

Myocardial Bridging: Therapeutic and Clinical Development

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Abstract

Background: The myocardial bridge constitutes one of the main differential diagnoses of coronary artery disease. However, it remains an underdiagnosed condition and its physiopathological mechanisms and therapeutics are yet to be elucidated.

Objectives: To analyze and describe the clinical and therapeutic evolution of patients with an angiographic diagnosis of myocardial bridge, comparing the data with that in the current literature, in order to clarify the patients' clinical profile and prognosis.

Methods: The results of coronary angiographies carried out from 2003 to 2007 in a Laboratory of Hemodynamics were reviewed; the analysis of patients' files was carried out and selected patients were interviewed.

Results: The frequency of myocardial bridge diagnosis was 3.6%. The mean age of patients was 56.8 years (SD = 11.83; CI = 0.73). The anterior descending artery was affected in isolation in 100% of the cases. After the selection, the analysis and interview of 31 patients were carried out. There was no correlation between symptoms and degree of angiographic narrowing observed in the studied patients. The drug treatment included the use of beta-blockers, calcium-channel antagonists, platelet antiaggregants and/or nitrates and resulted in clinical improvement in 30%, absence of alterations in the clinical picture in 60% and symptom worsening in 10% of the patients. One patient presented sudden death; two patients underwent angioplasty followed by significant clinical improvement and none of the patients underwent surgical procedures.

Conclusion: Most of the patients with myocardial bridge have a good prognosis, but in the long term, there are not enough data, obtained from a large sample of symptomatic patients, to draw definitive conclusions. (Arq Bras Cardiol 2010;94(2): 175-181)

Key Words: Coronary artery disease/therapy; myocardial bridging; angioplasty, transluminal, percutaneous coronary; cohort studies.

Introduction

The myocardial bridge (MB) is a congenital anomaly of the coronary arteries that usually affect the left anterior descending artery (ADA), when one or more myocardial bundles cross or involve a segment of the epicardial coronary artery, which crosses the intramural portion of the myocardium, below the muscular bridge. The MB constitutes one of the main differential diagnoses of coronary artery disease (CAD) and can manifest as typical or atypical angina pectoris and, more rarely, as acute myocardial infarction (AMI) or sudden death.

It is a relatively common and usually benign pathology among the general population, affecting mainly patients at low risk for CAD; however, when symptomatic, it can manifest

as unstable or stable angina, cardiac arrhythmias (ventricular tachycardia and supraventricular tachycardia), AMI and sudden death, with the latter two being rare¹.

It remains underdiagnosed due to the fact that only a minority of patients present symptoms as well as the lack of availability and, consequently, the restricted use of more accurate diagnostic methods and therefore, its physiopathological mechanisms and therapeutics have not been fully elucidated.

The present study aimed at analyzing the clinical and therapeutic evolution of patients with an angiographic diagnosis of myocardial bridge, from 2003 to 2007, comparing the data with those in the current literature, in order to elucidate the clinical profile and the evolution of these patients.

Methods

This is an observational, cross-sectional cohort study that used statistical tools for the analysis of the level of significance of 5%. The researcher was fully responsible for the costs of the

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materials used in the study. The study was carried out through the analysis of medical files and by applying a questionnaire to a group of selected patients.

The study was carried out in a Laboratory of Hemodynamics after being approved by the Ethics Committee of the institution. The patients' confidentiality and the use of the data for scientific research purposes only were warranted. After the pre-selection of the medical files, the patients or their tutors were contacted, the study objective was explained to them and the Free and Informed Consent Form was signed by all participants, which had been previously approved by the Scientific Committee in Research and Ethics of the Institution.

Patients aged 20 to 75 years that had been submitted to a coronary angiography from 2003 to 2007, due to suspected and undergoing investigation for probable CAD, in whom the angiographic results showed the presence of MB, were evaluated. Patients who did not present associated cardiac pathologies capable of producing angina symptoms were also assessed: cardiac valvular disease or myocardopathies that had been previously diagnosed or were diagnosed at the coronary angiography and the presence of CAD at the coronary angiography.

Patients with neurological pathologies that prevented the correct use of the questionnaire and those who available data were incomplete or outdated, which prevented the contact for the interview, were excluded.

Although the patients were included in the study based on the coronary angiographies carried out before the study, all of them were carried out according to the protocol of the Laboratory of Hemodynamics of the institution, following the Judkins technique, in which the phenomenon of systolic compression of the descending coronary artery can be clearly observed.

