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Percutaneous Coronary Intervention of Left Main Stenosis in the Era of Drug-eluting Stents

A Case Report of Stent Thrombosis 3 Years Post Implantation

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Summary

Stent thrombosis remains the Achilles heel of drug-eluting stents. Although rare, stent thrombosis is a potentially fatal complication of coronary stenting necessitating dual antiplatelet therapy. This case of very late stent thrombosis highlights some of the problems that remain unsolved in coronary stenting. Furthermore, it reminds us that the risk of stent thrombosis persists even years after successful stenting.

Introduction

Until recently, coronary artery bypass grafting (CABG) has been the recommended treatment of left main (LM) stenosis and 3-vessel coronary artery disease (3-VD). However, owing to the continuous emergence of technical refinements and operators gaining expertise, percutaneous coronary intervention (PCI) with coronary stenting has been suggested as a potential alternative to CABG.

Antiproliferative drug-eluting stents (DES) have proved superior to bare-metal stents (BMS) [1] as well as conventional PCI in reducing the rate of in-stent restenosis. Interim results of the SYNTAX (Synergy Between PCI and CABG) trial thus indicate equal rates of hard endpoints in LM and 3-VD patients treated with either CABG or PCI with DES implantation, but a higher rate of revascularization in the latter [2].

Unfortunately, coronary stenting holds the risk of stent thrombosis (ST). It has been suggested that DES may be excessively susceptible to late (> 6 months) and very late (> 12 months) ST. In the era of DES, the frequency of ST is 1.8–2.7 % [3, 4] within 6 to 15 months of follow-up despite dual antiplatelet therapy.

ST is a rare, but potentially fatal complication of coronary stenting and has emerged as an issue of major concern. The following case report describes very late ST of the left anterior descending coronary artery (LAD) in a patient who underwent DES stenting.

Case Report

A 58-year-old woman (51 kg, 164 cm) was admitted to a regional hospital presenting with a 3-week history of increasing chest pain. She was predisposed to cardiovascular disease by hypertension, hypercholesterolaemia, previous smoking and a family history of ischaemic heart disease.

Electrocardiographic and biochemical examination demonstrated myocardial ischaemia consistent with the diagnosis of non-ST-segment elevation myocardial infarction (NSTEMI), and treatment with aspirin, clopidogrel and low-molecular-weight heparin was initiated. However, due to persistent chest pain despite adjunctive nitroglycerine infusion, the patient was transferred for acute coronary angiography (CAG) at an invasive cardiology center.

CAG performed from the femoral artery revealed a 90 % distal LM stenosis (Fig. 1A) involving the bifurcation of the circumflex and the left anterior descending coronary artery as well as a 60 % stenosis of the right coronary artery. Echocardiography showed ventricular hypertrophy, yet left ventricular ejection fraction was normal (60 %). The patient was offered participation in the ongoing SYNTAX trial. She refused, though, preferring PCI rather than randomization.

PCI was performed, and sirolimus stents (Cypher®) were deployed under intravascular ultrasound (IVUS) guidance. The patient underwent LM bifurcation stenting with 3 stents (crush technique) and right coronary stenting with a single stent. Subsequent angiographic results were excellent (Fig. 1B). Antithrombotic therapy consisting of unfractionated heparin, aspirin and a GPIIb/IIIa inhibitor (abciximab) was initiated in the catheterization laboratory.

One hour after leaving the catheterization laboratory, the patient developed severe hypotension. Acute echocardiography showed no pericardial effusion, and subsequent acute CAG showed no signs of acute stent occlusion or coronary artery rupture. Nevertheless, blood tests revealed a considerable drop in haemoglobin level (from 7.5 to 5.1 mM) suggesting an internal haemorrhage of non-cardiac origin. The patient received intravenous fluid and 4 portions of blood and was transferred to the intensive care unit. Acute abdominal ultrasonography identified a 10 × 10 cm retroperitoneal haematoma. No intervention was instituted, except discontinuation of abciximab, and the bleeding stopped spontaneously.

The patient was discharged on lifelong aspirin treatment and clopidogrel for 12 months. Six months later, a re-angiography was performed; still, the results were excellent.

