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Hypertrophic Cardiomyopathy With Mid-Ventricular Obstruction and Splenic Infarction Associated With Paroxysmal Atrial Fibrillation: A Case Report

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Abstract

A 54-year-old woman had been treated for hypertrophic cardiomyopathy and paroxysmal atrial fibrillation since 1992. She was admitted with paroxysmal atrial fibrillation which was resolved by medical treatment. However, on the next day, left lateral chest pain appeared. Computed tomography disclosed a low density area in the spleen. She received anticoagulant therapy under a diagnosis of splenic infarction, and the pain disappeared. Echocardiography showed hypertrophic cardiomyopathy with mid-ventricular obstruction. She was treated with cibenzoline to prevent paroxysmal atrial fibrillation attack and attenuate the hemodynamic load. After treatment, the pressure gradient decreased from 41 to 7 mmHg.

This patient with hypertrophic cardiomyopathy suffered a rare isolated splenic infarction associated with paroxysmal atrial fibrillation.

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Key Words

■ **Cardiomyopathies, hypertrophic (mid-ventricular obstruction)**
■ **Atrial fibrillation (paroxysmal)** ■ **Thromboembolism (splenic infarction)**
■ **Computed tomography**

INTRODUCTION

Atrial fibrillation occurs in as many as 5% to 15% of patients with hypertrophic cardiomyopathy[1–3]. Moreover, atrial fibrillation in patients with hypertrophic cardiomyopathy is often associated with thromboembolism complications[4–6]. The brain, kidney and femoral artery are the most common locations of thromboembolism[7–9]. We report a rare case of hypertrophic cardiomyopathy with isolated splenic infarction due to paroxysmal atrial fibrillation.

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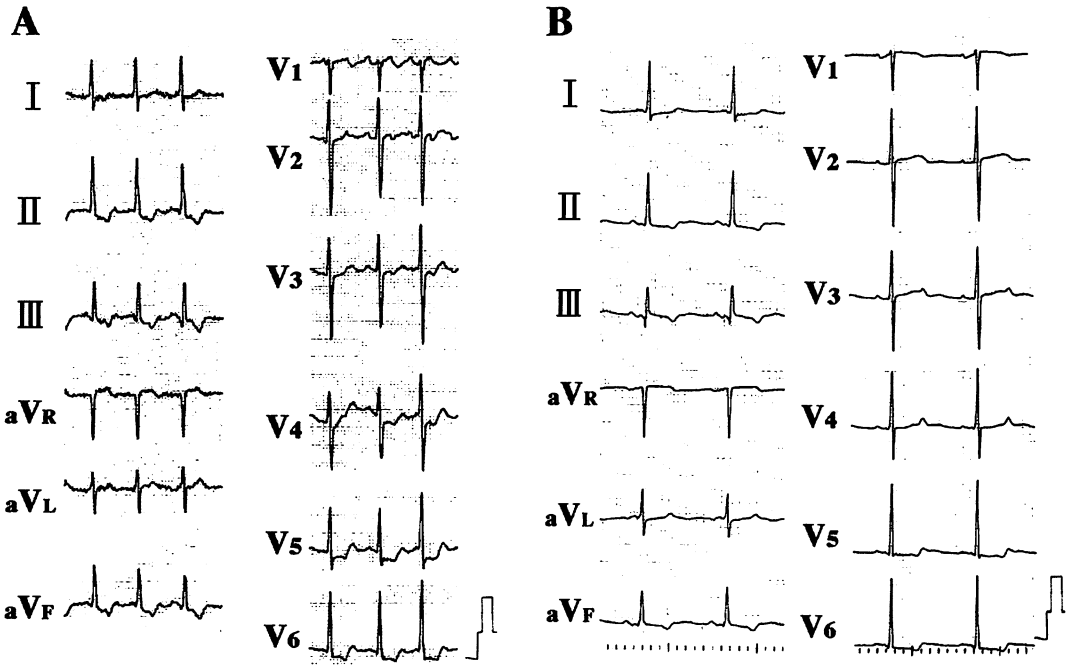

Fig. 1 Electrocardiograms recorded during an attack of paroxysmal atrial fibrillation (*A*) and on admission (*B*)

A: Atrial fibrillation with tachycardia was observed.

B: Left ventricular hypertrophy with negative T wave and ST depression was observed.

