

A Case of Stress-Induced Cardiomyopathy Related with the Postpartum Period

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ABSTRACT

Stress-induced cardiomyopathy is a relatively rare, unique entity that has only recently been widely appreciated. It is characterized by transient left ventricular regional wall motion abnormalities (with a peculiar apical ballooning appearance), chest pain or dyspnea, ST-segment elevation and/or T wave inversion and minor elevations of the cardiac enzyme levels. The patients in the previous series were usually women over 50 years of age and a triggering event was identified in most cases; these included severe emotional distress or an acute medical illness. Although reports of single episodes of stress-induced cardiomyopathy are not infrequent in the recent medical literature, we report here on a case of stress-induced cardiomyopathy in a young woman, and this was related with the postpartum period as a stressful condition. (**Korean Circulation J 2007;37:388–392**)

KEY WORDS : Cardiomyopathies ; Stress ; Postpartum period.

Introduction

Recent reports have described left ventricular dysfunction that resembles acute myocardial infarction, but these cases have displayed normal coronary arteries and a shape resembling a takotsubo (a Japanese pot for fishing for octopus) on the left ventriculography.^{1,2)} This transient entity is typically precipitated by acute emotional stress or physical stress. Stress-induced cardiomyopathy is also called “takotsubo cardiomyopathy” or “broken-heart syndrome.”³⁾ However, its origin and the detailed clinical features remain unknown. To the best of our knowledge, the present report may be the first case of a stress-induced cardiomyopathy in a young Korean woman and this illness was related with her postpartum period.

Case

A 29-year-old female with a history of her first normal

spontaneous vaginal delivery 10 days previously was hospitalized because of sudden onset chest pain. She had no previous medical problems and no significant family history. One hour before admission, she developed severe chest pain and called the emergency medical service (EMS); she visited a primary clinic and was checked with an electrocardiogram (ECG). It showed normal sinus rhythm and a normal QT interval (Fig. 1A). She was immediately referred to our hospital due to sustained chest pain, but she collapsed during transportation. When the EMS arrived at the emergency department, she was found to be in ventricular fibrillation (Fig. 1B); the patient was then defibrillated by the EMS team (Fig. 1C). On admission to the hospital, she had a pulse rate of 152 beats/min, a blood pressure of 100/70 mmHg and a respiratory rate of 24 breaths/min. Her physical examination was essentially normal. A chest X-ray demonstrated no abnormality. The complete blood count was within normal limits, and the electrolytes and thyroid function tests were also within normal limits. The cardiac enzymes were elevated with a troponin-T level of 2.76 ng/mL (normal: 0.00-0.10 ng/mL), and a CK-MB level of 51.38 ng/mL (normal: 0-5 ng/mL). The serum brain natriuretic peptide concentration was elevated to 1,055 pg/mL and the serum norepinephrine was 195.7 pg/mL (normal: 0-600 pg/mL). Post-resuscitation ECG showed sinus tachycardia with

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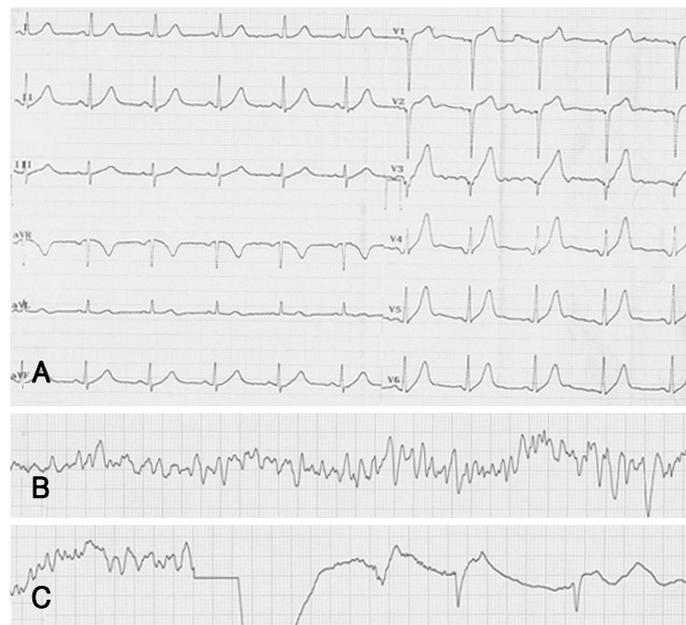


