Very late thrombosis of bare-metal stent: Apropos of a case

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ABSTRACT
The very late thrombosis of a conventional stent is an uncommon event, although its consequences can be catastrophic for the patient, being associated with an increase in the long-term mortality rate. Here is exposed the case of a 60-year-old man who presented an acute inferior myocardial infarction due to definitive thrombosis of the conventional stent, after 24 months of implantation.

Key words: Bare metal stent, Thrombosis, Acute myocardial infarction, Percutaneous coronary intervention

INTRODUCTION
With the introduction of coronary stents in the therapeutic arsenal for percutaneous coronary intervention there was a significant reduction in the occurrence of acute occlusions (associated with coronary dissection, intracoronary thrombus and elastic recoil), and the incidence of restenosis (associated with negative arterial remodeling and neointimal hyperplasia), characteristically associated with the use of balloons.

However, despite coronary stents have substantial advantages over the use of balloon angioplasty, stent thrombosis is a serious problem that may manifest as myocardial infarction and sudden death1.
The arrival of drug-eluting stents (DES) was thought to wipe out this complication but only 5 years after the first study supporting the use of these devices and their spectacular results, several meta-analyses seemed to indicate an increase in long-term mortality. The culprit?... very late stent thrombosis, which until then, had been underemphasized in case reports. Hence, stent thrombosis would become the great threat after DES implantation. But this phenomenon is not exclusive to DES since, although very rare, late stent thrombosis has also been described in conventional bare metal stents (BMS).

We present the case of a patient with very late thrombosis 24 months after BMS placement.

**CASE REPORT**

A 60-year-old man with a history of essential hypertension had a BMS Apolo Small 3.0 x 18 mm stent for severely occluded proximal right coronary artery in the context of acute inferior myocardial infarction in January 2015. He was on dual antiplatelet therapy for a month and continued taking oral aspirin.

After percutaneous coronary intervention, the patient was enrolled in a cardiac rehabilitation program until reentering the workforce. He remained asymptomatic until January 2017 when he was admitted in his health area due to oppressive, stabbing chest pain of more than 30 minutes with irradiation to the neck, jaw and left arm, associated with profuse sweating and nausea. A 12-lead electrocardiogram on presentation showed ST segment elevation ≥ 2 mm in DII, DIII, aVF, and V3 to V6, with ST segment depression in DI and aVL (Figure 1), consistent with acute inferior wall myocardial infarction. He was urgently commenced on fibrinolytic treatment with recombinant streptokinase with the recommended dose, but either alleviation of symptoms was achieved, nor regression to normal electrocardiographic pattern. So we managed his transfer for

![Figure 1. 12-lead electrocardiogram at admission, showing ST-segment elevation in DII, DIII, aVF, V3-V6.](image)
urgent coronary angiography, demonstrating acute occlusion of the right coronary artery with evidence of a thrombus at the site of stenting (Figure 2A). Percutaneous coronary intervention was performed and another 3.0 × 23 mm bare metal stent was implanted and good angiographic result was achieved (Figure 2B y C).

The patient made an uneventful recovery and was discharged without any symptoms. Thereafter, he has remained asymptomatic.

**COMMENT**

Stent thrombosis is unusual (<1%), but with high morbidity and mortality (20-40%)⁴. With BMS, most thromboses usually occur in the first days after implantation. It is an exceptional complication after the first month. Hence, dual antiplatelet therapy is prescribed during this period⁵.

However, the identification of new late thrombotic events (beyond the traditional 30 days) and the potential risk of increased frequency of this complication over time, turned this phenomenon—which until then received scarce attention in case reports—into a latent threat⁵, raising great concern and controversy in the cardiological community.

Since the beginning, stent thrombosis was classified according to angiographic, clinical and time parameters. Although these classifications were extensive and did not fully encompass the expectations to detect the true dimension of the problem. Therefore, new standardised definitions of stent thrombosis were obtained, which included the concepts of definite, probable and possible thrombosis. Similarly, the temporal classification was modified to adjust to the new thrombosis tendency, and the «very late» definition was suggested when it occurred after one year of stent implantation¹⁻³,⁶.

Its mechanisms are little understood, but several factors are known to stimulate it (Box). Moreover, it has been suggested that stent thrombogenicity depends largely on the electropositive charge of its metallic coating, which seems to be modified by its composition, configuration and size, and consequently the protective «defense» response of the vessel; in addition to the presence of late-onset atheromatous plaques in the stent (neoatherosclerosis)⁷⁻⁹.

On this point, we should comment that, although DES, compared to BMS have notable advantages in relation to the appearance of thrombosis, are equally susceptible to the multifactorial cause. However, the association of new related factors is possible, considering the complexity of these devices: metallic platform (material, geometry, strut thickness), polymer (composition, disposal, thickness, biocompatibility, thrombogenicity, pro-inflammatory potential, biodegradability or lack of polymers) and anti-proliferative drug (molecular composition, biological actions, doses, releasing kinetics)¹⁻³.

The treatment of this major adverse coronary event consists of immediate restoration of artery blood flow after the occurrence of the event. Most of the time it is successfully treated with mechanical thrombectomy, balloon angioplasty or implantation of another stent (BMS or DES). Coronary artery bypass grafting should be considered when patients do

**Figure 2.** A. 30º left anterior oblique angiographic view showing total occlusion of proximal right coronary artery. B. Stent implantation. C. Angiographic result.
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not benefit from the previous options\textsuperscript{1,3,10}.

Very late stent thrombosis is extremely rare. Hence, it requires a closer interaction with the patient so that the risk of occurrence can be identified and stratified individually.

REFERENCES


\textbf{Box.} Predisposing factors to stent thrombosis.

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\textbf{Related to patient} \\
- Diabetes mellitus \\
- Kidney disease \\
- Severe left ventricular dysfunction \\
- Poor adherence and resistance to antiplatelet therapy \\
- Heparin-induced thrombocytopenia \\
\hline
\textbf{Related to procedure} \\
- Implantation of multiple stents \\
- Persistent dissections \\
- Residual thrombi \\
- Stent malapposition \\
- Long lesions and long stents \\
- Small vessels \\
- Complex lesions \\
- Slow coronary flow pattern \\
\hline
\textbf{Related to stent and intimal endothelium} \\
- Incomplete endothelization \\
- Delayed hypersensitivity \\
- Physical properties of the stent \\
- Endothelial dysfunction \\
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