Slow coronary flow phenomenon associated with exercise-induced myocardial ischemia

Egzersize bağlı miyokard iskemisine yol açan koroner yavaş akım fenomeni

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Slow coronary flow in the presence of normal epicardial coronary arteries frequently has an asymptomatic course; however, some reports have showed its pathologic potential. A 35-year-old male patient was admitted with a history of exertional angina (Canadian class II) of a year duration. During treadmill exercise stress testing, he experienced typical angina pectoris and a 2-mm ST-segment depression. Coronary angiography revealed slow flow in the right coronary artery without any other coronary pathology. Medications with aspirin, nitroglycerin, and a beta-blocker were instituted, after which relief of symptoms was observed. His complaints did not recur during a follow-up period of a year.

Key words: Angina pectoris; blood flow velocity; chest pain/etiology; coronary angiography; coronary circulation; coronary disease; exercise test; vascular resistance.

Epikardiyal koroner arterlerin normal olup, koroner akımın yavaş olması sıklıkla asemptomatik bir seyre sahiptir; ancak, bazı raporlar bu durumun patolojik potansiyeli olduğunu göstermektedir. Otuz beş yaşında bir erkek hasta bir yıldır var olan egzersiz anginası (Kanada sınıf II) nedeniyle yatırıldı. Treadmil egzersiz testi sırasında hastada tipik angina pektoris gelişti ve 2 mm'lik ST segment çökmesi görüldü. Koroner anjiyografide sağ koroner arterde yavaş akım dışında bir soruna rastlanmadı. Aspirin, nitrogliserin ve beta-blokerle medikal tedaviye başlanmasının ardından hastanın semptomları geriledi ve bir yıllık takip süresi boyunca şikayetlerin bir daha tekrarlamadığı görüldü.

Anahtar sözcükler: Angina pektoris; kan akım hızı; göğüs ağrısı/etyoloji; koroner anjiyografi; koroner dolaşım; koroner hastalık; egzersiz testi; vasküler direnç.

Patients with chest pain and normal epicardial coronary arteries represent a nonhomogeneous and often unclassifiable group. In this spectrum, slow coronary flow (SCF) characterized by slow dye progression to the distal branch of the coronary artery in the absence of any major epicardial stenosis is a distinct angiographic phenomenon. In 1972, Tambe et al.[1] were the first to coin the term in six cases with angina pectoris. However, since that time, its clinical significance has remained unclear.

In this article, we reported a young patient with a history of exercise-induced angina, in whom coronary arteriography revealed slow flow in the right coronary artery.

CASE REPORT

A 35-year-old man was admitted to the cardiology department with a history of exertional angina (Canadian class II) of a year duration. His blood pressure was 110/70 mmHg, heart rate was 57/min, and heart sounds were normal. Systematic inquiry showed no further significant symptoms. There was no family history of coronary artery disease. He had been a smoker (one pack/day) for 12 years. The resting 12-lead ECG was unremarkable. Chest radiography revealed a normal cardiac silhouette and lung fields. Transthoracic echocardiographic evaluation showed a normal left ventricular function and no regional wall motion abnormality. The patient underwent a treadmill exercise stress test using the Bruce protocol. He experienced typical angina pectoris at 4 min of exercise, with nearly a 2-mm ST-segment depression in D_2, D_3, aVF derivations. Cardiac catheterization was performed for suspected coronary artery disease. During left heart catheterization, systemic arterial pressure was normal and there was no gradient across the aortic valve. A left ventricular angiogram obtained in the right anterior oblique view revealed no regional wall motion abnormality. A cineangiogram of the left coronary artery in

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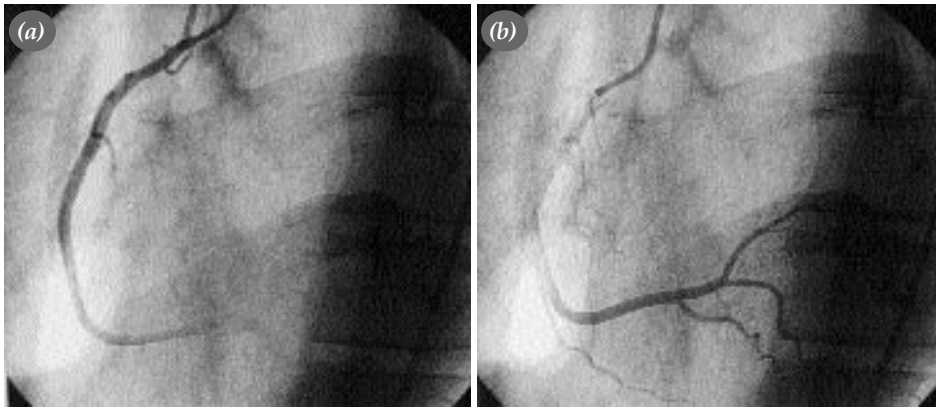

