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ORIGINAL ARTICLE

Clinical Profile of Patients with Angiographically Proven Myocardial bridging

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ABSTRACT

Background: Myocardial bridging (MB) is a frequently encountered coronary anomaly, and its prevalence varies, ranging from 1.5% to 16%. The present study evaluates the clinical profile of MB in patients undergoing coronary angiography at a tertiary care center in coastal Karnataka, India.

Methodology: This prospective study was performed among patients presenting to the Department of Cardiology and Department of Emergency Medicine from July-2017 to May-2018. All the patients presenting with chest pain, dyspnoea, or fatigue; and planned for coronary angiography (CAG) of age 30 and above who had MB were included in the study. Patient data, including electrocardiogram(ECG), echocardiography(ECHO), and routine blood investigations, were recorded in the pre-designed form. The study was carried out after attaining Institutional ethical clearance.

Results: The patient cohort was predominantly composed of males (75.7%) with an average age of 55.2±0.97 years. Among 140 patients with MB, 75.7% of cases were classified as grade I, 20.7% as grade II, and 3.6% as grade III. Significant association was observed between MB grades and clinical factors, such as diastolic dysfunction, active smoking, and diabetes. Electrocardiographic abnormalities included left ventricular hypertrophy (25%), inferior wall myocardial infarction(8%), left bundle branch block (2.9%), and T-wave inversion (2.9%). Echocardiographic findings included concentric LVH (22.9%), regional wall motion abnormality (12.1%),

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and hypertrophic cardiomyopathy(3.6%). CAG unveiled various pathologies, including mild coronary artery disease(28.6%), single vessel disease(15%), double vessel disease(1.4%), branch vessel disease(5%), and multivessel disease(2.1%).

Conclusion: MB was prevalent among patients undergoing CAG. Significant association was found between grades of MB and diastolic dysfunction, active smoking, and diabetes mellitus.

KEYWORDS

• Myocardial Bridging • Coronary Angiography • Echocardiography • Acute Coronary Syndrome

INTRODUCTION

Myocardial bridging (MB) describes an anatomical variant in which a portion of an epicardial coronary artery tunnels through the myocardium and undergoes transient compression during systole. Although often incidental, MB has been linked to ischemic events, arrhythmias, and even sudden cardiac death in some individuals.[1–3] Reported prevalence varies markedly by detection method: angiographic series cite rates of 0.5 % to 2.5 %, whereas autopsy studies document frequencies up to 86 %.[4–6] The mid-segment of the left anterior descending artery (LAD) is most commonly involved, though marginal and diagonal branches may also be bridged in nearly one-fifth of cases.[4]

Ferreira and colleagues classified bridges by depth into superficial types, which generally do not impede flow, and deep types, which can significantly constrict the vessel.[5] Noble et al. further graded systolic narrowing as follows: Grade I (<50 % luminal reduction), Grade II (50–75 %), and Grade III (>75 %).[7] Morphologically, bridge lengths range from approximately 2.3 to 42.8 mm and thicknesses from 1.0 to 3.8 mm, with mean values of 14.6 ± 9.0 mm and 1.23 ± 1.32 mm, respectively.[8,9]

In this prospective cohort at a tertiary centre in coastal Karnataka, we sought to characterise the clinical profile, angiographic features, and associated risk factors in patients with angiographically confirmed MB.

METHODOLOGY

Study Design

This prospective observational, time-bound, single-arm study was conducted among patients who presented to the cardiology and emergency medicine departments from July 2017 to May 2018. Patients who were 30 years old and above and had chest pain, dyspnea, fatigue, or planned for coronary angiography (CAG) were included in the study. A total of 140 patients with myocardial bridging identified on angiograms were included in this study. The patient's chest pain was categorized based on the presence of substernal chest pain or discomfort that was provoked by exertion or emotional stress and was relieved by rest and/or nitroglycerin. Chest pain was classified as "typical angina" if all three descriptors were present and "atypical" if less than three descriptors were present, as defined by the American College of Cardiology/ American Heart Association 2002 Guideline Update on Exercise Testing.[9] All patients underwent electrocardiogram (ECG) and echocardiogram (ECHO) tests, as well as blood investigations including renal function test, thyroid stimulating hormone (TSH), Glycated hemoglobin (HbA1c), and complete blood cell counts (CBC). The ECG was examined for ischemia and infarction changes; and left ventricular hypertrophy (LVH) was diagnosed using voltage criteria. ECHO was used to detect Concentric LVH, diastolic dysfunction, and regional wall motion abnormality. Coronary angiography was performed through the radial or femoral route, and normal saline was used for hydration before and after the procedure.

