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TWO STRIKES: A CASE STUDY OF CONSECUTIVE ST-ELEVATION MYOCARDIAL INFARCTIONS (STEMI) IN TWO DIFFERENT CORONARY ARTERIES

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ABSTRACT

An acute ST-elevation myocardial infarction (STEMI) followed by reinfarction within a short period of time is typically due to stent thrombosis. However, a STEMI caused by occlusion of one vessel followed by a repeat infarction due to occlusion of a different vessel which was seemingly innocent a few hours earlier is extremely rare. We present the case of a 61-year-old male with a past medical history of prediabetes, hyperlipidemia, tobacco use, and gastroesophageal reflux disease who presented to the emergency department with complaints of chest pain. His initial electrocardiogram (EKG) revealed ST elevation in leads II, III and aVF with reciprocal changes in leads I and aVL. He promptly underwent cardiac catheterization and had percutaneous coronary intervention with placement of two drug-eluting stents (DES) in the right coronary artery (RCA). At that time coronary angiography revealed 50% stenosis of the left anterior descending (LAD) artery and 60% stenosis of the second diagonal branch artery. Shortly after the procedure he was asymptomatic, and the post procedure EKG demonstrated resolution of the ST elevations. However, within 2 hours he developed chest pain and was found to have new ST elevations in the anterolateral leads. Repeat cardiac catheterization revealed patent RCA stents with subtotal occlusion of the LAD and another DES was placed. After the second procedure the patient remained hemodynamically stable, EKG changes resolved, and he was kept on eptifibatide infusion for 18 hours after which he was switched to dual antiplatelet therapy and ultimately discharged home.

KEYWORDS

ST-segment elevation myocardial infarction, recurrent myocardial infarction, early recurrence of myocardial infarction, STEMI

LEARNING POINTS

- Physicians should promptly address the recurrence of symptoms following an initial ST-elevation myocardial infarctions (STEMI) and be proactive regarding follow-up with the appropriate investigations.
- Although recurrence of STEMI within a few hours is extremely rare, the first 2 weeks following an initial STEMI is a critical time and patients should be educated on symptoms that will require further evaluation.
- The mortality associated with early recurrent myocardial infarction is up to 50% in 5 years so these patients require strict outpatient follow-up and counseling to minimize risk factors.

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INTRODUCTION

An acute ST-elevation myocardial infarction (STEMI) is associated with significant morbidity and mortality. Profound importance has been placed on improving the techniques to manage these events which has resulted in a significant reduction in the mortality. However, patients remain at a higher risk of other cardiovascular events[1]. Recurrence of an infarction, moreover within a short time after the previous one, poses an even higher risk of mortality. We present this case to arouse awareness of the possibility of recurrent STEMI within a remarkably short period of time and to highlight the importance of prompt action.

CASE DESCRIPTION

A 61-year-old male with a past medical history of prediabetes, hyperlipidemia, gastroesophageal reflux disease, and chronic tobacco use (20 pack-years) and a significant family history of cardiac disease presented to the emergency department complaining of chest pain. He described the pain as left-sided pressure radiating to his left shoulder and arm, accompanied by diaphoresis, upon awakening from sleep. He reported experiencing mild chest discomfort for the preceding 24 hours. Emergency medical services activated a code for STEMI after performing an electrocardiogram (EKG) on-site. The patient received 325 mg of aspirin and sublingual nitroglycerin en route, reporting slight improvement in pain.

Upon presentation, he appeared diaphoretic with a blood pressure of 136/90 mmHg, heart rate of 66 bpm, and otherwise unremarkable vitals. EKG revealed ST elevation in leads II, III, and aVF with reciprocal ST depression in leads I and aVL *(Fig. 1)*. He promptly received intravenous heparin and a loading dose of 180 mg of ticagrelor before undergoing cardiac catheterization. Angiography demonstrated subtotal occlusion of the mid to distal right coronary artery (RCA), 50% stenosis of the left anterior descending (LAD) artery, and 60% stenosis of the second diagonal branch artery *(Fig. 2A and B)*.

