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HISTOLOGICAL ANALYSIS OF CORONARY ATHEROSCLEROSIS AT MYOCARDIAL BRIDGES: A CADAVERIC STUDY

Anatomy

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ABSTRACT

Introduction: Coronary artery disease resulting from atherosclerosis has reached epidemic proportions in India, being the primary cause of death globally and accounting for one-third of all fatalities. Presently, its prevalence stands at 14% in urban areas and 7% in rural regions of India. By 2016, the number of CAD patients had risen to 24 million. CAD was responsible for the most deaths (18% of total deaths), while stroke ranked fifth (7% of total deaths) in India. Atherosclerotic plaques typically develop in specific areas of the coronary vasculature where blood flow is disrupted. One such area is near myocardial bridges, a congenital condition that manifests in various clinical presentations. **Aims and Objectives:** The current study aimed to examine myocardial bridges across various branches of the coronary arteries and assess the association of bridges with atherosclerosis in hearts lacking a history of cardiac disease. **Materials and Methods:** This study was conducted at Department of Anatomy and Forensic Medicine, KGMU Lucknow UP, India, using 50 adult human hearts without known cardiac disease. The hearts were dissected and examined to determine the number and location of myocardial bridges (MB). 78 tissue samples were taken at 3mm intervals from the coronary arteries above, below, and distal to the MB. These samples underwent whole histological slide preparation and were observed microscopically to assess for atherosclerosis. **Results:** In present study out of 50 cases 26 (52%) had MB (20 male, 6 female). Total 78 segments were evaluated which include proximal, below and distal to the myocardial bridge. Majority of atherosclerosis was present proximal to bridges i.e.,77%(p=<0.001). **Conclusion:** In hearts without a history of cardiac disease, the proportion and severity grade of atherosclerosis are notably elevated at the proximal ends of myocardial bridges. Identifying these high-risk zones for atherosclerosis could pave the way for targeted preventive strategies in the future.

KEYWORDS

Myocardial Bridges, Atherosclerosis, Coronary arteries & Cardiac Disease.

INTRODUCTION

Coronary arteries normally lie subepicardially, but when an artery submerges into the myocardium and then reappears subepicardially after a short intramural course, it is termed an embedded coronary artery, with the myocardium above it referred to as a myocardial bridge. The concept of myocardial bridging was first described by Reyman in 1737[1]. Initially considered benign, clinical evidence now shows that myocardial bridges can lead to several complications including ischemia, acute coronary syndrome, ventricular septal rupture, arrhythmias, exercise-induced atrioventricular conduction block, coronary spasm, and even sudden death. This is due to disrupted blood flow beneath myocardial bridges, leading to significant hemodynamic changes, particularly at the proximal end where arteries are highly prone to atherosclerosis. Myocardial bridges may initiate the development of atherosclerotic plaques or accelerate their progression in the proximal coronary artery segment, increasing the risk of acute coronary syndrome when atherosclerosis is superimposed on myocardial bridges. Therefore, there is a critical need for histological studies of coronary arteries to investigate the impact of blood flow dynamics on arterial walls beneath myocardial bridges, especially in hearts without documented cardiac disease history. This study aims to contribute to understanding the prevalence of atherosclerosis associated with myocardial bridges through histological examination.

MATERIAL & METHODS

The study was a cross-sectional observational study conducted from September 15 to August 16. It involved 50 adult human hearts of both sexes, sourced from the Department of Anatomy and Forensic Medicine at KGMU, U.P. Lucknow. All necessary permissions and approvals were obtained from the institutional ethical committee. Criteria for inclusion encompassed individuals aged 16 to 70 years who had died from both natural and unnatural causes. Exclusion criteria were applied to decomposed bodies and cases where death was attributed to documented cardiac causes, toxins, or a history of chest pain. After procurement, the hearts underwent dissection to identify and document the number and location of myocardial bridges (MBs). Subsequently, 78 tissue sections were sampled from the coronary arteries positioned proximal, below, and distal to the MBs. These tissue samples were processed using whole histological slide preparation techniques and analyzed under a microscope to assess the presence of atherosclerosis.