Noble *et al.'s* myocardial grading system was utilized: Grade I for < 50% narrowing, Grade II for 50-75% narrowing, and Grade III for > 75% narrowing.[7] Coronary artery disease is classified as mild if the lesion is causing less than 50% stenosis, sub-critical if the lesion is causing 50-70% stenosis, and significant if the stenosis is greater than 70%.

The Coronary Slow Flow Phenomenon (CSFP) can be diagnosed using the TIMI flow grade or TIMI frame count. A TIMI-2 flow grade (i.e., requiring ≥3 beats to opacify the vessel) or a corrected TIMI frame count of >27 frames is commonly used. The latter is based on images acquired at 30 frames/second and a correction factor of 1.7 for the LAD.[10]

ECG Stress Testing

The exercise stress testing is performed using the Bruce protocol. This protocol involves continuous monitoring of heart rate, blood pressure, and a 12-lead electrocardiogram throughout the test. The test will be stopped if there are any cardiac symptoms or if there is more than 2mm horizontal or downsloping ST-segment depression measured 80 ms after the J-point for more than 3 consecutive beats. A positive outcome is defined by the occurrence of angina, ischemia (horizontal or descending ST-segment depression >1 mm, or ST-segment elevation), or inotropic failure (a fall of systolic arterial blood pressure [SBP] > 10 mmHg).[11]

A negative result is determined when the peak heart rate surpasses 85% of the expected rate for age without the occurrence of angina or ischemia. The test will be labeled inconclusive if the specified criteria are not met. An inconclusive interpretation is assigned when the peak heart rate >85% of the expected rate for age cannot be achieved (e.g., due to beta-blockade) or in the presence of intermediate ECG changes (e.g., T-wave changes without ST-segment shift) without pain.

Statistical Analysis

Data cleaning was conducted using Microsoft Excel, and statistical analyses were performed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Shapiro–Wilk test. Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data and as median with interquartile range (IQR) for non-normally distributed data. Categorical variables were presented as frequencies and percentages. Associations between Clinical factors and Different grades of MB were evaluated using the Chi-square test or Fisher's exact test, as appropriate. A p-value of <0.05 was considered statistically significant.

Ethical Considerations

We ensured all ethical guidelines were followed. The study protocol was approved by the Institutional Ethics Committee (Reg. No. IEC: 418/2017). This study was conducted in accordance with the principles of the Declaration of Helsinki after obtaining written consent from the patients. The confidentiality and privacy of all patients were safeguarded throughout the study.

RESULTS

Demographics

A total of 140 patients undergoing coronary angiograms who had MB were enrolled in the study. Participants' mean age was 55.2±0.97, with most being male patients (75.7%). Out of 140, 66 (47.14%) and 35 (25.2%) were reported to have hypertension and diabetes mellitus, respectively. Of 140, 61 (43.6%) and 17 (12.1%) were active smokers and alcoholics, respectively (Table 01).

Table 1: Demographics and Clinical Features

Variable	Frequency (%)
Mean Age (Years ± SD)	**55.2 ± 0.97**
Age Group	
30-39	11 (7.9)
40-49	35 (25.0)
50-59	40 (28.6)
60-69	39 (27.9)
70-79	12 (8.6)
80-89	3 (2.1)
Gender	
Male	106 (75.7)
Female	34 (24.3)
Co-Morbidity	
HTN	66 (47.14)
DM	35 (25.2)
Social Habit	
Smoking	61 (56.4)
Alcohol	17 (12.1)
Presented With	
Atypical Angina	82 (58.6)
Typical Angina	35 (25.0)
Dyspnoea on Exertion	23 (16.4)
SBP	
100-119	26 (18.57)
120-139	55 (39.28)
140-159	47 (33.57)
160-179	8 (5.71)
180-200	4 (2.85)