In August 2008, after nearly 3 years without any cardiac symptoms despite daily physical activity, the patient was acutely admitted to a regional hospital with sudden onset of severe chest pain. An ECG revealed ST-segment elevation in

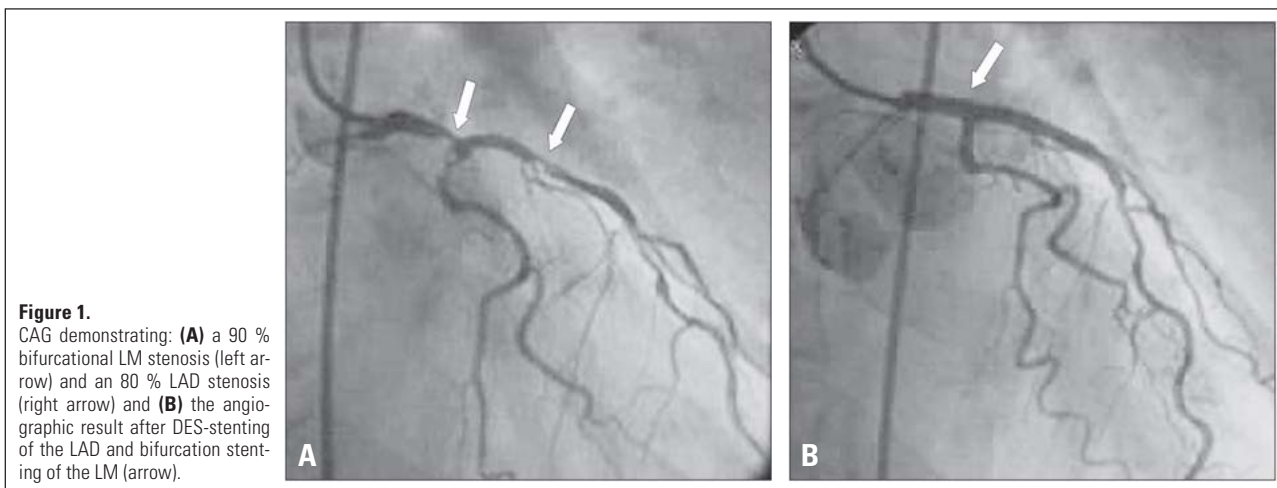


Figure 1. CAG demonstrating: (A) a 90 % bifurcational LM stenosis (left arrow) and an 80 % LAD stenosis (right arrow) and (B) the angiographic result after DES-stenting of the LAD and bifurcation stenting of the LM (arrow).

