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Coronary "Muscular Bridge": A benign anomaly or a dangerous defect to be treated?

Paolo Angelini*

Summary

The subject of MB's of the coronary arteries is typically discussed in the literature on the basis of unusual, but alarming and critical presentations. We propose an update of the currently available concepts and information focusing on the fact that a comprehensive understanding of such a frequent congenital anomaly needs to openly account for its apparent compatibility with normal function, for most of the life of the patient. Recent observations in clinical cases included the use of pressure-wire, Doppler flow-wire, intravascular ultrasound imaging. Each new method introduces additional diagnostic signs, which are characteristic of MB's. Unfortunately, we remain with great uncertainties as to the reasons why a specific clinical event (myocardial infarction, ventricular fibrillation, sudden death, typically) occurs. As a consequence, even though both interventional cardiologists and cardiovascular surgeons have perfected effective interventions to correct the (systolic) compression of the affected coronary artery, both the indication to intervene and the proof of effectiveness are still quite incomplete.

Resumen

"PUENTE MUSCULAR" CORONARIO:
¿TRATAMIENTO PARA UNA ANOMALÍA BENIGNA
O PARA UN DEFECTO PELIGROSO?

En la literatura médica el tema de puente miocárdico de coronarias está discutido típicamente, en el contexto de presentaciones clínicas inusuales, severas y alarmantes. En este artículo revisamos los conceptos actuales en el tema, con la idea fundamental de que una presentación adecuada de esta frecuente anomalía congénita coronaria debe de tener en cuenta que ella es comúnmente compatible con función normal cardíaca en gran parte (o toda) la vida de un paciente. Recientes adelantos diagnósticos han involucrado el uso de hilo de presión, Doppler de flujo y ultrasonidos intravasculares. Cada uno de estos nuevos métodos introduce signos diagnósticos nuevos, que son característicos de esta entidad. Desafortunadamente quedan grandes incertidumbres sobre las causas específicas de eventos clínicos en la vida de un determinado portador de puentes musculares (infarto, arritmias ventriculares, muerte súbita, etc.). Como consecuencia, aun si el cardiólogo intervencionista y el cirujano cardiovascular han perfeccionado instrumentos eficaces y simples para corregir el defecto (compresión sistólica), sea las indicaciones a intervenir, sea las pruebas de eficacia son todavía muy incompletas.

Key words: Coronary muscular bridge. Doppler flow-wire. Intravascular ultrasound imaging.

Palabras clave: Puente muscular coronario. Doppler de flujo. Ultrasonido intravascular.

In humans, most frequently, the larger conductive coronary arteries (and veins) run subepicardially and are visible on the surface of the heart, on gross inspection. Only in some animal species and occasionally in humans, proximal

coronary arteries are observed to deepen into the myocardium for a variable tract.¹ Angiographically, such event is diagnosed by the systolic phasic compression of a coronary segment or the milking effect.² Such anomalous course of a

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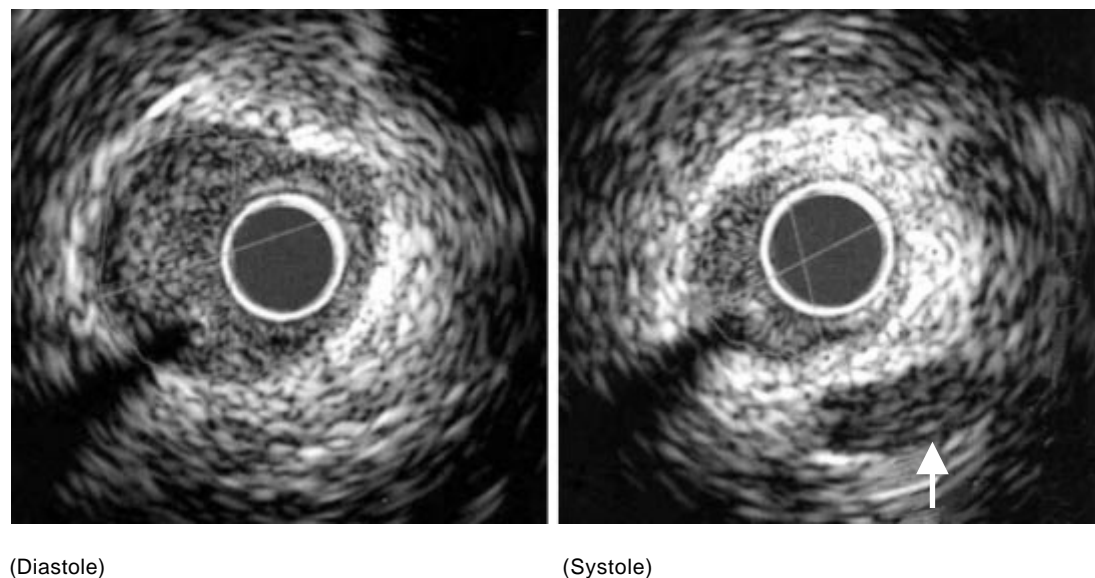


