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Case Report

Simultaneous left anterior descending and right coronary stent thrombosis after aspirin withdrawal

Abstract

ST-segment elevation myocardial infarction is usually caused by plaque rupture and subsequent thrombosis of a single culprit vessel. In rare occasions, simultaneous thrombosis of 2 coronary arteries occurs, which is usually associated with a worse prognosis. Although surgery provokes hemodynamic stress, leading in some instances to myocardial ischemia due to supply/demand mismatch, other factors may also contribute to postoperative myocardial infarction. We present a case of postoperative simultaneous left anterior descending and right coronary stent thrombosis that followed cessation of long-term aspirin therapy in a patient with stable coronary artery disease. This case raises concerns with drug-eluting stents due to the higher potential for late stent thrombosis related to delayed endothelialization of the stent struts. Physicians should be very cautious when deciding to withdraw antiplatelet therapy preoperatively to avoid rebound coronary thrombosis.

A 68-year-old man with a medical history of hypertension, dyslipidemia, and coronary artery disease with derug elluting stent deployment of the right coronary artery (RCA) and left anterior descending artery (LAD) 4 and a half years earlier, presented to the hospital for elective abdominal surgery. The patient had a history of metastatic colon cancer with previous biliary stent placement and presented with obstructive jaundice for which Roux n-Y hepaticojejunostomy was planned. He denied any cardiac symptoms including exertional chest pain or shortness of breath and was compliant with his cardiac medications, which comprised aspirin as the sole antiplatelet therapy and 100 mg of daily extended release metoprolol. The patient was seen by his cardiologist for a preoperative cardiac assessment who confirmed that he had a normal clinical examination and electrocardiogram (ECG) and that his last stress test ruled out any reversible ischemia. Preoperative laboratory tests were unremarkable except for a mild elevation of bilirubin and alkaline phosphatase suggestive of biliary obstruction. The patient was cleared for surgery without any further workup with the agreement that aspirin therapy could be stopped 1 week before surgery.

The patient had a prolonged procedure for 3 hours during which he remained hemodynamically stable, maintaining good urine output, without significant blood loss or evidence of ventricular arrhythmias on the monitor. He was sent to post anesthesia care unit, fully conscious, and hemodynamically stable and was ready to be transferred to the intensive care unit when all of a sudden he had a cardiac arrest and lost consciousness. The monitor revealed ventricular fibrillation (Fig. 1) that reverted with a precordial thump after 1 round of epinephrine and atropine. The patient subsequently regained full consciousness, started to talk normally, and began experiencing chest pain. An ECG was immediately performed (Fig. 2) and revealed ST-segment elevation in leads II, III, and aVF and widespread ST-segment depression in leads V_2 to V_6 and lead I and aVL, a picture suggestive of acute inferior ST-segment elevation myocardial infarction (STEMI). The presence of ST-segment elevation in lead III greater than lead II and ST-segment depression in lead aVL greater than lead I suggested that the RCA is the culprit vessel.

The patient was given 600 mg of clopidogrel, 2.5 mg of IV metoprolol, 100% oxygen via a nonrebreather mask, IV nitroglycerin and then sent to the catheterization suite. Coronary angiography revealed total RCA occlusion due to in-stent thrombosis in addition to subtotal proximal LAD occlusion with an evident stent thrombus as shown in Fig. 3. In the catheterization suite, the patient had a second episode of cardiac arrest due to ventricular fibrillation. CPR was initiated and maintained for 30 minutes. Intubation was performed; amiodarone and lidocaine drip were given; however, the patient was pronounced dead after all resuscitative efforts failed.

Surgery is associated with increased catecholamine production leading to vasoconstriction, hemodynamic stress [1], and subsequent myocardial ischemia as a result of supply/demand mismatch. The propensity of the surgical stress to induce plaque rupture was previously demonstrated by Cohen and Aretz [2] and Dawood et al [3]. In the presented case, a patient with a previously stable coronary