We categorized atherosclerotic changes in the arterial walls into four grades:

- Grade 0: No atherosclerotic changes observed.
- Grade I: Less than 25% of the arterial wall affected by atherosclerotic changes, characterized by minimal changes, fatty streaks, or early-stage preatheromas.
- Grade II: Atherosclerotic changes affecting 25% to 50% of the arterial wall, typically manifesting as atheromas or fibroatheromas.
- Grade III: More than 50% of the arterial wall affected by atherosclerotic changes, which may include hemorrhage, thrombosis, or calcification.

RESULTS

In the current study of 50 hearts, 26 hearts were found to have myocardial bridges (MBs), with 20 bridges in males and 6 in females. A total of 78 segments were evaluated, including proximal, below, and distal to the MBs. At the proximal segment, Grade I (Fig 1) and Grade II (Fig 2) atherosclerosis were observed at equal frequencies of 38.5%, while Grade I atherosclerosis was seen at 11.5% in the distal segment.

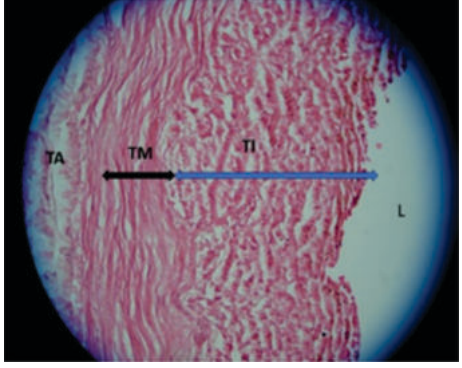

Figure 1: Photomicrograph of coronary artery proximal to myocardial

bridge (Transverse section) showing <25% (Grade I) atherosclerotic changes (H & E 100x) TI-tunica intima, TM-tunica media, TA- Tunica adventitia,L-Lumen

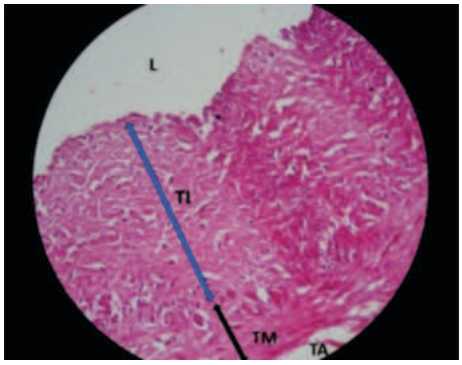

Figure 2: Photomicrograph of coronary artery at proximal to myocardial bridge (Transverse section) showing 25%-50% (Grade II) atherosclerotic changes (H & E 100x) TI-tunica intima, TM-tunica media, TA- Tunica adventitia, L-Lumen

Table -1 Distribution Of Atherosclerosis In Relation Of Myocardial Bridges At Their Site

	Grade 0	Grade I	GradeII	Grade III	Total	P value
Proximal	6 (23.1%)	10 (38.5%)	10 (38.5%)	0	26	p<0.001#
Below	26 (100%)	0	0	0	26	
Distal	23 (88.5%)	3 (11.5%)	0	0	26	
Total	55	13	10	0	78	
2=44.8 (df=4); p<0.001						
Chi-square test / #Fisher exact test used. P<0.05 significant						

The majority of atherosclerosis was concentrated at the proximal segment, where the prevalence and severity (grade) were significantly higher compared to the below and distal segments. None of the segments exhibited Grade III atherosclerosis (Table 1), and no atherosclerosis was observed below the MBs in any of the cadaveric hearts. This trend was consistent across both genders, with no significant gender-based differences observed in atherosclerosis grades at different locations (p>0.05). Evaluation of the association between atherosclerosis and MB location revealed a significant relationship (p<0.001).

DISCUSSION

The extent of atherosclerosis associated with myocardial bridges depends on several factors including their location, thickness, length, and degree of contractility. Angiographic assessments indicate myocardial bridges are present in 1.5% to 16% of cases, with autopsy studies showing rates as high as 80% [2]. Typically, the segment proximal to the bridge is more prone to developing atherosclerotic plaques, while the tunneled segment is often spared. This observation is supported by cellular and ultrastructural studies.

