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Stress-Induced Cardiomyopathy Presenting as Acute Myocardial Infarction

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Stress-induced cardiomyopathy is described as an acute cardiomyopathy that occurs under the influence of an excessive level of catecholamine related to intense emotional stress. A 64-year-old woman presented with symptoms of acute myocardial infarction after emotional upset, but her coronary angiographic findings were revealed to be normal. Diffuse T wave inversions were observed in her electrocardiograms with akinetic wall motions sparing the basal segments in her left ventriculography. After four months, her electrocardiogram and echocardiogram findings had completely returned to normal. The precise diagnosis of this acute cardiomyopathy must be emphasized because it can initially be misdiagnosed as acute coronary syndromes. However in complete contrast to acute myocardial infarction, it has a rapid and favorable recovery with hardly any sequelae after a few months.

Key Words: Stress-induced cardiomyopathy, acute myocardial infarction, catecholamine

INTRODUCTION

Stress-induced cardiomyopathy was first described by Marilyn et al. in 1980 through their analysis of 11 victims who showed cardiac changes, focal myocardial necrosis or myofibrillar degeneration.[1] They interpreted these myocardial changes as catecholamine mediated lesion which is related to the lethal potential of stress. The typical characteristics of this acute stress-induced cardiomyopathy have been described by Pavin et al. as sudden onset immediately following intense emotional stress, chest pain associated with vomiting followed by shock, acute respiratory distress related to severe pulmonary edema, major impairment in left ventricular contractility sparing the basal segments without any ventricular dilatation, diffuse reversible changes in ECG measurements, and rapid and favorable recovery with hardly any sequelae.[2]

Here, we report a case of stress-induced cardiomyopathy presenting with symptoms mimicking acute myocardial infarction. To our knowledge, this is the first case of stress-induced cardiomyopathy in Korea.

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CASE REPORT

A 64-year-old woman visited our emergency room because of severe chest pain after becoming angry. Earlier in the evening, she had been subjected to an unexpected tax surveillance which made her very upset. She reported that she had never been as angry as this before, and that it was the worst day of her life. She suffered chest discomfort with nausea and vomiting. Her initial blood pressure was 83/51 mmHg and her hepatojugular reflux was positive. Crackles were audible in the lower part of both lungs, with evidence of pulmonary edema in a chest radiograph. An electrocardiogram taken on her arrival showed horizontal ST segment elevation in leads II, III, and aVF (Fig. 1A). Her initial laboratory findings revealed that creatinine phosphokinase, creatinine phosphokinase-MB, and lactate dehydrogenase were elevated to 293 IU/L (24 - 204

IU/L), 40 U/L (1-16 U/L), and 943 IU/L (240-480 IU/L) respectively. There was no evidence of neutrophilia or eosinophilia in this patient, and the titer of C-reactive protein was 0.04 mg/dl initially. On echocardiogram, the ejection fraction was about 30% and global akinetic wall motions were observed except in the basal portion (Fig. 2A and 2B).

The patient was initially diagnosed as having acute myocardial infarction, and coronary angiography was performed the following day. The coronary angiograms were normal, but akinetic wall motions characteristically sparing the basal segments, were found on left ventriculography (Fig. 3 and Fig. 4). The left ventricular end diastolic pressure was 19 mmHg on hemodynamic findings. Finally, she was diagnosed with acute stress-induced cardiomyopathy and was treated with carvedilol, aspirin, captopril, atenolol and spironolactone. Her electrocardiograms changed to show diffuse T wave inversions (Fig. 1B and 1C). The wall motions in her echocardiograms returned to almost normal findings after 2 weeks. Her electrocardiograms and echocardiograms returned completely to normal after four months (Fig. 1D, Fig. 2C, and 2D).

DISCUSSION

In 2001, Tsuchihashi et al. reported a heart syndrome exhibiting acute onset, transient reversible left ventricular apical wall motion abnormalities with chest symptoms, electrocardiographic changes, and minimal myocardial enzymatic release which mimics acute myocardial infarction in patients without angiographic stenosis on coronary angiogram.[3] They termed this transient left ventricular apical ballooning as "takotsubo shaped cardiomyopathy" and found that it is related to various exacerbated systemic disorders, noncardiac surgical procedures, and psychotic

