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Mini Review

Nitroglycerin-Induced Coronary Collapse in Myocardial Bridging-Pathophysiology and Clinical Implications

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ABSTRACT

Myocardial Bridging (MB) is a congenital anomaly in which an epicardial coronary artery, commonly the mid-Left Anterior Descending (LAD), tunnels through the myocardium rather than coursing along its surface. While typically benign, dynamic systolic compression of the bridged segment can precipitate ischemia, arrhythmias, or even infarction. Nitroglycerin, a classic anti-anginal agent, paradoxically aggravates MB-related obstruction by increasing vessel wall compliance and amplifying systolic narrowing. This mini-review summarizes current understanding of MB pathophysiology, diagnostic imaging modalities, and management strategies, highlighting the paradoxical pharmacologic effect of nitrates observed in a recent case of nitroglycerin-induced coronary collapse.

Keywords: Myocardial Bridging, nitroglycerin, ischemia, collapse

INTRODUCTION

Myocardial bridging was first described anatomically in the 18th century, but its clinical importance remains underrecognized [1]. The reported prevalence ranges from 0.5%-5% on coronary angiography to over 60% in autopsy or Computed Tomography (CT) studies [2]. Although many bridges are asymptomatic, compression of the tunneled segment during systole may cause angina, myocardial ischemia, arrhythmias, or sudden cardiac death.

Our recently published case report described a 48-year-old woman with LAD bridging who developed near-occlusive (99%) systolic collapse after intracoronary nitroglycerin administration [3]. This was successfully reversed with calcium-channel blockade and ultimately treated surgically. This case provides a framework to review the paradoxical behavior of nitrates in MB and discuss optimal diagnostic and therapeutic approaches.

LITERATURE REVIEW

During systole, myocardial contraction compresses the intramural arterial segment, reducing luminal diameter and delaying early diastolic relaxation. Key determinants of severity are multifactorial. First, the physical anatomy and the parallel relationship of the depth and length of the bridge significantly influences the degree of infarction. Secondly, the sympathetic response of the heart rate and contractility can decrease diastole. Finally, the presence of vasospasm or hypertrophy can amplify wall tension [4,5].

Unlike atherosclerotic narrowing, MB-related ischemia arises from dynamic mechanical compression, not plaque formation. Proximal to the bridged segment, turbulent flow can promote endothelial injury and atherogenesis, whereas the tunneled portion itself remains spared.

Nitroglycerin relaxes vascular smooth muscle *via* nitric-oxide–mediated cGMP pathways, reducing preload and myocardial oxygen demand. In MB, however, vasodilation of non-bridged segments increases the compliance differential between normal and tunneled arteries. Quantitative angiography and Intravascular Ultrasound (IVUS) demonstrate that nitroglycerin has two major effects. It increases diastolic diameter of adjacent segments, while it also enhances systolic narrowing within the bridged zone by up to 20%-40% [6].

This results in exaggerated "milking" of the vessel and delayed diastolic filling. Reflex tachycardia further shortens diastole, compounding ischemia. The phenomenon underscores why nitrates are contraindicated or used with caution in symptomatic MB [1].

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DISCUSSION

Current interventions highlight key modalities that are used in the early detection and management of myocardial bridging. Coronary angiography classically demonstrates a transient "step-down, step-up" systolic pattern, known as the milking effect, but often underestimates MB prevalence since compression may be missed without provocation. Coronary CT Angiography (CCTA) offers superior spatial resolution and sensitivity compared to invasive angiography, effectively visualizing both the anatomic bridge and dynamic vessel compression [7].

Utilizing Intravascular Ultrasound (IVUS) reveals the hallmark "half-moon" echolucent halo over the tunneled segment, quantifies luminal narrowing, and assesses wall morphology, while Optical Coherence Tomography (OCT) refines this by evaluating intimal integrity and confirming the absence of atherosclerosis. Functional imaging modalities, such as myocardial perfusion imaging and Fractional Flow Reserve (FFR), determine physiologic relevance. Findings suggestive of stress-induced defects or FFR <0.75 indicate hemodynamically significant MB [8].

Medical management remains first-line, focusing on reducing heart rate and contractility. β-blockers are preferred for decreasing systolic compression by prolonging diastole. Non-dihydropyridine calcium channel blockers (verapamil, diltiazem) are useful alternatives, especially when vasospasm is present. Nitrates should be avoided because they can exacerbate obstruction through increased wall compliance and reflex tachycardia [1,9].

Percutaneous Coronary Intervention (PCI) is reserved for refractory cases due to high risks of in-stent restenosis, fracture, or perforation from mechanical stress [10]. Surgical options, including myotomy (unroofing) or LIMA–LAD bypass, serve as definitive therapies for severe, symptomatic MB unresponsive to medication, with symptom resolution rates exceeding 90% [11].

Recognition of MB-related ischemia is critical, particularly in younger patients or those without coronary atherosclerosis who experience nitrate-worsened angina.

Future research priorities should focus on defining standardized imaging criteria, and elucidating molecular factors influencing vascular compliance within the bridged segments. Furthermore, developing personalized pharmacotherapy that mitigate systolic compression without reflex hemodynamic effects can maximize therapeutic results.

CONCLUSION

Myocardial bridging represents a dynamic, potentially reversible cause of myocardial ischemia. The paradoxical response to nitroglycerin emphasizes the need for individualized therapy guided by detailed imaging and hemodynamic assessment. Clinicians should recognize MB as a distinct pathophysiologic entity and exercise caution when using nitrates in these patients. A multidisciplinary approach, incorporating cardiology, cardiac imaging, and surgery, optimizes outcomes and advances understanding of this underappreciated condition.

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