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Very late stent thrombosis of a paclitaxel-eluting stent after left main coronary artery stenting

Summary

We report the case of a 60-year-old male who was treated by implantation of one drug-eluting stent in the distal left main coronary artery and the ostial left anterior descending artery for an acute anterior ST-elevation myocardial infarction and who developed stent thrombosis and cardiogenic shock seven-hundred thirty days after stent implantation by complete occlusion of the distal left main coronary stem.

This case report highlights that very late stent thrombosis (>1 year) may occur with drug-eluting stents, which requires careful clinical follow-up and particularly so with stents implanted into the left main coronary artery.

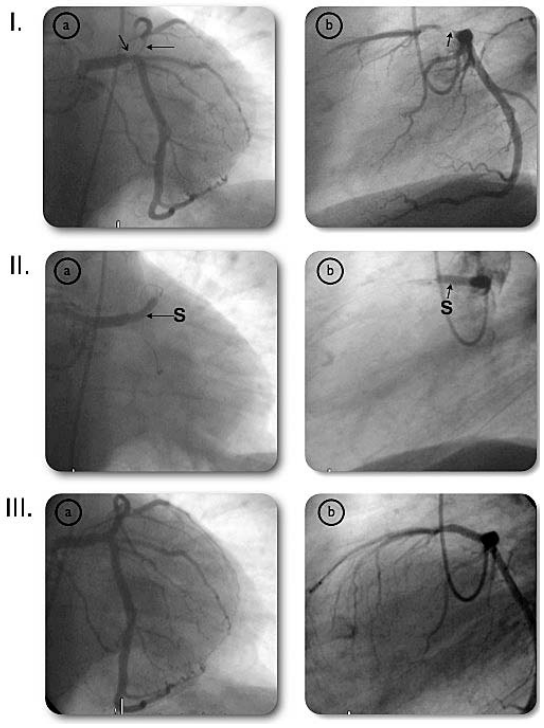

Figure 1
Panel I: Coronary angiography (a: LAO cranial, b: LAO 90°) revealing a thrombotic lesion on the ostium of the left anterior descending artery (arrow) with involvement of the distal left main coronary artery (light arrow). Panel II: Coronary angiography during percutaneous coronary intervention. The 3.5/16 mm paclitaxel-eluting stent (S, Taxus®, Boston Scientific) efficiently covered the distal left main coronary lesion, leading to temporarily left main occlusion (contrast in the left main coronary artery). Panel III: End result after percutaneous coronary intervention.

Case report

A 60-year-old white male was admitted with acute anterior ST-elevation myocardial infarc-

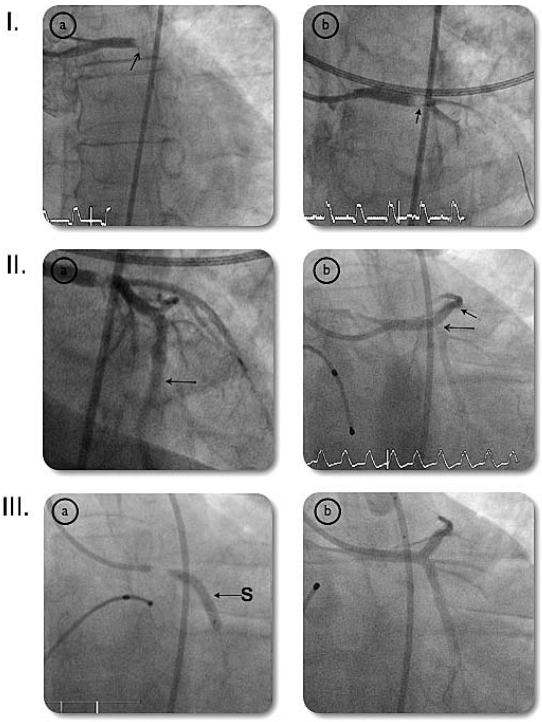

Figure 2
Panel I: Coronary angiography (a & b: anteroposterior view) revealing a total occlusion of the distal left main coronary artery due to stent thrombosis (b: arrow shows a large thrombus). Panel II: Coronary angiography during percutaneous coronary intervention (a: anteroposterior view, b: LAO cranial) with restoration of TIMI 2 flow in the LAD after to be restored (b: arrow) and demonstrating resting high-grade stenosis of the ostium of RCX (b: light arrow) Panel III: a: Implantation of a 3.5/10 mm titanox-eluting stent (S, Titan-2®, Hexacath). b: End result after percutaneous coronary intervention.