CASE

A 54-year-old woman had been treated under a diagnosis of hypertrophic cardiomyopathy since 1992. Paroxysmal atrial fibrillation had occurred twice in 1992. However, she had discontinued the treatment since 1996. She was admitted to another hospital because of paroxysmal atrial fibrillation on June 16, 1997 (**Fig. 1−A**). Disopyramide (50 mg) was administered intravenously. After 9 hr, her cardiac rhythm returned to sinus, but she complained of left lateral chest pain on the next day. Computed tomography disclosed a low density area in the spleen (**Fig. 2−A**). She received anticoagulant therapy under a diagnosis of splenic infarction, and the pain disappeared. Thromboembolism was not found in other organs such as the brain, kidneys and lower extremities. She was transferred to our hospital to receive the appropriate treatment for hypertrophic cardiomyopathy and to prevent paroxysmal atrial fibrillation on July 22, 1997.

On admission her pulse rate was 60/min and blood pressure was 128/72 mmHg. Auscultation of the heart revealed a big fourth heart sound at the apex and systolic murmur of Levine Ⅲ at the third intercostal region. Electrocardiography on admission showed negative T wave in the Ⅱ, Ⅲ, aV_F and V_6 leads, and ST depression in the Ⅰ, aV_L, V_5 and V_6 leads (**Fig. 1−B**). Paroxysmal atrial fibrillation accompanied tachycardia of 120/min (**Fig. 1−A**). Chest radiography showed cardiomegaly. **Fig. 3** shows the two-dimensional echocardiographic study. Interventricular septal thickness and left ventricular posterior wall thickness were 13 and 10 mm, respectively. Left ventricular diastolic dimension and left atrial dimension were 47 and 29 mm, respectively. Systolic anterior movement of the mitral valve was not observed. The systolic peak flow velocity at the region of the mid-ventricle was 3.2 m/sec and the calculated pressure gradient was 41 mmHg (**Fig. 4−A**). Based on these echocardiographic findings, the diagnosis was hypertrophic cardiomyopathy with mid-ventricular obstruction. Left atrial thrombus was not observed even by transesophageal echocardiography. Coronary angiography showed the intact left and right coronary arteries. End-diastolic left ventricular pressure was 13 mmHg. Computed tomography

