

Myocardial Bridging Causing Exertional Angina in a Young Adult: A Case Report

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Case Report

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Abstract

Myocardial bridging (MB), a congenital coronary anomaly marked by an intramyocardial arterial course, is increasingly implicated in ischemia among patients without obstructive coronary disease. This case underscores the clinical importance of MB in young, low-risk individuals presenting with angina-like symptoms. A 30-year-old Ethiopian male with no cardiovascular risk factors reported recurrent exertional chest pain radiating to the left arm. Initial assessment revealed peaked T-waves on electrocardiography (ECG) and cardiac troponin levels below detectable thresholds. Coronary angiography revealed systolic compression (“milking effect”) of the distal left anterior descending artery (LAD) due to intramyocardial tunneling, confirming ischemia with nonobstructive coronary arteries (INOCA). Despite optimal β -blocker therapy (metoprolol titrated to 75 mg/day), symptoms persisted, prompting surgical supra-arterial myotomy, which resulted in symptom resolution. This case highlights the imperative to suspect MB in patients with unexplained angina, particularly when traditional atherosclerotic risk factors are absent. Angiography remains the gold standard, whereas β -blockers are first-line therapy. Surgical interventions can be efficacious in refractory cases. Further research is needed to standardize diagnostic and therapeutic approaches for this underrecognized condition.

Background

Myocardial bridging (MB) is a congenital coronary anomaly that involves systolic luminal narrowing of an intramyocardial arterial segment (1). It is detected incidentally in 0.5–16% of angiographic studies and predominantly affects the left anterior descending artery (LAD) (2)(3).

Although often asymptomatic, MB is linked to exertional angina, acute coronary syndrome, arrhythmias, syncope, and sudden cardiac death (4). Emerging evidence identifies MB as a frequent yet underrecognized contributor to myocardial infarction with nonobstructive coronary arteries (MINOCA), either independently or synergistically with other mechanisms (5). Accurate MB diagnosis has critical therapeutic implications (6).

We present a 30-year-old male with exertional angina and ischemia secondary to LAD bridging, illustrating the necessity of MB consideration in young, low-risk patients without atherosclerosis.

Case Presentation

A previously healthy 30-year-old Ethiopian male with no family history of cardiovascular disease or psychosocial risk factors presented to the emergency department with a 48-hour history of intermittent left-sided chest pain. Symptoms included pressure-like discomfort lasting 30–60 minutes per episode, occurring at rest and exacerbated by exertion, with radiation to the left arm. A similar episode one month prior, provoked by cycling and relieved by rest, prompted outpatient ECG evaluation and subsequent cardiology referral.

Admission vital signs were stable: blood pressure 122/78 mmHg, heart rate 79 bpm, respiratory rate 16/min, and SpO₂ 95% (room air). The physical examination was unremarkable. High-sensitivity cardiac troponin I was undetectable (< 1.5 ng/L; reference < 14 ng/L). The results of laboratory studies, including complete blood count, metabolic panel, and inflammatory marker data, were normal(Table 1). Chest x-ray was normal. Transthoracic echocardiography demonstrated preserved left ventricular ejection fraction (LVEF: 65%) without structural abnormalities. A 12-lead ECG revealed sinus rhythm with a rate of 65 and no significant ST or T wave changes to suggest myocardial ischemia (Fig. 1) and a QT interval (QTc) of 360 ms (normal: 360–460 ms). Prior ECG findings were unchanged, and exercise stress testing failed to elicit symptoms or ischemic changes. Coronary angiography revealed dynamic systolic compression (“milking effect”) of the distal LAD due to intramyocardial tunneling, which was consistent with MB (Fig. 2). Atherosclerotic lesions were absent.

Table 1
Laboratory Investigations

Investigation	Result	Unit	Reference Range
Troponin High Sensitive	< 1.5	ng/L	17–50
CBC			
WBC	8.56		3.9–10.1
Neutrophils	57.5	%	40.4–74.6
Hemoglobin	17.3	g/dL	12.7–17.1
Hematocrit	48.6	%	34.4–48.3
Platelet	222	/μL	100–300
Liver Panel			
Alkaline Phosphatase	78	U/L	55–149
ALT (SGPT)	29	U/L	0–41
AST (SGOT)	18.1	U/L	0–40
Hepatitis B Surface Antigen	NEG		Negative
Hepatitis C Virus	NEG		Negative
ESR	5	mm/hr	0–22
Electrolyte Panel			
Sodium	132	mmol/L	136–145
Potassium	4.15	mmol/L	3.5–5.1
Chloride	98	mmol/L	98–107
Renal Function Test			
Creatinine	0.71	mg/dL	0.7–1.2
Urea	25.7	mg/dL	16.6–48.5
Lipid Panel			
HDL cholesterol	49.0	mg/dL	35–65
LDL cholesterol	69.6	mg/dL	0-100
Total cholesterol	117.9	mg/dL	0-200
Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; ESR, erythrocyte sedimentation rate; FBS, fasting blood sugar.			

