

Clinical Characteristics, Angiographic Features, And Prognostic Implications of Myocardial Bridging: A Retrospective Study in Myanmar

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ABSTRACT

Background

Myocardial bridging (MB) is a congenital anomaly where an epicardial coronary artery tunnels intramuscularly. Though traditionally considered a benign variant, its clinical relevance remains controversial, particularly regarding the risk of early hospital readmission and symptom burden in patients with non-obstructive coronary artery disease.

Methods

We conducted a retrospective cohort study at a 700-Bedded Hospital in Pyin Oo Lwin, reviewing 600 consecutive diagnostic coronary angiograms performed from January 2021 to December 2024. Patients with isolated MB—defined as systolic compression of 50% or greater without concomitant atherosclerosis—were evaluated against a propensity-matched control group. The primary endpoint was the incidence of Major Adverse Cardiovascular Events (MACE) at 12 months.

Results

Isolated MB was identified in 21 patients (3.5% prevalence). The cohort was predominantly male (81%) with a mean age of 49 ± 11 years. The left anterior descending artery was involved in 95% of cases. At 12 months, the MB group exhibited a significantly higher MACE incidence compared to controls (14.3% vs. 2.4%; $p=0.04$). Crucially, this difference was driven entirely by recurrent hospitalizations for unstable angina (14.3% vs. 2.4%; $p=0.03$). There were zero cases of cardiovascular death, non-fatal myocardial infarction, or revascularization. Multivariable Cox regression identified bridge length exceeding 25 mm as an independent predictor of symptom recurrence.

Conclusions

Isolated MB demonstrates excellent short-term survival but notable morbidity. The high rate of recurrent angina requiring hospitalization within one year indicates routine reassurance is insufficient. Symptomatic patients necessitate optimized medical therapy and close monitoring to effectively prevent early readmissions.

KEYWORDS: Coronary Angiography, Hospital Readmission, Myocardial Bridging, Prognosis

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INTRODUCTION

Myocardial bridging (MB) is defined anatomically by the dipping of an epicardial coronary segment into the myocardium. This configuration subjects the 'tunneled segment' to compression by the overlying 'myocardial

bridge' during the cardiac cycle. While theoretically possible in any coronary territory, this phenotype is overwhelmingly observed in the mid-Left Anterior Descending (LAD) artery. [1, 2]

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The historical understanding of MB has evolved significantly over the centuries. The condition was first described anatomically by Reyman in 1737. He noted the intramural course of coronary arteries during autopsy examinations. [3] In 1960, Porstmann and Iwig expanded the understanding of this anatomy by documenting its angiographic hallmark: the 'milking effect.' This term describes the unique hemodynamic profile of the bridged segment, characterized by transient systolic compression of the vessel that disappears during diastole. [4]

For decades following this discovery, MB was largely regarded as a benign and incidental finding. The rationale for this "benign" classification was physiological: since the majority of coronary blood flow (approximately 85%) occurs during diastole, systolic compression was theoretically believed to have minimal impact on myocardial perfusion. [5]

However, a significant diagnostic paradox exists between anatomical and clinical prevalence. Autopsy series using rigorous dissection techniques have reported high prevalence of MB (80% to 85%) in the general population. By contrast, conventional coronary angiography (CAG) detects MB in only 0.5% to 16% of patients. [1] This vast discrepancy suggests that the majority of myocardial bridges are thin, superficial, and hemodynamically silent. It is only when the bridge is sufficiently deep (encircled by thick muscle) or long that it produces the angiographic "milking effect." "Consequently, patients diagnosed in the catheterization laboratory likely represent a specific, high-risk subset exhibiting the most severe anatomical phenotype. [5, 6]

In recent years, the paradigm of MB as a purely benign has been challenged by accumulating evidence of its pathological potential with adverse cardiovascular sequelae. These include angina pectoris, myocardial ischemia, acute coronary syndromes (ACS), coronary spasm, exercise-induced arrhythmias (such as supraventricular tachycardia and ventricular fibrillation), and even sudden cardiac death, particularly in young athletes. [7] Resolving the paradox between this historically 'benign' classification and the observed 'malignant' clinical sequelae requires a more nuanced understanding of coronary hemodynamics. [8]

