Case Report
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Acute Coronary Stent Thrombosis: A Rare Case Report

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ABSTRACT
Coronary stent thrombosis is an uncommon but potentially catastrophic complication of percutaneous coronary intervention, which can result in acute myocardial infarction and often death. The most consistent predictive factors include premature discontinuation of antiplatelet therapy, the extent of coronary disease, and stent number/length. We present a case of acute stent thrombosis in a patient with Acute Myocardial infarction undergoing Coronary Angiolplasty which occured within half an hour of stents placement along with a review of the literature.

Keywords: *coronary stent thrombosis; myocardial infarction; percutaneous coronary intervention*.

INTRODUCTION

Bare metal stents (BMS) were introduced in the late 1980s and have been replaced by drug-eluting stents (DES) over time due to decreased risk of myocardial infarction, cardiovascular complications, and mortality.[1] While each type of stent carries its own risks and benefits, stent thrombosis, most commonly seen in DES, remains a rare but often fatal complication. Drugs used in these stents may halt re-endothelization of smooth muscle and delay healing, inducing tissue growth expression and promoting thrombogenicity.[2] Stent thrombosis is a rare but potentially catastrophic complication of percutaneous coronary intervention (PCI), with an incidence of 0.1-1.7% and a mortality rate of over 45%.[3] While no single risk factor has been proven to predict stent thrombosis, the most consistent predictive factors include premature discontinuation of antiplatelet therapy, the extent of coronary disease, and stent number/length.[4]

CASE REPORT

A 45-year-old male with a past medical history of systemic hypertension with type 2 DM presented with central chest pain on and off since 10 days. The pain was intensified since morning. Pain was described as a "pressure-like" sensation and radiating to inner aspect of his left arm which was aggravated during and after having meal and associated with sweating. At presentation, vital signs showed a heart rate of 80 bpm with a respiratory rate of 16 breaths/minute, blood pressure of 130/80 mmHg, and oxygen saturation (SpO2) of 98% on room air. A thorough physical exam was unremarkable. Pertinent blood work showed a creatinine of 0.7 mg/dl (unknown baseline), Troponin I at 0.25ng/L, CPK-MB of 6.80 ng/L, total cholesterol of 285.4 mg/dl, triglyceride(TG) of 332.6 mg/dl, low-density lipoprotein (LDL) cholesterol of 167.6 mg/dl and VLDL of 66.5 mg/dl. Initial electrocardiogram (EKG) showed ST-elevations in leads II, III, aVF and leads V5-V6 with ST-depressions in I and aVL (Figure-1), consistent with acute inferolateral wall ST-elevation myocardial infarction (STEMI), requiring emergent cardiac catheterization.

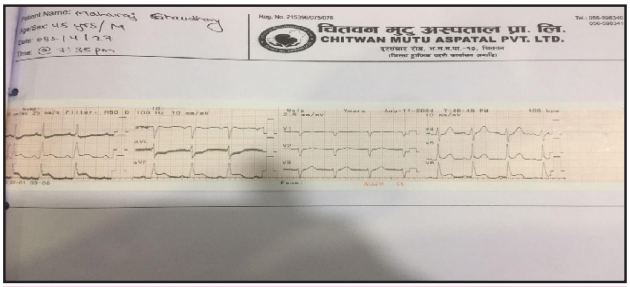

Figure 1. ECG showing ST elevation in II, III, aVF, V5, V6.

The patient underwent coronary angiography, which demonstrated triple vessel CAD with 80-90% stenosis in the middle left anterior descending artery

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(LAD), 95% stenosis in the left circumflex artery (LCX), and RCA with diffuse lesion, proximal to mid 70-80% stenosis and 100% occlusion distally with retrograde collaterals (CTO) from LAD. PCI was performed in the mid-LAD and distal LCX with successful placement of two everolimus-DES (EES) (3 mm x 36 mm and 2.75 mm x 12 mm, respectively) after thorough pre-dilatation with Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow (Figure 2 and 3). The procedure was completed without

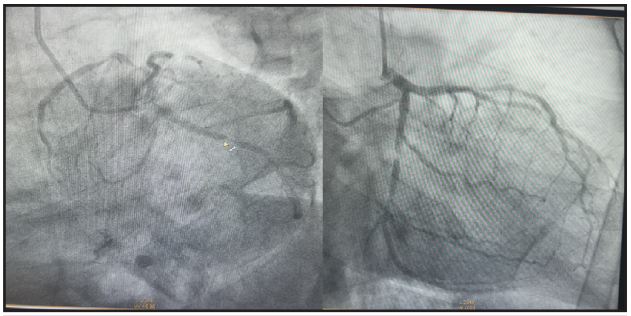

Figure 2. Coronary Angiography (CAG) showing significant stenosis in Mid LAD and Distal LCX.

