

# Apical hypertrophic cardiomyopathy coexisting with coronary artery disease: a case report

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## SUMMARY

Apical hypertrophic cardiomyopathy is a rare form of hypertrophic cardiomyopathy and may mimic coronary artery disease. There are very limited data about apical hypertrophic cardiomyopathy coexisting with coronary artery disease in the literature. We present an interesting case of coronary artery disease who was later found to have apical hypertrophic cardiomyopathy and a decrease in T wave negativity after coronary artery bypass grafting.

**Key words:** Apical hypertrophic cardiomyopathy, coronary artery bypass grafting, coronary artery disease

## ÖZET

**Koroner arter hastalığına eşlik eden apikal hipertrofik kardiyomiyopati: bir olgu sunumu**

Apikal hipertrofik kardiyomiyopati nadir görülen bir kardiyomiyopati türü olup, koroner arter hastalığını taklit edebilmektedir. Literatürde apikal hipertrofik kardiyomiyopati ve koroner arter hastalığı birlikteliği ile ilgili çok az veri bulunmaktadır. Bu yazıda sonradan apikal hipertrofik kardiyomiyopati varlığı saptanan ve koroner by-pass greft operasyonu sonrası T dalga negatifliğinde azalma tespit edilen bir koroner arter hastalığı olgusu sunulmaktadır.

**Anahtar kelimeler:** Apikal hipertrofik kardiyomiyopati, koroner arter by-pass greft, koroner arter hastalığı

## Introduction

Apical hypertrophic cardiomyopathy (ApHCMP) is a rare form of hypertrophic cardiomyopathy, which is diagnosed by giant T wave negativity on electrocardiogram (ECG) and “spade-like” configuration of the left ventricular (LV) cavity at end diastole on ventriculography (1,2). ApHCMP is accompanied by symptoms and ECG changes indicative of myocardial ischemia (3,4). There are very limited data about ApHCMP coexisting with coronary artery disease (CAD) (5-7). We present an interesting case of CAD who was later found to have ApHCMP and a decrease in giant T wave negativity after coronary artery bypass grafting (CABG).

## Case Report

A 52-year-old man who had previously documented CAD was referred to our department with a typical chest pain. He was a heavy smoker with hypercholesterolemia. He had a history of percutaneous coronary intervention (PCI) to the right coronary artery at another medical center 7 years ago. Physical examination was normal. His ECG showed sinus rhythm, LV hypertrophy (SV1+RV5 46 mm), and giant negative T waves (>10 mm) in leads V2-6, I and avL (Figure 1). On transthoracic echocardiographic examination left ventricular diameters were within normal limits and left atrial diameter was increased (41 mm). Interventricular septum and posterior wall thickness were 12 and 11 mm, respectively. However, apical segments of left ventricle could not be visualized because of poor image quality. Coronary angiography revealed 70% stenosis in the mid-portion of the left anterior descending artery (LAD), 80% stenosis in the proximal segment of the left circumflex coronary artery (Figure 2) and 98% stenosis in the proximal segment of the right coronary artery (RCA). Left ventriculography demonstrated a “spade-like” configuration on the right anterior oblique view (Figure 2). Surgical treatment was suggested and CABG (left

