

A case of myocardial muscular bridging causing severe hypotension during exercise-electrocardiography test

Egzersiz testi sırasında ciddi hipotansiyon gelişen miyokart köprüleşmesi olgusu

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Summary– Outlining the severity of the myocardial bridge (MB) is a critical step for selecting the appropriate option among medical, surgical, or angioplasty-based treatments. Invasive treatments are usually preferred if treatment-resistant symptoms are observed or ischemia is proven by tests such as fractional flow reserve or myocardial perfusion scintigraphy (MPS). In this report, we present a patient who developed severe hypotension during treadmill exercise test, even though there were no perfusion defects during adenosine-induced MPS. This case suggests MPS with adenosine is not a good choice for evaluating ischemia in MB patients, as it may cause false negative results.

Treadmill exercise test is frequently used for investigating coronary artery disease (CAD) in patients with typical or atypical chest pain. Exercise-induced hypotension as well as electrocardiographic changes during the test may also indicate cardiac diseases (hypertrophic obstructive cardiomyopathy, aortic stenosis, etc.) rather than CAD. Angiographically proven severe myocardial bridging (MB) is associated with severe hypotension, angina, and arrhythmia.^[1]

Herein, we present an MB patient who developed serious hypotension during treadmill test, although previous adenosine-induced myocardial perfusion scintigraphy (MPS) did not reveal any perfusion de-

Özet– Tıbbi, cerrahi ve anjiyoplastiye dayalı tedavi seçeneklerinden uygun olana karar verebilmek için kritik adım miyokart köprüleşmesinin (MK) ciddiyetini belirlemektir. Genellikle tedavide invaziv yöntemler; fraksiyonel akım rezervi ya da miyokart perfüzyon sintigrafisi (MPS) gibi yöntemlerle iskemi kanıtlanırsa veya tedaviye dirençli semptomların varlığında tercih edilir. Bu yazıda, adenosin ile yapılmış MPS’de perfüzyon defekti saptanmamasına rağmen koşu bandı egzersiz testinde ciddi hipotansiyon gelişen bir hastayı sunuyoruz. Bu olgu adenosinli MPS’nin, MK’si olan hastalarda iskemi değerlendirmesi için iyi bir seçenek olmaya-çağını düşündürmektedir.

fect. We also discuss the effectiveness of adenosine stress for inducing ischemia in patients with MB.

CASE REPORT

A 55-year-old-man was admitted to the emergency room with palpitation and presyncope. Physical examination findings were unremarkable. The electrocardiogram and cardiac biomarkers were normal, as were results of transthoracic echocardiography. His medical history indicated an atypical angina attack one month prior, but a perfusion defect had not been detected by adenosine-induced MPS. Treadmill test was performed, and at the end of the third stage, the patient developed severe hypotension (50/30 mmHg), dyspnea and near-syncope status. Ischemic ST-T wave changes were absent. The patient was hospital-

