Pak Heart J

"VERY LATE BARE-METAL STENT THROMBOSIS"

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ABSTRACT

Stent thrombosis (ST) is an acute thrombotic occlusion of a stent in a coronary artery. It is a serious complication. An acute myocardial infarction or sudden death is the most common presentation of stent thrombosis. According to Academic Research Consortium classification, ST can be early when occurring within 30 days, late when it occurs between 30 days to 1 year or very late when it occurs after 1 year. Very late ST after drug-eluting stent implantation is rare. But very late ST after bare-metal stent (BMS) implantation is unsual. Here we report a case of ST-elevation myocardial infarction occurring 2 years after BMS implantation (very late ST).

Key Words: Stent Thrombosis, Percutaneous Coronary Intervention, Bare Metal Stent.

INTRODUCTION

Stents have improved the safety and efficacy of percutaneous coronary interventions (PCI) by reducing acute vessel closure and by reducing re-stenosis rates compared with conventional balloon angioplasty.[1] However, stent thrombosis (ST) remains a serious complication of PCI particularly with drug-eluting stents (DES). ST is a rare condition but it is associated with 20–40% mortality and 50-70% incidence of acute myocardial infarction (MI) Academic Research Consortium (ARC) categorizes stent thrombosis as: **Definite ST:** Angiographic or pathologic proven, **Probable ST:** Unexplained death within 30 days of stent implantation or myocardial infarction in the territory of the implanted stent, **Possible ST:** any unexplained death after 30 days.[2]

It is also classified based on the time elapsed since stent implantation: **Early:** ST between 0-30 days post stent implantation, **Late:** ST >30 days after stent implantation, **Very Late:** ST >12 months after stent implantation.[2]

Early stent thrombosis is further categorized into acute (ST occurig within 24 hours) and sub-acute (ST occurring between 24 hours-30 days) events. ST occurs mostly within 30 days of stent implantation. Bare-metal stents (BMS) has been associated with early ST, whereas very late ST has been reported with drug-eluting stents (DES) and is due to delayed stent endotheliazation. The incidence

of definite very late ST with DES is 0.4–0.6% per year. While very late stent thrombosis (VLST), occurring beyond 1 year, has been increasingly described with the use of drug-eluting stents, VLST with bare-metal stents is not very common.[3] We report a case of Stent thrombosis occurring 2 years after BMS implantation (VLST).

CASE REPORT

A 73 year old man, resident of Gilgit, had sudden onset, severe chest pain radiating to left arm about 7 days prior to his admission at our hospital. He got admitted at local hospital in Gilgit with the diagnosis of acute anterior wall myocardial infarction. The facility of primary PCI was not available and streptokinase was not given due to history of peptic ulcer disease. He was managed with aspirin, clopidogrel and enoxaparin. He was then referred to us for further management. He was an ex-smoker and had a history of hypertension, IHD and renal dysfunction. In the past he had percutaneous coronary intervention (PCI) for stable angina (CCS III) in 2010, with placement of 1 drug-eluting stent in mid LAD (Xience 3.0 x 38 mm), 2 bare-metal stents in proximal to mid LAD (Express 4.5 x 24mm and Vision 4.0 x 15 mm) and 3 bare-metal stents in proximal to mid LCx (Vision 4.0 x 18 mm, Vision 3.5 x 15 mm and Micro Driver 2.75 x 14 mm) with overlapping of edges. At the time of presentation to our hospital he had ongoing chest pain. He was hemodynamically stable with Killip class II. ECG showed q's and ST segment elevation in anterior leads. His serum creatinine was raised to 2.1 mg/dl. As he had on going symptoms and ECG changes, we decided to proceed with coronary angiography & percutaneous coronary intervention. After informed consent patient was taken to cardiac catheterization laboratory.

Coronary angiography showed total occlusion of mid LAD in the previously stented segment with BMS and mild in-stent restenosis in LCx.

Left Main engaged with VL 3 guide. LAD wired with Cougar XT wire. Multiple aspiration done with Thrombuster II. Large thrombus burden persisted in mid LAD. Intracoronary abciximab was given. Residual thrombus persisted but TIMI III flow achieved. So we decided to stop here and kept the patient on i.v, abciximab infusion.

Relook angiography was performed after 48 hours which showed residual thrombus but less as compared to previous angiogram. Direct stenting of mid LAD (within stent) was done with Liberty (BMS) 4.5 x 16 mm. TIMI III flow achieved. Hospital course was uncomplicated and patient was discharged on the next day.