Advanced intravascular interrogation using IVUS and Doppler flow wires have revealed that the hemodynamic impact of MB is not confined to systole. While the compression is indeed systolic, the relaxation of the bridged segment is delayed, persisting into early and mid-diastole. This phenomenon, known as the "finger-tip phenomenon," significantly encroaches upon the diastolic fraction of the cardiac cycle, which is critical for coronary perfusion. Furthermore, the rapid systolic compression of the vessel creates a "water-hammer" effect—a retrograde pressure wave that propagates proximally. This results in disturbed, oscillatory shear stress in the segment proximal to the

bridge, which has been shown to accelerate the development of atherosclerosis. [9, 10] Conversely, the bridged segment itself is typically spared from plaque formation. This is likely due to the high wall shear stress and the absence of tensile stress during systole. [11]

This pathophysiology assumes critical clinical relevance under conditions of increased metabolic demand. Exercise induces tachycardia and increased myocardial contractility. Tachycardia shortens the duration of the cardiac cycle, but it disproportionately shortens diastole. In a patient with MB, where diastolic relaxation is already delayed, the window for coronary perfusion becomes critically narrow during tachycardia. Concurrently, the hypercontractile state of the myocardium exerts intensified mechanical crushing of the intramural artery. This creates a "dynamic stenosis" that worsens precisely when myocardial oxygen demand is highest. [6]

Clinically, this presents a significant challenge for the interventional cardiologist. Patients frequently manifest typical exertional angina, yet catheterization reveals angiographically non-obstructive coronary arteries (ANOCA) or only trivial luminal irregularities. [8] Once obstructive atherosclerotic Coronary Artery Disease (CAD) is ruled out, the diagnosis of MB is often made, but its significance is frequently downplayed. Patients are reassured that their heart is "structurally normal" and that they are not at risk of a heart attack. While this may be true regarding the risk of thrombotic myocardial infarction, it fails to address the functional ischemia causing their symptoms.

This disconnect often traps the patient in a maladaptive cycle of care: patients are discharged without targeted therapy, they return repeatedly to emergency departments with unremitting chest pain, incurring significant healthcare costs and psychological distress. [12, 13]

Moreover, the diagnostic sensitivity of conventional angiography is limited. Angiography is a 2D lumenogram that may miss thinner bridges or those that do not cause severe systolic compression but still induce hemodynamic disturbances. The use of nitroglycerin during angiography can accentuate the systolic compression by reducing preload and increasing reflex contractility. But it is not universally standardized in all labs. [14] Consequently, the true prevalence and clinical burden of symptomatic MB remain poorly defined, particularly within general hospital cohorts situated outside specialized academic centers. [15]

Therefore, the primary objective of this study was to evaluate the prevalence, angiographic characteristics, and specifically the short-term (12-month) prognostic implications of MB. By focusing on a general cohort of patients undergoing angiography for suspected ischemia, we aimed to determine whether the diagnosis of isolated MB is truly benign or if it is associated with a burden of early

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morbidity and hospital readmission that warrants a shift in management strategy.

MATERIALS & METHODS

Study Design and Population

This retrospective cohort study, conducted at a 700-bed military hospital in Pyin Oo Lwin, evaluated the clinical and prognostic impact of myocardial bridging (MB). The research protocol was approved by the Institutional Review Board. Investigators reviewed 600 (100%) consecutive diagnostic coronary angiograms performed between January 2021 and December 2024.

Inclusion Criteria (MB Group) and Exclusion Criteria

Patients were included in the MB group if they demonstrated isolated pathology, defined as a "milking effect" with systolic luminal reduction ≥ 50% without concomitant atherosclerosis. Major exclusion criteria included prior revascularization, hypertrophic cardiomyopathy, significant valvular disease, and an LVEF<35%. A control group of patients with normal or near-normal coronary arteries was selected and propensity-matched in a 2:1 ratio based on age, sex, and cardiovascular risk factors.

Angiographic Analysis

Two blinded interventional cardiologists performed the angiographic analysis using quantitative coronary angiography (QCA) to measure the reference vessel diameter (RVD) in end-diastole and the minimal luminal diameter (MLD) at maximal end-systolic compression. The percentage of systolic compression was calculated using the following formula:

$$\text{Systolic Compression (\%)} = \frac{\text{RVD (Diastole)} - \text{MLD (Systole)}}{\text{RVD (Diastole)}} \times 100$$

Compression was further categorized using the Noble classification, with Grade 2 representing 50% to 75% and Grade 3 representing >75% systolic compression.

Clinical Endpoints

The primary endpoint was Major Adverse Cardiovascular Events (MACE) at 12 months, defined as a composite of cardiovascular death, non-fatal myocardial infarction, target vessel revascularization, and hospitalization for unstable angina.

Statistical analysis

Statistical analysis was performed using SPSS version 26.0, employing Student's t-tests for continuous variables and Chi-square or Fisher's exact tests for categorical data.

