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Special article

A tale of two stents: perioperative management of patients with drug-eluting coronary stents

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Abstract Drug-eluting stents were introduced into clinical practice to decrease coronary stent restenosis rates. Though remarkably effective in reducing this complication, recent data reveal that drug-eluting stents pose a significant risk for late stent thrombosis, an event strongly correlated with discontinuation of anti-platelet therapy. Because anti-platelet agents are often discontinued perioperatively, patients with DES are at risk for perioperative stent thrombosis and myocardial infarction. Along with a review of the recent literature, we present two cases of patients with drug-eluting stents scheduled for renal transplantation. Two distinct antithrombotic management strategies illustrate the risk of either approach—bleeding and transfusion versus stent thrombosis and myocardial infarction.

1. Introduction

Drug-eluting coronary stents (DES), introduced into clinical practice in the United States in 2003, accounted for more than 8 of 10 coronary stents[1,2] [1] placed at the beginning of 2006. With nearly 6 million stents in place worldwide, DES represent an important advance in percutaneous coronary intervention (PCI) because they significantly reduce arterial restenosis rates and the subsequent need for repeat coronary intervention[1,2] [2-9]. However, as DES reduce the risk of restenosis, the concomitant delay in stent endothelialization after deployment significantly increases the risk of stent thrombosis for months or even years after deployment. In the last year, data suggesting that DES pose a significant risk for late stent thrombosis have continued to mount. Multiple case reports and observational studies describe late thrombosis of DES as an event associated with high mortality [10-15]. Ironically, many of these patients discontinued antiplatelet therapy based on medical advice in anticipation of elective noncardiac surgery [10-13].

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[2] Cordis Corp (a Johnson & Johnson Company), Miami Lakes, FL. Information available at cordis.com and personal communication (DEH, July 2006).

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In the two case reports presented, the challenges of perioperative management of such patients with DES are presented. The two distinct antithrombotic management strategies demonstrate the risks of either approach—bleeding and transfusion versus stent thrombosis and myocardial infarction (MI).

2. Case reports

2.1. Case 1

A 56-year-old man with end-stage renal disease secondary to type II diabetes mellitus and known coronary artery disease (CAD), presented for a living-related renal transplant. At the time of an MI suffered 7 years earlier, multivessel CAD was treated with balloon angioplasty and a bare metal stent (BMS) in the left anterior descending (LAD) artery. Eight months before scheduled renal transplantation, repeat cardiac catheterization showed a patent LAD BMS, total occlusion of the midcircumflex artery, an 85% stenosis of the obtuse marginal artery, and a complex lesion at the bifurcation of the right coronary artery (RCA). Two paclitaxel DES were placed in the RCA, and a paclitaxel DES was placed in the obtuse marginal artery lesion. The patient continued aspirin therapy and was started on clopidogrel 75 mg daily. Two weeks before his scheduled surgery, repeat coronary angiography showed a patent LAD BMS, a 40% occlusion within the obtuse marginal artery DES (in-stent restenosis), and a widely patent DES within the RCA. Because the patient was asymptomatic and reported moderate activity (biking 15 min/day) without shortness of breath or chest pain, no further coronary interventions were deemed necessary.

Preoperative medications included furosemide, metoprolol, olmesartan, rosuvastatin, insulin, clonazepam, and omeprazole. He discontinued clopidogrel and aspirin 7 days before surgery on the advice of the surgeon in consultation with his cardiologist. Electrocardiography (ECG) showed normal sinus rhythm with a left anterior fascicular block. Standard ASA monitors were placed in the operating room. After preoxygenation of the patient, anesthesia was induced with lidocaine, propofol, fentanyl, and cisatracurium. Metoprolol 2.5 mg was given twice shortly after induction to maintain the heart rate less than 80 beats per minute (bpm). The patient's oxygen saturation via pulse oximetry (Spo_2) was greater than 95%, and his systolic blood pressure was maintained between 120 and 180 mmHg throughout the anesthetic. Surgery proceeded without complications. An estimated blood loss of 750 mL was replaced with two units of packed red blood cells (PRBCs), 500 mL of 5% albumin, and 3800 mL of normal saline. The patient's trachea was extubated, and he was taken to the postanesthesia care unit (PACU) taking supplemental oxygen.

After an uneventful 45-min stay in the PACU, the patient was transferred to the surgical ward. Shortly thereafter, he began to complain of right-sided chest pain similar to that experienced during his previous MI. He was immediately treated with two mg of morphine, 5 mg of intravenous (IV) metoprolol, and 325 mg of oral aspirin. An ECG showed ST-segment elevation in leads III and aVF as well as reciprocal ST depression in leads V5 and V6. The patient was taken emergently to the cardiac catheterization laboratory.

Coronary angiography showed 100% occlusion of the RCA stent consistent with thrombus and the likely cause of the postoperative MI. Successful balloon angioplasty was followed by placement of a BMS within the previously deployed DES. Post-stent angiography showed no residual stenosis. The remainder of the angiographic examination was unchanged. A heparin infusion was initiated, and the patient received 300 mg of oral clopidogrel. The patient's troponin-I peaked at 167 ng/mL. Transthoracic echocardiogram before hospital discharge showed inferior and posterior wall hypokinesis, with an ejection fraction of 0.40, decreased from 0.75 preoperatively.

2.2. Case 2

A 54-year-old man with end-stage renal failure secondary to hypertension and type I diabetes mellitus was evaluated for deceased donor kidney-pancreas transplant. Cardiac catheterization before transplantation showed diffuse CAD (80% stenosis of the distal circumflex and midobtuse marginal arteries, 70% proximal RCA, and 99% posterior descending coronary artery). After complex angioplasty, two DES were placed in his RCA, one BMS in his posterior descending artery, and two DES in his left circumflex artery. Lifelong therapy with clopidogrel 75 mg daily and aspirin 81 mg daily was recommended.

After 6 months of combined antiplatelet therapy, the patient presented for kidney-pancreas transplant; clopidogrel and aspirin were discontinued one day before surgery on the advice of the surgical team. The intraoperative course was uneventful with immediate transplant graft function. Aspirin was resumed on postoperative day 1, and clopidogrel was restarted on day 2. That evening, he passed a single maroon stool with a decrease in hematocrit (Hct) from 35% to 26%. Two units of PRBCs were transfused, and IV octreotide therapy was instituted. On postoperative day 6, the patient's abdomen became distended, with associated hypotension and with a decrease in Hct from 34% to 25%. Urgent exploratory laparotomy identified a single source of bleeding at the anastomotic suture line in the left iliac vein, which was repaired. A total of 7 units of PRBCs was required to stabilize his Hct at 31%. The remainder of his hospital course was unremarkable. Clopidogrel and aspirin therapy were resumed three days after emergent exploratory laparotomy; no further bleeding episodes occurred.