

### **Myocardial Infarction in young patient on myocardial bridge revealed by ventricular arrhythmia: A case report and literature review**

#### **Abstract**

**Background:** Myocardial bridging (MB) is a congenital coronary anomaly with normal coronary epicardial artery taking an intra myocardial pathway also described as tunneled artery. This benign anatomical abnormality usually can manifest as an acute coronary syndrome or fatal ventricular arrhythmia.

**Methods:** We present an unusual case of a young male patient with a history of MI on MB who presented at the emergency room for constrictive retro sternal chest pain associated with palpitations evolving for 24 hours with ventricular tachycardia on the ECG reduced by pharmacological therapy. Coronary angiography showed a myocardial bridging at the level of the distal left interventricular artery without atheromatous lesions neither in the upstream or downstream of the vascular bed nor in the other coronary networks. Cardiac MRI findings showed a predominantly left biventricular dilated cardiomyopathy of ischemic origin with severe LV dysfunction.

**Discussion:** MB can be associated with serious manifestations such as syncope, myocardial ischemia, coronary spasm, supraventricular or ventricular arrhythmias, atrioventricular block, exceptionally a coronary syndrome or even sudden death. Our case is one of the rare case reports of MB manifested as myocardial ischemia complicated with ventricular arrhythmias in a young patient without an evident cardiovascular risk factor.

**Conclusion:** Management decision on myocardial bridge remains controversial but medical therapy consist of the first therapeutic line. This is a case of a complicated myocardial bridge with a proof of effectiveness of medical therapy.

**Keywords:** Myocardial Bridge, left interventricular artery, Myocardial Infarction, Ventricular arrhythmias

#### **Introduction**

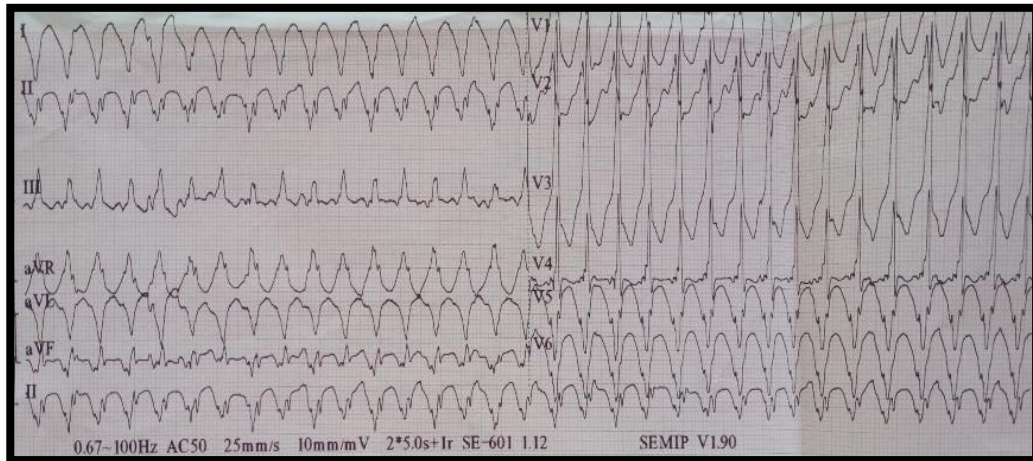
Myocardial bridging (MB) is a congenital coronary anomaly with normal coronary epicardial artery taking an intra myocardial pathway also described as tunneled artery. This benign anatomical abnormality usually can manifest as an acute coronary syndrome or fatal ventricular arrhythmia.

We report a case of a MB involving the anterior interventricular artery (AIV) confirmed by coronary angiography complicated by ACS and sustained ventricular tachycardia in a 38-year-old young subject without an evident cardiovascular risk factor.

