

A Rare Cause of Acute Myocardial Infarction: The Coronary Artery Ectasia

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Introduction

Coronary artery ectasia (CAE) is characterized by an abnormal dilatation of coronary arteries. More than half of CAE is due to atherosclerosis, and thus it has been considered as a variant of atherosclerotic coronary artery disease (CAD). It may result in angina pectoris, even in myocardial infarction due to impaired coronary blood flow [1-3]. In this report, we present a patient who developed ST elevation myocardial infarction due to multivessel ectasia.

Case Report

A 46 year-old male patient presented with severe, squeezing chest pain of an hour onset. ST-segment elevation was detected in DII, DIII, aVF, V5 and V6 and ST-segment depression was detected in V1, V2 and D1, aVL. Age, obesity and smoking were present as risk factors for coronary heart disease. On physical examination, heart rate was 77/min and arterial blood pressure was 140/80 mmHg. There were no pathologic findings except for a 2/6 degree systolic murmur on the mitral auscultation area. On his electrocardiogram, ST-segment elevation detected in DII, DIII, aVF, V5 and V6 derivations (Figure 1). Cardiac enzymes levels were elevated. Transthoracic echocardiography revealed a left ventricular ejection fraction of 50% and hypokinesia in inferior, posterior and lateral ventricle free walls. The patient was admitted to the coronary intensive care unit with the diagnosis of acute coronary syndrome. He underwent primer coronary angiography with the diagnosis of acute ST elevation myocardial infarction. Coronary angiography revealed coronary artery ectasia involving left main coronary artery, left anterior descending artery and circumflex coronary artery. Right common artery was normal. There was no stenotic lesion in coronary arteries (Figure 2). He underwent subcutaneous enoxaparin, oral aspirin, clopidogrel, atorvastatin metoprolol and ramipril. His chest pain did not recur following medical therapy in the coronary intensive care unit. Patient's symptoms rebounded significantly after anticoagulant, antiaggregant, and anti ischemic therapies. Then, ECG gradually showed the resolution of ST-segment elevation with Q-wave in the inferior leads (Figure 3). Creatine kinase (CK)-MB fraction of CK (CK-MB) and Troponin T values rose to 661 IU/L (normal range: 0-171 U/L), 37 U/L (normal range: <24 U/L) and 1,29 ng/mL (normal range: <0.04 ng/mL), respectively. Other hematological and biochemical tests revealed the following: prothrombin time 11 second (normal range: 10-14 second), and activated partial thromboplastin time 29 second (normal range: 25-36 second). Total cholesterol 221 mg/dL (normal range: 110-200 mg/dL), triglyceride 252 mg/dL (normal range: 0-200 mg/dL).

Discussion

CAE, or aneurysmal coronary artery disease, is defined as dilatation of an arterial segment to a diameter at least 1.5 times that of the adjacent normal coronary artery. CAE can be found in up to 5% of angiographic and in 0.22% to 1.4% of autopsy series [4-7]. It can be either diffuse, by affecting the entire length of a coronary artery, or localized. When the dilatation involves the entire vessel the word "ectasia" is used instead of aneurismal disease. Coronary artery ectasia or aneurysm is attributed to atherosclerosis in 50% of cases, whereas 20-30% has been considered to be congenital in origin. In the great majority of these

patients ectasia coexists with coronary artery disease. Only 10% to 20% of cases of CAE have been described in as-association with inflammatory or connective tissue diseases [1,5,6]. The presence of aneurysmal segments produces sluggish or turbulent blood flow, with increased incidence of typical exercise induced angina pectoris and myocardial infarction, regardless of the severity of coexisting stenotic coronary disease. This is due to the repeated dissemination of microemboli to segments distal to the ectasia, or to thrombotic occlusion of the dilated vessel [7,8]. Slow blood flow in the coronary artery may also be a causative factor [9]. Patients with pure ectasia [15% of the total population with CAE] have a more benign course, but 39% of them still present signs of previous myocardial infarction [9]. There is a higher incidence of adverse events in this population compared to people with normal coronary arteries [5]. Markis et al. [5] classified CAE in four types: type 1 includes diffuse ectasia involving two or three vessels, type 2 includes diffuse ectasia involving one vessel and discrete ectasia in another, type 3 includes diffuse ectasia in only one vessel, and type 4 includes localized or segmental ectasia in only one vessel. When our case was considered, the patient had presence of multiple ectasia in two major epicardial coronary arteries without any obstructive lesion. Based on these findings, he was accepted as having type 2 CAE.

The clinical spectrum of CAE is variable, including stable angina pectoris, unstable angina pectoris, vasospastic angina, and myocardial infarction. The most common symptom is exertional angina [5,10]. Tendency to thrombosis due to diminished coronary flow and vasospasm due to structural changes in the vessel wall may cause chest pain and even myocardial infarction [11].

In our case, collagen tissue diseases and malignancy, which are known to cause in situ coronary thrombosis, were excluded. No abnormality in blood coagulation tests was detected, as well. The patient was asymptomatic before the diagnosis of ST elevation myocardial infarction, which was understandable with the advanced age and diminished physical activity of the patient. After the diagnosis of ST elevation myocardial infarction, he was treated with anticoagulant, antiaggregant, and antiischemic therapies: β -blocker therapy was applied for antiischemic therapy. An angiotensin converting enzyme inhibitor and a statin were added to treatment due to their therapeutic effects on the endothelial dysfunction. After discharge, he described no acute chest pain or chronic exertional angina under medical therapy for three months.

