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Multivessel myocardial bridge involving left and right coronary arteries

Ponte miocárdica multiarterial comprometendo as coronárias esquerda e direita

José Augusto Rocha Araújo[1], Renato Áttila de Macedo Souza[1], Fabricio da Nóbrega Carvalho[1], Frederico Augusto de Lima e Silva[1]

[1] *Hospital de Messejana Dr. Carlos Alberto Studart Gomes, Fortaleza, CE, Brazil.*

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ABSTRACT - A 62-year-old female patient with hypertension and a 4-month history of angina pectoris, triggered by emotional stress. The electrocardiogram showed left ventricular hypertrophy and changes in ventricular repolarization. The coronary angiography showed a significant left ventricular hypertrophy with no intracavitary gradient, and the presence of a myocardial bridge over the middle third of the left anterior descending artery and the right coronary artery in its posterior descending branch. Myocardial bridge almost exclusively occurs in the left anterior descending artery. Reports of a myocardial bridge involving the left circumflex artery are rare, and even rarer is the involvement of the right coronary artery.

Keywords: Myocardial bridge; Microvascular angina; Coronary vessels/anatomy & histology

RESUMO – Paciente de 62 anos de idade, sexo feminino, portadora de hipertensão arterial e história de angina do peito, desencadeada por estresse emocional há 4 meses. O eletrocardiograma mostrava hipertrofia ventricular esquerda e alterações da repolarização ventricular. A cineangiocoronariografia mostrou importante hipertrofia ventricular esquerda sem gradiente intracavitário e a presença de ponte miocárdica comprometendo o terço médio da artéria descendente anterior e da coronária direita, em seu ramo descendente posterior. A ponte miocárdica tem como localização quase exclusiva a artéria descendente anterior. Relatos comprometendo a artéria circunflexa são raros e mais ainda a coronária direita.

Descritores: Ponte miocárdica; Angina microvascular; Vasos coronários/anatomia & histologia

INTRODUCTION

A myocardial bridge (MB) is a coronary anatomical variation, defined as an intramyocardial course of an epicardial coronary vessel.[1] It was recognized by autopsy, in 1737 by Reyman and first described angiographically by Portmann and Iwig, in 1960.[2] The frequency described in angiographic studies ranges from 0.5 to 16%. The lower frequency of MB observed angiographically contrasts with autopsy studies, which describe a frequency of 40 to 80%.[3] On average, MB is thought to be present in one-third of adults.[4] A high prevalence has been described in heart transplant patients and in patients with obstructive hypertrophic cardiomyopathy.[5,6] Traditionally, MB has been described as a benign condition, but it may cause myocardial ischemia, coronary spasm, acute coronary syndrome, arrhythmias, and transient ventricular dysfunction, and also sudden death has been associated with MB.[2]

The objective of this report was to present a rare case of a patient with an MB involving simultaneously the left anterior descending coronary artery and the right coronary artery.

CASE REPORT

A 62-year-old female patient, who was followed up for hypertension for 15 years. In the last 4 months, the patient presented intense burning precordial pain, radiating to the neck region, lasting approximately 2 minutes, triggered by emotional

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Corresponding author:
José Augusto Rocha Araújo
Rua Vicente Leite, 497/300 – Meireles
Zip code 60170-150
Fortaleza, CE, Brazil
E-mail: jaraujobr@yahoo.com

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stress, with spontaneous improvement and no relation to physical effort. The symptom has remained stable throughout this period. She makes regular use of 50mg captopril daily and 25mg hydrochlorothiazide daily. Upon clinical examination, blood pressure 131×83mmHg, and heart rate of 76bpm. Other clinical findings were normal. Her mother died suddenly at age 85. Two siblings have type 2 diabetes mellitus.

The electrocardiogram showed normal sinus rhythm, a heart rate of 75bpm, and left ventricular hypertrophy, with diffuse changes in ventricular repolarization (Figure 1). An exercise stress test was performed and considered abnormal, with ST-segment depression of 2 to 3mm at stage 2 of the Bruce protocol, with a descending ST-segment, which disappeared in the recovery period. The control electrocardiogram after the exercise test was the same as the baseline. No symptoms were reported.

The coronary angiography showed significant hypertrophy of the left ventricle, but with no intracavitary gradient. The left and right coronary arteries were free of obstructive lesions, but the left anterior descending artery presented significant systolic constriction in the middle third — around 80%. The right coronary artery showed a large posterior descending branch, the middle segment of which occluded completely during systole (Figure 2). After an angiographic study, we opted for therapeutic optimization, with the addition of a beta-blocker (25mg atenolol twice daily) and a calcium channel blocker (20mg nifedipine twice daily). The symptoms disappeared since then, with the patient under 6-month outpatient clinic follow-up.

Figure 1. Resting electrocardiogram showing left ventricular hypertrophy with changes in ventricular repolarization.

