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Myocardial Bridge Syndrome: A Case Report and Literature Review

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Abstract **Case Report**

Introduction: Myocardial bridging (MB) is a congenital coronary artery malformation. Various clinical symptoms related to MB have been reported; however, subxiphoid pain has rarely been reported. Moreover, it is rarer for electrocardiography (ECG) to show such obvious ischaemia in patients with MB. ***Objectve:*** To report the case of a 65-year-old patient in whom the diagnosis was made during coronary angiography, and whose management was pharmacological with good progress. ***Case Report:*** The patient was 65 years old, with no cardiovascular risk factors other than age and male gender, a former smoker with 20 BPs, admitted to our clinic for management of stress angina that had been present for 3 years prior to his admission, associated with NYHA stage II dyspnea. On admission, the patient was conscious, hemodynamically and respiratory stable, with no signs of right or left heart failure. The ECG was in regular sinus rhythm, with negative T waves in septo-apical, extended to the right leads. Troponin was positive at 110ng/l. TTE was performed showing a non-dilated, non-hypertrophied LV with preserved systolic function LVEF at 72%, preserved segmental and global kinetics, non-dilated atria free of echoes, absence of significant mitro-aortic valve disease, LV size and longitudinal systolic function preserved. A coronary angiography was performed showing a Milking aspect of the anterior interventricular artery, associated with coronary-ventricular fistulas, without significant atheromatous lesions. ***Discussion and Conclusion:*** The myocardial bridge corresponds to a coronary anomaly, most often congenital, in its anatomical relationship to the myocardial muscle, which more frequently affects the anterior interventricular artery. Despite technical advances, angiography remains the reference diagnostic method with a typical image of milking systolic compression. In most cases benign and asymptomatic, it can nevertheless be responsible for serious and even fatal complications. Several therapeutic alternatives are available, ranging from pharmacological treatment to interventional treatment.

Keywords: Myocardial ischemia; Myocardial bridge; Milking effect; Coronary angiography.

INTRODUCTION

The Myocardial Bridge (MB) is defined, anatomically, as being a segment of coronary artery covered, in its epicardial path, by a band of myocardium, thus forming a ``bridge'' (bridge) over this vascular portion. It is a very common congenital coronary anomaly, the incidence of which is between 5 to 85% at autopsy and 0.5 to 2.5% on coronary angiography [1].

Most often asymptomatic, otherwise manifest clinically following a Milking effect in systole heart disease, MB can cause ischemia and even myocardial infarction, left ventricular dysfunction, rhythmic complications and sudden death.

The peak of clinical manifestation occurs during the third and fourth decades of life. MB provokes myocardial ischemia through different mechanisms including supply-demand mismatch, endothelial dysfunction, coronary microvascular dysfunction and external mechanical compression. The association between MB and atherosclerotic disease is controversial. Recent studies established a significant association of MB with myocardial infarction and non-obstructive coronary artery disease.

The first line medical treatment is based on beta-blockers and calcium channel blockers. Ivabradine is used in second line therapy. Invasive approaches involving percutaneous coronary intervention, coronary artery bypass graft and myotomy are performed in patients with symptoms refractory to maximally tolerated medical treatment.

The choice of revascularization technique depends on anatomical characteristics, clinical

condition and physician experience. Available data derived from anecdotal evidence view the lack of randomized clinical trials.

We report the case of a 65-year-old patient in whom the diagnosis was made during coronary angiography, and whose management was pharmacological with good progress.

CASE REPORT

The patient was 65 years old, with no cardiovascular risk factors other than age and male gender, a former smoker with 20 BPs, admitted to our clinic for management of stress angina that had been present for 3 years prior to his admission, associated with NYHA stage II dyspnea.

On admission, the patient was conscious, hemodynamically and respiratory stable, with no signs of right or left heart failure.

The ECG was in regular sinus rhythm, with negative T waves in septo-apical, extended to the right leads. Troponin was positive at 110ng/l.

