Two Cases of Cerebral Embolism Caused by Apical Thrombi in Midventricular Obstructive Cardiomyopathy

Ikuko Takeda¹, Mayu Sekine², Hayato Matsushima³, Naohisa Hosomi¹, Takeshi Nakamura¹, Toshiho Ohtsuki¹, Takemori Yamawaki¹ and Masayasu Matsumoto¹

Abstract

Midventricular obstructive hypertrophic cardiomyopathy (MOHC) is a rare form of cardiomyopathy that was demonstrated to have caused embolic stroke in two patients. In both cases, the embolic sources of stroke were thrombi in an apical aneurysm caused by turbulent stasis of blood flow and subsequent injury of myocardial endocardium. Even without atrial fibrillation, apical aneurysm can induce embolic stroke in MOHC.

Key words: cerebrovascular disease/stroke, midventricular obstructive cardiomyopathy, apical thrombus


DOI: 10.2169/internalmedicine.50.5079

Introduction

Most patients with hypertrophic cardiomyopathy (HCM) have asymmetric septal hypertrophy and subaortic obstruction. Midventricular obstructive hypertrophic cardiomyopathy (MOHC) presents as atypical intraluminal stenosis of the midventricle followed by a pressure gradient between the apical and basal chambers. A previous study reported the incidence of concealed apical aneurysm with midventricular cavity obliteration to be approximately 1.5% of all HCM cases (1). Atrial fibrillation and the dilated phase of HCM are associated with embolic stroke (2); however, in 9.8% of cases of HCM, a definite origin of stroke could not be identified (3). We describe herein two MOHC patients with cerebral embolism induced by apical thrombi.

Case Report

Case 1

A 42-year-old woman who had MOHC was hospitalized because of nausea and difficulty speaking. On arrival, she had right facial palsy, dysarthria and ataxia of the right extremities. Laboratory data revealed an increased white blood cell count (12,400/mm³), D-dimer (1.1 μg/mL), and N-terminal proBNP (3484 pg/mL). Electrocardiography (ECG) on admission showed a sinus rhythm with deep T wave inversion. MRI showed acute cerebellar infarcts and occlusion of the basilar artery (Fig. 1). Transthoracic echocardiography revealed a left midventricular obstruction followed by the apical aneurysm; on ultrasound contrast, a mural thrombus was observed (Fig. 1). Transesophageal echocardiography did not demonstrate the apical thrombus or any other sources of embolus. Eight days after admission, MRI revealed left cerebellar, right pontine, and left cerebral peduncle infarcts. The patient was treated with unfractionated heparin and warfarin, and her symptoms gradually improved.

Case 2

A 65-year-old man was admitted with syncope and was diagnosed with ventricular tachycardia and midventricular obstructive cardiomyopathy. On the third day after admission, he had left hemiparesis, dysarthria and unilateral spatial neglect. Laboratory data revealed increases in blood sugar (376 mg/dL), D-dimer (4.0 μg/mL), and BNP (717 pg/mL). ECG detected a sinus rhythm with a deep negative T wave inversion in leads V3-V6. CT showed no infarction and perfusion CT demonstrated hypoperfusion and a prolonged...
apical aneurysm (Fig. 2) and continuous wave Doppler re-
mean transit time (MTT) in the right frontotemporal lobe.

mean transit time (MTT) in the right frontotemporal lobe. Transthoracic echocardiography revealed two thrombi on an apical aneurysm (Fig. 2) and continuous wave Doppler re-
corded the paradoxical diastolic flow from the apex to the base of the left ventricular cavity. One hour and 37 minutes later, the patient was treated with intravenous thrombolysis with tissue plasminogen activator and his condition improved. Warfarin was administered to prevent recurrence.

Discussion

Among the cases of HCM, the main cause of ischemic stroke is atrial fibrillation and the dilated phase of HCM (3). Paroxysmal or chronic atrial fibrillation ultimately occurs in 20% to 25% of HCM patients (4, 5) and causes 23% of the cumulative incidence of vascular events (2). We encountered and thoroughly examined two cases of stroke, possibly caused by a thrombus of akinetic apical aneurysm as possible sources of embolism. Few patients with normal left ventricular systolic performance who have never experienced stroke have demonstrated thrombus in an apical aneu-
rysm (6). A thrombus is induced by turbulent stasis of blood flow and injured myocardial endocardium by both systolic midventricular obstruction and diastolic paradoxical jet flow (7, 8) from the apex to the base of the left ventricular cavity.

Transthoracic echocardiography was superior to trans-
esophageal echocardiography in revealing the thrombus of apical aneurysm in Case 1. Because apical thrombi can cause emboli, MOHC patients without atrial fibrillation re-
quire transthoracic echocardiography to locate embolic sources.

The authors state that they have no Conflict of Interest (COI).

References

1. Maron MS, Finley JJ, Bos JM, et al. Prevalence, clinical signifi-

© 2011 The Japanese Society of Internal Medicine
http://www.naika.or.jp/imindex.html