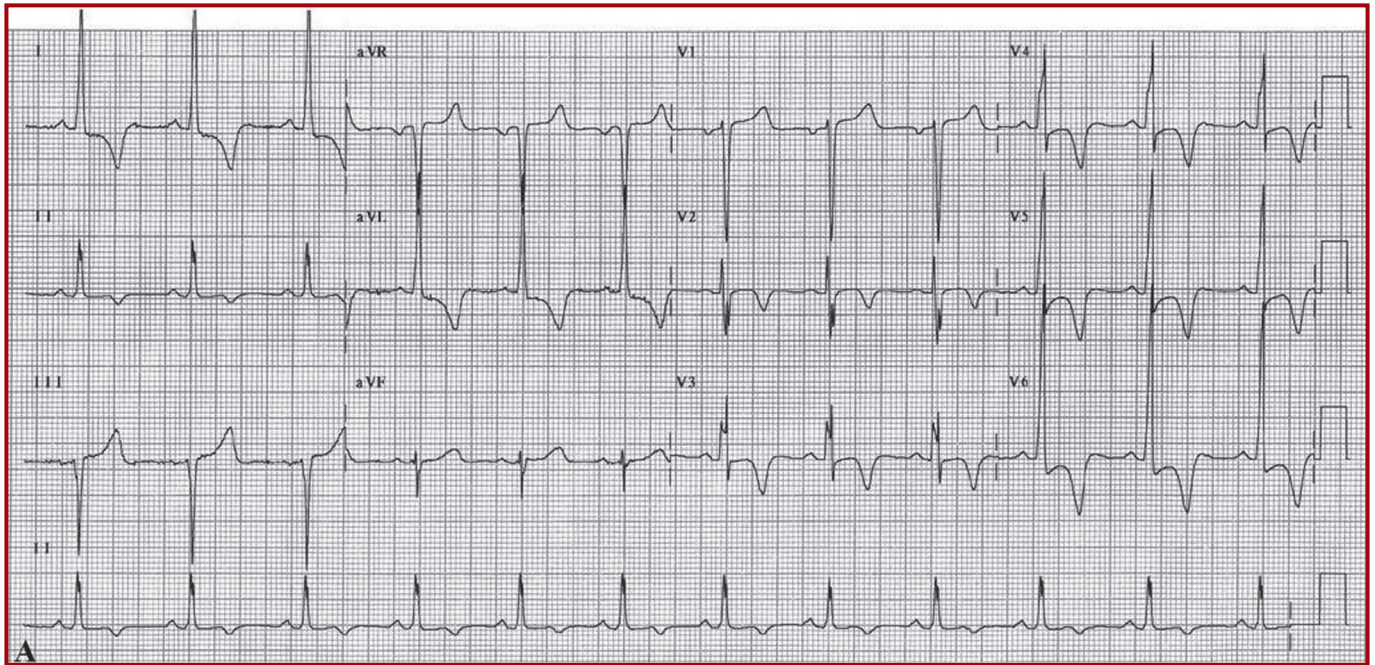
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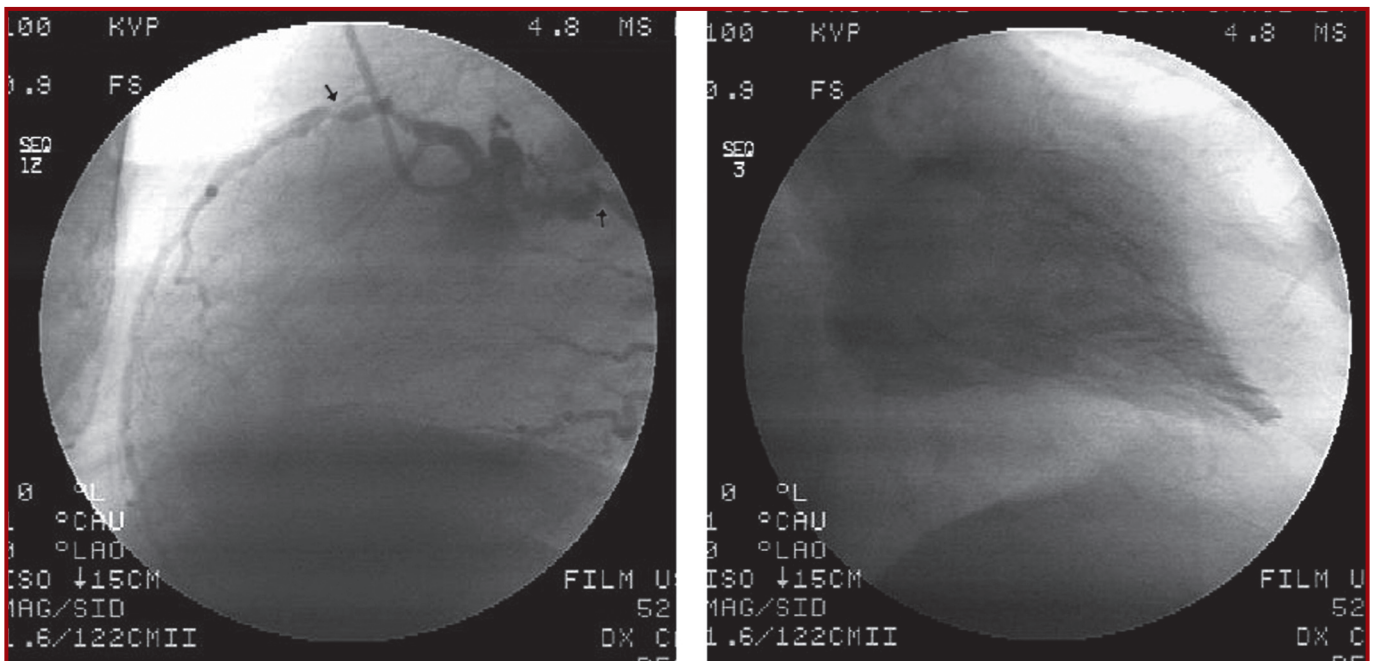
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**Figure 1.** Electrocardiogram shows left ventricular hypertrophy and giant negative T waves (>10 mm) in leads V2-6, I and avL



