Clinical Profiles and In-patient Outcomes of Patients with Myocardial Bridging Versus Obstructive Coronary Artery Disease: A Single Center Retrospective Study

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Abstract

Background: Myocardial bridging (MB) is a congenital coronary anomaly characterized by an epicardial coronary artery taking an intramuscular course, causing systolic compression of the tunneled segment. In comparison to coronary artery disease (CAD), myocardial bridges have been uncommonly associated with acute coronary syndromes and sudden cardiac death. Evidence of accelerated atherosclerotic plaque formation proximal to the bridged segment may increase the risk for future adverse cardiac events in these patients.

Methodology: This Single–Center Retrospective Study included 323 adult in-patients who underwent coronary angiography for suspected myocardial ischemia in 2022. Clinical information and in-hospital outcomes were obtained by review of medical records.

Results: Myocardial bridging was observed in 31 out of 323 patients (9.60%), with the majority in the mid-left anterior descending artery (87.10%). MB was more prevalent in females (56.62%), and these patients were younger than patients with obstructive CAD (56.9 versus 63.6 years). Chronic Coronary Syndrome was more prevalent in the MB group (82.62%). The coronary segment proximal to the area with MB showed the concurrent presence of obstructive CAD in 16.12% and non-obstructive CAD in 29.03% of cases. In-hospital mortality occurred in 4.44% of the studied population. However, there were no mortalities in the MB group.

Conclusion: Among patients admitted for suspected myocardial ischemia, 9.6% had MB. These patients were younger and, more often, female. Obstructive and non-obstructive CAD were noted in bridged vessels. Although patients with obstructive CAD have a higher risk of experiencing in-hospital death and cardiac complications, evidence of increased atherosclerotic plaque formation in bridged vessels has important implications for future adverse cardiac events and repeat hospitalizations in the MB population. Aggressive risk factor modification, emphasis on long-term follow-up, and the establishment of clinical practice guidelines are therefore necessary for patients with MB.

Keywords: Myocardial bridging, coronary artery disease, clinical profiles, in-patient outcomes

Introduction

Myocardial bridging (MB) is a congenital coronary anomaly with an epicardial coronary artery taking an intramuscular course and is tunneled under an overlying muscular bridge.[1] It is characterized by systolic compression of the tunneled segment, which remains clinically silent in the vast majority of cases.[2] The prevalence in autopsy studies has been reported as high as 86%,[3] with a mean of 25% similar to more recent evaluations using coronary computed tomography angiography (CCTA).[4] In contrast, the prevalence of MB reported by coronary angiography is lower than in autopsy and CCTA studies, ranging from 0.5 to 16%, and remains ≤40% with the use of provocative testing.[3] There is a high prevalence (41%) of MB in adults with hypertrophic cardiomyopathy (HCM).[5] Myocardial bridging is usually considered in patients who are at low risk for CAD but with angina or evidence of myocardial ischemia.[3] Previous studies examining patients with MB showed a male predominance (89%) with a mean age of 51 years, typically younger than patients with CAD.[4] The median age of patients with MB associated with HCM is even younger at 29 years (range 5-90).[5] Coronary artery disease is a leading cause of mortality worldwide. Men are generally at increased risk for CAD in comparison to women, and the prevalence increases with age.

