J Med Sci 2008;28(1):035-038
http://jms.ndmctsgh.edu.tw/2801035.pdf

Apical Hypertrophic Cardiomyopathy Mimicking Coronary Artery Disease with Unstable Angina

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We report the case of an animal bite with a right forefoot avulsion wound and amputation of the fifth toe, for an emergency debridement operation under spinal anesthesia. An initial mistaken diagnosis of coronary artery disease with unstable angina was made because of the presence of resting angina, with the corresponding electrocardiographic findings. The patient was ultimately diagnosed with apical hypertrophic cardiomyopathy with normal coronary arteries based on his previous medical records. The outcome was favorable after treatment with a β-blocker.

Key words: apical hypertrophic cardiomyopathy, coronary artery disease, unstable angina

INTRODUCTION

Apical hypertrophic cardiomyopathy (ApHCM) was initially described by Japanese investigators as a form of nonobstructive hypertrophic cardiomyopathy associated with specific electrocardiographic findings of giant precordial negative T waves and a typical ace-of-spades configuration of the left ventriculogram, and characteristic echocardiographic evidence of localized apical hypertrophy[1]. Studies of ApHCM in Japanese and Western populations have suggested the rare occurrence of cardiovascular mortality and morbidity[1-3]. However, severe complications associated with ApHCM have been described, including sudden cardiac death, severe, arrhythmias, and apical infarction with apical aneurysm[4-6]. Here, we report a patient with typical ischemic chest pain and electrocardiographic ST-segment depression and inverted T waves, who presented for emergency surgery. The presence of resting angina with these electrocardiographic findings led to a mistaken initial diagnosis of coronary artery disease with unstable angina.

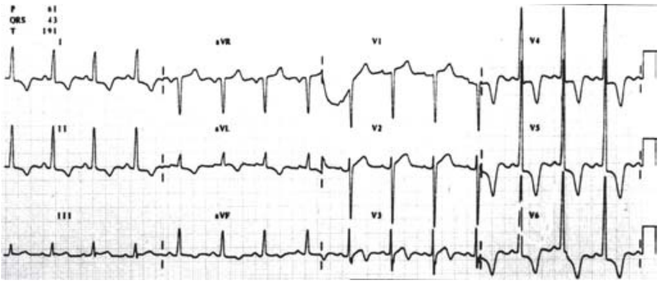

Fig. 1 Twelve-lead electrocardiogram demonstrates giant negative T waves in precordial leads.

CASE REPORT

A 60-year-old man (height 170 cm, weight 75 kg) was sent to our emergency department with an animal bite, with a right forefoot avulsion wound and amputation of the fifth toe, for emergency debridement surgery. The patient suffered from crushing retrosternal chest pain, which had at that time lasted for 2 h. His preoperative blood pressure was 150/80 mmHg, with a heart rate of 90 beats/min. He had experienced exertional angina and easy fatigue for two years. His previous history revealed no abnormalities except borderline arterial hypertension without regular medical control and heavy smoking for 30 years. His preoperative laboratory data were unremarkable. An electrocardiogram showed an ST-segment depression and giant inverted T waves in leads I, II, aVL, and V4-V6 (Figure 1).

After a diagnosis of coronary artery disease with unstable angina, nitroglycerin (10 μg/min) and morphine (2 mg) were administered intravenously, and we chose spinal

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Received: July 6, 2007; Revised: October 11, 2007; Accepted: December 11, 2007

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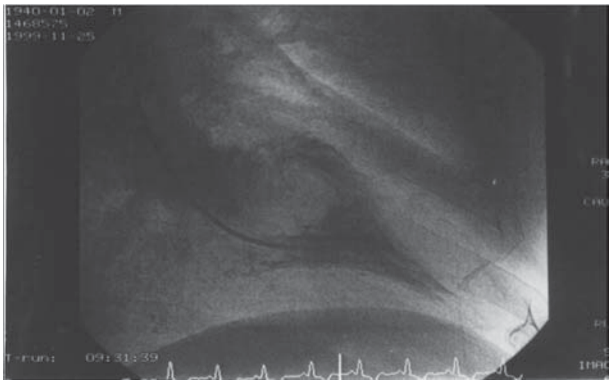

Fig. 2 Left ventriculography revealed a spade-like configuration at end-diastole.

anesthesia for the emergency operation. Following an uncomplicated lumbar puncture at the L4-5 interspace using a 25-G spinal needle, 12 mg of tetracaine was administered, and a satisfactory sensory block to the level of T10 was achieved. A decrease in his systolic blood pressure from 150 to 80 mmHg and an increase in his heart rate from 90 to 120 beats/min were noted, and the patient complained of worsening angina symptoms over the left shoulder area, but no evolutional changes were apparent on electrocardiographic monitoring. Ephedrine (10 mg) and crystalloid solution were given, and his blood pressure increased to 100/60 mmHg, but tachycardia (120 beats/min) persisted.

