Very late stent thrombosis in a bare-metal stent, 9 years after implantation

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ABSTRACT

تعتبر التجلطات للدعامات الشريانية الغير مغلفة بالعقاقير من الحالات النادرة الحدوث بعد مرور عام من زراعتها. نستعرض في هذا التقرير حالة أُصيبت بتجلطات في هذا النوع من الدعامات بعد مرور تسعة أعوام على زراعتها في الشرايين التاجية للمريض، والذي لم يكن ملتزما بتناول العقاقير المضادة للصفائح (Aspirin) والتي عادة ما تحمي من هذا النوع من التجلطات. هنا تتضح أهمية الالتزام بهذه العقاقير حتى بعد مرور مدة طويلة من إجراء عملية زرع الدعامات.

Very late bare-metal stent thrombosis occurring beyond one year after stenting is uncommon. We report a case of bare-metal stent thrombosis presenting as acute myocardial infarction. The patient's stent was implanted 9 years ago, however, he was not adherent to his antiplatelet therapy (aspirin). This case highlights the importance of antiplatelet therapy in patients with stents even many years after implantation.

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Stent thrombosis (ST) is an uncommon complication that can happen after deploying the stent during the first few weeks. It is an acute thrombotic occlusion in the stented segment of a coronary artery, usually presenting as ST-segment elevation, such as myocardial infarction. It has been classified according to the Academic Research Consortium) as either early stent thrombosis (occurring within 30 days), or as late stent thrombosis (LST), (30 days to one year), or very late stent thrombosis (VLST), occurring beyond one year. The VLST is usually described and associated with the use of drug-eluting

stents.² Late stent thrombosis with bare-metal stents is very uncommon, therefore, we report a case of very late thrombosis of a bare-metal stent occurring more than 9 years after implantation. We present this case with VLST presented in Saudi Arabia, and to reemphasize the importance of anti-platelet therapy, even after a long time from implanting any stent.

Reports. A 73-year-old Saudi male, known to have diabetes mellitus, and chronic myeloid leukemia (CML) in remission, presented to the emergency room (ER) in December 2008 complaining of retrosternal chest pain, and shortness of breath for 6 hours. His medical history included percutaneous coronary intervention (PCI) in 1999 with 2 stents sized 3.0-18 mm and 2.5-16 mm in the proximal and mid segment of the left anterior descending artery (LAD). Simultaneously, he also had 2 stents in the right coronary artery (RCA). He was discharged on aspirin and was stated to be doing well until April 2004 when he presented with angina. The coronary angiogram (CA) was carried out and showed that the 2 stents in the LAD showed mild in-stent restenosis, and the RCA stents were widely patent with no signs of restenosis. The left circumflex showed a tight lesion proximally, a 3.0-18mm stent was implanted; all the stents he had were bare-metal stents. He was started on aspirin (ASA) and plavix for one year, and later continued on ASA. In March 2005 he was diagnosed to have CML, for which he responded very well to chemotherapy. When he was brought to the ER, he was in a state of remission. His complete blood count, including white blood cells, hemoglobin, and platelet counts were found to be within normal limits. His medication history revealed only iminitab, but he was not compliant to cardiac treatment including ASA. His 12 lead echocardiogram showed new left bundle branch block, with hypertented T wave anteriorly (Figure 1). The CA showed total thrombotic occlusion of the previously stented proximal LAD (Figures 2a & 2b). The circumflex, as well as the RCA stents showed mild in stent restenosis. We used a whisper wire and crossed the lesion easily. We

used a 2.0-20 mm voyager balloon at 8 atmospheres. The partial antegrade flow was established with some residual thrombus overlying the stented segment of the proximal LAD and in stent stenosis (Figures 3a & 3b). We deployed 2 stents (TAXUS) DES 3-28 mm and 2.5-16 mm at 14 and 16 atmospheres, then we post dilated with 3.0-15 mm (Maverick) high pressure balloon distally and a (Maverick) 4.0-11 mm proximally. We achieved an excellent angiographic result with thrombolysis in myocardial infarction trial

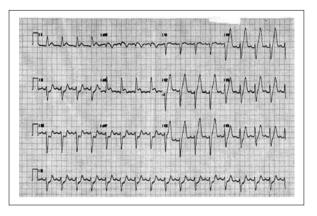


Figure 1 - A 12 lead ECG showed new left bundle branch block, and T-tented segment.

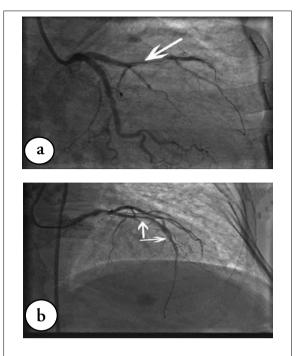


Figure 3 - Left coronary angiogram in a) right anterior oblique (RAO) caudal view and in RAO cranial b) showing residual thrombous and underlying stent stenosis.

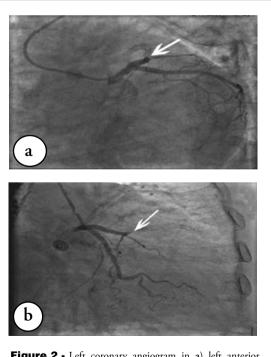


Figure 2 - Left coronary angiogram in a) left anterior oblique and right anterior oblique b) showing total thrombotic occlusion of the left anterior descending at the proximal edge of the previous stent.