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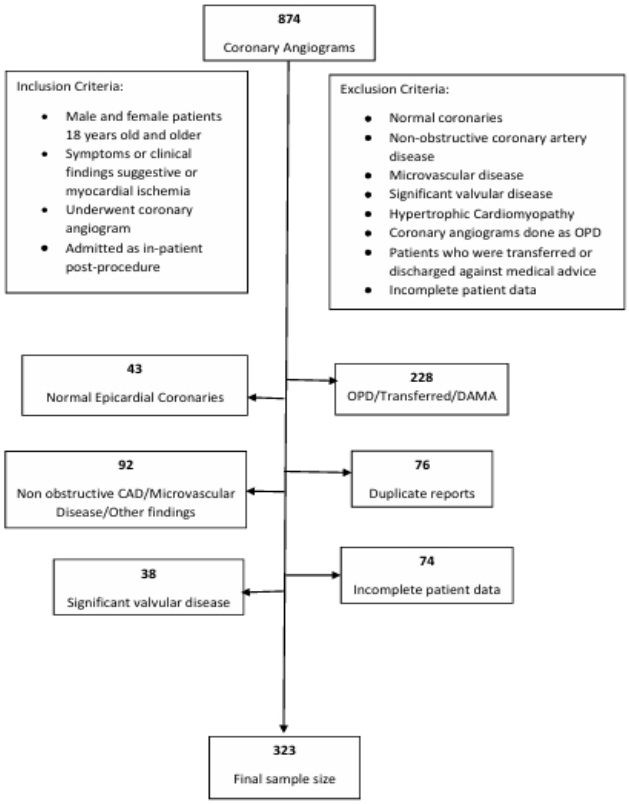

Figure 1: Research Flow Diagram

Modifiable risk factors for CAD include hypertension, dyslipidemia, diabetes mellitus, smoking, and obesity. Up to 50% of patients with MB have traditional risk factors for CAD, and the clinical presentation typically varies.[4] Myocardial bridges have been associated with exertional angina, myocardial ischemia, acute coronary syndromes, ventricular arrhythmias, stress cardiomyopathy, and sudden cardiac death.[2] However, these complications are rare, and most myocardial bridges are thought to be benign and have no clinical significance at the time of identification. Data on MB in the Philippines is limited, and there are currently no major cardiovascular society guidelines on the management of MB. The evidence on the therapy of MB is mainly based on the known pathophysiology and hemodynamics of the disease. The 5-year survival in patients with isolated MB is generally good at 97.5%. However, reports of patients with coexistent CAD experiencing myocardial infarction and persistent CCS Class I-II angina raise concern about the true significance of MB.[4]

Significance of the Study

There is evidence of accelerated atherosclerotic plaque formation that develops proximal to the bridged segment but spares the tunneled segment.[5] Increases in local wall tension and stretch may induce endothelial injury retrograde systolic flow and contribute to proximal atherosclerotic plaque formation.[6,7] Progression of atherosclerosis may predispose an individual with a myocardial bridge to future adverse cardiac events, repeat hospitalizations for recurrent angina, and increased risk for cardiac mortality. The lack of established clinical practice guidelines for patients with MB poses a challenge in the management, treatment goals, and long-term follow-up of these patients.

Methodology

The study utilized a descriptive, correlational retrospective cohort design. Medical history, records, and angiogram results of 874 patients from January to December 2022 in a PCI-capable tertiary hospital in Cebu City, Philippines, were reviewed. An analysis was done to correlate clinical profiles and treatment outcomes of admitted patients with MB and those with obstructive coronary disease. The study was done in Chong Hua Hospital, a 660-bed capacity, private, tertiary, PCI-capable tertiary hospital in Cebu City, Philippines.

The study population was composed of patients who were admitted and underwent coronary angiography for suspected acute or chronic coronary syndromes based on symptoms and/or clinical findings suggestive of myocardial ischemia. A total of 874 coronary angiograms done in the year 2022 were reviewed and analyzed. Coronary angiograms were done as an outpatient and were excluded. Patients with angiographically normal coronaries, non-obstructive CAD, microvascular disease, severe valvular disease, hypertrophic obstructive cardiomyopathy, and stenosis of non-dominant branches were similarly excluded. After applying the appropriate inclusion and exclusion criteria, 323 adult patients were included in the study (*Figure 1*).

Chart data of the included study population were collected through the Medical Records section after the protocol's approval by the same hospital's Ethics Review Board. The angiographic diagnosis of MB was defined as systolic narrowing of an epicardial coronary artery produced during systolic compression. A comparative group with obstructive CAD was selected after applying relevant inclusion and exclusion criteria. Other laboratory results pertinent to the study were 12L-electrocardiogram, Troponin I, 2D echocardiography, and non-invasive stress testing. The information on clinical presentation, cardiovascular risk factors, hyperlipidemia burden, and in-hospital outcomes was obtained by chart review of patients' medical records.

