

Case Report

Hypertrophic cardiomyopathy comorbid with myocardial infarction: a case report and literature review

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Received May 22, 2017; Accepted October 18, 2017; Epub January 15, 2018; Published January 30, 2018

Abstract: Hypertrophic cardiomyopathy (HCM) is a clinically and morphologically heterogeneous genetic cardiovascular disease that is often associated with unfavourable prognosis, unrelenting progression, and premature death. Unfortunately, current treatment strategies are ineffective. HCM-associated coronary artery disease (CAD) is an extremely rare type of lesion that is regarded as the most common non-traumatic cause of sudden death among young adults. In the current case, a 31-year-old man complaining of typical exertional dyspnoea and chest distress for 3 days was admitted to the hospital in May 2016. Electrocardiogram (ECG), echocardiography excision, and laboratory examinations were performed. Coronary angiography (CAG) revealed diffuse plaque infiltration throughout the coronary arteries. The patient was diagnosed with HCM-associated CAD via ECG, echocardiography, and CAG. Blood flow improved after percutaneous coronary intervention in the middle segment of the right coronary artery. The patient did not appear to suffer from chest pain or other symptoms during the follow-up period.

Keywords: Hypertrophic cardiomyopathy, myocardial infarction, coronary angiography, hypercholesterolemia

Introduction

Hypertrophic cardiomyopathy (HCM), an inherited cardiomyopathy that occurs in approximately 1 in 500 individuals, is characterized by left ventricular hypertrophy (LVH) without underlying pressure overload [1]. HCM is the most common non-traumatic cause of sudden death among young adults. Assessments of the clinical outcomes of patients with HCM have indicated that annual HCM-related mortality rates range from 3% to 6%; however, the mortality rate is significantly elevated among HCM patients with coronary artery disease (CAD). Angina pectoris and myocardial infarction caused by atherosclerotic CAD are being reported with increasing frequency among HCM patients [2]. The clinical symptoms of HCM are highly variable and include asymptomatic to severely limiting dyspnoea and/or chest pain, atrial fibrillation, and ventricular arrhythmia. The subset of HCM patients with chest pain may have angina pectoris or even myocardial infarction (MI). A previous study showed that 19% of HCM patients with CAD exhibited narrowing of the luminal diameter by >60% in one or more coronary arteries [3]. HCM is difficult to diagnose

and treat when the disease is comorbid with other heart diseases, such as CAD. HCM patients and CAD patients typically present with similar clinical symptoms and ECG changes due to the increased oxygen demand of the hypertrophied ventricular myocardium in HCM. Patients with HCM are more likely to present with non-ST-elevation MI [4]. In HCM patients with left ventricular (LV) outflow tract obstruction, the metabolic demand is further increased due to the greater workload caused by substantially elevated intraventricular pressure [5]. In cases of HCM, clinically asymptomatic or atypical chest pain may be misleading and result in a failure to recognize coexisting CAD. Here, we report a rare case of an HCM patient with CAD who was only 31 years of age.

Case report

In May 2016, a 31-year-old man presented to our centre at the Second Affiliated Hospital of Harbin Medical University with exertional dyspnoea and chest distress 3 days prior to admission. The dyspnoea and chest distress had been triggered 3 days prior by exertion and after meals, and these symptoms were relieved