Investigation	Result	Unit	Reference Range
Triglycerides	154.6	mg/dL	0-150
FBS	86	mg/dl	70–100
Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; ESR, erythrocyte sedimentation rate; FBS, fasting blood sugar.			

The patient was diagnosed with INOCA secondary to myocardial bridging and was initially treated with metoprolol 25 mg twice daily. After one month of follow-up with no improvement in symptoms despite adherence to the treatment, the dosage was gradually increased to 75 mg daily. Despite the increased dose, his symptoms persisted. Consequently,the patient underwent surgical intervention in the form of supra-arterial myotomy, which resulted in marked symptomatic improvement. Postoperatively, the patient experienced significant relief from exertional chest pain and currently remains under regular cardiology follow-up with a favorable prognosis.

Given the surgical intervention, adherence to post-operative care and the risk of potential complications were relevant considerations; however, no adverse events were reported during the follow-up period. The patient expressed satisfaction with the treatment process and highlighted a notable improvement in his quality of life following surgery. He continues to be monitored routinely to assess long-term outcomes.

Discussion

First documented pathologically by Reyman (1737) and angiographically by Portmann and Iwig (1960) (7, 8), MB involves coronary artery traversal through the myocardium rather than its typical epicardial path. Prevalence varies widely: pathologic analyses indicate a mean frequency of 25% (range: 5–86%), aligning with coronary computed tomography findings (9). MB is more prevalent in heart transplant recipients and hypertrophic obstructive cardiomyopathy (HOCM) patients, where hyperdynamic contractions unmask latent bridges (10). The mid-LAD segment is most commonly involved (11), as observed here.

Although frequently incidental, symptomatic MB may manifest as ischemia, acute coronary syndrome (12, 13), arrhythmias (e.g., supraventricular tachycardia (14) ventricular tachycardia, or atrioventricular block), myocardial stunning, syncope, or sudden death (15–17). The MB prevalence in MINOCA cohorts (2.9%) exceeds that in atherosclerotic populations (5)

Diagnostic modalities include coronary angiography (gold standard), intravascular ultrasound (IVUS), coronary CT angiography (CCTA), and optical coherence tomography (OCT) (18). Angiography in this case demonstrated systolic LAD narrowing (“milking effect”), distinguishing MB from fixed stenosis. IVUS further delineates bridge anatomy via the “half-moon” sign (persistent echolucency between the artery and myocardium) (19) Noninvasive techniques (CT, MRI, echocardiography) are less utilized; their unavailability precludes their use here. Notably, angina and stress-induced ischemia are inconsistently present in MB, and postdiagnostic management lacks consensus (20).

Therapeutic strategies include pharmacologic agents like β -blockers, calcium channel blockers to reduce systolic compression and improve perfusion (20), stenting, surgical myotomy, or CABG. β -blockers are preferred despite the limited number of comparative trials. Nitrates may worsen symptoms via coronary vasodilation and sympathetic activation, whereas aspirin/statin use is unwarranted without atherosclerosis (19). A study of 15 patients with severe myocardial bridging revealed that intravenous esmolol, a short-acting beta-blocker, effectively reduced Doppler flow velocities during tachypacing, returning them to baseline levels and normalizing the diastolic-to-systolic flow velocity ratio. Patients also experienced symptomatic relief and improvement in stress-induced ST depression (20).

Surgical treatment with dissection of the overlying myocardium (surgical unroofing) is often limited to patients who are refractory to medical therapy (1). In a small series of patients, favorable clinical outcomes have been observed; however, there remains a risk of serious complications, including right ventricular perforation and left ventricular aneurysm (21). In our patient's case, the symptoms of angina did not improve even after the metoprolol dosage was increased to 75 mg orally per day, which led to the decision for surgical intervention. Consequently, he underwent supra-arterial (LAD) myotomy. When surgery is indicated, the decision to opt for myotomy or CABG is determined on an individual basis. In instances where arteries run deeply through the septum, nearing the right ventricular subendocardium and making surgical access to the intramuscular coronary artery challenging, CABG is preferred over myotomy, with the left internal mammary artery (LIMA) being the optimal graft choice (22). However, with a high graft failure rate, CABG has demonstrated limited effectiveness. There is limited information regarding the prognosis of surgical interventions for symptomatic myocardial bridging. In a follow-up study involving 11 patients who underwent surgical intervention, no major adverse cardiac events were reported, suggesting that surgical intervention is relatively safe (23). The long-term prognosis for isolated MB is favorable, with a 97.5% 5-year survival rate in one cohort (11).