Abbreviations: DM: diabetes mellitus; HTN: hypertension; SBP: systolic blood pressure

Clinical Features

Out of 140 participants, 82 (58.6%) reported experiencing atypical angina, 35 (25%) reported experiencing typical angina, and 23 (16.4%) reported experiencing dyspnea on exertion. Among the 140 participants, 55 (39.28%) were reported to have a systolic blood pressure (SBP) of 120-139, and 47 (33.57%) were reported to have an SBP of 140-159 (Table 1).

ECG Findings

35 out of 140 individuals (25%) had left ventricular hypertrophy (LVH), 11 (8%) had inferior wall myocardial infarction, 4 (2.9%) had left bundle branch block, and 4 (2.9%) had T-wave inversion (Table 2).

Table 2: ECG, ECHO and Coronary Angiography Findings

Variable	Frequency (%)
ECG Findings	
LVH	35 (25.0)
IWMI	11 (7.9)
LBBB	4 (2.9)
RBBB	1 (0.7)
LWMI	1 (0.7)
AMWI	1 (0.7)
T-Wave Inversion	4 (2.9)
ECHO Findings	
Concentric LVH	32 (22.9)
RWMA	17 (12.1)
HCM	5 (3.6)
Diastolic Dysfunction	52 (37.1)
Coronary Angiography	
Normal	30 (21.5)
Mild	40 (28.6)
SVD	21 (15.0)
DVD	2 (1.4)
Subcritical	3 (2.1)
BVD	7 (5.0)
MVD	3 (2.1)
Slow flow coronaries	34 (24.3)
Myocardial Bridging	
Grade I	106 (75.7)
Grade II	29 (20.7)
Grade III	5 (3.6)

Abbreviations: AMWI: Anterior wall myocardial infarction; **BVD:** Branch vessel disease; **DVD:** Double vessel disease; **ECG:** Electrocardiogram; **ECHO:** Echocardiogram; **HCM:** Hypertrophic cardiomyopathy; **IWMI:** Inferior wall myocardial infarction; **LBBB:** Left bundle branch block; **LVH:** Left ventricular hypertrophy; **LWMI:** Lateral wall myocardial infarction; **MVD:** Multivessel disease; **RBBB:** Right bundle branch block; **RWMA:** Regional wall motion abnormality; **SVD:** Single vessel disease.

ECHO Findings

Out of the 140 participants, 32 (22.9%) reported having concentric left ventricular hypertrophy (LVH), 17 (12.1%) reported having regional wall motion abnormality (RWMA), and 5 (3.6%) reported having hypertrophic cardiomyopathy (HCM). Among the 140 participants, 52 (37.1%) reported having diastolic dysfunction (Table 2).

Coronary Angiography Findings

The distribution of coronary artery disease is mentioned in Table 2. Out of a total of 140 patients, 106 (75.7%) had Grade I, 29 (20.7%) had Grade II, and 5 (3.6%) had Grade III myocardial bridging. (Table 2).

Factors Associated with Myocardial Bridging

A statistically significant association was observed between myocardial bridging grades and the presence of diastolic dysfunction ($p = 0.006$), smoking status ($p = 0.020$), and diabetes mellitus ($p < 0.05$). The prevalence of diastolic dysfunction increased progressively with higher grades of myocardial bridging (30.2% in Grade I, 55.2% in Grade II, and 80% in Grade III). However, no consistent linear trend was observed for smoking or diabetes mellitus across the grades of myocardial bridging, despite the statistically significant associations. (Table 3).