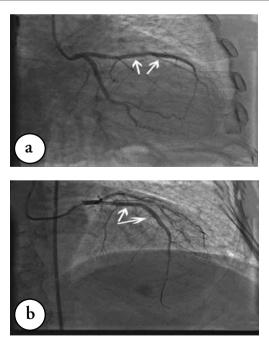


Figure 4 - Left coronary angiogram in a) right anterior oblique (RAO) caudal and in RAO cranial b) showing thrombolysis in myocardial infarction trial 3 flow in the left anterior descending and good angiographic result.

(TIMI) 3 flow in the LAD (Figures 4a & 4b). He was admitted to the coronary care unit on IV Abcixmab, where he was doing well after the procedure and during his hospital stay. He was discharged 3 days later in a good condition on dual anti-platelets, ASA, and Plavix for life, as he was high risk to have stent thrombosis, especially with the diethylstilbestrol stent. We explained the dangerous consequences of stopping his medication before discharge.

Discussion. Since the advent of coronary stenting has started, the practice of interventional cardiology has dramatically improved following the acute procedural success and has also reduced the restenosis rates observed with balloon angioplasty alone.3 Although the great achievement in PCI procedures, stent thrombosis, which is associated with 20-40% mortality, became the new concern of PCI procedures, occurring in up to 4.7%.4 Fortunately, stent thrombosis has been overcome using high-pressure inflation and dual anti-platelet therapy.⁵ It has been demonstrated that the aggressive use of antiplatelet agents such as aspirin and clopidogrel decreased the incidence of early and late stent thrombosis to less than 1%.5,6 Stent thrombosis in bare-metal stents occurs mainly within 30 days (early).7 The VLST is increasingly being recognized as a complication of

Table 1 • Previous studies for very late stent thrombosis (ASA - aspirin).

Authors	Year	Number of cases	Duration to the thrombotic event	Antiplatelet
Sarkees ML et al ¹²	2009	1	13 year	ASA discontinuation and in stent stenosis
Katayama T et al ¹³	2007	1	10.5 years	On ASA
Lemesle G et al ¹⁴	2008	2 case	8 years 6 years	On ASA On ASA+flavix but evidence of stenosis
Finechi M et al ¹⁵	2008	1	7 years	/
Ramos AR et al ¹⁶	2007	2	5 years 3 years	On ASA+flavix On ASA
Celik T et al ¹⁷	2005	1	3 years	On ASA +flavix
Hayashi T et al ¹⁸	2004	2	31 months 27 months	No antiplatelets On ASA
Saito T et al ¹⁹	2007	1	29 months	On ASA
Manjappa N et al ²⁰	2006	1	2 years	On ASA
Wenaweser P et al ²¹	2005	2	21 months 20 months	/
Ishikawa T et al ²²	2004	1	19 months	/
Celik T et al ²³	2005	1	16 months	On ASA stent under expansion
Mauri L et al ²⁴	2007	8	/	/

drug-eluting stents, where it may be related to delayed endothelialization,1 and rarely occurs with the use of bare-metal stents outside the context of brachytherapy.8 Although millions of bare-mental states have been implanted since it was invented, we could only find 26 cases of late stent thrombosis occurring with the use of bare-metal stents published in the literature (Table 1). The duration between the initial stent placement and the subsequent thrombotic event ranged from one and a half years to less than 13 years. Only 5 cases had the event after more than 5 years, and in all cases there was no clear cause, and no clear relation to antiplatelet compliance, except in one case. In our patient, the time to the acute thrombotic event from the PCI was 9 years. We could not identify any potential explanation for this acute event except for the possibility that he had only been intermittently compliant with aspirin. In addition, there was evidence of significant in stent stenosis.

The presumed causes of bare-metal stent thrombosis, both early and late, include noncompliance with antiplatelet agents, and in stent restenosis, chronic inflammation and neointima, and atherosclerosis that lead to clot formation, and exercise-induced procoagulant state, small stent size, and under deployment of the stent. Impaired response to antiplatelet therapy has also been reported. Since there is no published literature on the specific causes of VLST, it is conceivable and probable that many mechanisms that led to early ST and LST may also be responsible for VLST. The role of intravascular ultrasound (IVUS) in investigating the possible pathophysiological mechanisms is very important and cannot be underestimated.

There is no specific management of VLST, as with any thrombotic lesion the restoration of perfusion to TIMI grade 3, most commonly by a variety of percutaneous techniques such as balloon angioplasty, thrombectomy, or re-stenting, depends on the underlying presumed cause, with subsequent long-term (if not lifelong) dual antiplatelet therapy. However, these patients are at higher risk for recurrent thrombosis

In conclusion, the VLST with the use of a bare-metal stent is a rare, but catastrophic event. The mechanism is not fully understood, and is likely to be different from that of drug eluting stents. The optimum size of the stent and adequate implantation with high pressure during deployment, full expansion with good post-dilatation under the guidance of IVUS after stent implantation, and the importance of compliance to dual antiplatelets for longer duration are the factors to be taken into consideration. The duration of antiplatelet therapy for patients with coronary artery stents remains to be determined, which is a key factor to avoid this serious complication.

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