CASE REPORT

Multiple myocardial bridges associated with left-ventricular dysfunction, intermittent left bundle branch block, and cardiac memory: A case report

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Abstract

Myocardial bridges are a congenital anomaly in which a segment of the coronary artery takes a “tunneled” intramuscular course. Few reports have associated myocardial bridges with left-ventricular dysfunction in patients with ischemia. Intermittent left bundle branch block is a conduction disturbance that has been described to be associated with myocardial bridges and cardiac memory. This study reports unusual associations of multiple myocardial bridges, angina, left-ventricular dysfunction, intermittent left bundle branch block, and cardiac memory.

KEYWORDS

angina, cardiac memory, intermittent left bundle branch block, left ventricular dysfunction, myocardial bridging

1 | INTRODUCTION

The suspicion and study for diagnosis of coronary disease includes evaluation of the patients' symptoms and assessment of their risk factors. It is essential at the time of decision making to request diagnostic studies and to follow specific protocols. Unusual findings on diagnostic studies should provide reason to suspect and confirm other pathological conditions with similar symptoms.

Under such conditions, using new diagnostic techniques can provide a better evaluation, thereby allowing correct diagnosis and an adequate treatment plan.

2 | CASE PRESENTATION

A 46-year-old man consulted the cardiology department for stable angina CF II of 1 year of duration. He had a history of arterial hypertension (HTA) treatment with enalapril and had a previous history of smoking (10 years ago). His father had HTA and had died at the age of 50 years from an aortic aneurysm.

His vital signs included: heart rate, 80 beats/min; blood pressure, 120/90 mmHg; and respiratory rate, 16 breaths/min. He appeared

healthy, with no cardiac heaves or lifts. His lungs were clear without any rales or wheezes. The point of maximal impulse (apical impulse) was noted at the mid clavicular line, in the fifth intercostal space, and normal S1 and S2 paradoxical splitting was observed, with regular rate and rhythm. No murmurs, S3 and S4, friction rub, jugular venous distention or pulsations, and peripheral edema was noted.

Routine 12-lead electrocardiogram (ECG) showed sinus rhythm with left bundle branch block (LBBB; Figure 1a).

A New ECG was performed as control and showed normalization of the QRS duration (80 msec), evidencing intermittent LBBB, rate-dependent LBBB, and changes involving the ST and T waves. T-wave changes correspond to the phenomenon of cardiac memory (Figure 1b).

Transthoracic echocardiography (TEE) was performed, which provided evidence of moderate deterioration in the left ventricular systolic function (LVSD) and left ventricular ejection fraction (LVEF) by 38%. Mild dilatation of the left ventricle was also observed with hypokinesia in the apical, lateral, septal, and lateral walls. The left atrium and right chamber were normal. An abnormal left ventricular diastolic pattern (grade I) was noted.

To assess myocardial ischemia, myocardial perfusion imaging was performed with decreased markers in segments of the anterior,

FIGURE 1 (a) Electrocardiogram at day 1, sinus rhyming 80 bpm with complete left bundle branch block (QRS duration >120 ms, eliminating normal septal Q waves in lateral leads); tall R waves are produced in the lateral leads [I, V5–V6] and deep S waves in the right precordial leads [V1–V3]; and broad or notched R waves in the lateral leads. (b) Electrocardiogram for control (day 3), sinus rhythm 70 bpm, normalization in QRS duration 90 ms and negative T waves from V2 to V5

apical, septal, and inferior wall. LVEF was 38%. The patient was diagnosed with angina CF II with myocardial ischemia in the described segments.

Cardiac magnetic resonance imaging (MRI) with late gadolinium enhancement was performed (Figure 2). This is a useful technique for diagnosis of cardiomyopathy, quantification of ventricular function,

establishing etiology, determining prognosis, and stratifying risk in cardiomyopathy, and showed left ventricular diastolic diameter (LVDD) of 58 mm; LV mass of 138, cardiac output of 2.8, and LVEF of 38%. The absence of the absence of scars of myocardial infarction, noncompaction cardiomyopathy, edema, deposition of other pathological substances, myocardial inflammation, fibrosis, and fatty infiltration of myocardium

FIGURE 2 Cardiac magnetic resonance imaging: (a) spoiled fast gradient echo, short-axis view with LVDD 58 mm; (b) spoiled fast gradient echo four-chamber view; (c) black blood image in the vertical long-axis view; (d) phase-sensitive inversion recovery in the vertical long-axis view; (e) phase-sensitive inversion recovery in vertical two-chamber view; (f) phase-sensitive inversion recovery in four-chamber view; (g) T2 turbo spin echo black blood short-axis view (perfusion). Left ventricular mass, 138. Cardiac output 2.8. Left ventricular ejection fraction (LVEF) 38%. The absence of fatty infiltration or edema. Late enhancement with gadolinium negative for fibrosis

involved the right or left ventricle. There was no fatty infiltration or edema. Late enhancement with gadolinium was negative for fibrosis.

Coronary computed tomography angiography (CCTA) was recommended to exclude atherosclerotic coronary artery disease. The coronary artery calcium score was 0, indicating no coronary artery atherosclerosis. Multiple myocardial (MB) bridges were found (Figure 3). A CCTA showed MB in coronary arteries, in the left anterior descending artery (LAD) in middle third the presence of MB. Coronary ramus intermedius (RI), two branches in portion distal had

with MB. Left circumflex artery in portion distal with MB are evidential for the study performed. RCA without atherosclerotic lesions. This is called multiple myocardial bridges.