Results

Demographics and Clinical Profiles of Patients with MB and Obstructive CAD. Presented in *Table I* are the demographic and clinical characteristics of patients with angiographically determined MB and obstructive CAD admitted due to clinical suspicion of myocardial ischemia. Eight patients who presented with both MB and Obstructive CAD were excluded from the comparative analysis, resulting in 315 patients. Patients with MB are, on average, younger than those with CAD, with a mean of 56.9 years versus 63.6 years, respectively ($p = 0.015$). CAD is more prevalent in males (n = 220,

Table I. Patient Demographics and Clinical Profiles, n = 315

Characteristics	Patients with		P-Value
	Angiographically-determined Myocardial Bridging (MB) *n* = 23	Angiographically-determined Coronary Artery Disease (CAD) *n* = 292	
Demographic Profile			
Age *in years*, mean ± SD	56.96 ± 11.86	63.61 ± 10.59	0.015 *
Sex			
Male	10 (43.48)	220 (75.34)	0.001 *
Female	13 (56.52)	72 (24.66)	
BMI, mean ± SD	25.65, ± 4.70	26.42 ± 4.48	0.451
History of Smoking	2 (8.70)	92 (31.51)	0.021 *
Pack years, mean ± SD	14.00 ± 1.41, *n* = 2	25.06 ± 23.57, *n* = 92	0.512
Comorbid Conditions			
Hypertension	11 (47.83)	259 (88.70)	<0.001 *
Dyslipidemia	3 (13.04)	55 (18.84)	0.490
Diabetes Mellitus	7 (30.43)	179 (61.30)	0.004 *
Chronic Kidney Disease	0 (0.00)	36 (12.33)	0.074
Other	4 (17.39)	71 (24.32)	0.453
Clinical Presentation			
Chest pain	9 (39.13)	160 (54.79)	0.147
Dyspnea	7 (30.43)	110 (37.67)	0.489
Epigastric Pain	1 (4.35)	10 (3.42)	0.816
Clinical CAD (Asymptomatic)	8 (34.78)	49 (16.78)	0.031 *
Other	1 (4.35)	6 (2.05)	0.473
Hyperlipidemia Burden			
LDL *in mg/dL*	79.13 ± 51.75, *n* = 12	96.93 ± 50.18, *n* = 125	0.187
HDL *in mg/dL*	48.41 ± 19.28, *n* = 12	44.40 ± 15.51, *n* = 125	0.591
TG *in mg/dL*	76.33 ± 48.14, *n* = 12	130.45 ± 94.04, *n* = 125	0.005 *
Clinical Diagnosis			
Chronic Coronary Syndrome	19 (82.61)	143 (48.97)	0.002 *
Unstable Angina	1 (4.35)	23 (7.88)	0.539
Non-ST Elevation MI(NSTEMI)	2 (8.70)	58 (19.86)	0.189
ST Elevation MI (STEMI)	1 (4.35)	66 (22.60)	0.039 *

Note: Values are presented in frequency (percentage) unless otherwise stated; * Significant at $p < 0.05$

75.34%, $p = 0.001$), while MB is more prevalent in females (n = 13, 56.52%, $p = 0.001$). Patients with CAD are more likely to have a history of smoking, hypertension, and diabetes mellitus, with hypertension being the most common medical comorbidity (n = 259, 88.7%, $p = <0.001$) in this group. There were no patients with chronic kidney disease in the MB group. The most common indication for coronary angiogram in both groups was chest pain. Clinical findings suggest that CAD in an asymptomatic patient is more common in the MB group (n = 8, 34.78%). Triglyceride levels are significantly higher in patients with obstructive CAD, with a mean level of 130.45 (SD ± 94.04). A clinical diagnosis of Chronic Coronary Syndrome is more prevalent in the MB group (82.61%, $p = 0.002$), while ST Elevation MI (STEMI) was more common in the CAD group (22.60%, $p=0.039$). Other characteristics were observed to be comparable between the two groups.

