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RESEARCH ARTICLE

A CASE REPORT OF FULL REVASCULARIZATION OF ACUTE ST ELEVATION MYOCARDIAL INFARCTION PATIENT FOUND TO HAVE 2 CULPRIT LESIONS IN 2 DIFFERENT VESSELS

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ABSTRACT

ST-elevation myocardial infarction is commonly caused by intracoronary plaque rupture with thrombus formation and acute vessel closure in single coronary artery that is labelled as the culprit. The simultaneous thrombotic occlusions of more than one coronary artery in the setting of STEMI is a rare entity, which is not fully addressed in the current STEMI guidelines, although more recent studies suggest a benefit of complete revascularization compared to culprit vessel-only treatment in the setting of STEMI. In this report we presented a case of STEMI with 2 culprit vessels.

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INTRODUCTION

33-year-old male, smoker, epileptic, non-diabetic, non-hypertensive with negative family history of coronary artery diseases or sudden cardiac death. presented to emergency department with acute anginal chest pain of 2 hours duration, associated with nausea and vomiting. His blood pressure was 135/75, heart rate 60 beat per minute, respiratory rate 14 breath per minute. His initial electrocardiogram showed inferior, septal and lateral ST elevation with lateral reciprocal ST depression, his emergency laboratory results were within normal range including complete blood count, kidney function test, coagulation profile, creatine kinase and troponin level. He was sent emergently for coronary catheterization laboratory to clarify his coronaries and proceed with the appropriate revascularization strategy. Patient pre-medicated with chewable aspirin 325 mg, intravenous unfractionated heparin 4000 I.U., oral ticagrelor 180 mg. Emergent bedside echocardiogram showed ejection fraction of 50% with mild inferior-basal hypokinesia without valvular dysfunction or pericardial effusion.

His coronary angiogram showed normal left main coronary artery, distal left descending artery thrombotic total occlusion, normal left circumflex artery, dominant right coronary artery with distal thrombotic total occlusion. Decision taken to proceed with primary percutaneous coronary intervention, patient given bolus does of tirofiban intravenously, then distal left anterior descending artery lesion wired then pre dilated using complaint balloon 2*15 then 3*28 drug eluting stent implanted with final TIMI III flow.

Distal right coronary artery lesion also wired and pre dilated using complaint balloon 2*15 then 3*32 drug eluting stent implanted with final TIMI III flow. After revascularization patient sent to coronary care unit for observation and tirofiban infusion maintenance dose and intravenous nitroglycerin as he still complaining of anginal chest pain less severe than presenting one. The day next patient still getting anginal pain without significant electrocardiogram changes, so he was sent to catheterization laboratory again and another coronary angiogram done which showed patent stents with TIMI III flow in both stented arteries, and his pain managed medically.

3 days later, patient discharged home as he is free of symptoms. Next clinic follow up visit was 3 months later, thrombophilia screen, homocysteine level, coagulation profile and connective tissue screen were within normal ranges, Toxicology screen also reported free.

CONCLUSION

Acute coronary syndrome can be caused by more than one culprit vessels. Full revascularization strategy can be adopted, as both lesions have thrombus and should managed emergently especially in ST elevation setting, continuous anginal pain, hemodynamic or electrical instability or less than TIMI III flow.

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