A case of multivessel coronary ectasia resulting in myocardial infarction

Miyokard infarktüsüne yol açan çokdamar koroner ektazisi: Olgu sunumu

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Coronary ectasia is an infrequent angiographic lesion commonly seen in stenotic coronary arteries. It may result in angina pectoris, even in myocardial infarction due to impaired coronary blood flow. A 70-year-old woman presented with chest pain that occurred after physical activity and lasted nearly 30 minutes. There were no pathologic findings except for a 2/6-degree systolic murmur on the left sternal border. Electrocardiography showed T-wave inversions in leads III, AVF, and V1-V3. Her chest pain did not recur following medical therapy in the coronary intensive care unit. Coronary angiography performed with the diagnosis of acute non-ST elevation myocardial infarction revealed segmental ectasia in both the left anterior descending and right coronary arteries without any obstructive lesion. The patient was discharged with medical therapy. She was followed-up for six months without chest pain.

Key words: Coronary angiography; coronary vessel anomalies/complications; dilatation, pathologic; myocardial infarction/etiology.

Koroner arter ektazisi genellikle aterosklerotik koroner arter hastalığında görülen nadir bir anjiyografik lezyondur. Bozulmuş koroner kan akımına bağlı olarak angina pektorise, hatta miyokard infarktüsüne bile yol açabilir. Yetmiş yaşında kadın hasta, fiziksel aktivite sonrası oluşan ve yaklaşık 30 dakika süren göğüs ağrısı şikayetiyle başvurdu. Fizik muayenede, sol sternal sınırda 2/6 şiddetinde duyulan sistolik üfürüm dışında patolojik bulguya rastlanmadı. Elektrokardiyografide III, AVF ve V1-V3 derivasyonlarında T dalgası dalgalanmaları gözlendi. Koroner yoğun bakım ünitesinde uygulanan ilaç tedavisinden sonra hastada göğüs ağrısı olmadı. Akut ST yükselmesiz miyokard infarktüsü tanısıyla uygulanan koroner anjiyografide sol ön inen arter ve sağ koroner arterde segmental ektazi görüldü; herhangi bir tıkayıcı lezyona rastlanmadı. İlaç tedavisi verilerek taburcu edilen hastada altı aylık takip dönemi içinde göğüs ağrısı tekrarlamadı.

Anahtar sözcükler: Koroner anjiyografi; koroner damar anomalisi/komplikasyon; dilatasyon, patolojik; miyokard infarktüsü/etyoloji.

Coronary ectasia (CE) is an infrequent angiographic lesion commonly seen in patients with stenotic coronary arteries. It may result in angina pectoris, even in myocardial infarction due to impaired coronary blood flow. In this report, we present a patient who developed non-ST elevation myocardial infarction due to multivessel ectasia.

CASE REPORT

A 70-year-old woman was admitted to the emergency room with a complaint of chest pain that occurred after physical activity and lasted nearly 30 minutes. She described it as if something exerted a weight on the chest. Age, hypertension, and hyperlipidemia were present as risk factors for coronary heart disease. On physical examination, heart rate and arterial blood pressure were 98/min and 160/100 mmHg, respectively. There were no pathologic findings except for a 2/6-degree systolic murmur on the left sternal border. On her electrocardiogram, T-wave inversions were noted in leads III, AVF, and V1-V3 (Fig. 1). The patient was admitted to the coronary intensive care unit with the diagnosis of acute coronary syndrome. She was treated with salicylate, enoxoparine, clopidogrel, metoprolol, and intravenous nitroglycerin, during which no chest pain occurred. Cardiac troponin T, which was negative on admission, increased to 0.247 ng/dl at the 12th hour. She underwent coronary angiography with the diagnosis of acute non-ST elevation myocardial infarction. Coronary