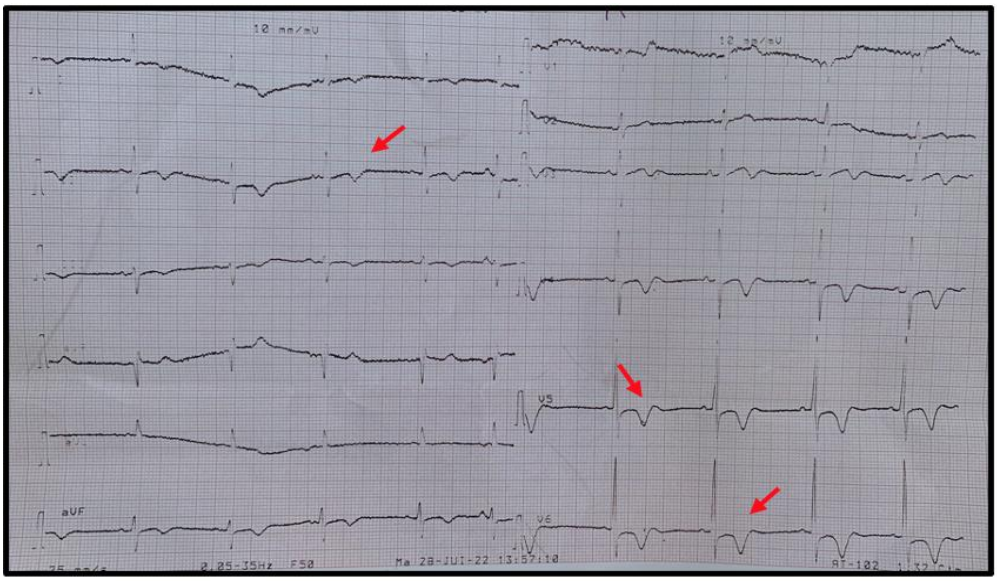

Figure 1: ECG showed negative T waves in septo-apical, extended to right leads

TTE was performed showing a non-dilated, non-hypertrophied LV with preserved systolic function LVEF at 72%, preserved segmental and global kinetics, non-dilated atria free of echoes, absence of significant mitro-aortic valve disease, LV size and longitudinal systolic function preserved.

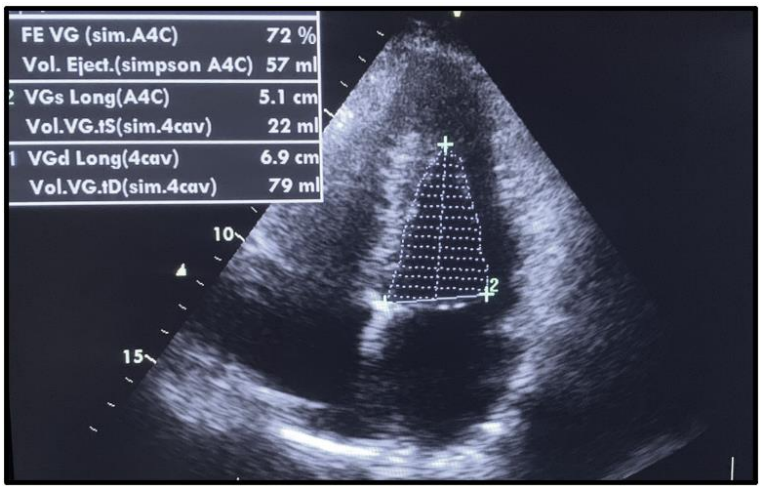

Figure 2: TTE showed a non-dilated, non-hypertrophied LV with preserved systolic function, LVEF 72%, and preserved segmental and global kinetics

A coronary angiography was performed showing a Milking aspect of the anterior interventricular artery, associated with coronary-ventricular fistulas, without significant atheromatous lesions.