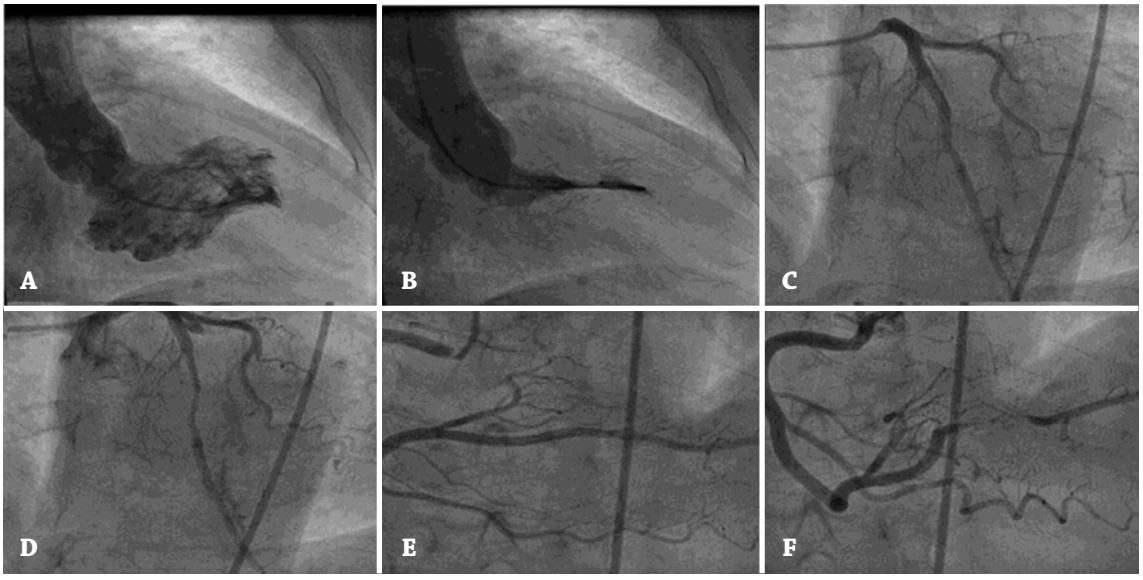

Figure 2. Coronary angiography. (A and B) Left ventricle in diastole and systole, with ventricular cavity obliteration, characterizing significant myocardial hypertrophy; (C and D) left anterior descending coronary artery, in diastole and systole, respectively, with significant systolic constriction in the middle third; (E and F) right coronary artery and its posterior descending branch, during diastole and systole, with severe compression of middle third segment.

DISCUSSION

MB is more commonly found in the middle third of the left anterior descending coronary artery,[4] and the typical angiographic finding is a systolic constriction of the epicardial arterial segment. The occurrence of MB in other coronary segments is rare. A recent report describes the case of a patient with MB involving the left main coronary artery, the left anterior descending artery and the left circumflex artery.[7]

The incidence of MB in the right coronary artery is quite rare and, when it occurs, it usually involves the posterior descending branch,[8] and this is often associated with myocardial hypertrophy.[9] The present case involves the left anterior descending artery and the right posterior descending artery, with typical myocardial bridging images. Although the clinical and electrocardiographic findings are nonspecific, angiographic findings are rare, and, as far as we know through literature review, there is no similar case in the medical literature, therefore this report is probably the first to be published.

Coronary angiography is considered the gold standard diagnostic method because it shows dynamic images of the segmental compression of the vessel during cardiac systole.[4] However, the angiographic finding provides little or almost no information from a functional point of view.[10] New diagnostic modalities, such as coronary computed tomography, intravascular ultrasound and coronary flow reserve, enabled better anatomical and functional analyses of the consequences of systolic compression, including coronary flow effects.[10] Bourassa et al.[11] analyzed patients with MB and myocardial ischemia and concluded that two mechanisms are responsible for the symptoms: (1) phasic compression of the vessel during systole associated with persistent reduction of diastolic diameter; (2) increased flow velocities, retrograde systolic flow, and reduction of coronary flow reserve, detected by intracoronary Doppler.

Although MB is most often considered a benign condition, its association with angina-type chest pain, coronary spasm, myocardial ischemia, acute coronary syndrome, ventricular dysfunction, and even sudden death has been occasionally reported.[12]

In symptomatic patients, therapy may be attempted to improve quality of life, although there is no evidence of a favorable effect on morbidity and mortality.[4] Treatment consists primarily of pharmacological therapy, although percutaneous coronary intervention, myotomy, and myocardial revascularization surgery may be considered in highly selected cases of patients refractory to optimized medical therapy.[13] Aggressive modification of risk factors and the use of antiplatelet agents should be considered because of the risk of atherosclerosis.[13] In symptomatic patients, the use of beta-blockers remains the first option, because they decrease heart rate, increase the diastolic perfusion period, and reduce the contractility and compression of coronary arteries.[14] Calcium channel blockers are frequently used, in addition to beta-blockers, for their vasodilator effect in patients with possible concurrent vasospasm. Pure vasodilators, such as nitrates, are not indicated because they may worsen symptoms due to increased systolic compression of the tunneled artery, tachycardia and dilation of the proximal vessel, which may aggravate the reversal of flow in the coronary artery in the proximal segment of the MB.[15]

Percutaneous coronary intervention with stent implantation in MB symptomatic patients can normalize coronary flow and eliminate symptoms.[16] However, problems such as perforation during release,[17] stent fracture,[18] intrastent restenosis,[19] and thrombosis,[20] have limited its use.

MB surgical treatment involves both supra-arterial myotomy and coronary artery bypass graft.[13] Potential complications of myotomy include ventricular wall perforation, aneurysm formation, and postoperative bleeding. Regarding myocardial revascularization surgery, the major concern is graft failure. Surgery with the use of the left internal mammary artery results in a higher rate of occlusion when compared to saphenous vein grafts.[21]

Beta-blockers and calcium-channel antagonists are the first-line therapy for MB. For highly selected cases with refractory symptoms, coronary artery bypass graft or percutaneous coronary intervention with drug-eluting stent can be considered as options in the treatment.[21, 22]

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None.

CONFLICTS OF INTEREST

The authors declare there are no conflicts of interest.

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