## **Case Report**



# Multi-arterial Myocardial Bridge: Uncommon Clinical and Anatomical Presentations

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We report the case of a 42-year-old man with no risk factors for coronary artery disease admitted with atypical chest pain. The electrocardiogram performed after intravenous injection of nitrate revealed ST-segment elevation in leads V1 to V4. The coronary angiography showed myocardial bridges in the three coronary arteries, besides an unusual length of the left anterior descending artery (80 mm). The patient progressed well following the discontinuation of nitrate use and introduction of beta-blockers and calcium channel antagonists.

## Introduction

Despite several series reporting the clinical progress of patients with myocardial bridges, little is known as to whether this entity is benign or not. There is even considerable disagreement about its incidence, with discrepant results when angiographic findings are compared to pathologic diagnoses. Although ultrasound and intracoronary Doppler techniques have enhanced the knowledge of ischemia mechanisms, these have not yet been completely explained. We intend to report the case of a myocardial bridge with uncommon clinical and anatomical presentations.

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A 42-year-old male patient was admitted to the cardiology emergency room with a complaint of mild continuous precordialgia over the previous 12 hours, without irradiation, triggered by physical exertion and resistant to common analgesics. The patient had no risk factors for coronary heart disease. On examination, his arterial blood pressure was 135/85 mmHg and the heart rate was 82 bpm. Segmental evaluation did not show any abnormalities.

The electrocardiogram showed sinus rhythm and T-wave inversion in leads DI and aVL. CK-MB activity was normal, whereas troponin was slightly elevated. The patient was started on 1 mg/kg/12h enoxaparin, 200 mg aspirin, 600 mg clopidogrel, and nitroglycerin. After initiation of intravenous nitrate infusion, the patient reported worsening of chest

## **Key words**

Coronary artery bypass; coronary angiography, nitroglycerin, myocardial ischemia.

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discomfort; another electrocardiogram was performed which revealed ST-segment elevation in leads V1 to V4.

Emergency coronary angiography showed severe systolic arterial lumen stenosis of the long segment of the anterior descending artery (80 mm), septal branches, distal portions of diagonal and marginal branches of the circumflex artery, as well as slight systolic narrowing of the arterial lumen in the acute marginal branch of the right coronary (Figure 1). Left ventriculography showed that the contractile function was preserved. Intravenous nitroglycerin was discontinued and the patient was started on an intravenous beta-blocker associated with an oral calcium channel antagonist. The patient progressed asymptomatically with resolution of the ST-segment elevation. Doppler echocardiography showed no anatomical or functional abnormalities. The patient was discharged with a regimen of 200 mg aspirin, 100 mg metoprolol, and 240 mg diltiazem.

Later the patient underwent coronary angio-tomography that showed muscle bundles involving the coronary arteries described above, including the elongated anterior descending artery (80 mm), though not very deep (Figure 2).

Thirty days after the initiation of clinical treatment, the patient was submitted to myocardial perfusion scintigraphy associated with an ergometric stress test, but no ischemic areas were observed. Due to the double blockade, the patient only achieved 74% of his maximum heart rate, although with excellent aerobic capacity.

The previously described clinical treatment was maintained and the patient was allowed to start a physical activity program under supervision.

## **Discussion**

We believe this is the first report ever of a myocardial bridge involving the diagonal and septal branches of the anterior descending artery, the marginal branch of the circumflex artery, and the acute marginal branch of the right coronary associated with a rather unusual lengthening of the anterior descending artery (80 mm) manifested as high-risk unstable angina.

In reviewing 2,000 consecutive coronary angiographies performed at our unit, we found an incidence of 8.2% cases of myocardial bridges. We believe this high incidence is due to the nitrate routinely used in diagnostic coronariographies performed using the trans-radial route, which is the case for most of our patients. Despite the mean incidence of myocardial bridge detected by diagnostic coronary angiography being around 2%, when a provocation test is used, such as the infusion of nitroglycerin, acetylcholine, or papaverine, the

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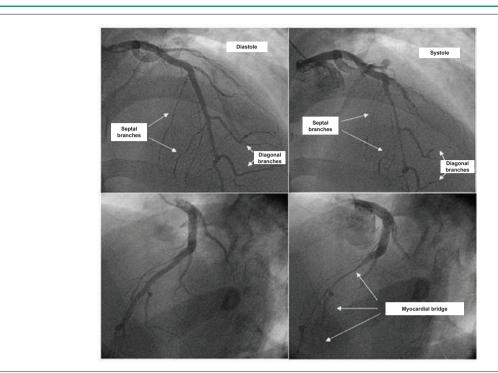


Fig. 1 - Long myocardial bridge in the anterior descending artery and involvement of septal branches and distal portions of diagonal branches.

