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Clinical case

Milking phenomenon secondary to intramyocardial bridging in a patient with trifascicular block

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Phenomenon of order secondary to intramyocardial bridge in a patient with trifascicular block

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SUMMARY

Introduction: Intramyocardial bridges are an anatomical modification of the coronary circulation consisting of abnormal sections in the thickness of the myocardium that, by forming tunneled segments and incorporating into the epicardium, cause vascular milking called the

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Milking phenomenon during extrinsic systolic compression, which is responsible for the clinical manifestations and electrocardiographic alterations.

A clinical case is described with the aim of communicating to the scientific community the clinical, electrical and coronary angiographic characteristics of a patient with acute coronary syndrome with trifascicular block and Milking phenomenon due to intramyocardial bridging.

Clinical caseA 56-year-old woman with a history of hypertension, obesity, and frequent hospitalizations for unstable angina episodes was admitted for acute unstable angina plus trifascicular block. An intramyocardial shunt was confirmed in the distal mid-segment of the left anterior descending artery. Anti-ischemic therapy was optimized with carvedilol, after permanent pacemaker implantation.

Discussion: Intramyocardial bridges cause dynamic reduction of the lumen of the epicardial coronary vessel and when it is greater than 50% it causes the Milking phenomenon, which is more frequent in the middle and distal segments of the left anterior descending artery. Its evolution is favorable and they respond adequately to therapy with ß blockers.

Conclusions: The diagnostic correlation facilitated the application of an invasive approach with permanent transvenous stimulation that allowed the initiation of beta-blocker treatment, which would not have been possible due to the intraventricular conduction disorder.

Keywords: Intramyocardial shunt; Milking phenomenon; Coronary angiography; Acute coronary syndrome; Coronary artery disease.

ABSTRACT

Introduction: Intramyocardial bridges are an anatomical modification of the coronary circulation consisting of abnormal sections in the thickness of the myocardium that, when forming tunnel segments and incorporating into the epicardium, cause vascular milking, the so-

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called milking phenomenon, during extrinsic systolic compression, which is responsible for the clinical manifestations and electrocardiographic alterations.

A clinical case is described with the aim of providing the scientific community with the clinical, electrical and coronary angiographic characteristics of a patient with acute coronary syndrome with trifascicular block and milking phenomenon due to intramyocardial bridging.

Case Report:A 56-year-old woman with a history of hypertension, obesity, and frequent admissions for episodes of unstable angina was admitted for acute unstable angina plus trifascicular block and intramyocardial bridging was confirmed in the distal middle segment of the left anterior descending artery. Optimization of anti-ischemic therapy was based on carvedilol after implantation of a permanent pacemaker.

Discussion: Intramyocardial bridges cause a dynamic reduction of the coronary epicardial vessel lumen and when it is greater than 50% it causes a milking phenomenon, which is more frequent in the middle and distal segments of the left anterior descending artery, its evolution is favorable and responds adequately to ß-blocker therapy.

Conclusions: The diagnostic correlation facilitated the application of an invasive approach with permanent transvenous stimulation, allowing the initiation of beta-blocker treatment, which would not have been possible due to the intraventricular conduction disorder.

Key Words: Intramyocardial bridge; Milking phenomenon; Angiography; Acute coronary syndrome; Coronary artery disease.

SUMMARY

Introduction: The intramyocardial bridges are an anatomical modification of the coronary circulation constituted by abnormal sections in the thickness of the myocardium that, when segments are formed in the tunnel and incorporated into the epicardial system, cause vascular

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order, or the so-called order phenomenon, during extrinsic systolic compression, responsible for clinical manifestations and electrocardiographic alterations.

A clinical case is described with the objective of informing the scientific community about the clinical, electrical and coronary angiographic characteristics of a patient with acute coronary syndrome with trifascicular block and milking phenomenon due to intramyocardial bridging.

Case Report:A 56-year-old woman, with a history of high blood pressure, obesity and frequent hospitalizations for episodes of instável angina, was hospitalized for acute instável angina with trifascicular block and confirmation of an intramyocardial bridge in the middle distal segment of the anterior descending artery. The optimization of anti-ischemic therapy was based on carvedilol after implantation of definitive pacemaker.

Discussão: The intramyocardial bridges cause a dynamic reduction in the lumen of the coronary epicardial vessel and when this is greater than 50% it causes a phenomenon of order, more frequently in the middle and distal segments of the anterior descending artery, its evolution is favorable and it responds adequately to therapeutics with ß-blockers.