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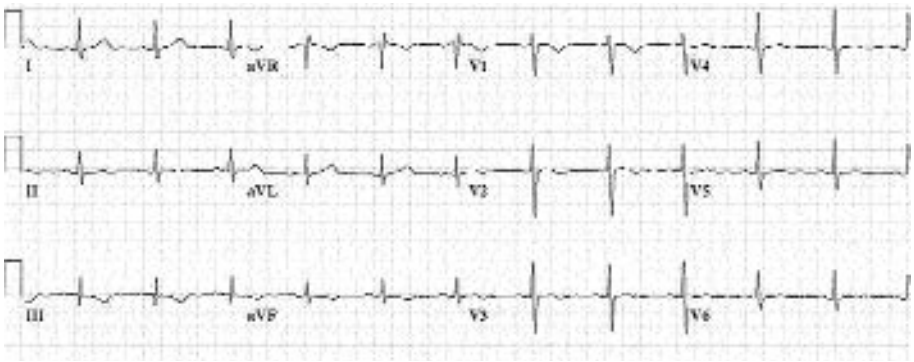

Figure 1. Electrocardiogram showing T- wave inversions in leads III, AVF, and V1-V3.

angiography revealed a diminutive circumflex artery, and segmental ectasia in both the left anterior descending (LAD) and right coronary (RCA) arteries without any obstructive lesion (Fig. 2). The patient was discharged with medical therapy. She was followed-up for six months without chest pain.

DISCUSSION

Coronary ectasia is defined as a ≥1.5-fold diffuse or localized luminal dilatation of the coronary artery compared to the diameter of normal segments. In most cases, CE is related to obstructive atherosclerotic coronary artery disease.[1,2] A uniform definition does not exist and the terms "ectasia" and "aneurysm" have been used inaccurately. It has been suggested to define the dilatation of the vessel as ectasia when the involvement is diffuse rather than segmental.[3] A luminal dilatation of 1.5 to 2.0-fold of normal diameters is regarded as CE, and an aneurysm is considered with expansion exceeding this range.[4,5] The incidences of CE and isolated CE without any other cardiac disorder vary in different studies. Pinar Bermudez et al.[6] evaluated 4,332 patients angiographically, of whom 147 patients had CE (3.4%), and 33 patients (0.7%) had CE with non-significant coronary stenosis. Most of the ectasia cases were men (91.2%) and patients with CE had a younger age.[6] Gulec et al.[7] evaluated 7,342 patients who underwent coronary angiography and reported the overall incidences of CE and isolated CE as 3.2% and 0.12%, respectively.

Markis et al.[8] classified CE in four types: type 1 includes diffuse ectasia involving two or three vessels, type 2 includes diffuse ectasia involving one vessel and discrete ectasia in another, type 3 includes diffuse ectasia in only one vessel, and type 4 includes localized or segmental ectasia in only one vessel.

