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THROMBOEMBOLIC NSTEMI FROM A 9.4 × 7.3 MM MID-LEFT CIRCUMFLEX ARTERY ANEURYSM WITH A ZERO CALCIUM SCORE AND NON-OBSTRUCTIVE CORONARY ARTERIES

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ABSTRACT

Introduction: Coronary artery aneurysms (CAAs) are uncommon and can cause acute coronary syndromes due to thromboembolism despite the absence of obstructive coronary disease.

Case description: We report the case of a 51-year-old patient with hypertension and hypercholesterolemia with the chief complaint of chest pain and elevated troponin, which is typical of non-ST-segment elevation myocardial infarction (NSTEMI). Coronary angiography showed non-obstructive coronary arteries and a calcium score of 0 with 9.4 x 7.3 mm middle-left circumflex artery aneurysm with thrombosis. Thrombus aspiration was effectively done without stenting. The patient was treated using dual antiplatelet therapy and statin therapy, and clinical improvement was achieved. The case in point demonstrates an infrequent pathophysiology of NSTEMI due to thromboembolism in a tiny aneurysm without severe atherosclerosis. Multimodality imaging was essential for diagnosis. Management was conservative. Transthoracic echocardiography revealed mild left ventricular hypertrophy with inferior hyperkinesia along with preserved ejection fraction. The patient was hemodynamically stable throughout hospitalization and was discharged on optimal medical therapy having no recurrent symptoms.

Conclusion: NSTEMI may be caused by small CAAs through thromboembolism even though the coronary artery calcium score is zero. Personalized care (thrombus aspiration and optimal medical therapy) can yield excellent outcomes.

KEYWORDS

Coronary artery aneurysm, NSTEMI, thromboembolism, zero calcium score, non-obstructive coronary artery disease

LEARNING POINTS

- A non-ST-segment elevation myocardial infarction can occur in the absence of obstructive coronary disease because of thromboembolism from a small coronary aneurysm, even with a coronary artery calcium score of zero.
- Multimodality imaging is crucial for precise diagnosis along with management of coronary artery aneurysms.
- Conservative management with thrombus aspiration along with medical therapy may be effective in selected patients.

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INTRODUCTION

Coronary artery aneurysm (CAA), which is defined as localized dilatation of a coronary artery that is more than 1.5× the surrounding vessel diameter, is rare, with a reported prevalence of 0.3-5% in angiographic studies[1]. Even though CAAs are often incidental findings they can cause complications such as thrombosis and distal embolization.

In adults, CAAs are usually linked to atherosclerosis, and other causes include inflammatory diseases, congenital defects, and iatrogenic factors[2]. CAAs cause acute coronary syndrome (ACS) that are uncommon and generally associated with the formation of thrombus as opposed to the rupture of the plaque.

CASE DESCRIPTION

The patient was a 51-year-old Emirati male patient diagnosed with hypertension and hypercholesterolemia who presented with acute chest pain. He had a history of inferior ST-segment elevation myocardial infarction, with no diabetes, smoking or family history of premature coronary disease.

At admission, he was hemodynamically stable. In addition, ischemic symptoms and the absence of ST elevation on the electrocardiogram (ECG), the troponin level was high and therefore a diagnosis of NSTEMI was made. Dilated inferior Q waves without fresh ischemic alterations were observed on ECG *(Fig. 1)*.

The laboratory results showed high total cholesterol (6.8 mmol/l) and low-density lipoprotein cholesterol (2.9 mmol/l), normal haemoglobin A1C and C-reactive protein *(Table 1)*.

Transthoracic echocardiography revealed mild left ventricular hypertrophy, inferior hypokinesia and maintained ejection fraction (50-55%).

Coronary angiography showed that the coronary arteries were non-obstructed and right dominance. Computed tomography coronary angiography (CTCA) was performed and showed a coronary artery calcium score of zero, a 9.4 × 7.3 mm aneurysm in the mid left circumflex artery and a thrombus *(Fig. 2)*.

Thrombus aspiration was successfully done without placing stents because there was no major stenosis. The patient

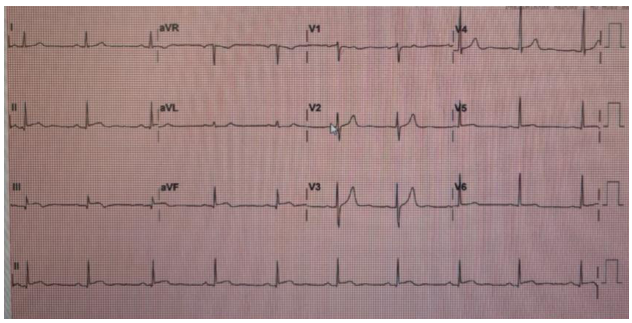


Figure 1. Admission 12-lead electrocardiogram showcasing normal sinus rhythm with old inferior Q waves and no acute ischemic changes.

