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Case Report

Post-operative simultaneous very late two-vessel drug eluting stent thrombosis with sparing of bare metal stent in a Jehovah's witness after clopidogrel withdrawal

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ABSTRACT

Very late stent thrombosis is a feared complication after drug-eluting stent (DES) implantation. Several factors related to the patient, generation and type of the deployed stent, procedure, and premature antiplatelet withdrawal are known to contribute to this complication. Herein, we describe a case of a Jehovah's witness patient who developed simultaneous two-vessel 1st generation DES thrombosis 5.4 and 3.5 years after deployment [with sparing of the bare metal stent (BMS)] in the immediate post-operative period secondary to clopidogrel withdrawal. The case was complicated by ST-segment elevation myocardial infarction, cardiogenic shock, and a ventricular fibrillation cardiac arrest requiring urgent percutaneous coronary intervention. The acute thrombosis of DESs with sparing of the BMS exemplify how they are more prone to this complication due to delayed endothelialization of stent struts and neoinitimal coverage.

<**Learning objective:** Extreme caution should be excercised when attempting to withdraw antiplatelets immediately before surgery in patients with drug eluting stents even if the recomended 12-month period of dual antiplatelet therapy has elapsed because of the risk of very late stent thrombosis.>

Introduction

Despite the prominent success of drug-eluting stents (DESs) over bare metal stents (BMS) in suppressing neointimal proliferation and reducing in-stent restenosis, recent concerns have risen because of the higher risk of late (31 days–1 year) and very late (after 1 year) DES thrombosis. The risk of stent thrombosis increases in the perioperative period secondary to catecholamine production promoting vasospasm, increased platelet activation, and decreased fibrinolysis. Moreover, preoperative interruption of antiplatelet therapy to minimize blood loss can trigger a rebound increase in platelet activation further intensifying the chance of stent thrombosis. The American College of Cardiology (ACC)/American Heart Association (AHA) recommends continuing dual antiplatelet therapy (DAPT) for 12 months after DES deployment because of lack of evidence for protection beyond that period in randomized trials and this recommendation has aptly guided percutaneous coronary intervention (PCI) care over the past decade. The perioperative period is a unique state with lack of clear-cut recommendations for patients with DES deployed >12 months who are still on antiplatelet therapy and anticipating surgery. In this instance, the choice to withhold antiplatelet therapy preoperatively to minimize blood loss or continue them to avoid cardiovascular complications is challenging. The presented case draws attention to the entity of very late DES thrombosis in the postoperative period and invites further research for ideal perioperative handling of antiplatelet therapy.

Case report

A 54-year-old man with a past medical history of hypertension, dyslipidemia, paroxysmal atrial fibrillation, obstructive sleep apnea, and coronary artery disease presented to the hospital for elective bilateral knee replacement. He had 3 previous stents

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deployed for recurrent anginal chest pains: in February 2003, a BMS 2.75/12 mm Express II (Boston Scientific, Natick, MA, USA) for a 90% mid left anterior descending (LAD) lesion; in October 2003, a sirolimus-eluting stent (SES) 3.5/13 mm Cypher (Cordis Corporation, Johnson & Johnson, Warren, NJ, USA) for a 90% mid circumflex (LCX) lesion; and in September 2005, a paclitaxel-eluting stent (PES) 3/8 mm Taxus (Boston Scientific) for an 80% proximal LAD lesion. His last coronary angiography in July 2007 revealed no occlusive disease. He denied any exertional chest pain or shortness of breath and was compliant with his medications including warfarin (for atrial fibrillation), clopidogrel (sole antiplatelet agent), sotalol, and lisinopril. He was a Jehovah's witness (JW) who would not receive any blood products. Given this cardiac history, he was sent to his cardiologist for preoperative clearance. To minimize blood loss, it was decided to stop clopidogrel five days prior to surgery and to stop the warfarin and start low molecular weight heparin bridging to be stopped 12 h prior to surgery and to continue perioperative beta-blockade.