After reviewing the patient's previous medical records, it was found that this patient had been diagnosed with apical hypertrophic cardiomyopathy seven months previously. The diagnosis was confirmed at that time by (1) an electrocardiogram that demonstrated typical left ventricular hypertrophy with giant negative T waves; (2) an echocardiographic study showing apical hypertrophy with a slight intracavitary pressure gradient; and (3) catheterization that revealed normal coronary arteries with typical spade-shaped ventriculogram at the end diastole (Figure 2). After treatment with a β-blocker (esmolol; 5 mg), the patient's angina symptoms were relieved. His vital signs remained stable and no complications were noted.

Serial postoperative electrocardiograms showed no new changes, and plasma concentrations of cardiac isoenzymes were within normal limits (creatinine kinase [CK]: 115 U/L; CK-MB: 9 U/L; troponin I: 0.12 ng/mL).

DISCUSSION

Apical hypertrophic cardiomyopathy (ApHCM) is considered a form of hypertrophic cardiomyopathy, with a relatively favorable prognosis in most patients[7]. This apical variant appears to be far more common in Japanese patients than in Western populations (25% vs 2% of hypertrophic cardiomyopathies, respectively)[8]. The incidence of ApHCM in Taiwan is relatively high, similar to that in Japan[9]. There is strong evidence of male predominance[2,9] and some evidence of genetic transmission in first-degree relatives of patients with ApHCM[10].

Patients with ApHCM can experience typical angina resulting from apical ischemia. It has been suggested that the imbalance of coronary flow directed to the hypertrophic apex may be one of the mechanisms of ischemia in the apical form of hypertrophic cardiomyopathy[4]. A variety of mechanisms have been proposed to explain the angina observed in hypertrophic cardiomyopathy: impaired diastolic filling leading to impaired coronary flow, relatively insufficient myocardial perfusion of the increased myocardial mass[4], and intramyocardial small vessel disease[11]. ApHCM can coexist with coronary artery disease, systemic hypertension, or both[5].

The presence of infarct in the absence of coronary stenosis is well documented in hypertrophic cardiomyopathy. Apical segmental dysfunction and/or aneurysm have been reported and may be related to myocardial infarction, to massive myocardial degeneration of unknown etiology and the ensuing replacement fibrosis, or to small vessel disease[11]. In some cases, it seems to be associated with the presence of a mild ventricular obstruction, which may facilitate the formation of a left ventricular aneurysm by increasing the pressure in the apical chamber[5].

The preoperative evaluation of patients with hypertrophic cardiomyopathy should focus on evaluating the possibility of significant dynamic obstruction, malignant arrhythmia, or myocardial ischemia. The results of echocardiography and Holter monitoring should ideally be reviewed with a cardiologist. The general principles of management of hypertrophic cardiomyopathy include the minimization of sympathetic activation, maintenance of adequate preload and afterload, avoidance of tachycardia, and increased myocardial contractility[12]. The drugs of choice for therapy include β-blockers and calcium channel blockers. These classes of drugs have both negative inotropic and chronotropic effects that decrease the outflow tract obstruction. They may also improve diastolic filling of the ventricle[12]. Phenylephrine and other pure α-adrenergic agonists are ideal vasopressors in these patients because they do not augment contractility, but increase systemic vascular resistance. Regional anesthesia has been considered relatively contraindicated in patients with hypertrophic cardiomyopathy because of the risk of decreased preload and afterload, causing a decrease in diastolic function or stenosis in the left ventricular outflow tract. Nonetheless, epidural anesthesia alone[13] or combined with spinal anesthesia[14] has been used safely for vaginal delivery in patients with hypertrophic cardiomyopathy. These reports indicate that, if adequate preload is preserved and hypotension is avoided with volume replacement and vasoconstrictors, regional anesthesia need not be precluded in patients with hypertrophic cardiomyopathy.

In conclusion, ApHCM can be misdiagnosed as coronary artery disease with unstable angina because of its typical ischemic chest pain and marked ST depression and deep symmetric T-wave inversion. However, echocardiography is a useful tool for its quick and accurate diagnosis and can guide further therapy.

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