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Case Report

Cardiocentro ERNESTO CHE GUEVARA SANTA CLARA CUBA

Long myocardial bridging in the left anterior descending artery causing acute coronary syndrome

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ARTICLE INFORMATION

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Figures
Images from complementary tests are shown with the patient's consent.

Abbreviations
LAD: left anterior descending artery
MB: myocardial bridging

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ABSTRACT

Coronary arteries usually run along the outer surface of the heart. In some cases, small segments of them take a "tunneled" intramuscular course forming the so-called myocardial bridging. This anatomical feature may lead to a sudden systolic narrowing of the "tunneled" segment (milking effect), thereby impairing coronary blood flow in both systole and diastole; which further reduces coronary reserve. Myocardial bridging mainly affects the middle-distal segments of left anterior descending (LAD) artery and may cause anything from chest pain to sudden death. So far, it seems that the use of beta-blockers and anti-calcium agents is the most effective therapeutic option for symptomatic cases. We now report a case where the clinical presentation of this anatomical feature was an acute coronary syndrome. Timely, accurate angiographic diagnosis allowed for adequate therapeutic adjustments to improve the patient's symptomatology.

Keywords: *Myocardial bridging, Intramyocardial course of coronary artery, Milking, Acute coronary syndrome*

Puente miocárdico largo en la arteria descendente anterior como causa de síndrome coronario agudo

RESUMEN

Las arterias coronarias principales generalmente tienen un trayecto epicárdico. En algunos casos, pequeños segmentos de éstas se introducen en el interior del miocardio formando los denominados puentes miocárdicos. Esta particularidad anatómica puede producir un estrechamiento sistólico súbito del trayecto tunelizado (fenómeno de *milking*) y afectar el flujo coronario tanto en sístole como en diástole, con la consiguiente reducción de la reserva coronaria. Afecta principalmente a la arteria descendente anterior en sus segmentos medio y distal. Su presentación clínica puede ir desde un cuadro anginoso hasta la muerte súbita. Hasta el momento, parece ser, que el uso de betabloqueadores y anticálcicos es la opción terapéutica más efectiva en los casos sintomáticos. Se expone un caso en el que un síndrome coronario agudo fue la forma de presentación de esta variante anatómica y el oportuno diagnóstico angiográfico permitió realizar los reajustes terapéuticos necesarios para mejorar la sintomatología del paciente.

Palabras clave: Puente miocárdico, Trayecto coronario intramiocárdico, *Milking,* Síndrome coronario agudo

INTRODUCTION

Blood is supplied to the heart by the coronary arteries, whose main branch-

es generally take an epicardial course, that is, they run all along the surface of the myocardium. In some patients, small segments of these arteries take a "tunneled" intramuscular course under a "bridge" of overlying myocardium; having varying intramyocardial course distances. This arrangement frequently results in vessel compression during systole (extrinsic compression or milking effect) −as consequence of the external pressure exerted on the artery by the myocardial fibers− which may be confounded with atheromatous stenosis or coronary spasm in coronary angiography imaging[1].

Thorough observation of a normal or near-normal caliber artery in diastole rules out such diagnoses and helps confirm this anatomical feature. Muscle overlying the intramyocardial segment of an epicardial coronary artery was first mentioned by Reyman in 1737 and was termed myocardial bridge by Polacek in 1961, name by which it is still known[1].

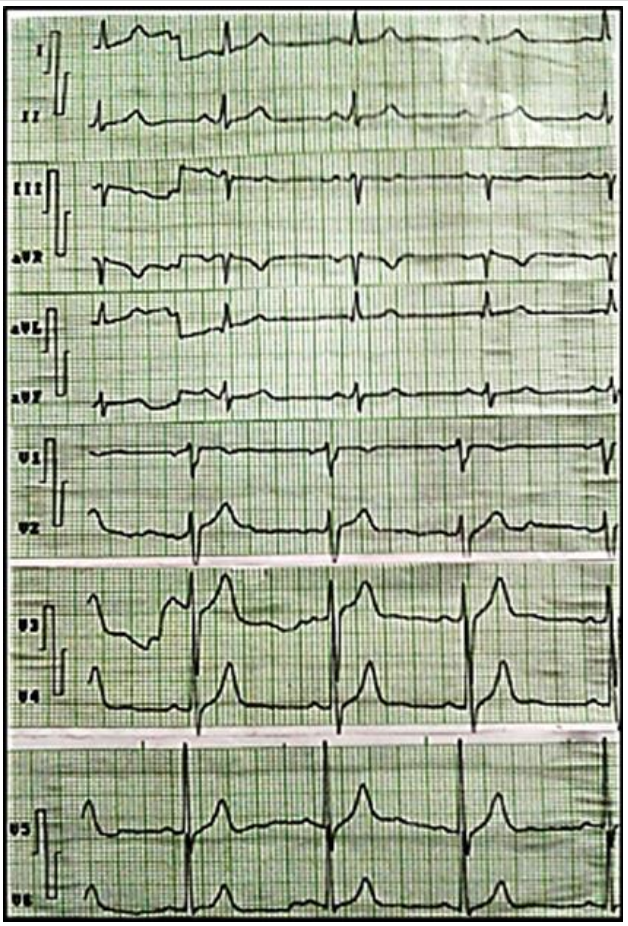

Figure 1. Standard 12-lead electrocardiogram on admission.