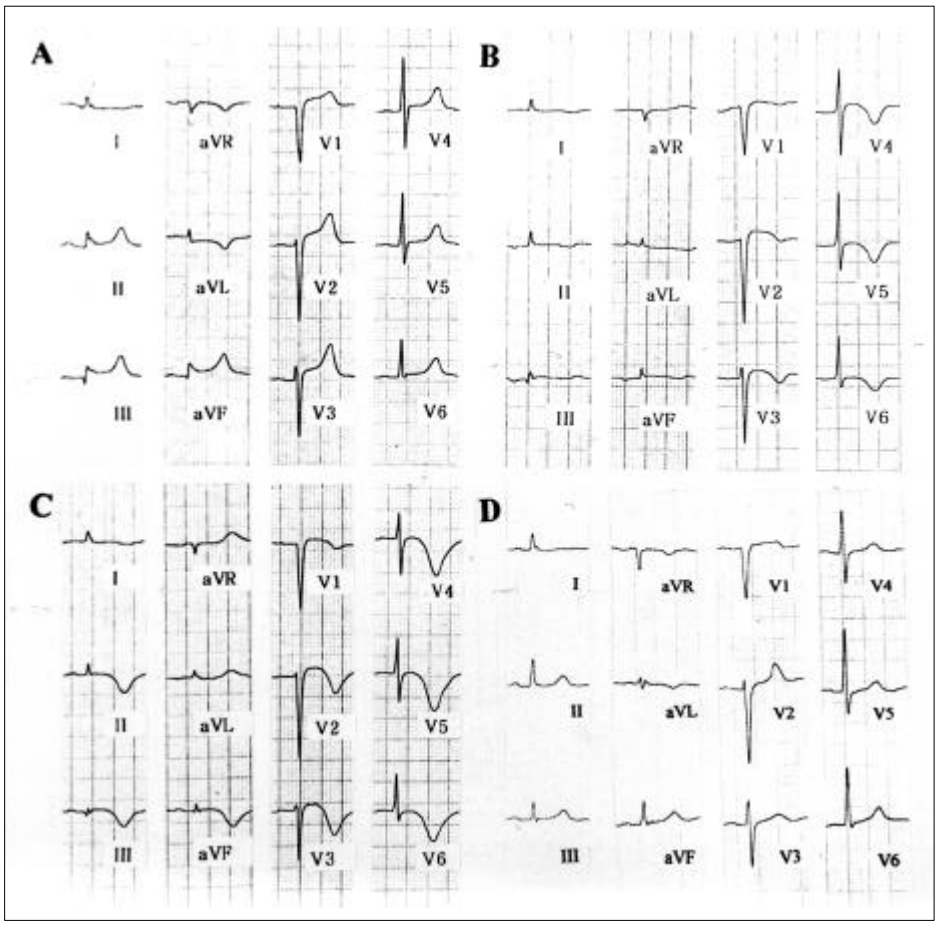

Fig. 1. (A) Electrocardiogram on arrival, showing horizontal ST segment elevation in leads II, III, and aVF, mimicking acute inferior wall infarction. (B) Electrocardiogram after 6 hours showing newly-appeared T wave inversion in precordial leads with normalization of ST segment elevation in leads II, III, and aVF. (C) Electrocardiogram after 1 day showing globally inverted T waves. (D) Electrocardiogram after 4 months. Repolarization patterns have gradually changed into the previous normal form.

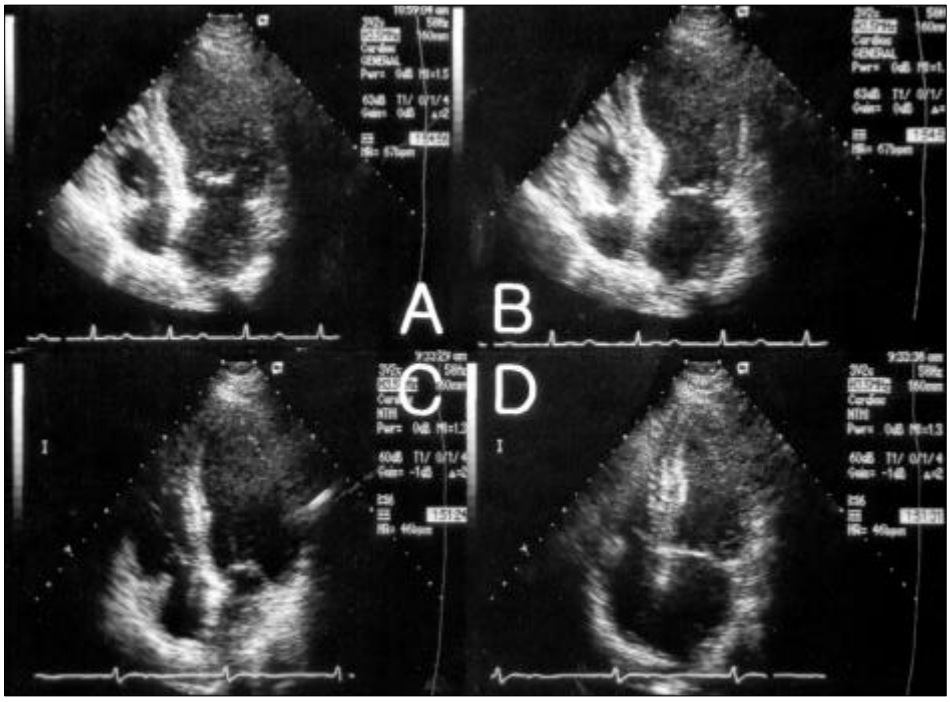

Fig. 2. Echocardiograms during diastole (A) and systole (B) on arrival. They show impaired contractility sparing the basal segments. Echocardiograms during diastole (C) and systole (D) after 4 months, which show normal contractility with no regional wall motion abnormality.

