Cardiovascular and Metabolic Science

Clinical Case

Vol. 31 No. 4
October-December2020

doi: 10.35366/97546

# Is coronary artery myocardial bridging always a benign condition?

## ¿Los puentes musculares arteriales coronarios son siempre una condición benigna?

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**Keywords:**
Myocardial bridge, acute coronary syndrome, cardiogenic shock.

***Palabras clave:***
*Puentes musculares, síndrome coronario agudo, choque cardiogénico.*

**ABSTRACT**

**Introduction:** The myocardial bridging (MB) is a rare clinical entity, with a prevalence of 0.15-16% in angiographic series and 5-86% in autopsy series; considered of benign course; commonly associated with hypertrophic cardiomyopathy and infrequently with acute coronary syndromes (ACS). **Justification:** Report this rare association of pathologies, with few reports in the literature. **Clinical case:** 61-years-old male, without previous chronic degenerative diseases, current smoker. He started 4 hours prior to admission with sudden anginal chest pain, dyspnea, and profuse diaphoresis. Electrocardiogram: left bundle branch block, sinus rhythm with HR 44 bpm. Troponin I: 0.6 µg/L. A diagnosis of STEMI with cardiogenic shock was integrated. Emergency coronary angiography was performed reporting epicardial arteries without obstructive lesions, with the presence of muscle bridges in the left anterior descending artery in the middle and distal segments, in the first diagonal and the vertical segment of the right coronary artery with severe milking phenomenon. Ventriculogram: with mild anteroapical hypokinesia and asymmetric septal hypertrophy of the left ventricle without intraventricular gradient or Brockenbrough-Braunwald phenomenon. No clinical improvement despite specific treatment management, so a successful supra-arterial decompression myotomy was performed. Vasoactive amines were withdrawn, with an adequate postoperative clinical course, so he was discharged home asymptomatic. Control echocardiogram without alterations of ventricular mobility, remaining asymptomatic at six months of follow-up. **Conclusions:** Although infrequent as in our case, the association of hypetrophic cardiomyopathy and MB may occur with ACS. This association has not been ruled out as a possible cause of ischemia and sudden death in these patients. The literature supports the use of beta-blockers and supra-arterial myotomy or coronary bypass as surgical treatment. Percutaneous treatment is not recommended. There is no consensus for its management, and treatment must be individualized in each patient.

***RESUMEN***

***Introducción:*** *El puente miocárdico (PM) es una entidad clínica poco frecuente, con prevalencia 0.15-16% en series angiográficas y 5-86% en series de autopsias; considerada de curso benigno; comúnmente asociado con miocardiopatía hipertrófica e infrecuentemente a síndromes coronarios agudos (SICA).* ***Justificación:*** *Reportar esta rara asociación de patologías, con escasos reportes en la literatura.* ***Caso clínico:*** *Masculino 61 años, sin enfermedades crónico degenerativas previas, tabaquismo positivo. Inició 4 horas previas a su ingreso con dolor torácico anginoso súbito, disnea y diaforesis profusa. Electrocardiograma: bloqueo de rama izquierda, ritmo sinusal con FC 44 lpm. Troponina I: 0.6 µg/L. Se integró diagnóstico de IAMCEST con choque cardiogénico. Se realizó coronariografía de emergencia reportando arterias epicárdicas sin lesiones obstructivas, con presencia de puentes musculares en la descendente anterior en el segmento medio y distal, en la primera diagonal y en el segmento vertical de la coronaria derecha con fenómeno de* milking *severo. Ventriculograma: con hipocinesia anteroapical leve e hipertrofia septal asimétrica del ventrículo izquierdo sin gradiente intraventricular, ni fenómeno de Brockenbrough-Braunwald. Sin mejoría clínica a pesar del manejo tratamiento específico, por lo que se realizó miotomía de descompresión supraarterial exitosa. Se retiraron las aminas vasoactivas, cursando con adecuada evolución clínica postquirúrgica, por lo que se egresa a su domicilio asintomático. Ecocardiograma de control sin alteraciones de la movilidad ventricular, permaneciendo asintomático a los seis meses de seguimiento.* ***Conclusiones:*** *Aunque infrecuente como en nuestro caso, la asociación de miocardiopatía hipertrófica y PM pueden cursar con SICA. No se ha descartado esta asociación como una posible causa de isquemia y muerte súbita en estos pacientes. La literatura apoya el uso de betabloqueadores y miotomía supraarterial o bypass coronario como tratamiento quirúrgico. No se recomienda el tratamiento percutáneo. No existe un consenso para su manejo y el tratamiento debe individualizarse en cada paciente.*