Prevalence of MB and Its Location. As shown in *Table II*, MB was observed in 31 out of the 323 individuals admitted for clinical suspicion of myocardial ischemia, indicating a prevalence of 9.60% in patients admitted for Coronary Angiogram after application of exclusion criteria. The majority of myocardial bridges were located in the middle portion of the LAD (n = 27, 87.10%), with only a small percentage in the proximal (6.45%) and distal (6.45%) portions of the same vessel. There was one case of MB in the Left Circumflex Artery (LCx), and no cases were recorded in the Right Coronary Artery (RCA). Only one case had two myocardial bridges in the proximal LAD and proximal LCx. Considering the territory supplied by the LAD, the preferential location of MB in this vessel, specifically in the middle segment, predisposes these patients to a higher ischemic burden.

Atherosclerotic Disease in the Obstructive CAD Population. Shown in *Table III* is the distribution of atherosclerotic disease within the population of individuals with obstructive CAD, which totaled 292 patients. The LAD is the most commonly affected vessel (n = 259, 88.7%). Analysis of the data in this population showed that 36.6% (n = 106) of patients in this group have a one-vessel disease, 26.7% (n = 78) have a two-vessel disease, and 36.0% (n = 105) have significant three vessel involvement. Significant LM involvement is seen in 15.41% of cases, while 3 out of 292 patients had isolated LM disease (1.0%).

In-hospital treatment outcomes of Patients with MB and Obstructive CAD. Table IV provides data on the clinical outcomes for 315 individuals. In-hospital death occurred

Table II. Prevalence and Location of the Myocardial Bridge

Characteristics	Values
Prevalence – *population size* is 323	31 (9.60)
Location, *n = 32*	
Left Anterior Descending Artery (LAD)	31 (96.8)
Proximal	2 (6.45)
Middle	27 (87.10)
Distal	2 (6.45)
Right Coronary Artery (RCA)	0 (0.00)
Left Circumflex Artery (LCx)	1 (3.23)
Proximal	1 (3.23)

Note: Values are presented in frequency (percentage)

Table III. Atherosclerotic Disease in the Obstructive Coronary Artery Disease Population

Characteristics	Values n (%)
Prevalence – *population size* is 292	292 (100.00)
Location	
Left Anterior Descending Artery (LAD)	259 (88.70)
Left Circumflex Artery (LCx)	145 (49.66)
Right Coronary Artery (RCA)	171 (58.56)
LM Involvement	45 (15.41)

Note: Values are presented in frequency (percentage)

in 4.44% of the population, with cardiac causes of death occurring in 85.8% of mortalities. Cardiogenic shock was seen in all cardiac deaths, while congestive heart failure occurred in 75% of cardiac mortalities. Non-cardiac causes of death were less common (42.85%), with septic shock accounting for the majority of cases. All in-hospital deaths occurred exclusively in the obstructive CAD group. Cardiac complications were relatively frequent, affecting 31.51% of patients in the obstructive CAD group. Congestive heart failure was the most common cardiac complication in the obstructive CAD group (n = 72, 23.17%) and the only cardiac complication recorded in the MB group (n=1, 4.35%) in a patient who presented with STEMI. Cardiogenic shock, ventricular arrhythmias, and heart blocks occurred in 14.04%, 9.59%, and 2.05% of cases in the obstructive CAD group, respectively. There were no recorded cases of cardiac rupture in both study groups.

Comparison of in-hospital outcomes between patients with angiographically-determined MB and obstructive CAD is also seen in *Table IV*. The data indicates that patients with CAD have a higher risk of experiencing cardiac complications, mostly in the form of congestive heart failure, during their hospital stay compared to patients with MB. Other outcomes, such as cardiac death, non-cardiac death, cardiogenic shock, heart blocks, and ventricular tachycardia/fibrillation, were not seen in the MB group.

