Transient mid-ventricular ballooning syndrome is a variant form of Takotsubo cardiomyopathy (TTC). The precipitating factors and clinical course are similar, and the clinical manifestations mimic those of acute coronary syndrome, which are characterized by transient left ventricular dysfunction without significant coronary artery disease. Takotsubo cardiomyopathy with transient left bundle branch block (LBBB) is rare. Herein, we describe the case of a 64-year-old woman who had a transient mid-ventricular apical ballooning syndrome. Electrocardiography (ECG) revealed a transient LBBB.

Key Words: Mid-ventricular ballooning • Takotsubo • Transient left bundle branch block

INTRODUCTION

Takotsubo cardiomyopathy (TTC), also known as transient left ventricular apical ballooning syndrome, was first described in the Japanese literature in 1991 by Dote et al.1 TTC accounts for 0.7 to 2.5% of admissions for suspected acute coronary syndrome.2 Transient mid-ventricular ballooning syndrome is a variant form of TTC. The precipitating factors and clinical course of the nonapical type are mostly similar to those of the apical type.3,4 However, TTC has been rarely reported in Taiwan.4-7

The syndrome is characterized by the abrupt onset of angina-like chest pain, mild creatine kinase and troponin I elevations, ischemic electrocardiogram (ECG) changes and transient wall motion abnormalities in the absence of obstructive epicardial coronary artery disease. The clinical presentation mimics acute myocardial infarction but without evidence of coronary artery stenosis on angiography, and the left ventricular (LV) dysfunction can completely regress.

The main electrocardiographic finding at presentation is ST-segment elevation followed by T-wave inversion. In a minority of cases, left bundle branch block (LBBB) may be the ECG pattern of TTC presentation.8 We herein describe such a case with transient mid-ventricular ballooning syndrome and transient LBBB at presentation.

CASE REPORT

A 64-year-old woman with a history of type 2 diabetes mellitus was brought to our emergency room with severe dyspnea for 2 hours. She had developed chest tightness and dyspnea for 5 days after receiving news of her son’s death. Her blood pressure was 148/87 mmHg, pulse 98 beats per minute, respirations 22 per minute, and temperature 36 °C. The heart rhythm was regular, and other physical examinations were unremarkable. Her chest roentgenogram showed mild cardiomegaly with pulmonary edema. The ECG showed sinus rhythm with
left bundle branch block pattern (Figure 1A). The patient’s creatine kinase and creatine kinase, muscle and brain (CKMB) levels were 132 and 2.8 IU/L respectively, and troponin I 0.318 ng/ml (normal range < 0.5 ng/ml). The white-cell count was 8430 per cubic millimeter, hematocrit 42%, blood sugar 241 mg/dl, and the prothrombin and partial-thromboplastin times and the values for nitrogen, creatine, and electrolytes were normal. Intravenous diuretics and supplemental oxygen were administered, and the patient’s dyspnea improved a little. A cardiologic consult was obtained, and echocardiography showed hypokinesis over the mid-portion of the LV and an ejection fraction of 38%. A cardiac catheterization was done immediately and revealed no evidence of coronary artery stenosis (Figures 2A, B). The contrast left ventriculography demonstrated hypokinesis over the mid portion of the anterior wall and normal contractility of the basal and apical portions (Figures 2C, D). No LV outflow pressure gradient was apparent. The patient was diagnosed with stress cardiomyopathy. Follow-up creatine kinase and CKMB levels were 100 and 3 IU/L respectively, and troponin I was measured 0.183 ng/ml 12 hrs later. A repeat ECG performed 2 days later (Figure 1B) demonstrated resolution of the LBBB, and the appearance of T-wave inversion in I, aVL, and V3-6 leads. The patient remained stable and was transferred to the general ward on admission day 3. She was discharged on admission day 4 and remained well without chest pain or dyspnea. Two weeks later, repeat echocardiography showed complete resolution of the LV wall motion abnormalities, and the ejection fraction had improved to 65%.

DISCUSSION

One review of relevant studies has suggested that TTC accounts for 0.7 to 2.5% of patients presenting with a suspected ACS. Over the last decade, there have been many articles published on the topic of TTC. A modified version of the Mayo Clinical Criteria for the clinical diagnosis of transient LV apical ballooning syndrome has been made.9 This includes (1) transient hypokinesis, akinesis, or dyskinesis of the LV and mid-segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; (2) absence of significant coronary stenosis (more than 50% of the luminal diameter) or angiographic evidence of acute plaque rupture; (3) new ECG abnormalities (ST-segment elevation and/or T-wave inversion); and (4) absence of pheochromocytoma or myocarditis.