RESULTS

Baseline Demographics and Clinical Characteristics

From the cohort of 600 diagnostic angiograms, 21 (3.5%) patients met the inclusion criteria for isolated, significant Myocardial Bridging. These patients were

matched with 42 controls. The baseline characteristics are detailed in Table 1.

Table 1: Baseline Clinical Characteristics

Variable	MB Group (n=21)	Control Group (n=42)	P-Value
Age (years)	49±11	50±10	0.45
Male Sex, n (%)	17 (81.0%)	34 (81.0%)	1.00
BMI (kg/m2)	26.4±3.0	27.2±3.5	0.38
Hypertension, n (%)	10 (47.6%)	20 (47.6%)	1.00
Diabetes Mellitus, n (%)	4 (19.0%)	9 (21.4%)	0.82
Dyslipidemia, n (%)	8 (38.1%)	17 (40.5%)	0.85
Current Smoker, n (%)	11 (52.4%)	23 (54.8%)	0.86
Clinical Presentation			
Stable Angina	13 (61.9%)	22 (52.4%)	0.46
Atypical Chest Pain	6 (28.6%)	18 (42.9%)	0.27
Unstable Angina	2 (9.5%)	2 (4.8%)	0.47

Continuous variables compared using the independent Student's t-test (t); categorical variables compared using the Chi-square test (χ2) or Fisher's exact test.

The mean age of the MB cohort was 49 ± 11 years, notably younger than the typical population presenting with obstructive atherosclerosis. The cohort was predominantly male 17 (81.0%). There were no statistically significant differences between the MB and Control groups regarding traditional cardiovascular risk factors, including hypertension, diabetes mellitus, dyslipidemia, and smoking status, confirming the efficacy of the matching process.

Angiographic Characteristics

The detailed angiographic analysis of the MB cohort is presented in Table 2. The Left Anterior Descending (LAD) artery was the site of bridging in 20 (95.2%) of cases. The mean lesion length was 19.4 ± 5.9 mm. Regarding the severity of systolic compression, 15 (71.4%) of patients had Grade 2 compression (50-75%), while 6 (28.6%) had Grade 3 compression (>75%).

Table 2: Quantitative Coronary Angiography (QCA) Analysis

Angiographic Parameter	Value (n=21)
Vessel Involved	
Left Anterior Descending (LAD)	20 (95.2%)
Left Circumflex (LCx)	1 (4.8%)

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Right Coronary Artery (RCA)	0 (0%)
Segment Location	
Mid	19 (90.5%)
Distal	2 (9.5%)
Reference Vessel Diameter (mm)	3.08±0.40
Minimum Luminal Diameter (Systole) (mm)	1.16±0.31
Bridge Length (mm)	19.4±5.9
Systolic Compression (%)	62.1±13.5
Noble Classification	
Grade 2 (50-75%)	15 (71.4%)
Grade 3 (>75%)	6 (28.6%)

Clinical Outcomes at 12 Months

The median follow-up duration was 12 months (Interquartile Range: 10-14 months). Clinical outcomes are summarized in Table 3. The primary endpoint (MACE) occurred significantly more frequently in the MB group compared to controls (14.3% vs. 2.4%; p=0.04). This difference was driven entirely by re-hospitalization for unstable angina. Importantly, there were zero cases of cardiovascular death, zero non-fatal myocardial infarctions, and zero requirements for revascularization in the MB group during the 12-month follow-up period.

Table 3: Comparison of Clinical Outcomes (MACE) at 1 Year

Outcome	MB Group (n=21)	Control Group (n=42)	Hazard Ratio (95% CI)	P-value
Total MACE	3 (14.3%)	1 (2.4%)	4.80 (1.1-21.3)	0.04
Cardiovascular Death	0 (0%)	0 (0%)	—	1.00
Non-fatal MI	0 (0%)	1 (2.4%)	—	1.00
Hosp. for Unstable Angina	3 (14.3%)	1 (2.4%)	5.30 (1.05-26.8)	0.03
Target vessel revascularization	0 (0%)	0 (0%)	—	1.00

Comparison between groups performed using the Chi-square test or Fisher's exact test for event frequencies; Hazard Ratios calculated via Cox proportional hazards regression.

Predictors of Adverse Events

To identify which patients with MB are at risk for recurrent symptoms within the first year, we performed Cox proportional hazards regression (Table 4).