This protocol did not include the intracoronary administration of nitroglycerin.

Results

The results of 3,375 coronary angiographies, carried out from 2003 to 2007, were assessed, of which 123 presented the phenomenon of systolic constriction of the left ADA (Figure 1), with a diagnosis of MB. The frequency of diagnoses of MB in coronary angiographies performed within this period was 3.6%. The mean age of patients was 56.8 years (SD=11.83; CI=0.73). All patients presented only one affected coronary artery and the left ADA was the affected one in 100% of the cases. Thirty-four patients (27.6%) presented CAD that affected the proximal segment of the bridge at different degrees. The involvement of the left ADA at the coronary angiography was classified as Grade 1 ($\leq 49\%$), grade 2 (50-74%) and grade 3 ($\geq 75\%$) (Table 1).

After the patient selection, 81 were excluded from the study: 68 due to associated CAD (55.28%), 17 due to myocardopathy (13.82%) and 21 due to valvulopathy (17.07%). A total of 42 patients were selected for the study, of

Table 1 - Degree of narrowing of the anterior descending artery at the coronary angiography

Degree of narrowing	Patient % (n°)
Grade 1: < 49%	50.41 (62)
Grade 2: 50%-74%	36.59 (45)
Grade 3: > 75%	13.01 (16)
Total	100 (123)

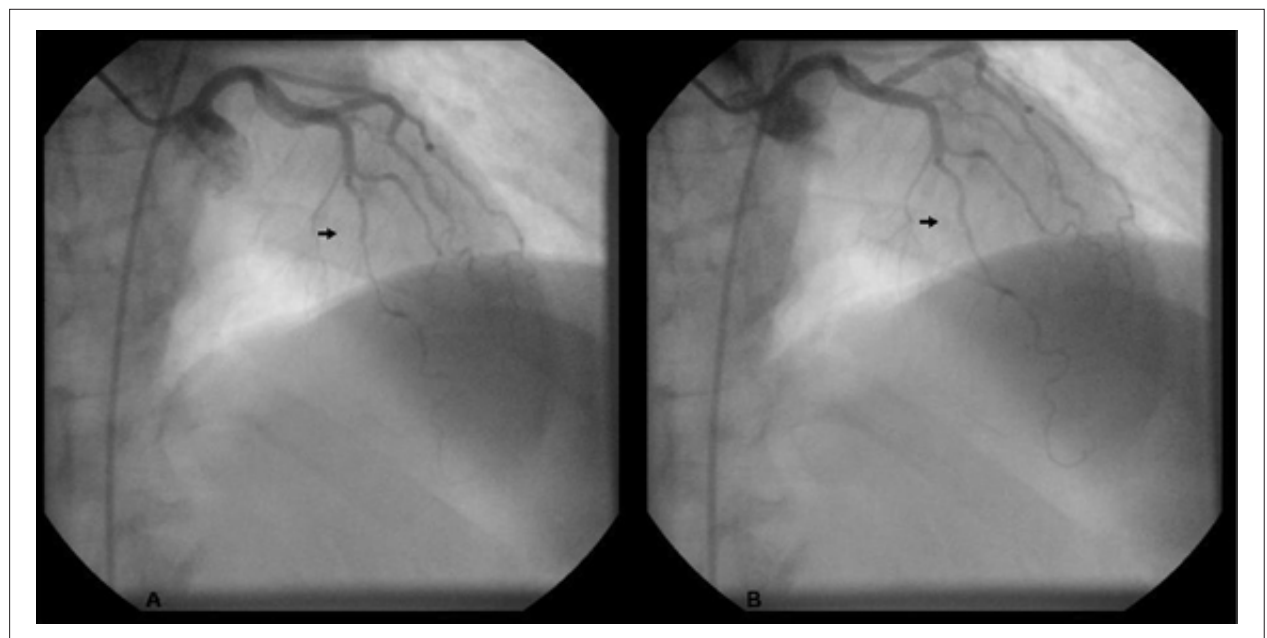


Figure 1 - Coronary angiography performed in a 56-year-old female patient that presented compression of the left anterior descending artery, with an 80% decrease in vessel lumen diameter (arrow) at systole (A) and persistence of the phenomenon at diastole (B), with slight vessel dilatation (arrow).

whom one presented a neurological pathology that prevented the interview to be carried out and in 10 cases, the interview could not be carried out as the available data were incomplete or outdated, which prevented the contact with these patients, resulting in the analysis and interview of 31 patients.

