

Apical Hypertrophic Cardiomyopathy Mimicking Acute Coronary Syndrome

Akut Koroner Sendromu Taklit Eden Apikal Hipertrofik Kardiyomiyopati

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ABSTRACT

Apical hypertrophic cardiomyopathy is a rare form of hypertrophic cardiomyopathy and it can be mistaken for coronary artery disease due to the symptoms and electrocardiography findings. In this report, we aimed to present a patient referred to our clinic with complaints of chest pain and electrocardiography findings who had been misdiagnosed as non-ST elevation acute coronary syndrome.

Key Words: Acute coronary syndrome; cardiomyopathies; cardiomyopathy, hypertrophic.

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ÖZET

Apikal hipertrofik kardiyomiyopati; hipertrofik kardiyomiyopatinin seyrek bir formudur, semptom ve elektrokardiyografi bulguları nedeniyle koroner arter hastalığı ile karışabilir. Bu olgu sunumunda tipik göğüs ağrısı ve elektrokardiyografi bulgularıyla başvuran ve yanlışlıkla ST yükselmesi olmayan akut koroner sendrom tanısı alan hastayı sunmayı amaçladık.

Anahtar Kelimeler: Akut koroner sendrom; kardiyomiyopatiler; kardiyomiyopati, hipertrofik.

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INTRODUCTION

Apical hypertrophic cardiomyopathy is one of the subgroups of hypertrophic cardiomyopathy with its specific morphological and clinical features. Although it is more common in Japan population, it can be detected rarely also in western populations^(1,2). It has a better prognosis compared to hypertrophic cardiomyopathy, but some complications may be observed with apical hypertrophic cardiomyopathy such as atrial fibrillation, myocardial infarction and sudden death^(3,4).

CASE REPORT

A 55-years-old female patient presented to the emergency department with the chest pain which occurred after exertion and was persistent in resting also for 30 minutes. The pain was diffuse in middle line of the chest. The patient had not any known risk factor except hyperlipidemia. On physical examination; blood pressure was found as 130/70 mmHg and the heart rate as 78 beat/minute. No significant findings was found in the auscultation. On her electrocardiography (ECG), there were ST depression and T negativity at the anterolateral derivations (Figure 1). On echocardiologic examination, wall motion defect (dyskinesia) was found in the left ventricular apical region, but detailed evaluation could not be made due to echogenicity (Figure 2). The patient was considered as non-ST elevation acute coronary syndrome and hospitalized in the coronary intensive care unit. Morphine sulfate was administered due to her pain. Antiagregant, anticoagulant and anti-ischemic therapies were introduced. No elevation was observed in cardiac enzymes of the patient during the follow-up. On coronary angiographic evaluation of the patient, the coronary arteries were normal. There was an ace of spades

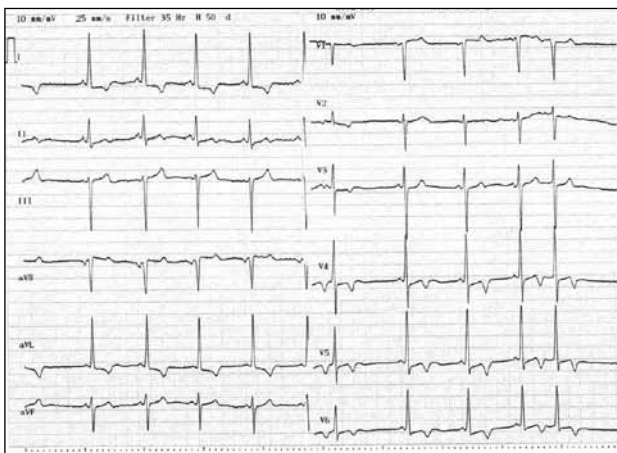


Figure 1. 12 lead electrocardiogram shows ST depression and T negativity at the anterolateral derivations.