Table 4: Predictors of MACE in MB Patients (Cox Regression)

Variable	Univariable HR (95% CI)	P-Value	Multivariable HR (95% CI)	P-Value
Age > 50 years	1.05 (0.30 - 3.40)	0.92	-	-
Hypertension	1.35 (0.35 - 5.10)	0.65	-	-
Bridge Length > 25 mm	2.90 (1.15 - 7.50)	0.02	2.65 (1.05 - 6.70)	0.04
Systolic Compression > 70%	2.80 (0.90 - 8.50)	0.07	2.40 (0.80 - 7.20)	0.11

In the multivariable analysis, Bridge Length > 25 mm (HR 2.65; p=0.04) was identified as an independent predictor of recurrent angina.

Forest plot displaying hazard ratios (HRs) with 95% confidence intervals derived from Cox proportional hazards regression analysis was shown in Figure 1.

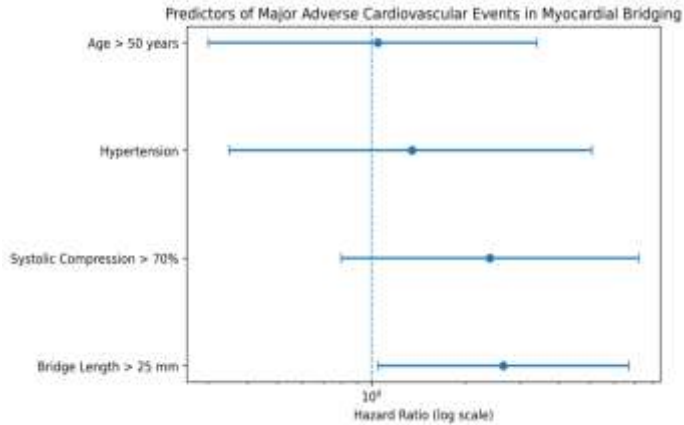


Figure 1: Predictors of Major Adverse Cardiovascular Events in Patients with Myocardial Bridging

Hazard ratios are plotted on a logarithmic scale, with the vertical reference line indicating no effect (HR = 1.0). Among the evaluated clinical and angiographic variables, bridge length >25 mm was identified as an independent predictor of major adverse cardiovascular events at 12 months. Other variables did not demonstrate a statistically significant association.

DISCUSSION

This 4-year retrospective analysis of 600 coronary angiograms from a tertiary care center provides critical

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insights into the "prognostic paradox" of myocardial bridging. Our data reaffirm that myocardial bridging is largely benign with respect to hard clinical outcomes. There was no death recorded during 12-month follow-up. However, this apparent safety masks a meaningful burden of early clinical events. Notably, 14.3% of patients required rehospitalization for unstable angina within one year of diagnosis. This rehospitalization is calling into question the adequacy of simple reassurance and highlighting the need for a more individualized and proactive management strategy.

Prevalence and Phenotype

Our observed angiographic prevalence of 3.5% aligns with major historical registries, such as the Coronary Artery Surgery Study (CASS). CASS reported rates between 0.5% and 5% depending on the definition used. [16] The demographic profile of our cohort—predominantly males with a mean age of 49 years—is distinct from the typical CAD population. In general, population mean age tends to be older with a higher burden of comorbidities. This reinforces the clinical maxim that MB should be highly suspected in younger patients presenting with exertion-related chest pain with no traditional risk factors for atherosclerosis. The exclusive involvement of the LAD in 95% of our cases also corroborates the well-established anatomical predilection of this anomaly. [2]

The Survival vs. Morbidity Paradox

The most striking finding of our study is the dichotomy between survival and symptoms. The absence of cardiovascular death and myocardial infarction in our MB cohort supports the "benign" classification when considered solely in terms of life expectancy. This is likely because MB is a stable anatomical substrate. In atherosclerosis process, the plaque can rupture and cause acute thrombotic occlusion. In myocardial bridging, the vessel itself is typically smooth and free of plaque at the site of compression. [11] However, a 'benign' prognosis does not equate to an asymptomatic course. The significantly higher rate of readmission compared to matched controls (14.3% vs 2.4%) indicates that these patients suffer from real, functional ischemia that impacts their quality of life. The mechanism driving this early readmission is likely the failure of initial medical management or patient education. When patients are discharged with a diagnosis of "non-obstructive CAD," they may not receive adequate rate-control therapy. Consequently, daily physical exertion triggers a tachycardia-dependent ischemic cascade. The failure to suppress this exercise-induced tachycardia permits diastolic shortening and intensifies compressive force, leading to recurrent angina and subsequent emergency department visit. [13]