Fig. 1. Electrocardiogram in the primary clinic and the EMS ambulance before admission. A: the twelve-lead electrocardiogram done at the primary clinic revealed a normal sinus rhythm and a normal QT interval (corrected QT interval: 0.430 second). B: the rhythm strip demonstrating ventricular fibrillation. C: the rhythm strip taken during defibrillation by the EMS team. EMS: emergency medical service.

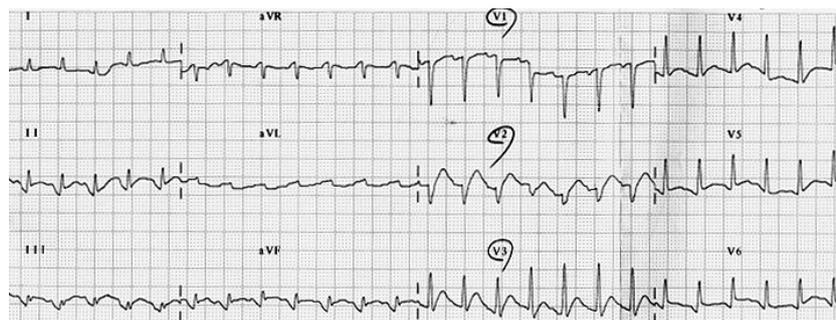


Fig. 2. The twelve-lead electrocardiogram obtained upon admission (post-resuscitation) depicts mild ST segment elevation in the II, III and aVF leads and in the V1-V6 leads.

mild ST-segment elevation in the II, III and aVF leads and the V1-V6 leads (Fig. 2). The presentation was clinically consistent with acute coronary disease. A pre-procedural transthoracic echocardiogram revealed regional systolic dysfunction of the left ventricular walls with hypokinesia of the mid-apical segments and hyperkinesia of the basal segments with an ejection fraction of 38% (Fig. 3). She underwent cardiac catheterization, which revealed normal coronary arteries, but spasm of the left anterior descending artery was induced by ergonovine-provoked testing; she was without chest pain or ST segment change on the ECG (Fig. 4). Left ventriculography showed akinesis of the left ventricular apex and at the mid-portion too (Fig. 5). The remainder of the myocardium contracted normally. There was an area of anterior and apical akinesis, systolic ballooning of the apex and hypercontraction of the basal segment in the configuration of a takotsubo. Computed tomography of the brain revealed no abnormalities. She was managed conserva-

tively and her symptoms resolved. A subsequent transthoracic echocardiogram performed 2 weeks later revealed resolution of the wall motion abnormalities (Fig. 3). The patient was discharged home following symptomatic improvement.

Discussion

Transient left ventricular apical ballooning syndrome is a cardiac syndrome that's characterized by transient LV dysfunction, electrocardiographic changes that can mimic acute myocardial infarction and minimal release of myocardial enzymes in the absence of obstructive coronary artery disease.⁴⁾ Although the exact cause of the syndrome remains unknown, many underlying mechanisms have been proposed, including diffuse epicardial arteries spasm, coronary microcirculation dysfunction, catecholamines-induced myocardial dysfunction and neurologically mediated myocardial stunning.^{5,6)} Acute

