Transient Left Ventricular Apical Ballooning in a Patient With Bicuspid Aortic Valve Created a Left Ventricular Thrombus Leading to Acute Renal Infarction

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A 44-year-old woman had tako-tsubo-like ventricular dysfunction with chest pain and ST segment elevation on the ECG. Echocardiography revealed a bicuspid aortic valve with moderate to severe aortic regurgitation. She developed mild heart failure during the clinical course, but the medication (furosemide, enalapril, and aspirin) had to be stopped because of skin eruptions. Four weeks after ceasing the antiplatelet agent, she was re-admitted with acute renal infarction. Enhanced chest computed tomography revealed a filling defect in the left ventricle and echocardiography showed a high echogenic mass in the left ventricular apical wall. These findings strongly suggested that the renal infarction was caused by an embolism derived from a left ventricular thrombus that formed during the clinical course of the transient left ventricular apical ballooning. Anticoagulation therapy with urokinase and warfarin successfully lysed the thrombus. Left ventricular thrombus should be considered a complication of transient left ventricular apical ballooning, especially in patients with organic heart disease. (Circ J 2004; 68: 1081–1083)

Key Words: Bicuspid aortic valve; Left ventricular thrombus; Renal infarction; Tako-tsubo-like ventricular dysfunction

Discussion

Direct evidence of a LV thrombus associated with tako-tsubo-like ventricular dysfunction has not been demonstrated, although there have been reports regarding embolic complications of this disorder. Visser et al reported that akinetic LV wall in the setting of myocardial infarction is...
Fig 1. Left ventriculography indicates apical akinesis with ballooning that does not correspond to the perfusion area of any single coronary artery (a, b). Transthoracic echocardiography shows apical ballooning and moderate to severe aortic regurgitation (c) and transesophageal echocardiography shows a horizontal-type bicuspid aortic valve (d).

Fig 2. Acute renal infarction confirmed by abdominal CT (a) and a filling defect in the left ventricle on chest CT (b). Echocardiography also demonstrates a high echogenic mass attached to the left ventricular apical wall (c). Enhanced chest CT and echocardiography (d) after the anticoagulation regimen shows that the left ventricular apical thrombus has disappeared.
an important cause of LV thrombus? Given that the LV thrombus in the present case was caused by a wall motion abnormality, its clinical appearance seems rather late. In a typical case, the wall motion abnormality recovers within 1 month of onset,3,4 so any thromboembolic complications occur in the earlier period. The absence of chest symptoms and ECG abnormalities before and after the onset of renal infarction make a recurrence of the tako-tsubo-like ventricular dysfunction highly unlikely and therefore the precise cause of the late thrombus formation was not clarified in the present patient case.

Embolic complication has been reported in patients with a severely degenerated bicuspid valve accompanied by a thrombus or infected valves.8,9 The present patient did not have a severely degenerated aortic valve and there was no obvious thrombus associated with the aortic valve, so it is unlikely that the bicuspid aortic valve was directly involved in the systemic embolization. We speculate, however, that the presence of aortic regurgitation with the bicuspid valve may have contributed to thrombus formation through turbulent flow, in addition to the delayed recovery of heart failure.

In summary, we report a case of transient LV apical ballooning with LV thrombus and acute renal infarction after cessation of antiplatelet drug therapy. With restoration of LV apical wall motion and warfarin therapy, the LV thrombus disappeared. LV thrombus should be considered not only an early but also a delayed complication of transient LV apical ballooning, especially in a patient with organic heart disease.

References