Left main coronary artery aneurysm occlusion presenting as myocardial infarction with cardiogenic shock: Patient saved by securing left anterior descending artery at cost the of other vessels

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#### Abstract

Coronary artery aneurysms and ST-segment elevation myocardial infarction (STEMI) are seldom encountered in clinical practice and pose a management challenge. We describe the case of a patient diagnosed with anterior wall myocardial infarction with a complete occluded left main coronary artery (LMCA). After the establishment of partial blood flow within the

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## CONFLICT OF INTEREST

The authors declare no conflict of interest.

## CONSENT STATEMENT

Consent was obtained from the patient's wife as he underwent urgent PCI.

#### ABSTRACT

Coronary artery aneurysms and ST-segment elevation myocardial infarction (STEMI) are seldom encountered in clinical practice and pose a management challenge. We describe the case of a patient diagnosed with anterior wall myocardial infarction with a complete occluded left main coronary artery (LMCA). After the establishment of partial blood flow within the LMCA was achieved, a 6 x10 mm aneurysm was discovered in the LMCA. To the best of our knowledge, this case appears to be the first successful percutaneous treatment of a completely obstructed aneurysmal LMCA.

**Keywords:** coronary artery aneurysm; left main coronary artery, ST-segment elevation myocardial infarction, stent

#### INTRODUCTION

It is unusual for a ST-elevation myocardial infarction (STEMI) to be caused by a left main coronary artery (LMCA) aneurysm [1]. The pathogenesis of STEMI in aneurysms is perplexing, and its therapy is still debatable. Case studies have been used primarily to describe pharmacological therapy, percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), and hybrid approaches depending on the severity of coexisting coronary artery stenosis.

### CASE REPORT

A 40-year-old male smoker and to bacco chewer presented to the emergency department eight hours after onset of resting angina. The patient's history was unremarkable without any record of similar symptoms experienced earlier. He also did not have a family history of ischemic heart disease. His physical examination revealed a heart rate of 116 beats/min, blood pressure of 90/66 mmHg, respiratory rate of 20 breaths/min, and oxygen saturation (SPO<sub>2</sub>) of 88%. Electrocardiography showed ST elevation in V2-V6, I, and aVL, suggestive of anterior wall myocardial infarction (AWMI). During hospitalization, the patient developed hemodynamically unstable ventricular tachycardia (VT) which was treated with DC cardioversion. The patient was immediately rushed to the cath-lab for primary PCI.

After engaging the LMCA with a 6F EBU 3.5 Launcher guiding catheter (Medtronic Inc, Minneapolis, USA) an occlusion within the LMCA was encountered. Initial considerations included the possibility of a dissection induced by the guide catheter or a complete thrombotic occlusion of the LMCA, given the absence of observable branches or distal vasculature beyond this occlusion point (Fig 1. & Video 1). Due to complaints of persistent chest pain by the patient, cautious advancement of the workhorse guide wire was

performed, leading to an extension of staining. Finally, the lesion was crossed using a Fielder FC guidewire (polymer jacket and hydrophilic wire, Asahi Intecc, Aichi, Japan) originally intended for wiring the left anterior descending artery (LAD) and achieving some flow (**Fig. 2**), but ultimately went into a diagonal branch (**Video 2**). As the thrombosuction catheter was unable to cross, the LMCA lesion was pre-dilated with a  $2 \times 10 \text{ mm}$  semi-compliant balloon.

After establishment of partial blood flow within the LMCA (Fig. 2), a 6 x10 mm aneurysm was discovered in the LMCA (Fig. 3 & Video 3). The case was discussed with among the cardiothoracic surgical team. The decision was made to pursue revascularization using PCI. Two other Fielder FC wires were introduced into both the LAD and the ramus intermedius (RI). Dilation of the RI was also performed with a 2 x 10 mm semi-compliant balloon (Fig. 4 & Video 4) While both LAD and RI achieved thrombolysis in myocardial infarction (TIMI) grade I flow, efforts to wire the left circumflex artery (LCx) were unsuccessful (Video 5). Consequently, a decision was made to employ a 5F TIG diagnostic catheter (Terumo Corporation, Shibuya-Ku, Tokyo) to perform an angiogram of the right coronary artery (RCA)s via the radial route. This imaging revealed the RCA was normal, however, a grade 3 retrograde collateral circulation was evident from the RCA to the LCx, suggestive of LCx chronic total occlusion (CTO) (Fig. 5 & Video 6).

