

A Case Report of Successful Fibrinolysis in a Multiple Coronary Ectasia ST-segment Elevation Myocardial Infarction Validated by Coronary Angiography

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Authors' contributions

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

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Case Report

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ABSTRACT

Coronary artery ectasia (CAE) often presents in the form of an acute coronary syndrome (ACS) caused by slow blood flow leading to thrombus formation in ectatic coronary arteries. The usual approach is thrombectomy with intracoronary thrombolysis but often does not guarantee immediate blood flow. A 45-year-old male presented with anginal chest pain and was diagnosed with ST-segment elevation myocardial infarction (STEMI), managed immediately with tissue plasminogen activator (t-PA) followed later with coronary angiography revealing diffused Multiple coronary ectasia MCEA with no identified culprit lesions afterward. The patient was followed up periodically, with favorable outcomes on daily aspirin, direct oral anticoagulant (DOAC), and guideline-directed medical therapy (GDMT) for coronary artery disease (CAD) and reduced ejection fraction heart failure (HF-rEF). This case management strategy was dictated by available resources at the time of

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presentation; however, it signifies the importance and favorable outcome of thrombolysis in CEA/STEMI patients. Future large-scale studies are required toward defining the duration as well as the choice of long-term anticoagulation.

Keywords: *Coronary artery ectasia; ST-segment elevation myocardial infarction; Fibrinolysis; Thrombolysis.*

1. INTRODUCTION

Coronary artery ectasia (CAE) is a rare form of aneurysmal coronary artery heart disease, encountered while performing coronary angiography. It is described as segmental dilation with a diameter 1.5 times that of a normal adjacent coronary artery and differentiated from a coronary aneurysm by the involvement of more than 50% of the length of the vessel [1,2]. Furthermore, atherosclerosis is attributed as a major cause of CAE [3]. Acute coronary syndrome (ACS) can occur from altered blood flow occluding the ectatic segment with thrombus or by distal embolization [4]. CAE and cardiovascular disease related complication, occurs in only 0.3%-4.9% of the patients [5]. Regarding etiology, rheumatologic disorders, systemic inflammatory diseases, congenital and iatrogenic origin have all been implicated, while atherosclerosis is reported as accounting for more than 50% of cases [6]. We hereby report a case of acute anteroinferior ST-segment elevation myocardial infarction (STEMI) treated with systemic tissue-plasminogen activator (t-PA) infusion, followed by percutaneous coronary angiography revealing patent multi coronary artery ectasia (MCAE).

2. CASE REPORT

A 45-year-old male with tobacco use, not known to have any chronic medical illness, presented to

the emergency department complaining of retrosternal compressing chest pain for 4 hours, associated with sweating, after having a quarrel with his family members. He was hemodynamically stable with unremarkable physical examination. His presenting ECG showed ST-elevation myocardial Infarction (STEMI). Presenting ECG (Figure 1A) showing ST-segment elevation in the anterolateral and inferior leads. The patient was thrombolysed with a tissue plasminogen activator (t-PA) as the Primary PCI facility was not accessible at the time of presentation. Post t-PA ECG showed accelerated Idioventricular rhythm at 100 bp (Figure 2A), and (Figure 2B) ECG showing Anterior/inferior Q waves with partial reduction in st segment elevation.

His Echocardiogram revealed normal-sized left ventricle with an ejection fraction of 25-30%, severely hypokinetic LAD territory. Later, the patient was transferred to another hospital for Coronary Angiography for possible further intervention. His coronary angiogram exhibited an ectatic left main diffusely ectatic atherosclerotic type III Left anterior descending (LAD) artery without significant lesion (Figure 3A), a dominant ectatic atherosclerotic left circumflex artery without significant lesion (Figure 3B), and a non-dominant right coronary artery free of ectasia (Figure 3C). The patient was discharged on Aspirin, Apixaban, high-dose statin, and guideline-directed anti-failure therapy.

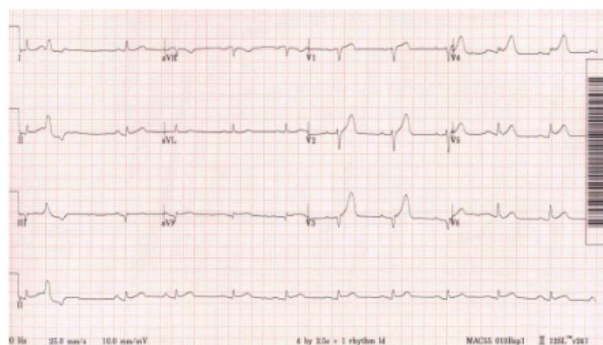


Figure 1A: ECG showing ST-segment elevation in the anterolateral and inferior leads.

