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Case report

Coronary artery perforation: How to treat it?

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ABSTRACT

Coronary artery perforation fortunately represents a rare complication of coronary catheterization but, if not properly and promptly treated, it is burdened by a high mortality rate. Rates of coronary perforation may be potentially higher when atherectomy devices are used or very complex calcified lesions are treated. Cardiac tamponade constitutes the most severe clinical consequence.

We report the case of an intra-stent coronary perforation at the end of revascularization of a non-ST elevation myocardial infarction (NSTEMI), followed by an immediate impairment of hemodynamic compensation, due to significant pericardial effusion and subsequent cardiac tamponade.

The use of covered stents has revolutionized the management of coronary perforation and this has meant that the use of emergency CABG has decreased over the years with satisfactory immediate and short-term outcomes, reducing the incidence of acute cardiac tamponade and mortality without surgery.

Introduction

Percutaneous coronary intervention (PCI) is today the gold standard for treatment of coronary lesions, however the possible complications deserve proper attention and treatment modality. Coronary perforation represents fortunately a rare complication (incidence among 0.2–0.6%) and it is burdened by a high mortality rate if not properly and promptly treated [1,2].

We report the case of an intra-stent coronary perforation at the end of revascularization of a non-ST elevation myocardial infarction (NSTEMI), followed by immediate impairment of hemodynamic compensation due to significant pericardial effusion and subsequent cardiac tamponade.

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Case report

We present the case of a 67-year-old Caucasian man with arterial hypertension, and also a former smoker, who in 2008 was admitted to our department for lateral NSTEMI. A coronary angiography was performed in our catheterization laboratory with the evidence of critical stenosis (70–90%) of the distal left anterior descending (LAD) coronary artery, which also appeared dilated with a sequence of moderate stenosis at the middle segment. An occlusion of the distal left circumflex artery (LCX) was also present rehabilitated by collateral homocoronaric circulation and moderate stenosis of the right coronary artery (RCA).

After an ineffective attempt at revascularization of the distal LCX, the patient was discharged after medical therapy optimization (aspirin, beta-blocker, ACE inhibitor, statin, ranitidine).

Due to the persistent unstable angina, a myocardial scintigraphy was performed in January 2013 which showed moderate/severe inducible ischemia with low workload involving the inferolateral myocardial wall. The patient refused a further coronary angiography.

In November 2013 he was referred to our emergency room because of the onset of localized pain in the left shoulder. A diagnosis of NSTEMI was made (ST segment depression in the anterior side extended max 3 mm in V3-5, ST segment elevation max 1 mm in aVR) (Fig. 1) and he was transferred to our cardiology care unit (CCU).

At admission Troponin I was 0.035 ng/ml (normal value <0.012 ng/ml), eGFR (MDRD) 65.4 ml/min/1.73 mq, Killip class 1 and high GRACE risk score (153).

The patient was treated with acetylsalicylic acid (300 mg orally) and subcutaneous enoxaparin (6000 IU); an urgent coronary angiography with right femoral artery access was performed showing extremely calcified coronary vessels, and critical stenosis of middle LAD with occluded apical segment rehabilitated by collateral homocoronaric circulation (Fig. 2, panels A and B). Distal LCX and obtuse marginal (OMB) were occluded and rehabilitated by homocoronaric collateral circulation (Fig. 2, panel C), a RCA with critical stenosis along its posterolateral and posterior interventricular branches of small caliber (Fig. 2, panel D).

So we decided to administer prasugrel 60 mg orally and to perform PCI on middle LAD beyond the critical and extremely calcified stenosis with the guide wire BMW ABBOTT (a second ABBOTT BMW guide wire placed on diagonal branch) and with implantation of a bare metal stent (BMS) MULTI-LINK ABBOTT 5 mm × 18 mm (issued to 16 atm, estimated stent diameter 5.33 mm).

