MYOCARDIAL BRIDGING: A CASE STUDY

PONTE MIOCÁRDICA: ESTUDO DE CASO

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ABSTRACT

Introduction: The myocardial bridge integrates one of the possible differential diagnosis of Coronary Artery Disease (CAD) as a result of usually having an asymptomatic progress, but it may present itself with angina pectoris and more rarely with myocardial infarction or even sudden death. **Case report:** A female patient, 57 years old, asymptomatic at the first clinical evaluation, without precordialgia, hypertensive, underwent a routine cardiopulmonary stress test (CPX), which revealed a ST depression, in CM5 of 1,5 mm in relation to repose. **Commentary:** The study concluded that the clinical repercussions of Myocardial Bridging were due to the increment of tension during heart contraction in consequence of the increase of end diastolic pressure at the left ventricle due to arterial hypertension influence, what justifies the degree of myocardial hypertrophy.

Keywords: Myocardial bridging; Coronary artery disease; Chest pain

RESUMO

Introdução: A ponte miocárdica constitui um dos diagnósticos diferenciais da doença arterial coronariana que geralmente cursa de forma assintomática, mas pode manifestar-se com angina, e mais raramente, infarto agudo do miocárdio ou morte súbita. **Relato do caso:** Paciente do gênero feminino, 57 anos, assintomática em avaliação clínica inicial, sem precordialgia e hipertensa foi submetida a teste de esforço cardiopulmonar de rotina, o qual apresentou um infradesnivelamento de ST, em CM5 de 1,5 mm em relação ao repouso. **Comentários:** As repercussões clínicas da ponte miocárdica ocorreram devido ao incremento da tensão durante a sístole, em consequência do aumento da pressão diastólica final no ventrículo esquerdo, devido à influência da hipertensão arterial, o que justifica o grau de hipertrofia miocárdica.

Palavras-chave: Ponte miocárdica; Doença da artéria coronariana; Dor torácica

INTRODUCTION

Myocardial bridging (MB) can be defined as an anatomical variation in which a coronary artery segment that normally courses epicardially becomes intramural[1,2]; this variation usually affects the anterior descending artery via compression during systole, and it is reversible in diastole[3]. MB is one of the main differential diagnoses of coronary artery disease that is usually asymptomatic, but can manifest as angina and more rarely as acute myocardial infarction or sudden death, suggesting clinical relevance3-6.

MB is still underdiagnosed because few patients are symptomatic, the use of more accurate diagnostic methods is limited, and its pathophysiological mechanisms and treatment have not been fully elucidated[3]. Autopsy studies have found this anatomical variation in 15.0 to 85.0% of cases, but *in vivo* cases, it varies from 0.5 to 4.5% in conventional coronary angiography[7]. Clinical diagnosis should be considered in patients with chest pain and no risk factors for cardiovascular disease. In complementary exams, the most common finding in coronary cineangiography is the compression of the coronary segment during systole that is reversed in diastole.

The report describes the case of a patient with angina before the MB was demonstrated in the anterior descending artery.

CASE REPORT

Female patient, 57 years old, BMI 32.7 kg/m^2, asymptomatic in a previous clinical assessment, without precordial pain, smoker, hypertensive, with a paternal family history of fulminant acute myocardial infarction. She was taking telmisartan, simvas-

tatin, and calciferol.

She underwent a cardiopulmonary stress test on a treadmill using the ramp protocol with respiratory gas analysis (Metalyzer 3B); the patient was classified in the functional II (NYHA) group with poor cardiorespiratory performance (AHA). The test lasted 10 minutes and covered 0.74 km with a maximum power of 310.9 w; a maximum heart rate (HR) of 128 bpm (predicted 163 bpm), a maximum VO_2 of 16.45 ml/kg.min (predicted 29.87 ml/kg.min), a maximum VO_2/FC ratio of 11.4 ml/b (predicted 16.3 ml/b), and a maximum VE of 49.6 l/min (predicted 53.1 l/min).

Blood pressure was proportional to the effort made, with adequate recovery. A chronotropic deficit occurred, but with an adequate reduction in HR in the first minute of active recovery, with a satisfactory return of parasympathetic autonomic activity after exercise.

