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CORONARY MYOCARDIAL BRIDGE, A LONG TERM FOLLOW UP STUDY

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ABSTRACT

Myocardial bridging is a congenital anomaly in which a segment of a coronary artery takes a "tunneled" intramuscular course under a "bridge" of overlying myocardium. This causes vessel compression in systole, resulting in hemodynamic changes that may be associated with angina, myocardial ischemia, acute coronary syndrome, left ventricular dysfunction, arrhythmias, and even sudden cardiac death. India is being laden with increasing incidences of coronary heart disease out from clinical presentation through a simplified investigation profile their morbidity may be addressed in a distinguished manner which has a less malignant course of progression. The present study is designed with an objective to identify the clinical spectrum of presentation of coronary myocardial bridge and to evaluate such cases with simple conventional noninvasive and invasive tests. All cases of angina attending the medical and cardiology OPD for the first time at the central hospital, S.E.C. Railway, Bilaspur (CG) between April 2011 to March 2016. After basic investigations all angina cases were evaluated by resting ECG and Echocardiography. Cases with normal resting Echo study were subjected to Tread Mill Test (TMT) with Bruce protocol. Total 1362 (female =354) cases of angina attended for first time in OPD. Out of which 271 (72%) cases had atherosclerotic coronary artery disease (CAD), 61(16%) cases were having CMB and 43 (12%) cases had normal coronaries. 54 patients out of 61 of CMB remained free of symptoms where as 29 out of 43 cases of CAD on medical management. First-line therapy involves medical treatment with beta-blockers and non-dihydropyridine calcium-channel blockers. From the present study, the researchers observed better results with beta-blockers added with nikorandil. A prospective randomized trial is required to identify the best treatment strategy for patients with myocardial bridging.

ORIGINAL RESEARCH ARTICLE

INTRODUCTION:

Since the initial angiographic imaging by Portsmann in 1960,[1] newer diagnostic modalities such as coronary computed tomographic angiography (CCTA),[2] intravascular ultrasound (IVUS),[3] intracoronary Doppler[4], and fractional flow reserve (FFR)[5] have enabled fuller analysis of

the anatomic and hemodynamic consequences of the systolic compression, including pathological effects on coronary flow.[6] Despite this increased understanding, treatment options remain limited. Medications such as beta-blockers and calcium-channel blockers remain first-line therapy, with surgical myotomy reserved for refractory cases. While percutaneous coronary intervention in the form of stenting has been used, serious complications such as stent fracture and coronary perforation have been reported. This manuscript summarizes our current understanding of the hemodynamic alterations in myocardial bridging, especially as it relates to the observed clinical sequelae, describes the anatomical characteristics on angiography and non-invasive imaging, and explores current treatment options including medical and invasive therapies.[7]

Coronary myocardial bridging (CMB) is a congenital variant of a coronary artery in which a portion of an epicardial coronary artery (most frequently the middle segment of the left anterior descending [LAD] artery)[7] takes an intramuscular course. This results in vessel compression during systole. While frequently asymptomatic, in many cases this condition may be responsible for adverse complications including angina, myocardial ischemia, acute coronary syndromes, left ventricular dysfunction and stunning, arrhythmias, and even sudden cardiac death.

The degree of myocardial ischemia and resulting symptoms appear on first glance to be out of proportion to the degree of compromise in coronary blood flow by myocardial bridging. As the majority of coronary filling occurs in diastole (with mean flow systolic to diastolic ratios measured in one study of 0.22 and 0.85 in the LAD and the RCA, respectively), systolic compression of the artery should have only a blunted impact on total effective myocardial perfusion.[8]

Reported rates of myocardial bridging differ based on the mode of evaluation. Numerous autopsy series have been performed, with rates reported from 5%–86%, with a mean of 25%. The largest study by Risse et al[9] involving 1056 patients found an intramyocardial coronary artery course in 26% of patients. These rates are much higher than angiographically reported bridging, which typically detects systolic compression at rates from 0.5%–12%.[10–12]

India is being laden with increasing incidences of coronary heart disease mostly in younger population due to faulty lifestyle. A subset of congenital coronary artery anomaly may also contribute to this morbidity which is usually under-considered. If such cases are fished out from clinical presentation through a simplified investigation profile their morbidity may be addressed in a distinguished manner which has a less malignant course of progression[13].