### **Case Report**

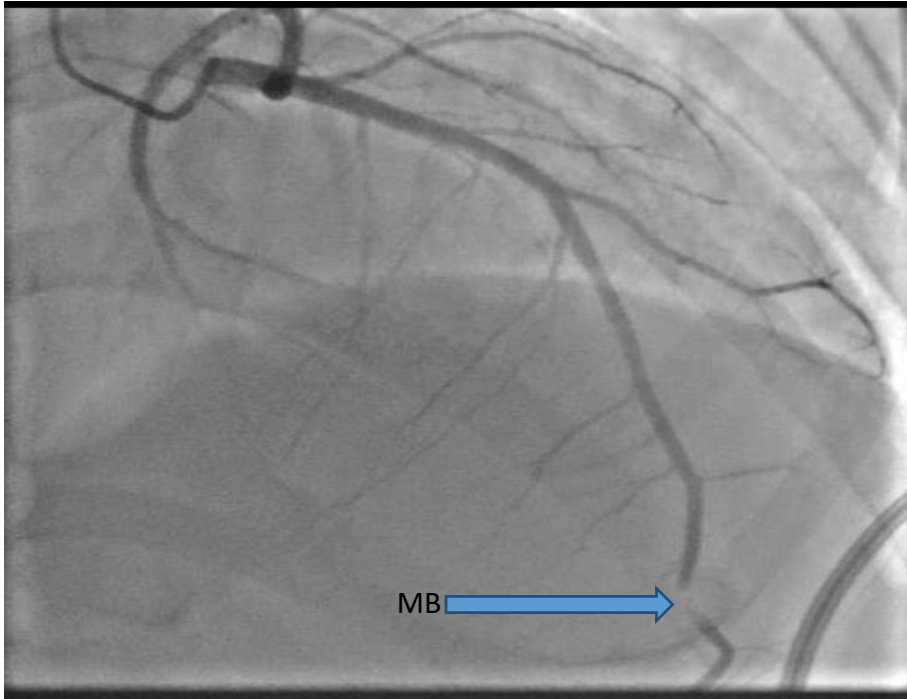
A 38-year-old patient, chronic consumer of traditional plant-based toxic products presented to the emergency room for constrictive retro sternal chest pain associated with palpitations evolving for 24 hours. Patient has a history of an infero-lateral myocardial infarction (MI) one month interval before admission to our emergency department with a documented coronary angiography diagnosis of a myocardial bridge at the level of the distal segment of the anterior interventricular artery without atheromatous lesions upstream or downstream of the vascular bed or on the other coronary networks. Clinical examination noted blood pressure at 100 / 70mmhg, heart rate at 230 beats/min, respiratory rate at 17 cycles / min, oxygen saturation at 99% in open air, and a body temperature of 36.5 ° C. The cardiorespiratory physical examination was normal.

The ECG showed a sustained ventricular tachycardia at 250bpm with right delay (Figure 1). The diagnosis of ventricular rhythmic disorder on myocardial bridging secondary to myocardial ischemia was initially retained.



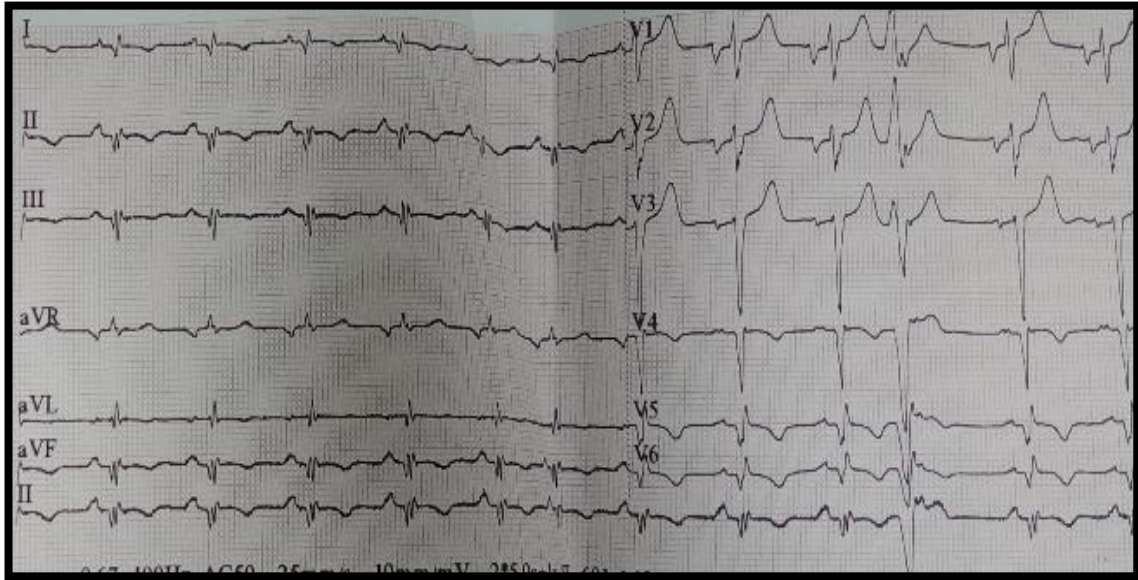
**Figure 1:** ECG showing sustained ventricular tachycardia with right delay.

