

Left Ventricular Apical Thrombus Formation in a Patient With Suspected Tako-Tsubo-Like Left Ventricular Dysfunction

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A 74-year-old woman with hypertension and bronchial asthma had chest discomfort at rest and 4 days later was admitted to her nearby hospital because of the sudden onset of right hemiparesis. The hemiparesis had almost disappeared within 24 h of onset, but because an electrocardiogram showed sinus tachycardia and diffuse symmetrical T-wave inversion, she was referred for cardiac examination. Coronary angiography did not reveal any significant coronary artery stenosis, but left ventriculography revealed severe hypokinesis of the left ventricular apical region, which contained a 4×4-mm solid thrombus moving freely with a wavy motion. Moreover, the activity of both protein C and protein S had decreased. The thrombus disappeared after 2 weeks of anticoagulant treatment with warfarin. Her clinical course suggested that the transient cerebral ischemic attack was caused by embolism of the left ventricular thrombus associated with 'tako-tsubo-like left ventricular dysfunction'. (*Circ J* 2003; 67: 556–558)

Key Words: Cerebral ischemia; Protein C; Tako-tsubo; Thrombus

We have reported a novel cardiac syndrome that is characterized by a transient left ventricular apical wall motion abnormality with chest symptoms, electrocardiogram (ECG) changes and minimal myocardial enzymatic release mimicking acute myocardial infarction, but without significant coronary artery stenosis.^{1–4} Because the end-systolic left ventriculogram looked like a tako-tsubo, used in Japan for trapping octopuses, we proposed the name 'tako-tsubo-like left ventricular dysfunction'. Other clinical studies in Japan have reported a predominance of elderly females and a relatively favorable prognosis^{4,5} and several case reports^{5–7} have revealed transient left ventricular outflow obstruction as a complication of tako-tsubo-like left ventricular dysfunction.

We present a patient who developed transient cerebral ischemic attack probably because of a left ventricular apical thrombus that developed as a complication of tako-tsubo-like left ventricular dysfunction.

Case Report

A 74-year-old woman with hypertension and bronchial asthma experienced chest discomfort at rest on November 1, 2000, followed by general fatigue and 4 days later she was admitted to her nearby hospital because of the sudden onset of right hemiparesis. Computed tomography 5 h after onset could not find a lesion responsible for the hemiparesis, the symptoms of which almost disappeared within the next 24 h. However, because the ECG showed sinus tachy-

cardia and diffuse symmetrical T-wave inversion, she was referred for cardiac examination on November 8, 2000.

The patient was fully conscious, her pulse rate was

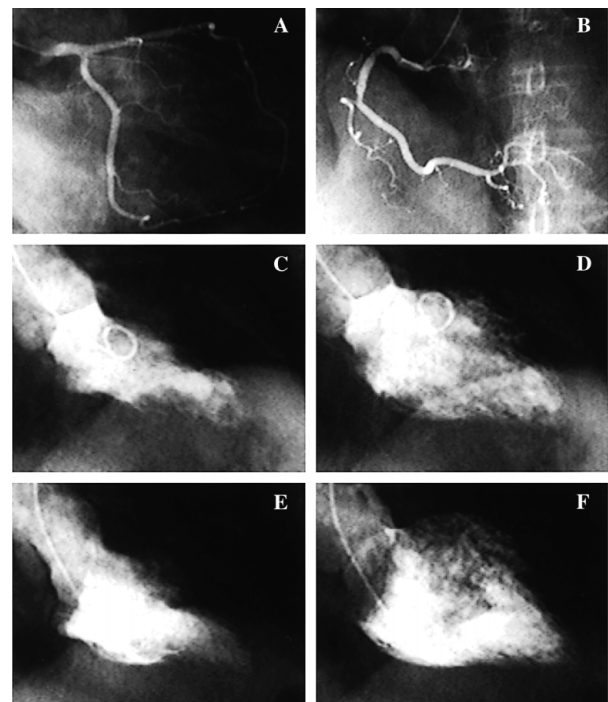


Fig 1. Coronary angiography revealed no significant coronary artery stenosis (A, B). Left ventriculography revealed severe hypokinesis of the left ventricular apical region, which contained a thrombus (C, D) that disappeared 2 weeks later after anticoagulant treatment with warfarin (E, F).