CONCLUSION

Although anatomically benign, myocardial bridging (MB) is a critical yet underrecognized etiology of ischemia in young, low-risk patients. This case demonstrates that MB mimics atherosclerotic angina, necessitating distinct diagnostic and therapeutic approaches. A 30-year-old male with exertional symptoms and dynamic ECG changes was diagnosed with LAD bridging, reinforcing the role of the MB in INOCA evaluations when atherosclerosis is absent.

Timely diagnosis prevents mismanagement: β -blockers (e.g., metoprolol) are first-line, while nitrates and antiplatelets should be avoided. Favorable prognoses are typical, although severe cases require vigilant monitoring.

This report advocates heightened MB suspicion in young adults with unexplained angina. Future research must prioritize diagnostic standardization and comparative therapeutic trials to optimize care for this overlooked population.

Declarations

Ethics approval and consent to participate: St. Paul's Hospital Millennium Medical College Institutional Review Board deems ethical approval unnecessary, as this is a rare case encountered during clinical practice and does not involve experiments on humans or animals.

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Availability of data and materials: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no conflicts of interest.

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Authors' contribution: H.A. conceptualized the study, drafted the manuscript, and led critical revisions for intellectual coherence. A.W., contributed substantially to the conceptual framework, directed the comprehensive literature synthesis, provided expert interpretation of clinical evidence, and established rigorous citation standards. N.G. conducted clinical evaluations, analyzed diagnostic data, and integrated patient findings. A.A. contributed in imaging interpretation and prepared visual documentation. T.J. oversaw methodological design and supervised scientific rigor. All authors reviewed, revised, and approved the final manuscript