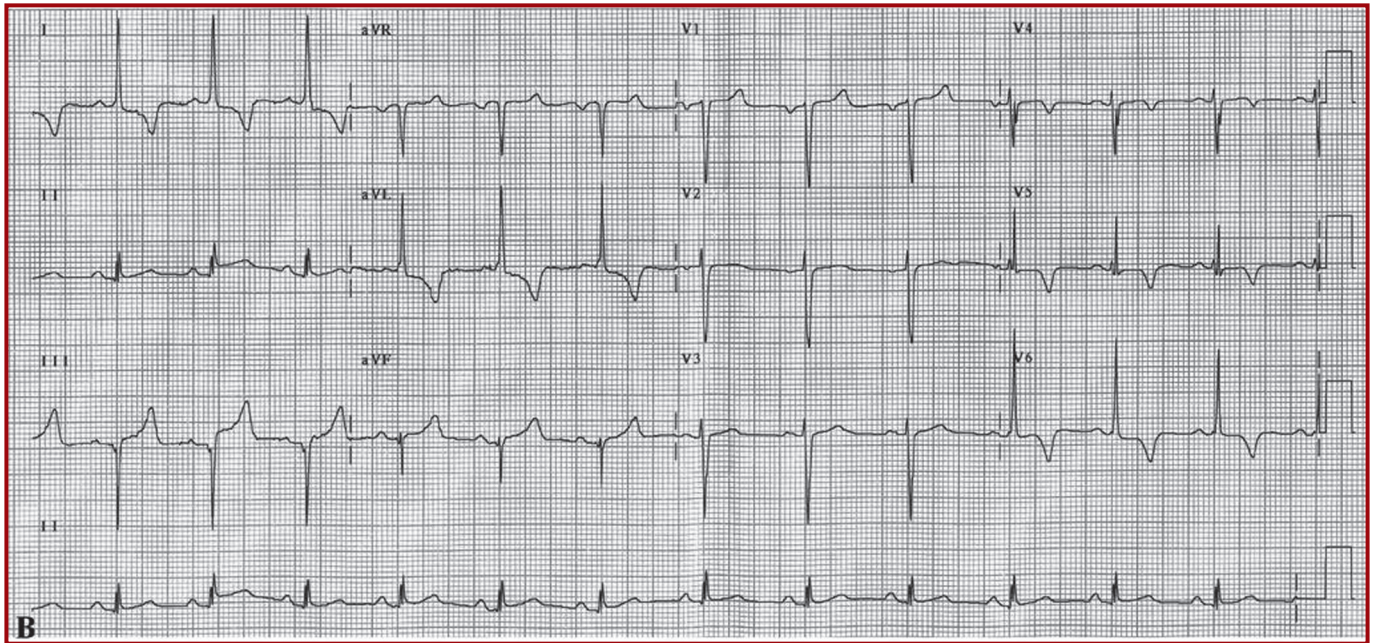
**Figure 2.** Coronary angiogram shows 70% stenosis in the mid-portion of the left anterior descending artery and 80% stenosis in the proximal segment of the left circumflex coronary artery (left). A "spade-like" configuration of left ventricle at the right anterior oblique view (right)

internal thoracic artery to the LAD and saphenous vein grafts to the obtuse margin branch and to the RCA) was performed 4 weeks later. Subsequent ECGs showed decreases in both R and S waves (SV1+RV5 27 mm) and negative T waves (3-6 mm). Negative T waves also returned to positive in leads V2-3 (Figure 3).

### Discussion

Although ApHCMP is not associated with sudden cardiac death and has a benign prognosis in terms of

cardiovascular mortality, one third of the patients experience serious cardiovascular complications, such as myocardial infarction, congestive heart failure and arrhythmias (7). There are only a few case reports about ApHCMP coexisting with CAD (5,6). Furthermore, most of the ApHCMP patients with myocardial infarction have normal coronary arteries (7). On the other hand, ApHCMP may be confused with myocardial ischemia or acute coronary syndrome (3,4). In our case, the T wave negativity primarily suggested



**Figure 3.** Decreases in R, S and negative T waves (3-6 mm) on the postoperative electrocardiogram

progression of CAD as the patient had a known history of CAD and PCI. Moreover, a significant stenosis of LAD was supporting this observation. However, left ventriculography showed an ApHCMCP coexisting with CAD. Our patient was treated with CABG. To our knowledge, this is the third case of CABG performed for CAD coexisting with ApHCMCP. Two similar cases have been previously reported (5,6).

Typical giant T wave negativity may decrease in some patients with ApHCMCP. Horita et al. have reported two cases of ApHCMCP, in whom giant T waves resolved during 10 years (8). The reason of this change was the development of dilated cardiomyopathy in one patient and CAD in the other. Eriksson et al. have also showed that the development of myocardial infarction usually causes loss of giant T wave negativity (7). Our case had no myocardial infarction or dilated cardiomyopathy, and decrease in T wave negativity and amplitudes of R and S waves were observed after CABG. The possible reasons of these changes might be results of CABG, such as decreasing myocardial ischemia due to CAD and/or anatomical changes in chest wall.

In conclusion, despite its low incidence, giant negative T waves on ECG may be considered as ApHCMCP even in the presence of documented coronary artery disease, and T wave negativity may decrease after revascularization.

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