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Case Report

INTERVENTIONAL TRAPS IN ECTATIC CORONARY ARTERIES: A CASE REPORT

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ABSTRACT – Coronary artery ectasia or aneurysms are dilatation of an arterial segment to a diameter at least 1.5 times that of the adjacent normal. Blood flow through such arteries is disturbed and turbulent, which, with the activation of endothelium, leads to chronic thrombosis in the blood vessel wall. Percutaneous coronary interventions in ectatic / aneurysmal vessels carry a high risk of complications, primarily a "no-reflow" phenomenon. No-reflow is common in patients with acute coronary syndrome, especially ST elevation myocardial infarction (STEMI). In this article, we present the occurrence of the no-reflow phenomenon in a stable patient undergoing percutaneous intervention due to a significant stenosis of the aneurysmally altered right coronary artery. Despite the rapid placement and optimization of stents and the applied drug therapy combined with thromboaspiration periprocedurally, it was not possible to establish TIMI 3 flow. However, after the initial failure and development of periprocedural STEMI, the patient was successfully stabilized with unexpectedly good recovery of infarcted myocardial function. Control coronarography 6 weeks after the initial event confirmed a proper stent patency with normal TIMI 3 flow.

Key words: *aneurysms, acute coronary syndrome, thromboaspiration, no-reflow.*

Introduction

Ectatic and/or aneurysmically dilated coronary arteries (CAE) are diagnosed in up to 5% of all coronary angiograms. Such changes are mostly asymptomatic and detected by chance, and in a small number of cases they may lead to the development of acute coronary syndrome. The treatment may be medical (conservative), endovascular or surgical. Endovascular treatment presents a major challenge in interventional cardiology with possible unpredictable complications.

Definition and classification

According to the definition, CAE is a coronary artery dilatation > 1.5 times in relation to the reference lumen of the vessel and the prevalence of 1.2 - 4.9% (1). The two basic phenotypes are coronary ectasia and aneurysm. If the enlargement is segmental, it is an aneurysmal type, whereas coronary ectasia refers to a diffuse dilation of the coronary arteries. Aneurysms are further subdivided into saccular, if the transverse diameter is larger than the longitudinal one, and fusiform, if the longitudinal diameter is larger (1). Ad-

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ditionally, they can be subdivided according to the diameter into small (lumen < 5 mm), moderate (5-8 mm) and large (> 8 mm). Depending on the location, right coronary arteries (RCA) are the most commonly affected > 40%, followed by the left anterior descending artery (LAD, 32%). The left main and circumflex arteries (ACx) are the least commonly affected (1). In adulthood, the most common cause of these changes is atherosclerotic heart disease, while genetic factors and inflammatory diseases, such as vasculitis (Kawasaki), are the most common at a younger age (2).

Clinical presentation and therapy

CAEs are asymptomatic in most patients and are detected incidentally during routine coronary angiography or MSCT coronary angiography. However, in some patients, they may cause a clinical picture of stable angina pectoris and may sometimes be the cause of acute coronary syndrome (3). In asymptomatic patients, the risk of rupture or distal embolization should be assessed based on the morphology and extent. Therapy should focus on the treatment of standard risk factors for atherosclerotic heart disease and the use of antiplatelet therapy. In the case of multivessel ectasia and aneurysms, the use of oral anticoagulant therapy may be taken into consideration. In large saccular aneurysms with a risk of rupture, endovascular or surgical treatment is recommended (3). Endovascular treatment with stent grafts is performed to isolate the aneurysm, while surgical treatment of excisions is performed exclusively in giant aneurysms.

Patients with symptoms of stable angina should have functional evidence of ischemia before deciding on the endovascular treatment. In case of unfavourable anatomy or multi-vessel coronary disease, surgical revascularization should be preferred (Figure 1) (3).

Acute coronary syndrome in patients with CAE occurs due to distal embolization of thrombogenic plaque or primary rupture of atherogenic plaque (4). Percutaneous coronary intervention (PCI) is the treatment of choice in patients with acute coronary syndrome. However, PCI on the responsible ectatic vessel in acute myocardial infarction results in significantly poorer revascularization results, caused by a high percentage of no-reflow phenomena, especially in patients with STEMI. Therefore, thromboaspiration and intracoronary administration of glycoprotein IIb/IIIa inhibitors (GP IIb/IIIa) are necessary in most cases. Given the difficult anatomy, the primary goal of treatment is to establish TIMI 3 flow, based on balloon dilatation, thrombectomy and using GP IIb/IIIa inhibitors, initially in a bolus and then in continuous infusion for 24 - 48 hours (3).

