Changing ischemic symptoms despite stable coronary anatomy

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Abstract
A 67-year-old man with known ischemic heart disease was referred to our Cardiology Department because of dyspnea on effort and exertional angina. An exercise stress test showed significant progression of ischemic signs on the electrocardiogram, also involving the area of a totally occluded coronary artery. Repeated angiography confirmed the absence of angiographic changes to support the clinical progression of the ischemic syndrome.


Keywords: Angina, chronic ischemic heart disease, chronic total coronary occlusion, dyspnea, exercise stress testing

Introduction
Chronic total coronary occlusions are observed in 35–50% of patients with significant coronary disease undergoing diagnostic angiography [1,2]. It is expected that the extent of the corresponding ischemic region remains essentially unchanged unless a significant progression of atherosclerotic lesions in other vessels occurs, but, as often documented, there is no close relationship between the severity of epicardial coronary stenosis and that of the ischemic syndrome. We report here the case of a patient with chronic total occlusion of the right coronary artery, who presented with a worsening of his ischemic syndrome despite no appreciable changes in coronary angiographic features.

Case report
A 67-year-old man was admitted to our Cardiology Department in October 2008. His cardiac history had begun in 2002, when he was admitted after an acute myocardial infarction. Subsequently, he presented occasional episodes of chest pain and, in January 2007, he underwent an exercise stress test, as part of a preoperative evaluation for abdominal surgery. At peak exercise ST-segment depression was present in anterior leads from V4 to V6 and in the inferior leads, in the absence of any symptom (125 W, 6.5 metabolic equivalent of task units [METS], maximal heart rate 131 beats/min; maximal blood pressure 210/100 mm Hg) (Figure 1).

In April 2007, a stress scintigraphy confirmed inducible ischemia in the proximal inferolateral segments. Coronary angiography revealed a total occlusion of the right coronary artery (Figure 2), a 50% proximal stenosis and a 70% mid-distal stenosis of the first obtuse marginal branch and an intercoronary collateral circulation between the left circumflex coronary artery and the distal right coronary artery (Figure 3; Rentrop 1).

Given the absence of anginal symptoms, no percutaneous coronary intervention was performed in that setting. Five months later, the patient started to complain of dyspnea on effort and occasional exertional angina; in the meantime he had been scheduled to undergo surgical repair of an inguinal hernia. A second exercise stress test was therefore performed which documented ST-segment depression in leads DII, DIII, aVF, and V5–V6 (100 W, 5.3 METS,
maximal heart rate 110 beats/min; maximal blood pressure 165/90 mm Hg) (Figure 4).

The patient was admitted to our division. His cardiovascular risk profile included hypertension, diabetes mellitus, and dyslipidemia; other routine blood chemistry was normal. An echocardiogram revealed normal left ventricular contractile function and volume. Coronary angiography was repeated and confirmed total occlusion of the right coronary artery and the 50% proximal and 70% mid-distal stenosis of the first obtuse marginal branch (Figure 5). With the aim of improving the collateral circulation to the distal right coronary artery, angioplasty (Figure 6) and stenting (Cypher 3.0/8) (Figure 7) were performed on the marginal branch.

The patient was discharged receiving aspirin, clopidogrel, verapamil, furosemide, and candesartan.

Ten days after the procedure, he performed an exercise stress test that showed ST-segment depression in leads DI, DII, and DIII, in the absence of symptoms (125 W, 6.1 METS, maximal heart rate 108 beats/min; maximal blood pressure 200/80 mm Hg) (Figure 8).

Comment

In patients with chronic angina, changes in ischemic threshold are commonly attributed to progression of coronary atherosclerotic lesions. Here, however, we have reported a case of changing severity of ischemia, in the absence of significant progression of coronary atherosclerosis in other vessels.

At the time of the first exercise stress test, the patient was almost asymptomatic for angina or dyspnea;
onset of his symptoms was associated with an extension of ischemic signs in a control exercise stress testing. Indeed, a similar pattern on the electrocardiogram was present at lower working load, lower METS, and lower double-product values. We hypothesized that a revascularization procedure might improve the collateral circulation and myocardial perfusion, and therefore we performed percutaneous transluminal coronary angioplasty and stenting on the only treatable lesion. Nevertheless, the exercise stress test undertaken after this “successful” percutaneous coronary intervention showed a similar electrocardiographic pattern at equivalent METS and double-product values, confirming the absence of any obvious link between coronary anatomy and progression of the ischemia over time.

This case report emphasizes that we frequently adopt the position of passive bystander in the management of chronic ischemic heart disease. For this reason, we perhaps need to see beyond the plaque and have the confidence to leave plaques alone; alternative mechanisms, including microvascular dysfunction and coagulation abnormalities, may be important determinants of chronic ischemic heart disease. The awareness of several underlying mechanisms could help us to avoid ineffectif, expensive, and possibly harmful revascularization procedures and to optimize treatment by other means.

REFERENCES