OBJECTIVES:

- To identify the clinical spectrum of presentation of coronary myocardial bridge
- To evaluate such cases with simple conventional noninvasive and invasive tests
- To follow up symptomatic CMB cases with optimum medical management

MATERIAL AND METHODS

All cases of angina attending the medical and cardiology OPD for the first time at the central hospital, S.E.C.Railway, Bilaspur(CG) between April 2011 to March 2016 were included. All the selected patients were informed about the procedure of the study and written consent was taken from all the patients. The inclusion and exclusion criteria were laid down:

Inclusion Criteria:

- Patients irrespective to their age and both gender were included in the study.
- Patients with or without hypertension or diabetes mellitus were included.

Exclusion Criteria:

- Patients who did not give consent for the study.
- Patients with a history of coronary artery (atherosclerotic) disease with or without a history of revascularization were excluded from this study.

After basic investigations, all angina cases were evaluated by resting ECG and Echocardiography. Cases with normal resting Echo study were subjected to Tread Mill Test (TMT) with Bruce protocol. Those with positive TMT were taken up for conventional (invasive) coronary angiography. The patients with CMB were taken up as cases for study.[14]

Patients with atherosclerotic coronary artery disease on medical management were taken as a control group. Both the cases and controls were followed up for long term (3 to 5 years).

RESULTS

Demographic Distribution of selected populations:

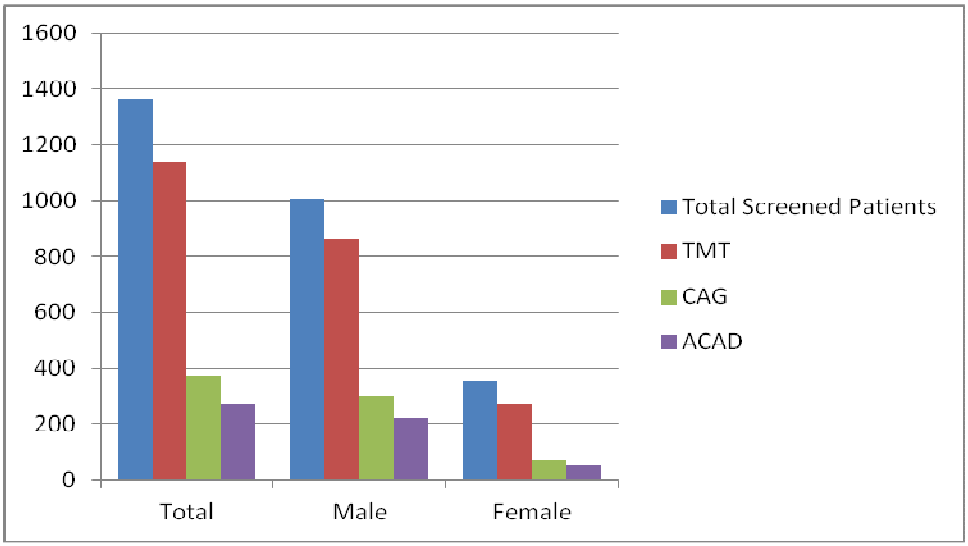

Fig 1: Genderwise distribution of patients selected for the study.

Total 1362 (female =354) cases of angina attended for the first time in OPD. Total cases subjected to TMT after basic evaluations were 1138 (female 273). Total cases found to be positive 404 (female 137). Out of these only 375 (female 71) cases were taken up for coronary angiography (CAG). Out of which 271 (72%) cases had atherosclerotic coronary artery disease (CAD), 61(16%) cases were having CMB and 43 (12%) cases had normal coronaries.

