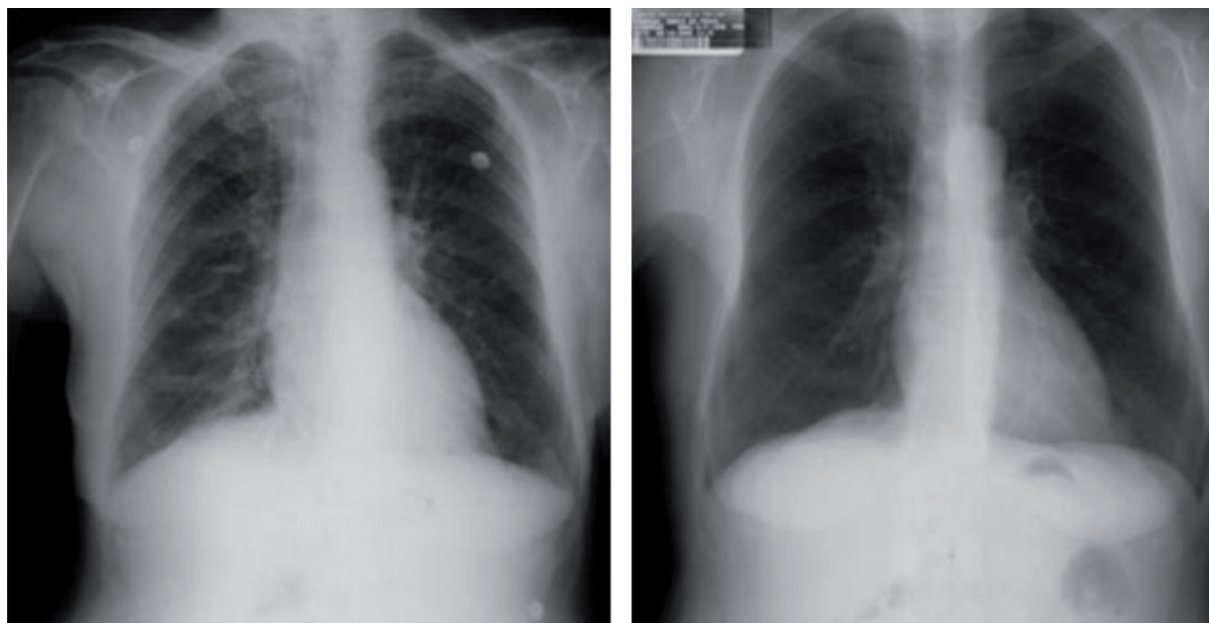


Fig. 2 - Aspects of the conventional radiological assessment of the thorax obtained at the hospital admission in the emergency room (left panel) and before hospital discharge (right panel).

of acute myocardial infarction with ventricular dysfunction, it can course with signs of congestion, and, as in the case reported here, the marked ST-segment depression is the most common alteration at the initial ECG. Other alterations, such as deep negative T waves and prolonging of the QT interval, are also often reported.

Among the most commonly described manifestations of stress-induced cardiomyopathy, the function of the distal and apical segments of the left ventricle is depressed and there is compensatory hyperkinesis of the basal walls, producing the characteristic aspect of apical ballooning during the systole. A striking aspect that can suggest the diagnosis is the detection of left ventricular segmental parietal dysfunction that extends beyond the irrigation zone of a single coronary vessel.

One of the most interesting aspects of this syndrome is its triggering following emotional stress situations (death of family members, natural disasters, financial catastrophes). It is postulated that the physiopathological mechanism responsible for the syndrome is an intense adrenergic discharge, inducing multivessel coronary spasm (possibly at microcirculatory level, but one cannot rule out its occurrence in sub-epicardial vessels), causing myocardial stunning⁵. In this sense, the stress-induced cardiomyopathy is quite similar to other situations of catecholamine excess, such as those associated to brain injuries, pheochromocytoma, scorpionism and the accidental infusion of sympathomimetic drugs, clinical situations in which the transient left ventricular dysfunction also occurs^{7,8}.

As it mimics them in several aspects, and despite its relatively low frequency, the stress-induced cardiomyopathy must be considered a differential diagnosis for patients suspected of having acute coronary syndrome, especially when they appear to be emotionally unstable and when the extension of the ischemic abnormalities at the ECG or the severity of the left ventricular dysfunction exceed the evidence of myocardial necrosis assessed by the increase in enzymatic levels; similarly, this diagnosis must be considered when the coronary angiography confirms the absence of obstructive lesions. Its prompt recognition and intensive treatment with pharmacological agents or mechanical circulatory support is indicated, as the complete recovery of the ventricular function is expected within a few days, with a favorable prognosis seen in almost all of cases³.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

There were no external funding sources for this study.