Table 3: Factors Associated with Myocardial Bridging

Variables	Myocardial Bridging			p-value
	Grade I (n=106) (%)	Grade II (n=29) (%)	Grade III (n=5) (%)	
SBP Group				
100-119	19 (17.9)	5 (17.2)	2 (40)	0.370
120-139	43 (40.6)	12 (41.4)	0 (0)	

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Variables	Myocardial Bridging			p-value
	Grade I (n=106) (%)	Grade II (n=29) (%)	Grade III (n=5) (%)	
140-159	39 (36.8)	7 (24.1)	1 (20)	
160-179	3 (2.8)	4 (13.8)	1 (20)	
180-200	2 (1.9)	1 (3.4%)	1 (20)	
ECG Findings				
LVH	25 (23.6)	8 (27.6)	1 (20)	0.104
IWMI	3 (2.8)	6 (20.7)	2 (40)	
LBBB	3 (2.8)	1 (3.4)	2 (40)	
RBBB	1 (0.9)	0 (0)	0 (0)	
LWMI	1 (0.9)	0 (0)	0 (0)	
AMWI	1 (0.9)	0 (0)	0 (0)	
T-Wave Inversion	3 (2.8)	1 (3.4)	0 (0)	
ECHO Findings				
Concentric LVH	23 (21.7)	7 (24.1)	2 (40)	0.091
RWMA	8 (7.5)	8 (27.6)	1 (20)	
HCM	4 (3.8)	1 (3.4)	0 (0)	
Diastolic Dysfunction	32 (30.2)	16 (55.2)	4 (80)	0.006*
ST Depression Stage				
Stage II	14 (13.2)	1 (3.4)	0 (0)	0.571
Stage III	26 (24.5)	6 (20.7)	1 (20)	
Stage IV	1 (0.9)	1 (3.4)	0 (0)	
Coronary Artery Disease				***0.085***
Mild	33 (31.1)	6 (20.7)	1 (20)	
SVD	13 (12.3)	5 (17.2)	3 (60)	
DVD	1 (0.9)	1 (3.4)	0 (0)	
Subcritical	2 (1.9)	1 (3.4)	0 (0)	
BVD	4 (3.8)	2 (6.9)	(20)	
MVD	1 (0.9)	2 (6.9)	0 (0)	
Slow flow	30 (28.3)	4 (13.8)	0 (0)	0.118
Smoking	41 (38.7)	19 (65.5)	1 (20)	0.020*
Alcohol	12 (11.3)	4 (13.8)	1 (20)	0.806
Diabetes Mellitus	27 (25.5)	8 (27.6)	0 (0)	<0.05*
Hypertension	47 (44.3)	16 (55.2)	3 (60)	0.493

Abbreviations: AMWI: anterior wall myocardial infarction; BVD: branch vessel disease; DVD: double vessel disease; ECG: electrocardiogram; ECHO: echocardiogram; HCM: hypertrophic cardiomyopathy; IWMI: inferior wall myocardial infarction; LBBB: left bundle branch block; LVH: left ventricular hypertrophy; LWMI: lateral wall myocardial infarction; MVD: multivessel disease; RBBB: right bundle branch block; RWMA: regional wall motion abnormality; SVD: single vessel disease.

DISCUSSION

Our angiographic prevalence (approximately 6% in those undergoing catheterisation) aligns with prior series reporting 0.5 %–2.5 % detection by angiography, yet is lower than autopsy-based estimates.[6,7] Consistent with existing data, bridged segments predominantly occurred in male patients and presented a decade earlier than typical atherosclerotic coronary disease cohorts.[12,13] Unlike many studies where typical exertional angina predominates, over half of our patients reported atypical chest pain, suggesting that the symptom spectrum may vary by population or referral pattern.[14]

Significant ST-segment depression was observed in the precordial leads of patients during the exercise treadmill test, particularly in stage 2 and stage 3. Although this

depression resolved during the early recovery stage, patients with coronary artery disease exhibited persistence of the depression in the late recovery stage. Significant ischemic ST-segment depression >0.1 mV was identified in 28% to 67% of patients in whom the treadmill test was done.[15] Huang et al. demonstrated that patients who had abnormal exercise treadmill tests and angiographic evidence of myocardial bridge also had abnormal myocardial SPECT perfusion defects.[16]

Diastolic dysfunction was significantly associated with higher-grade bridges in our cohort, possibly reflecting a demand–supply mismatch in the presence of dynamic systolic compression.[17]