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*Received: 24/07/2020*
*Accepted: 13/11/2020*

**How to cite:** Serrano AC, Guzmán RD, Palacios GEC. Is coronary artery myocardial bridging always a benign condition? Cardiovasc Metab Sci. 2020; 31 (4): 137-140. https://dx.doi.org/10.35366/97546

## INTRODUCTION

Myocardial bridging (MB) is a congenital abnormality defined by a segment of a coronary artery that takes a «tunneled» intramuscular course under a bridge of overlying myocardium, resulting in systolic compression.[1] Its incidence ranges from 1.5 to 16% when assessed by angiography and up to 86% in some autopsy series.[2]

Traditionally, MB has been considered a benign condition; however, severe bridging of the major coronary arteries can produce silent ischemia, stable angina, acute coronary syndromes, stress cardiomyopathy, or malignant arrhythmias possibly leading to sudden cardiac death.[3]

The bridged coronary segment compressed at systole (milking effect), contributes to myocardial supply-demand mismatch and development of ischemic symptoms. The mechanism is not completely understood.[4] Pathophysiologic factors that may exacerbate myocardial bridges are the patient's age, heart rate, left ventricular hypertrophy, and the presence of coronary atherosclerosis, since all of these may worsen the supply-demand mismatch imposed by the bridge, reducing coronary reserve.[5] Herrmann et al. suggested that the wrapping of the myocardial bridge by myocardial fibers contracting in systole limits the vasodilation capacity to nitroglycerin at the myocardial bridge, compared to the anatomically unrestricted conditions and vasodilation in the segments proximal and distal to it. Also, previous data suggest that MB is associated with the development of atherosclerosis proximal to the tunneled artery, but currently, no data demonstrate this as an independent factor.[6] Cay et al. showed that systolic compression > 50% of the native lumen might predict future cardiac events.[7] A higher prevalence of MB and/or milking arteries has been already demonstrated in patients with hypertrophic cardiomyopathy (HCM) than in healthy subjects.[8] And children with HCM were found to carry an increased risk for ventricular arrhythmias and sudden death if systolic compression was > 80%. These factors also can contribute to explaining cardiovascular outcomes.[9]

It is extremely rare for multiple arteries or segments to be affected simultaneously by MB. Only symptomatic patients or those with straight forward clinical signs of ischemia require treatment. The cornerstone treatment is medical with beta-blockers and calcium channel blockers, being the regimen of choice.[10] In contrast to atherosclerotic MI, the role of PCI in MB ACS is very limited due to the adverse outcome reported in the literature.[11] Surgical treatment with dissection of the overlying myocardium is reserved for patients with persistent symptoms refractory to medical therapy, in whom ischemic changes are proven, and for those with high-risk markers such as life-threatening ventricular arrhythmias, aborted sudden death, or nonfatal myocardial infarction.[12]

## CASE PRESENTATION

61-years-old male, without previous chronic degenerative diseases, current smoker. He started 4 hours before admission with sudden anginal chest pain, dyspnea, and profuse diaphoresis. Electrocardiogram: left bundle branch block, sinus rhythm with HR 44 bpm. Troponin I: 0.6 µg/L. A diagnosis of STEMI with cardiogenic shock was integrated. Emergency coronary angiography was performed reporting epicardial arteries without obstructive lesions, with the presence of muscle bridges in the left anterior descending artery in the middle and distal segments, in the first diagonal and the vertical segment of the right coronary artery with severe milking phenomenon *(Figure 1)*. Ventriculogram: with mild anteroapical hypokinesia and asymmetric septal hypertrophy of the left ventricle without intraventricular gradient or Brockenbrough-Braunwald phenomenon *(Figure 2)*. No clinical improvement despite specific treatment management, so a successful supra-arterial decompression myotomy was performed. Vasoactive amines were withdrawn, with an adequate postoperative clinical course, so he was discharged home asymptomatic. Control echocardiogram without alterations of ventricular mobility, remaining asymptomatic at six months of follow-up.