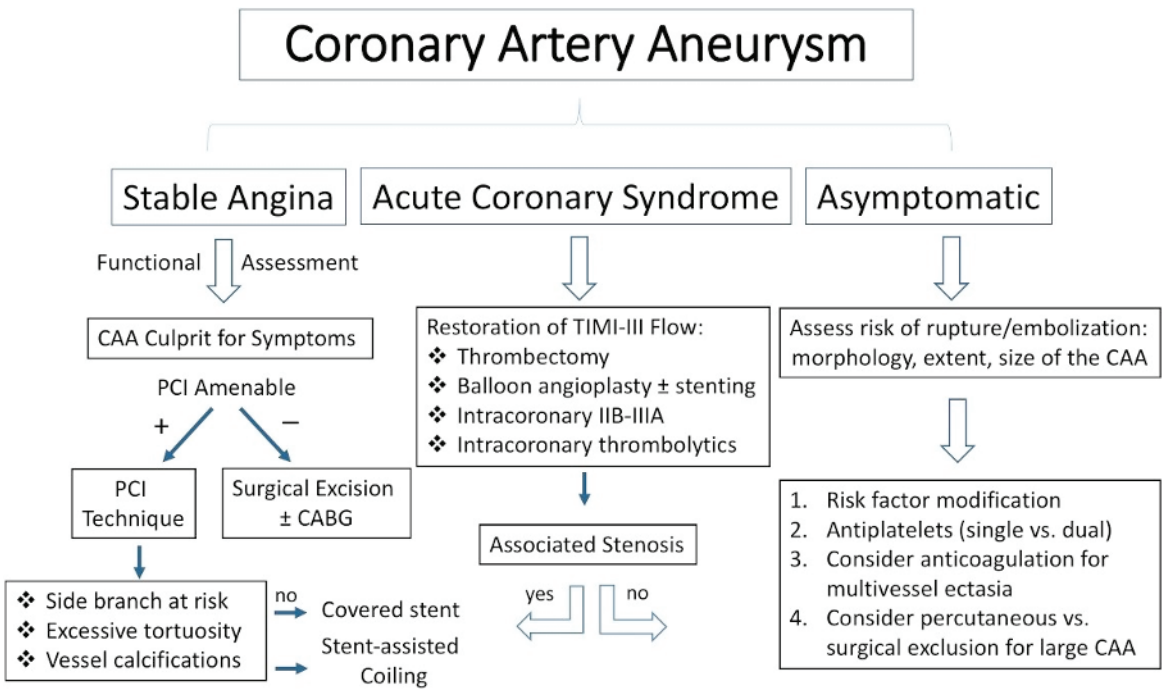


Figure 1. Algorithm for the management of patients with coronary artery aneurysms.

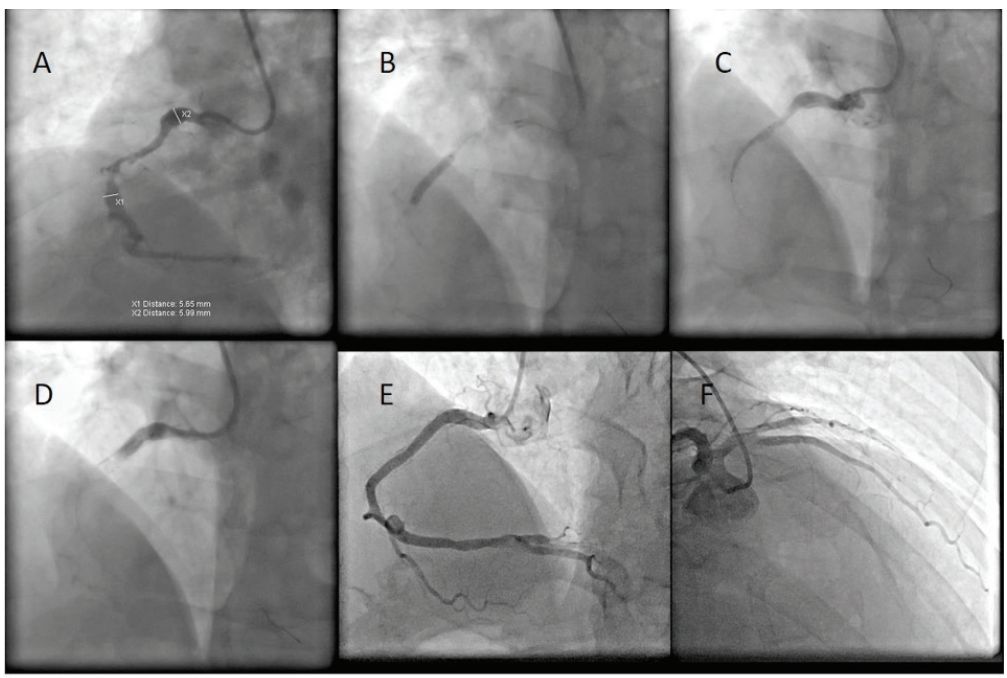
Case report

A 54-year-old patient, with a history of acute non-ST-elevation myocardial infarction (NSTEMI) and PCI of ectatic ACx five years ago, was hospitalized due to worsening of anginal symptoms. Invasive cardiac treatment was performed through the cardiology day hospital, which established significant LAD stenoses, the stenosis of first diagonal branch (Dg1) and significant ectatic RCA stenosis. Based on the findings, an elective PCI in two acts was planned.