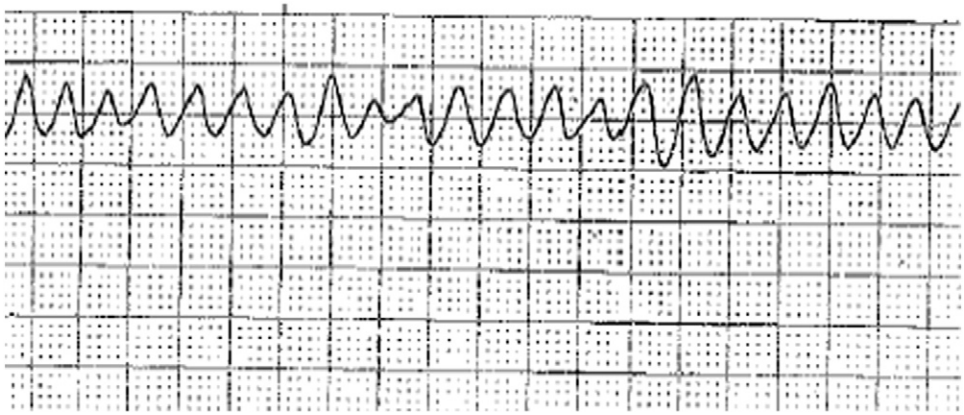

Fig. 1 Rhythm strip recorded during the arrest revealing ventricular fibrillation.

artery disease had a fatal postoperative STEMI after simultaneous occlusion of both RCA and LAD stents. Immediate coronary evaluation suggested that a more serious pathology than just supply/demand mismatch was responsible for the simultaneous late in-stent thrombosis. A clue was provided from the patient's history, when he was advised to discontinue aspirin (the sole antiplatelet medication that he was taking) 1 week before surgery.

In most scenarios, STEMI is caused by plaque rupture and subsequent thrombosis of a single culprit vessel. Rarely, simultaneous thrombosis of more than 1 coronary artery occurs [4-6] and is usually associated with a worse outcome [7]. On autopsy examination, Burke and Virmani [8] found that 25% to 50% of patients who died of MI had multiple acute coronary thromboses. Goldstein et al [7] found that up to 32.8% of patients with acute MI had thrombi in nonculprit arteries. Simultaneous coronary thrombosis is usually not due to local plaque pathology but rather due to an underlying systemic thombophilic or inflammatory state (also known as "pancoronaritis") causing diffuse atherosclerotic plaque destabilization [9]. Other reported factors for simultaneous stent thrombosis include premature cessation of antiplatelet therapy, antiplatelet resistance, antithrombin III deficiency [10], thrombocytosis [11], cocaine use [12], and secondary thrombosis due to systemic hypotension after first stent thrombosis [13].

Aspirin withdrawal in patients with coronary artery disease was found to be an independent predictor of mortality. Discontinuation of chronic aspirin therapy in patients