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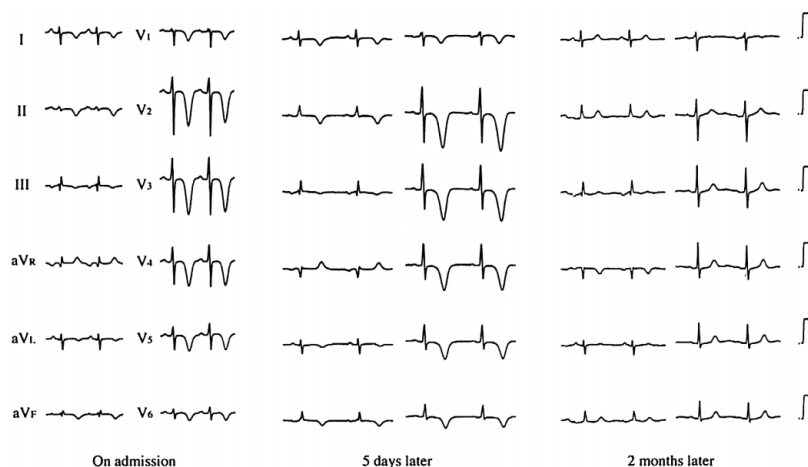


Fig 2. Serial ECG.

104 beats/min, and blood pressure was 120/70 mmHg. The peripheral pulses were normal and she did not have edema. Her neurologic examination was negative. Serum creatine kinase concentration was normal. Echocardiography revealed severe hypokinesis of the left ventricular apical region, which contained a 4×4-mm solid thrombus, but there was no suggestion of hypertrophic or dilated cardiomyopathy. Cardiac catheterization was carefully performed via the left brachial artery to ascertain the cause of the apical asynergy. Coronary angiography could not find significant coronary artery stenosis, but left ventriculography confirmed severe hypokinesis of the left ventricular apical region (ejection fraction: 40%) and a solid thrombus moving freely with a wavy motion. The activity of both protein C (34%; normal: 64–146%) and protein S (27%; normal: 60–150%) was decreased. Anticoagulant treatment with warfarin was initiated and the thrombo test was meticulously kept in the therapeutic range. Calcium antagonist (amlodipine 5 mg) was also added to the treatment because we could not exclude coronary spasm as the cause of the tako-tsubo-like left ventricular dysfunction. Two weeks later, while still receiving warfarin and calcium antagonist, repeated left ventriculography revealed that the thrombus had disappeared and the left ventricular apical asynergy had almost resolved (ejection fraction: 53%). There were no adverse cardiovascular events during hospitalization and she was discharged on the 16th day. Two months later, her ECG had normalized.

Discussion

The present patient, who was an elderly woman, had left ventricular apical asynergy with chest discomfort and diffuse symmetrical T-wave inversion. Although the left ventricular apical asynergy was not typical in part because the left ventriculogram was obtained 7 days after the onset of chest symptoms, her clinical course was consistent with that of tako-tsubo-like left ventricular function, and to the best of our knowledge, this is the first case of a left ventricular apical thrombus associated with the syndrome.

Left ventricular thrombosis can occur with acute myocardial infarction and is associated with increased risk of embolism.^{8,9} Anterior location, impaired ejection fraction ($\leq 35\%$) and apical dyskinesis or aneurysm¹⁰ are related to the appearance of thrombus after acute myocardial infarction.

Systemic embolism occurred in 4–63% of patients with echocardiographically documented ventricular thrombus and 0–4% of those without a thrombus.¹⁰ Those findings and the patient's clinical course suggest that the transient cerebral ischemic attack was caused by embolism of the left ventricular thrombus associated with the tako-tsubo-like left ventricular dysfunction.

In the present patient, the apical asynergy was probably the most important cause of thrombus formation, as well as the decreased activity of both protein C and protein S. Ozkutlu et al reported a case of a left ventricular thrombus that was caused by acquired transient protein C deficiency from hepatitis, sepsis and heart failure,¹¹ and Yetkin et al recently reported that activated protein C resistance was an independent risk factor for left ventricular thrombosis in patients with anterior myocardial infarction.¹²

Tako-tsubo-like left ventricular dysfunction usually resolves within several weeks. In this respect, asynergy of tako-tsubo-like left ventricular dysfunction is quite different from that of acute myocardial infarction. Therefore, if the thrombus remains within the apical region, the risk of embolism is relatively high and in selected cases in which there is malignant change of the morphologic features, surgical thrombectomy should be considered.

In conclusion, a number of patients with tako-tsubo-like left ventricular dysfunction have been reported, especially in Japan,^{13,14} but this is the first report of an association with a left ventricular apical thrombus. It is important to recognize that tako-tsubo-like left ventricular dysfunction can induce thrombosis, so cardiac catheterization must be performed with caution to prevent systemic embolism, even if an apical thrombus is not seen during echocardiography.

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