Myocardial bridging is largely considered to be a benign condition, relatively common among the general population; which primarily afflicts patients at low risk for coronary artery disease. When symptomatic, this condition may be associated with adverse complications including stable or unstable angina, arrhythmias, (ventricular and supraventricular tachycardia), acute myocardial infarction and sudden death, although the last two are infrequent[2].

A number of angiographic series have shown an incidence of 0.82 to 4%, while targeted autopsy studies have yielded results of 23 to 55%. Such difference is due, on the one hand, to the fact that not all myocardial bridging (MB) generate systolic compression, at least detectable; and, on the other hand, to the angle or depth, or both, of the muscle fibers. Myocardial bridges are either single or multiple and may appear in isolation or coexisting with other coronary lesions[3]. They are most frequently localized to the middle-distal segment of the left anterior descending (LAD) artery[4].

This anatomical variant remains underdiagnosed due to the small number of symptomatic patients, limited availability of more accurate diagnostic methods and consequently their restricted use; therefore, its pathophysiological mechanisms (beyond extrinsic compression) and treatment have not yet been fully elucidated[3].

CASE REPORT

We present the case of a 45-year-old mixed-race man who had been a smoker for over 30 years; who claimed that approximately one month prior to medical consultation was presenting with oppressive exertional chest pain progressively increasing in frequency, intensity and duration, radiating to the left arm, neck and jaw; which prompted him to present to the emergency room.

No suggestive findings were found on physical examination; heart rate and blood pressure were 72 beats per minute and 120/80 mmHg, respectively. His electrocardiogram on presentation showed tall, symmetrically peaked T-waves in V_2-V_6 (**Figure 1**), with no changes in subsequent serial electrocardiograms. He was considered to have non-ST segment elevation acute coronary syndrome given the symptomatology and electrocardiographic findings. Therefore, subsequent admission to the Cardiology Department for study and treatment was agreed upon.

Laboratory tests evidenced: negative cardiac troponins and total creatine kinase-MB fraction. All other blood tests were also within the normal range with blood glucose at 5.4 mmol/L, creatinine 108 µmol/L, cholesterol 3.4 mmol/L and triglycerides 1.12 mmol/L.

When taking into account the negative result for biomarkers of myocardial necrosis the patient was defined as having unstable angina. He underwent transthoracic echocardiography which proved to be normal, without disorders of global and segmental left ventricular wall function at rest; left ventricular ejection fraction of 64%, and normal dimensions of the heart chambers and great vessels.

The patient was initially commenced on anti-ischemic treatment but he had recurrence of pain. It was thereby decided to perform coronary angiography which reported that epicardial coronary arteries (left main coronary artery, LAD artery, circumflex artery, and right coronary artery) and their main branches did not present significant lesions. The LAD artery was found to be a well-developed vessel with an MB of roughly 30 mm, partially occupying its middle and distal segments (**Figure 2**).

Treatment was readjusted based on the angiographic diagnosis and doses of atenolol 25 mg/day and amlodipine 10 mg/day were added; resulting in alleviation of symptoms and hospital discharge. The patient has been followed-up in regular outpatient appointments and has had a satisfactory outcome.

COMMENT

Discussions about MB's ability to cause myocardial ischemia may be influenced by the group of patients analyzed[5]. Its clinical symptomatology is variable and will be conditioned by the degree of coronary blood flow impairment[6]. When systolic vessel compression is greater than 50% and especially 70%, the coronary flow reserve will always be reduced[5]. While frequently asymptomatic, this condition in many cases may be responsible for adverse complications, mainly chest pain on both exertion and rest. Its spectrum ranges from angina to acute myocardial infarction or sudden death[6].

The previously mentioned case report depicts a long MB with significant systolic reduction of the vessel caliber whose clinical presentation was that of a non-ST segment elevation acute coronary syndrome, defined as unstable angina.