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Figures

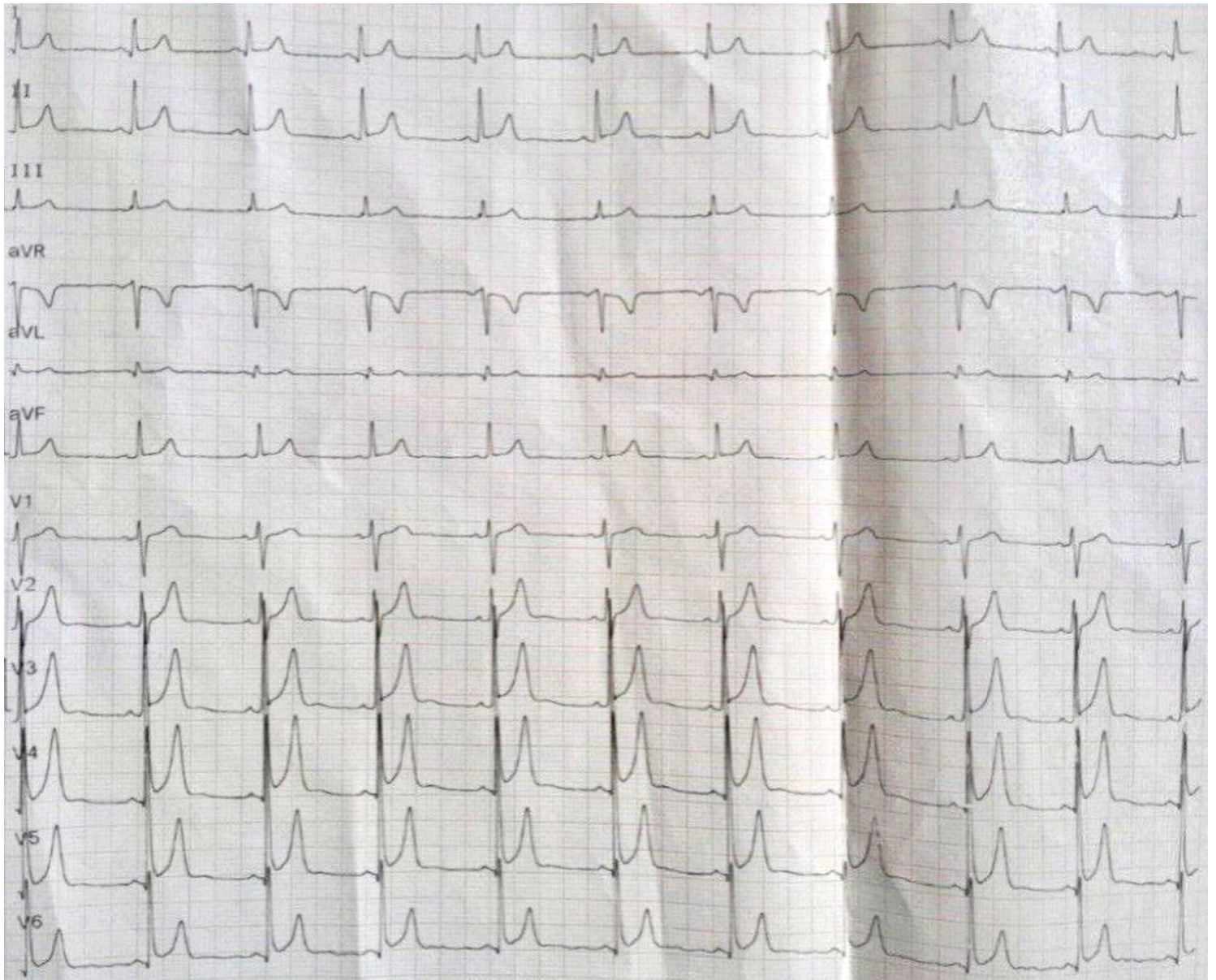


Figure 1

Electrocardiogram (ECG) Findings

ECG at 65 bpm demonstrates normal sinus rhythm, normal axis, and normal intervals (PR, QRS, QTc 360 ms). Tall T waves in lead V2 are a non-specific finding. No ST-segment changes or ischemic patterns are present.

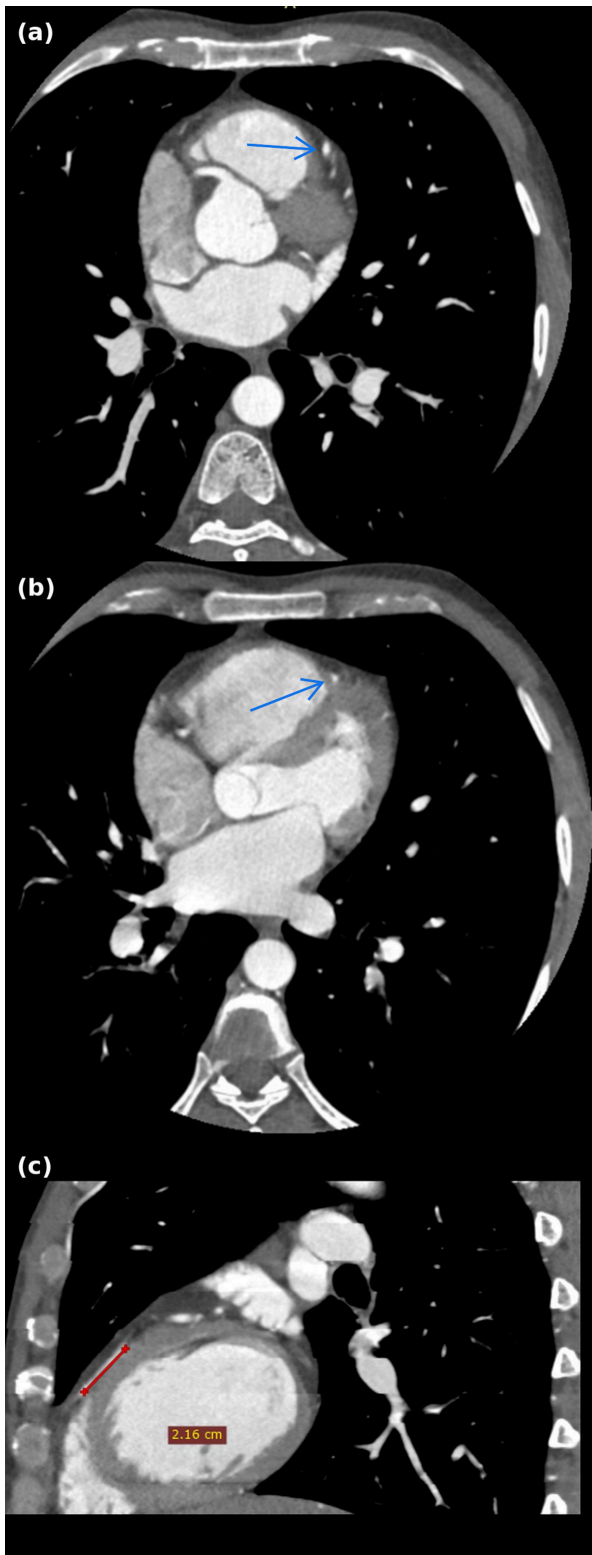


Figure 2

CT Angiographic Findings Demonstrating Myocardial Bridging of the LAD

(a) Normal LAD course within epicardial fat (blue arrow). (b) Systolic compression of the LAD (blue arrow) by overlying myocardium. (c) Axial view showing the intramyocardial course of the mid-LAD (arrows) with a tunnel length of 2.16 cm (double-headed arrow).

