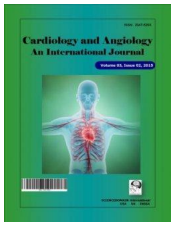

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Severe Coronary Artery Ectasia with ST Elevation MI: A Challenging Situation

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aim: To report a case of huge coronary artery ectasia presenting with acute myocardial infarction; a relatively rare finding encountered during coronary angiography.
Presentation of Case: A young male presented with chest pain and profuse sweating at a local hospital. Electrocardiogram showed Infero-posterior STEMI. Patient received streptokinase. His symptoms settled however the electrocardiogram changes did not resolve. He presented at our hospital after 24 hours with chest discomfort. He was vitally stable and a murmur of MR was audible. His Troponin-I was raised and electrocardiogram showed ST elevations with Q waves. Coronary angiogram showed giant ectasia and occluded right coronary artery (RCA). Percutaneous coronary intervention of RCA was done; with TIMI II flow but still had some residual thrombus. The patient was kept on Tirofiban infusion. His CRP and homocysteine levels were raised. Dual antiplatelet, statin, ACE Inhibitor, beta blocker with vitamin B12 and folic acid supplement were continued.
Discussion: Coronary artery ectasia is a form of atherosclerosis seen in 0.3–4.9% of coronary angiography procedures. It is described as dilation of the coronary arteries >1.5 times compared to adjacent normal vessel. An excessive expansive remodeling with enzymatic degradation of the extracellular matrix is considered to be the major pathophysiologic process. Clinical importance

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inclines on its association with acute coronary syndrome.
Conclusion: A case of huge coronary artery ectasia presenting with acute myocardial infarction and successfully treated with PCI.

Keywords: ST elevation myocardial infarction (STEMI); coronary artery ectasia (CAE); percutaneous coronary intervention (PCI).

1. INTRODUCTION

Coronary artery ectasia (CAE) is a well-recognized but relatively rare finding faced during diagnostic coronary angiography [1–3]. It is commonly described as inappropriate dilation of the coronary arteries exceeding more than 1.5 times the largest diameter of an adjacent normal vessel [1]. Although several mechanisms are proposed; the pathophysiology of CAE is still underrecognized. Similarly no consensus stands about the natural history and management of this condition due to scarcity of data.

2. PRESENTATION OF CASE

A 37 year old man; presented with history of chest pain, vomiting and profuse sweating at a local hospital. Chest discomfort lasted for four hours. ECG was done that revealed Infero-posterior wall ST-Elevation MI. He received streptokinase at the local hospital and his symptoms settled, however ECG changes did not resolve. He was referred to a tertiary care hospital after 24 hours with mild chest discomfort. He had a sedentary life style and history of 20 pack year smoking and tobacco chewing. He denied any other addiction. His family history was negative for premature ischemic heart disease. He was not taking any regular medicines before this event.

Clinical examination of this young overweight man (BMI-29) revealed pulse of 110 beats/minute, blood pressure of 130/70 mmHg. Jugular veins were not distended. Cardiac auscultation revealed 2/6 pansystolic murmur at apex radiating to axilla and chest examination was normal.

Blood tests showed normal complete blood count, urea, creatinine and electrolytes. His Troponin-I was raised (106 ng/ml) and electrocardiogram (ECG) showed ST elevations in inferior leads along with Q waves (Fig. 1). Patient received antiplatelets, statin and heparin in emergency department. We decided to proceed with coronary angiogram and revascularisation due to recurrence of symptoms and persistant ST elevations. Coronary angiogram showed giant ectasia of left anterior descending (LAD) artery and right coronary artery (RCA) (Figs. 2 and 3). The left circumflex artery (LCx) was normal. The ectatic right coronary artery was full of thrombus and totally occluded from distal segment before dividing into posterior descending artery (PDA) and posterior left ventricular branch (PLV) (Fig. 3). RCA was engaged with Judkins Right 4 (JR4) 6 French guiding catheter. PDA was wired with Runthrough Intermediate wire. Manual aspiration was done with thrombuster catheter. Large amount of thrombus was extracted. After thrombus extraction, PLV was faintly visible. The PLV branch was wired with another Runthrough Intermediate wire, manual aspiration of thrombus was done and the thrombus was extracted. After extracting large amount of thrombus the blood flow in distal RCA improved however there was still some residual thrombus (Fig. 4). Patient was kept on glycoprotein IIb/IIIa inhibitor (Tirofiban) infusion for 48 hours and remained asymptomatic post procedure. The patient was worked up for cause of coronary artery ectasia. He was found to have raised CRP and homocysteine levels; 20 mg/dl and 45.2 micromoles/dl respectively. His vitamin B12 level was within normal range. Autoimmune profile and syphilis serology were negative. Dual antiplatelet therapy, statin, ACE Inhibitor and beta blocker were continued. Vitamin B12 and folic acid were added to his treatment. He underwent an echocardiogram that revealed mild left ventricular dysfunction with ejection fraction of 45% and segmental wall motion abnormalities correlating to the right coronary artery territory. Moderate mitral regurgitation with regurgitant fraction 41% and structurally normal valve was observed. The patient was subsequently discharged from the hospital in stable condition. He was counseled with regards to smoking cessation and life style modification. At follow-up clinic visit (after two weeks) he is stable and asymptomatic.