Trans-thoracic ultrasound revealed a bi-ventricular dilated heart disease with hypokinesia of the anterior, anterolateral and inferior walls at the level of its septal and lateral segments with a 20% LVEF measured by biplane Simpson. Coronary angiography performed for residual angina showed a Myocardial Bridge along the distal interventricular artery (milking syndrome effect) without associated atheromatous lesions and coronary spasm after vasoactive test. (Figure 2).



**Figure 2:** Coronary angiogram showing a myocardial bridge (MB) of the distal portion of the anterior interventricular artery  
("Milking" effect)

Cardiac MRI revealed a predominantly left biventricular dilated cardiomyopathy (LVTDV= 177ml / m<sup>2</sup>, RVTDV = 119ml / m<sup>2</sup> of ischemic origin with severe LV dysfunction), with the presence of late contrast enhancement in the form of late enhancement of the endocardial under transmural region involving the apex, the anterolateral wall at the level of the three segments, the anterior and inferior walls at the level of the middle and apical segments. His blood analyses revealed an elevation of the ultrasensitive troponin at 6389ng / ml. The blood electrolytes, kidney and liver function, blood sugar and hemostasis results were normal. Blood analyses for the search of COVID- 19 infection came out negative for SARS-COV2. The final diagnosis retained is myocardial ischemia complicated by heart disease arrhythmia on myocardial bridge.



**Figure 3:** ECG after pharmacological reduction showed frequent ventricular extrasystole.

A treatment based on a pharmacological reduction of the ventricular tachycardia was performed as a first-line treatment with the I.V infusion of an attack dose of cordarone<sup>R</sup> (Amiodarone hydrochloride) 300mg in 30minutes after assuring hemodynamic stability of the patient. A reduction of the ventricular rate under the maintenance dose of cordarone<sup>R</sup> was marked initially with the appearance of monomorphic ventricular extrasystoles treated

with a cardioselective beta-blocker such as bisoprolol 2.5 mg per day (Figure 3) titrated to its maximum tolerated dose by the patient.

Complementary treatment with aspirin at a dose of platelet anti-aggregation, beta-blocker (bisoprolol 2.5mg per day), anti-vitamin K type acenocoumarol (sintrom 40mg 1 / 2cp per day) and spironolactone (aldactone 50mg 1 / 2cp per day) was initiated in the patient on discharge from the hospital.

The clinical outcome was favorable after 1 month of treatment with disappearance of symptoms and regression of the number of ventricular extrasystoles on the 24hr control ECG holter (15 Ventricular extrasystole max / H; 142 / 24H versus 49 Ventricular extrasystole max / H; 721 / 24H).

## **Discussion**

Congenital coronary artery is characterized by an intra myocardial pathway of a segment of an epicardial coronary artery (1, 3), the myocardial bridge (MB) was initially described from autopsy of cases by Reymann in 1737 and then using coronary angiography method by Portsmann in 1960 (2,3).