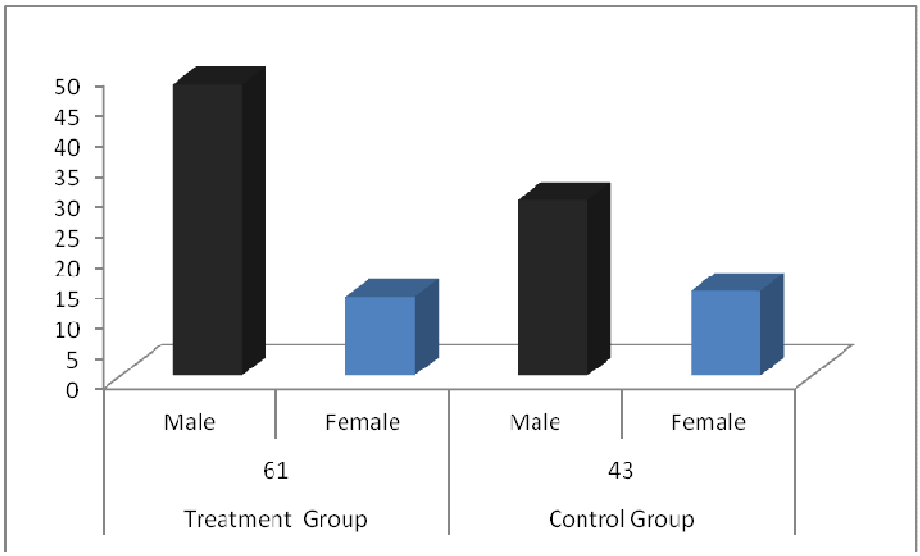

Fig 2: Patient distribution among the groups.

All 61 CMB cases (female13) were enrolled as study cases and 43 (female 14) out of 271 (16%) of CAD cases who were placed on medical management were taken as control. Ages in male ranged from29 to72 and in female 27 to 66 years. Follow up for subsequent 3 years showed better symptom-free intervals in CMB(cases) group with Beta blocker and Nicorandil than (control) group of CAD on optimum medical management.

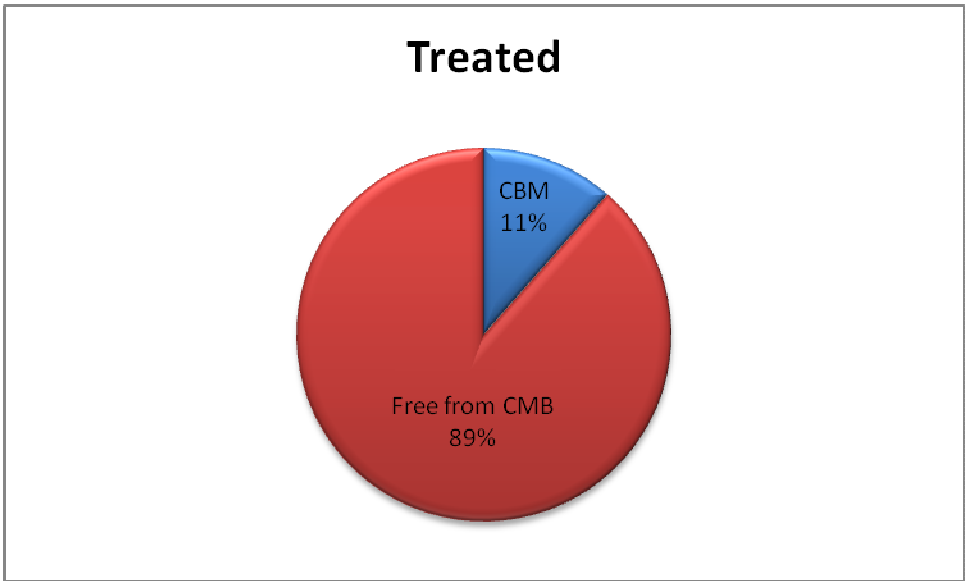

Fig 3: Percentage of patients recovering from the symptoms of CMB after treatment in the test group (n= 61)