It has been suggested that myocardial bridges that are longer, wider, and thicker may increase the risk of clinical events.[18] Despite the high prevalence of bridging in the general population, there is a low incidence of exercise-related events, which supports the concept that most myocardial blood flow occurs during diastole. It is believed that reduced coronary blood flow reserve may be the reason for ischemia or myocardial infarction (MI) in these patients.[17]

Histopathological studies indicate that the tunnelled arterial segment exhibits a thinner intima and lower expression of atherogenic mediators—such as endothelial nitric oxide synthase, endothelin-1, and angiotensin-converting enzyme—rendering it relatively protected from plaque formation. In contrast, the proximal segment, exposed to altered shear stress, demonstrates a higher propensity for atherosclerosis. This dichotomy may underlie the observation that significant coronary lesions often localise just proximal to the bridge.[19,20]

In our study, we observed significant left anterior descending (LAD) bridging in patients presenting with acute inferior wall myocardial infarction (MI). Subsequently, these patients recovered after revascularization without experiencing cardiogenic shock. The clinical significance of myocardial bridging in patients with inferior wall MI and shock was elucidated by Yano K *et al*[21] They noticed that patients developed shock despite successful reperfusion of the infarct-related lesion (IRL), suggesting that myocardial bridging plays a role in left ventricular function during the acute stage of inferior wall MI.[21] We did not find any patients with positive troponin-T and myocardial bridging in the absence of coronary artery disease. Brolin EB *et al*. suggested that there is no causal link between myocardial bridging and myocardial infarction (MI) in cases without obstructive coronary atherosclerosis (MINOCA).[22] Interestingly, coronary atherosclerosis was frequently observed in vessels with myocardial bridging. Thus, myocardial bridging may be an important risk factor for coronary artery disease. While beta-blockers remain first-line therapy to reduce compression and improve diastolic filling, adjunctive statin therapy may benefit those with coexistent slow-flow phenomena by improving endothelial function.[19,23]

CONCLUSION

In conclusion, myocardial bridging was frequently observed among patients undergoing coronary angiography, particularly in middle-aged males presenting with atypical angina, with Grade I being the predominant form (75.7%), followed by Grade II (20.7%) and Grade III (3.6%). A significant association was identified between the severity of myocardial bridging and the presence of diastolic dysfunction, while significant associations with smoking and diabetes mellitus were also noted, although without a consistent trend across bridging grades. These findings underscore the potential interplay between myocardial bridging severity and structural cardiac changes. Prospective studies with larger sample sizes and longer follow-up periods are warranted to clarify the prognostic implications and therapeutic strategies in this subgroup of patients. Additionally, myocardial bridging can be a possibility in asymptomatic patients (those without angina during treadmill testing) who exhibit positive treadmill test results with ST-T changes noted during peak exercise, which subsequently disappear in early recovery.

List of Abbreviations:

CAG – Coronary Angiography

CBC – Complete Blood Cell Counts

CSFP – Coronary Slow Flow Phenomenon

ECG – Electrocardiogram

ECHO – Echocardiography

eNOS – Endothelial Nitric Oxide Synthase

HbA1c – Glycated hemoglobin

HCM – Hypertrophic Cardiomyopathy

IEC – Institutional Ethical Committee

IRL – Infarct-Related Lesion

LAD – Left Anterior Descending

LVH – Left Ventricular Hypertrophy

MB – Myocardial Bridging

MI – Myocardial Infarction

MINOCA – Myocardial Infarction with Non-Obstructive Coronary Arteries

RWMA – Regional Wall Motion Abnormality

SBP – Systolic Blood Pressure

SPECT – Single-Photon Emission Computed Tomography

SPSS – Statistical Package for Social Science

TIMI – Thrombolysis in Myocardial Infarction

TSH – Thyroid Stimulating Hormone

Clinical Trial Number: Not Applicable.

Human Ethics and Consent to Participate Declarations: All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1964 and later versions. Informed consent was taken. The study protocol was approved by the Institutional Ethics Committee (Reg. No. IEC 418/2017).

Availability of Data and Materials: The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests: The authors declare that they have no competing interests.

Consent to Participate: All participants have given written consent.

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