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Case Report Open Access

Apical Hypertrophic Cardiomyopathy Presenting Mimicking Acute Coronary Syndrome at an Advanced Age

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Abstract

At the emergency room, advanced ages with chest pain and ST-T changes in the electrocardiogram (ECG) could be easily considered as acute coronary syndrome (ACS) and transferred to catheterization laboratory for urgent coronary angiogram (CAG). In our case, these patients were finally diagnosed as apical hypertrophic cardiomyopathy (AHCM) after urgent CAG subsequent to echocardiogram. AHCM is common in Asian populations and it represents a condition may mimic ACS. We report two elderly cases of AHCM who presented with chest pain mimicking an ACS in the emergency room and review the literature on the late development of AHCM.

Keywords: Acute coronary syndrome; Apical hypertrophic cardiomyopathy; Advanced age

Introduction

Chest pain and ST-T changes in the electrocardiogram (ECG) could be easily regarded as acute coronary syndrome (ACS) in the emergency room [1,2]. However, the correlation between the ST-T change in the ECG and late development of Apical Hypertrophic Cardiomyopathy (AHCM) is still unknown.

Case Report

Patient 1

A 71-year-old woman with chest pain was admitted at night in the emergency room. She had no risk factors of coronary artery disease (CAD). Normal finding of ECG and echocardiogram were already identified 5 years ago (Figure 1A and 1C). Persistent chest pain led to cardiac catheterization laboratory for urgent CAG due to dramatic change of ST-segment and T-wave in the anterolateral leads on ECG (Figure 1B). CAG showed normal coronary artery and the subsequent 2D echocardiogram showed, in both parasternal short axis and apical 4-chamber views, an asymmetric hypertrophy of left ventricular (LV) apex with a 15 mm maximum wall thickness at the age of 72 years (Figure 1D).

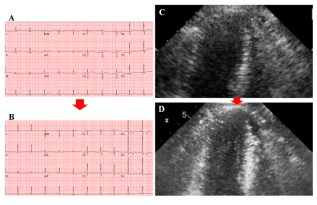

Figure 1: Normal finding of ECG and echocardiogram

Patient 2

A 69-year-old asymptomatic woman without risk factors of CAD was identified with a normal finding of ECG during routine health check-up in 2006 (Figure 2A). After 5 years, she visited emergency room at night due to severe chest pain. An ECG revealed changes of ST-segment and T-wave in the anterolateral leads with high R wave (Figure 2B). Urgent CAG also showed normal coronary artery and in particular, on 2D echocardiogram, both a parasternal short axis and an apical 4-chamber views of the LV noted asymmetric apical hypertrophy with a maximum wall thickness 19 mm at the age of 74 years (Figure 2C and 2D).

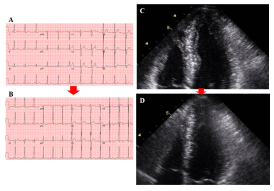

Figure 2: Normal finding of ECG during routine health check-up

Discussion

AHCM was first reported in Eastern Asia,where it is represented by 13% to 25% of the whole HCM. In the Western country, AHCM is less common and has been described in 3% to 11% of all HCM patients [3,4]. AHCM is a form of HCM which has a peculiar geographic distribution, being a common morphologic expression in middle-aged Asian people but relatively rare in Western ones **4**. AHCM was morphologically defined as focal LV hypertrophy confined to the most distal region of the apex below the papillary muscle level. In addition, ECG findings such as a ST change, T wave inversions and high voltage QRS complexes in precordial leads could serve as sensitive diagnostic marker in patients with AHCM even though ECG changes that occur throughout the progression of AHCM may evolve over several years

[**5-7**]. However, the clinical features of AHCM are variable with 30–40% of patients reported to be asymptomatic and the most frequently encountered symptom is chest pain [**4**]. Even if the pathophysiology of angina remains unclear, the AHCM could be the result of LV volume overload by the coronary artery–LV shunt or could be the cause of multiple coronary artery microfistular, possibly due to disarray of myocardial cells. Therefore, the main mechanism of myocardial ischemia is the coronary steal phenomenon. In addition, AHCM can also cause ischemia by altering the oxygen demand and supply balance to the hypertrophic myocardium [**4**,**8**].

Yamaguci et al. [**3**] suggested that abnormal change in the hypertrophic process of the myocardium becomes manifested during middle-age in response to unknown stimuli, assuming that the disease process is entirely based on genetic factors. So far, many previous reports revealed that these patients present at a relatively middle age with T wave inversions of ECG that frequently result in hospitalization for suspected CAD [**1**,**2**]. In general, phenotypic expression of AHCM in these patients seems to evolve over several years at a relatively middle age. However, the occurrence of late, and relatively abrupt, development of AHCM at an advanced age has been rarely reported. In addition, the late development of AHCM in the elderly may represent an example of gene-environmental interaction required for phenotypic manifestations. Although it is not easy to prove causality of development of AHCM, Chung et al. [**9**] suggested that the sustained hypertension may be a triggering factor in the late development of AHCM. In our two cases, no triggering pathologic factor such as hypertension was noted in the rapid development of AHCM at the relatively advanced age. The ECG finding showed newly appeared ST-T change or T wave inversion. However, biochemical analysis of biomarkers of myocardial damage from these two patients showed within normal range. Final echocardiogram finding established a correct diagnosis of AHCM which is characterized by LV thickening confined to the apex (Figures 1 and 2). In emergency room, echocardiogram guided by experienced medical personnel is required for the advanced ages with chest pain and ST-T change on the ECG may be helpful on decision-making for correct diagnosis and treatment.

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