Fig. 1. (a, b) The slow progression of the dye in the right coronary artery in the left anterior oblique views.

the left anterior oblique projection showed that both the left anterior descending (LAD) and left circumflex (Cx) coronary arteries were normal. However, both the left and right anterior oblique views showed a marked antegrade filling of the right coronary artery (RCA) without any stenosis or spasm (Fig. 1). The degree of slow antegrade filling was assessed using the TIMI frame count method. The frame counts for the LAD and Cx were normal (34 and 24 frames, respectively), while it was 48 frames for the RCA. Medications with aspirin, nitroglycerin, and a beta-blocker were instituted, after which relief of symptoms was observed. His complaints did not recur during a follow-up period of a year.

DISCUSSION

Although acute coronary syndrome is mainly due to an obstructive atherosclerotic lesion, multiple nonatherosclerotic causes of angina pectoris and acute myocardial infarction have been reported. Also, in angiographic evaluation as many as 30% of patients presenting with angina pectoris can be found to have normal coronary arteries.[2,3] In this group of patients, SCF with otherwise normal epicardial coronary arteries represents a not negligible subgroup. However, whether or not SCF causes myocardial ischemia is still unclear. To our knowledge, there are only a few reports suggesting the clinical significance of this phenomenon. Coronary slow flow phenomenon was first described by Tambe et al.[1] in six patients with chest pain, of which four had typical and two had atypical angina. All these patients had an abnormal resting ECG; one might have had a previous inferior myocardial infarction, and three had ischemic exercise tests. This phenomenon was attributed to the abnormally high small-vessel resistance.[1] Later, Mangieri et al.[4] noted that slow flow of dye into main coronary vessels was not reversed by intracoronary nitroglycerin administration, but was normalized following dipyridamole intravenous infusion in patients with SCF, a finding that supported the role of high small-vessel resistance. More recently, Kurtoglu et al.[5] reported normalization of SCF after administration of 75 mg of dipyridamole three times daily for a month. Goel et al.[6] showed that definitively positive exercise test results were more common in SCF patients than in those with normal coronary flow. Moreover, in a previous study, we reported impaired vascular endothelial function in patients with SCF.[7] Cesar et al.[8] demonstrated ST depression in 11.7% of patients with SCF. Przybojewski and Becker,[9] reported myocardial infarction due to SCF in a 51-year-old woman with a previous history of palpitation and episodes of retrosternal pain. They observed severe angina pectoris and ST-segment elevation after the first injection, accompanied by slow flow of contrast in the LAD. Kapoor et al.[10] reported three cases with angina pectoris and SCF, in whom ST-segment elevation occurred after treadmill exercise testing in two, and during general anesthesia induction in one. Our patient was relatively young and there was no apparent factor such as surgical intervention to induce ST-segment changes.

Slow coronary flow phenomenon has a distinct significance within this spectrum of patients with chest pain and normal epicardial coronary arteries. Moreover, no optimal therapeutic approach exists for this distinct angiographic finding. It has been demonstrated that coronary flow returns to normal in patients with ST-segment elevation after the administration of dipyridamole[4,5] or sublingual nifedipine.[10] In this context, considering the role of microvascular abnormalities in the pathogenesis of SCF phenomenon, vasodilator agents such as calcium-channel blockers and/or dipyridamole may be recommended in the treatment of these patients. However, in our patient, ST-segment depression, not elevation occurred during exercise, and we preferred a beta-blocker due to its anti-ischemic properties.

In our opinion, SCF phenomenon is not purely an incidental angiographic finding; on the contrary, it may lead to angina pectoris and true myocardial ischemia.

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