The patient's surgery was scheduled on March 6th 2009 (73 months after mid LAD stent, 65 months after LCX stent, and 42 months after proximal LAD stent). Following surgery and in the post anesthesia care unit, he started experiencing chest pain. Electrocardiogram revealed ST-segment elevation in leads V5 and V6 with ST-segment depression in leads V1–V3 conforming to a lateral wall ST-segment elevation myocardial infarction (STEMI) (Fig. 1). Shortly thereafter, he became unresponsive due to ventricular fibrillation cardiac arrest requiring repeated defibrillation (Fig. 2). Following restoration of spontaneous circulation, dopamine and norepinephrine were initiated for cardiogenic shock and the trachea was intubated and the patient was mechanically ventilated and transferred to the catheterization suite. Coronary angiography revealed total LCX occlusion with a thrombus at the site of the previous DES and a 90% stent thrombosis in the proximal LAD DES with sparing of the mid LAD BMS which showed only in-stent restenosis (Fig. 3). AngioJet thrombectomy (Medrad Inc., Warrendale, PA, USA) was used to debulk the thrombus load in the LCX. There was still significant thrombus burden, so dilatation of the LCX stent was performed using a Quantum Maverick 3/15 mm balloon (Boston Scientific) at 9 atmospheres. A Driver 3.5/18 mm BMS (Medtronic, Santa Rosa, CA, USA) was then successfully deployed followed by thrombolysis in myocardial infarction (TIMI) 3 flow. The same Maverick was used to dilate the thrombus laden stent in the proximal LAD; post-percutaneous transluminal coronary angioplasty (PTCA) there was a TIMI 3 flow. The patient experienced multiple episodes of ventricular fibrillation during the procedure requiring cardiopulmonary resuscitation and multiple electrical defibrillations. He was given clopidogrel 600 mg, aspirin 300 mg, bivalirudin, and an intra-aortic balloon pump was placed to augment coronary perfusion. The patient had a peak troponin level of 600 μg/L (this significant leak is likely related to repeated defibrillation) and a left ventricular ejection fraction of 20% prior to discharge. His condition gradually improved and he was discharged when stable two weeks after surgery.

Discussion

Despite the distinct success of DES compared to BMS in suppressing neointimal proliferation and reducing rates of in-stent restenosis, the significantly higher risk of very late DES thrombosis remains a major concern [1]. The various proposed mechanisms of very late DES thrombosis include delayed endothelialization and neoinitimal coverage, intrastent hypersensitivity reaction to polymers [2], stent fracture [3], and late stent malapposition [4]. Incomplete stent apposition is highly prevalent in patients with very late DES thrombosis compared with respective controls (77% vs. 12%; $p < 0.001$), suggesting a role in the pathogenesis of this adverse event [5]. Imai and colleagues demonstrated that peri-stent contrast staining (PSS) (defined as an abnormal angiographic findings at sites of DES implantation, suggesting contrast staining outside the stent struts) found within 12 months after SES implantation was associated with very late stent thrombosis (8.2% in PSS vs. 0.2% in non-PSS) [6]. In an in vivo case-controlled study by Guagliumi and colleagues, the presence of positive vessel remodeling as imaged by intravascular ultrasound was associated with late stent thrombosis after DES implantation ($p = 0.003$) [7]. Cessation of DAPT remains the single most important predictor of stent thrombosis [8] and can trigger a rebound increase in platelet activation [9,10]. This is especially concerning in the perioperative period due to the accompanying increased catecholamine

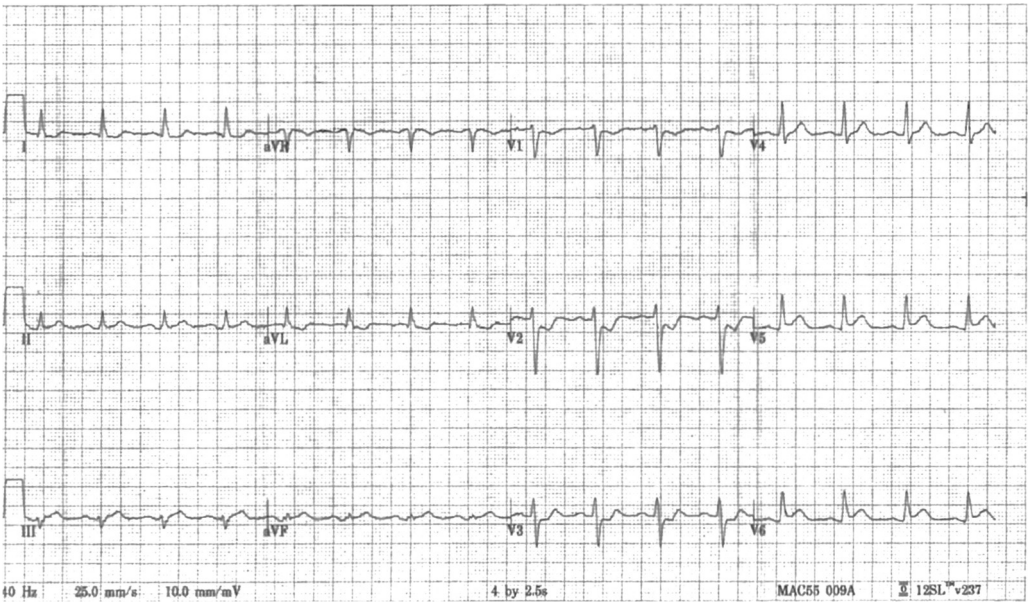

Fig. 1. Electrocardiogram during chest pain revealing ST-segment elevation in leads V5, V6 with reciprocal ST-segment depression in V1–V3 suggestive of a lateral ST-elevation myocardial infarction. Notice also the subtle ST-segment elevation in the inferior leads.