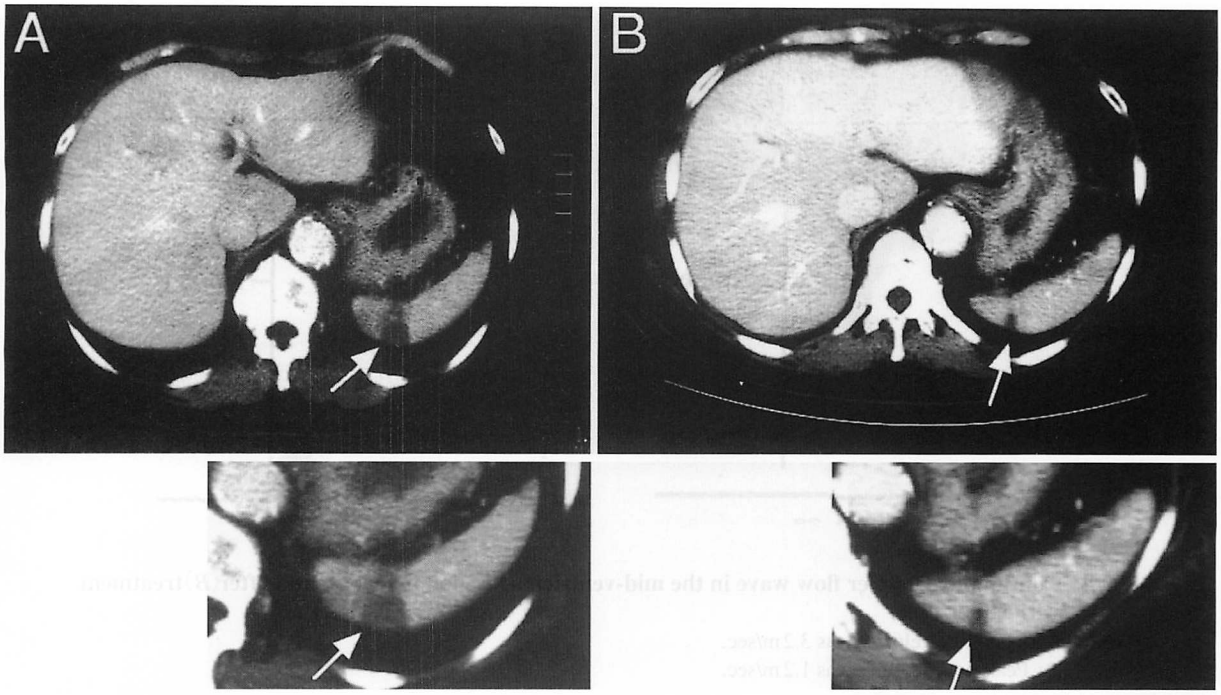

Fig. 2 Computed tomography scans of the splenic level
A large low density area (*arrows*) was found in the spleen at the episode of infarction (*A*), and this low density area (*arrows*) was markedly decreased 2 months after the episode (*B*).
Lower panels are enlarged.

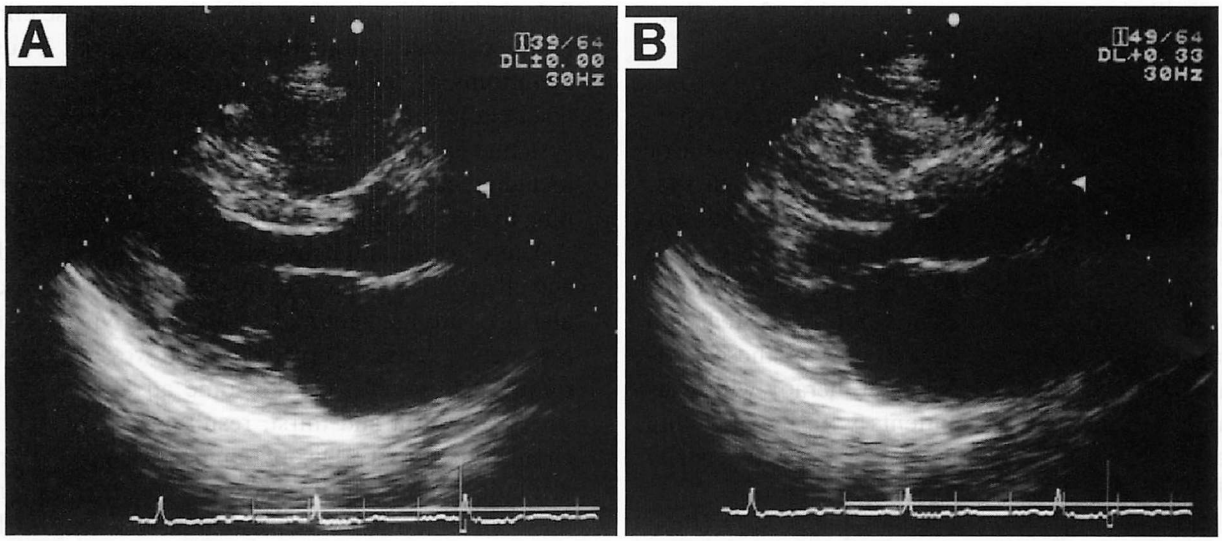

Fig. 3 Stop frame of two-dimensional echocardiography obtained in the parasternal long-axis view at end-diastole (*A*) and end-systole (*B*)

performed 2 months after the episode of thromboembolism showed marked reduction of the low density area in the spleen (**Fig. 2–B**).

The patient had sinus bradycardia and sinus pause of 2.2 sec suggesting the existence of sick sinus syndrome, so she was treated with cibenzoline and warfarin to prevent paroxysmal atrial fibrillation attack and attenuate the hemodynamic