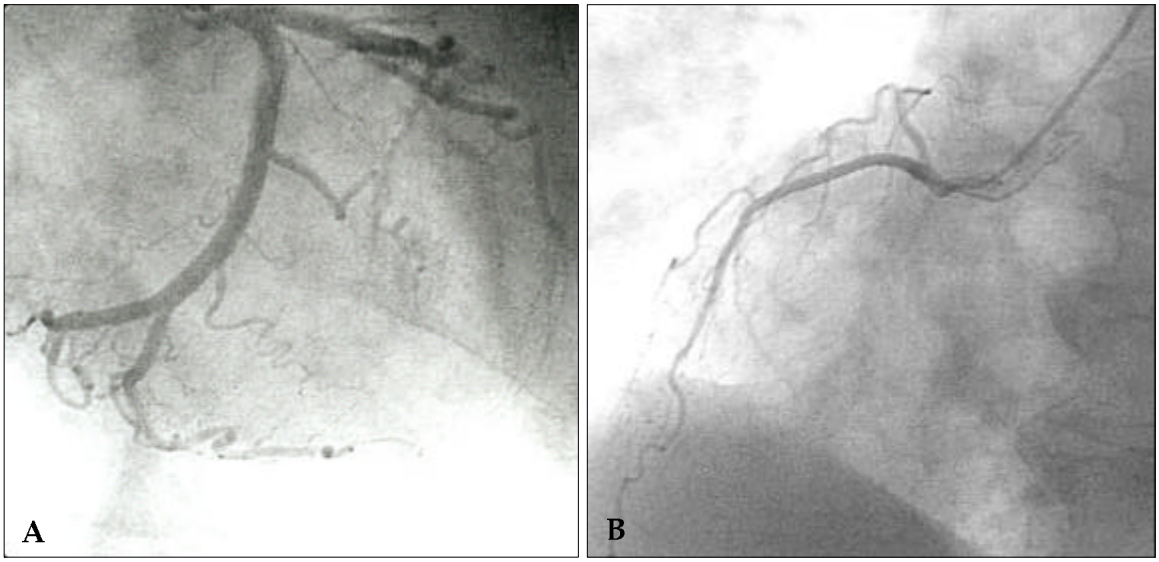

Fig. 3. (A) Left anterior oblique view of coronary angiographic findings at arrival, showing normal findings. (B) Right anterior oblique view of coronary angiographic findings at arrival, showing normal findings.

exposure. Triggering factors such as emotional exposure and mental stress are known to be important contributors, since they have been shown to increase heart rate, blood pressure, and myocardial oxygen demand mediated at least in part by catecholamine secretion.[4] We speculate that the acute stress of an abrupt tax surveillance led to a catecholamine surge in our patient. In fact, the myocardial toxicity of high concentration endogenous or exogenous catecholamines has been demonstrated to be responsible for acute cardiomyopathy.[2]