The mean age at the symptom onset among the studied patients was 45.9 years (CI=11.7; SD=4.2) and in 50% of them, the diagnosis of MB took \geq one year. The analysis of the degree of narrowing of the left ADA was performed (Table 2). The patients' symptoms were classified according to the classification proposed by the Canadian Cardiovascular Society Grading Scale for angina pectoris, before and after the treatment (Table 3).

According to this classification, patients with grade 1 of systolic narrowing at the coronary angiography presented the classifications CCS-I (31.6%), II (31.6%) and III (31.6%). Patients with grade 2 of narrowing were classified as I (11.1%), II (11.1%), III (66.7%) and IV (11.1%). Patients with grade 3 were classified as II (33.3%), III (33.3%) and IV (33.3%).

A patient who died due to sudden death was excluded from the analysis of symptoms; this patient presented grade 1 systolic narrowing of the ADA. Two patients that underwent angioplasty presented grade 1 narrowing and were both classified as CCS-III; they were later reclassified as classes I and II after the procedure.

Based on these data, one concludes that there is no correlation between symptom classification and the degree of angiographic narrowing observed in the studied patients. However, the intensity of these symptoms also depends on the number, thickness, location and length of the MB, which vary from patient to patient and can even vary in a same patient, from one evaluation to another. The adverse events reported by the patients were: ischemia and acute coronary syndrome, AMI and sudden death (Chart 1).

There was a significant variation regarding the time of treatment performed and the therapeutic regimens used by

the patients. The drug treatment included the use of beta-blockers, calcium-channel antagonists, platelet antiaggregants and/or nitrates. Two patients underwent angioplasty and none of the patients was submitted to surgical procedure. Nitrates were used by 43.3% of the patients, associated or not to other drugs, in spite of reports of the worsening of symptoms and clinical picture in the literature. However, it was not possible to assert the real impact of these medications on the clinical outcome of these patients.

Of the patients that presented CCS-I classification before treatment, all remained at the same classification after the treatment. Of the patients that presented CCS-II classification before the treatment, 37.5% remained at the same class after the treatment, 50% became class I, with clinical improvement and 12.5% presented clinical worsening and became class III. Of the patients that presented CCS-III classification before treatment, 53.8% remained at the same classification after the treatment, 30.8% presented improvement to classes I or II and 15.4% presented symptom worsening and became class IV. Of the patients that presented CCS-IV classification before the treatment, 50% remained at the same classification after the treatment and the other 50% presented clinical improvement and became class III. This group included two patients with classification III that underwent PCI and maintained the same drug treatment, presenting clinical improvement.

In conclusion, 30% of the patients presented clinical improvement, 60% did not present alterations in the clinical picture and 10% presented symptom worsening, in spite of the treatment (Chart 2).

Discussion

The myocardial bridge is a congenital anomaly^{2,3} that results from the failure in the synchronic development of the myocardium and the coronary branches⁴, in which a segment of one epicardial coronary artery is involved by a cardiac muscle bundle, called "tunneled segment", crossing the intramural portion of the myocardium, below the myocardial muscle bridge⁵. The frequency of MB diagnoses in this study was 3.6%. Study data have shown a significant variation regarding the frequency, incidence and prevalence rates and the angiographic results. The angiographic prevalence of myocardial bridges is $< 5\%$ ⁶⁻⁸, a fact attributed to thin myocardial bridges that cause little compression to the tunneled segment and are present in most cases⁸.

Several studies have tried to elucidate the association between myocardial bridges and atherosclerosis. It is known

