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Case Report

An Unusual Case of Recurrent Stent Thrombosis

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Abstract

Stent thrombosis is a dramatic event resulting in ST elevation MI with high mortality rate. In general subacute stent thrombosis is more common. Risk factors associated with stent thrombosis can be clinical, angiographic or procedural factors. Herein we report a case of recurrent stent thrombosis on second generation p2y12 inhibitors and an extensive workup for coagulation disorders was negative. Ultimately the patient underwent CABG. The cause for recurrent stent thrombosis in our case remains a mystery..

***Keywords**: Stent Thrombosis; ST Segment Elevation*

Introduction

Stent thrombosis is a dramatic event resulting in ST segment elevation myocardial infarction with high mortality rate [1]. Previous studies have reported incidence rates of recurrent stent thrombosis of 10% to 20% on long term follow up [2]. Clinical, angiographic and procedural factors predispose to stent thrombosis. Noncompliance to medical therapy, diabetes, renal failure, young age, smoking, presence of malignancy, and stenting for myocardial infarction (primary angioplasty) are usually the predisposing factors. Small vessel diameter, the use of long stents and multivessel disease are the angiographic factors predisposing to stent thrombosis. Stent undersizing, bifurcation stenting, and dissection are the procedural factors predisposing to stent thrombosis. Discontinuation of antiplatelet therapy within the first 30 days after stenting is arguably the most important predictor of stent thrombosis [3]. Herein in this case report we want to highlight repeated episodes of subacute stent thrombosis in a patient on potent antiplatelet agents. Ultimately the patient underwent coronary artery bypass graft (CABG).

Case Report

A 39 years old male obese, smoker, hypertensive, diabetic patient suffered from non ST segment elevation anterior wall myocardial infarction [NSTEMI] on 26.09.2020 was admitted to a peripheral hospital. Coronary angiography revealed sub -total left anterior descending artery [LAD] occlusion in the proximal segment patient was loaded with clopidogrel and aspirin. He underwent percutaneous transluminal coronary angioplasty [PTCA] to proximal LAD and Xience V stent drug eluting stent was implanted and subsequently patient was discharged on 28.09.2020 after stabilization. Patient developed acute onset chest pain with ST segment elevation in anterior leads on 30.09.2020. He was re-admitted and check coronary angiography revealed subacute- stent thrombosis. Patient was given prasugrel. Repeat PTCA was done and thrombosuction and balloon dilatation was done and there was distal stent edge dissection and an overlapping XIENCE V STENT was put. After the procedure patient again developed retrosternal chest pain after 6 hrs and he was brought to the emergency of Jaypee Hospital for further management. Since the patient was continuing to have chest pain and there was persistent ST segment elevation in leads V2-V5 he was taken for urgent coronary angiography which revealed recurrent sub-acute stent thrombosis (LAD stent 100% total occlusion (Figure 1).

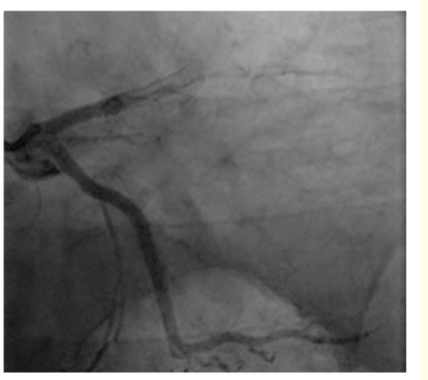

Figure 1: *Lad 100% stent occlusion.*

After loading Patient with ticagleror [180 mg] he was taken up for high risk angioplasty. Lesion was crossed with whisper.014 wire and balloon dilatation was done using Sapphire Balloon 2.5 x 15 mm at 10 atm and intracoronary abciximab bolus was given (Figure 2).

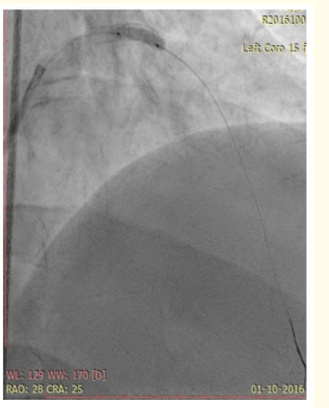

Figure 2: *Balloon dilatation with 2.5 - 15 mm balloon.*

Thrombosuction was done with 6F thrombuster 3 passes were made (Figure 3).

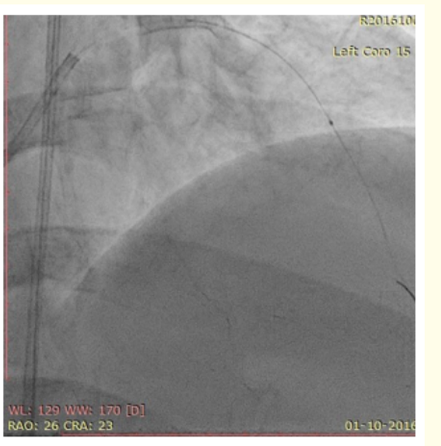

Figure 3: *Thrombosuction with 6f thrombuster catheter.*

We do not have intravascular ultrasound in our laboratory. TIMI III flow was achieved in LAD but there was thrombus burden present in first Diagonal and the diagonal could not be wired because of overlapping stents (Figure 4).