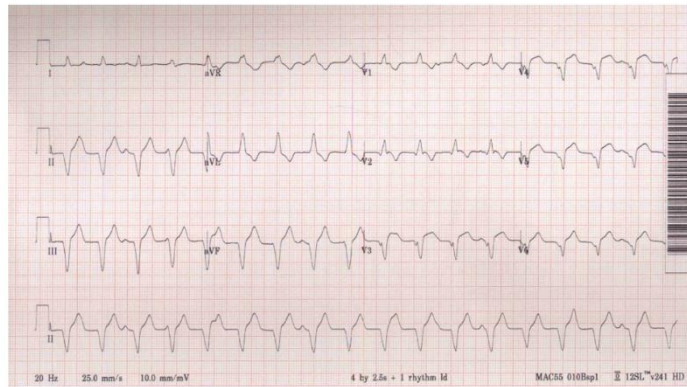


Figure 2A: Post t-PA ECG; showing accelerated Idioventricular rhythm at 100 bpm.

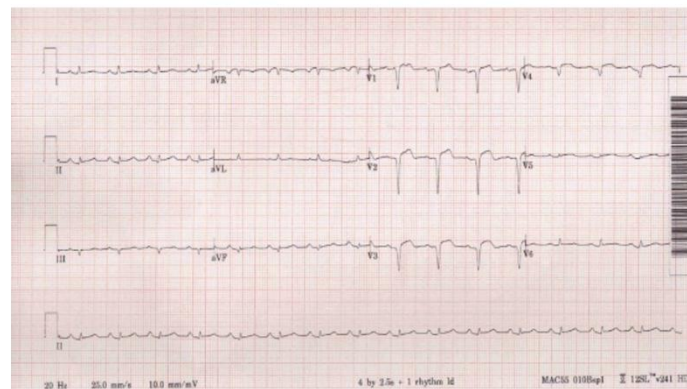


Figure 2B: ECG showing Anterior/inferior Q-waves with partial reduction in ST segment elevation.

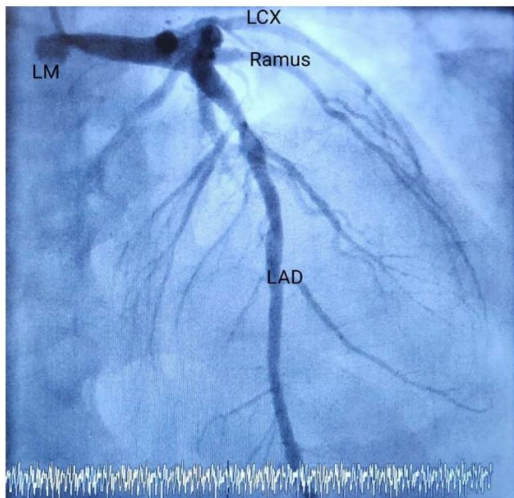


Figure 3A: RAO cranial view of ectatic LM and LAD showing diffusely atherosclerotic type III anterior descending artery without significant lesion.

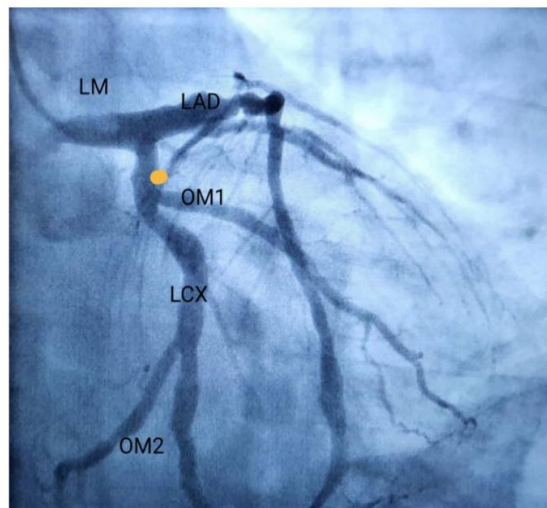


Figure 3B: RAO caudal view for LCX, a dominant ectatic atherosclerotic left circumflex artery without lesion.

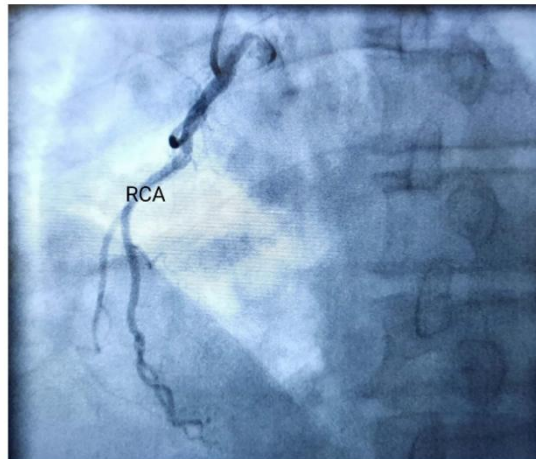


Figure 3C: RAO for RCA showing non-dominant RCA free of disease.