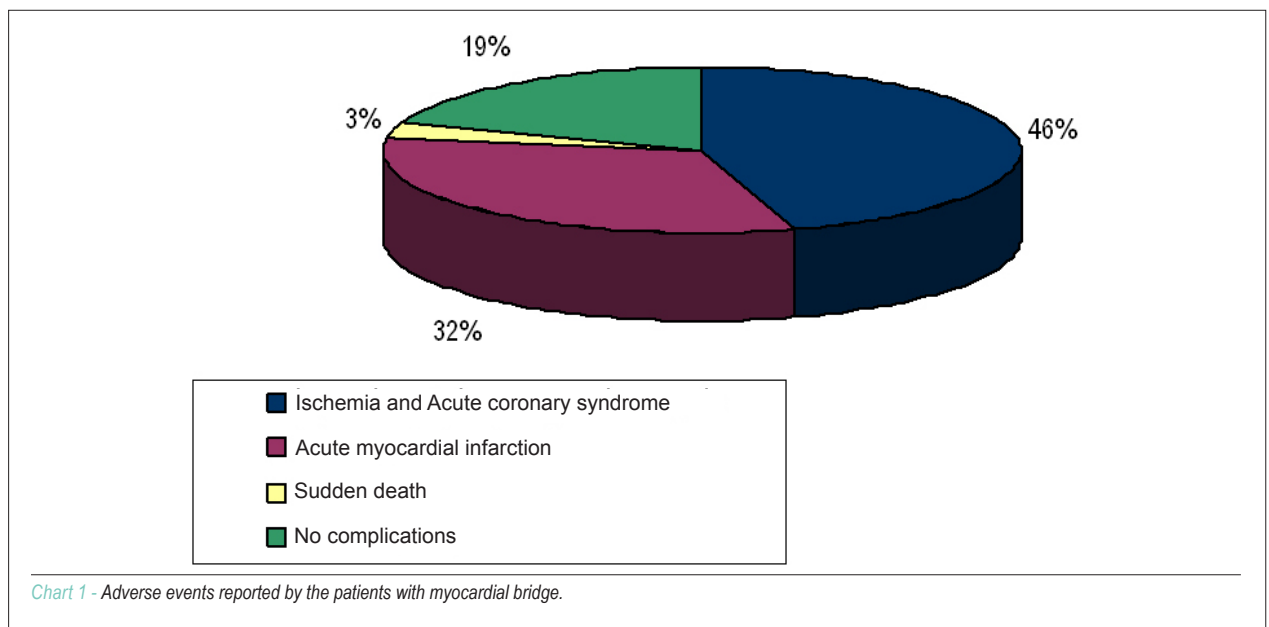
Table 2 - Degree of narrowing of the anterior descending artery at the coronary angiography of the patients selected for the study

Degree of narrowing	Patient % (n°)
Grade 1: < 49%	61.29 (19)
Grade 2: 50%-74%	29.03 (09)
Grade 3: > 75%	9.68 (03)
Total	100 (31)

Table 3 - Canadian Cardiovascular Society Grading Scale for Angina Pectoris

Class I – Usual physical activity, e.g. walking or climbing stairs, does not cause angina; angina is evoked by strenuous and/or rapid work or recreation.
Class II – Slight limitation of ordinary activities, e.g. after walking 2 blocks, climbing one flight of steps, under normal circumstances, after meals, in the cold, wind, in the morning, or when under emotional stress
Class III – Marked limitation of ordinary activities, e.g. walking 1-2 blocks or climbing stairs under normal circumstances.
Class IV – Inability to carry out any physical activity without discomfort—angina may be present at rest ..

Source: Stable Angina Guideline. Arquivos Brasileiros de Cardiologia, 2004. Modified from Campeau, Circulation, 19761.



that the area below the MB is spared in atherosclerotic disease, whereas the proximal area is prone to the development of atherosclerosis^{9,10}. In the present study, 27.6% of the patients presented involvement of the proximal segment of the bridge due to varying degrees of CAD. Disorders in blood flow contribute to the development of atherosclerosis in the proximal segment of the bridge¹¹, modulating the production of vasoactive substances by the endothelial cells, affecting vascular cell functions such as the thrombogenic potential, blood flow regulation and vascular tonus^{12,13}.

The typical angiographic finding of the MB is the systolic reduction of the epicardial coronary artery diameter and the persistence of this reduction during diastole¹⁴. Its transient nature and the dynamics of the obstruction help the differential diagnosis of fixed coronary stenoses. The left ADA is the most commonly affected artery¹⁵ and 100% of the patients in the present study presented the isolated form of left ADA involvement. However, other arteries can be involved, such as the left circumflex artery and the right coronary artery⁴. The quantification of the involvement of the coronary artery was carried out according to Noble et al⁶, in which the narrowing of the ADA during systole was classified as: Grade 1 ($\leq 49\%$); Grade 2 (50%-74%); and Grade 3 ($\geq 75\%$).