This morphological abnormality, as in our patient, mainly affects the anterior interventricular artery (AIV) (3, 4) without known risk factors for atherosclerosis apart from consumption of herbal plants (2, 5).

The prevalence of myocardial bridges varies depending on the screening methods. It is relatively low on angiography of the order of 0.8 to 4.9% compared to that reported by autopsy studies (2, 6, 14).

In the majority of cases, this is a benign abnormality and patients are asymptomatic because the myocardial perfusion is predominantly diastolic (3, 5, 8). However, as reported by

Hosticue et al. It can be associated with serious manifestations such as syncope, myocardial ischemia, coronary spasm, supraventricular or ventricular arrhythmias (VT), atrioventricular block, exceptionally a coronary syndrome or even sudden death (2,7,8, 9). The particularity in our case, is the richness of the clinical presentation of sustained ventricular tachycardia at 230 beats per minute tolerated by the patient during 24hrs before admission without hemodynamic instability with an altered ventricular ejection fraction.

The hemodynamic influence of the bridge on coronary vascularization depends on the length, depth (> 2mm), number of tunneled arteries and the severity of the systolic reduction in diameter (5,9). This effect of myocardial bridging may be exacerbated by some factors such as age, heart rate, left ventricular hypertrophy and coronary atherosclerosis (5,10,13). Mechanisms of myocardial ischemia are complex and include systolic compression, coronary spasm, coronary dissection and proximal athero-thrombotic changes to the bridged segment due to endothelial dysfunctions secondary to turbulent forces induced by the "milking" effect (6,7,9,10).

Despite the progress in imagery cardiac, angiography remains the gold standard examination with the typical appearance of systolic stenosis also called the <<milking>> effect at the level of the epicardial artery (5,7,13) In clinical case the diagnosis is evident on angiography with typical <<milking>> effect as in our case. A systolic reduction of at least 70% in mean diameter of the tunneled artery is generally considered significant, it may persist and reach 35% reduction in diastole (2,14). According to Hirokib et al. this decrease in systolic and diastolic coronary flow can alter the coronary reserve in these patients (6) and thus explain the sign of "half moon" observed on endocoronary ultrasound (IVUS). An obvious diagnosis on angiography, the use of nitroglycerin, or functional tests such as

fractional flow reserve (FFR), IVUS, and optical coherence tomography (OCT) can be of great help (2,3). Myocardial bridging can also be viewed simply and reliably on the Coroscanner (3,9,11).

Various therapeutic strategies are available for the management of patients with symptomatic myocardial bridges (2,3,6). Certain associated comorbidities such as coronary artery disease as in our patient, Takotsubo cardiomyopathy, hypertrophic cardiomyopathy may affect the choice of treatment (2,12). The Schwarz classification in 3 stages from A to C, can be used as a guide to choose between a medical, endovascular or surgical treatment. Beta-blockers (BB) are recommended in first intention in this therapeutic pyramid because of their chronotropic and inotropic effects that prolong the duration of diastole, which improves coronary perfusion and reduces systolic compression of the bypass segment (2,3,5). Our patient received bisoprolol as a beta-blocker (BBs) at maximum tolerated dose. According to the literature, calcium channel blockers (CCBs) are an alternative in case of intolerance or contraindication to BBs (2, 3) Ivabradine may be an alternative to BBs and CCBs, nitrates are contraindicated in patients' carriers of symptomatic myocardial bridging (2,6). Anti-platelet aggregation treatment also finds its place in view of the increased risk of major cardiovascular events (2, 3,6). In our case, because of the recurrence of cardiovascular ischemia event, acetyl salicylic acid as an anti-aggregant platelet therapy and acenocoumarol as anticoagulant therapy was administered to the patient.

Surgery by coronary artery bypass grafting associated or not with decompression myotomy may be proposed when the symptoms persist despite appropriate medical treatment (2, 3) whereas in our case a medical treatment was opted in the first place. Revascularization by placing a stent presents significant complications such as restenosis, intra-stent thrombosis, stent fracture; It should only be used when patients are not surgical candidates (2, 4, 15).

