

## Acute Multiple Cerebral Infarction in a Patient with an Accessory Mitral Valve

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### Abstract

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A 96-year-old woman developed hemiparesis 2 weeks after orthopedic surgery. Magnetic resonance imaging revealed multiple cerebral infarctions in the bilateral hemisphere. Transthoracic echocardiography revealed a mobile structure attached to the anterior mitral leaflet that protruded toward the left ventricular outflow tract. The structure was identified as an accessory mitral valve. Doppler echocardiography showed that there was no significant left ventricular outflow obstruction. This is a rare case of a silent accessory mitral valve that was detected after multiple cerebral infarctions.

**Key words:** accessory mitral valve, left ventricular outflow obstruction, cerebral infarction

(Intern Med 56: 153-155, 2017)

(DOI: 10.2169/internalmedicine.56.7649)

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### Introduction

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An accessory mitral valve (AMV) is a rare congenital cardiac malformation that may be accompanied by other congenital heart diseases, including left ventricular outflow tract obstruction (1). We herein report the case of a patient with an AMV that was diagnosed after multiple cerebral infarctions.

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### Case Report

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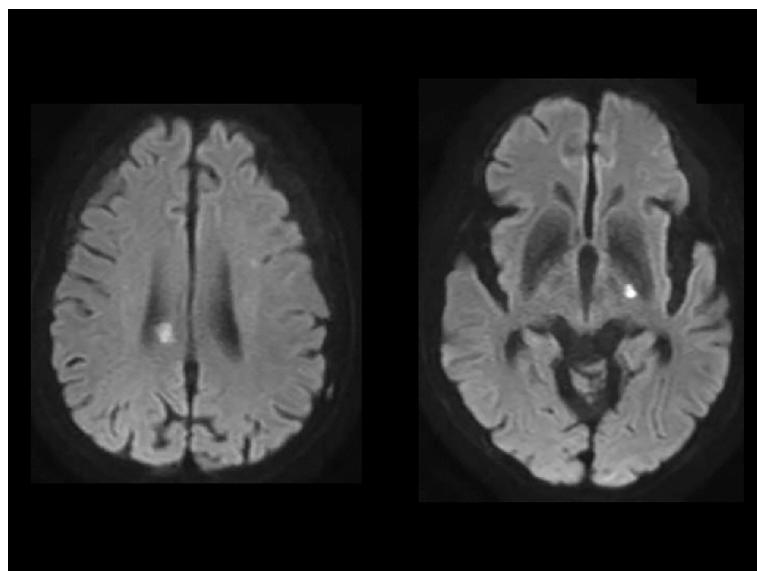
A 96-year-old woman fractured her thigh and was admitted to our hospital. Two weeks after open surgery, she noticed right hemiparesis. Diffusion-weighted magnetic resonance imaging (MRI) of the brain revealed multiple acute cerebral infarctions at the right parietal lobe and the posterior limb of the internal capsule (Fig. 1). A physical examination revealed that her body temperature was 36.9°C, her blood pressure was 135/77 mmHg, and her pulse rate was 84 beats per minute. Auscultation revealed normal respiratory sounds. A Levine 2/6 diastolic murmur and Levine 2/6

systolic murmur were detected at the second right sternal border. A blood analysis revealed mild anemia (hemoglobin, 10.3 g/dL), hypercoagulability (D-dimer, 4.1 µg/mL), dyslipidemia (low density lipoprotein (LDL)-cholesterol level of 161.0 mg/dL), and a high plasma b-type natriuretic peptide level of 162.7 pg/mL. A 12-lead electrocardiogram (ECG) showed left axis deviation of the QRS wave, a high R wave in lead V5, and mild ST segment elevation in leads V1 to V3 (Fig. 2, left panel). Chest radiography revealed cardiomegaly with a cardiothoracic ratio of 63% (Fig. 2, right panel). A Holter electrocardiogram showed frequent premature atrial contraction, but no atrial fibrillation. A transthoracic echocardiogram showed a normal left ventricular (LV) wall thickness (the interventricular septum and LV were both 11 mm in thickness) and wall motion (LV end-diastolic dimension, 32 mm; LV end-systolic dimension, 18 mm; LV ejection fraction, 76%). There was a mobile structure originating from the ventricular side of the proximal part of the anterior mitral leaflet that was also attached to the interventricular septum as a chordae tendineae-like structure, which matched the diagnostic criteria for an AMV (Fig. 3). Color Doppler ECG showed mild aortic regurgita-