In some cases, when the angiographic results disclose normal coronary arteries, the use of provocative tests through pre-load decrease and adrenergic stimulation with nitroglycerin and orciprenaline, respectively, can enhance the systolic compression of the tunneled segment, thus establishing the diagnosis in up to 40% of the cases^{7,16}. Such procedures were not carried out in the present study.

The clinical diagnosis of MB must be considered in patients with angina symptoms, in the absence of risk factors or evidence of ischemia¹⁷. Although it is a malformation present since birth, symptoms onset does not occur until the third decade of life. The mean age of symptom onset presented

by the patients was 45.9 years (CI=11.7; SD=4.2). The late symptom manifestation can be explained by the systolic tension increase in the myocardial wall, as a consequence of the heart growth due to the elevation in the left ventricular diastolic-end pressure, through the association with arterial hypertension (myocardial hypertrophy) and with the eventual decrease in the coronary flow due to atherosclerotic processes that occur later¹⁸.

A considerable number of patients with an angiographic diagnosis of MB present associated cardiac valvular, muscular or atherosclerotic disease, which independently affects the clinical presentation and the response to treatment⁶. Such patients were excluded from the present study.

It has been well established the association between MB and complications such as ischemia and acute coronary syndrome¹⁹, reported by 56% of the studied patients; AMI²⁰ affecting 40% of the patients and sudden death²¹, which was the cause of death of one patient in the present study. Other complications are: coronary spasm²²; ventricular septum rupture²³, supraventricular paroxysmal tachycardia²⁴; ventricular tachycardia²⁵ and exercise-induced atrioventricular block²⁶, which were not reported by the patients in the present study.

The drug therapy is the first-choice treatment for symptomatic patients with MB. Interventions are reserved for patients with angina that is refractory to drug treatment. The drug treatment consists in the use of negative inotropic and chronotropic agents, such as adrenergic receptor blockers or calcium-channel blockers and the use of anti-platelet agents, with the objective of relieving symptoms and signs of myocardial ischemia and reducing the risk of future cardiac adverse events²⁷. Treatment duration is not well clarified among the several studies.

In the present study, there was a significant variation regarding the time of treatment and the therapeutic regimens

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used by the patients, which prevented the individualized analysis of each drug class.

The use of beta-blockers results in decreased heart rate, increased diastolic time and reduced contractility and systolic compression of the vessel, with a consequent return to normal of the ST-segment alterations at the ECG, in addition to the improvement of clinical symptoms of angina and signs of ischemia^{28,29}.

The use of calcium-channel antagonists can be particularly useful when there is a contraindication for the use of blockers or, first choice, when there is a suspected coronary vasospasm³⁰. The use of nitrates must be avoided, as while they improve cardiac contractility, they worsen the degree of systolic narrowing of the coronary artery, which can aggravate symptoms^{16,31,32}. In spite of the presented studies, the nitrate was used in 43.3% of the patients in association or not with other drugs. However, the real impact of the use of these drugs on the clinical outcome of these patients cannot be established.

Stent implants prevent the phasic compression of the coronary lumen, eliminate the abnormalities of the diastolic flow and the maximum elevation of the intracoronary systolic pressure, normalizing the clinical symptoms¹³. After the stent implant, the systolic compression of the anterior descending artery disappears and the luminal diameter increases as well as the transversal section of the artery and the coronary flow reserve. However, according to Haager et al³³, analyses after 7 weeks showed moderate to severe stenosis of the stents in 45% of the patients, which required a new intervention in 36% of the patients.

The two-year clinical and angiographic follow-up showed good results, with improvement of the angina symptoms and absence of cardiac events³³. However, long-term favorable

results have been described by other authors^{34,35}. In the present study, two patients with grade 1 angiographic systolic narrowing underwent angioplasty, presenting significant symptom improvement, free of adverse events, after one and two years of follow-up.