The pathophysiology of MB originates from the many and diverse biomechanical forces generated inside it; and will cause impairment of the adjacent segments. Therefore, all histopathological manifestations observed, differ between the tunneled segment and those proximal and distal to it. The proximal

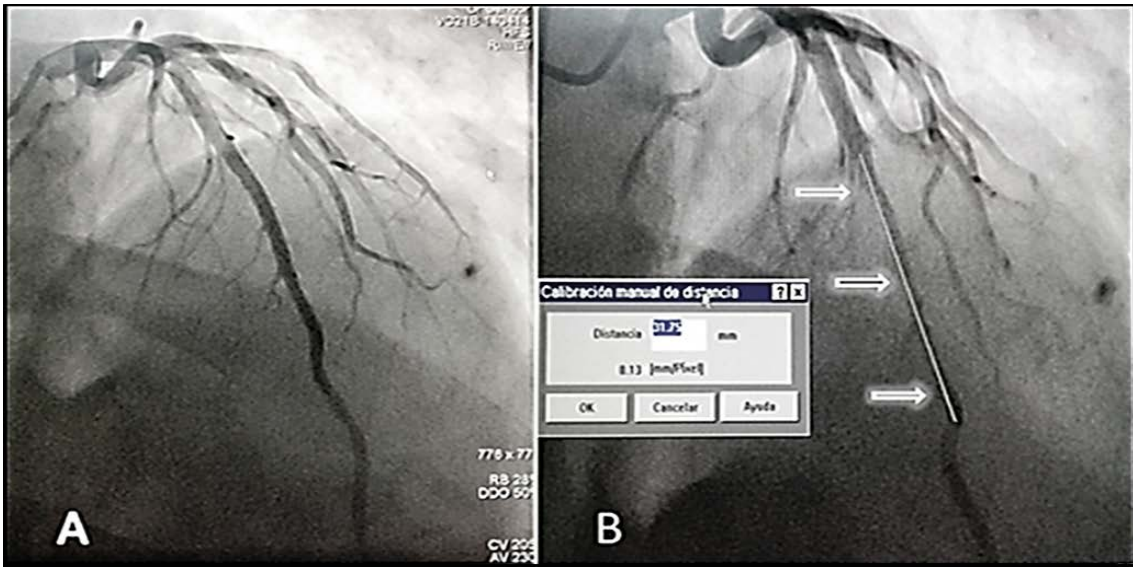

Figure 2. Left coronary angiography (anteroposterior view). **A.** Normal left anterior descending artery during ventricular diastole **B.** Extrinsic compression (milking) of 31.75 mm in the middle-distal segment during systole.

segment is likely to develop atherosclerosis, which is mainly attributable to hemodynamic disturbances such as low shear stress generated by the MB at this level. The proximal mechanical stress resulting from arterial compression within the MB and the angle at which the vessel enters the myocardium may also play a role. A constrictive vascular remodeling in the tunneled segment is frequently observed, yet with no associated atheromatous disease. As for dynamic obstruction, it has been documented that myocardial ischemia is not exclusively related to systolic vascular compression, but that it persists into diastole[7] since adequate relaxation is not achieved, which may cause diastolic flow disorders[6].

The current gold standard method for diagnosing MB is coronary angiography. This anatomical variant's angiographic behavior and its hemodynamic disturbances will depend on multiple factors including bridge length, depth/thickness, orientation of myocardial fibers to the artery and the nature of tissue between them. The angiographic finding is a systolic stenosis that disappears during diastole, thus observation of a normal caliber vessel during this phase of the cardiac cycle, confirms the so-called myocardial bridging[6].

Angiographically, MBs are almost exclusively spotted in the middle-distal segments of LAD artery and may commonly range from 4 to 25 mm in length; being less frequent in the diagonal and marginal branches. It was precisely the LAD artery and the aforementioned segments that were involved in the previous case report. Although in this particular case the MB course is strikingly long, about 30 mm, presumably resulting in reduced coronary flow reserve and leading to subsequent clinical expressions.

Increased heart rate, contractility/flow velocity, short diastolic perfusion time and exercise-induced coronary spasm may cause chest pain in patients with MB. Bearing this in mind, first-line therapy remains medical treatment with increased doses of beta-blockers since they have a negative inotropic and chronotropic effect[6]. A good alternative would be the use of calcium channel blockers, which also add vasodilatory properties[7]. In general, the use of pure vasodilators such as nitrates should be avoided in these specific patients, as they increase systolic stenosis and lower the ischemic threshold, which may aggravate symptoms[6,7].

The patient had a positive response to treatment adjustments (betablockers and calcium channel blockers) since his symptomatology improved after being started on these medications as primary treatment.

Knowing this anatomical variant allows for the application of specific treatments or optimization of previous ones, which is extremely important as it may condition its prognosis.

CONCLUSIONS

Although most patients with an MB are presumed to be asymptomatic or present mild symptoms, there are some cases −like the one in this paper− where it would lead to acute coronary syndrome related to triggering factors; thus requiring hospitalization and treatment modifications.

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