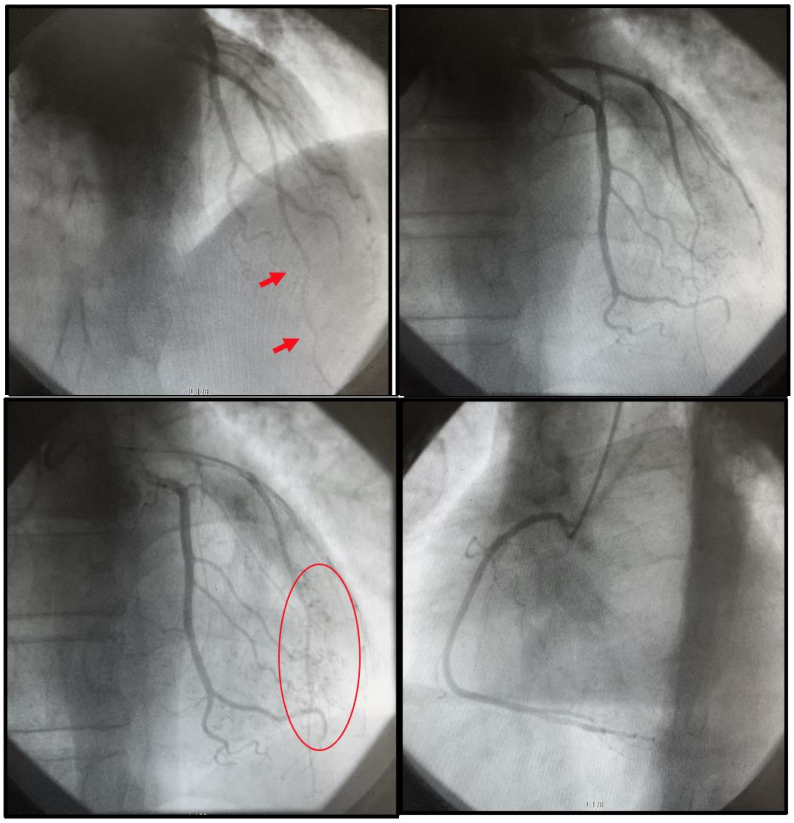

Figure 3: Coronary angiography showed a Milking appearance of the AVI, associated with coronary-ventricular fistulas, without significant atheromatous lesions

The patient was put on 5 mg of bisoprolol with good improvement of the symptomatology.

DISCUSSION

The Myocardial Bridge (MB) is a congenital coronary artery malformation that was discovered 100 years ago. This particularity is defined as a segment of coronary artery covered, in its epicardial path, by a strip of myocardium, thus forming a "bridge" over this vascular portion. In clinical practice, this corresponds to a coronary artery entity characterized by a systolic compression of the coronary artery: this is the milking effect. In histology, there is no direct contact between the myocardial bridge and the adventitia, but rather an interposition of adipose and connective tissue [2].

Myocardial bridging is a common finding (5%-86%) depending on the imaging modality/autopsy series [3]. The detection rate of coronary angiography (CAG) is 2%−6%, that of coronary computed tomography angiography (CCTA) is 19%−22%, and that of autopsy is 33%−42%. The CCTA data are closer to the autopsy data. Therefore, CCTA is more sensitive than CAG, and intravascular imaging and can be used for general diagnosis [4].

The middle segment of the left anterior descending coronary artery is the most frequent location of MB; however, bridging on the circumflex branch of the left coronary artery and right coronary arteries has also been reported.

The reported prevalence of MB in literature is highly variable. This wide variation ensued from the heterogeneity of definitions and modes of evaluation used in the conducted studies.

The phenomenon was first mentioned in the literature in the middle of the 18th century by Reyman [2] and then described again at the beginning of the 20th century, but without knowing its full significance. Although it is an anatomical peculiarity, bridging is not a rarity: the incidence at autopsy ranges from 5 to 85% depending on the series. At coronary angiography, however, the incidence is only 0.5 to 2.5%. This discrepancy is explained by the fact that angiographic detection depends on several factors: the length of the muscle bridge, the orientation of its fibers with respect to the vessel, the extent of compression, and the nature of the tissue interposed between the myocardium and the coronary [1]. The advancement of endo-coronary imaging modalities, diagnostic and functional tests has contributed to better understanding the hemodynamic and physiological significance of MB [2].

In most cases, it is a congenital anomaly, although acquired forms have been described [4], resulting from a geometrical modification of the left ventricle corresponding to myocardial hypertrophy.

Other mechanisms could be involved but remain, at present, imperfectly known.

Atherosclerosis does not seem to play a role in the reduction of luminal diameter: the bridging site is spared but the part immediately proximal to the bridge is affected by varying degrees of atheromatosis [6]. It is indeed in this portion that the flow is the most turbulent, itself a well-known atherogenic factor.

The clinical manifestation is according to the anatomy of the segment concerned, as well as by the concomitant atheromatous changes and a possible myocardial ischemia that the authors Ferreira AG, Ge J *et al.*, [3, 4] report in their articles as the contraction of the overlying myocardium compresses the artery; this compression may persist in diastole, when the majority of coronary blood flow occurs. Increased heart rate, short diastolic infusion time, increased myocardial contractility and blood flow rate, as well as exercise-induced coronary spasm can all cause ischemia in patients with MB.

For many years, MB was considered an entirely benign phenomenon. This was chiefly based on the observation that almost all (~85%) coronary blood flow occurs during diastole, while MB is characterized by systolic arterial compression. Therefore, only approximately 15% of coronary blood flow is at risk of being compromised by significant MB, a seemingly clinically irrelevant fraction. The reality, however, is more complex and is characterized by the interplay between anatomic and physiologic.