Angiographic Findings on Vessels with MB. Table V presents other angiographic findings in bridged vessels. Obstructive CAD was present in 16.12% of cases, indicating that a notable portion of the MB population has clinically significant stenosis in the bridged vessel. Non-obstructive CAD is observed in 29.03%, indicating that more than a quarter of individuals with MB have non-obstructive stenosis in the same vessel. All obstructive and non-obstructive CAD cases were seen proximal to the bridged segment. Diffuse luminal irregularities were found in 19.35% of cases, highlighting irregularities in the coronary artery lumen that may not necessarily be obstructive but may result from altered hemodynamics and increased shear stress. Tortuous vessel configurations were noted in 9.68% of individuals. Ectatic vessels (enlarged coronary arteries) and microvascular disease (small vessel disease) were less common, with each occurring in 3.23% of cases. Notably, 35.48% of the vessels with MB were disease-free.

Table IV. The In-Hospital Outcomes of Patients with Myocardial Bridging and Obstructive Coronary Disease

In-Hospital Outcomes	Overall *n* = 315	Patients with		*p-Value*
		Myocardial Bridging (MB) *n* = 23	Coronary Artery Disease (CAD) *n* = 292	
In-hospital Death	14 (4.44)	0 (0.00)	14 (4.79)	0.283
Cardiac Death	12 (3.81)	0 (0.00)	12 (4.11)	0.322
Cardiogenic Shock	12 (3.81)	0 (0.00)	12 (4.11)	0.322
Congestive Heart Failure	9 (2.86)	0 (0.00)	9 (3.08)	0.393
Cardiac Rupture	0 (0.00)	0 (0.00)	0 (0.00)	—
Non-cardiac Death	6 (1.90)	0 (0.00)	6 (2.05)	0.488
Sepsis/Septic Shock	5 (1.59)	0 (0.00)	5 (1.71)	0.527
Respiratory Failure	5 (1.59)	0 (0.00)	5 (1.71)	0.527
Multiorgan Failure	4 (1.27)	0 (0.00)	4 (1.37)	0.572
Cardiac Complications	93 (29.20)	1 (4.35)	92 (31.51)	0.006 *
Congestive Heart Failure	73 (23.17)	1 (4.35)	72 (24.66)	0.026 *
Cardiogenic Shock	41 (13.02)	0 (0.00)	41 (14.04)	0.054
2nd Degree/Complete Heart Block	6 (1.90)	0 (0.00)	6 (2.05)	0.488
Ventricular tachycardia/fibrillation	28 (8.89)	0 (0.00)	28 (9.59)	0.120
Cardiac Rupture	0 (0.00)	0 (0.00)	0 (0.00)	—

Note: Values are presented in frequency (percentage); * Significant at 0.05

Table V. Angiographic Findings on Vessels with Myocardial Bridging

Findings	Frequency (%)
Obstructive CAD	5 (16.12)
Non-obstructive CAD	9 (29.03)
Luminal Irregularities	6 (19.35)
Tortuosity	3 (9.68)
Ectasia	1 (3.23)
Microvascular Disease	1 (3.23)
Disease-free	11 (35.48)

Discussion

Myocardial bridging occurs when a portion of an epicardial coronary artery takes an intramuscular course and is tunneled under an overlying muscular bridge.[1] This congenital coronary anomaly results in systolic compression of the tunneled portion of the coronary artery and remains clinically silent in the majority of cases. The prevalence of MB in autopsy studies has been reported as high as 86%,[3] with a mean of 25%, similar to more recent evaluations using coronary computed tomography angiography (CCTA).[4] In contrast, the prevalence of MB reported by coronary angiography is lower than in autopsy and CCTA studies, ranging from 0.5 to 16%, and remains ≤40% with the use of provocative testing.[3] Myocardial bridging was observed in 9.60% of patients in this study, with the majority of myocardial bridges located in the mid-LAD (87.10%). Only one patient had two myocardial bridges (3.2%), making these phenomena uncommon. Consistent with previous studies, myocardial bridges are most commonly localized in the middle segment of the left anterior descending coronary artery (LAD).[6] The preferential location of MB in the mid-LAD predisposes patients to a high ischemic burden and the development of symptoms.

