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Coronary Artery Ectasia with Acute Myocardial Infarction: A Case Report

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Abstract

Coronary artery ectasia (CAE), a rare condition characterized by abnormal dilation of the coronary arteries, has increasingly been recognized as a potential risk factor for acute myocardial infarction (AMI). While CAE is often asymptomatic, it can predispose individuals to thrombus formation, leading to occlusions and subsequent myocardial damage. Our case is a 36-year-old male with no prior medical problem who presented with chest pain and shortness of breath and was diagnosed as a non-STEMI. The coronary angiogram revealed multiple ectatic segments in the coronary artery with 100% stenosis in the circumflex branch. Coronary artery ectasia represents an emerging risk factor for acute myocardial infarction. The complex relationship between these two conditions is essential for improving diagnosis, prevention, and treatment strategies. Presentation of CAE is usually asymptomatic but when present with occlusive symptoms, management is guided by the extent of occlusion similar to coronary artery disease.

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Introduction

Coronary artery ectasia (CAE) is defined as the abnormal dilatation of the epicardial coronary artery exceeding 1.5 times than the normal adjacent coronary artery.[1] Coronary artery ectasia (CAE) was described in 1761 by Morgagni. CAE is an infrequent coronary angiographic finding with a frequency ranging from 1.5% to 5% varied by region.[2-4] CAE has clinical presentations and implications similar to CAD, such as stable angina and acute coronary syndrome (ACS) secondary to coronary thrombus formation or vasospasm.[5,6] The etiology of coronary artery ectasia can be mysterious. Atherosclerosis claims to be the principal etiologic in adults, while Kawasaki disease is the most common cause in children and young adults.[7] Studies have reported that 85-91% of patients with CAE were found to have CAD.[8,9] Most of the patients are asymptomatic, while some may present with symptoms of CAD.[10] Here we present a case of a male with CAE who presented with symptoms of non-ST elevation myocardial infarction (NSTEMI).

Case Presentation

A 36-year old male patient, who was non-diabetic, normotensive, smoker, hailing from Sadar Upazila, Mymensingh, was admitted into our hospital and presented with severe chest pain for 8 hours, which was at the center of the chest. The patient also had complains of shortness of breath, which was more marked on exertion and not relieved by rest. On examination, the heart rate was 90/min., blood pressure was 100/70 mmHg, respiratory rate was 30 breaths/min, and basal crepitations were present in the both lungs. His initial ECG showed an ischemic change in the inferior and lateral lead (Fig. 1). His serum troponin level was elevated at 9.00 ng/ml (normal level:<0.04ng/l) An echocardiogram showed fair left ventricular function (LVEF=53%) with inferior

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and lateral hypokinesia (Fig. 2). The brain natriuretic peptide (BNP) level was 373 pg/ml (Normal level <100 pg/ml) Liver function tests and serum creatinine were found normal. The patient was admitted with a working diagnosis of non-ST segment elevation myocardial infarction (NSTEMI). Coronary angiography showed that the RCA – long ectatic vessel in its proximal to distal; LAD – ectatic vessel and 70-80% stenosis in its proximal segment; LCX – total occlusion in its distal segment (Fig. 3). PCI was challenging due to the necessity of using large-size devices. The patient was referred to a specialized cardiac center for surgical coronary artery vascularization with coronary artery bypass grafting (CABG) and further management.

Fig.1: Inferior and lateral ischemia as seen in ECG of the patient.

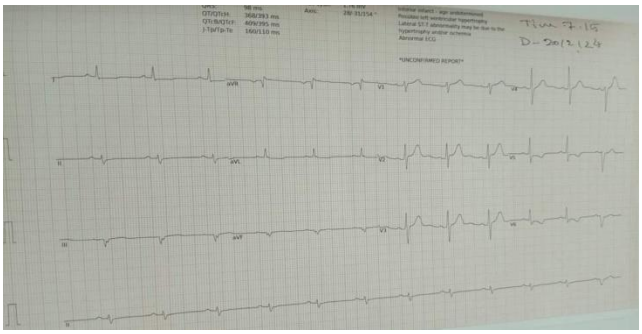
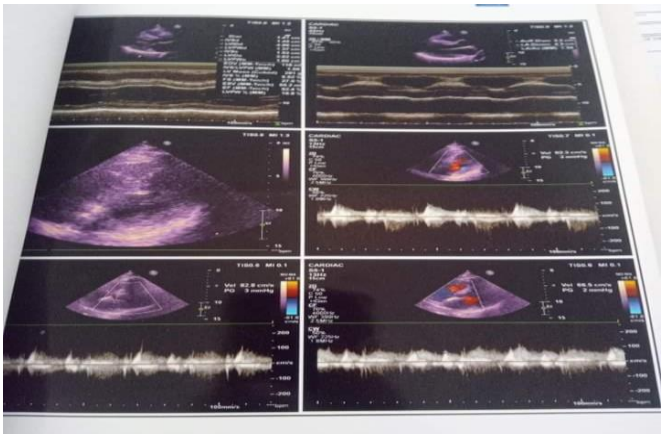