Conclusões:A diagnostic correlation facilitates the application of an invasive approach with permanent transvenous stimulation, allowing the initiation of treatment with beta-blockers, which would not have been possible due to intraventricular conduction disturbance.

Keywords: Get intramyocardial; Phenomenon of order; Angiography; Acute coronary syndrome; Coronary disease.

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Introduction

Cardiac tissue is nourished through the main coronary arteries and normally runs epicardially, surrounding the myocardium from its surface. (1-3) In less than 1% of patients, this epicardial course is modified, such that segments with an intramural course are described, covering a certain length of the vessel, which can range from 4 to 80 mm. (1,2,4)

This congenital anomaly of the coronary circulation is characterized, anatomically, by sections that are found in the thickness of the myocardium and give rise to tunneled segments that, after penetrating the interior and then joining the epicardium, are called intramyocardial tracts or bridges (IMP). (2,3,5) Meanwhile, in each cardiac cycle, an extrinsic systolic compression of the arterial portion that is located between the muscle fibers is generated and causes a kind of milking or Milking phenomenon (FeMilk), by its meaning in English, when the constriction is greater than 50% of the vessel lumen. (2,3)

Its usual location is the middle and distal portion of the left anterior descending artery; it has a benign course, but is often associated with chronic exertional angina, arrhythmias, acute coronary syndromes, arterial spasms, ventricular septal defects due to septal rupture, ventricular dysfunction, and even sudden death. (1-3,5,6)

Although coronary angiographic reports only report an incidence of 0.6% to 4.0%, some postmortem investigations identify PIM in 23% to 55% of autopsies performed; in fact, not all intramyocardial tracts cause FeMilk and in many cases their behavior is asymptomatic, which represents a greater diagnostic challenge in the context of acute coronary syndrome. (2,3)

Likewise, this anatomical variety is underestimated in practice because it does not manifest clinically and because it lacks classic signs in traditional imaging studies that make this malformation evident. (2-4)

On the other hand, adrenergic blocking drugs and non-dihydropyridine calcium antagonists are essential pillars of proven efficacy in the implementation of anti-ischemic therapies; however,

due to their recognized negative chronotropic effect, their prescription in the context of atrioventricular (AV) and intraventricular conduction disorders constitutes a real therapeutic challenge, as prior cardiac electrical stimulation is required through the implantation of a permanent pacemaker to guarantee an optimal heart rate.

Investigating the diagnostic guidelines that imply the timely clarification of this phenomenon and lead to accurate therapeutic treatment justifies the presentation of this clinical case; the primary objective was to inform the scientific community about the diagnostic characteristics and therapeutic treatment of a patient with Milking's phenomenon associated with trifascicular block.

Case presentation

This is a 56-year-old mixed-race woman with a history of obesity, diabetes, and hypertension, with a history of multiple admissions for episodes of exertional anginal pain with no electrocardiographic changes. She was admitted to the emergency intensive care unit with intense, oppressive, burning, and work-related retrosternal pain radiating to the neck, jaw, and left shoulder. This pain was also accompanied by anxiety, sweating, and coldness, and did not resolve after three doses of sublingual nitroglycerin, requiring opioids for complete relief.

Vital parameters: BP: 130/90 mmHg FC: 44bpm FR: 18 rpm Temp.: 36.3 °C

To the physical examination

Normal chest expansion. Normal breath sounds were heard. No rales were heard. Bradycardic, arrhythmic, low-pitched, and low-intensity heart sounds were heard. No third or fourth sounds were heard. No murmurs were heard. No pericardial friction rub was observed. No thermal gradient or jugular vein engorgement was observed. No edema was observed. Pulses were full and symmetrical (Table 1).

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Table 1. Results of the Hemochemical analysis.

Analytics	Results	Analytics	Results
Hemoglobin (g/L)	120.0	Cholesterol (mmol/L)	6.6
Blood glucose (mmol/L)	5.9	Triglycerides (mmol/L)	1.9
Creatinine (µmol/L)	78.9	Uric acid (µmol/L)	320.0

It was interpreted as a trifascicular block in the context of an acute coronary syndrome without ST-segment elevation, which is why the patient was admitted for prognostic stratification and therapeutic application, which included implantable permanent electrical stimulation (Figure 1).

