Myocardial bridging
Clinical and angiographic profile
in last 5 years; a study of 129 cases.

Sunita Viswanathan****, A George Koshy ***

**Aims of study** : To assess the clinical and angiographical profile of myocardial bridging from consecutive coronary angiograms done over last 5 years at Medical college, Thiruvananthapuram. To assess the risk of cardiovascular events and the risk of accelerated atherosclerosis in isolated myocardial bridging.

**Methods** : Consecutive coronary angiograms done at Medical college Thiruvananthapuram from 04/02/2005 to 31/03/2010 were reviewed for myocardial bridging. A total of 10492 coronary angiograms were reviewed. Myocardial bridges with systolic lumen reduction of more than 50% were considered for analysis. Quantitative coronary angiography (QCA) was used for analysis. Clinical presentation as well as correlation with structural heart disease and coronary heart disease was assessed.

**Results**: Incidence of myocardial bridges was 1.23%. Of the 129 patients with myocardial bridges 63 (48.8%) had associated significant coronary artery disease. Remaining 66 (51.2%) patients presented with isolated bridges. Out of these 66 patients with isolated myocardial bridges, 7 (10.6%) patients presented with acute myocardial infarction and 3 (4.5%) presented with cardiac arrhythmias. Of the 63 patients with significant coronary disease 11 (17.5%) patients had single vessel disease and they had the culprit lesion and myocardial bridge seen in the same vessel. Most common location of myocardial bridge was mid LAD (64.9%) followed by distal LAD (23.8%). Length of bridge was < 10 mm in 18.2%, between 11-20 mm in 46.2% and > 20 mm in 35.7%. Mean percentage of systolic obliteration by the bridge was 74.5%. 100% systolic obliteration was seen in 7.79% of isolated bridges and in 9.79% of bridges with CAD. Among 120 rhematic heart disease patients who underwent coronary angiogram, 10 (8.3%) patients had myocardial bridging. 12.1% of all HCM patients who underwent coronary angiograms had myocardial bridging.

**Conclusion**: Myocardial bridging can be lethal- can accelerate atherosclerosis, can precipitate acute MIs and life threatening arrhythmias.

**Introduction**
Myocardial bridging occurs when a band of cardiac muscle overlies an intramural segment of a coronary artery, the intramural segment being referred to as a “tunneled” artery. LAD is the vessel most commonly involved; however, diagonal branches are occasionally involved as well as the posterior descending right coronary artery or obtuse marginal branches of the circumflex artery. The clinical, hemodynamic, and prognostic significance of this entity has remained controversial, some investigators suggesting that it was a benign condition. Meanwhile there are also reports that patients with myocardial bridging are in fact at increased risk for clinical symptoms and cardiac events. However among patients with angiographically documented myocardial bridging, a significant percentage have concomitant atherosclerotic, muscular, or valvular heart disease, which independently affect clinical outcome as well as treatment strategy.

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Methods
Consecutive coronary angiograms done at Medical college Thiruvananthapuram from 04/02/2005 to 31/03/2010 were reviewed for myocardial bridging. A total of 10492 coronary angiograms were reviewed. Myocardial bridges with systolic lumen reduction of more than 50% were considered for analysis. Quantitative coronary angiography (QCA) was used for analysis. Clinical presentation as well as correlation with structural heart disease and coronary heart disease was assessed.

Statistical Analysis
Statistical analysis was carried out for 66 patients with isolated myocardial bridges and for 63 patients with myocardial bridge and coronary artery disease after categorizing each variable. Patient's age, sex, incidence of diabetes, hypertension, dyslipidemia and mean levels of total cholesterol, LDL and HDL were compared. Also incidence of myocardial bridging in rheumatic heart disease and hypertrophic cardiomyopathy & mean ejection fraction of isolated myocardial bridges were analyzed in relation to normal population undergoing coronary angiogram. Fisher test and Chi-square test with Yates correction were used for categorical variables and unpaired T test was used for continuous variables. A p value of less than 0.05 was considered statistically significant.

Observations
Among the 10492 angiogram reviewed, the prevalence of myocardial bridging was 1.23%. Total number of patients with myocardial bridging was 129 of which 66(51.2%) had isolated myocardial bridging and 63 (48.8%) had coronary artery disease in addition. Of the 66 patients with isolated myocardial bridges 7 patients (10.6%) presented with acute ST elevation myocardial infarction and 3 patients (4.5%) presented with cardiac arrhythmias. 11 patients (17.5%) in the 63 patients with myocardial bridging and coronary artery disease (CAD) had single coronary disease and myocardial bridge in the same vessel.

