CASE REPORT

Acute in-situ coronary thrombosis during elective coronary angiography

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Original submission:
15 September 2008;
Revised submission:
10 November 2008;
Accepted:

Med Glas 2009; 6(1): 131-133

ABSTRACT

This report presented a case of in situ intracoronary thrombosis of the proximal left anterior descending artery (LAD), causing significant transient myocardial ischemia during elective coronary angiography in a patient with known coronary artery disease (chronic occlusion of the circumflex artery, significant stable left main stenosis) and a severe vasovagal reaction during femoral artery puncture. He was treated successfully with local fibrinolytic therapy, whereas entire diagnostic procedure was completed successfully. There were no cardiac wall motion abnormalities after the procedure, and the rest of the hospitalization was uneventful.

Key words: coronary angiography, complications, thrombosis, myocardial infarction

INTRODUCTION

One of the well described complications during elective coronary angiography is coronary vasospasm provoked by severe vasovagal reaction (1, 2). The role of coronary vasospasm in acute myocardial infarction has been already discussed in the literature (3). However, there is still insufficient data on the relationship between coronary vasospasm and acute intracoronary thrombosis (4).

After a systematical Medline® and OVID® search, a series of reports on coronary vasospasm with or without intracoronary thrombosis and myocardial infarction following percutaneous coronary intervention or local anesthesia administration was found (4, 5). There were no reports of intracoronary thrombosis during elective diagnostic coronary angiography. We reported a case of intracoronary thrombosis causing significant myocardial ischemia during elective coronary angiography, treated promptly with local fibrinolytic therapy, in a patient with severe vasovagal reaction during femoral artery puncture.
CASE REPORT

A 51 year old Caucasian male underwent elective coronary angiography at the Internal Medicine Department of Slavonski Brod General Hospital. He had a history of a non ST-elevation myocardial infarction (NSTEMI) five months before admission to our hospital, with normal treadmill test and echocardiography findings at the time of discharge. During those five months he had one episode of prolonged chest pain, without new ECG changes and without elevated troponin levels. He was continuously taking acetylsalicylic acid, beta blocker and an ACE inhibitor.

Patient underwent standard preparation procedures for elective heart catheterization with oral hydration, and proper anxiolytic therapy. All the standard laboratory results, including coagulation, were normal. During femoral artery puncture he had a severe vasovagal reaction, causing extreme bradycardia (heart rate 30 bpm) and a significant blood pressure drop (systolic blood pressure 60 mmHg). Atropine 1 mg was administered intravenously with a rapid injection of 500 mL of saline solution, followed shortly by hemodynamic stabilization, thus allowing the procedure to continue. Patient had no chest pain, and felt only slight dizziness.

After the insertion of a 6 french catheter into the left coronary artery no blood pressure drop or arrhythmias were observed. After contrast injection in the left coronary artery a 50% stenosis of the left main coronary artery (LM) and a thrombus occluding the proximal LAD were noted, together with a chronic occlusion of the left circumflex artery (CxA) (Figure 1 A, B). There were no signs of ongoing coronary vasospasm. In situ thrombosis of proximal LAD was diagnosed, and 5000 IU of unfractioned heparin was administered intracoronary. At that time ST-elevation in lateral leads occurred, together with non-sustained ventricular tachycardia, and patient reported chest pain. A total of 250000 IU of streptokinase was then administered through the catheter in the left coronary artery. Prompt recanalization with TIMI 3 flow within left anterior descending artery occurred. LM stenosis with no signs of defect was still visible, there were no signs of coronary vasospasm, and no significant atherosclerotic lesions in the segment of the artery where thrombosis was witnessed (Figure 1 C, D). ST-elevation in lateral leads diminished, with normal ST-segment at the end of the procedure, and patient did not report chest pain. There were no thrombi in the perfusion systems or catheters during, and after the whole procedure. Procedure was completed with right coronary catheterization, showing only borderline atherosclerotic changes through the middle segment of the right coronary artery. At the end of the investigation the patient was in a circulatory stable condition and had no chest pain. Troponin values showed minimal myocardial necrosis (Troponin I-ADV 1.03 IU), whereas creatine kinase values remained normal. Echocardiography revealed no segmental contractility defects. The remaining two days of hospitalization were uneventful, and the patient was discharged with no symptoms at rest. He was scheduled for conservative management and follow-up, and referred to a cardiac surgeon.

Figure 1. Right anterior oblique (RAO) view of the left coronary artery during the procedure: A) acute occlusion of the proximal left anterior descending artery (LAD), B) atherosclerotic stenosis of the left main (LM), and a chronic occlusion of the CxA, C) RAO view of the left coronary artery after intracoronary injection of heparin and streptokinase showing recanalization of the lesion free LAD with coronary vasospasm in the distal part of the artery D) RAO projection of the lesion free and spasm free LAD with normal coronary flow (D. Prvulovic, 2007.)
Myocardial infarction is a rare, but known complication of elective coronary angiography, caused mainly by coronary artery embolisation. Emboli are mostly plaque derived, or thrombi formed within catheterization instruments. Coronary thrombosis causing myocardial ischemia is also described during percutaneous coronary interventions during which guidewires remain in the arteries for significant period of time. However, there were no thrombi detected after careful inspection of all devices used during the procedure, and the procedure did not include guidewire insertion within any major coronary artery. Also, there was no evidence of air embolism. We encountered no cases describing acute coronary thrombosis of a major artery witnessed during elective diagnostic procedures causing myocardial ischemia, and treated successfully with local fibrinolytic therapy. Today, primary percutaneous coronary intervention (PCI) and use of GP IIb/IIIa inhibitors are preferred therapeutic options in cases of coronary thrombosis and myocardial infarction (6). However, at that time GP IIb/IIIa inhibitors were not available to our site, and primary PCI was not yet established as a routine procedure. Although intracoronary fibrinolysis is abandoned in the modern era of and stent implantation, some recent data suggest that this therapeutic option should be reconsidered for the treatment of acute myocardial infarction (7).

Previously described vasovagal reaction with severe transient hypotension most probably caused transient coronary vasospasm followed by an abnormal flow and in situ thrombosis in a lesion-free proximal LAD in this patient (8). The same process within the LM atheromatous lesion could provoke plaque rupture, causing in situ thrombosis and embolisation of the proximal LAD, and a catheter tear or manipulation at the site of atheromatous lesion in the LM should also be taken in consideration (9). However, it is less likely that a lesion of that severity would be completely cured by intracoronary heparin and streptokinase injection. Nevertheless, this case brings to light another complication of elective coronary angiography that was successfully cured during the procedure, and probably witnesses one of the three well known pathophysiological processes in coronary thrombus formation: rheologic abnormality (10).

REFERENCES