Additionally, a shelf-like 50% lesion in the proximal RCA raised concerns for possible dissection *(Fig. 2B)*. Percutaneous coronary intervention (PCI) involved balloon angioplasty followed by placement of two drug-eluting stents (DES) in the mid RCA. However, this was complicated by dissection proximal to the stent, necessitating placement of another DES in an overlapping fashion. Subsequent angiography identified another lesion in the distal RCA, treated with another DES. Repeat angiography revealed 0% residual stenosis in the RCA *(Fig. 2C)*, and the patient reported relief from chest pain. Post procedure EKG revealed resolution of the ST segment elevation.

The patient was then transferred to the coronary care unit and 2 hours later, complained of sudden severe substernal chest pain. EKG revealed new ST elevation in anterolateral leads V1-V6 *(Fig. 3)*, prompting a return to the catheterization lab. Repeat angiography demonstrated a patent RCA stent with TIMI 3 flow but subtotal LAD occlusion with plaque rupture and TIMI 1 flow, which was addressed with DES placement *(Fig. 4A and B)*. Further angiography revealed a second diagonal branch dissection, managed conservatively given restored flow, small caliber of the vessel and significant bridging *(Fig. 4C and D)*.

The patient's chest pain resolved, and he remained hemodynamically stable post procedure. Repeat EKG post procedure revealed resolution of ST segment elevation. He received eptifibatide infusion for 18 hours and continued on dual antiplatelet therapy (DAPT). Echocardiography revealed normal left ventricular function but right ventricular dilation, likely secondary to chronic obstructive pulmonary disease due to smoking. He developed a left groin hematoma postoperatively, which was resolved with conservative management. Extensive counseling against smoking and adherence to DAPT was provided. He was discharged with plans for follow-up with cardiologist, cardiac rehabilitation, and continuation of DAPT.

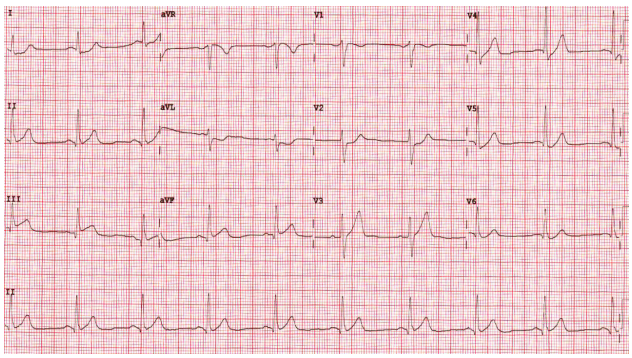

Figure 1. Electrocardiogram showing ST elevation in leads II, III, and aVF with reciprocal ST depression in leads I and aVL.

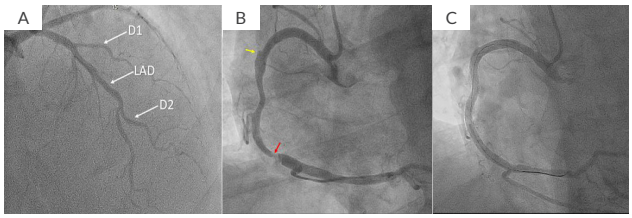

Figure 2. First coronary angiogram revealing: A) Moderate disease in left anterior descending artery (LAD). D1- First diagonal branch, D2- Second diagonal branch; B) Right coronary artery showing subtotal occlusion in the mid to distal area (red arrow), and a shelf-like 50% lesion in the proximal right coronary artery (yellow arrow); C) Right coronary artery with restored flow post intervention.

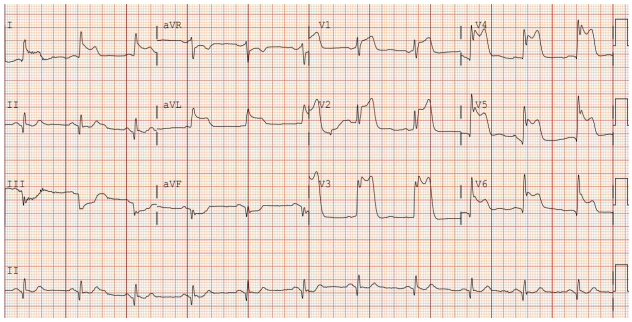

Figure 3. Electrocardiogram showing ST elevation in anterolateral leads V1-V6.

