An Unusual Cause of Sub acute Stent Thrombosis — A Case Report and Review of the Literature

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Abstract
Stent thrombosis (ST) is an ominous clinical development that has a significant impact on clinical outcomes. Stent thrombosis is often multifactorial. Stent thrombosis can be manifested by serious complications such as nonfatal and fatal myocardial infarction and marked hemodynamic instability. Early diagnosis and expedient thrombus management is the key to a favorable outcome, especially for patients who present with ST-elevation Myocardial Infarction (STEMI). Percutaneous coronary interventions (PCI), including additional stenting with or without thrombectomy, is effective in restoring vessel patency. Every effort should be made to determine the cause of stent thrombosis. Even successful PCI for stent thrombosis is associated with a larger infarct and poorer outcome than in patients with de novo STEMI. We report a case of 58 year old male admitted in our center with stent thrombosis, manifesting as STEMI. Heparin-induced thrombocytopenia and thrombosis (HIT) was identified as the cause of stent thrombosis. Coronary angiography and PCI was carried out with direct thrombin inhibitor. The condition of the patient improved and was discharged after 5 days.

ABBREVIATIONS
ST: Stent Thrombosis; STEMI: ST Elevation Myocardial Infarction; HIT: Heparin Induced Thrombocytopenia; PCI: Percutaneous Coronary Intervention

INTRODUCTION
Stent thrombosis, the most feared complication related to coronary stent placement is relatively rare; however, its consequences can be fatal, as most ST cases are associated with acute coronary syndrome (ACS) or sudden death. The Academic Research Consortium has proposed criteria for the timing and definitions used to document stent thrombosis in clinical studies. The Timing of stent thrombosis is defined as acute (<24 hours), subacute (24 hours to 30 days), late (30 days to 1 year), and very late (after 1 year) [1]. Early stent thrombosis (<30 days) rates for elective PCI in stable coronary artery disease patients ranges from 0.3% to 0.5%, but stenting for acute coronary syndromes demonstrated early stent thrombosis rates as high as 3.4% and 1.4% for STEMI and non-ST-elevation myocardial infarction (NSTEMI), respectively [2,3]. Treatment for ST almost always requires emergent repeat PCI, although optimal reperfusion is only achieved in two-thirds of patients [4]. We report a case of 58 year old male admitted in our center with stent thrombosis, manifesting as STEMI. Heparin-induced thrombocytopenia and thrombosis was identified as the cause of stent thrombosis. Coronary angiography and PCI was carried out with direct thrombin inhibitor. The condition of the patient improved and was discharged after 5 days.

CASE PRESENTATION
58 year old male patient admitted inother hospital with acute onset of chest pain at rest associated with sweating since 6 hours. He is a chronic smoker since 20 years, hypertensive since 10 years and diabetic since 5 years. Troponin T was positive. Coronary angiography was done which showed a significant lesion in LAD. A drug eluting stent 2.75 x 43 was deployed in LAD. The patient was discharged from that hospital after 3 days (Table 1: Timeline of events). Patient again developed acute onset of chest pain, at rest and associated with profuse sweating in morning 1 am after 4 days of discharge. Patient was admitted in same hospital.

Echocardiogram showed basal, mid, distal anteroseptal, anterior and anterolateral segment hypokinesia.
Ejection fraction was 30%. Routine blood investigations were normal except the platelet count. Platelet count was 84,000/mm³. Review of the previous blood reports done showed last platelet count of 248,000/mm³. Patient was given unfractionated heparin, infusion followed by bolus 6 hourly in the previous hospital, as per the discharge card. We suspected heparin-induced thrombocytopenia and thrombosis as the cause of patient's present condition. We planned for coronary angiography with direct thrombin inhibitors. Bivalirudin was not available in our hospital. It took us 2 hours to arrange bivalirudin. Patient was taken for coronary angiography after 1 hour of bivalirudin infusion.

Coronary angiogram showed total thrombotic occlusion of LAD at the proximal end of the stent (Figure B). Lesion was crossed with PTCA wire and thrombus aspiration was done. Check shoot after thrombosuction showed good result with residual lesion at the distal end of the stent. This area was balloon dilated and stented with 2.75 x 18 mm Drug eluting stent. Proximal half of the previous stent was dilated with 3 x 13 mm noncompliant Balloon. Check shoot showed good result (Figure C). Patient was shifted to intensive care unit and bivalirudin infusion was continued for 4 hours. Within 24 hours the general condition of the patient improved significantly and was shifted to ward. Electrocardiogram is shown in the Figure D. Platelet count improved to 112,000/mm³ in the next 24 hours. Patient was discharged on 6th day of the procedure on dual antiplatelets (aspirin and prasugrel), metoprolol, ramipril, statin and eplerenone. Patient is asymptomatic at 6-week followup.

**DISCUSSION**

Stent thrombosis, a devastating complication of stent implantation although fortunately rare (occurring in approximately 0.5–1% of patients within 1 year), most commonly presents as an acute myocardial infarction [5]. Stent thrombosis has been associated with 30-day mortality rates of 10–25%. Moreover, approximately 20% of patients with a first stent thrombosis experience a recurrent stent thrombosis episode within 2 years [6]. The factors contributing to early ST are divided into patient-related factors, lesion-related factors, and procedure-related factors. Stent thrombosis occurs more frequently in complex patients and lesions, especially in those with acute coronary syndromes, thrombus containing lesions, diabetes mellitus, chronic kidney disease and diffuse disease, small vessels, and bifurcation lesions requiring multiple stents. The procedure-related factors include poor stent expansion, stent malapposition, and edge dissections. Very rarely early stent thrombosis occurs due to heparin-induced thrombocytopenia.

Heparin-induced thrombocytopenia (HIT), when associated with thrombosis (HITT) is an adverse immune-mediated drug
Thrombocytopenia due to HIT may be either relative (50% decrease from baseline) or absolute (less than 150,000/mm³). Various laboratory tests, including Enzyme Immunoassay, Serotonin Release Assay or heparin-induced platelet activation testing are available for the diagnosis of HIT with varying sensitivities and specificities but are performed only in certain laboratories.

Heparin is an essential drug in the treatment of ACS and it is used during PCI. Five previous cases of acute and sub-acute coronary stent thrombosis secondary to HIT have been reported by Cruz D et al, Gallagher MJ et al, Hussain F et al, Shin HW et al and Narasimha D et al. When stent thrombosis due to HIT is suspected, all heparin products should be discontinued and PCI is performed using alternative anticoagulants like direct thrombin inhibitors. In our patient, the diagnosis of HIT was based on “4T score”. Our patient had 4T score of 7, which indicate high probability of HIT. Coronary angiography and PCI was performed using bivalirudin. With the help of thrombus aspiration and small additional stent at the distal end of the previous stent, the adequate result was achieved. In this situation, Intravascular ultrasound (IVUS) or optical coherence tomography (OCT) is ideal to rule out procedure-related and stent related causes of stent thrombosis like stent underexpansion, malapposition or edge dissection. Platelet counts improved during the hospital stay as shown in Table.

CONCLUSION

Although very rare, whenever early stent thrombosis occurs, we should always consider HIT if the patient had been exposed to heparin previously. Diagnosis is simple. The management of patients with stent thrombosis due to HIT, undergoing PCI is challenging. Immediate cessation of all heparin products and initiation of alternative anticoagulation is recommended. There are no specific guidelines or studies addressing anti-platelet therapy after coronary stenting in HIT patients.

REFERENCES