In the first act, PCI LAD - Dg was performed, first by dilating the diagonal branch with stent implantation (Xience 3.0/15 mm), and then by revascularisation of LAD-Dg with the provisional technique (3.5/23 mm with POT 4.5/8 mm) with the optimal final angiographic result. After one month, the elective PCI was performed on ectatic RCA, 6.0 mm in diameter and with 80% QCA stenosis in the proximal segment. After the initial predilatation of the lesion, a "no-reflow" phenomenon occurred. Two DES stents (5.0/22 mm) were placed in the middle and proximal segments, which were then post-dilated with a balloon (NC Emerge 6.0/12mm) but without the establishment of the TIMI 3 flow. Intracoronary (i.c.) drug therapy was applied; nitroglycerin 300 mcg, adenosine 100 mcg i.c. but still without establishing the flow. A balloon dilatation (SC 3.0 / 15 mm) was then made to the RCA junction, followed by thromboaspiration (Export 6FR), still with no flow. The patient became hemodynamically unstable and developed STEMI infarction with the development of ventricular fibrillation. The patient was defibrillated, sedated, placed on mechanical ventilation, and transferred to the coronary care unit. Following intensive care measures were performed, the patient was clinically stabilized and separated from the respirator after 24 hours. Echocardiography displayed loss of contractility but the extent of damage was smaller than expected, given the size of the supply area of RCA, and initially more dominant loss of right ventricular function, which recovered significantly during the hospital stay. The patient was discharged 12 days after the initial periprocedural infarction. After 6 weeks, re-coronarography was performed and showed normal patency of stents in RCA with TIMI 3 flow (Figure 2).

Discussion

Percutaneous coronary interventions on ectatic blood vessels in acute coronary syndrome are associated with a higher prevalence of no-reflow phenomena.



A RCA before procedure; B predilatationNC4,0/20mm at 12 Atm; C position of 1st. stent 5,0/22 ; D „no reflow" E normalTIMI 3 flow after 6 weeks ; F normal flow through LAD DG1

Figure 2. Percutaneous coronary intervention of the ectatic right coronary artery.

The term "no-reflow" implies functional and structural changes in microcirculation, primarily caused by distal embolization and ischemic or reperfusion injury. Yip et al. reported no-reflow in as many as 68% of patients with STEMI on aneurysm-altered vessels (5).

The occurrence and frequency of the no-reflow phenomenon in clinically and hemodynamically stable patients is unknown. A total of 78 patients participated in the study conducted by Joo et al., 60% of who had acute coronary syndrome and 40% stable angina pectoris. In the group of patients with coronary aneurysms, a higher incidence of major adverse cardiovascular events (MACE) was recorded in 26% of cases, compared with 2.2% in the group of patients without coronary aneurysm (6).

In the case presented here, PCI was conducted on an ectatic vessel in stable coronary heart disease. A probable pathophysiological mechanism of no-reflow was the rupture of soft and chronically thrombosed plaque caused by balloon predilatation, which led to distal embolization and massive thrombosis (7).

Intracoronary drug administration and thrombectomy are key therapeutic options for the treatment of no-reflow phenomena. Annibali et al. recommend an algorithm based on the presence or absence of hypotension and bradycardia, and, depending on hemodynamic parameters, recommend the use of adenosine, norepinephrine or nitroprusside as the first therapeutic options, while the use of GP IIb/IIIa inhibitors is reserved as a second line of treatment (Figure 3) (7).

Thromboaspiration is also one of the key methods for treating the no-reflow phenomenon. Namely, the routine use of thromboaspiration in STEMI has not led to a significant reduction in mortality and is not recommended according to most guidelines of the European Society of Cardiology (8).

If the earlier mentioned methods fail to establish an adequate TIMI 3 flow, extensive myocardial necrosis occurs, which in our case led to the development of transmural infarction, complicated with malignant arrhythmia. However, after initial stabilisation, a relatively favourable course of recovery and coronary angiography performed 6 weeks after the primary adverse event, our patient showed a favourable outcome with TIMI 3 flow. This late spontaneous establishment of TIMI 3 flow has been noted in previous researches of thrombolytic therapy era (9). Functional and structural changes in microcirculation are influenced by reactive hyperaemia, transient blockage by microthrombi and neutrophils, and microvascular spasm (10). It is believed that the phenomenon can be prevented or treated only at a very early stage of coronary recanalization, and the resulting changes will become reversible and spontaneously stabilized within 24 hours of the no-reflow phenomenon (11).