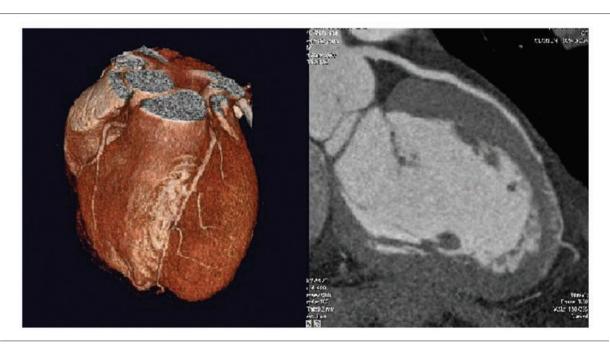


Fig. 2 - Small depth of the anterior descending artery in the myocardium as opposed to the marked systolic narrowing detected by coronary angiography.

incidence may reach up to 40% of the cases<sup>2</sup>. The angiographic extent of myocardial bridges in the anterior descending artery observed in several series is between 11 and 44 mm<sup>3</sup>.

Similar to other previously reported cases, the physiopathology of the clinical syndrome in this patient is not very clear. One hypothesis is the link between myocardial bridges and coronary spasms. In a study using acetylcholine

infusion in the left coronary arteries of patients with myocardial bridges versus patients with normal coronary arteries, spasms were more frequently observed in the group with myocardial bridges (73% X 40%, p=0,0006), and these spasms are capable of triggering ischemic syndromes<sup>4</sup>. Transient or prolonged thrombotic phenomena as a cause of acute coronary syndromes have also been described in patients

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with myocardial bridges<sup>5</sup>. In our case, however, the most likely cause of the unbalance between oxygen supply and oxygen consumption was the continuous infusion of nitroglycerin. Hongo et al<sup>6</sup> used intracoronary ultrasound to study the response to nitroglycerin in 39 patients with myocardial bridges. They were able to clearly show a reduced coronary lumen under the myocardial bridge during systole, and a prolongation of this reduction during the initial phase of the diastole. This delay in coronary lumen dilation during diastole may account for the occurrence of myocardial ischemia<sup>6</sup>.

Some studies tried to correlate the marked degree of systolic narrowing and the greater intramyocardial depth of the tunneled coronary segment as poor prognosis factors in patients with myocardial bridges; these authors even tried to associate these factors with sudden death by means of autopsy studies<sup>7,8</sup>. Due to the failure to demonstrate the depth of the myocardial coronary by means of coronary angiography, our patient underwent coronary angio-tomography. The coronary arteries were only partially involved by myocardial fibers, contrasting with the significant systolic narrowing observed.

In symptomatic patients, clinical treatment with betablockers and/or calcium channel antagonists is recommended<sup>2</sup>. Percutaneous intervention (stenting)<sup>9</sup> or surgical treatment (myotomy or myocardial revascularization surgery)<sup>10</sup> is indicated only for a minority of patients with symptoms or positive functional tests who are under optimized clinical treatment.

In retrospective studies, all of them conducted with small numbers of patients, a benign clinical progression is suggested for patients with myocardial bridges. However, reports on angina, coronary spasms, ventricular arrhythmias, acute myocardial infarction, and sudden death among patients with myocardial bridge indicate the existence of a subgroup of patients with a higher risk for adverse events<sup>1</sup>. The lack of controlled prospective studies makes it difficult to establish definite prognostic factors for this condition.

In this case, the asymptomatic progression associated with negative myocardial perfusion scintigraphy reinforces the success of clinical treatment so far.

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#### Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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