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Acute Myocardial Infarction due to Double Coronary Artery Thrombosis: Double Trouble. Case Report from Two Subjects

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Abstract

Acute ST elevation myocardial infarction (STEMI) from simultaneous thrombosis of more than one coronary artery is an uncommon entity. The outcome can be devastating due to the large myocardium that is affected. The mechanism of simultaneous multivessel coronary occlusion is not clear. We should consider conditions such as essential thrombocythemia, multivessel spasm, hypercoagulable state, cocaine abuse, endocarditis, left ventricular clot emboli, or a paradoxical emboli. Traditional risk factors such as diabetes, hyperlipidemia, and smoking also play a role. Here we describe two cases wherein simultaneous thrombotic occlusion were noted in the same setting.

Index terms— myocardial infarction, double coronary occlusion, cardiogenic shock.

1 Introduction

imultaneous thrombosis in more than one coronary artery is an uncommon finding seen in nearly 2.5% of all patients undergoing primary percutaneous intervention (PCI). (1) The incidence is low as most of these patients die from sudden cardiac death (SCD). Autopsy studies in SCD patients revealed a higher incidence of nearly 50%. (2) Several theories are postulated for simultaneous thrombosis including the presence of a hypercoagulable state or increased catecholamine surge and inflamematory response caused by the occlusion of the first artery causing thrombosis of the other, but the cause is still unclear [1]. In this study we present two cases of double coronary thrombosis myocardial infarction and discuss our management of the case, the possible causes and the outcomes of this high-risk presentation.

2 II.

3 Case Report

4 Patient 1

A 25 year old male patient with no risk factors presented to our institute with complaints of severe retrosternal chest pain of 2 hours duration with diaphoresis. No prior history of exertional angina or breathlessness.

On examination he had a pulse rate of 34 bpm, BP of 100/60 mm Hg. Cardiac examination was unremarkable.

ECG revealed ST elevation in leads V2-V6, II, III, aVF with 2:1 AV block (Fig. **??**). He had received two doses of Reteplase 10 mg each 30 minutes apart. The pain was persistent and repeat ECG showed ST elevation in leads II, III, aVF with resolution of ST elevation in precordial leads (Fig. **??**). A coronary angiogram was subsequently performed which revealed Mid LAD 80% thrombus containing lesion and Mid RCA total occlusion (Fig. **??**, 4, Video 1, 2).

The RCA lesion was crossed using a guide wire. This was followed by deployment of a drug eluting stent (DES), with good angiographic results (Fig. **??**, Video 3). The patient was given Gp IIb/IIIa inhibitor. A check CAG after one day revealed mild disease in Mid LAD (Fig. **??**, Video 4). The patient was discharged on Ecosprin 75 mg OD, Ticagrelor 90 mg BID, Rosuvastatin 40 mg OD, ACE inhibitor and beta blocker.

5 Patient 2

A 46 year male patient, smoker presented to us with complaints of sudden onset retrosternal chest pain with radiation to left shoulder with sweating. No prior history of exertional angina or breathlessness.

On examination he had a pulse rate of 94 bpm and BP of 100/60 mm Hg. Cardiac examination was unremarkable.

ECG revealed ST elevation in leads aVR with ST depression in leads II, III, aVF, V2-V6, I, aVL (Fig. **??**). A diagnosis of acute posterior wall MI was made. A coronary angiogram was performed which revealed total occlusion of Proximal LCX and Mid RCA (Fig **??**, 9, Video 5, 6). The LCX lesion was crossed using a guide wire. The lesion was predilated with a 2.5 X 15 mm Across HP balloon @ 10 atm followed by deployment of a 2.75 x 28 mm Xience prime DES and 2.5 x 12 mm Xience Xpedition DES (Fig. **??**0, Video 7).

The RCA lesion was crossed using a guide wire. The lesion was predilated with a 2.5 x 15 mm balloon followed by deployment of a 2.5 x 23 mm Xience prime DES, postdilated with a 2.75 x 12 mm NC balloon with good angiographic results (Fig. **??**1, Video 8).

6 S

III.

7 Discussion

Acute STEMI is usually due to occlusion of a single coronary artery, but rarely we come across occlusion in more than one coronary arteries simultaneously. The incidence is reported to be around 1.7 to 4.8 % of all primary PCI (1), but autopsy studies in patients of SCD have reported simultaneous occlusion of more than one coronary vessel in 50% of the cases.

(2) The low incidence is owing to higher complications in such cases wherein death occurs before reaching the hospital. Simultaneous multi-vessel coronary thrombosis can occur secondary to cocaine abuse, anti-thrombin III deficiency, idiopathic thrombocytopenic purpura, as well as thrombophilias such as antiphospholipid antibodies, factor V Leiden deficiency, and essential thrombocytosis. (3) The most common presentation is cardiogenic shock or sudden cardiac death which occurs in 40-50% of the cases factors involved in simultaneous acute thrombosis of multiple coronary arteries is not clear, possible factors include (a) catecholamine surge with heightened inflammatory response caused by the acute occlusion of 1 vessel, resulting in a second coronary arterial occlusion; (b) hypotension from hemodynamic instability due to the occlusion of 1 coronary artery, resulting in stasis and acute occlusion in another artery with a preexisting lesion; (c) prolonged coronary vasospasm (cociaine use or Prinzmetal's angina (5) (d) hypercoagulable states due to malignancy and thrombocytosis (6) and (e) coronary embolism (7).

Multiple coronary artery thrombosis with ST-segment elevation MI are uncommon. Isolated cases of simultaneous occlusion of coronary artery are reported in the literature, as a result defining treatment strategies is difficult. Revascularization should be done as early as feasible and mechanical support may be necessary to improve the outcome.

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