Study Association

This study is not associated with any graduation program.

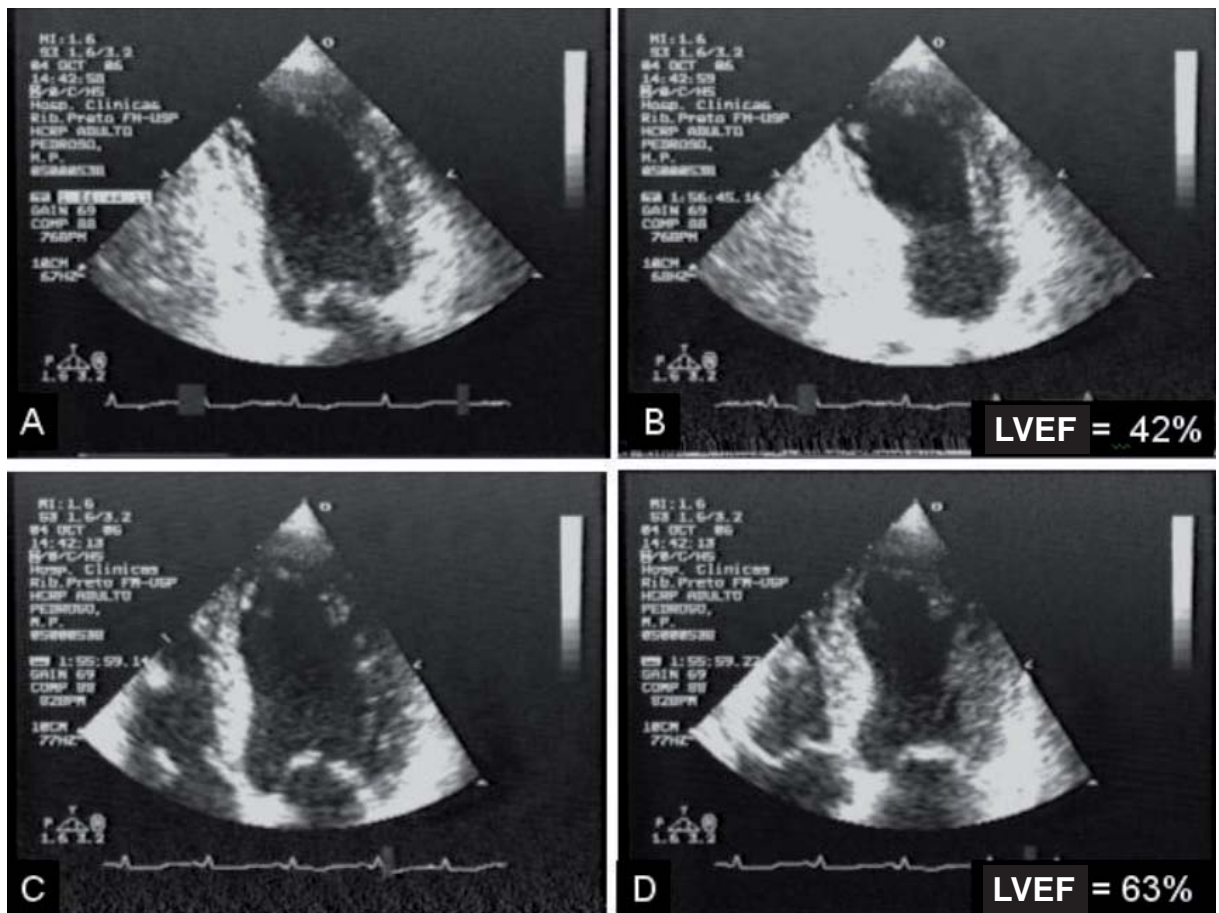


Fig. 3 - Illustrative images of the transthoracic echocardiographic assessment obtained on the 4th day of clinical evolution (A and B) and before hospital discharge, 3 weeks later (C and D). A and B show images of the diastolic and systolic pictures, respectively, obtained at the apical two-chamber view. A significant involvement of the segmental parietal mobility in the medium and apical portions of the left ventricle can be observed with hyperkinetic compensatory movement of the basal segments. At the later study (C and D), the complete reversion of the abnormalities can be observed.