DISCUSSION

Stents have reduced rate of acute vessel closure and re-stenosis compared with conventional balloon angioplasty, but stent thrombosis (ST)is a serious complication of PCI.[1]

Stent thrombosis is an acute thrombotic occlusion of the stented segment in a coronary artery. It usually presents as ST-segment elevation myocardial infarction (STEMI). Very late stent thrombosis (VLST) has been reported with bare metal stent (BMS) implantation, although the annual incidence is much lower than that with drug-eluting stents. In a meta-analysisof RCTs, ST at 1 year was similar for DES andBMS.[4-6] Beyond 1 year, ST was greater with DES. In a meta-analysis of 13 RCTs restricted to primary percutaneous coronary intervention (PPCI), ST was observed with similar frequency upto 1-year follow-up

Figure 1

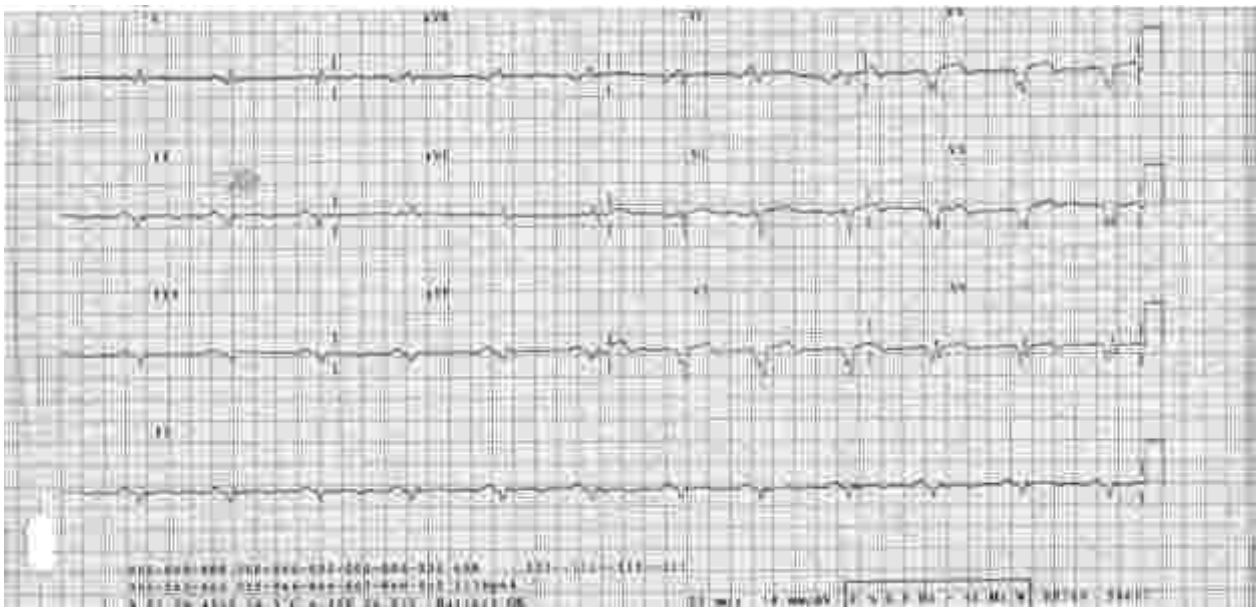
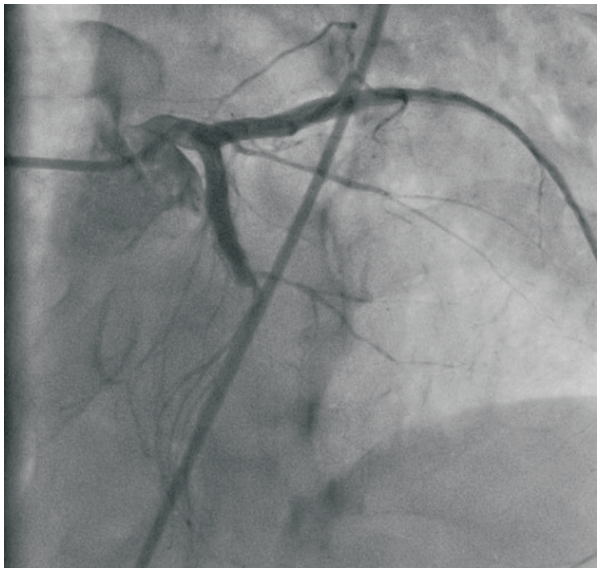


Figure 2



(2.7% DES, 2.6% BMS).[2] The incidence of definite very late ST after DES implantation appears to be approximately 0.4–0.6% per year. Very late stent thrombosis (VLST) in patients with BMS implantation is much lower (0.1% per year) than that after drug-eluting stent implantation.[3,7] Recently, a study is published which compared the risk of ST among BMS, First and Second generation DES.[8] The risk of very late stent thrombosis was higher with first degeneration DES (2.2%) as compared with BMS (1.5%).The risk of stent thrombosis was similar with both (DES & BMS) beyond 1 year.