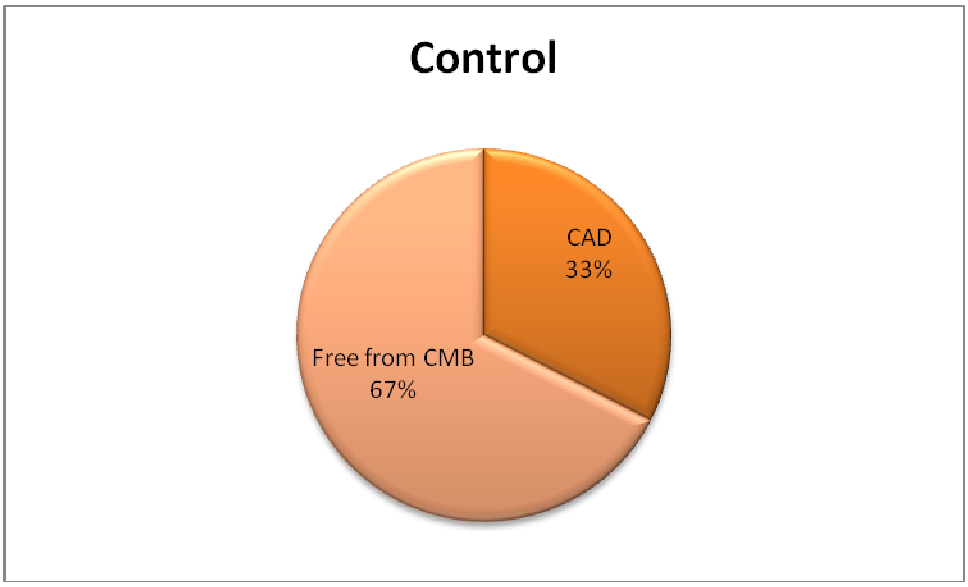

Fig 4: Percentage of patients recovering from the symptoms of CAD after clinical management in the control group (n= 43)

54 patients out of 61 of CMB remained free of symptoms whereas 29 out of 43 cases of CAD on medical management (p=0.05)

DISCUSSION

CMB is a congenital coronary artery anomaly up to a prevalence rate of 26% in patients with angina[9]. There is a paucity of studies that have established the natural history of the condition It may affect an individual at an early stage in the form of exertional angina (most common form). It can be confused with early atherosclerotic angina.

The proximal segment of the tunneled artery undergoes early atherosclerotic changes may play the role of a nidus for an acute obstructive coronarypathy[3]. If diagnosed early fatal outcomes can be considerably reduced by therapeutic interventions. During exertional tachycardia, the heart rate and increased force of contraction causing systolic compression of the tunneled artery causes impairment of distal blood supply. Since tachycardia cut shorts diastole so also major coronary feed in the LAD artery and it thus recycles the myocardial ischemia[15]**.** First-line therapy for such patients thought to be experiencing symptoms secondary to myocardial bridging consists of beta-blockers[16] and non-dihydropyridine calcium-channel blockers[17]**.** Another study has found the benefit of installing intra coronary Nicorandil by dilating the tunneled segment during both diastoles as well as systole. In our study Nicorandil 5mg twice daily have been found to be more effective in reducing symptoms especially when added with low dose long-acting beta blockers even after 3 years of following up[18].

CONCLUSIONS

Myocardial bridging is a congenital anomaly in which an epicardial coronary artery takes an intramyocardial course. Flow alterations from this condition can cause accelerated atherosclerosis in the coronary segment immediately proximal to the bridged segment. First-line therapy involves medical treatment with beta-blockers and non-dihydropyridine calcium-channel blockers. We have observed better results with beta-blockers added with nicorandil. A prospective randomized trial is required to identify the best treatment strategy for patients with myocardial bridging, with a larger subject size.

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