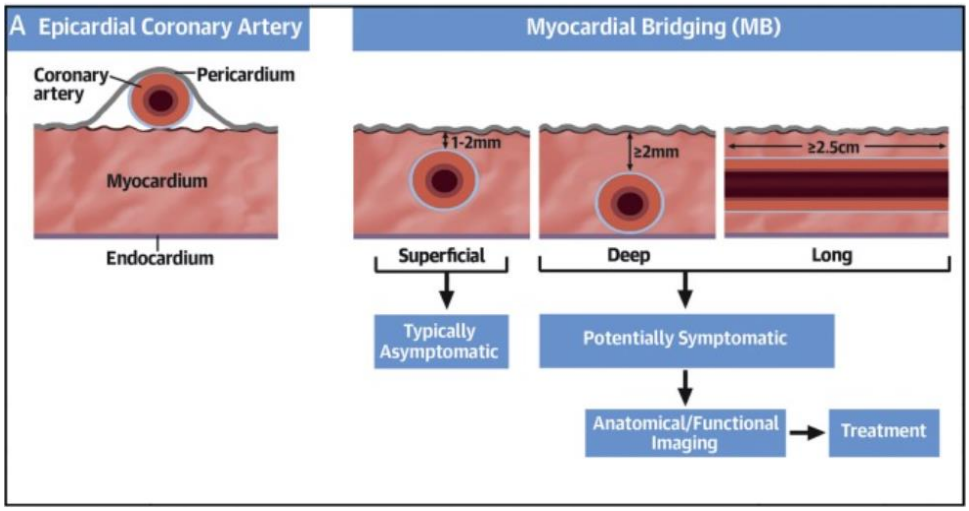

Figure 4: Anatomic propertis of Myocardial Bridging [5]

The mechanical compression is the main player in the pathophysiology of ischemia in MB. In fact, coronary filling occurs in diastole, and subsequently a transient systolic narrowing should not significantly impair the myocardial perfusion [8]. However, IVUS based studies revealed a persistent narrowing of coronary diameter during the early diastolic phase compromising coronary blood flow [9, 10]. There is an important role of this expansion of systolic compression into diastole in the pathophysiology of myocardial ischemia in MB. Up to 51% continual reduction in tunneled segment diameter was noted during diastole for a mean time of 136ms [11].

For this reason, situations that decrease diastolic coronary filling time like tachycardia may precipitate or amplify the clinical manifestations of MB.

A parallel interrelation between the degree of systolic compression and reduction in coronary diastolic lumen and myocardial perfusion was also observed. In the presence of significant obstructive coronary artery disease (>50% luminal stenosis), MB is considered critical. It can have detrimental impact on the left ventricular systolic and diastolic functions affecting the stroke volume. This negative outcome is exacerbated by the length of bridging segment and coexistence of arterial hypertension [6].

Intra-coronary Doppler has been used to assess the hemodynamic significance of MB. The spike and dome sign also called finger-tip phenomenon is likely suggestive of MB. This characteristic pattern reflects the fast increase in flow velocity at early diastole followed by quick decline in late diastole due to the delay in relaxation [7].

Rather than shortening diastolic time that compromises coronary filling and subsequently myocardial perfusion aforementioned, systolic kinking results in endothelial dysfunction and reduced expression of vasoactive-agents, like nitric oxide synthase, angiotensin converting enzyme and endothelin-1[6]. All of these changes predispose the bridged segment to vasospasm and thrombus formation leading to acute ischemic event [8].

Initially considered as a silent anatomical variant, the clinical relevance of MB has been debated for a long time. MB is associated with serious cardiovascular outcomes, like major adverse coronary events, myocardial infarction, myocardial ischemia and sudden death. Recently, the association between MB and MINOCA (myocardial infarction with non-obstructive coronary arteries) has been raised [9]. It is noteworthy that the peak of clinical manifestation of MB occurs during the third and fourth decades of life. To date, in the absence of randomized clinical trials, there are no standard guidelines for the optimal management of MB [10].

Medical management with negative inotropic and negative chronotropic agents is considered first-line therapy [11]. β Receptor blockers can slow down the heart rate, increase the diastolic filling time and reduce the arterial pressure in the tunnel section. Calcium channel blockers (CCBs) are also commonly used to treat symptomatic myocardial bridges, especially for patients with myocardial infarction. CCBs are preferred in patients with receptor blocker contraindications (such as bronchospasm).