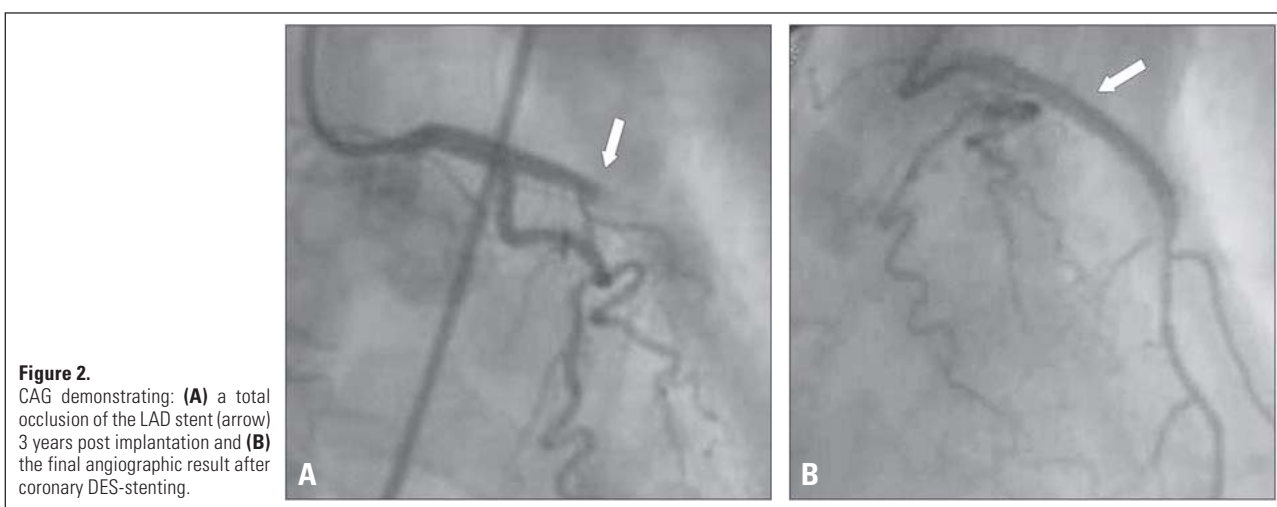


Figure 2. CAG demonstrating: (A) a total occlusion of the LAD stent (arrow) 3 years post implantation and (B) the final angiographic result after coronary DES-stenting.

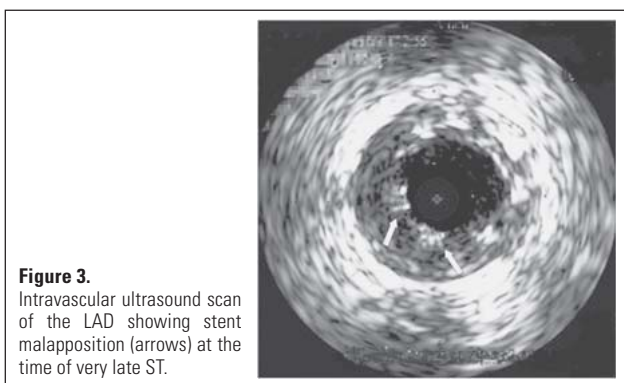


Figure 3. Intravascular ultrasound scan of the LAD showing stent malapposition (arrows) at the time of very late ST.

leads V1–V5 as well as in I and II consistent with the diagnosis of anterior STEMI. Haemodynamics were stable. Clopidogrel 600 mg and unfractionated heparin 10,000 IU were administered pre-hospitally, and the patient was transferred for primary PCI (transfer time 90 minutes).

CAG demonstrated total occlusion of the distal part of the LAD stent (Fig. 2A). Abciximab was administered and thrombus aspiration was attempted without success. Balloon angioplasty was performed, but due to distal embolization only TIMI 1 flow was present. IVUS suggested malapposition of the LAD stent, perhaps due to intravessel oedema, incomplete expansion or negative remodelling (Fig. 3). Two additional

sirolimus stents were implanted at the site of the stenosis. IVUS now showed optimal stent expansion, and TIMI 3 flow was present at the end of the procedure (Fig. 2B). Echocardiography revealed severe hypokinesia of the anterior wall and septum. Ejection fraction was 25–30 %.

The patient was stabilized on diuretics, an ACE-inhibitor and a beta-blocker, and was transferred to the regional hospital after a few days. At 1-month follow-up, ejection fraction had increased to 30–35 % and the patient was in NYHA-II class.

Discussion

The present case report illustrates that the use of DES in the treatment of complex LM stenoses is encumbered with potential drawbacks. The patient suffered very late ST 3 years after angiographically successful stenting and developed compromised cardiac function. At the time of ST, IVUS suggested malapposition of the LAD stent, which might have formed a nidus for the development of ST.

During the initial PCI procedure, a severe haemorrhagic incident occurred. This stresses the risk of using the femoral approach in combination with triple antiplatelet therapy and anticoagulation for complex PCI-procedures, where vigorous antithrombotic therapy is required.

In this particular case the PCI procedure of ST was complicated by distal embolization, which may have contributed to the resultant poor ventricular function. The use of thrombus aspiration and in some cases distal protection devices might be an option when treating ST.

A potential drawback of DES is the suggested propensity of these stents to cause late and very late ST, which is thought to result primarily from delayed re-endothelialization. Thus, the risk of ST may be reduced by intensified antiplatelet therapy. We suggest a thorough assessment of the regime for anti-thrombotic treatment of complex LM stenoses and 3-VD treated with PCI. In some cases, prolonging the dual antiplatelet therapy beyond current recommendations may be beneficial. However, the potential benefit might be outweighed by an increase in major bleedings.

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