Fig 1. (A, B). Diastolic (A) and systolic (B) IVUS images of a left anterior descending coronary artery; which showed a 50% diameter stenosis in systole at coronary angiography. The systolic cross-sectional luminal area is 2.7 mm², whereas the diastolic is 5.4 mm² (50% area stenosis). In A, the characteristic “half moon” sign (arrows) is present.¹⁴

coronary artery is also called myocardial or muscular bridge (more properly, it should be called “coronary underpass”) and it is observed more frequently in humans at the junction of the proximal to the mid segment of the left anterior descending branch of the left coronary artery, but it has also been described in all the other coronary branches, even though less frequently.

In humans, authors have reported a 25-85% incidence of MB's as investigated by anatomic inspection with or without techniques of microdissection.^{1,3} Clinically, MB's are diagnosed by coronary angiography, whose sensitivity is clearly augmented by the administration of vasodilators (especially nitroglycerin) and myocardial inotropic agents (like isoproterenol or dobutamine), but decreased by purely tone-enhancing agents (ergonovine or neosynephrine).³

Quite consistently, MB's are more frequently encountered in patients with hypertrophic idiopathic cardiomyopathy, especially those with subaortic stenosis.^{1,3} Acquired hypertrophy (as in senile aortic stenosis or hypertension) does not seem to be equally accompanied by increased incidence of MB's.³ In a general population submitted to coronary angiography, the incidence of MB's is between 0.15 and 5% (depending especially on technical factors like the administration of nitroglycerin and the diagnostic criteria that were used).⁴

Functionally, there are no consistent clinical manifestations of MB's, but several cases of MB's have been presented in the literature or observed by clinicians, as accompanied by chest pains, dyspnea, myocardial infarction, ventricular arrhythmias and/or sudden death.⁴

During stress testing, most typically neither chest pain, nor EKG changes, nor especially myocardial reversible scintigraphic deficit of the dependent territory are encountered, but anecdotal exceptions have been presented in the literature (because they are rare).^{4,5} Recently both pressure wire (subselective pressure monitoring during adenosine challenge), intravascular ultrasounds imaging and intravascular Doppler flow measurements have been used to characterize muscular bridges. Typically, MB's are accompanied by mild decrease in coronary flow reserve.⁶⁻⁸ Intravascular ultrasound imaging reveals either a pattern of lateral flattening (parallel to the epicardial plane) or concentric narrowing, in deeper MB's⁷ (Fig. 1). Deep MB's frequently are accompanied by a characteristic halo, called “half moon” sign, probably an artifact due to catheter compression (Fig. 1). Doppler wire recordings typically feature a characteristic decrease or reversal in systolic flow, with increased early peak velocity and mean diastolic flow: the “spike and dome” sign.^{6,8}

The discussion is still open regarding the association of MB's and increased coronary spasticity (inside the affected segment).⁹ Most likely, only occasional patients do have elective, enhanced spasticity at ergonovine or acetylcholine testing, with or without an accompanying clinical history of Prinzmetal angina.¹⁰