Symptomatic myocardial bridging is generally of good prognosis under medical treatment, few patients will require surgery (9, 11). Our patient responded well to first-line treatment, improving chest pain while maintaining sinus rhythm before discharge from hospital.

### **Conclusion**

Although very common but most often benign, MB can have serious manifestations that could be either ischemic, arrhythmic or both. Medical treatment for MB should be considered as first-line therapy in most patients before invasive treatment, and abstinence therapy should be avoided in confirmed symptomatic patients.

### **Informed consent**

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

### **References :**

1. Yukio I, Yoko K, Ehiichi K, Toshiharu I. (1) Coronary Events Caused by Myocardial Bridge. *Ann Vasc Dis.* 2009;2(2):79-94.
2. Murtaza G, Mukherjee D, Gharacholou SM, Nanjundappa A, Lavie CJ, Khan AA, et al. An Updated Review on Myocardial Bridging. *Cardiovascular Revascularization Medicine.*2020;21(9):1169-79.
3. Daoud B, Pongas D, Beauchant T, Terrazonni S, Villemant D, Pernès J-M. Un pont myocardique ischémique. *Sang Thrombose Vaisseaux.*2016;28(2):85-90.
4. Ripa C, Melatini MC, Olivieri F, Antonicelli R. Myocardial bridging: A 'forgotten' cause of acute coronary syndrome – a case report. *Int J Angiol.* 2007;16(3):115-8.
5. Tarantini G, Migliore F, Cademartiri F, Fraccaro C, Iliceto S. Left Anterior Descending Artery Myocardial Bridging: A Clinical Approach. *J Am Coll Cardiol.*2016;68(25):2887-99.
6. Teragawa H, Oshita C, Ueda T. The Myocardial Bridge: Potential Influences on the Coronary Artery Vasculature. *Clin Med Insights Cardiol.*2019;13:1-6.
7. Corban MT, Hung OY, Eshtehardi P, Rasoul-Arzrumly E, McDaniel M, Mekonnen G, et al.

- Myocardial bridging: contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. *J Am Coll Cardiol*. 2014;63(22):2346-55.
8. Raouf Madhkour Hatem Ksouri Jacques Noble Fabien Praz Bernhard Meier. Le pont myocardique : mise au point. *Rev Med Suisse*.2019;55(655) :1232-38.
  9. Zhu C-G, Liu J, Liu W-D, Xu Y-L, Wu N-Q, Guo Y-L, et al. Myocardial infarction caused by myocardial bridging in a male adolescent athlete. *J Cardiovasc Med (Hagerstown)*. 2012;13(2):138-40.
  10. Möhlenkamp S, Hort W, Ge J, Erbel R. Update on myocardial bridging. *Circulation*. 12 nov 2002;106(20):2616-22.
  11. Tarantini G, Cademartiri F. Myocardial bridging and prognosis: more evidence but jury still out. *European Heart Journal - Cardiovascular Imaging*. 2013;14(6):515-7
  12. Krishnaswami A, Maurer MS, Alexander KP. Contextualizing Myocardial Infarction: Comorbidities and Priorities in Older Adults. *The American Journal of Medicine*.2017;130(10):1144-7.
  13. Papp S, Bárczi G, Karády J, Kolossváry M, Drobni ZD, Simon J, et al. Coronary plaque burden of the left anterior descending artery in patients with or without myocardial bridge: A case-control study based on coronary CT-angiography. *Int J Cardiol*.2021;327:231-5.
  14. Matta A, Canitrot R, Nader V, Blanco S, Campelo-Parada F, Bouisset Fet al. Left anterior descending myocardial bridge: Angiographic prevalence and its association to atherosclerosis. *IHJ*.2021, 73(4):429-433
  15. Lee MS, Chen C-H. Myocardial Bridging: An Up-to-Date Review. *J Invasive Cardiol*.2015;27(11):521-8.



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Video of Myocardial Bridging 'Milking effect'