Premature discontinuation of recommended dual antiplatelet therapy with aspirin plus a thienopyridine has been identified as a major risk factor for stent thrombosis.[1]

Figure 3

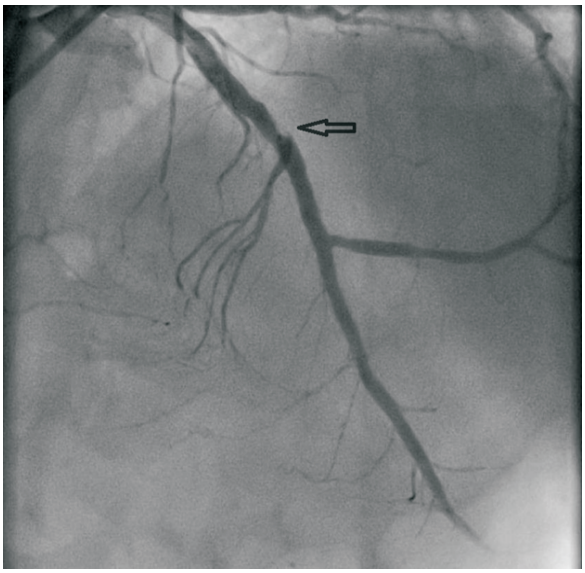
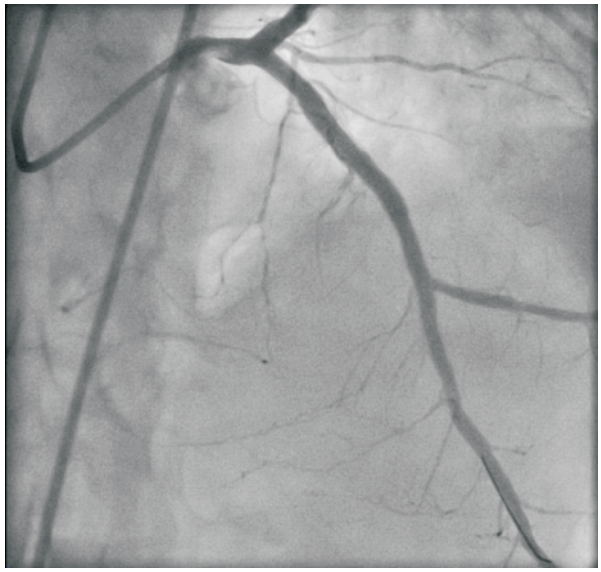


Figure 4



In addition to antiplatelet therapy discontinuation, many other risk factors for stent thrombosis have been identified.[2] Patients with acute coronary syndromes, renal failure, low ejection fraction, or diabetes mellitus are known to have increased risk for stent thrombosis. Procedurally incomplete stent expansion or apposition, smaller target vessels with smaller diameters, placement of multiple stents, stents placement over an existing thrombus or over existing stents. Finally, stent features, the polymer coating, strut thickness, and longer length are also associated with an increased risk of stent thrombosis.[1]

The mechanisms of VLST after BMS implantation is unknown and it could be different from DES. Disruption of in-stent neo-atherosclerosis has been implicated in the pathogenesis of VLST of BMS.

Nakazawa et al, reported that in-stent unstable neo-atherosclerosis, such as ruptured plaquesand thin-cap fibro-atheroma, was observed in BMS.[9]

CONCLUSION

Disruption of neo-atherosclerosis inside the stents could be an important underlying mechanism of VLST after BMS implantation. Recent guidelines from the American Heart Association, American College of Cardiology, and Society for Cardiovascular Angiography and Interventions recommends to continue dual antiplatelet therapy with aspirin indefinitely plus thienopyridine (clopidogrel) for at least one year, may be continued beyond one year if the risks of in-stent thrombosis is high and bleeding risk is low.

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