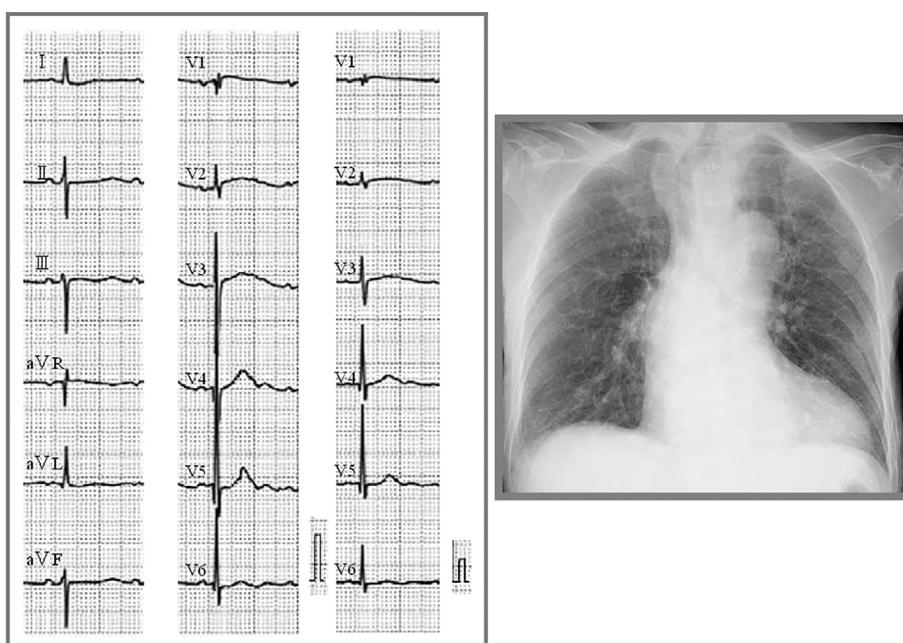
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Received for publication April 28, 2016; Accepted for publication May 29, 2016

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**Figure 1.** Diffusion-weighted magnetic resonance imaging of the brain reveals multiple acute cerebrovascular lesions at the right parietal lobe (left panel) and the posterior limb of the internal capsule (right panel).



**Figure 2.** A 12-lead electrocardiogram (left panel) showing the left axis deviation of the QRS wave, a high R wave in lead V5, and mild ST segment elevation in leads V1 to V3. A chest radiograph (right panel) showing cardiomegaly with a cardiothoracic ratio of 63%.

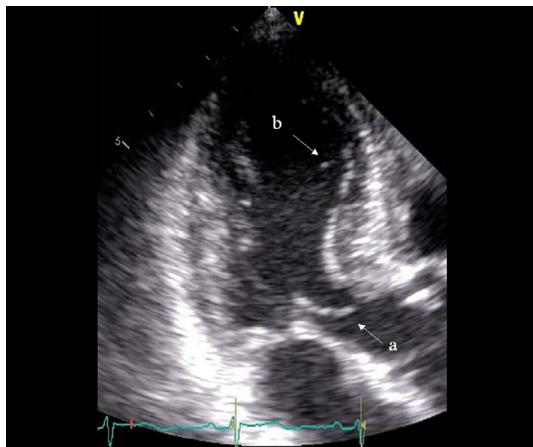
tion. Surgical treatment was not considered because the AMV did not cause left ventricular outflow obstruction. The patient was treated with warfarin and was discharged after rehabilitation.

## Discussion

An AMV is a rare congenital cardiac anomaly that has been estimated to be present in 1 per 26,000 echocardiograms (2). Although its embryonic mechanism is unclear,

the abnormal development of the endocardial cushion tissue may be involved. Transthoracic echocardiography is a useful and noninvasive modality for diagnosing AMVs. In the present case, transthoracic echocardiography showed an AMV that originated from the ventricular side of the anterior mitral leaflet, forming a sail-like appearance, which was also attached to the interventricular septum. These findings met the diagnostic criteria for an AMV.

Left ventricular outflow obstruction is frequently observed; almost 80% of AMV patients present moderate or



**Figure 3.** A transthoracic echocardiogram from the apical long axis view showing a mobile structure attached to the anterior mitral leaflet that protruded toward the left ventricular outflow tract forming a sail-like appearance (a). It was also attached to interventricular septum as a chordae tendineae-like structure (b).

severe outflow obstruction (3). However, the present case there was no left ventricular outflow obstruction.

There have been reports that cerebral embolisms may occur in an AMV (4). Rovner et al. reported that an AMV might accumulate platelets and other debris that may become detached and cause a cerebral embolism (4). In the present case, acute cerebral infarctions occurred in the bilateral hemisphere. As this patient did not have atrial fibrillation, valvular heart disease, dilated cardiomyopathy, or an intracardiac mass (5), the cardiogenic embolism might have

been caused by the AMV. Moreover, this case suggests that some patients with a silent AMV may live their natural life span without any of the symptoms that are generally associated with an AMV.

In summary, we reported a rare case of an AMV without left ventricular outflow obstruction that was diagnosed after multiple cerebral infarctions.

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

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