At the end of first BMS implantation, we observed an aspect of minus in the stenosis downstream to the distal edge (plaque shift, stenosis accentuation?), so we proceeded to implantation of an overlapping BMS MULTI-LINK ABBOTT 4.5 mm × 18 mm (issued to 14 atm, diameter reached 4.8 mm) and post-expending the point of overlap with the balloon of the second stent. Angiography performed after implantation of these two overlapping BMS showed the presence of intra-stent coronaric perforation type III [1] with hemorrhage (Fig. 3, panels A and B) and progressive and rapid reduction in blood pressure and bradycardia. Due to rapid hemodynamic destabilization, after the placement of an intra-aortic balloon pump (IABP), a covered stent GRAFTMASTER (Jostent Graftmaster, Abbott Vascular) 4.5 mm × 16 mm (inflated up to cover the perforation) was placed (Fig. 4) but the angiographic acquisitions in the course of the placement of the covered stent and at the end of the placement revealed multiple perforations (difficult to assess precisely because of severe hypotension, systolic blood pressure 60 mmHg) with slow flow in the middle-distal segment of LAD (Fig. 5). For this reason we proceeded to the positioning of an overlapping covered stent GRAFTMASTER 4.5 mm × 19 mm. A subsequent

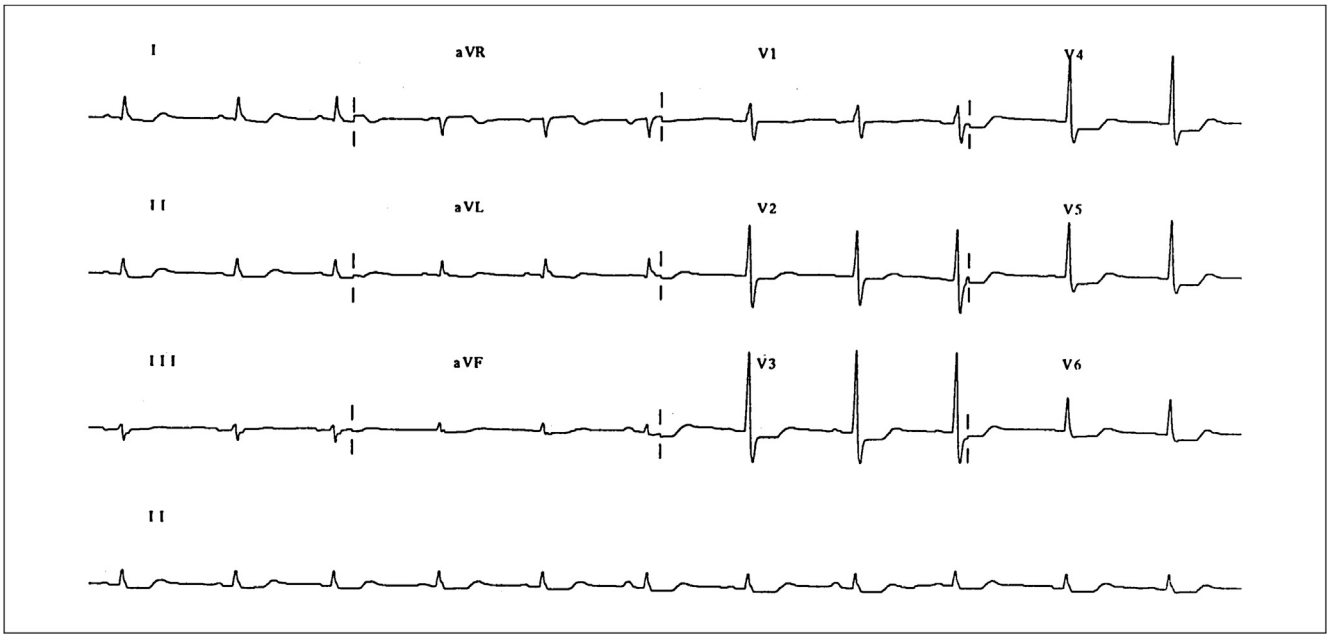

Fig. 1 – ECG at the admission.