Recently, a group of Japanese investigators carried out quite a precise and sophisticated study of the endothelial function in MB's.¹¹ This group concluded that the increased shear stress found at MB's could lead to both a peculiar elongation of the endothelial cells (spindle, in place of polygonal in shape) at this level, while examined on election scanning microscopy,¹² and dramatic changes in the expression of vasoactive agents. Namely, the expression of endothelial nitric oxide synthase, endothelin-1 and angiotensin converting enzyme are clearly decreased at the muscular bridge, as identified by immune histochemical techniques.¹¹ The authors suggested that such findings relate to the substantial absence of arteriosclerotic changes at the level of MB's, in contrast with increased amount of the same changes of the edges of the MB's (especially at their proximal one).¹¹ Considering the possibility that one of the mechanism involved in the causation of clinical events in carriers of MB's could be spasm, the above-mentioned new observations of endothelial dysfunction could be quite relevant. In MB's carriers, who present with acute myocardial infarction angiographic findings sometimes mention the presence of luminal thrombosis, which could also be the likely consequence of endothelial dysfunction.^{3,4}

Concluding such brief review of the pathophysiological mechanism possibly involved in clinical manifestations of MB's, we can summarize that the likely mechanisms are either systolic compression of the MB's segment with a reduced coronary reserve (but in a degree that is not able to cause clinically important or resting ischemia), or enhanced spastic tendency or intravascular thrombosis.

How frequently (and why?) does a muscular bridge become symptomatic? The simple, indirect evidence provided by the fact that such congenital anomalies are widely recognized as compatible with a normal function, both in animals and in humans, tend to suggest that generally MB's are a benign, frequent anomaly that does not require treatment. On the either side, the recurrent presentations of exceptional case repor-

ts in the literature should advice a prudent and rationale attitude, open to the possibility that indeed some patients, either by congenital features of particular severity of the MB's, or by acquired complicating conditions, could become threatened at a certain stage of their life.^{4,10}

The cardiologist should then be alert to the specific features of an individual presentation and should carry out careful diagnostic studies in such cases at the times of the acute events, and compare them to the ones in patients with similar angiographic features but without clinical symptoms.

If a muscular bridge at the end of the diagnostic process, should come to be considered as involved in the causation of symptoms and/or of a grave prognosis, interventions are available, but they should not be considered as a routine indication for any muscular bridge. Both stent angioplasty and surgical treatment have been considered and employed effectively in MB's. Stent angioplasty is particularly interesting because, in the scenario of a relatively simple procedure, it has demonstrated in a few anecdotal cases that both symptoms of angina tend to resolve and coronary hemodynamics normalize.¹²⁻¹⁵ Similarly, surgery of unroofing (resecting the muscular bridge) and aortocoronary bypass have again being reported in small series or individual case reports to cure effectively angina presentation.

Both kinds of interventions have their potential limitations. A few reports are already in the literature mentioning the risk of coronary rupture, following stent implantation.¹⁶ Possibly, such unusual complications is due to unsuspected oversizing of the vessel, which may be smaller than the reference diameter, at the muscular bridge (hypoplasia). Additionally, stents inside MB's reportedly have an increased restenosis rate.¹⁶ The event seems to be particularly frequent when the deployed stent does not cover totally the muscular bridge. Internal mammary arterial bypass is usually preferred to vein grafting, especially in young patients, but its use suffers from an increased rate of failure due to the absence of a significant baseline stenosis, which may lead to graft failure as it prevents the establishment of fistulous flow.¹⁸ Unroofing of deep MB's (the ones that most typically require intervention) is usually uneventful but it may cause perforation into the ventricular cavity (typically the right).¹⁸ This is a tracherous complication, especially when surgery is carried out without extracorporeal circulation.¹⁹

In summary, MB's seem to continue to present as a complex and frequently confusing subject in cardiology. While most of them seem to constitute an innocent anatomic variant, some indeed may cause clinical repercussions by different, generally difficulty-to-prove mechanisms. Interventions are uncertain in their indications and not routine in their executions or results. In general, it should be recognized that the simple

findings of a muscular bridge in patients presenting with chest pain, nor those who present with sudden death, are not by themselves proof of a casual relationship. The most urgent task for the cardiology community is to devise diagnostic tests capable of reproducing clinical events (typically, angina) while allowing to establish an experimental condition, which documents objectively ischemia.