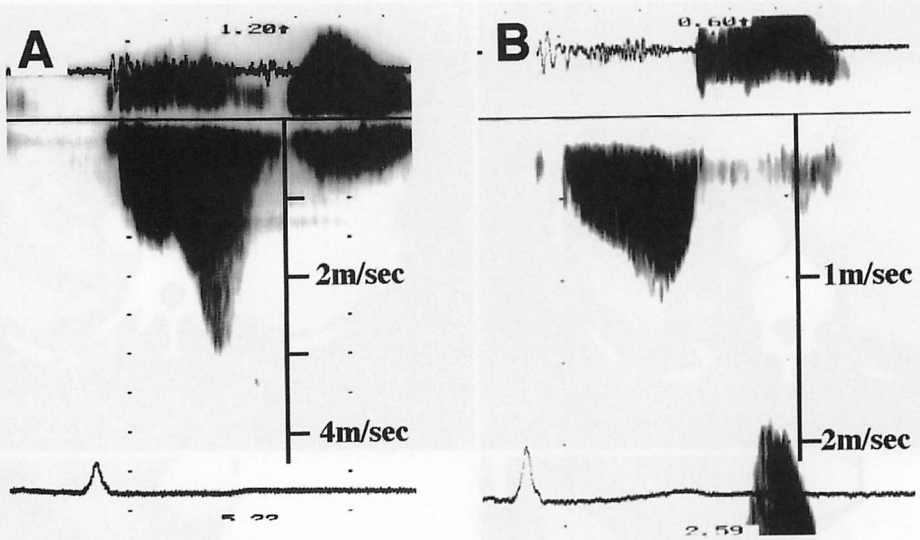

Fig. 4 Continuous Doppler flow wave in the mid-ventricular region before(*A*)and after(*B*)treatment with cibenzoline
A: Peak flow velocity was 3.2 m/sec.
B: Peak flow velocity was 1.2 m/sec.

load. After treatment with cibenzoline, the systolic peak flow velocity decreased from 3.2 to 1.2 m/sec, and the pressure gradient decreased from 41 to 7 mmHg (**Fig. 4−B**). In addition, paroxysmal atrial fibrillation attack has not occurred for 3 months.

DISCUSSION

Previously, we treated 24 patients with hypertrophic cardiomyopathy who suffered transient or persistent atrial fibrillation. The incidence of systemic thromboembolism in these 24 patients was 7.1% per patient year[7]. In addition, there was no significant difference in the incidence of thromboembolism between patients with persistent and transient atrial fibrillation. In view of these findings, we think that anticoagulant therapy with warfarin is mandatory for patients with hypertrophic cardiomyopathy and transient or persistent atrial fibrillation.

The present patient suffered splenic infarction. Thromboembolism associated with atrial fibrillation in patients with hypertrophic cardiomyopathy usually occurs in the brain, kidney or femoral artery[7−9]. Splenic infarction occurs as a consequence of several cardiovascular disorders. Autopsy series have substantiated the presence of splenic thromboembolic lesions in up to 9% of patients who have died early after an acute myocardial infarction[10]. Clinical series, in contrast, report a much low incidence of splenic infarction[11,12]. Splenic infarction is often difficult to diagnose clinically without a high index of suspicion. Thus, symptoms of fever, tachycardia, and left-upper-quadrant pain and tenderness in a patient at risk for systemic embolization should prompt examinations including computed tomography or ultrasonic scanning to rule out splenic infarction[13].

The class Ia antiarrhythmic drug disopyramide and cibenzoline can markedly attenuate left ventricular pressure gradient in patients with hypertrophic obstructive cardiomyopathy[14,15]. However, disopyramide has several troublesome adverse effects, such as dysuria and thirst, resulting from its anticholinergic activity. Disopyramide treatment is very difficult to continue in patients who suffer from these adverse effects. Cibenzoline has only little anticholinergic activity and can also improve the diastolic function. In our patient, cibenzoline was very effective both to ameliorate the hemodynamics and to prevent the attack of paroxysmal atrial fibrillation.

要　約

発作性心房細動により脾梗塞を合併した心室中部閉塞性肥大型心筋症

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症例は54歳の女性で，1992年から近医にて肥大型心筋症として加療されていた．1997年6月，発作性心房細動にて近医を受診し，薬物による除細動により洞調律に回復した．翌日，左側胸部痛が突然出現した．腹部コンピューター断層撮影により脾臓に低吸収領域を認め，脾梗塞と診断し，抗凝固療法を行ったところ左側胸部痛は消失し，脾臓の低吸収領域は縮小した．心エコー図法により心室中部に閉塞を生じる肥大型心筋症を認め，同部位の圧較差は41 mmHgであった．発作性心房細動の予防および圧較差の改善を目的にシベンゾリンを投与した．シベンゾリンの治療により圧較差は41から7 mmHgに低下した．

本症例は，発作性心房細動にて脾梗塞を併発した心室中部閉塞性肥大型心筋症であり，まれな症例と思われた．

J Cardiol 1999; 33(5): 273–277

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