

Muscular bridge causing non- ST-segment elevation myocardial infarction

ST-elevasyonsuz miyokard infarktüsüne neden olan miyokardiyal köprüleme

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Introduction

Coronary arteries and their major branches are usually located sub-epicardially. Some individuals, however, have regions in which a bunch of cardiac muscle fiber passes over those vessels like a bridge. This anatomical structure, called myocardial or coronary arterial bridge, is responsible for the narrowing of the artery at each systolic contraction. The left anterior descending coronary artery (LAD) is the vessel involved in majority of the cases. Muscular bridge may alter the hemodynamics of the coronary circulation in susceptible individuals.

Muscle bridges are more common in men than in women and tend to affect patients in their fourth decade of life (1). The prevalence rate of muscular bridges in angiographic studies is ranged from 0.5 to 33% (2). The clinical significance of myocardial bridges varies, and most patients are asymptomatic. However, angina, ventricular fibrillation, cardiac arrhythmias, and sudden death have been reported in association with myocardial bridges (3). Myocardial bridging rarely causes myocardial ischemia (4).

Case Report

A 43-year-old man was admitted to our emergency department with dyspnea and exertional chest pain. He had experienced shortness of breath and chest pain with effort for about four years. Four years ago, he underwent cardiac catheterization, which revealed normal coronary arteries. He was a non-smoker and normotensive. He had a family history of coronary artery disease and hypertension.

His physical examination was without pathological findings except cold sweating. His systolic and diastolic blood pressures were 100 and 60 mmHg respectively. Heart rate was noted as 68 beats per minute. Electrocardiography showed sinus rhythm without any sign of ischemia. Blood samples were carried out. At the beginning, cardiac enzymes were within normal limits. Triglyceride level was found to be 520 mg/dl. The patient was evaluated as unstable angina pectoris and anti-anginal therapy was begun at

admission. Six hours later, cardiac enzymes were found to be significantly elevated (Creatine kinase (CK): 1593 U/L (0-200 U/L), CK-MB: 97 U/L (0-42 U/L) and Troponin-I: 5.3 ug/l (0.1-0.8 ug/l)). Non-ST elevated myocardial infarction (MI) was diagnosed.

Coronary angiography revealed suspicion of thrombosis and 95 % luminal narrowing by systolic compression in the mid segment of the left anterior descending coronary artery at left anterior oblique cranial position (Fig. 1 and 2). Right coronary artery and left ventriculography were normal. Single photon emission computed tomography (SPECT) imaging with thallium-201 was performed. Moderately extended, mildly severe (+1) reversible ischemic defect was reported.

The patient was prescribed metoprolol, statin, nitroglycerine and acetylsalicylic acid. During the clinical follow-up parenteral diltiazem was also given because of the short term sinus tachycardia.

Discussion

Myocardial bridges are relatively uncommon congenital anomalies of coronary arteries recognized by the characteristic angiographic 'milking effect' or systolic compression of a discrete coronary segment (1). Although there are few reports of right coronary artery involvement, LAD is the vessel affected in the majority of cases (5).

Several studies have shown that the phasic systolic vessel compression of the coronary artery persists as a vessel diameter reduction into diastole. This incomplete relaxation of the bridge during diastole results in increased intracoronary flow velocities, reduced diastolic coronary flow, retrograde coronary flow, and a reduction in coronary flow reserve, resulting in a lowered ischemic threshold (6).

It is well known, that the main pathogenic features of acute coronary syndromes consists of atherosclerotic plaque disruption and thrombus formation (7). Induction of ischemia solely by a myocardial bridge has been demonstrated and different underlying mechanisms such as thrombus formation, vasospasm, en-



Figure 1. Angiographic demonstration of systolic compression of the mid-portion of the left anterior descending coronary artery by the muscle bridge

dothelial dysfunction or impaired coronary flow reserve have been proposed to explain this (8). It has also been suggested that myocardial bridges are involved in the development of atherosclerosis (8). The severity of symptoms induced by myocardial bridges has been related to the localization of the bridge, its length and depth, and the presence of left ventricular hypertrophy or an increased intraventricular pressure (8). Although most patients are asymptomatic, common symptoms associated with muscle bridging can range from angina pectoris to myocardial infarction, ventricular tachycardia and sudden death (9).

Myocardial bridges can be an incidental finding at the time of coronary angiography. As a rule, a significant "milking effect" is associated with 70% lumen diameter reduction during systole and 35% lumen diameter reduction during mid-to-late diastole (10).

Although the underlying pathogenesis of acute coronary syndrome consists of atherosclerotic plaques and thrombus formation, in the present patient the reason was muscular bridging. The results of endothelial injury might come out with a myocardial infarction. This patient was not a smoker, but he has a family history of coronary artery disease and he had hypertriglyceridemia. In this patient the explanation of acute non-ST-elevation MI might be endothelial injury and severe vasospasm. There were no atherosclerotic lesions in the major coronary arteries on coronary angiography. Cardiologists had a suspicion of thrombus formation in the mid portion of LAD but an exact diagnosis could not be made. There was a systolic coronary arterial luminal narrowing at the same level with the suspected thrombus. The patient was decided to be followed-up with medical therapy.

This is a case of acute coronary syndrome caused by coronary vasospasm in the setting of myocardial bridging. Acute isc-

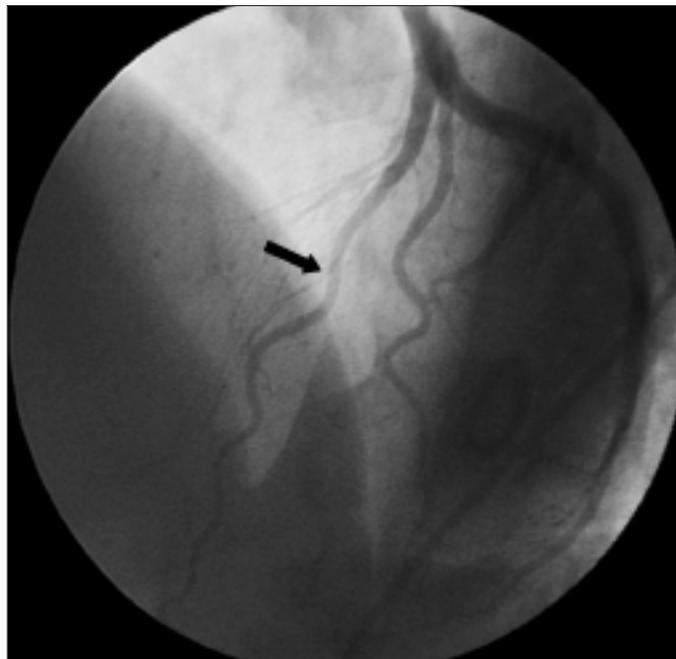


Figure 2. Myocardial bridging of left anterior descending coronary artery during diastole

hem complications associated with myocardial bridging are resolved by beta-blockers, acetylsalicylic acid, nitroglycerine and statins.

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