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Left Main Coronary Aneurysm: A Rare but a Serious Cause of Acute Myocardial Infarction

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Abstract Left main coronary artery (LMCA) aneurysms are rare with incidence of 1 in 1000 cases among patients undergoing coronary angiography. Coronary artery aneurysm is defined as coronary arterial segment dilation > 1.5 fold greater than normal adjacent coronary segments. Atherosclerosis is the primary cause of coronary aneurysms but also has been reported in other conditions like arterial vasculitis (e.g., Kawasaki disease, Takayasu arteritis), or infection (e.g., syphilis, mycosis). LMCA aneurysms are especially associated with serious complications including thrombosis, distal embolization, spontaneous rupture or dissection or even sudden cardiac death. The optimal treatment for LMCA aneurysm is still controversial and the available results have been based mainly on case reports and not on controlled trials. The lack of consensus about managing LMCA is not the only challenge here. The lack of individualized treatment or precision medicine in this area makes managing these cases at bedside even more challenging.

***Keywords**: left main coronary artery, aneurysm, myocardial infraction*

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1. Introduction

Coronary artery aneurysms are uncommon with an approximate incidence of 5.3%. [1,2,3,4] Left main coronary artery (LMCA) aneurysms are especially rare; accounting for only 1 in 1000 cases among patients undergoing coronary angiography. [5] It remains very controversial whether surgical repair for LMCA aneurysm is necessary or if conservative medical treatment is equally efficient. Here we describe a 50-year old female patient presenting with acute myocardial infarction whose her angiogram revealed a large fusiform aneurysm of the LMCA. She was treated conservatively with aspirin, statins and coumadin and had uneventful course over 9 months of follow up.

2. Case Presentation

A 50-year-old female, with medical history notable for anxiety depressive disorder, presented with acute retrosternal chest pain that started 8 hours prior to admission to hospital. The pain started at rest and was radiated to throat and both shoulders. She denied any history of dyspnea, orthopnea, tobacco use, fever, sweating or vomiting. After the administration of sublingual nitroglycerin, her chest pain was substantially reduced. The physical exam was unremarkable. Her ECG revealed ST-segment depression and T wave inversion in leads I and aVL and T wave inversion in V1 and V2 (Figure 1). Initial cardiac troponin I level was 2.5 ng/ml and reached 38 ng/ml during her hospitalization so she underwent a subsequent cardiac angiogram that showed a 20 x 15 mm fusiform aneurysm of the distal portion of the LMCA extending into both proximal left anterior descending coronary artery (LAD) and left circumflex artery (LCX) (Figure 2). Echocardiogram showed an ejection fraction of 55% with mildly hypokinetic inferior segment of the left ventricle. CT coronary angiogram showed Distal left main aneurysm containing mural thrombus measures 1.5 cm extending into the proximal LAD and 1.2 cm aneurysm in the proximal circumflex without mural thrombus (Figure 3). CTA of head, neck, chest and abdomen showed normal intracranial arteries, thoracic and abdominal aorta and normal renal and iliac arteries. She refused to undergo surgical repair of the coronary artery aneurysms so she was managed conservatively and discharged home on aspirin, statins and Coumadin. The patient remained asymptomatic at 3, 6 and 9 months follow up visits with no reported cardiovascular events.

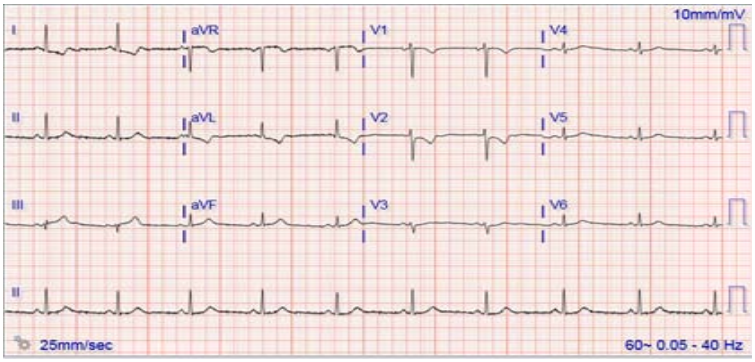

Figure 1. ECG revealed ST-segment depression and T wave inversion in leads (I and aVL) and T wave inversion in V1 and V2

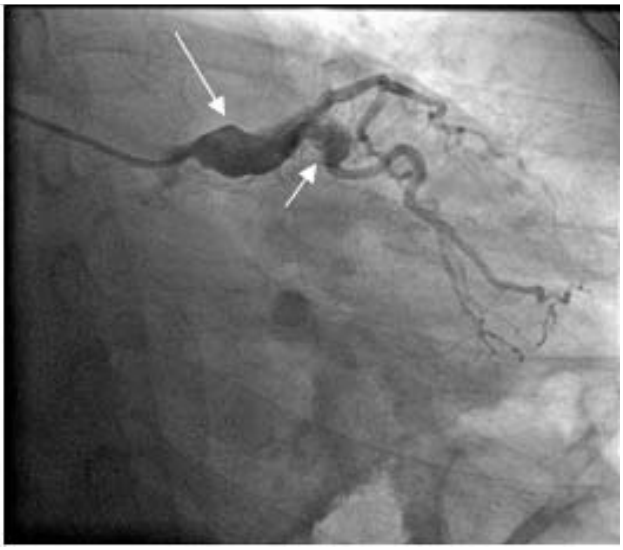

Figure 2. Coronary angiogram revealed an aneurysmal dilatation of distal left main coronary artery (LMCA) extended into left anterior descending artery (LAD) (long arrow) and aneurysmal dilatation left circumflex artery (LCA) (short arrow).

