

Persistent angina after myocardial revascularization: a case report

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Abstract

A 56-year-old woman with stable angina was admitted to our Cardiology Department; because of the presence of symptoms and signs of ischemia during a stress test she was a candidate for coronary evaluation. At 1 and 6 months of follow up, despite a complete coronary revascularization, the patient continued to complain of angina; stress testing continued to show signs of myocardial ischemia. This is an example of an individual with persisting angina despite “successful” coronary myocardial revascularization.

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Introduction

Revascularization of coronary epicardial stenoses by percutaneous angioplasty and stenting (percutaneous coronary intervention) is expected to improve angina pectoris and myocardial ischemia. Surprisingly, several clinical trials have incidentally reported the persistence of angina and ischemia after “successful” procedures.

We report the case of a woman with chronic stable angina who, after a “successful” coronary revascularization, remained symptomatic for angina and stress-induced ischemia.

Case report

A 56-year-old woman was admitted to our Cardiology Department in April 2005, with stable angina pectoris. She had a history of typical anginal pain and dyspnea for moderate exercise since 2004. Her risk factors for coronary artery disease included arterial hypertension, hypercholesterolemia, and obesity (body mass index 30 kg/m²). In February 2005, because of increasing intensity and duration of chest

pain, she underwent an exercise stress test that was stopped at the 1st minute of the third step (modified Bruce protocol) because of chest pain and downward sloping ST-segment depression (> 2 mm) in precordial leads V₄–V₆ (*Figure 1*). Maximal heart rate was 143 beats/min and maximal blood pressure was 150/80 mm Hg (rate–pressure product [RPP] 21 450). She was prescribed ticlopidine, simvastatin, and transdermal nitrates.

At the time of her admission to hospital, physical examination and routine blood examinations were normal, except for total cholesterol 260 mg/dL, high-density lipoprotein cholesterol 59 mg/dL, low-density lipoprotein cholesterol 177 mg/dL, and triglycerides 120 mg/dL. Echocardiography showed a normal contractile function and a normal left ventricular ejection fraction (60%).

Coronary angiography showed a single-vessel disease of the left anterior descending coronary artery (LAD), with proximal stenosis and a long distal stenosis (*Figure 2*). Fractional flow reserve at the level of the proximal stenosis was 0.88 at baseline and 0.70 after intracoronary adenosine. Successful percutaneous transluminal coronary angioplasty (PTCA) and stenting (drug eluting stents: Cypher 3.0 × 18 mm



Figure 1. Signs of exertional ischemia during stress testing: downward sloping ST-segment depression in precordial leads.

plus 3.0 × 8 mm) of the proximal lesion were performed (Figure 3).

Treatment on discharge included candesartan and hydrochlorothiazide, ticlopidine, and simvastatin (the patient was allergic to ramipril and aspirin).

One month later, the exercise stress test was repeated, but it was stopped at the beginning of the 2nd step of the modified Bruce protocol because of chest pain and downward sloping ST-segment depression (> 2 mm) in leads V₄–V₆. Maximal heart rate was 172 beats/min and maximal blood pressure was 150/90 mm Hg (RPP 25 800) (Figure 4). Calcium-channel blockers (60 mg three times a day) and nitrates (20 mg three times a day) were added to the medication because of persistent chest pain on effort.

In May 2005, the patient was re-admitted to our Cardiology Department. Coronary angiography showed a patent proximal LAD. Fractional flow reserve at the level of the distal LAD stenosis was measured and was 0.83 at baseline and 0.71 after



Figure 2. Coronary angiography, showing a single-vessel disease of the left anterior descending coronary artery: proximal stenosis and a long distal stenosis.



Figure 3. Percutaneous transluminal coronary angioplasty and stenting of the proximal lesion. Two drug eluting stents were used (Cypher 3.0 × 18 mm and 3.0 × 8 mm).

intracoronary adenosine. PTCA followed by implantation of a drug eluting stent was performed at this level (Figure 5).

One month after the second procedure, an exercise stress test was again stopped, at the beginning of the 3rd step of the modified Bruce protocol, because of chest pain and downward sloping ST-segment depression in precordial leads V₄–V₆ (Figure 6). Maximal heart rate was 161 beats/min and maximal blood pressure was 220/110 mm Hg (RPP 35 420). Drug therapy was modified by the addition of isosorbide mononitrate, verapamil, and bisoprolol.