Anatomical Predictors of Symptoms

Our analysis identified bridge length >25 mm as a significant independent predictor of recurrent symptoms. This finding is biologically plausible and rooted in the laws of fluid dynamics. According to Poiseuille's law, resistance to flow in a tube is directly proportional to its length. A longer segment of compression creates a greater cumulative resistance to blood flow. Furthermore, a longer bridge implies a larger mass of overlying myocardium, which may exert a more prolonged compressive effect that extends further into diastole. This "delayed relaxation" is the critical period where perfusion is compromised. Therefore, clinicians should pay particular attention to the length of the tunneled segment on angiography, as patients with long bridges are at higher risk of treatment failure. [5, 17]

Therapeutic Implications

The high rate of early readmission suggests that the current standard of care with observation or anti-anginal therapy is inadequate. Our data strongly supports the aggressive use of beta-blockers as the first-line therapy. Beta-blockers exert their therapeutic effect in Myocardial Bridging through two key mechanisms. First, negative chronotropy, which lengthens the diastolic filling time and allows the vessel to uncoil. Second, negative inotropy, which decreases the compressive force of the myocardial bridge.

Conversely, the use of nitrates should be strictly avoided in these patients, although they are the standard of care for atherosclerotic angina. Nitrates reduce preload, which decreases the size of the ventricular chamber. A smaller ventricle results in "systolic buckling" of the artery, paradoxically increasing the degree of systolic compression. Additionally, nitrates can cause reflex tachycardia, further exacerbating the diastolic perfusion mismatch. The recurrence of symptoms in our cohort may reflect the inadvertent use of nitrates or insufficient dosing of beta-blockers. [6, 12, 18]

The Role of Revascularization

Notably, despite a background procedural volume of 177 percutaneous coronary interventions (PCI) during the study period, no revascularization was attempted within the MB cohort (n=21).

This reflects a conservative institutional philosophy that aligns with current guidelines. Percutaneous intervention with stenting for MB is fraught with complications. The dynamic compression of the stent by the myocardium carries a high risk of stent fracture. The mechanical irritation of stent can lead to aggressive neointimal hyperplasia and high rates of in-stent restenosis. Surgical unroofing (myotomy) or CABG is generally reserved for patients with refractory symptoms who fail maximal medical therapy. The fact that patients with destructive symptoms were readmitted but did not undergo revascularization suggests that they were ultimately managed with medical therapy, or that a

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relatively high threshold for invasive intervention exists in our setting. [19, 20]

Study Limitations

Several limitations must be considered when interpreting our results. First, the retrospective design relies on the accuracy of medical records and the subjective visual assessment of angiography. We did not routinely employ functional testing such as Fractional Flow Reserve (FFR) or Instantaneous Wave-Free Ratio (iFR), which are considered the gold standards for assessing the hemodynamic significance of a bridge. [21, 22] Consequently, our cohort may have included myocardial bridges that were anatomically visible but physiologically insignificant. At the same time, some hemodynamically important bridges may have been under recognized because they appeared mild on visual assessment. Second, the sample size of 21 patients is relatively small, which limits the statistical power to detect rarer outcomes like MI or death, although it is sufficient to demonstrate the difference in readmission rates. Finally, the 12-month follow-up period is relatively short; a longer duration would be necessary to assess the potential development of proximal atherosclerosis over time.

CLINICAL PERSPECTIVES

Competency in Medical Knowledge

Myocardial bridging is often clinically categorized as a benign anatomical variant. However, this study demonstrates that patients with significant systolic compression ($\geq 50\%$) experience a high rate of recurrent hospitalizations for unstable angina (14.3%) within just 12 months of diagnosis. Clinicians should recognize that the hemodynamic burden of bridging impacts quality of life immediately, necessitating aggressive heart rate control.

Translational Outlook

Future research needs to focus on the optimal medical regimen to prevent early readmission. Randomized trials comparing aggressive beta-blockade versus calcium channel blockers in newly diagnosed MB patients are needed to establish best practices for reducing the burden of hospitalization in the first year.

CONCLUSIONS

In conclusion, our findings indicate that myocardial bridging represents a distinct clinical entity characterized by a form of “benign malignancy,” with excellent survival yet a disproportionate impact on patient morbidity and healthcare utilization. A diagnosis of myocardial bridging should therefore prompt a dedicated management strategy emphasizing strict heart rate control and structured patient education, rather than being dismissed as an incidental angiographic finding. Furthermore, identification of risk markers such as bridge length may help stratify patients at

higher risk for early readmission and guide closer clinical follow-up to mitigate recurrent hospitalizations.

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CONFLICTS OF INTERESTS

The authors declare no conflict of interest.

FINANCIAL DISCLOSURE

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