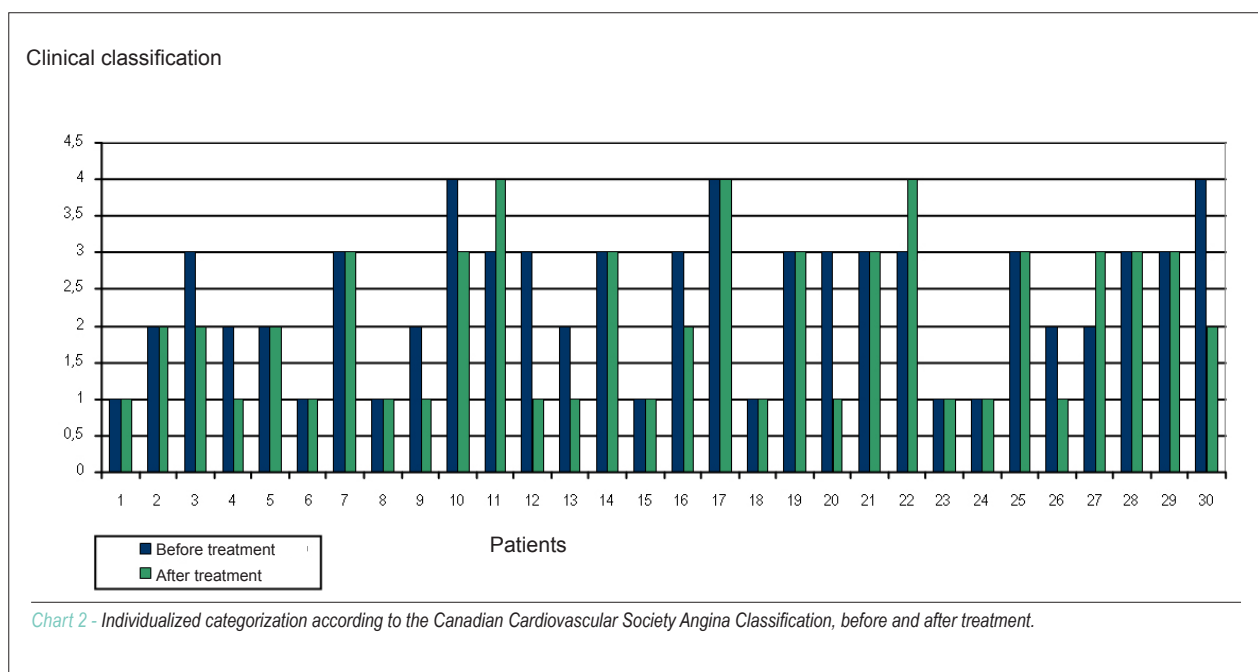
Before the current era of percutaneous coronary intervention, the surgical myotomy was considered the treatment of choice for patients with persistent symptoms, despite the intensive drug therapy^{27,36}. The surgical decompression of the tunneled artery results in the disappearance of the angina symptoms and ischemia in patients with severe systolic compression of the ADA³⁷. Another alternative is the myocardial revascularization surgery, with the anastomosis of the internal mammary artery to the left ADA³³. None of the patients in the present study underwent surgical procedures.

The long-term prognosis of MB is good, regardless of the severity of the systolic narrowing of the luminal diameter of the coronary artery³⁸.

In this study, the analysis of 31 patients showed that only one patient died, which was attributed to sudden death due to myocardial bridge; angioplasty was performed in two patients, with long-term significant clinical improvement; and 96.7% of the patients underwent clinical pharmacological therapy. In another study, during a 43-month follow-up of a group of 35 patients with MB, a patient died due to sudden cardiac death; 20% of the patients persisted with CCS I-II angina classification; one patient needed percutaneous revascularization for symptom improvement and 63% of the patients had kept the same drug therapy by the end of the study³⁹.

Limitations

This article aimed at describing the clinical and therapeutic evolution, as well as the long-term prognosis of patients with



MB. For this retrospective study, patients were selected from a database of a Laboratory of Hemodynamics that functions as a reference center in the region, which does not statistically reflect the data of the local population. As the patients were selected based on the data of the coronary angiographies previously carried out during a 5-year interval, it was not possible to carry out a follow-up and perform uniform noninvasive diagnostic tests in all patients, as well as confirm the reported complications, which were solely based on the report of the interviewed patients.

It was only possible to indicate the number of patients in whom these tests were performed and the type of test that was carried out, as well as their results. Finally, the criteria on which the decision of maintaining the long-term treatment were based are out of the scope of the present study, being influenced by the cardiologist's criterion.

Conclusion

Myocardial bridges affect individuals with angina symptoms in the absence of risk factors or evidence of ischemia, in which

symptom onset does not take place before the third decade of life. Most patients with myocardial bridges have a good prognosis, but in the long term, there are not enough data obtained from a large number of symptomatic patients that have presented an elevated degree of systolic and diastolic compression and ischemia evidence for definitive conclusions to be drawn.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

There were no external funding sources for this study.

Study Association

This study is not associated with any post-graduation program.

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