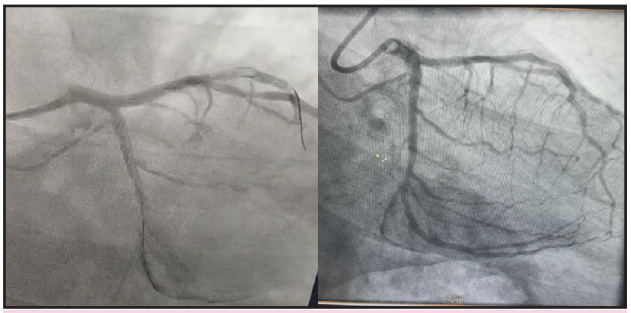

Figure 3. Coronary Angiography (CAG) after deployment of stents in Mid-LAD and LCX with DES.

complications.

Within 30 minutes of stents placement, the patient presented with severe chest pain with repeat EKG showing worsening inferolateral ST elevations and new ST elevations in anterior leads (Q-RBBB in V1)

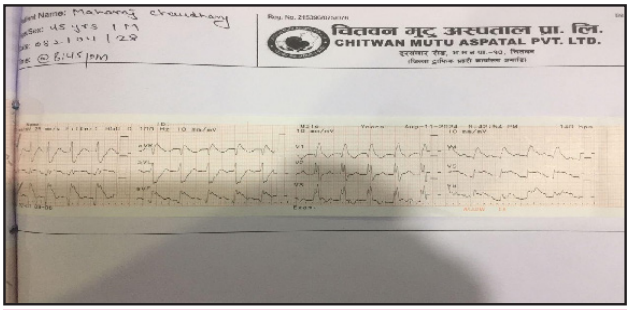

Figure 4. Electrocardiogram after 30 mins of stent deployment with severe chest pain showing ST elevation in Inferior and Anterior leads.

in V1-V4 (Figure 4).

Given his age and acuity of symptoms in the setting of worsening EKG, a repeat angiogram (Figure 5) was obtained immediately to ensure patency of the stent, which revealed a thrombotic stent occlusion in

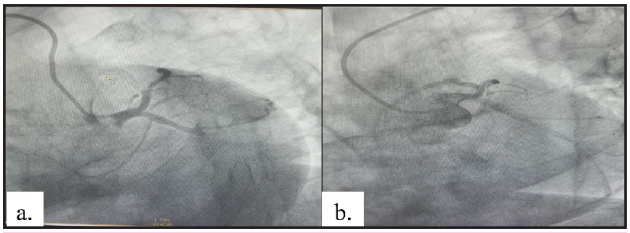

Figure 5. a) CAG showing Stent thrombosis in Mid-LAD and Distal LCX with (b) re-perform PCI.

both LAD and LCX.

Following the procedure, he was started on tirofiban infusion for 48 hours along with dual antiplatelet therapy (DAPT) with ticagrelor. He developed pulmonary edema on next day(Chest x-ray figure 5) for which he was kept on mechanical ventilation. Repeat angiography was done on day 3 which showed TIMI flow grade III(Figure 5b). Patient self-extubated on day 4 following which maintained saturation via n/p @4l of O2.Echocardiogram revealed regional wall motion

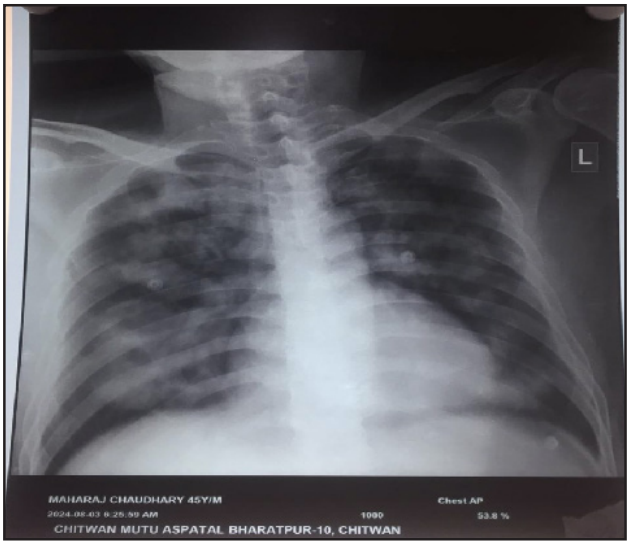

Figure 6. CXR showing Pulmonary edema after redo angioplasty of stent thrombosis.