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Fig. 1. Electrocardiogram 1.

Fountain: Electrocardiogram 1. ESC. HC: 1299355. Hospital "Carlos Manuel de Céspedes".

Second-degree AV block (2:1) + Complete left bundle branch block (Trifascicular block). HR: 43 bpm, Aoqrs: - 90º. ST and T typical of intraventricular disorder.

A permanent pacemaker was implanted in VVIR mode (Biotronik), with a heart rate of 60 bpm and amplitude and sensitivity of 1.5 mV (Figure 2).

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Fig. 2. Electrocardiogram 2.

Fountain: Electrocardiogram 2. Long DII. ESC. HC: 1299355. "Carlos Manuel de Céspedes" Hospital.

Pacemaker-dependent rhythm (PPM) HR: 75 bpm, QRS: 120 ms.

The echocardiogram revealed:Mild left ventricular (LV) remodeling. Hypokinesia of the septal (middle and apical), lateral (apical), and apical segments (segment 17). LV ejection fraction 55% by SIMPSON volumetric method.

Conclusion: Ischemic heart disease in the territory of the anterior descending artery (LAD) with preserved systolic function.

The patient continued to experience episodes of precordial pain during hospitalization, so a coronary angiography was performed, which revealed main epicardial coronary arteries without significant atherosclerotic lesions and a tunneled mid-distal course of the left anterior descending artery (LAD), causing systolic compression of 50% of the vessel lumen, which is responsible for the patient's ischemic symptoms. (Figure 3).

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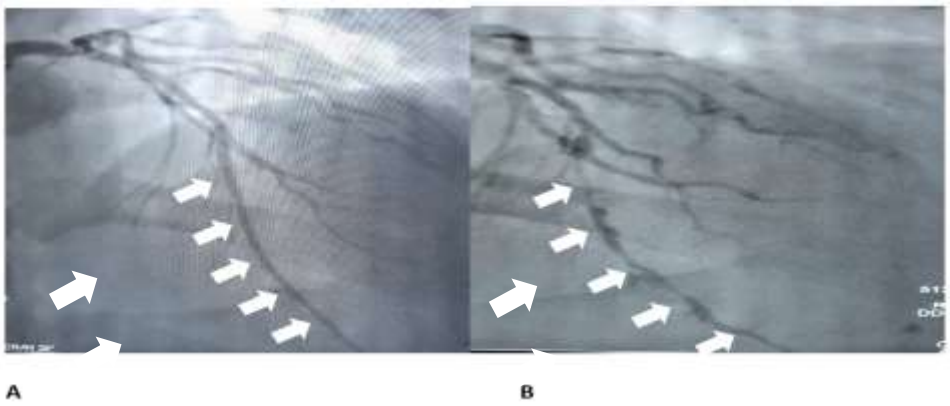

Fig. 3. Coronary angiography.

Fountain: Courtesy of the Santiago de Cuba Cardiocenter. ESC. HC: 1299355.

Coronary angiography. Right anterior oblique projection, RAO 10˚ and Skull 30˚.

(TO) Course of the DA artery during diastole. (B) Intramyocardial course of the DA artery, which constricts more than 50% of the artery during systole.

Once this abnormality of the coronary circulation was confirmed and the trifascicular blockade was corrected with the implantation of the PPM, treatment was applied with Carvedilol 12.5 mg/day, Amlodipine 10 mg/day, Captopril 25 mg/day, Spironolactone 25 mg/day and Aspirin (81 mg/day), which led to satisfactory progress and discharge of the patient with follow-up in the outpatient clinic for pacemaker programming and ischemic heart disease.

Discussion

Intramyocardial bridges are a common finding in coronary angiographic studies, however, current research differs in their behavior depending on whether they are imaging studies or autopsies, being higher in the latter, between 15 and 85% compared to hemodynamic studies with images that show an incidence between 0.5 and 15%. (2,6)

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The PIM and its hemodynamic phenomenon called Milking due to its systolic milking effect on the coronary artery, is considered a normal anatomical variant according to De la Torre and collaborators; (2) on the contrary, Peral (6) considers the PIM a congenital abnormality of the coronary circulation while referring to the description of acquired cases as a result of the increase in myocardial mass as evidenced in ventricular hypertrophy secondary to arterial hypertension, valvular heart disease (aortic stenosis), or in primary entities such as hypertrophic cardiomyopathy, where it is related to more severe symptoms and a high risk of cardiovascular complications.