There is no conflict of interest.

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tion. He complained of typical chest pain two hours before admission and his ECG displayed 3 mm ST-elevation from V_1 to V_5. His only known cardiovascular risk factor was smoking cigarettes (40 packs/year). He underwent emergency coronary angiography (fig. 1-Ia: LAO cranial, fig.1-Ib: LAO 90°), which revealed a thrombotic lesion on the ostium of the left anterior descending artery (LAD, arrow) with involvement of the distal left main coronary artery (light arrow), and percutaneous coronary intervention was undertaken. The lesion was treated by dilatation with Maverick 3.5/20 mm balloon and stent implantation using a 3.5/16 mm paclitaxel-eluting stent (Taxus®, Boston Scientific), which efficiently covered the distal left main coronary lesion, as demonstrated by temporarily left main occlusion (fig. 1-II). Because the result was satisfactory (fig. 1-III), no further intervention was performed (dilatation of the circumflex ostium or final kissing balloon). The recovery was uneventful and the patient was discharged after 3 days. Aspirin® was prescribed for the remainder of his life and clopidogrel was prescribed for 12 months.

Seven-hundred thirty days after stent implantation, the patient complained of acute chest pain. During his transport to the cathlab, the patient presented pulseless electrical activity (PEA) requiring chest compressions, endotracheal intubation and administration of pressors. The coronary angiography (fig. 2) was repeated and revealed a total occlusion of the distal left main coronary artery due to stent thrombosis (fig. 2-I, the arrow shows a large thrombus). The patient underwent percutaneous coronary intervention under chest compressions, which allowed a TIMI-2 flow in the LAD to be restored (fig. 2-II, arrow). A resting high-grade stenosis of ostium of RCX (fig. 2-II, light arrow) was finally treated with implantation of a 3.5/10 mm titanox-eluting stent (Titan-2®, Hexacath). Although the result was satisfactory with restoration of efficient blood flow in the left coronary tree, the left ventricular ejection fraction remained severely depressed and the patient died in cardiogenic shock. The family refused autopsy.

Discussion

Drug-eluting stents have been shown to effectively reduce neointimal hyperplasia, which make them a competitive alternative to coronary artery bypass surgery for patients suffering from left main coronary artery disease.

There is, however, a growing body of concerns about late-occurring stent thrombosis in the current drug-eluting stent era [1]. When very late stent thrombosis (ST >1 year after implantation) remains the exception with bare-metal stent (2% of all stent thrombosis) [2], about one third of drug-eluting stent – ST appear very late [1]. Very late ST is most likely a multifactorial process related to disturbed vasomotion, incomplete reendothelialisation,chronic inflammation with positive vessel remodeling, incomplete stent apposition and / or insufficient inhibition of platelet aggregation. Accordingly, recent investigations have reported variables consistently associated with ST following drug-eluting stent implantation, such as discontinuation of antiplatelet therapy, brachytherapy, bifurcation stenting, acute coronary syndromes, renal insufficiency, current smoking, left main coronary PCI and diabetes [3, 4]. Furthermore, incomplete stent apposition and stent underexpansion have been identified in IVUS studies [5, 6], and hypersensitivity reactions and localised inflammation have been recognised in autopsy proven ST cases [7, 8].

Whereas the exact causes leading to stent thrombosis in this particular case will stay unanswered (lack of final kissing balloon, stent underexpansion or malapposition, hypersensitivity, aspirin® resistance?), this current example highlights however that late stent thrombosis may occur with drug-eluting stents, which requires careful clinical follow-up and particularly so with stents implanted into the left main coronary artery.

References

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