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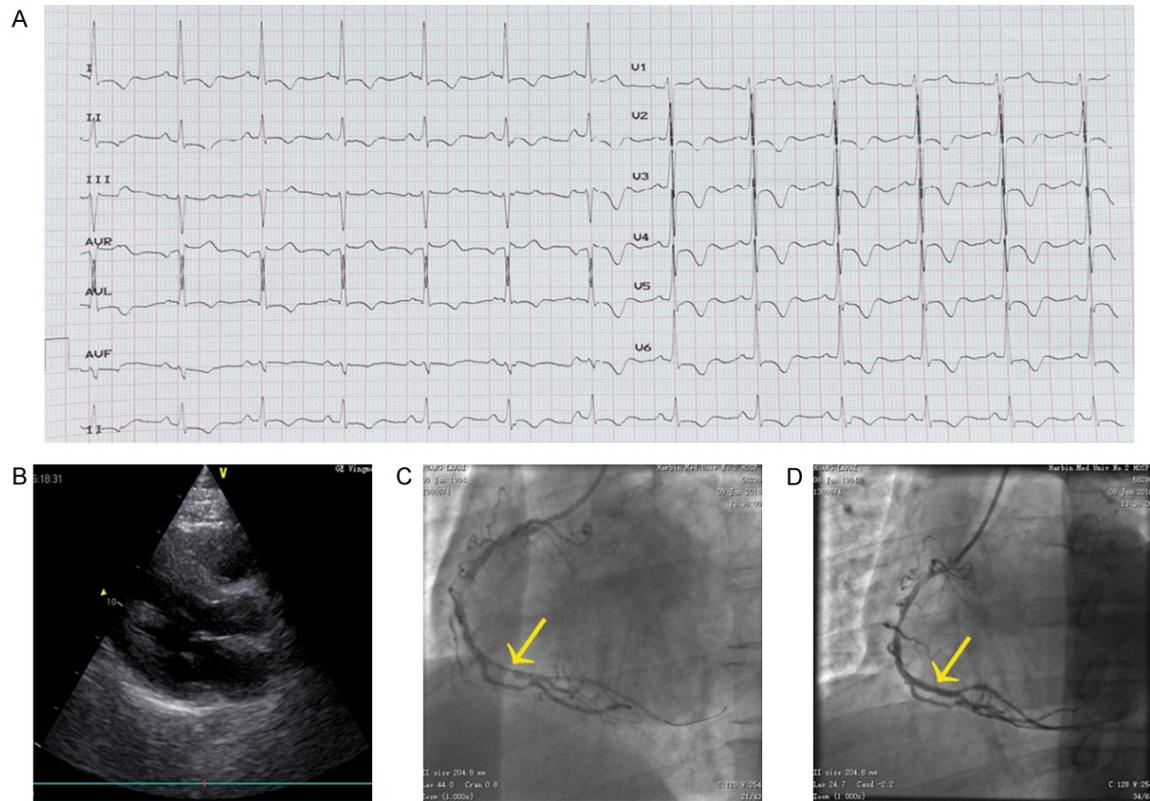


Figure 1. Hypertrophic Cardiomyopathy-associated Myocardial Infarction. A. An electrocardiogram revealed ST depression and deep T wave abnormalities in leads I, II, aVL, and V2-V6. B. The echocardiogram obtained at admission demonstrates significant non-obstructive hypertrophic cardiomyopathy. C. Coronary angiography before percutaneous coronary intervention. The arrowhead indicates the site of occlusion in the right coronary artery. D. Coronary angiography after percutaneous coronary intervention showing that the right coronary artery occlusion had been resolved.

after rest. The patient had a history of hypercholesterolemia. The patient had smoked 10 cigarettes daily for the past 10 years and had a history of mild drinking. A physical examination revealed a blood pressure of 140/80 mmHg. A prescribed 12-lead electrocardiogram (ECG) revealed sinus rhythm (heart rate: 76 beats/min), ST depression, and deep T wave abnormalities in leads I, II, aVL, and V2-V6 (**Figure 1**). Echocardiography revealed LV hypertrophy that was predominantly in the basal septal segments and no LV outflow tract obstruction or systolic anterior motion (SAM). The thicknesses of the interventricular septum and LV posterior wall were 19.3 mm and 17.2 mm, respectively (**Figure 1**). Left ventricular volume and left ventricular ejection fraction (LVEF) were normal (LVEF was 66%), but regional contraction was altered in the hypertrophic segments. The patient was diagnosed with non-obstructive HCM.