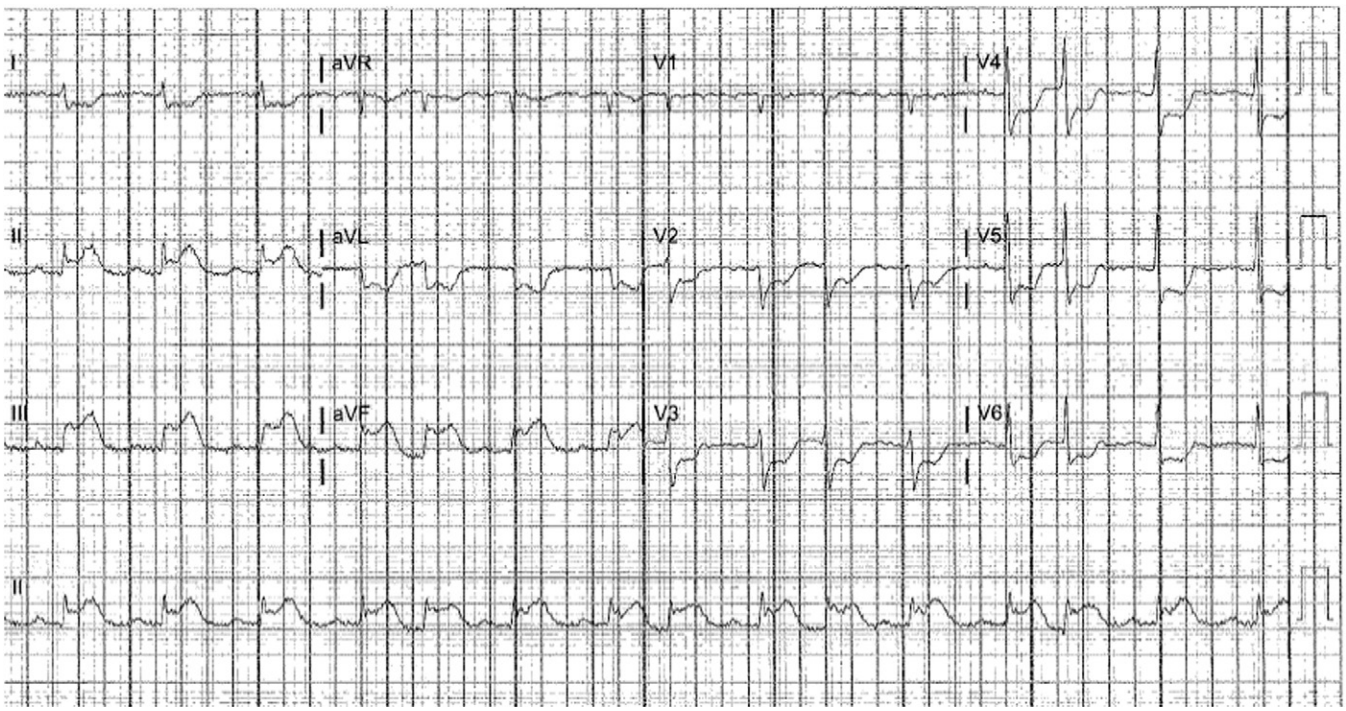

Fig. 2 Electrocardiogram after the cardiac arrest revealing ST-segment elevation in the inferior leads with ST-segment depression in the anterior and lateral precordial leads suggestive of acute inferior MI.

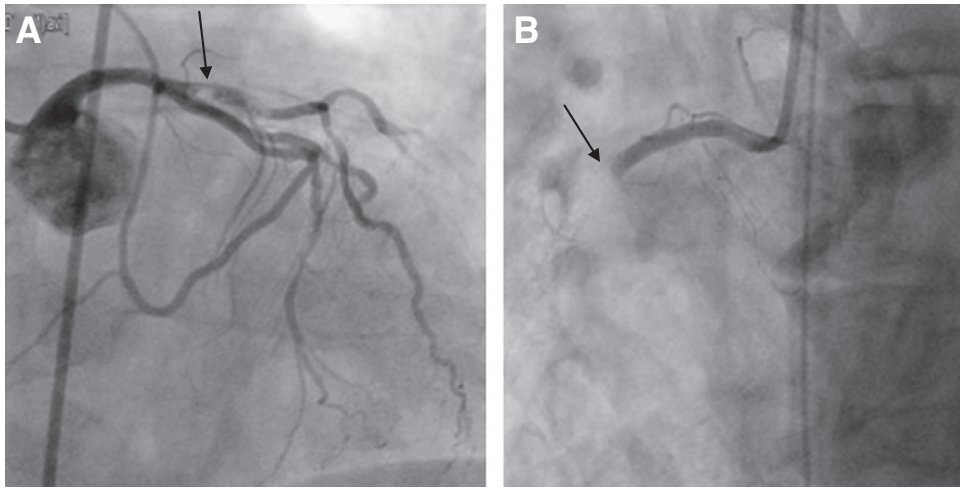

Fig. 3 Subtotal proximal LAD occlusion with an evident thrombus (panel A). Total mid RCA occlusion (panel B).

with stable coronary artery disease especially before surgery poses a potential risk of MI [14]. This was demonstrated by Collet et al [15] when he concluded increased 30-day rates of death and MI in patients with recent aspirin withdrawal. Ferrari et al [16] recognized a higher incidence of STEMI in patients who stopped aspirin therapy. There is also experimental evidence of a rebound increase in platelet activation and platelet aggregation in patients with atherosclerosis in whom aspirin therapy was discontinued [17,18]. Unexpectedly, data from the long-awaited STRTAGEM trial revealed that there was no difference in terms of occurrence of major thrombotic events between preoperative maintenance and interruption of aspirin [19]. There are concerns with drug-eluting stents due to the potential for late stent thrombosis related to delayed endothelialization of the stent struts [20]. McFadden et al [20] reported 4 cases of late stent thrombosis that occurred soon after interruption of antiplatelet therapy. Danenberg et al [21] reported a case of late stent thrombosis 2 years after implantation due to cessation of aspirin therapy 1 week before circumcision.

The presented case clearly exemplifies the phenomenon of post aspirin withdrawal simultaneous coronary thrombosis. We aim to create awareness of this rare presentation, the potential risks resulting from preoperative withdrawal of antiplatelet therapy, and emphasize the importance of imaging both left and right coronary systems and not just the culprit vessel to avoid missing a second occlusion especially if the ECG provides a clue for infarction at different territories. The report also sheds light on the phenomenon of late stent thrombosis due to delayed endothelialization of drug-eluting stents.

Appendix A. Supplementary data

Supplementary data to this article can be found online at doi:10.1016/j.ajem.2011.12.037.

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