3. DISCUSSION

Coronary artery ectasia (CAE) is defined as localized or diffuse dilatation of coronary artery lumen exceeding the largest diameter of an adjacent normal vessel >1.5 times [1]. The reported incidence of CAE varies between 0.3–4.9% of patients undergoing coronary angiography procedure [1,3]. CAE detection rate may rise with the use of modern non-invasive imaging technologies like computed tomography and magnetic resonance coronary angiography [4].

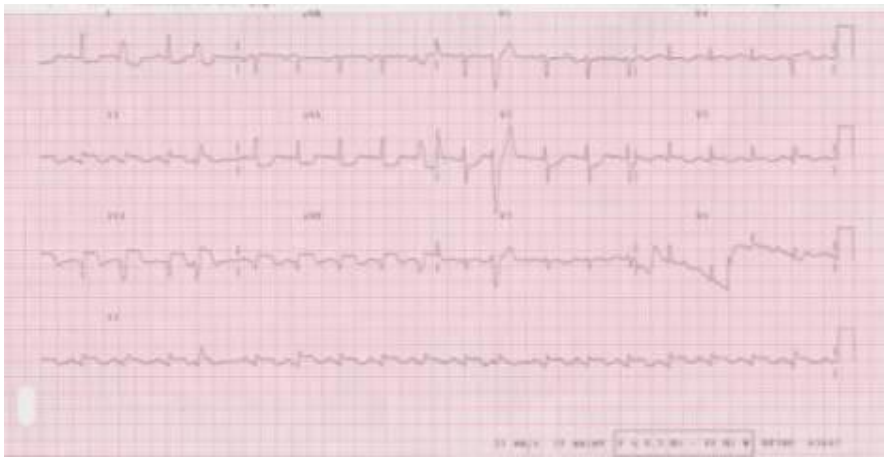

Fig. 1. Electrocardiogram showing ST segment elevations in inferior leads along with Q waves and reciprocal changes in lateral leads

