CASE REPORT

Reversible T-Wave Inversions and Neurogenic Myocardial Stunning in a Patient with Recurrent Stress-Induced Cardiomyopathy

Yasushi Akutsu, M.D., Ph.D.,^{*,‡} Kyouichi Kaneko, M.D., Ph.D.,^{*}
Yusuke Kodama, M.D., Ph.D.,^{*} Hui-Ling Li, M.D., Ph.D.,^{*,‡} Jumpei Suyama, M.D.,
Ph.D.,[†] Tsutomu Toshida, M.D., Ph.D.,^{*} Hiroyuki Kayano, M.D., Ph.D.,^{*}
Akira Shinozuka, M.D., Ph.D.,[†] Takehiko Gokan, M.D., Ph.D.,[†]
and Youichi Kobayashi, M.D., Ph.D.^{*}

From the ^{*}Division of Cardiology, Department of Medicine; [†]Department of Radiology, Showa University School of Medicine, Tokyo, Japan; and [‡]Department of Internal Medicine (Cardiology), Clinical Trial Center for Clinical Pharmacology, Showa University Karasuyama Hospital, Tokyo, Japan

A 72-year-old female was diagnosed as a stress-induced cardiomyopathy from apical ballooning pattern of left ventricular dysfunction without coronary artery stenosis after the mental stress. ECG showed the transient T-wave inversions after the ST-segment elevations. By the mental stress after 1 year, she showed a transient dysfunction with similar ECG changes again. T-wave inversions recovered earlier, and cardiac sympathetic dysfunction showed a lighter response corresponding to the less severe dysfunction than those after the first onset. Wellens' ECG pattern was associated with the degree of neurogenic myocardial stunning with sympathetic hyperinnervation caused by mental stress.

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Wellens' syndrome is characterized by dynamic symmetric T-wave inversion in the precordial leads in patients with acute coronary syndrome caused by the subocclusion of the left anterior descending coronary artery.¹ The development of transient, prominent inverted T waves in the precordial leads after an acute coronary event has been interpreted as an ECG marker of ischemia-induced, reversible myocardial dysfunction. Recently, this ECG pattern has been described in patients with stress-induced cardiomyopathy who have no stenosis of coronary artery although the pathophysiological basis of this ECG pattern remains to be elucidated.² We report a relationship between Wellens' ECG pattern and cardiac sympathetic nervous system

activity in a patient with recurrent stress-induced cardiomyopathy

CASE REPORT

A 72-year-old female who was presented with dyspnea after the earthquake showed the electrocardiographic ST-segment elevations in all leads excluding an aV_R (Fig. 1). The left ventricular wall motion showed an apical ballooning pattern without coronary artery stenosis (Fig. 2A). The serum concentrations of nor-adrenaline and dopamine were high (0.74 ng/mL and 0.03 ng/mL), and the creatinine phosphokinase (CK), CK myocardial

Address for correspondence: Yasushi Akutsu, M.D., Ph.D., Division of Cardiology, Department of Medicine, Showa University School of Medicine, 1–5–8 Hatanodai, Shinagawaku, Tokyo 142–8666, Japan. Phone: +81-3-3784-8539; Fax: +81-3-3784-8622; E-mail: hzn01233@s02.itscom.net

Figure 1. The course of electrocardiographic findings after the first and second onsets of stress-induced cardiomyopathy.

band (MB), and troponin I were increased (392 IU/L, 31.1 ng/mL, and 5.2 ng/mL). The 4-hour delayed phase scintigraphic images after the injection of iodine-123 metaiodobenzylguanidine (^{123}I -MIBG) showed the severe sympathetic dysfunction in the apex to inferior wall at 1 week and 2 months (Fig. 2C). The reduced apical wall motion recovered at 1 week (Fig. 2B). The ECG was changed to invert T waves in all leads excluding aV_R , and the inverted T waves recovered at 6 months completely (Fig. 1). After 1 year of the first onset, she was presented with dyspnea again by the intolerable mental stress, and showed the electrocardiographic ST-segment elevations in leads of I, II, III, aV_L , aV_F , and V_{1-4} similarly. The CK, CKMB, and troponin I were not so increased as the values after the first onset (161 IU/L, 12.5 ng/mL, and 0.07 ng/mL). The left ventricular wall motion showed a transient reduction of the apex to anteroapical wall motion without a typical takotsubo pattern (Fig. 2B), and ^{123}I -MIBG scintigraphy showed a light sympathetic dysfunction in the apex at 1 week and 2 months (Fig. 2C). The reduced wall motion recovered quickly. The ECG was changed to invert T waves, and the inverted T waves recovered at 2 months (Fig. 1).

DISCUSSION

Stress-induced cardiomyopathy (Takotsubo cardiomyopathy) can be a serious but reversible cause of acute myocardial dysfunction immediately after a trigger of mental stress such as an earthquake, and less than 5% of the patients have a recurrence within 1 year.³ The apical-basal gradients of beta-adrenergic receptors and sympathetic hyperinnervation, and the myocardial response of catecholamine play the potential role in the pathophysiology of stress-induced cardiomyopathy.⁴ On the condition of sympathetic hyperinnervation, the increased spillover of norepinephrine from the sympathetic nervous system in the skeletal muscle and heart leads to the increased plasma concentration and depleted stores in the heart. Therefore, using the radiotracer ^{123}I -MIBG which is taken up into presynaptic cardiac sympathetic nerves, the cardiac sympathetic dysfunction as a low uptake of ^{123}I -MIBG, that is to say the neurogenic myocardial stunning, has been reported in patients with stress-induced cardiomyopathy.⁵ In a patient with recurrent stress-induced cardiomyopathy, the ^{123}I -MIBG imaging demonstrates a lighter response of cardiac sympathetic nervous system to the mental stress corresponding to the less severe cardiac

Figure 2. A. The left ventriculography with an apical ballooning pattern and normal coronary arteries after the first onset of stress-induced cardiomyopathy. B. The course of echocardiographic findings after the first and second onsets of stress-induced cardiomyopathy. C. The course of polar maps on 4-hour delayed iodine-123 metaiodobenzylguanidine tomographic imaging after the first and second onsets of stress-induced cardiomyopathy.

dysfunction than those of the first onset. More important was that the number of ECG leads with inverted T waves, the maximal amplitude of T-wave inversion, and the time course of recovery from the T-wave inversions were associated with the degree of neuronal myocardial stunning with sympathetic hyperinnervation caused by mental stress.

Marra et al.⁶ found the significant linear correlations between the apicobasal ratio of myocardial edema estimated by T₂-weighted signal intensity on magnetic resonance imaging and the maximal amplitude of T-wave inversion and sum of the amplitudes of T-wave inversion. They concluded that the apicobasal gradient of myocardial edema is a cause of diffuse negative T waves observed in patients with apical ballooning. Myocardial edema is usually present in the regions with abnormal systolic function, and is associated with negative T waves. Therefore, when the edema disappears, the inverted T waves would be

normalized. The degree of neurogenic myocardial stunning induced by sympathetic hyperinnervation regulates the extent of transient myocardial edema, which may influence the process of T-wave inversions.

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