3. DISCUSSION

CEA reports are expanding with increased Coronary angiography utilization for ACS management, it is attributed to atherosclerosis in 50% of cases, whereas 20-30% of cases are congenital in origin, and only 10-20% of CAE cases are associated with various other disorders; inflammatory, or connective tissue diseases [2]. The proximal and middle segments of the RCA are the most common sites for CAE, followed by the proximal left anterior descending and left circumflex arteries [7].

CEA is classified into Type I- diffuse ectasia in 2 or 3 vessels; Type II-diffuse ectasia in 1 vessel and local ectasia in the other; Type III- diffuse ectasia in 1 vessel; and Type IV- local ectasia in 1, 2, or 3 vessels. Accordingly, a patient with diffuse dilatation of multiple vessels would be classified as Type I, with an incidence of 5.8% of all CAE patients [8]. A retrospective study conducted on the Saudi population by Almansori et al, showed CEA prevalence was higher and more diffuse reaching 6%, compared to the previously reported 1.2-4.9 % [1,2] A total sample of 1115 patients underwent coronary angiograms, 6% (total 67 patients) had CEA, and 43% of the sample had a severe form of ectasia (types I and II) 21% out of the 43% were type 1-similar to our case [9]. They attributed the increased prevalence in the Saudi population to increased atherosclerotic coronary artery disease, and/ or the possibility of increased genetic susceptibility to the development of coronary artery ectasia.

The reported risk factors of CAE are male gender, smoking, and hyperlipidemia, while diabetes mellitus and age are inversely associated risk factors. Plaque rupture is the most known cause of ACS; nevertheless, it can also be caused by CAE through sluggish or turbulent blood flow in the ectatic vessel leading to thrombus formation and distal embolization [2,4,10]. CAE is caused by the initial endothelial damage that activates a series of inflammatory mediators that cause degeneration of the medial layer of the vessel. These structural alterations, together with the action of nitric oxide and other vasodilators, cause dilatation of the coronary artery with expansive remodeling [3].

Management strategies are currently based on personal experience, single-case reports, and small-series reports [4]. Facilitated PCI with thrombolysis is not recommended for ST-segment elevation MI, and primary PCI is superior to in-hospital initiated fibrinolysis; however, thrombolysis, regimens of intravenous thrombolytic agents were found to be as efficacious as intra-coronary delivery [11].

Gunduz et al, reported two MI cases with diffuse MCAE who were treated with t-PA infusion [3]. However, the final TIMI grade flow was not assessed in those patients. In contrast, our patient presented with ST-segment elevation MI (STEMI), treated with t-PA infusion within 90 minutes of presentation, and normal coronary flow was validated less than 6 hours later with coronary angiography. Should our patient have gone for primary PCI, a large thrombus burden

might have been an obstacle and PCI might have been deferred.

According to Mauro et al., intense antithrombotic treatment, including glycoprotein IIb/IIIa inhibitors and PCI deferral, was safe and effective for acute coronary syndrome patients who had a large intracoronary thrombus [12]. Oral anticoagulation therapy has been suggested to prevent thrombus formation in patients with CAE; however, no current data or evidence-based guidelines support this [10]. Aspirin was suggested in all patients because of the coexistence of CAE with ACS in the great majority of patients, even in patients with isolated coronary ectasia [13].

The role of combined antiplatelet therapy, with the addition of adenosine diphosphate inhibitors, has not yet been evaluated in prospective randomized studies; however, it has been shown that plasma levels of P-selectin, beta thrombomodulin, and platelet factor 4 are elevated in isolated CAE patients when compared with control participants who have angiographically normal coronary arteries, suggesting an increased platelet activation [13]. Based on the significant flow disturbances within the ectatic segments, and published data on warfarin, we postulate chronic anticoagulation with direct oral anticoagulants (DOACs) similar to warfarin is indicated [14].

4. CONCLUSION

Multivessel diffused coronary artery aneurysms is a rare clinical entity, and one of the less frequent causes of myocardial infarction. Management of which requires diligent medical and interventional therapy. Our case represents a favorable response to fibrinolytic therapy - alone- evident by a reperfusion idioventricular rhythm and patent coronaries. Therefore, we conclude fibrinolysis alone is effective in restoring normal coronary flow in CEA myocardial infarction and pre-define CEA.

CONSENT

Written informed consent was obtained from the patient for publication.

ETHICAL APPROVAL

The study was approved by King Abdullah International Medical Research Center (KAIMRC) (NRA22A/012/05).

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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