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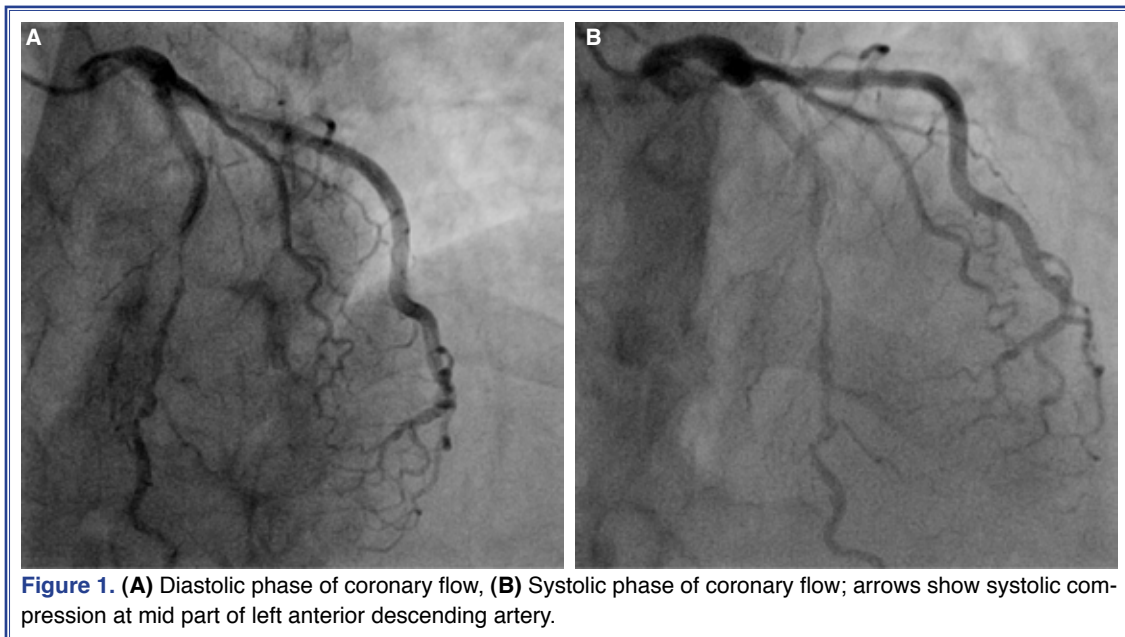


Figure 1. (A) Diastolic phase of coronary flow, **(B)** Systolic phase of coronary flow; arrows show systolic compression at mid part of left anterior descending artery.

ized. Cardiac biomarkers were normal at the 6th and 12th hours post-treadmill test. Coronary angiography revealed typical milking image (Figure 1) of MB at the mid part of the left anterior descending artery. The patient was prescribed metoprolol and acetylsalicylic acid before he was discharged without any symptoms on Day 3 of hospitalization. He was asymptomatic during the 2-month follow-up period.

DISCUSSION

Our patient exhibited hypotensive response during treadmill test, even though there was no perfusion defect on MPS. This phenomenon may be explained by increased systolic compression and shortened diastolic perfusion period (during tachycardia, decreased intracoronary pressure, hypovolemia), which increase the severity of MB due to its dynamic pathophysiology. In a case report, it was suggested that exercise results in a greater venous return, which leads to a more forceful myocardial contraction; as a result, a longer occlusion period would be promoted on the MB, and this causes an imbalance between the relaxation period and ventricular contraction period, consequently reducing cardiac output.^[2]

Another issue is the appropriateness of adenosine stress-induced MPS to detect ischemia in MB cases. Several adenosine receptors were identified over the last decade; some of them have negative chronotropic and dromotropic effects by modulating sinoatrial and

atrioventricular nodal conduction, while others induce coronary vasodilation.^[3] Previously, perfusion scintigraphy with dipyridamole was used to detect the severity of MB.^[4] To the best of our knowledge, use of adenosine-induced scintigraphy has not been reported in MB cases. The specificity or sensitivity of adenosine-induced scintigraphy in the diagnostic accuracy of MB is unknown. Adenosine may overshadow the severity of MB, since negative chronotropic and dromotropic effects counter the tachycardia-inducing effect. In our case, we could not detect any perfusion defects, while we did encounter a severe hemodynamic collapse. It is difficult to decide on the criteria for determining MB severity. Additionally, it was challenging for us to determine the more significant test result (severe hypotensive response in treadmill test or normal perfusion in MPS) in terms of follow-up treatment. We decided to treat the patient until all symptoms were alleviated.

Hypotensive response to exercise test is an important finding for the diagnosis and prognosis of CAD. Hypotensive response is also an absolute indication for coronary angiography after treadmill exercise test. Although an ischemic area could not be detected during MPS, a severe MB may mimic CAD. MB can be listed as a rare cause of abnormal hypotensive response to exercise test. After diagnosis of MB, individualized imaging methods should be used to determine treatment and follow-up strategy. In our

opinion, MPS with adenosine is not a good choice for evaluating ischemia in MB patients, as it may cause false negative results.

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