## DISCUSSION

Acute management of MB associated with a STEMI remains a challenge. Patients can be

managed with medications, via surgical approach or via percutaneous coronary intervention (PCI) although controversial.[13] The use of PCI has been limited by the presence of in-stent restenosis, stent fracture, and perforation during stent deployment. Coronary artery bypass grafting and supraarterial myotomy are the surgical strategies that may improve the quality of life in symptomatic adult patients. In cases where multiple arteries or segments are affected simultaneously by MB, supraarterial myotomy seamed the most reasonable choice.[14]

In this case, we determined that the cause of the patient's clinical condition was related directly to MB since the echocardiogram showed a non-obstructive asymmetric septal hypertrophic cardiomyopathy and the coronary angiography had no obstructive coronary artery disease or another pathological finding that could explain the patient's critical state except for several myocardial bridges on multiple arteries with severe milking effect, refractory to optimal medical treatment.

## CONCLUSIONS

MB treatment is still challenging, and there is little information about prognosis and outcomes in these patients. Although myocardial bridging is mostly asymptomatic and can be an incidental finding on angiography or autopsy, symptomatic patients with severe milking effect may present myocardial ischemia, acute coronary syndromes, exercise-induced dysrhythmias, myocardial stunning, transient ventricular dysfunction, syncope, or even sudden death.[15] The