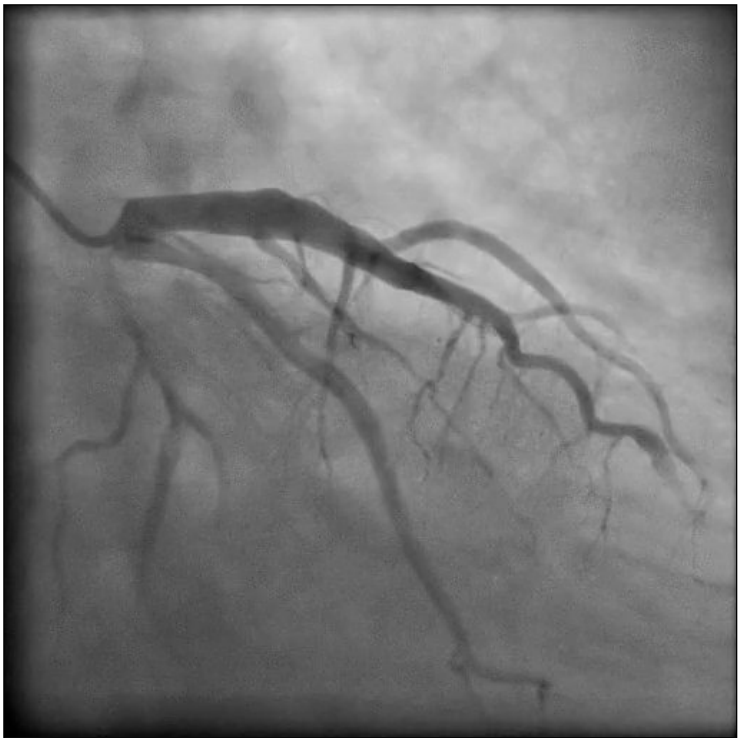

Fig. 2. Coronary angiogram showing ectatic left anterior descending artery

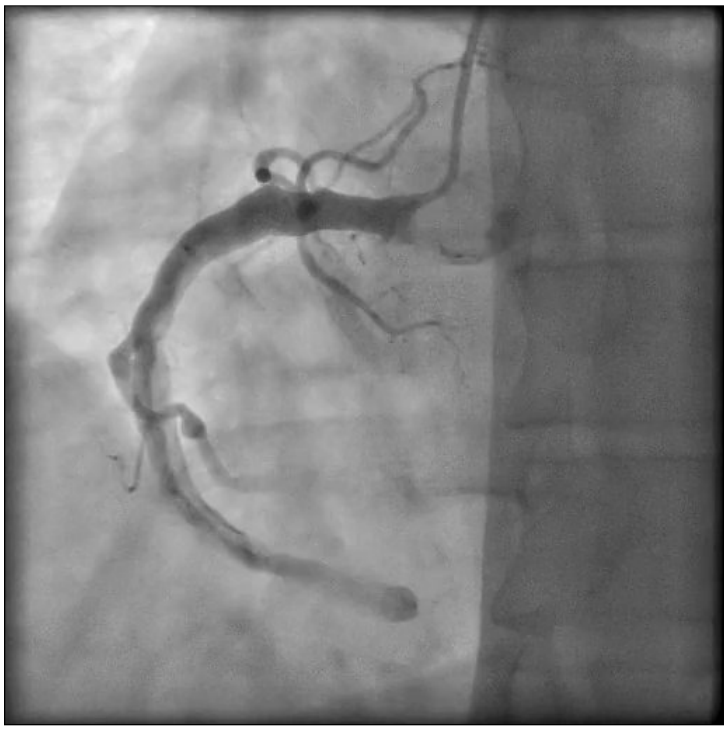

Fig. 3. Coronary angiogram showing ectatic right coronary artery filled with thrombus and totally occluded

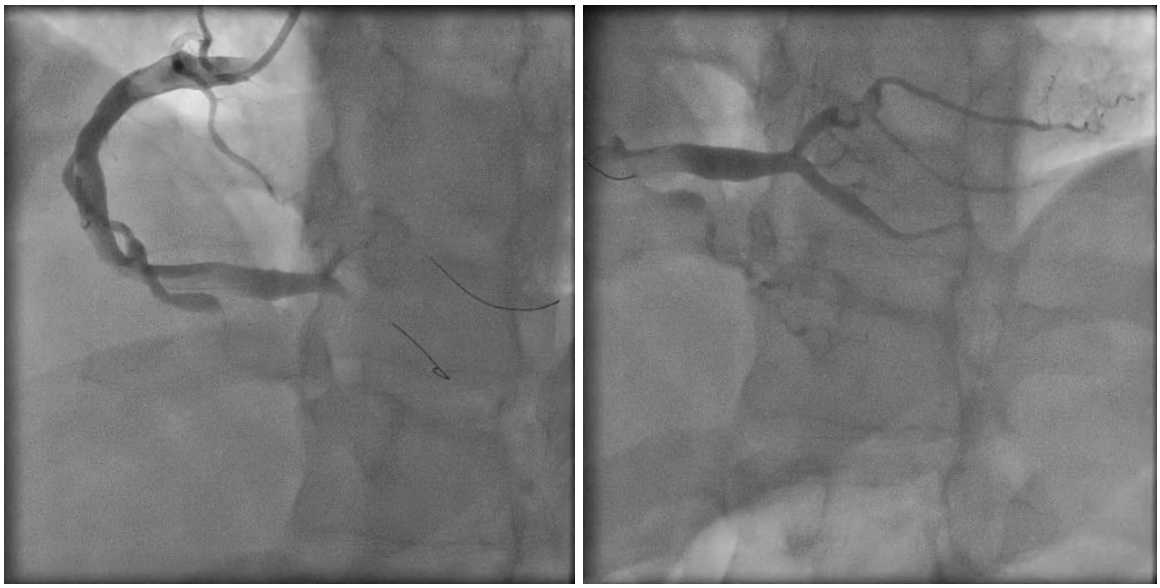

Fig. 4. Coronary angiogram: ectatic right coronary artery after thrombus aspiration showing flow in PDA and PLV branches and some residual thrombus