Fig. 2: Fair left ventricular function



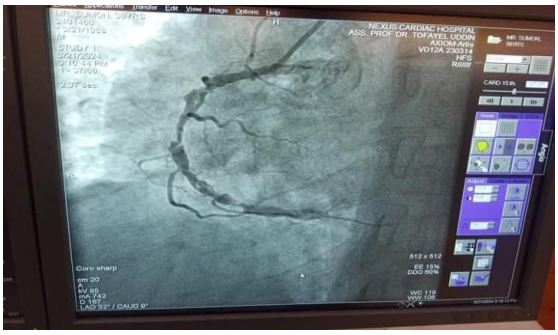


Fig. 3: Coronary angiography shows – a) RCA Long ectatic vessels proximal to distal; b) LAD ectatic vessels and 70-80% proximal stenosis; and c) LCX total occlusion of the left circumflex distal segment.

Discussion

The definition of CAE includes dilation of the coronary artery involving a diameter greater than the adjacent normal coronary artery by 1.5 times Based on the morphology and the extent of the coronary arteries' involvement, we can divide CAE into different groups. Classification based on morphology includes the

following types[11]:

1. **Saccular:** the transverse diameter is more than the longitudinal dimension.
2. **Fusiform:** the transverse diameter is lower than the longitudinal dimension. Classification based on the extent includes the following types:
   a) Type 1: diffuse ectasia involving aneurysmal lesions in two vessels
   b) Type 2: discrete ectasia in one vessel with diffuse ectasia in another vessel
   c) Type 3: one vessel having diffuse ectasia
   d) Type 4: one vessel having discrete ectasia

In a single observational study involving approximately 5,000 patients, CAE was reported mostly in proximal-mid-RCA (68%), followed by proximal LAD in 60% of cases, and LCX in 50%. Left main coronary artery ectasia (LMCA) is an infrequent occurrence, which accounts for 0.1% of cases.[12,13] In both children and adults, the most common cause was found to be Kawasaki disease (widespread inflammation of primarily medium-sized muscular arteries).[14] However, the exact pathophysiology of coronary artery ectasia is unknown. Coronary artery ectasia appears to be exaggerated expansive remodeling of the external elastic membrane resulting in luminal expansion. Enzymatic degradation of the extracellular matrix by matrix metalloproteinases and other lytic enzymes and thinning of the tunica media associated with severe chronic inflammation is the principal pathogenesis of expansive remodeling.[15] CAE is also related to apical hypertrophic cardiomyopathy with high wall tension. In iatrogenic mechanisms, e.g., percutaneous coronary interventions, including balloon angioplasty, stent placement, and atherectomy, can lead to the formation of aneurysms or ectasias. The mechanism is the injury of the media of the blood vessel.[16] In general, males are more prone to CAE than females, and the biggest risk factor is hypertension. A low incidence of CAE has been found in diabetic patients. This could be attributed to negative remodeling and matrix metalloproteinases (MMP) downregulation in response to atherosclerosis.[17] The habit of smoking has been associated with patients suffering from CAE than patients with CAD. Regardless of smoking, another independent predictor of CAE is cocaine use.[18]

Coronary angiography is the gold standard procedure for diagnosing CAE. The disturbances in washout and blood flow filling related to the severity of ectasia can be shown with the help of angiography. The local dye deposition in the dilated coronary segment and the segmental backflow phenomenon known as delayed antegrade dye filling are the signs of stagnant and turbulent flow signs indicated in angiography.[19,20] Nowadays, CAE can be diagnosed with the help of other modalities such as coronary CT angiogram (CTA) and coronary magnetic resonance angiogram (MRA).[21] In designing a treatment plan for patients with CAE, all risk factors must be taken into consideration along with the coexisting diseases. Treatment is directed towards risk reduction (management of atherosclerosis and hypertension management of CAD and obstructive lesions if found; thus, treatment can include medical, angioplasty, and surgical modalities.[22,23] Percutaneous coronary intervention (PCI) can be used for patients with obstructive CAD with CAE when medical treatment fails.[24] Our patient was treated with medical therapy as non-ST elevated myocardial infarction. After one month CAG was done and diagnosed with triple vessel disease. As PCI was challenging due to the necessity of using large-size devices, we chose surgical coronary artery vascularization with coronary artery bypass grafting (CABG).

Conclusion

CAE is an uncommon condition CAE may present with the classic symptoms of AMI, including chest pain, dyspnea, diaphoresis, and nausea. The precise mechanisms underlying CAE-associated AMI are not fully understood. The diagnosis of CAE in patients with AMI can be challenging, Coronary angiography is often required to visualize the ectatic segments of the coronary arteries. The management of CAE-associated AMI is generally similar to that of AMI. Further research is necessary to elucidate the underlying mechanisms and develop targeted therapeutic approaches for patients with CAE-associated AMI.

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