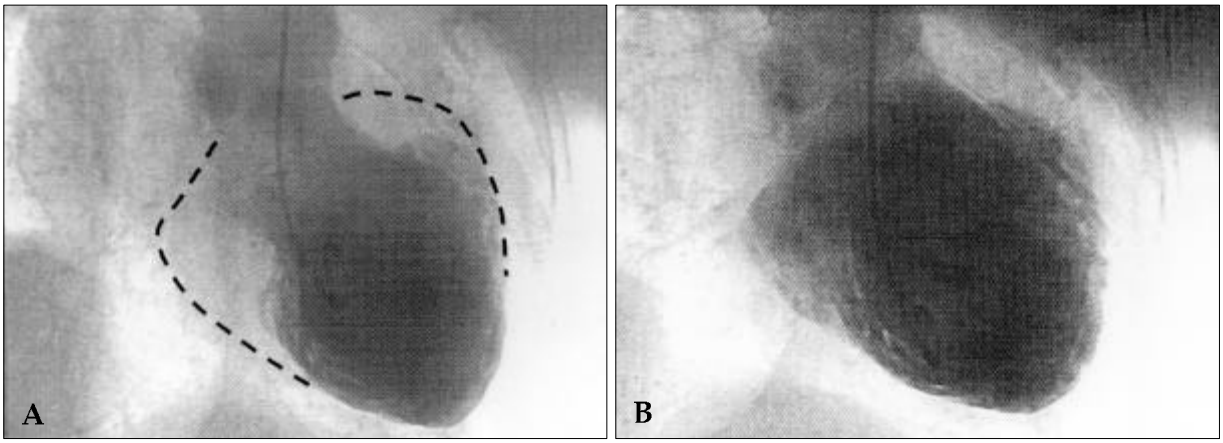

Fig. 4. (A) Left ventriculogram during systole showing impairment of the left ventricular contractility sparing the basal segments. The outline of the left ventricular cavity during diastole is marked with dashed lines in the figure. (B) Left ventriculogram during diastole showing no ventricular dilatation. The ejection fraction was about 30% and akinetic wall motions were observed in the anterolateral, apical, and inferior regions.

The marked impairment of the left ventricular contractility sparing the basal segments mimicked extensive apical myocardial infarction in our patient, but the possibility of this being the correct diagnosis is low due to the following reasons. First, there must be an extremely high cardiac enzymatic elevation in such extensive acute myocardial infarction, whereas in our patient there was only a minimal elevation which returned to normal within a few days. Second, the regional wall motion abnormalities of our patient couldn't be explained by any territory of the three coronary arteries. Third, coronary angiographic findings were normal without any luminal irregularities. Fourth, all the symptoms and abnormalities in this patient occurred after a single, definite emotional upset. Fifth, although she received only minor conservative management during her admission, the regional wall motion abnormality returned to normal rapidly and completely. Sixth, although she received no further medication such as calcium channel antagonist or nitrate after her discharge, there was no evidence of recurrence during follow up of more than a year.

Although it is still uncertain why the left ventricular apex is selectively vulnerable and subsequently forms balloons in acute stress-induced cardiomyopathy, it may be explained by the fact that excessive catecholamine surge induces coronary vasoconstriction that leads to hypoxia, and that myocardial lesions resulting from the hypoxia occurs predominantly at an area of distal coronary vascularization, i.e., the left ventricle apex.[2] Besides, Tsuchihashi et al. have described three other anatomical and physiologic factors that might contribute to left ventricular apical wall motion abnormalities; the absence of a three-layered myocardial structure in the left ventricular apex, the easy loss of elasticity after excessive expansion in the left ventricular apex since it is the border zone of the perfusion area of major coronary arteries, and the delayed functional recovery from global dysfunction in the left ventricular apex.[3] The reversibility of left ventricular apical lesions is related to the shortness of myocardial exposure to excessive catecholamines, resulting in a state of stunning with cellular metabolic abnormalities rather than necrosis.[2]

The changes of electrocardiograms in stress-induced cardiomyopathy are also reversible and hence frequently mistaken for acute coronary syndromes. They affect repolarization with T wave flattening, lengthening of the QT interval and even ST segment elevation as in acute myocardial infarction. In our patient, the electrocardiogram on arrival showed horizontal ST segment elevation in leads II, III, and aVF mimicking acute inferior wall infarction. However, the electrocardiogram 6 hours later showed newly-appeared T wave inversion in precordial leads with normalization of the ST segment elevation in leads II, III, and aVF. The electrocardiogram after

1 day showed globally inverted T waves, which we speculate were due to the effects of catecholamines.[5] Finally, the electrocardiogram after 4 months had gradually changed into the previous normal form.

The prognosis of stress-induced cardiomyopathy is excellent. Heart condition improves very rapidly without any specific treatment and ventricular function returns to normal within several weeks.[6] Normal wall motion returns when the moderately hypokinetic area has disappeared and the left ventricular ejection fraction has improved to more than 60%.[7] Lee et al. analyzed 13 patients prospectively and reported that the ejection fraction recovered within 6.3 ± 5 days. Most of their patients revealed favorable clinical course, in complete contrast to that of acute myocardial infarction.[8] The absence of angiographically significant coronary artery disease is considered as another unique feature which predicts good prognosis, as opposed to acute myocardial infarction.

Although the exact etiology remained unknown, we could conclude that stress-induced cardiomyopathy that occurs under the influence of intense emotional stress is of clinical importance because it mimics acute myocardial infarction. Clinicians should be aware of this unusual combination of symptoms: acute cardiomyopathy presenting as sudden onset of chest pain immediately following emotional upset, major impairment in left ventricular contractility sparing the basal segments without any ventricular dilatation, diffuse reversible changes in ECG measurements, and normal coronary angiographic findings. More data in the form of clinical and experimental studies may be necessary for the identification of its pathogenesis.

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