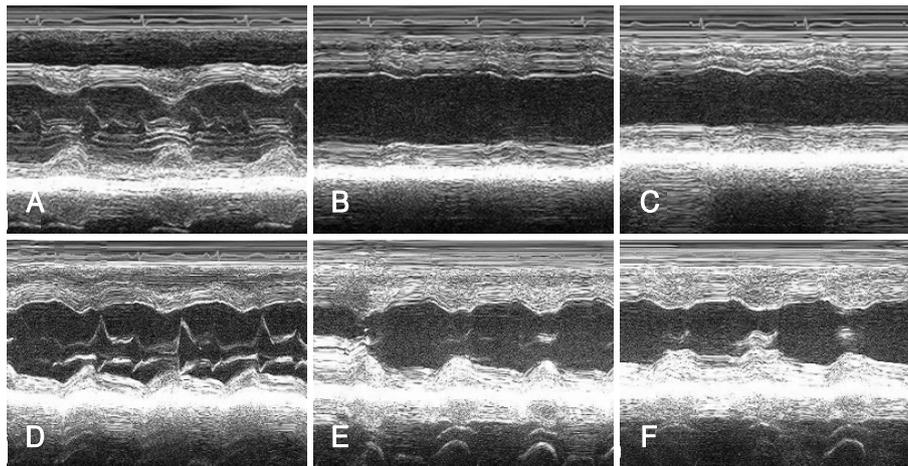


Fig. 3. Echocardiography depicts the reversible apical wall motion abnormality. A: the echocardiogram obtained upon admission depicts hyperkinesis of the basal segments, which was observed via the M-mode. B: hypokinesis of the mid-portion of the left ventricle was observed. C: hypokinesis of the apex was observed. D: echocardiograms taken 14 days after admission showed no interval change of the basal segment. E: improvement of the mid-ventricular wall motion was observed on the follow up echocardiograms. F: improvement of the apical wall motion is demonstrated.

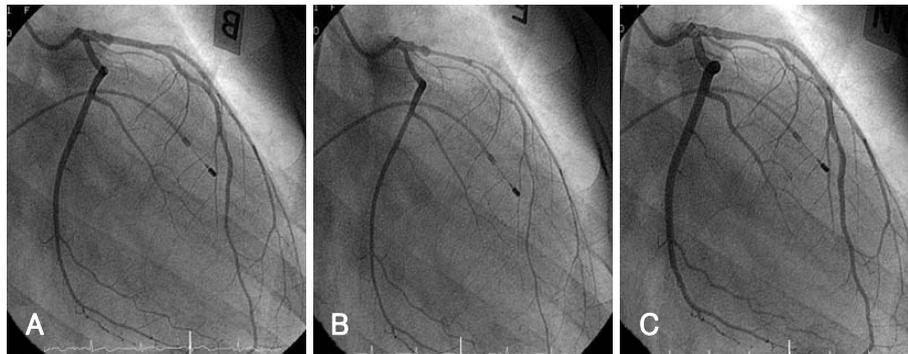


Fig. 4. A: the left coronary artery in the cranial right anterior oblique and caudal views showing the coronary arteries with no obstructive lesions. B: a spasm provoked by ergonovine was observed in the left anterior descending artery. C: it was relieved by nitroglycerin.

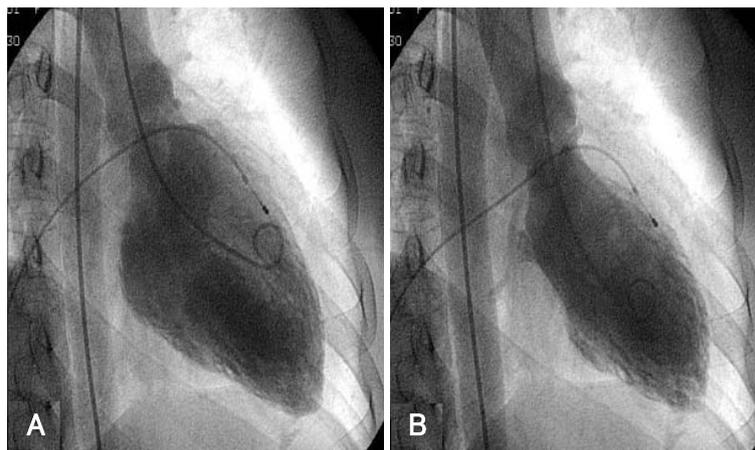


Fig. 5. Representative left ventriculogram from the patient: images obtained in diastole (A) and systole (B). Left ventriculography revealed akinesis of the left ventricular apex and the mid-portion.