The data in our study shows that patients with MB, on average, are younger than patients with obstructive CAD, with a mean age of 56.9 versus 63.6, respectively. MB was also more prevalent in females (56.62%) than CAD, which was more prevalent in males (75.34%). Consistent with known risk factors for atherosclerotic disease, hypertension, diabetes mellitus, and smoking were more common in the CAD population. Only 8.6% of the MB population had a history of tobacco, and none of the patients in this group had a diagnosis of chronic kidney disease. Triglyceride levels were significantly higher in the obstructive CAD group than in the MB group (130.45 $\pm$ 94.04 versus 76.33 $\pm$ 48.14). Chest pain was the most common indication for Coronary Angiogram in both groups. However, patients with MB were more likely to have asymptomatic myocardial ischemia detected only on non-invasive imaging such as 2D echocardiography and stress testing (34.78%, $p = 0.031$). The clinical diagnosis of Chronic Coronary Syndrome (CCS) is more prevalent in the MB group (82.62%, $p = 0.002$), while ST elevation MI (STEMI) was more common in the CAD group (22.6%, $p = 0.039$). It is of note that there was one patient in the MB group who presented with STEMI (4.35%) and two patients who presented with NSTEMI (8.7%), indicating that although CCS is the most common clinical diagnosis in patients with MB, acute coronary syndromes uncommonly occur. Myocardial bridges have been associated with exertional angina, myocardial ischemia, acute coronary syndromes, ventricular arrhythmias, stress cardiomyopathy, and sudden cardiac death.[2] Previously asymptomatic patients may develop symptoms with the development of diastolic dysfunction, left ventricular hypertrophy, coronary vasospasm, or microvascular dysfunction.[2] Additionally, symptoms and ischemia may be provoked by exercise and tachycardia.

There is evidence of accelerated atherosclerotic plaque formation that develops proximal to the bridged segment but spares the tunneled segment.[15] Increases in local wall tension and stretch may induce endothelial injury and contribute to proximal atherosclerotic plaque formation.[16,17] However, this fixed proximal plaque is likely not the sole mechanism of myocardial ischemia in these patients.[15] Using frame-by-frame quantitative coronary angiography, vessel compression of bridged arteries was shown to extend into diastole, resulting in decreased coronary artery perfusion.[18,19] Combining intracoronary doppler flow and pressure measurements with coronary angiography demonstrated evidence of a persistent decrease in diastolic vessel diameter and reduced flow reserve in patients with symptomatic bridges.[16] Given this, ischemia is likely provoked by tachycardia and increased contractility during stress or exercise, resulting in decreased diastolic coronary artery filling time, impairment of coronary blood flow, and worsening of systolic and diastolic compression of the artery.[3] Previously asymptomatic patients may develop symptoms with the development of diastolic dysfunction, left ventricular hypertrophy, coronary vasospasm, or microvascular dysfunction.[2] Pathologic studies suggest that bridged vessels that are long and deeper than 3 mm beneath the epicardium are at high risk for cardiac events.[20]

The LAD was the most commonly affected artery in the obstructive CAD population. In the obstructive CAD group, 36.6% of patients had a one-vessel disease, 26.7% had a two-vessel disease, and 36.0% had significant three-vessel involvement. Significant LM involvement was seen in 15.41% of cases, while only 1% of patients had isolated LM disease. The overall mortality in the population studied was low at 4.44%, with cardiac death comprising the majority of mortalities at 85.8%. The most prevalent cause of cardiac mortality was cardiogenic shock, which was seen in all cardiac deaths. Non-cardiac mortalities were a less common cause of death, with septic shock accounting for the majority of cases. There were no mortalities in the MB group and no recorded cases of cardiac rupture in either group.