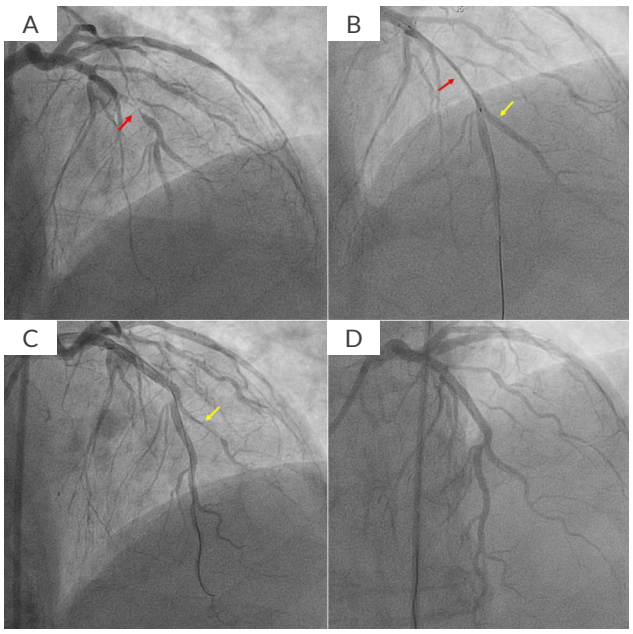

Figure 4. Second coronary angiogram revealing: A) New subtotal occlusion of the left anterior descending artery (LAD); B) Drug eluting stent being placed in the LAD (red arrow) prior to the second diagonal branch (D2) which has moderate disease (yellow arrow); C) Dissection in the second diagonal with disruption in flow (yellow arrow); D) LAD and D2 with restored flow post intervention.

DISCUSSION

An acute ST elevation myocardial infarction (STEMI) is due to the occlusion of a coronary artery usually secondary to plaque rupture or erosion or dissection of a coronary artery leading to thrombus formation. Dyslipidemia, diabetes mellitus, hypertension, chronic tobacco use, and family history of coronary artery disease are the major risk factors for the development of a STEMI.

The usual presentation involves only one major coronary artery occlusion however, rarely the simultaneous occlusion of two coronary arteries has been reported. A systematic review was conducted in 2015 outlining the various presentations and characteristics of patients with reported simultaneous multi-vessel occlusion. 56 patients were studied, and the results revealed the most commonly affected arteries to be the left anterior descending and the right coronary artery, while the most common presentation was cardiogenic shock[2].

A different but equally deadly presentation is that of early recurrent myocardial infarction following an acute STEMI. A study conducted in 2021 analyzed the characteristics and outcomes of patients readmitted for STEMI within 90 days of having had an acute myocardial infarction. It was found that the highest number of early recurrent myocardial infarctions took place within the first 2 weeks of discharge with the vast majority being within 2 days. Stent-related events accounted for a large proportion of the recurrent episodes. Lack of intervention during the initial myocardial infarction posed a higher risk for recurrence as well[1].

Recurrent myocardial infarction in a different coronary artery within a few hours of the initial STEMI suggests that there was rapid progression of plaques in a non-stented vessel enough so to cause complete occlusion. To the best of our knowledge there has only been one similar case reported in the literature[3]. Oftentimes patients presenting with a STEMI have more than one coronary plaque apart from the culprit lesion. Plaque progression is thought to happen in 2 distinct ways, slow linear progression, and rapid stepwise progression. A study in 2021 identified that lipid rich plaques, thin cap fibroatheromas and layered plaques were associated with rapid progression. The layered plaques suggest previous plaque disruption and subsequent healing[4].

The mechanism behind the unusually quick progression of a supposedly innocent lesion to becoming the second culprit is still unclear but some different hypotheses have been proposed. In the previously reported case, it was thought to be due to an inflammatory process related to the patient's urosepsis and disseminated intravascular coagulation[3]. Postinfarction inflammation characterized by inflammatory cell infiltration and activation of immune responses could also predispose to further adverse events[5]. Despite being given anti-inflammatory medications as per the guidelines our patient went on to develop reinfarction which suggests other factors yet to be properly studied must also be at play.

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