Six months later the patient was still complaining of persistent angina and a stress test was again stopped, at the 2nd step of the modified Bruce protocol, because of chest pain and horizontal ST-segment depression in precordial leads V₄–V₆ (Figure 7). Maximal heart rate was 137 beats/min and maximal blood pressure was 220/120 mm Hg (RPP 30 140).



Figure 4. Exertional ischemia during a stress test, showing downward sloping ST-segment depression in precordial leads.

Case report

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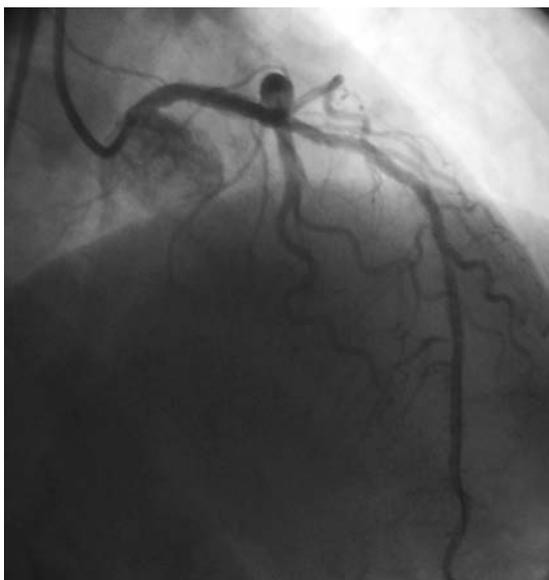


Figure 5. Coronary angiography performed in May 2005, showing a patent proximal left anterior descending artery (LAD). Percutaneous transluminal coronary angioplasty followed by implantation of a drug eluting stent was performed at the level of the distal LAD.

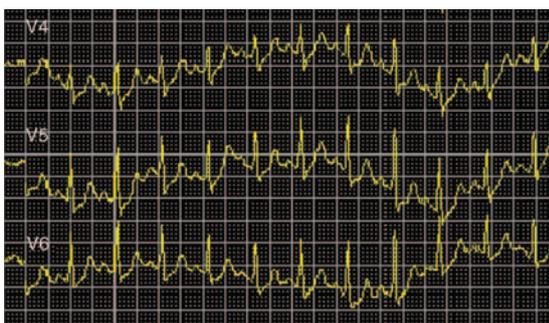


Figure 6. Exercise stress test performed 1 month after the second procedure, showing a downward sloping ST-segment depression in precordial leads V₄-V₆.

A single photon emission computed tomography perfusion study was consistent with a diffuse defect in subendocardial perfusion.

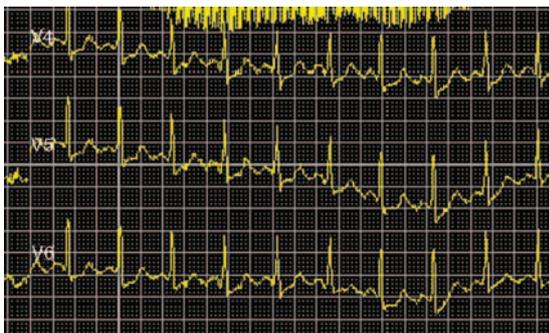


Figure 7. Exercise stress test performed 6 months after the second procedure. Again, there is a downward sloping ST-segment depression in precordial leads V₄-V₆.

Discussion

A case is reported of “persistent angina” – that is, angina and ischemia persisting after a complete and “successful” coronary recanalization. Several clinical trials have shown that patients may remain symptomatic for angina and in need of antianginal agents after coronary revascularization [1–5]. This observation has been confirmed in recent trials, including trials with drug eluting stents [3–5]. A growing body of evidence suggests that persistent angina is an emerging problem for the clinician [6].

In this patient, an angiographically “successful” myocardial revascularization was obtained, but the patient was never relieved from angina. “Persistence” of angina in such patients may be related to microvascular dysfunction. Several clinical and experimental studies have shown that microvascular dysfunction may result in paradoxical vasoconstriction of microvessels when metabolic demand increases [7–9].

Classical “hemodynamic” antianginal treatments were ineffective in our patient. Metabolic agents such as trimetazidine may offer an attractive alternative for patients with “persistent” angina [10]. ■

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