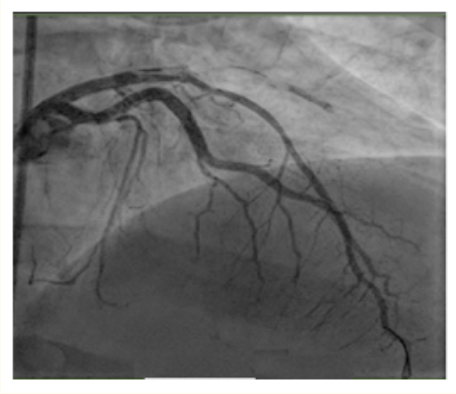

Figure 4: *TIMI 3 flow in lad and thrombus in diagonal.*

Post procedure he was kept on abciximab infusion 17 ml/hr and low dose heparin infusion. Post procedure his troponin I was raised and there was resolution of ST segment elevation and chest pain. In the ICU he had an episode of ventricular tachycardia which was DC Cardioverted. He was managed with cordarone infusion, DAPT, heparin and low dose inotropes. His 2 D Echo revealed RWMA in LAD territory with LVEF 30 - 35%. Patient remained asymptomatic and was shifted to the ward. Check coronary angiography was done after 7 days and it showed proximal 100% in stent occlusion in proximal stent) (Figure 5).

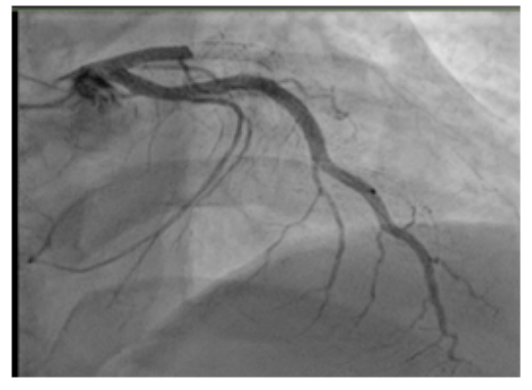

Figure 5: *100% stent occlusion.*

Distal LAD was filling retrogradely by type II collaterals from RCA and LCx. Since the Patient was asymptomatic patient was kept on medical therapy and CABG was advised. Patient also underwent an extensive hypercoagulable workup for coagulation disorders. The patient tested negative for antinuclear antibody [ANA], IGA/IGM anticardiolipin antibody, homocysteine, protein C, protein S, factor V assay, and antithrombin111 deficiency. Patient was kept on triple antiplatelet therapy [aspirin, ticagrelor and cilostazol] and he was discharged.

Subsequent to discharge patient again had chest pain and he underwent PET Scan which revealed viability in LAD territory and subsequently patient underwent CABG with lima to lad and diagonal [sequential].

Discussion

Subacute stent thrombosis has been classified by the Academic Research Consortium (ARC) as definite, probable, or possible and further subclassified according to time as acute (0 to 24h), subacute (1 to 30 days), late (31 to 360 days), and very late (> 360 days) [5]. The high incidence of subacute stent thrombosis with bare metal stents was a major limitation. Stent thrombosis is a life-threatening complication of percutaneous coronary intervention but rarely occurs because of 3rd generation drug eluting stents and potent antiplatelet agents [prasugrel, ticagleror]. The most common reasons for subacute stent thrombosis are early antiplatelet therapy discontinuation, number and length of the stents, acute coronary syndrome, diabetes, smoking, and bifurcation or ostial disease [4]. Early stent thrombosis (EST) (less than 30 days after stent implantation) is a rare but severe complication which could present as ST-elevation myocardial infarction (STEMI) or sudden cardiac death within the first 30 days after stent implantation [5]. Previous studies have shown that several patient-related variables are associated with EST during ACS, such as suboptimal antiplatelet administration, insulin-requiring diabetes, hypertension and baseline renal insufficiency [6]. In addition to several other independent predictors, such as final stent minimal luminal diameter, non-administration of thienopyridine prior to percutaneous coronary intervention (PCI) and high baseline haemoglobin levels [7]. Small case series have identified a number of possible contributors to recurrent stent thrombosis, including hypercoagulable states, increased platelet turnover, and rheumatological dis-eases [8]. Recent studies have shown that adequate anti-platelet effects are not achieved in 5 - 45% of the patients taking aspirin and in 4 - 30% of patients taking clopidogrel and therefore suggest that many patients are hyporesponsive to the anti-platelet agents [9]. Though the focus has shifted to late ST since the BASKET LATE trial was reported, most series reporting DES thrombosis have shown that acute or subacute stent thrombosis still outnumbers late events and hence remains an important entity [10]. Stent thrombosis (ST) represents a potentially life-threatening and fatal outcome following PCI (e2) which is associated with a mortality rate of between 5 and45%, as well as a recurrence rate of 15 - 20% at 5 years [11].

Conclusion

We report this case to highlight the repeated occurrence of subacute stent thrombosis despite being treated with newer generation P2Y12 inhibitors prasugrel and ticagrelor and the absence of any hypercoagulable disorders on an extensive coagulation workup. The limitation was that we could not do intravascular ultrasound to determine the mechanism since it was not available in our laboratory. For us it remains a mystery as to the cause of recurrent stent thrombosis in our patient.

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