Our intended approach involved the placement of a covered stent in the LM-LAD. However, due to the delayed availability of the designated stent and the patient's compromised hemodynamic state, a 3.5 x 20 mm Promus Elite stent (Everolimus Eluting Platinum Chromium Coronary Stent System, Boston Scientific Corporation, Marlborough, USA) was deployed from LMCA to LAD (Fig. 6 & Video 7). Subsequent to stent implantation a 4 x 10 non-compliant balloon was employed for post-dilatation. This yielded improved blood flow in the LAD and diagonal. Notably, on intravascular ultrasound (IVUS) imaging, the LM aneurysm was visualized as a radiolucent shadow with layers and scintillating blood shadow surrounding the stent struts and the stent appeared to dangle, creating a potential nidus for thrombus formation (Fig. 7 & Video 8). In an endeavor to exclude the aneurysm, a 3.8 x 26 mm Graftmaster covered stent (Coronary Stent Graft System, Abbott Vascular, Santaclara, USA) was subsequently deployed from ostial LMCA, encompassing the LCx and small sized RI extending up to the LAD/diagonal bifurcation, (Video 9) and post-dilated with a 5 x 8 mm non-compliant balloon.

Finally, TIMI II flow in both LAD and diagonal was achieved (Fig. 8 & Video 10). Post procedure IVUS revealed bright echo reflection due to the covered stent and echo drop out was observed behind the multiple layers of stents (Fig. 9 & Video 11). The patient was discharged on aspirin, clopidrogrel, rivoraxaban and high dose statin. At 3-month follow-up, angiography revealed TIMI III flow into the LAD and its branches (Fig. 10 & Video 12) and complete obliteration of the aneurysm. The patient's symptoms improved, and he was symptomatically better.

### DISCUSSION

The term "coronary artery aneurysms" (CAAs) refers to localized irreversible dilatation of a coronary artery to a diameter more than 1.5 times that of the adjacent normal coronary segments. In contrast, coronary artery ectasias (CAE) occur as widespread arterial dilations wherein the length of the dilated segment is more than 50% of the diameter [2, 3]. Morgagni published the first pathological description of a coronary artery aneurysm [2]. With the advanced application of coronary tomography (CT) and invasive coronary angiography, an anticipated increase in detection rate of CAAs exists.

Although the exact prevalence of CAAs is difficult to determine due to their asymptomatic nature, it is likely underestimated – reported prevalence ranges from 0.3–5%. RCA is most affected (40–70%), followed by the LAD (32.3%) and LCx (23.4%) [3, 4,]. An aneurysm in the left main coronary artery aneurysm (LMCAA) is a very rare finding occurring in 0.1% cases as reported by Topaz et al. [5]. A review recently published identifies atherosclerosis (40%) as the most common cause of LMCAAs, followed by inflammatory (12%) and congenital (9%) factors. Compared to stable angina (43%) and acute coronary syndromes (ACS) (32%), which were more often the first clinical signs, 14% of LMCAA were incidental observations. 18.6% of the patients were presented as STEMI [6].

Atherosclerosis is the leading cause of CAAs, accounting for approximately half of the cases. Other potential causes include vasculitis (Kawasaki disease and Takayasu arteritis), infections (bacterial, fungal, mycobacterial, Lyme, syphilitic, septic emboli, mycotic aneurysm, and HIV), connective tissue disorders (Marfan syndrome and Ehlers-Danlos syndrome), congenital conditions, fibromuscular dysplasia, and traumatic (post PCI).

While most patients are asymptomatic and are diagnosed incidentally, symptomatic patients often present with either effort angina or ACS owing to concurrent obstructive atherosclerotic disease or local thrombosis and embolisation [7, 8]. Rarely, it can present as cardiac tamponade due to CAA rupture or symptoms due to compression of adjacent structures. LMCAAs might even manifest as an incidental anterior mediastinal mass and syncope [9, 10]. Occasionally, a systolic murmur can be detected over the precordial region [3].