Due to its clinical characteristics, TTC is frequently misdiagnosed as acute coronary syndrome (ACS), or myocarditis. Since the ECG and symptoms cannot reliably distinguish TTC from ACS, identification of TTC requires diligence on the part of the diagnosing physician and sound clinical judgment. Differentiating from TTC from ACS is important, because of different treatment strategies and prognoses. If TTC is suspected, fibrinolytic therapy should be avoided and emergency coronary angiography should be performed in any patient presenting with ST-segment elevation. Furthermore, patients with ACS benefit from secondary preventive pharmacotherapy, whereas in patients with TTC, the LV wall motion abnormalities usually resolve without any specific treatment. Cardiac magnetic resonance seems to be a useful technique to differentiate TTC (characterized by the absence of delayed gadolinium hyperenhancement) from myocardial infarction and myocarditis, in which delayed hyperenhancement is present. Therefore, cardiac magnetic resonance can add valuable information for all patients with suspected TTC for further differential diagnosis and guidance of medical therapy.10

The underlying pathophysiology, which is often preceded by a recent stress inducing event (physical or emotional) in many cases, is still not well understood. However, several mechanisms for reversible cardiomyopathy have been proposed, including multivessel epicardial spasm,1 microvascular dysfunction and catecholamine-induced myocardial stunning.2 Several reports have suggested that wall motion abnormalities associated with TTC might not exclusively be located in apical segments, but may also occur in midventricular segments of the LV.3,4,11 Kurowski et al. reported that transient midventricular LV ballooning was observed in 40% of patients with transient LV dysfunction; no differences in demographic, clinical, angiographic, laboratory parameters, or outcome were found between these patients and patients with apical ballooning.3 The presentation, clinical features, and transient nature of the midventricular LV ballooning cases are similar to those of transient LV apical ballooning and
Transient Left Bundle Branch Block

Figure 1. (A) A 12-lead ECG showed left bundle branch block. (B) ECG demonstrating resolution of left bundle branch block with T-wave inversion in leads I, aVL, and V3-6 leads.
suggest a shared pathophysiologic etiology.\textsuperscript{11}

The ECG findings associated with TTC are numerous, but the most common abnormality is ST-segment elevation. Typically, the elevation is present in the precordial leads, but it may be seen in the inferior or lateral leads, nonspecific T-wave abnormality, or new bundle branch block, and in some cases, a normal ECG may be the finding at presentation.\textsuperscript{8} A recent study by Dib et al. showed that new onset of LBBB was found in 1 of 105 patients with TTC.\textsuperscript{8} They also concluded that the ECG features at presentation do not correlate with the magnitude of ventricular dysfunction or long-term outcomes. Thus, the ECG in patients with TTC does not provide significant prognostic information. The presence of new onset of LBBB is associated with adverse outcome in patients with acute myocardial infarction. However, when adjusted for age, baseline characteristics, and concomitant diseases, LBBB does not appear to be an independent predictor of poor outcome in patients with TTC.\textsuperscript{12}

Transient LBBB has not been reported in mid-ventricular ballooning syndrome. Our patient had normal coronary arteries, despite the appearance of LBBB and concomitant chest pain at heart rate of 96 bpm and disappearance of LBBB at heart rate of 78 bpm. This finding indicated that this was not a heart rate-dependent LBBB. A likely explanation for the appearance of LBBB in this case is that the deterioration of the coronary flow reserve resulted in myocardial ischemia. Several studies have suggested that microvascular dysfunction may be a potential cause for stress cardiomyopathy. Reduced coronary flow reserve in Doppler flow-wire measurements, and higher thrombolysis in myocardial infarction (TIMI) frame counts in all three major epicardial coronary vessels after onset of TTC, indicate the presence of impaired

\begin{figure}[h]
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\caption{Coronary angiography (A: left coronary artery; B: right coronary artery). No coronary artery stenosis was seen. A left ventriculogram showed hypokinesis of the mid-portion of the left ventricle and normal contraction of the basal and apical portions (C: end-diastole and D: end-systole).}
\end{figure}

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microvascular flow. Several nuclear studies have been performed recently, and the investigators interpreted the nuclear imaging findings of reduced myocardial perfusion in the absence of obstructive coronary lesions as direct evidence for impaired coronary microcirculation as a causative mechanism of the syndrome.3

CONCLUSION

Although new-onset LBBB is usually associated with myocardial ischemia, this case suggests that TTC should also be considered in the differential diagnosis of patients with acute coronary syndrome and transient LBBB.

REFERENCES