The incidence of CAE is higher in men (2.2%) than in women (0.5%) [3]. In the CASS registry postmortem incidence is reported 1.4%. Atherosclerosis is considered to be the main etiologic factor responsible for >50% of cases in adults [1,3]. Kawasaki disease is the most frequent etiology in children. Coronary artery ectasia is considered to be a result of excessive

expansive remodeling, which happens as a result of enzymatic degradation of the extracellular matrix and thinning of the vessel tunica media. Patients with CAE without obstructive coronary artery disease may present with positive stress tests, angina pectoris or acute coronary syndromes (ACS). Ectatic artery may be a source of thrombus formation with distal embolization, vasospasm or spontaneous artery rupture as the most catastrophic sequela. CAE is classified according to the coronary artery diameter as small (5mm), medium (5–8 mm) and giant (more than 8mm). There are significant histopathological similarities between ectasia and atherosclerosis. The vessel lumen may be narrowed, retained or dilated with advancement of atherosclerosis process. Some atherosclerotic plaques, as a consequence of a phenomenon called 'positive arterial remodeling, do not decrease luminal size, probably due to expansion of the tunica media and external elastic membrane. This finding may be the reason of ectasia or aneurysms of coronary arteries. Positive remodeling is basically a compensatory mechanism to retain luminal size during the progression of atherosclerosis. Significantly raised levels of C-reactive protein (CRP) and vascular endothelial growth factor (VEGF) were isolated in patients with CAE, which suggests extensive inflammation and neovascularization in ectatic vessel wall [5,6]. CAE is associated with angiotensin-converting enzyme DD genotype and familial hypercholesterolemia suggesting a genetic preponderance [7,8]. There is controversial association between hypertension and CAE [3]. CAE is linked to apical hypertrophic cardiomyopathy with high wall tension during ventricular systole. Right coronary artery is the most commonly involved vessel in the coronary tree (40–61%) which is followed by left anterior descending artery (15–32%) and left circumflex artery (15–23%) [1,3,4]. Solitary left main trunk ectasia is almost an exception. CAE may coexist with aneurysms of additional arterial beds, mainly abdominal aorta, even with venous varicosities. Coronary angiogram is gold standard for diagnosing coronary ectasia. Extent of CAE and corrected TIMI frame count are associated with severity of angina in patients with CAE [9]. CAE in patients younger than 50 years may be suggestive of connective tissue disorders or vasculitis. In such situations further workup to identify the cause should be considered, as was done in this case. The clinical features suggesting coronary aneurysms secondary to Kawasaki disease are the history of Kawasaki disease, matching symptoms, Asian race, proximal location of coronary artery aneurysms, giant aneurysms (more than 8 mm), young age and lack of significant coronary stenosis (>50%). Accurate diagnosis of Kawasaki disease is crucial because the treatment strategies differ from conventional atherosclerotic ectasias [10]. According to Markis classification of CAE, typeI and type II CAE have a worse prognosis than type III and IV. Mortality rate of CAE at 2 years is reported up to 15%. There is still no consensus for management of CAE amongst the experts. Risk factor modification and administration of aspirin to all CAE patients is logical due to the high coincidence with coronary artery disease and acute coronary syndrome. Statins may have an important role by inhibiting the enzyme matrix metalloproteinase [11]. Nitrates may cause steal phenomenon by dilating epicardial coronary arteries, and increase anginal symptoms [12]. Treatment of CAE with angina or myocardial ischemia includes aspirin, statin and anti-ischemic medications (calcium channel blocker, beta blocker and trimetazidine) [12]. Acute coronary syndrome (ACS) associated with CAE must be managed on an individual basis. The presence of thrombus may warrant to implement extra therapeutic decisions, like thrombolysis, heparin infusion or glycoprotein IIb/IIIa receptor inhibitors and thrombus aspiration during primary percutaneous coronary intervention (PCI) [13]. Long term anticoagulation is suggested by many authors however no randomized trial demonstrated its benefit. Percutaneous or surgical revascularization may be an option in patients with coexisting obstructive lesions and significant ischemia despite medical therapy [14]. Coronary intervention of stenosis adjacent to ectasia is in itself challenging with regards to optimal stent sizing, misplacement of stent, early stent thrombosis and restenosis. Additional care should be taken during stenting because adequate stent expansion and wall apposition is needed [15]. Covered stents offer superior angiographic results compared to the bare metal stents, but long term advantage has not been proven. The use of large sized peripheral stents in ectatic coronary arteries is also an option. In patients with evidence of enlargement of saccular aneurysms with high risk of rupture, surgical resection may be considered.

4. CONCLUSION

CAE is mostly a form of atherosclerosis seen in 0.3 4.9% of diagnostic coronary angiography procedures. An excessive expansive remodeling

with enzymatic degradation of the extracellular matrix and thinning of the vessel media as a result of chronic inflammation is considered to be a major pathophysiologic process. Clinical importance inclines on its association with acute coronary syndrome as was the case in our patient. Management options include risk factor modifications for coronary artery disease, anti-ischemic therapy, antithrombotic management and percutaneous or surgical revascularization.

CONSENT

Patient himself gave the written informed consent for case report.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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