References

1. POLACEK P, ZECHMEISTER A: *The occurrence and significance of myocardial bridges and loops on coronary arteries*. In: V. Krutna, ed. Monography 36, Opuscula Cardiologia. Acta Facultatis Medicinae Universitatis Brunenses. University J.E. Purkinje, Brno; 1968: 1-99.
2. PORTMANN W, IWIG J: *Die intramurale Koronarie im Angiogramm*. Fortschr Roentgenstr 92 (1960), pp. 129-132.
3. ANGELINI P, TRIVELLATO M, DONIS J, LEACHMAN RD: *Myocardial bridges: a review*. Prog Cardiovasc Dis 26 (1983), pp. 75-88.
4. ANGELINI P, VELASCO JA, FLAMM S: *Coronary anomalies. Incidence, pathophysiology, and clinical relevance*. Circulation. 2002; 105: 2449-2454.
5. KRAMER JR, KITAZUME H, PROUDFIT WL, ET AL: *Clinical significance of isolated coronary bridges: benign and frequent condition involving the left anterior descending artery*. Am Heart J 1982; 103: 283-288.
6. FLYNN MS, KERN MJ, AGUIRRE FY, ET AL: *Intramyocardial muscle bridging of the coronary artery – an examination of a diastolic “spike and dome” pattern of coronary flow velocity*. Cathet Cardiovasc Diagn 1994; 32: 36-39.
7. GE J, ERBEL R, RUPPRECHT HJ, ET AL: *Comparison of intravascular ultrasound and angiography in the assessment of myocardial bridging*. Circulation 89 (1994), pp. 1725-1732.
8. SCHWARZ ER, KLUES HG, VOM DAHL J, ET AL: *Functional angiographic and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: effect of short-term intravenous beta-blocker medication*. J Am Coll Cardiol 27 (1996), pp. 1637-1645.
9. TERAGAWA H, FUKUDA Y, MATSUDA K, ET AL: *Myocardial bridging increases the risk of coronary spasm*. Clin Cardiol 2003; 26: 377-383.
10. JOSHI SN, BLACKSHEAR JL: *66 year-old man with long-standing intermittent chest pain*. Mayo Clin Proc 2003; 78: 1405-8.
11. MASUDA T, ISHIKAWA Y, AKASADO Y, ET AL: *The effect of myocardial bridging of the coronary artery on vasoactive agents and atherosclerosis localization*. J Pathol 2001; 193: 408-414.
12. KLUES HG, SCHWARZ ER, VOM DAHL J, ET AL: *Disturbed intracoronary hemodynamics in myocardial bridging. Early normalization by intracoronary stent placement*. Circulation 1997; 96: 2905-2913.
13. MOHLENKAMP S, HOZT W, ERBEL R: *Update on myocardial bridging*. Circulation 2002; 106: 2616.
14. BOURASSA MG, BUTNARU A, LESPERANCE J, TARDIF JC: *Symptomatic myocardial bridges: overview of ischemic mechanisms and current diagnostic and treatment strategies*. JACC 2003; 41: 351-9.
15. HERING D, HORSTKOTTE D, SCHWIMMBECK P, ET AL: *Acute myocardial infarction caused by a muscle bridge of the anterior interventricular ramus: complicated course with vascular perforation after stent implantation*. Z Kardiol 1997; 86: 630-638.
16. HAAGER PK, SCHWARZ ER, VOM DAHL J, KLUES GH, REFFELMANN T, HANRATH P: *Long-term angiographic and clinical follow-up in patients with stent implantation for symptomatic myocardial bridging*. Heart 84 (2000), pp. 403-408.
17. ANGELINI P: *Some questions regarding myocardial bridges still require answers*. JACC 2003; 42: 394-5.
18. PRATT JW, MICHLEER RE, PALA J, ET AL: *Minimally-invasive coronary artery bypass grafting for myocardial muscle bridging*. Heart Surgery Forum 1999; 2: 250-253.