Multiple myocardial bridges with complete LBBB and LVSD were diagnosed.

Treatment with cardiovascular medication was initiated. Beta-blockers, statins, and aspirin were prescribed. Improvement of symptoms was noted post treatment, and the patient currently does not have angina.

FIGURE 3 Cardiac multislice computed tomography. Coronary artery calcium score was 0. LMA, without lesions; LAD artery, without lesions, with a deep intramyocardial course (bringing); DB, with intramyocardial course; LCX, first diagonal without lesions with intramyocardial course in distal third (bringing). RI, of moderate size with two rams intramyocardial course in middle third. RCA, dominant without lesions. View of an intramyocardial course in LAD (16.6 mm); (a) Cross-sectional view of intramyocardial course in LAD. LMA: left main coronary artery; LAD: left anterior descending; DB: diagonal branch; LCX: left circumflex; RI: Ramus intermedius; RCA: right coronary artery

3 | DISCUSSION

Myocardial bridging is a congenital anomaly in which a segment of the coronary artery takes a “tunneled” intramuscular course under a “bridge” of the overlying myocardium. Vessel compression in systole results in hemodynamic changes that may be associated with angina, myocardial ischemia, acute coronary syndrome, left-ventricular dysfunction, apical ballooning syndrome, or even sudden cardiac death. This was first described anatomically by Reyman in 1737 (Hostiuc, Rusu, Hostiuc, Negoï, & Negoï, 2017; Reyman, 1737).

Ferreira has characterized myocardial bridges of the LAD artery fas pathology into two distinct subtypes. The more common “superficial” bridges (approximately 75% of cases) involve the LAD artery that is situated normally in the intraventricular groove but is intersected by a muscle bundle perpendicularly or at an acute angle. There is also a “deep” variant, in which the LAD artery deviates toward the right ventricle and dives into the intra-ventricular septum, with an overlying longitudinal muscle bundle arising from the right ventricular apex and crossing the tunneled segment transversely, obliquely, or helically before terminating in the intra-ventricular septum (Ferreira et al., 1991).

Left bundle branch block (LBBB) is a conduction disturbance associated with increased cardiovascular morbidity, mortality, coronary artery disease, hypertensive heart disease, aortic valve disease, and cardiomyopathy. Myocardial bridge (MB) has been described as associated with disturbed conduction (Domínguez Domínguez & Costa Pérez-Herrero, 2015; Steg et al., 2012). It is possible to suspect and

confirm cardiac memory (pseudo-primary T waves) associated with intermittent LBBB through electrocardiography findings and confirm the diagnosis of cardiac memory. Rosebaum et al. and Shvilkinl et al. described and analyzed the cardiac memory criterions, these findings are accurate for the diagnosis of this in presence of intermittent LBB. It is possible to suspect and confirm the cardiac memory phenomena (pseudo-primary T waves) associated with intermittent LBBB using electrocardiography findings. Myocardial ischemia must be ruled out. It is essential that despite the presumed diagnosis is the called phenomenon of cardiac memory (Rosebaum, Blanco, Elizari, Lázzari, & Davidenko, 1982; Shvilkin, Huang, & Josephson, 2015; Figure 4).

Changes in left ventricular performance in patients with MB have been reported in a study of the LAD during rest and in the inotropic state. The association between chest pain and ischemia has been described as endothelial dysfunction, coronary vasospasm, and systolic compression in the absence of coronary artery disease (Angelini, Velasco, & Flamm, 2002). The association between MB and LVSD is uncommon. Myocardial stunning is described in the bibliography, in the reviews of this associations do not have mentioned the evidence of this. Referred this associations but do not have a report of this, its unusual presentation of MB and LVSD (Marchionni, Chechi, Falai, Margheri, & Fumagalli, 2002).

CCTA provides a three-dimensional image of excellent quality, including information on the origin, course, and anatomic relationships among the anomalous vessels, allowing differentiation between benign and malignant variants. These entities require different management

FIGURE 4 Analyzed electrocardiogram's according the Rosenbaum (in green) and Shvilkin (in orange) showed the cardiac memory criterions. With Rosembaum criteria (green circles): (a) Its present. The axis of QRS in complete LBBB is 5° and the T-wave axis in normally QRS beats is -1° . (b) The accumulation of the size of the T wave with abnormal activation of the ventricle, and that the T waves in the normally QRS beats are symmetrically negative in those leads where the aberrant QRS complex were negative (green circles). With Shvilkin criteria (orange circles): (a) In normal QRS had positive T wave in aVL, (b) Positive or isoelectric T wave in lead I, (c) Maximal precordial T-wave inversion is greater than the T-wave inversion in lead III

and treatment plans. CCTA is a noninvasive imaging modality with excellent diagnostic performance compared to that of invasive coronary angiography (ICA) and can identify patients with coronary artery disease or any other condition involving the coronary arteries, for example, malformation, anomalous origin, and MB (Konen et al., 2007).

4 | CONCLUSION

Patients with LVSD of indeterminate origin should be evaluated to rule out associations such as myocardial bridges as a cause of their cardiomyopathy. A novel diagnostic method such as CCTA is useful for diagnosis. It allows appropriate therapeutic decision-making post diagnosis. This would allow us to accurately determine the prevalence of the association described in these findings, found in the patient.

The presence of multiple myocardial bindings and LVSD associated with this. The intermittent LBBB with cardiac memory. Everything reported associated with the presence of MB, but unusual presence of all these in this case.

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CONFLICT OF INTEREST

None.

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