stress has been indicated as a common trigger for transient LV apical ballooning syndrome.⁵⁾⁷⁾ Several investigators have noticed that either emotional or physical stress often precedes left ventricular apical ballooning.⁸⁾⁹⁾ The cited examples include emotional stress associated

with a sudden accident, death/funeral of a family member, quarreling or severe anxiety. Several cases have been associated with underlying medical disorders such as pheochromocytoma, subarachnoid hemorrhage, exacerbation of bronchial asthma, noncardiac surgery, sepsis

and critical illnesses in patients.¹⁰⁾ The most common clinical presentations are chest pain and dyspnea, and these symptoms have been reported in 67.8% and 17.8% of the patients, respectively.⁴⁾ Cardiogenic shock(4.2% of the patients) and ventricular fibrillation(1.5%) have also reported.⁴⁾ Patients with stress-induced cardiomyopathy have clinical characteristics that are consistent with prior Japanese reports. To summarize those reports, the patients were generally female(81-93%) with an average age between 50 and 70 years.²⁾¹¹⁾¹²⁾ The clinical features observed in the present patient were consistent with those observed in the previous studies,¹⁾²⁾¹¹⁾¹³⁾ i.e., ST elevation, minimal myocardial enzymatic release and characteristic left ventricular apical asynergy with the absence of coronary artery stenosis, which are all typical of stress-induced cardiomyopathy. We supposed that the postpartum period was the emotional or physical stress that preceded the symptoms in our case. Abnormal Q waves following ST-segment elevation have often been reported as the typical electrocardiographic changes that occur during the acute phase in patients with stress-induced cardiomyopathy.¹⁾²⁾¹¹⁾¹³⁾ The value of the serum brain natriuretic peptide concentration (1,055 pg/mL) was very high in our case, suggesting congestive heart failure.¹⁴⁾ The value of the serum norepinephrine(195.7 pg/mL) was within the normal limits. Direct myocardial damage induced by catecholamine has been suggested to be one of the causes of stress-induced cardiomyopathy.¹⁵⁾ but this was unlikely in our case. Some cases of stress-induced cardiomyopathy have been reported to reveal coronary spasm.¹⁾²⁾¹¹⁾¹³⁾ Coronary spasm provocation testing should have been performed during the coronary angiography in this present case. A potentially more attractive hypothesis, in our judgment, is that transient apical akinesis results from coronary vasospasm. Kurisu et al.²⁾ prospectively studied 30 patients with takotsubo-like LV dysfunction, and most of whom had no obstructive coronary disease. These patients underwent provocative testing with acetylcholine or ergonovine. Ten percent of the patients had spontaneous coronary vasospasm with catheter engagement and 67% had spasm during the acute period when provoked by acetylcholine or ergonovine. These investigators noted that one patient had no evidence of epicardial coronary artery spasm, but delayed contrast filling was noted in the right coronary artery, implying microvascular dysfunction. Other investigators have implicated microcirculation dysfunction in these patients.¹⁶⁾ Recurrence of this syndrome is rare and this has been infrequently reported.¹⁷⁾ A recurrence rate of 2.7% has been reported in the largest series thus far, and this series consisted of 88 patients.⁵⁾ For the cases of recurrence, one may cautiously speculate that perhaps there is a genetic predisposition towards developing such a reversible syndrome. Of note, Japanese investigators have detected CD 36 deficiency in a patient

who developed stress-induced cardiomyopathy, suggesting that patients with certain genetic profiles may be more susceptible.⁸⁾

In conclusion, the present case shows the serious clinical course of stress-induced cardiomyopathy in which the left ventricular dysfunction of apical and mid-ventricular hypokinesis continued for about 2 weeks. As discussed above, most of these patients have generally been women with a mean age at presentation of approximately 50-70 years.⁵⁾ Our patient was young and the prognosis was generally thought to be good, yet it is important to recognize that there may be cases of stress-induced cardiomyopathy in young woman that are especially related with the postpartum period.