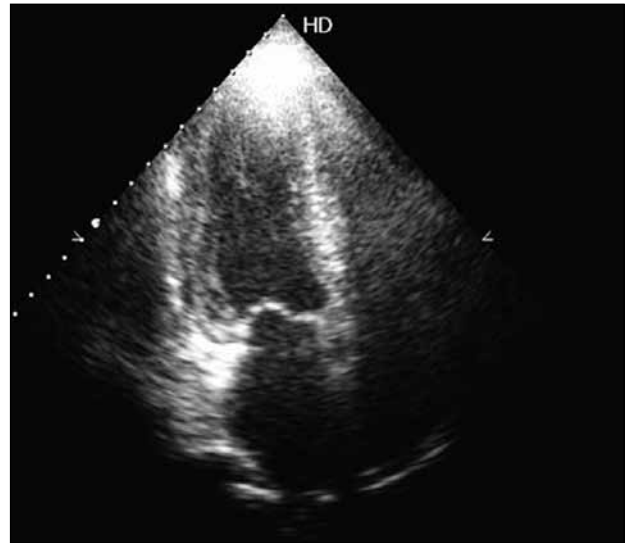


Figure 2. Apical four chamber view. Apex is dyskinetic, but no further information was available owing to limited image quality.

appearance on the left ventriculography and the diagnosis was set for apical hypertrophic cardiomyopathy (Figure 3). The patient was put on beta-blocker therapy and taken to follow-up.

DISCUSSION

Apical hypertrophic cardiomyopathy was described first by Sakamo et al. in a Japan patient in 1976. Clinical and morphological features of the disease were revealed

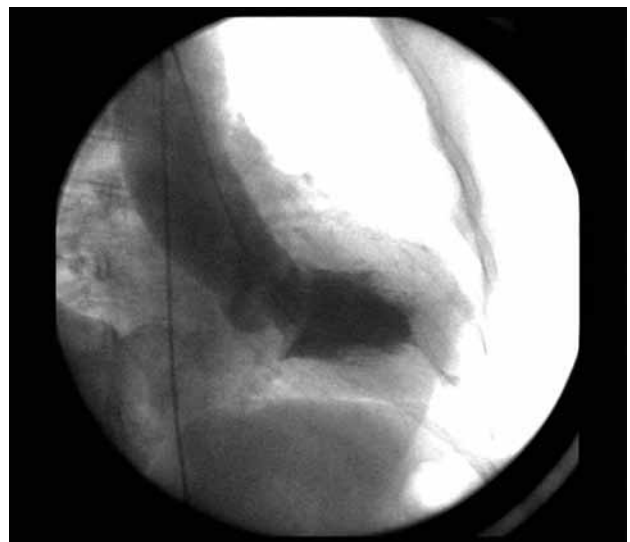


Figure 3. Characteristic ace of spades left ventriculogram demonstrating marked apical hypertrophic cardiomyopathy.

in 1979. Because the disease is common in Japan population, it is also known as Japan heart disease^(1,2). Apical hypertrophic cardiomyopathy is accounted for 13 to 25% of all the hypertrophic cardiomyopathy in Japan, whereas this rate is 1-2% in western countries^(5,6). In contrast to diffuse hypertrophy seen in hypertrophic cardiomyopathy, in this disease wall thickening is in the apex region under the papillary muscle^(1,2).

It has a better prognosis compared to hypertrophic cardiomyopathy, but some complications may be observed with apical hypertrophic cardiomyopathy such as atrial fibrillation, myocardial infarction and sudden death^(3,4). Genetic relationship demonstrated in hypertrophic cardiomyopathy could not found clearly in the patients with apical hypertrophic cardiomyopathy⁽⁷⁾.

These patients may present with typical chest pain due to ischemia of apical region. Increased myocardial tissue, small vessel disease and impaired vasodilator reserve have been proposed as the possible causes of myocardial ischemia^(8,9).

Apical hypertrophic cardiomyopathy has typical ECG findings. Repolarization impairment is observed in the anterolateral derivations in all the cases, while giant negative T waves can be monitored in about half of the cases⁽⁴⁾. These patients are often mistaken for coronary artery disease due to the symptoms and ECG findings^(10,11). Coronary arteries are normal on the coronary angiographic evaluation in most cases, although concomitant coronary artery disease was demonstrated in small number of the cases⁽⁴⁻¹³⁾.

Another typical finding specific to the disease is ace of spades appearance monitored in the left ventriculography^(1,2). Diagnosis of apical hypertrophic cardiomyopathy should be kept in mind in the patients with impaired repolarization identified in the anterolateral derivations. Transthoracic echocardiography is the first option for evaluation of the patients, but viewing the apical region may not be always possible because of artifacts or poor echogenicity⁽¹⁴⁾. In such situations, further examinations (magnetic resonance imaging, contrast ventriculography) may be considered.

CONFLICT of INTEREST

None declared.

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