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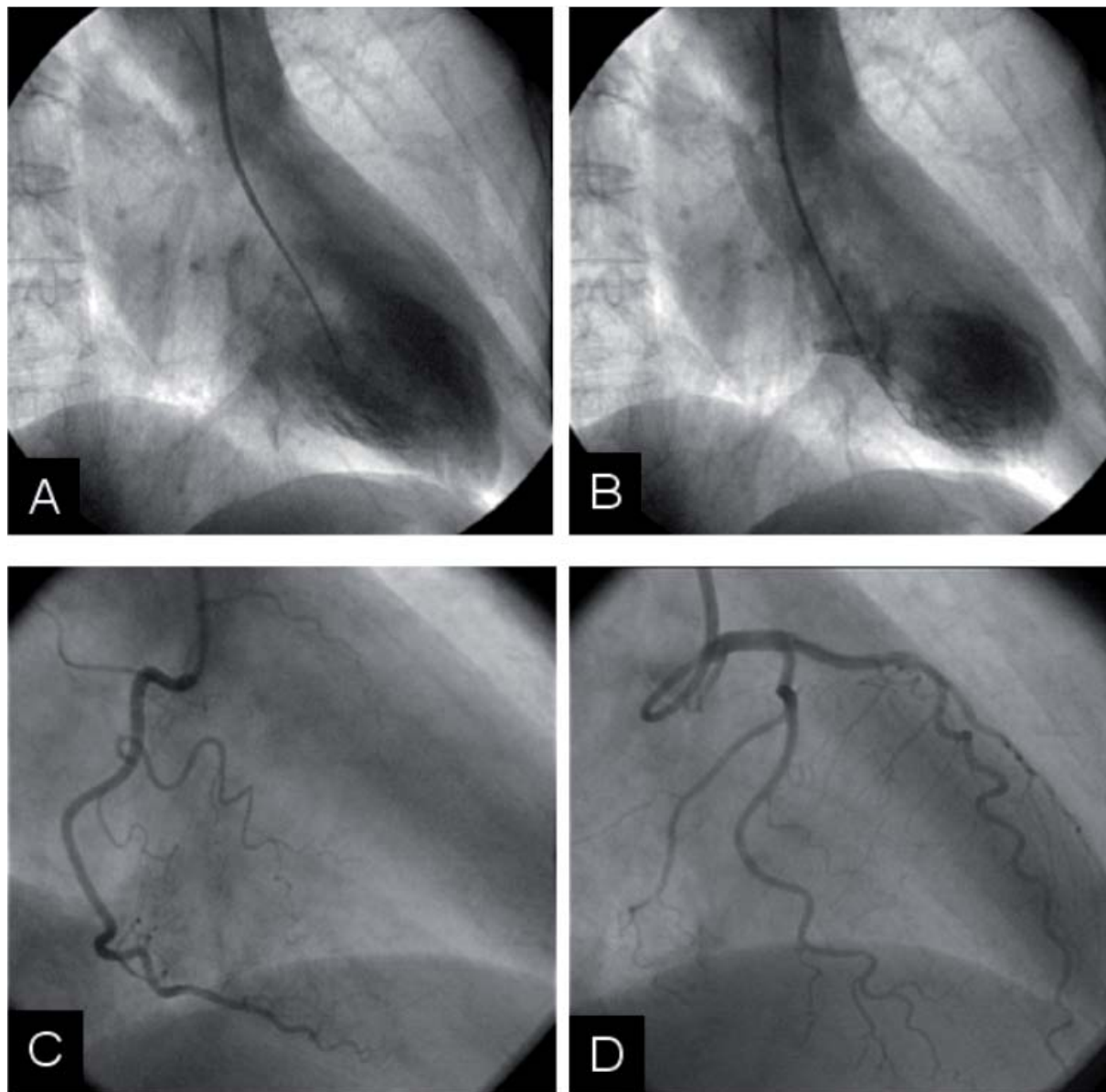


Fig. 4 - Coronary angiographic images of radiological contrast during cardiac catheterism performed on the 4th day of clinical evolution. The diastolic (A) and systolic (B) pictures show akinetic regions that give the left ventricle the typical aspect of apical ballooning. The right (C) and left (D) coronary angiographies show coronary artery circulation free of significant obstructive lesions.

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