The most effective diagnostic approach for CAAs likely involves coronary angiography coupled with IVUS. IVUS can help address drawbacks such as underestimation of CAA size due to intraluminal thrombi and differentiation between true and false aneurysms. Transthoracic echocardiography (TTE) is particularly helpful in the detection of abnormalities in the proximal LMCA and RCA, especially in children with Kawasaki disease. Computed tomography (CT) angiography is valuable for assessing larger CAAs and those with saphenous vein graft aneurysm. Optical coherence tomography (OCT) has limitations due to its narrow scanning diameter. OCT capabilities are constrained by the infrared scan's narrow diameter [7, 8].

Given the rarity of this cardiac entity and the lack of standard treatment guidelines, management approaches typically involve anticoagulation-based medical therapy, stenting using covered stents, or surgical intervention. Treatment decisions should be tailored to patient characteristics, clinical presentation, and the location and morphology of the aneurysms. According to a recent review, 53% of LMCAA patients received surgical treatment, while only 7% received percutaneous intervention. CAAs, especially LMCAAs, are associated with poor prognosis, with a reported 15% mortality rate after a median 8-month follow-up, when longitudinal data (n=81) were reported for LMCAA [6]

In the present case, as the patient suffered an acute AWMI with a completely occluded LMCA, it was decided to assess the anatomy of the LAD and LCx and then pursue revascularization. A planned approach to cross the lesion and wire the LAD resulted in inadvertent wiring of the diagonal artery. After discovering the LMCA aneurysm after pre-dilation, another wire was placed in the LAD and RI, however wiring and visualization of LCx proved unsuccessful. Consequent RCA angiography revealed complete LCx occlusion, filling retrogradely through the RCA. It was decided to stent LM-LAD up to LAD and the Diagonal bifurcation with a covered stent. Drug-eluting stents in such a LMCA aneurysm have a high chance of stent thrombosis. At the three-month follow-up, the patient's symptoms improved, and angiography revealed TIMI III flow and aneurysm completely obliterated. To the best of our knowledge, this case appears to be the first successful percutaneous treatment of a completely obstructed aneurysmal LMCA.

### CONCLUSION

LMCAAs can arise due to a variety of factors, the specific underlying cause in the present patient is most likely atherosclerosis. Treatment options typically encompass anticoagulation to mitigate the risks associated with thrombus formation, and surgical intervention is often the preferred approach. However, in the case of ACS, PCI becomes a viable choice if emergency bypass grafting is not feasible. Nevertheless, if the LM is implicated, a hybrid approach may be considered, especially if all vessels are unable to receive a bypass graft.

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#### FIGURE LEGENDS

- Fig 1: Complete thrombotic occlusion of the left main coronary artery
- Fig 2: Some flow restoration after diagonal wiring
- Fig 3: Post pre-dilation and wiring of the diagonal and left anterior descending artery
- Fig 4: Ramus intermedius wired with Fielder FC and balloon dilatation performed to achieve thrombolysis in myocardial infarction I flow
- Fig 5: Chronic total occlusion of the left circumflex artery filling retrogradely from the right coronary artery
- Fig 6: A  $3.5 \times 20$  mm drug-eluting stent placed in left main-left anterior descending artery and post dilated with a non compliant balloon
- Fig 7: Intravascular ultrasound showing aneurysm around stent
- Fig 8: Thrombolysis in myocardial infarction II flow achieved post  $3.8 \times 26$  mm Covered stent deployment and dilation by 5x8 mm non compiant balloon
- Fig 9: Intravascular ultrasound post covered stent
- Fig 10: 3-month follow up angiography showing thrombolysis in myocardial infarction III flow

## VIDEO LEGENDS

- Video 1: Occluded left main coronary artery
- Video 2: Crossing of wire across left main coronary artery
- Video 3: Appearance of left main coronary artery aneurysm
- Video 4: Diagnoal, left anterior descending artery and ramus wired and ramus dilated with semi compliant balloon
- Video 5: Thrombolysis in myocardial infarction I flow achieved in left anterior descending artery, diagonal, and ramus
- Video 6: Left circumflex artery 100% chronic total occlusion filling retrogradely from right coronary artery

Video 7: Drug-eluting stent placed in left main-left anterior descending artery

Video 8: Intravascular ultrasound showing left main aneurysm around the stent

Video 9: Covered stent deployment

 ${f Video}$  10: Final angiogram after covered stent

 ${f Video}~{f 11}:$  Intravascular ultrasound post covered stent

Video 12: Thrombolysis in myocardial infarction III flow oberved at 3-month follow-up



