A laboratory examination revealed a white blood cell count of 12.4×10^9 cells/L, a creatine kinase level of 1,256 U/L (normal range, 24-200 U/L), a creatine kinase isoenzyme MB (CK-MB) mass level of 16.4 $\mu\text{g/L}$ (normal, <3.6 $\mu\text{g/L}$), and a B-type pro-brain natriuretic peptide level of 452 pg/ml (normal, <125 pg/ml). Based on the laboratory test results, we performed coronary angiography (CAG), which revealed diffuse plaque infiltration throughout the coronary arteries as well as 50% stenosis in the proximal segment of the left anterior descending (LAD) artery; 70% stenosis in the proximal left circumflex (LCx) artery and the middle segment of the right coronary artery (RCA); and 90% occlusion in the distal segment of the RCA (**Figure 1**). Blood flow improved after percutaneous coronary intervention (PCI) in the middle segment of the RCA (**Figure 1**). After this procedure, the patient did not experience chest pain or exhibit any ST-T changes

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on ECGs. The patient did not appear to suffer from chest pain or other symptoms during the three-month follow-up period.

Discussion

In almost all HCM patients, cardiac enzyme levels are elevated although plaque rupture or stenosis is not observed by coronary angiography. A suggested mechanism is that increased wall stress leads to coronary spasm and squeezing, and the decreased coronary flow in the apex of the left ventricle then causes microvascular disorder, which subsequently leads to MI [6]. HCM coexisting with CAD was originally described in 1972 and is an extremely rare type of lesion that is regarded as the most common non-traumatic cause of sudden death among young adults. Revascularization is, however, performed in only a small number of patients with HCM. Timely CAG is the gold standard for diagnosing HCM comorbid with CAD. Our young patient's clinical symptoms and cardiac function were clearly alleviated after blood flow was improved in his RCA. Clinical recommendations suggest that all patients older than 45 years of age, particularly patients with angina pectoris or hyperlipidaemia, should undergo CAG in addition to a haemodynamic evaluation. Failure to recognize coexisting CAD may be responsible for the incomplete relief of angina and an increased rate of late postoperative mortality.

Levels of both natriuretic peptides and troponins predict the clinical risk of HCM independent of established risk factors [7]. In this young patient, increased trends were observed for CK-MB mass as well as troponin and natriuretic peptide levels. Perak et al. [8] reported that the burden of long-term atherosclerotic cardiovascular disease (ASCVD) was related to phenotypic heterozygous familial hypercholesterolemia (FH), which was defined as a low-density lipoprotein cholesterol level ≥ 190 mg/dL. Our patient's low-density lipoprotein cholesterol level was 7.25 mmol/L (normal range, 0.45-3.15 mmol/L), and his family members had also been diagnosed with hypercholesterolemia. Additionally, the patient had smoked cigarettes for 10 years. These characteristics may be one reason why this young man experienced MI and may explain the accelerated progression of HCM in combination with CAD. Given that hypercholesterolemia increases the risk for coronary heart disease, we consider

this marker to be important and suggest timely blood lipid measurements to enable the diagnoses of HCM comorbid with CAD.

In summary, HCM is difficult to diagnose and treat, especially when the disease is comorbid with other heart diseases, such as CAD. In the present case, a young man was diagnosed with HCM combined with CAD. His clinical symptoms and cardiac function were significantly alleviated after blood flow was improved in his RCA. Timely CAG is the gold standard for diagnosing HCM comorbid with CAD. Clinical recommendations suggest that all patients older than 45 years of age, particularly patients with angina pectoris or hyperlipidaemia, should undergo CAG in addition to a haemodynamic evaluation. Measurements of biomarkers may also help recognize and improve the management of HCM coexisting with CAD.

Acknowledgements

This work was supported by the National Natural Science Foundation of China (81771946, 81571749) and the Natural Science Foundation of Heilongjiang Province of China (H2015048).

Disclosure of conflict of interest

None.

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