Coronary atherosclerosis related to myocardial bridging has been predominantly studied in the left anterior descending artery (LAD) [3,4]. Although the precise mechanisms are not fully understood, literature suggests several contributing factors including tensile stress (TS), shear stress (SS), blood flow dynamics, and coronary wall motion.TS, also known as circumferential stress, is influenced by blood pressure and affects the arterial wall uniformly. Regions with reduced blood pressure or lumen size experience lower TS, potentially creating an atheroprotective environment [5,6,7]. Conversely, the proximal segment of a myocardial bridge typically exhibits higher TS compared to the tunneled segment, which may lead to structural and functional changes in vascular cells (particularly endothelial and smooth muscle cells) and contribute to atherosclerotic plaque formation [8,9]. Shear stress (SS) represents a crucial factor in the local hemodynamic environment, influencing both the protection against atherosclerosis in mural segments and the propensity for plaque formation in proximal segments [10]. SS is defined as the frictional force exerted by blood on the endothelial surface [11], calculated as the product of the velocity gradient near the vessel wall and blood viscosity, suggesting it varies with flow [12,13]. Low shear stress likely contributes to the development of atherosclerotic plaques proximal to myocardial bridges, whereas high shear stress may have a protective effect within the tunneled segment. Another possible mechanism contributing to the resistance of myocardial bridges to atherosclerosis involves coronary motion. The external support provided by the surrounding myocardium may reduce coronary wall motion, which could play a protective role. As long as the epicardial coronary segments remain closely attached to the beating heart, they experience periodic motion throughout the cardiac cycle. This pulsatile coronary motion influences coronary geometry and thereby affects the local hemodynamic environment. This interaction initiates a self-perpetuating cycle that drives the progression of atherosclerosis [7,14].

In our study, atherosclerosis was absent below myocardial bridges (MBs), with the majority of atherosclerotic lesions located in the proximal segments. Therefore, myocardial bridges appear to be spared from atherosclerosis, likely due to the surrounding myocardium creating a unique hemodynamic microenvironment that protects against plaque formation. Disturbances in blood flow, turbulence, and lipid accumulation in the proximal part contribute to plaque formation in the tunica intima, leading to complications and potentially sudden death. Ge et al. [12] conducted an intracoronary ultrasound and Doppler study on 69 patients, revealing a high incidence of atherosclerosis proximal to the bridges but no plaque within or distal to them. Klues et al. [13] observed the highest systolic pressure within the tunneled segment in a study of 12 patients using coronary angiography, but found no pressure gradient across the bridge. Robicsek et al. [8] found no apparent atherosclerosis in the intramyocardial segments of 92.3% of patients in their coronary angiographic study on myocardial bridges. Heribert et al. [15] reported a case of left anterior descending coronary artery stenosis detected in a patient with extensive myocardial bridging distal to the lesion, suggesting turbulent blood flow and increased wall stress proximal to the myocardial bridge may contribute to such lesions. Sandra et al. [16] compared calcium scores of LAD myocardial bridges in cardiac CT studies of 40 subjects with and without bridges, finding fewer atherosclerotic plaques in bridged segments, consistent with our histological findings. Atherosclerosis was consistently absent below myocardial bridges in all cases examined. These findings align with those reported by Ge et al. [12] and Robicsek et al. [8].

CONCLUSION

Atherosclerotic plaque tends to develop preferentially in regions of the coronary arteries where there is turbulence or disturbed blood flow. Interestingly, coronary arteries below myocardial bridges are often spared from this plaque buildup. However, the segments of these arteries that are proximal to myocardial bridges are notably more prone to developing atherosclerosis. Therefore, it is crucial to identify these proximal segments as high-risk zones for atherosclerosis. By pinpointing these areas and implementing comprehensive medical, interventional, and surgical therapies aimed at secondary prevention, we can potentially extend the life expectancy and significantly improve the quality of life for patients diagnosed with clinical coronary artery disease, as well as for individuals who do not have a documented history of cardiac disease.

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Conflict of Interest

None

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