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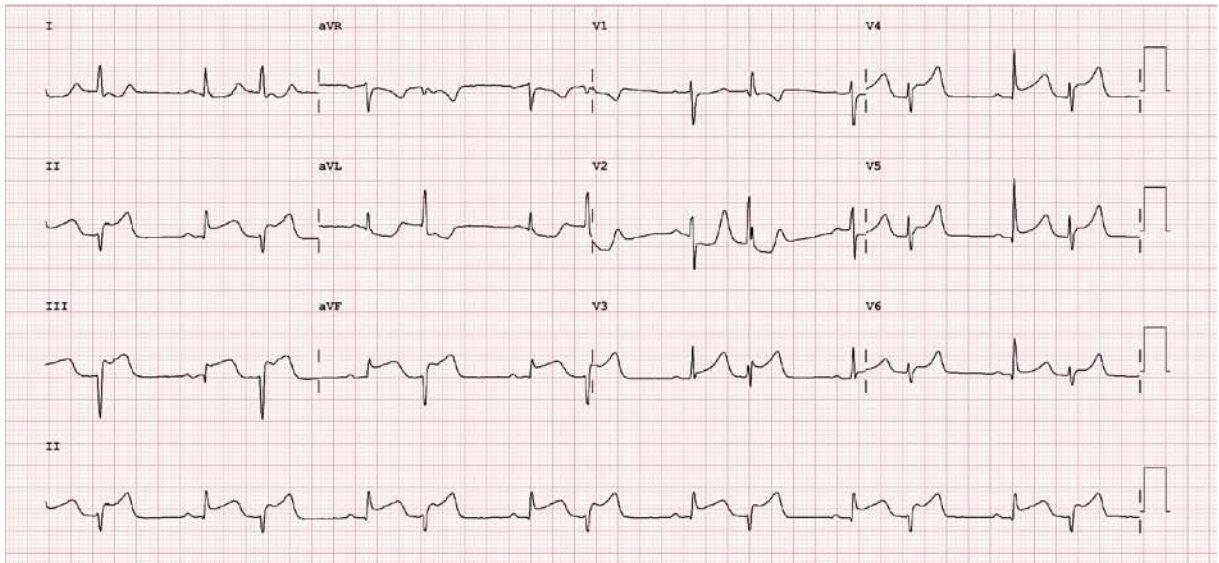


Figure 1: Electrocardiogram shows sinus rhythm and ST- segment elevation in the emergency room.

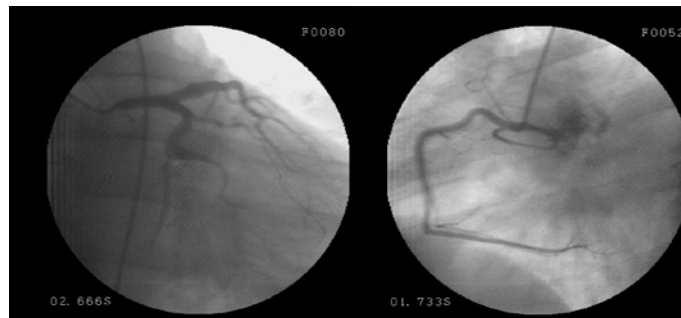


Figure 2: Coronary angiography revealed coronary artery ectasia involving left main coronary artery, left anterior descending artery and circumflex coronary artery. Right common artery was normal.

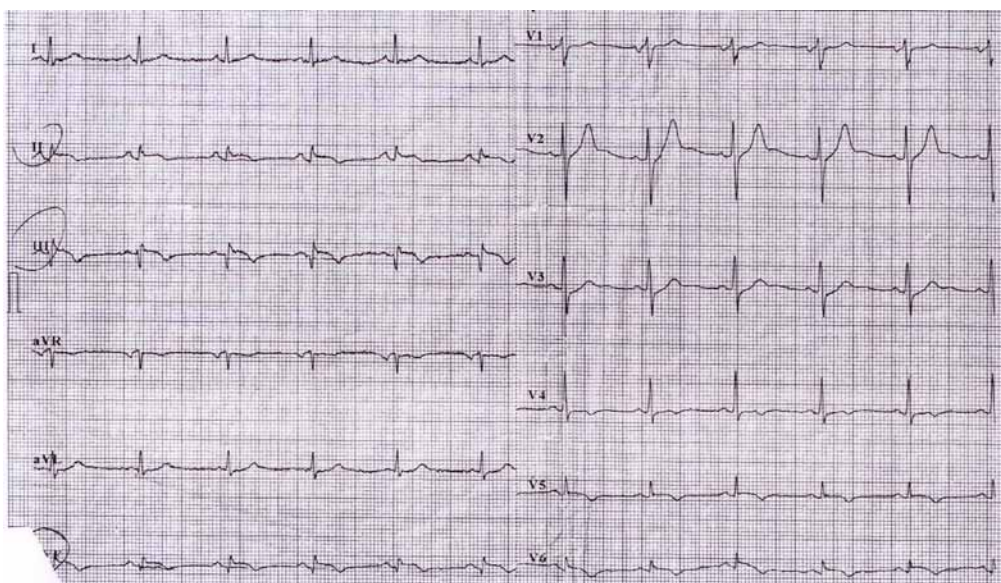


Figure 3: Nearly complete resolution of ST-segment elevation with Q-wave in the inferior leads after medical therapy.

In conclusion, coronary ectasia should not be considered merely dilatation of the vessel wall, because it may lead to cardiovascular complications. It is one of the less frequent causes of myocardial infarction and management of this angiographic entity requires meticulous medical therapy.

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