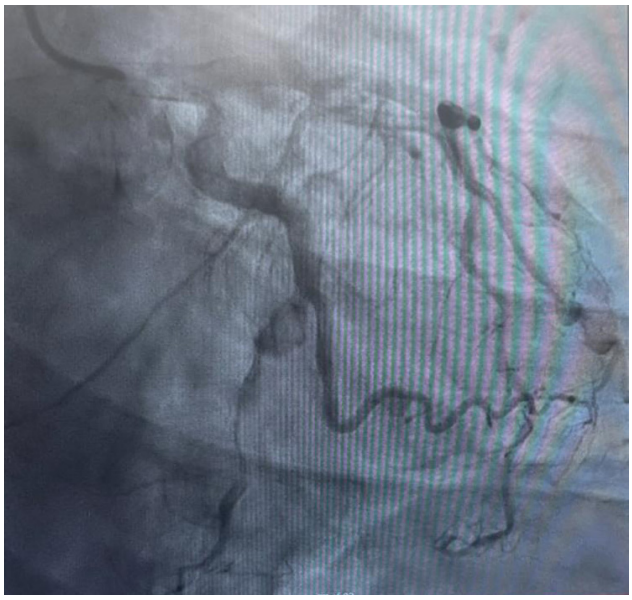


Figure 2. Coronary angiography (left anterior oblique caudal view) showing a small mid-left circumflex artery aneurysm (9.4 × 7.3 mm) with surrounding thrombus (arrow) in a patient with non-obstructive coronary arteries along with a calcium score of zero.

Parameter	Value	Reference range	Interpretation
High sensitivity Troponin (ng/l)	Elevated (peak not specified)	<14 (99th percentile)	Increased
HbA1c (%)	5.6	<5.7	Normal
C-reactive protein (mg/l)	0.8	<3	Normal
Total cholesterol (mmol/l)	6.8	<5.2	Increased
LDL-cholesterol (mmol/l)	2.9	<2.6	Increased
HDL-cholesterol (mmol/l)	1	>1 (men)	Normal
Triglycerides (mmol/l)	1.68	<1.7	Normal

Table 1. Laboratory investigations performed on admission (November 6-7, 2025). No serial measurements of troponin or lipids were reported in this admission.
Abbreviations: HbA1c, haemoglobin A1c; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

received dual antiplatelet therapy (aspirin and ticagrelor), high-intensity statin therapy and antihypertensive medications. At discharge, on day 5 he had no symptoms in stable condition.

DISCUSSION

This case indicates that thromboembolism caused by a small CAA is a rare cause of NSTEMI even in the absence of obstructive coronary artery disease.

Though CAAs are rare, they may cause thrombosis and embolization[3]. NSTEMI in a patient with a zero coronary artery calcium score and no obstruction of the coronary arteries is uncommon and indicates a non-atherosclerotic process[4].

The size of the aneurysm (9.4 mm) was in the small-to-moderate size range and yet represented a considerable thromboembolic risk. Smaller aneurysms are less well defined than giant aneurysms but may still lead to acute events unlike giant aneurysms which are usually associated with Kawasaki disease.

The imaging of multimodality was essential in this case. The coronary angiography made it possible to detect and treat thrombus, whereas CTCA made it possible to measure the aneurysm correctly and guarantee the lack of calcification[5,6].

Management strategies for CAAs are not standardized because of a scarcity of evidence. CAAs can be treated by medical therapy, percutaneous intervention, or surgery depending on aneurysm size, signs, and complications[5,6]. Conservative management in this instance, which included thrombus aspiration and dual antiplatelet therapy, was effective, and prevented the use of stents which were not necessary[7,8].

The case demonstrates that mechanisms other than plaque rupture can cause ACS. Further, it is important to take into account CAAs with atypical presentations.

There is an absence of obstructive coronary disease along with a zero coronary artery calcium score of zero. Hence, this case shows the significance of considering alternative mechanisms of myocardial infarction beyond conventional atherosclerotic plaque rupture[9].

CAA must be included in the differential diagnosis of ACS specifically when imaging and clinical findings are discordant. Moreover, tailored treatment strategies based on clinical along with anatomical factors are critical to optimize outcomes and avoid unnecessary invasive interventions[10].

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