REFERENCES

- 1) Kawai S, Suzuki H, Yamaguchi H, et al. *Ampulla cardiomyopathy (takotsubo cardiomyopathy): reversible left ventricular dysfunction with ST segment elevation. Jpn Circ J* 2000;64:156-9.
- 2) Kurisu S, Sato H, Kawagoe T, et al. *Takotsubo like left ventricular dysfunction with ST-segment elevation: a novel cardiac syndrome mimicking acute myocardial infarction. Am Heart J* 2002;143:448-55.
- 3) Virani SS, Khan AN, Mendoza CE, Ferreira AC, de Marchena E. *Takotsubo cardiomyopathy, or broken heart syndrome. Tex Heart Inst J* 2007;34:76-9.
- 4) Gianni M, Dentali F, Grandi AM, Sumner G, Hiralal R, Lonn E. *Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review. Eur Heart J* 2006;27:1523-9.
- 5) Bybee KA, Kara T, Prasad A, et al. *Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. Ann Intern Med* 2004;141:858-65.
- 6) Lee HH, Gwon HC, Kim BJ, et al. *Clinical manifestation of novel stress-induced cardiomyopathy mimicking acute myocardial infarction: single center prospective registry. Korean Circ J* 2002;32:1054-63.
- 7) Sato M, Fujita S, Saito S, et al. *Increased incidence of transient left ventricular apical ballooning (so-called "Takotsubo" cardiomyopathy) after the mid-Niigata Prefecture earthquake. Circ J* 2006;70:947-53.
- 8) Cherian J, Angelis D, Filiberti A, Saperia G. *Recurrence of stress-induced (takotsubo) cardiomyopathy. Cardiology* 2006;108:144-6.
- 9) Chang KY, Jeon HK, Chae JS, et al. *A novel cardiomyopathy mimicking acute myocardial infarction. Korean Circ J* 2002;32:608-12.
- 10) Park JH, Kang SJ, Song JK, et al. *Left ventricular apical ballooning due to severe physical stress in patients admitted to the medical ICU. Chest* 2005;128:296-302.
- 11) Tsuchihashi K, Ueshima K, Uchida T, et al. *Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. J Am Coll Cardiol* 2001;38:11-8.
- 12) Sharkey SW, Shear W, Hodges M, Herzog CA. *Reversible myocardial contraction abnormalities in patients with an acute non-cardiac illness. Chest* 1998;114:98-105.
- 13) Yamasa T, Ikeda S, Ninomiya A, et al. *Characteristic clinical findings of reversible left ventricular dysfunction. Intern Med* 2002;41:789-92.
- 14) Mukoyama M, Nakao K, Saito Y, et al. *Human brain natriuretic*

peptide: a novel cardiac hormone. Lancet 1990;335:801-2.

- 15) Akashi YJ, Nakazawa K, Sakakibara M, Miyake F, Sasaka K. Reversible left ventricular dysfunction "takotsubo" cardiomyopathy related to catecholamine cardiotoxicity. *J Electrocardiol* 2002;35:351-6.
- 16) Ako J, Kozaki K, Yoshizumi M, Ouchi Y. Transient left ventricular apical ballooning without coronary artery stenosis: a form of stunning-like phenomenon? *J Am Coll Cardiol* 2002;39:741-2.
- 17) Kushiro T, Saito F, Kusama J, et al. Takotsubo shaped cardiomyopathy with type I CD36 deficiency. *Heart Vessels* 2005;20:123-5.