A sub-group analysis was done on other angiographic findings on vessels with MB. Among patients in the MB group, 16.12% presented with obstructive CAD, and 29.03% presented with non-obstructive CAD in the segment proximal to the MB. Diffuse luminal irregularities were also noted in 19.35%. Eleven out of 31

patients had no other findings other than MB. Coronary atherosclerosis associated with MB has primarily been studied in the LAD. The segment proximal to the bridge frequently shows atherosclerotic plaque formation [8,12]. This is supported by studies on a cellular and ultrastructural level [21,22]: In contrast to proximal and distal segments, foam cells and modified smooth muscle cells were missing in patients' tunneled segments.[21] Extramural, epicardial segments in cholesterol-fed rabbits developed intimal atherosclerosis with accumulation of ApoB and proliferating cell nuclear antigens (PCNA) in smooth muscle cells of the intima.[22] These changes were not seen in any arterial wall component in tunneled segments.[22] Furthermore, endothelial cell permeability was increased in atherosclerotic and nonatherosclerotic portions of epicardial segments in high-cholesterol rabbits but not in tunneled or normal control arteries.[22]

Neither nonsignificant stenosis proximal to the bridge nor systolic compression of the tunneled segment alone can sufficiently explain severe ischemia and associated symptoms. Experimental LCX occlusion, initially during systole and then extending increasingly into diastole, resulted in a distinct shortening of inflow time with a significant reduction of epicardial, subendocardial, and distal coronary pressure.[29,30] After releasing the occlusion, diastolic flow increased in correspondence with the increasing duration of vessel occlusion despite a decrease in mean flow.[30] This increased diastolic/systolic flow ratio was later verified in patients.[13] Consistent with clinical findings,[13,26] the increase in diastolic flow could not fully compensate for the decrease in mean flow, resulting in reduced coronary flow reserve, which the impaired vasodilatory capacity of the resistance vessel could not explain.[30] An increase in the sympathetic drive during stress or exercise likely facilitates ischemia because tachycardia increases the systolic-diastolic time ratio at the expense of diastolic flow. Increased contractility during stress further aggravates systolic (and diastolic) compression.[32]

The evidence behind medical therapy in MB is limited. Nitrates are contraindicated as they can accentuate systolic compression of the bridged segment by vasodilating the adjacent non-bridged coronary segments and worsen the symptoms.[15] Calcium channel blockers may be beneficial if vasospasm is present. Beta-blockers relieve hemodynamic disturbances by decreasing peak heart rate, increasing diastolic filling time, and decreasing contractility and compression of the artery.[36] As a result, they are commonly used as first-line agents for patients with symptoms.

Conclusions and Recommendations

Myocardial bridging is a congenital anomaly frequently encountered in practice, and consideration has been taken in identifying and treating the subset of patients with symptomatic disease. Current imaging modalities such as coronary angiography have proven useful in the anatomic characterization of MB. As noted in this study, patients with MB were younger, more likely to be female, and had fewer medical comorbidities compared to patients with obstructive CAD. Several angiographic findings were noted in the MB study group, including a notable portion of the study group having clinically significant stenosis in the bridged vessel, some with non-obstructive CAD and luminal irregularities. Although patients with MB had a lower risk of experiencing in-hospital death and cardiac complications, evidence of accelerated atherosclerotic disease in bridged vessels has important implications for future adverse cardiac events, repeat hospitalizations for recurrent angina, and an increased risk for cardiac mortality in the MB population. Aggressive risk factor modification, emphasis on long-term follow-up, and the establishment of clinical practice guidelines are therefore necessary for patients with MB.

Given the low prevalence of MB, a multicenter study across PCI-capable institutions may be done to obtain a larger population size more representative of the Filipino population. Future areas of research in the MB group include symptom control in various treatment groups presenting with chronic coronary syndrome, the occurrence of future acute coronary events and repeat coronary angiography with or without revascularization, the development of angiographically-determined obstructive CAD in previously disease-free vessels with a myocardial bridge, comparison of various imaging modalities in the monitoring of atherosclerotic disease progression, all-cause mortality and cardiac mortality on 5 and 10-year follow-up.

Funding: None

Declaration of Interest: The author declares no competing interested

Conflict of interest: None

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