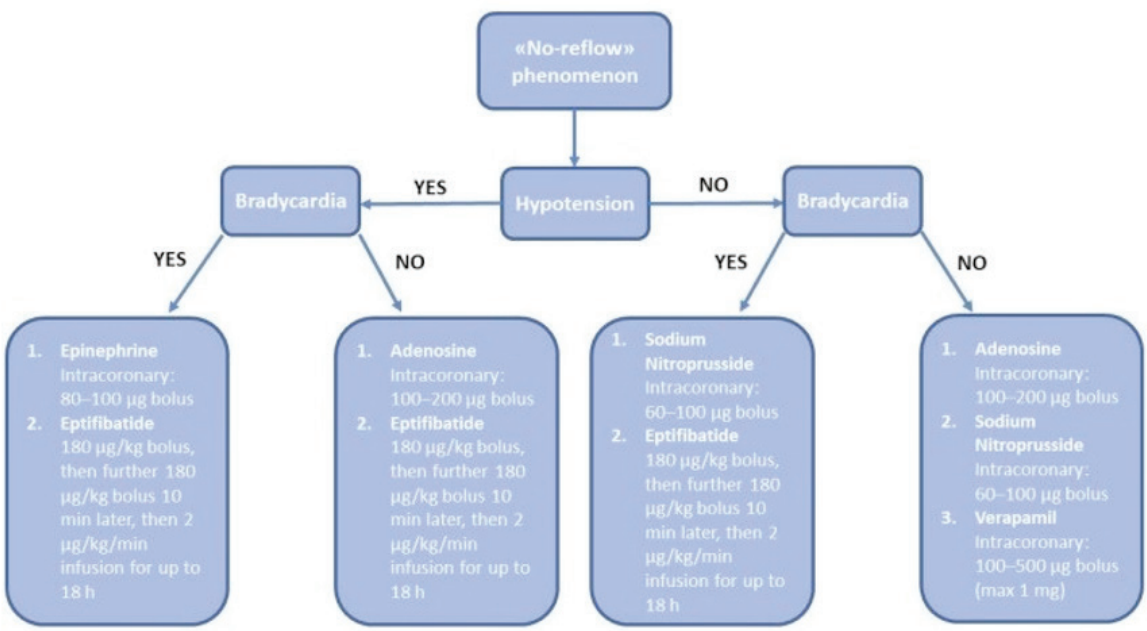


Figure 3. Algorithm for the treatment of the no-reflow phenomenon.

Our report is in accordance with the CARE guideline available through the EQUATOR network (http://www.equatornetwork.org/) and COPE guidelines (http://publicationethics.org/). All procedures in this study were performed in accordance with the ethical standards of the Helsinki Declaration and its later amendments or comparable ethical standards.

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Sažetak

INTERVENCIJSKE ZAMKE U KORONARNIM EKTAZIJAMA

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Koronarne ektazije i aneurizme su proširenja koronarnih arterija promjera > 1,5 puta u odnosu na referentni lumen krvne žile. Protok krvi kroz tako promijenjene arterije je poremećen, turbulentnog tipa, a što uz aktivaciju endotela dovodi do kronične tromboze u stijenci krvne žile. Perkutane koronarne intervencije kod ektatičnih / aneurizmatskih žila nose visok rizik od nastanka komplikacija, prvenstveno "no- reflow" fenomena. Pojava no-reflow je česta kod bolesnika sa akutnim koronarnim sindromom te osobito onih s transmuralnim infarktom (STEMI). U ovom članku predstavljamo pojavu "no-reflow" fenomena kod hemodinamski stabilnog bolesnika, podvrgnutog perkutanoj intervenciji zbog značajne stenoze aneurizmatski izmijenjene desne koronarne arterije. Unatoč brzom postavljanju i optimizaciji stentova te svoj primijenjenoj medikamentoznoj terapiji, uključujući i tromboaspiraciju, periproceduralno se ne uspijeva uspostaviti TIMI 3 protok. Nakon inicijalnog neuspjeha i razvoja periproceduralnog STEMI bolesnik se uspješno stabilizira te se prati neočekivano dobar oporavak funkcije infarktom zahvaćenog miokarda. Kontrolna koronarografija 6 tjedana nakon neželjenog događaja potvrđuje urednu prohodnost implantiranih stentova uz TIMI 3 protok.

Ključne riječi: *aneurizme; akutni koronarni sindrom; tromboaspiracija; no-reflow.*