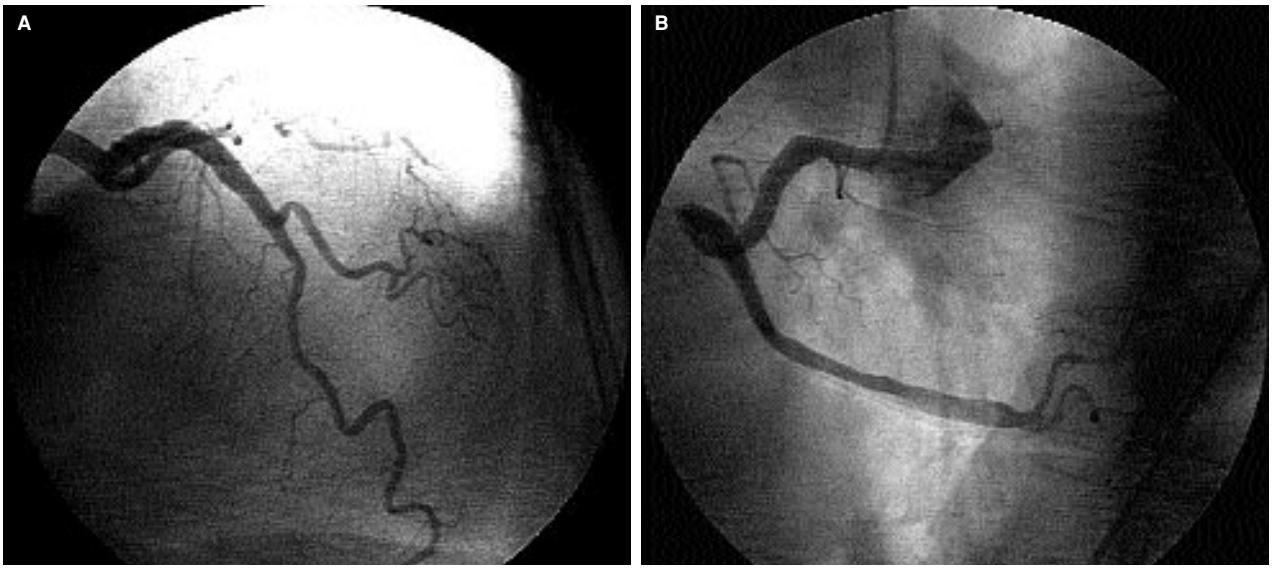

Figure 2. (A) Ectasia of the left anterior descending artery in an anteroposterior 40° cranial view (ectatic segment=3.6 mm, normal segment=1.9 mm). **(B)** Ectasia of the right coronary artery in the left anterior oblique view (ectatic segment=4.8 mm, normal segment=2.5 mm).

When our case was considered, the patient was somewhat atypical due to advanced age, female gender, and presence of multiple ectasia in two major epicardial coronary arteries without any obstructive lesion. Based on these findings, she was accepted as having type 2 CE.

Five-year follow-up of patients with CE showed similar mortality rates for patients with and without significant coronary artery stenosis. However, diffuse coronary ectasia and ectasia in the left main coronary artery were associated with higher mortality rates.[9] In patients with CE, coronary blood flow estimated with TIMI frame count (TFC) was slower.[10] In parallel with this finding, in our case, corrected TFC was 28.5 in the LAD, and 36.0 in the RCA.

The clinical spectrum of CE is variable, including stable angina pectoris, unstable angina pectoris, vasospastic angina, and myocardial infarction. The most common symptom is exertional angina.[7,8] Tendency to thrombosis due to diminished coronary flow and vasospasm due to structural changes in the vessel wall may cause chest pain and even myocardial infarction.[11] The mechanism of exercise-induced myocardial ischemia in isolated CE was investigated in two studies.[11,12] Kruger et al.[11] demonstrated evidence for exercise-induced myocardial ischemia through a coronary sinus stress test and an ergometric test in patients with nonobstructive, ischemic coronary artery disease (dilated coronaropathy). Signs of impaired coronary blood flow and evidence for ischemic response (considerable lactate production and significant ST-segment depression) were found more often with increasing coronary diameters.

Microvascular angina secondary to microvascular dysfunction has been proposed as another mechanism for positive stress tests and exertional angina in ectasia patients.[12] Endothelial dysfunction is known to be the leading cause for the development of microvascular dysfunction. Hence, blunted vasodilator response of the endothelium results in decreased coronary blood flow under the conditions requiring increased myocardial oxygen supply.[13]

In our case, collagen tissue diseases and malignancy, which are known to cause *in situ* coronary thrombosis, were excluded.[14] No abnormality in blood coagulation tests was detected, as well. The patient was asymptomatic before the diagnosis of non-ST elevation myocardial infarction, which was understandable with the advanced age and diminished physical activity of the patient. After the diagnosis of non-ST elevation myocardial infarction, she was treated with anticoagulant, antiaggregant, and anti-ischemic therapies. A calcium channel blocker (diltiazem) was applied in order to prevent coronary vasospasms and beta-blocker therapy was discontinued. An angiotensin converting enzyme inhibitor and a statin were added to treatment due to their therapeutic effects on the endothelial dysfunction. After discharge, she described no acute chest pain or chronic exertional angina under medical therapy for six months.

In conclusion, coronary ectasia should not be considered merely dilatation of the vessel wall, because it may lead to cardiovascular complications. It is one of the less frequent causes of myocardial infarction and management of this angiographic entity requires meticulous medical therapy.

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