abnormalities with Mild LV systolic dysfunction.

Upon stabilization, the patient was discharged on eighth day on DAPT (aspirin and ticagrelor), high-intensity rosuvastatin, and metoprolol succinate with outpatient follow-up with cardiologist. Patient was stable at 2 weeks, 4 weeks and 3 months follow-up.

DISCUSSION

PCI is the gold standard for coronary re-vascularization through angioplasty and placement of DES or BMS. The study of DES has helped cardiologists avoid restenosis and thrombosis by releasing of drugs[4], with current-generation stents including EES. Despite the well-studied literature and the minimally invasive nature of the procedure, a myriad of complications of both cardiac and non-cardiac origin have to be taken into consideration. A definite stent thrombosis is defined as an angiographic thrombus originating in the stent or within 5 mm of the stent, with or without vessel occlusion, associated with ischemic symptoms, acute changes in EKG, or changes in cardiac biomarkers. The timeframe begins once the patient has been undraped and removed from the catheterization laboratory. According to the Academic Research Consortium (ARC) revision, an acute stent thrombosis must occur within the first 24 hours after stent implantation (0.4% of cases).[3] Other time frames include subacute (24 hours to 30 days), late (31 days to one year), and very late (>one year) (incidence of 1%, 0.4%, and 0.5%, respectively).[3,5] In the case of our patient, occlusion and manifestation of symptoms occurred within 30 minutes of stent placement. Overall, stent thrombosis is characterized by platelet activation and aggregation, but causes are multifactorial and may include reduced left ventricular ejection fraction, malignancy, smoking, or factors concerning the procedure itself, such as stent malapposition or length, and dissections.[6] Other causative factors for thrombosis include small vessel lesions, chronic occlusive lesions, intramural hematomas, or bifurcation of lesions. Regardless, premature discontinuation of DAPT remains the front-runner for stent thrombosis.[6] DAPT relies on the synergy of cyclooxygenase-1 inhibitors (aspirin) and P2Y12 receptor inhibitors (clopidogrel, prasugrel, or ticagrelor) to prevent clot formation.[7] The length of DAPT depends on the stability of ischemic heart disease, the type of stent used, and the bleeding risk. Patients with acute coronary syndrome (ACS) have a recommended period of at least 12 months with aspirin and P2Y12-inhibitor therapy; whereas a shorter duration is feasible in patients with a history of bleeding.[8,9] Premature discontinuation of DAPT, particularly at six months in those with DES, with ACS and no overt signs of bleeding, represents an increased risk for myocardial infarction.[10,11] If necessary, therapy prolongation can be assessed with risk-assessment scales such as the PRECISE-DAPT score.[12] However, more recent studies have shown limitations such as irreversibility and hepatic conversion from pro-drug to active metabolite.[8] The approval of more novel P2Y2 inhibitors, such as prasugrel and ticagrelor, has increased the challenges and risk assessment when selecting an adequate agent to protect from ischemic events while balancing an acceptable bleeding risk. In the case of our patient, ticagrelor was chosen given the patient's age and superiority over other agents at reducing the one-year incidence of ischemic events without significant bleeding risks.[13]

CONCLUSION

Restenosis and occlusion of the coronary stent remain a complication of PCI despite years of investigation and, in some cases, no evident risk factors for occlusion. While the mechanisms of injury may include patient characteristics or direct complications of the procedure, these may develop at any stage after the procedure and at this time there is no fault-proof method to prevent it. In the meantime, DAPT remains the mainstay of therapy for preventing stent thrombosis, but its efficacy is also based on patient characteristics as well as compliance. While studies may show minor advantages for particular P2Y12 agents, analysis on efficacy and safety among the three agents show equal mortality rates at one year.

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