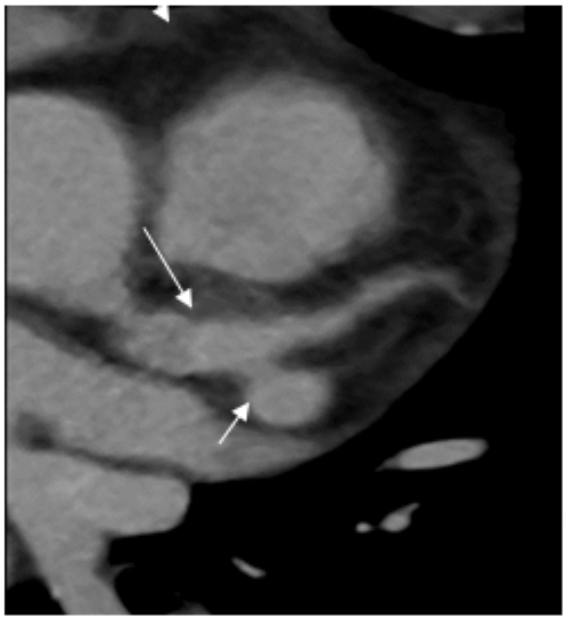

Figure 3. CT coronary angiogram showed Distal left main aneurysm containing mural thrombus measures 1.5 cm extending into the proximal LAD. This aneurysm is peripherally calcified. There is no luminal narrowing (long arrow). 1.2 cm aneurysm in the proximal circumflex without mural thrombus (Short arrow)

3. Background

Coronary artery aneurysm is defined as coronary arterial segment dilation > 1.5 fold greater than normal adjacent coronary segments or largest coronary artery or three times the diameter of coronary artery catheter. The patient might present with single or multiple aneurysms affecting the same or multiple coronary arteries. Morphologically, these aneurysms may be saccular or fusiform. Saccular aneurysms have a transverse diameter that is greater than the longitudinal diameter, whereas fusiform aneurysm have a dilatation along the long axis of vessel at least twice the diameter of the transverse dimension. [6] The majority of coronary aneurysms are seen in the right coronary artery (40-70%), followed by LAD (32.3%), LCX (23.4%), and LMCA (3.5%). [9,10,11]

Atherosclerosis is the primary cause of coronary aneurysms in adults especially among male patients with significant coronary artery disease like who have suffered a myocardial infraction in the past or have three-vessel atherosclerotic disease. [8] Other causes of coronary aneurysms include arterial vasculitis (e.g., Kawasaki disease, Takayasu arteritis), or infection (e.g., syphilis, mycosis). There are few cases reports on the development of coronary aneurysms after the placement of drug-eluting coronary stents and percutaneous coronary angioplasty. [7]

LMCA aneurysms are especially associated with serious complications. The sluggish blood flow in the aneurysm itself leads to higher tendencies for thrombus formation with subsequent embolization to distal coronary arteries. In addition, the weakened arterial wall places the patient at very high risk of vessel dissection and rupture, leading to angina pectoris, myocardial ischemia and/or infarction and sudden cardiac death. [12,13]

The optimal treatment for LMCA aneurysm is still controversial and the available results have been based mainly on case reports and not on controlled trials. The patients with evidence of atherosclerosis should receive guideline-directed medical therapy to modify their coronary artery disease risk factors and to decrease risk of thromboembolic complications through long term administration of antiplatelet, statin and anticoagulants with close follow up every 3 months. [14] However, there are no controlled studies showed; for how long we need to keep on dual antiplatelet and anticoagulant treatment, who at higher risk for thromboembolic complications or can we generalize this approach among patients without evidence of coronary atherosclerosis like in Kawasaki disease and

what is the best way to follow up these patients; clinically versus angiographically. In addition, these patients theoretically at higher risk of bleeding from dual antiplatelet and anticoagulant treatment. In other side, the surgical intervention, (including surgical removal of aneurysm and CABG, isolated CABG, stent graft or coil) is appropriate in symptomatic patients who have obstructive coronary artery or evidence of embolization leading to myocardial infraction and in patients with LMCAs with high risk of rupture. [15,16] Also here, there are no studies showed; survival benefits of this approach in comparison with who were treated medically, is it applicable in patients with complex aneurysm involving more than two coronaries like in our case, does it apply to patients without atherosclerosis, and what is the role of presence of aneurysmal mural thrombus with and without coronary luminal narrowing in deciding to treat medically versus surgical approach?

4. Conclusion

The lack of consensus about managing LMCA is not the only challenge here. The lack of individualized treatment or precision medicine in this area makes managing these cases at bedside even more challenging. The current approach used to make decisions about the management of LMCA does not take into account individual variability such as complexity of aneurysm, presence of multiple coronary aneurysms, atherosclerosis-related or not, and presence of mural thrombus with and without coronary luminal narrowing, feasibility and applicability of surgery and risk of bleeding. We believe that the variables should be used for risk stratification and subsequent decision making for management rather than using “one size fits all” approach.

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Conflict of Interest Statement

No conflict of interest in preparing this article.

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