In addition, CCBs cause vasodilation and may be beneficial to patients with vasospasm. If the ischaemia of myocardial bridge patients cannot be alleviated by drugs, percutaneous transluminal coronary intervention (PCI) is not impossible, but there are some disputes. Because the radial pressure of the myocardial bridge on the stent is significantly greater than that of atherosclerosis, there is a high risk of coronary perforation, stent rupture, in-stent restenosis (and in-stent thrombosis. After strict screening of patients, PCI could be considered in patients who had poor drug treatment effects, a short life expectancy or surgical contraindications [6].

Surgery is an effective treatment for symptomatic MB patients for whom drug treatment is ineffective, and these surgical techniques include coronary artery bypass grafting (CABG) and surgical unroofing. CABG is more suitable for patients with deep (> 5 mm) or long (> 25 mm) myocardial bridges [12, 13]. There is a risk of ventricular wall perforation after myocardial bridge resection, but Hemmati P *et al.*, confirmed that surgical unroofing is a safe option for patients with isolated MB [14].

Symptomatic patients should be treated conservatively interventional or surgical treatment, depending on the clinical situation depending on the clinical situation. Drug therapy remains the remains the gold standard of treatment for patients with symptomatic symptomatic myocardial bridges. Nevertheless, in case of refractory symptoms or proven ischemia, surgical treatment (myotomy surgical treatment (myotomy, coronary artery bypass grafting) represents the most the most effective and durable therapeutic alternative. Active stent angioplasty angioplasty with active stents could represent an alternative treatment alternative for patients who cannot be operated on despite a high risk of risk of restenosis and stent fracture. At the end of this this article, we propose the management algorithm shown in management algorithm shown in Figure 5 [5].

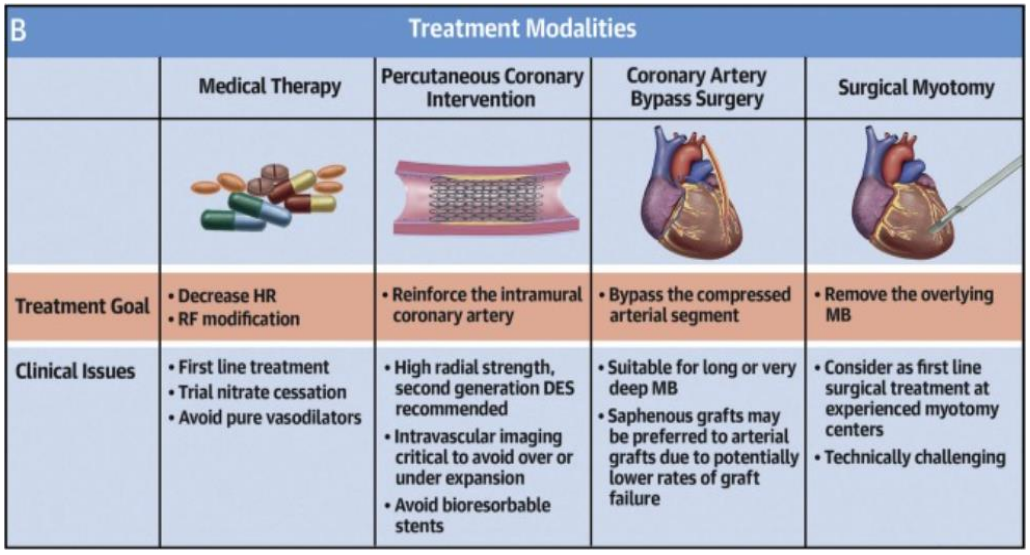

Figure 5: Overview of treatement modalities of Myocardial Bridging [5]

CONCLUSION

Myocardial bridging appears in many cases to be asymptomatic and, therefore, is often ignored diagnosis. It is one more and more reported anomaly, which attests to its frequency and the importance of looking for it. It can be asymptomatic or result in a clinic of myocardial ischemia or outright myocardial infarction. Its screening involves invasive cardiac exploration with treatment of choice for each patient.

MB is potentially implicated in the pathophysiological mechanisms of several cardiovascular entities like MINOCA and myocardial ischemia. Its association with atherosclerosis is ambiguous in view of the controversial results between old and recent data. Medical therapy including beta-blockers, non-dihydropyridine calcium channel blockers and ivabradine is the first therapeutic approach in symptomatic.

It should also be noted that it can be very useful to detect it in patients with coronary atherosclerosis who are to undergo bypass surgery since; in this case, the site of the distal anastomosis will inevitably be influenced. Nevertheless, it is advisable to keep this possibility in mind, especially in the presence of ischemic signs, and all the more so when the individual is young and an atheromatous context is not evident.

This interesting phenomenon is therefore not unknown to the medical world, it remains a subject for future studies to further improve our understanding.

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