Myocardial bridge: An unusual cause of asymptomatic ST-elevation during treadmill stress test causing functional ischaemia

Ioannis Karfis[1] MD, Christos Dresios[2] MD, Irini Kotsalou[1] MD, PhD, Mercurios Voyatzis[1] MD, Dimitrios Antoniadis[2] MD, Nikolaos Dimakopoulos[1] MD, PhD, FEANM-FESC

1. Nuclear Medicine Department and
2. Interventional Cardiology Department, 417 NIMTS Hospital, Athens, Greece

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Correspondence address:
Ioannis Karfis MD, 417 NIMTS Hospital, Nuclear Medicine Department Monis Petraki 10, 11521, Athens, Greece
Tel: +30 2107288380
E-mail: ykarfis@gmail.com

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Abstract

Myocardial bridges are congenital structural abnormalities with debated clinical relevance; they may be traditionally asymptomatic or associated with various clinical manifestations, some of which may be potentially fatal. We report on a 60 years old man, who underwent a follow-up stress/rest myocardial perfusion scintigraphy developing asymptomatic exercise-induced ST-elevation and demonstrating completely reversible ischaemia in the scintigraphic images. Subsequent coronary angiography showed no artery stenosis, but revealed an intramyocardial pathway of the left anterior descending artery throughout a myocardial bridge. *In conclusion*, to our knowledge, this is the first reported case of asymptomatic exercise-induced ST-elevation secondary to a myocardial bridge.

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Introduction

Myocardial bridges and loops are usually clinically silent congenital structural abnormalities, defined by an intramyocardial course of a major epicardial artery segment. Since perfusion occurs mostly in diastole, it is with each systole, that this tunnelled artery segment is compressed by contracting myocardium, inducing a phasic "*milking effect*" and a "*step down-step up*" phenomenon, which are characteristic angiographic findings in the setting of a myocardial bridge. Milking was initially thought as a solely systolic event and therefore with no significant haemodynamic impact on myocardial perfusion. However quantitative intravascular ultrasound studies demonstrated that vessel compression within the bridge is not limited to systole, but rather persists throughout a large part of diastole affecting thereby the predominant phase of coronary perfusion and causing functional ischaemia [1].

Numerous reports indicate a close association between a myocardial bridge and various clinical manifestations such as angina [2], acute coronary syndromes [3], arrhythmias including ventricular tachycardia [4], stunning [5], tacotsubo cardiomyopathy [6] or even sudden death [7]. Bridging has also been related to perfusion defects documented on myocardial scintigraphy [8]. Hereby, we describe a case of asymptomatic exercise-induced ST-elevation secondary to a myocardial bridge, with positive scintigraphic findings, the first such reported case according to our knowledge.

Case report

A 60 years old man with a history of percutaneous transluminal coronary angioplasty in the left anterior descending (LAD) and the right coronary artery (RCA) due to a non ST-elevation myocardial infarction (NSTEMI) 12 months before was referred to the department of Nuclear Medicine in order to perform a stress/rest technetium-99m-tetrofosmin (^{99m}Tc-T) myocardial perfusion scintigraphy. He was overweight, with no history of diabetes, hypertension or dyslipidaemia and no family history of coronary artery disease. Scintigraphy was performed as part of his follow-up treatment, a year after his NSTEMI.

Treadmill stress test was performed using the Bruce protocol while heart rate, blood pressure and 12-lead electrocardiogram (ECG) were monitored. The ST segment developed progressive elevation >1mm in V2-V3 leads, urging for early termination of the test at the third stage of Bruce protocol (total exercise duration: 8:40min, maximal achieved heart rate: 152min^{-1}, peak blood pressure: 180/90mmHg, METs: 10.1) and after intravenous administration of 295MBq of ^{99m}Tc-T. At early recovery we noticed that ST elevation worsened (>2mm in leads V1-V4) but progressively reversed until complete disappearance in about the 10thmin of recovery phase (Fig. 1). The patient received sublingual nitroglycerin, and he was admitted to cardiological intensive care unit for further observation. Throughout the whole procedure, he

was asymptomatic and haemodynamicaly stable. Monitored ECG remained free of any new changes and markers of myocardial injury were tested negative with no serial evolution. Echocardiography showed no ventricular wall motion abnormalities with a left ventricular ejection fraction (LVEF) value of 65%. Stress perfusion images were acquired 60min after ^{99m}Tc-T administration, and rest scintigraphy was programmed for the following day, and performed 30min after the injection of 295MBq of the same radiopharmaceutical. Single photon emission tomography (SPET) data were obtained using a large-field-of-view, double-headed γ-camera with high resolution collimators and a 64x64 matrix.

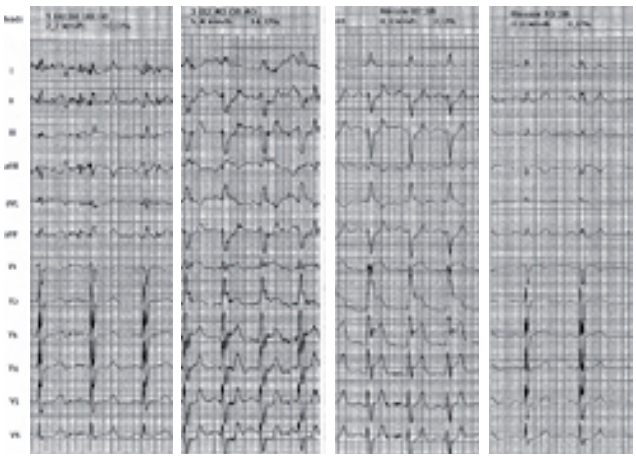

Figure 1. ECG during stress test: a) baseline (rest) trace, b) 3rd stage of Bruce protocol, 8:40min: early termination of the test because of ST-segment elevation in precordial leads. Patient asymptomatic. c) Recovery phase, 0:38min: worsening of ST-segment elevation. Patient still asymptomatic. d) Recovery phase, 10:38 min: normalisation of ST-segment.