The left anterior descending artery (LAD) in its middle and distal portions is the most frequent location, representing 43.2%; although some authors claim that coronary angiography can demonstrate that the LAD artery represents between 87.2% and 100% of cases affected with this anatomical variant. (1-3) However, cases of intramyocardial pathways in the right coronary artery have been described, but to a lesser extent, 18.4% according to Contreras et al. (1)

The hemodynamic impact affects systolic flow to a greater extent, but because coronary arteries are perfused during diastole, these tunneled segments interfere with both phases of the cardiac cycle and hypotheses are postulated that describe the milking of the vessel until mid- to late diastole. (2,3,7,8) On the other hand, other disorders are described such as delayed muscle relaxation, increased coronary flow velocity, retrograde flow and decreased diastolic lumen of the vessel, which together cause a reduction in coronary flow reserve and myocardial ischemia. (2)

These hemodynamic alterations depend largely on the thickness and length of the MFP, as well as its orientation relative to the myocardial fibers and the presence of connective tissue in the tunneled segment. (5) The development of atherosclerotic plaques in the proximal segment of the MFP is common, which is related to the low shear capacity at this level; although it is true

that ischemia occurs thanks to the aforementioned hemodynamic effects, atherosclerosis can aggravate ischemic symptoms. (3,5)

On the other hand, in the tunneled portion it is common to find negative remodeling of the vascular endothelium, but in the absence of atherosclerosis. (3,5)

The extension of the muscular bridge can range from 4 to 25 mm, although they can be longer. Peña Oliva S. et al. (3) described a case of 30 mm in length, which implies a marked affectation of the coronary reserve. Regarding the percentage of compression, it was classified into three groups: grade 1 less than 50%, grade 2 between 50-75% and grade 3 greater than 75%. (9,10)

- Coronary angiography is the most widely used conventional method for diagnosing PIM. Other noninvasive methods include three-dimensional multislice computed tomography, which is generally superior to coronary angiography. Among the invasive methods, intracoronary ultrasound is a novel method with a high diagnostic rate, although none is the absolute gold standard for diagnosing PIM.

  The non-pharmacological therapeutic strategy recommends the absolute modification of the individual risk factors of each patient and the medicament includes as a choice in these cases the ß blockers, with the best results obtained up to now due to their mechanism of action (1), also the calcium antagonists can be used with favorable results, as well as the antiplatelet agents, in the prevention of alterations related to the development of atherosclerosis and its complications. (1,3,5)

  From an interventional perspective, in symptomatic patients, stenting can improve systolic coronary perfusion, but is more associated with complications compared to this invasive procedure. (5) On the other hand, surgical procedures can be used when symptoms persist; however, recent research provides evidence of high clinical risk, such as myectomy, which reduces peripheral vessel pressure, and revascularization by bypass, although these have not shown any advantages over other techniques and pose a real challenge. (6,9,10)

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In this case, medical treatment was administered without the need for invasive therapeutic procedures that carry a higher risk of cardiovascular complications. To date, the patient remains asymptomatic with an excellent response to therapeutic stratification.

Conclusions

Permanent electrical stimulation due to the atrioventricular conduction disorder allowed the initiation of anti-ischemic pharmacological treatment, given the persistence of anginal symptoms secondary to intramyocardial bridging in the distal middle segment of the left anterior descending artery, with a good therapeutic response and favorable prognosis.

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Conflicts of interest

The authors declare no conflicts of interest related to the study.

Authorship contribution:

Marian Maité Rodríguez Peña: As lead author, she originated the idea for the article's topic; she participated in the conceptualization, data curation, formal analysis, research, methodology,

research design, software development, visualization, validation, writing (original draft), and writing–review and editing.

Julio Alberto Pérez Domínguez: contributed to the research design, data curation, formal analysis, and writing of the original draft.

Raúl Leyva Castro: collaborated on the methodology, research design, supervision, and writing – original draft.

José Alberto Sánchez Guerra: participated in data curation, formal analysis and methodology.

Addiel Matos Pupo: contributed to data curation and analysis of the study.

I, Marian Maité Rodríguez Peña, on behalf of the co-authors, declare the veracity of the content of the article: Milking phenomenon secondary to intramyocardial bridging in a patient with trifascicular block.