The pre-exercise electrocardiogram (ECG) showed sinus rhythm and regularity with no significant morphological changes. During exertion and recovery, the exam evidenced a ST depression at CM5 of 1.5 mm at peak exertion compared with rest (Figure 1). In addition, the test also evidenced a chronotropic deficit, a plateauing/dropping VO_2 pulse curve (normally related to myocardial ischemia), and a drop in SpO_2 during exertion.

Then, a coronary cineangiography was conducted as a complementary exam, showing muscle bundles involving the middle third of the anterior descending artery, with a slight depth, and an image suggestive of a stenosing lesion in this segment of the anterior descending artery (Figure 2).

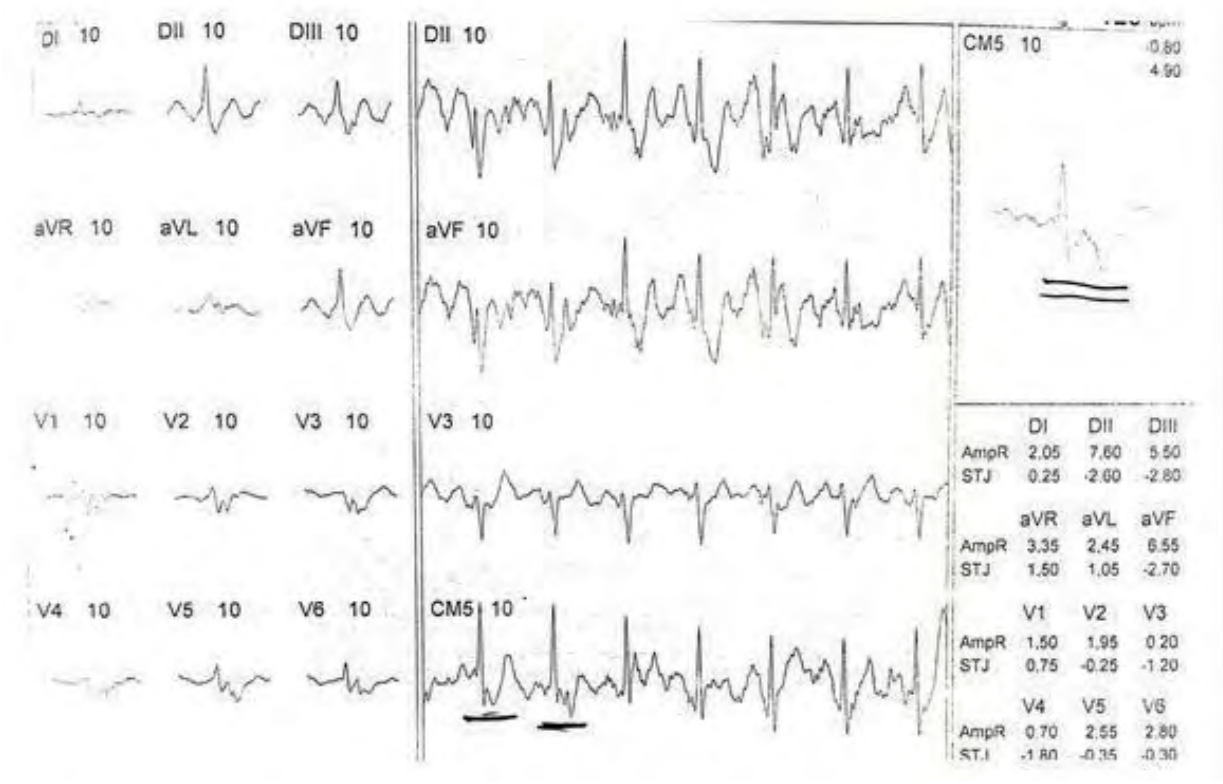

Figure 1. ECG during exertion showing ST depression at CM5 of 1.5 mm compared with rest.