Image processing and comparison revealed severe ischemia in the anterior, septal, apical and inferior myocardial wall at stress images, which was completely reversed in the respective rest images suggesting possible occlusion (or re-stenosis) of LAD and RCA (Fig. 2). However, coronary angiography performed few hours after the rest scintigraphy, evidenced no obstructive signs of previous interventions or in any other location. Careful reading of angiography images revealed a systolic compression effect (''*milking*'') in a LAD segment coursing through a myocardial bridge (Fig. 3) Remission of compression with restoration of blood flow was observed in the diastolic phase (Fig. 4).

The patient was discharged being treated with a β-blocker because of his coronary disease and with instructions to avoid activities requiring heavy effort.

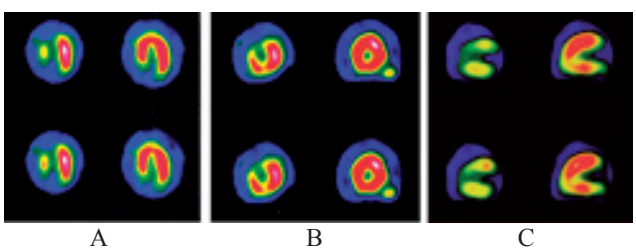

Figure 2. SPET images at stress (left column) and rest (right column) in transverse (**A**), coronal (**B**) and sagittal (**C**) axes, demonstrating severe but completely reversed ischemia in the anterior, septal, apical and inferior myocardial wall.

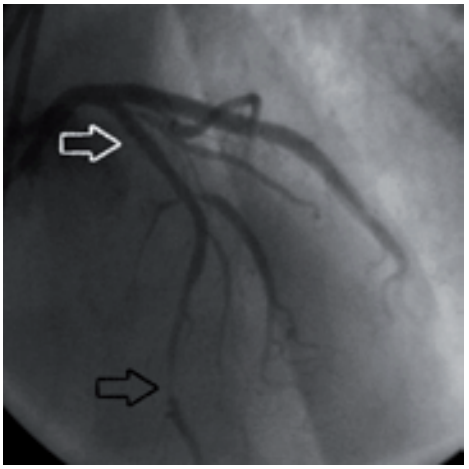

Figure 3. Left anterior oblique coronarography view in systole: Systolic compression in distal LAD due to a myocardial bridge (black arrow). White arrow indicates previous stent location in proximal LAD.

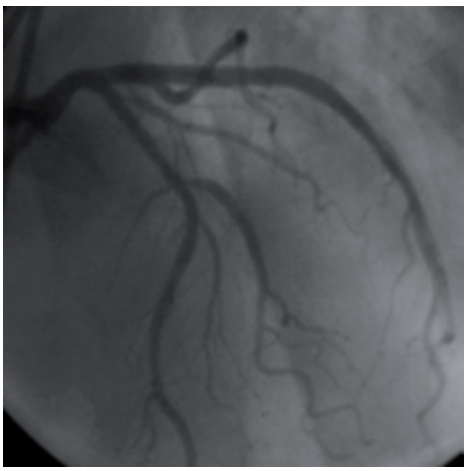

Figure 4. Left anterior oblique coronarography view in diastole: Remission of compression with restoration of blood flow.

Discussion

Exercise-induced ST-segment elevation during ergometric treadmill test, an uncommon finding by itself, occurs more often in patients with previous myocardial infarction (evidenced by the presence of Q waves on the ECG at rest) suggesting left ventricular wall motion dyssynergy (dyskinesia) or aneurysm [9]. In patients without Q waves on baseline ECG, as in the present case, this finding is thought to be related to ischaemia, caused by critical or subcritical coronary stenoses, as well as coronary artery spasm [10, 11], with aVR lead presenting substantial particularities [12].

Ischaemia evidenced in the ECG trace and confirmed by stress scintigraphy, was provoked by bridge-related vessel compression affecting the diastolic phase of myocardial perfusion. Moreover, the enhanced sympathetic drive during the exercise test aggravate ischaemia, since the resulting increased chronotropic and inotropic stimulation led to an increased systolic/diastolic time ratio at the expense of the diastolic flow; perfusion time is then further compromised. In addition, the restored coronary blood flow following angioplasty led to an improvement of myocardial wall motion, enhancing thus bridge visualisation. Careful reviewing of

coronarography images can increase the detection frequency of bridges from 1.7% to 9.7% [13].

Reports on ST-elevation associated with chest pain during an ergometric stress test, attributed to a myocardial bridge, have been recently published [14]. Myocardial bridges are almost exclusively spotted in the left anterior descending artery, whereas in the presence of two parallel LAD branches, one frequently takes an intramural course [15].

Since there was no evidence of any artery occlusion in the coronarography, the coexisted, fully reversible ischemia in the inferior myocardial wall was attributed to vasospasm or endothelial microvascular dysfunction [16].

Several therapeutic approaches have been considered for myocardial bridging including β-blockers, calcium-channel blockers, stents, minimally invasive coronary artery bypass grafting and surgical myotomy. However, percutaneous and surgical treatment did not achieve significant success. Therefore, drugs are currently preferred as medical treatment.

In conclusion, we present a case with arterial myocardial bridge as novel cause of asymptomatic exercise-induced ST-segment elevation. Although myocardial arterial "bridges" are often clinically silent, they can also induce dramatic ECG changes and positive scintigraphic findings without coronary arteries stenosis.

The authors declare that they have no conflicts of interest.

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