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 yanılsılıkla 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\(^1\text{-}\text{2}\). 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\(^3\text{-}\text{4}\).

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. Antiaggregant, 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 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

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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.

Figure 3. Characteristic ace of spades left ventriculogram demonstrating marked apical hypertrophic cardiomyopathy.
in 1979. Because the disease is common in Japan popu-
lation, 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 dif-
fuse 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 card-
miopathy, but some complications may be observed
with apical hypertrophic cardiomyopathy such as atrial fib-
-llation, myocardial infarction and sudden death[3,4]. Ge-
netic relationship demonstrated in hypertrophic cardiomi-
opathy 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 an-
terolateral 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 dis-
ase due to the symptoms and ECG findings[10,11]. Coro-
-ary 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 ventriculog-
raphy[1,2]. Diagnosis of apical hypertrophic cardiomy-
opathy should be kept in mind in the patients with impaired
repolarization identified in the anterolateral derivations.
Transthoracic echocardiography is the first option for eva-
ualuation of the patients, but viewing the apical region may
not be always possible because of artifacts or poor echo-
genicity[14]. In such situations, further examinations (mag-
netc resonanse imaging, contrast ventriculography) may
be considered.

CONFLICT of INTEREST

None declared.

REFERENCES

1. Sakamoto T, Tei C, Murayama M, Ichiyasu H, Hada Y. Giant T
 wave inversion as a manifestation of asymmetrical apical hypert-
 raphy (AAH) of the left ventricle. Echocardiographic and ultraso-

2. Yamaguchi H, Ishimura T, Nishiyama S, Nagasaki F, Nakanishi
 S, Takatsu F, et al. Hypertrophic nonobstructive cardiomyopathy
with giant negative T waves (apical hypertrophy): ventriculog-
graphic and echocardiographic features in 30 patients. Am J Cardiol
1979;44:401-12.

the natural history of apical hypertrophic cardiomyopathy during

4. Eriksson MJ, Sonnenberg B, Woo A, Rakowski P, Parker TG, Wig-
 gle ED, et al. Long-term outcome in patients with apical hypertrop-

5. Louie EK, Maron BJ. Apical hypertrophic cardiomyopathy: clinical
and two-dimensional echocardiographic assessment. Ann Intern

6. Yamaguchi H, Nishiyama S, Nakanishi S, Nishimura S. Electro-
cardiographic, echocardiographic and ventriculographic charac-
terization of hypertrophic non-obstructive cardiomyopathy. Eur


8. Lazzaroni E, Rolli A, Auriel E, Botti G. Clinical significance of coro-
-ary artery disease in hypertrophic cardiomyopathy. Am J Cardiol

of hypertrophic cardiomyopathy complicated by acute myocardial
infarction and ventricular tachycardia: slow coronary artery flow.

10. Seneviratne SL, Premawardena AP, Ranasinghe GW, Gunatilake
SB, De Silva HJ. Apical cardiomyopathy: an important differential

11. Iskandar SB, Dittus K, Merrick D. Uncommon cause of a common

12. Albanesi Filho FM, Lopes JS, Aron Diamant JD, Castier MB, Alex-
andre J, Esad R, et al. Apical hypertrophic cardiomyopathy asso-
ciated with coronary artery disease. Arq Bras Cardiol 1998;71:139-
42.

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