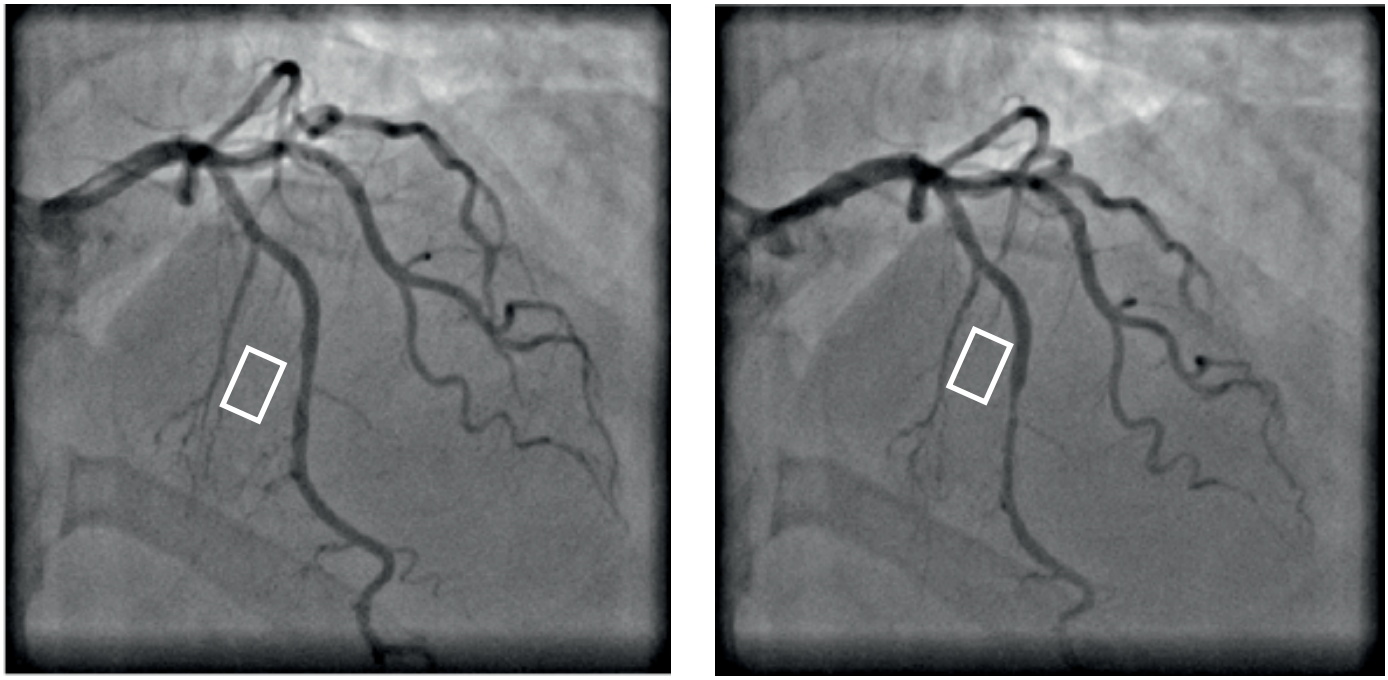

Figure 2. Coronary cineangiography showing a stenosing lesion, a myocardial bridge, in the projection of the anterior descending coronary artery in the middle third. A) Systole. B) Diastole.

COMMENTS

MB is a congenital anomaly that some authors suggest its origin in the embryonic period, concomitant with the formation of the coronary capillary network. Although MBs are congenital, symptoms in childhood are extremely rare, and the influence of extrinsic factors on the symptoms is unclear[4,5].

The most commonly involved artery is the anterior descending coronary artery, corroborating our finding and also other findings in the literature[4,5,8,9], but differs from a case report involving diagonal and septal branches of the anterior descending artery and a marginal branch of the circumflex artery[10]. The literature lacks data on MB affecting the diameter of the coronary artery branch.

The mechanism responsible for the symptoms is uncertain and controversial. Ventricular irrigation occurs almost exclusively during diastole, and the presence of MB alone would not explain the frequent symptoms, which require stress factors, such as physical exercise and psychological stress, to result in greater flow limitation and symptom onset.

Clinical diagnosis should be considered in patients with anginal symptoms and in the absence of risk factors or evidence of ischemia. The mean age of symptom onset reported was 45.9 years[4]; in this study, the age of diagnosis was 57 years. This finding may be explained by increased tension during systole as a result of increased end-diastolic pressure in the left ventricle due to the influence of hypertension, which justifies the degree of myocardial hypertrophy and arteriosclerotic processes, and may corroborate the findings and be demonstrated by the 1.5 mm depression described in CM5. The appearance of coronary spasm in the anterior descending coronary artery suggests an endothelial dysfunction in the segment, which may be the reason for the later presentation of symptoms[9].

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