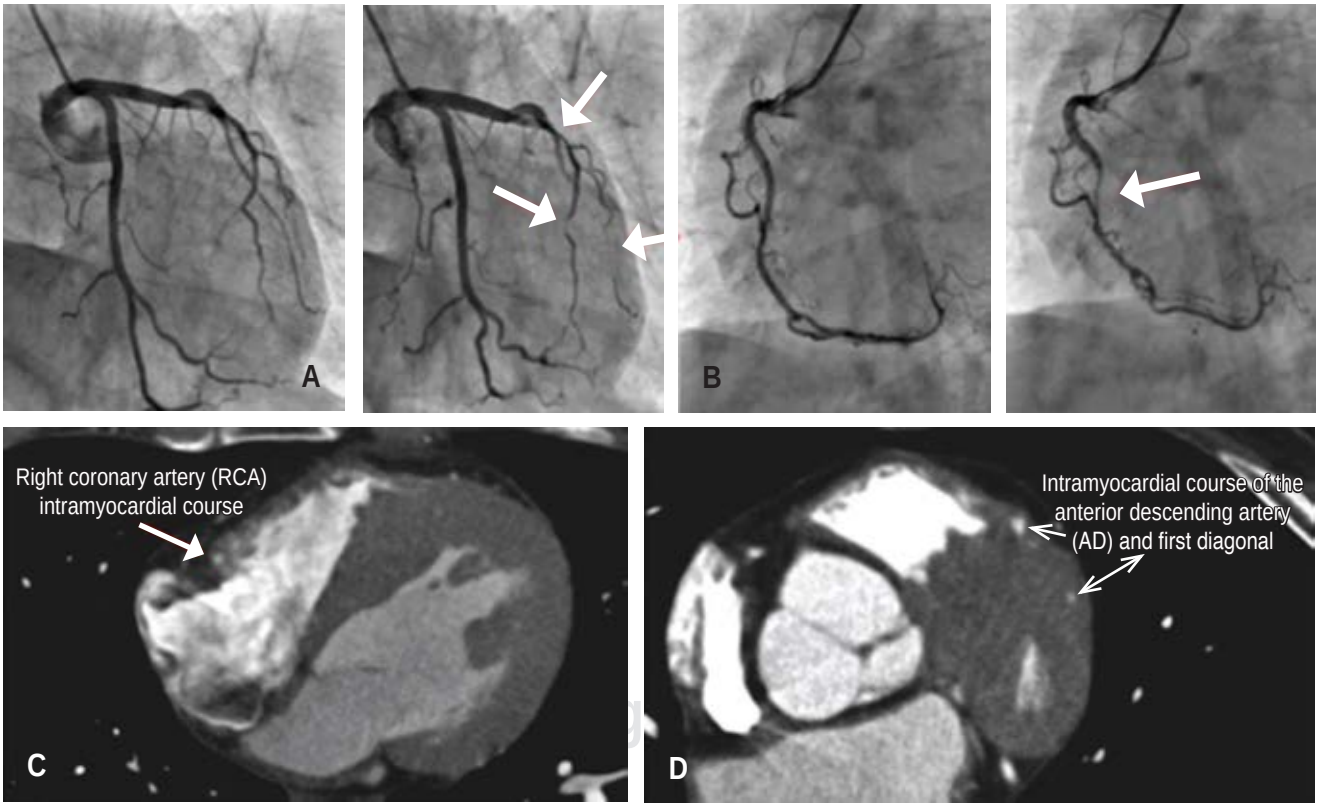


**Figure 1: A)** Myocardial bridging of the middle and distal segments of the left anterior descending coronary artery and first diagonal. **B)** Myocardial bridge of the vertical segment of the right coronary artery with milking phenomenon causing almost total occlusion of the vessels. **C)** Angiotomography shows the intramyocardial course of the right coronary artery. **D)** Angiotomography confirm myocardial bridging of the middle and distal segments of the left anterior descending coronary artery and first diagonal.

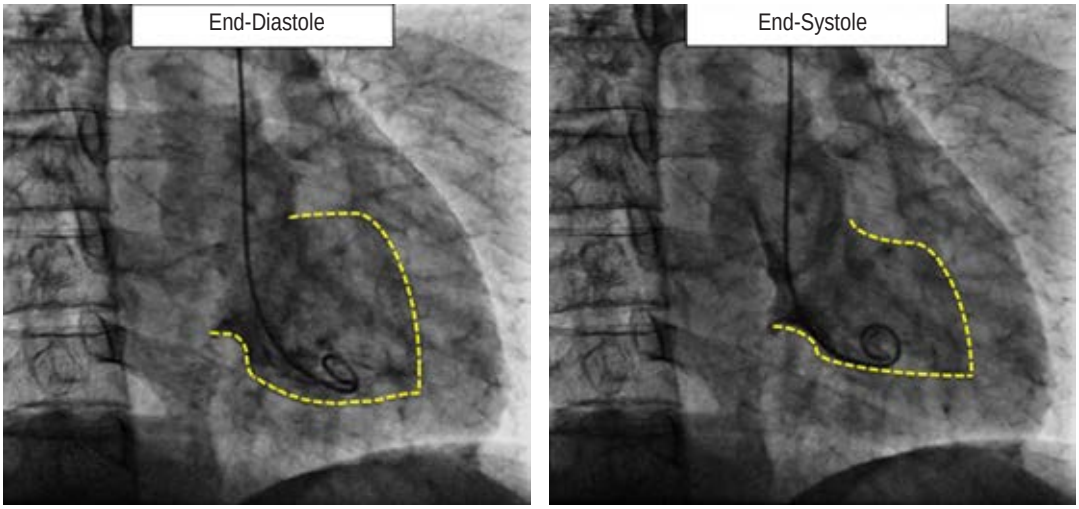


**Figure 2:** Ventriculogram: mild anteroapical hypokinesia and asymetric septal hypertrophy of the left ventricle.

association of hypertrophic cardiomyopathy and MB may occur with ACS. This association has not been ruled out as a possible cause of ischemia and sudden death in these patients.[9]

Medical treatment with beta-blockers and calcium channel blockers remains the mainstay of treatment; nevertheless in patients refractory to intensified medical therapy, surgical intervention, can be a feasible and safe treatment and it should be considered in these patients.[16]

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