Mean age of presentation of isolated bridges (47.3+/–11 years) was significantly lower than that of bridge and CAD (55.2+/–10.8 years). P value was 0.0001. Myocardial bridging was more common among males. (Table 1).

Diabetes mellitus was seen in 10.6% of isolated bridges and 36.5% of bridges with CAD. (P value 0.0007). Mean cholesterol level (174+/–50.5 mg%) as well as mean LDL level (109.8+/–43.4 mg%) in isolated bridges was statistically lower compared to 209.6+/–53.6 mg% and 138.8+/–51.9 mg% in the bridges and CAD group. (P value 0.017 and 0.030 respectively). Incidence of hypertension, dyslipidemia and mean level of HDL cholesterol were comparable between the two groups. (Table 1)

Among the 120 rheumatic heart disease (RHD) patients who underwent coronary angiogram 10 patients (8.3%) had myocardial bridging. Of these 10 patients 6 patients had severe mitral stenosis, 3 had severe mitral stenosis and moderate MR, and 1 had severe MR and moderate MS. None had aortic involvement of rheumatic disease. Occurrence of myocardial bridging among rheumatic heart disease patients was significantly higher compared to general population (8.3% Vs 1.23 % P value 0.0001). All RHD patients with myocardial bridging had bridging in the LAD territory. Apart from these patients there was one patient with MVP and severe mitral regurgitation and another with severe calcific aortic stenosis; both associated with myocardial bridging.

66 patients with hypertrophic cardiomyopathy (HCM) underwent coronary angiography and 8 of them (12.1%) had myocardial bridging. This was also significantly higher compared to the prevalence in general population (P value 0.0001).

<table>
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<tr>
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<th>Isolated bridge (66 patients)</th>
<th>Bridge + CAD (63 patients)</th>
<th>P value</th>
<th>Overall</th>
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<tr>
<td>Mean age (years)</td>
<td>47.3+/–11.0</td>
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<tr>
<td>Female (%)</td>
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<td>DM (%)</td>
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<td>36.5</td>
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<td>DLP (%)</td>
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<td>LDL(mean mg%)</td>
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<td>0.0300</td>
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<td>47.9+/–20.5</td>
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Clinical profile table 1
Isolated bridges
Among the 66 patients with isolated bridges 49 patients (74.2%) presented with chest pain, 4 patients with palpitation and syncope (6.1%) and 2 patients (3.0%) with breathlessness. 11 patients (16.7%) underwent coronary angiogram as preoperative evaluation. 18 out of 49 patients with chest pain (36.7%) presented as unstable angina/NSTEMI. 11 patients (22.5%) presented as STEMI. Effort angina was seen in 10 (20.4%) patients and atypical chest pain in another 10 patients (20.4%). 16 patients with isolated bridges underwent TMT (Treadmill test) prior to coronary angiography. 12 patients (75%) were positive for inducible ischemia whereas 3 patients(18.8%) were TMT negative and one (6.2%) inconclusive.

Analysis of patients with isolated bridges (without STEMI) showed a mean ejection fraction of 72+-/10 % which was significantly higher than general population. (P value 0.0001). Even patients presented with STEMI had preserved mean EF. (60+-/15.2 %)

STEMI in isolated myocardial bridging.
7 patients (10.6%) presented with STEMI among isolated bridges and all presented with anterior wall myocardial infarction (AWMI). 6 patients presented with chest pain and were thrombolysed & one presented with LV dysfunction due to old AWMI. Two of them were smokers and one had diabetes mellitus and one had dyslipidemia.

Isolated myocardial bridging presenting as cardiac arrhythmia
4.5% (3 patients) presented with palpitation and syncope and had cardiac arrhythmias. One patient had recurrent ventricular tachycardia and had to be treated with amiodarone and sotalol. Another patient presented with transient complete heart block and T inversions in inferior leads. Coronary angiogram showed mid RCA myocardial bridging. Third patient presented with paroxysmal atrial fibrillation

Quantitative Coronary Angiography
There were a total of 151 myocardial bridges. Most common location of myocardial bridge was mid LAD (98 bridges, 64.9%), followed by distal LAD (36 bridges, 22.8%). Septals were involved in 4 patients (2.6%), diagonals in 2 patients (1.3%); RCA in 1 patient (0.66%), RPDA in 3 patients (2.0%) and RV branch in 1 patient (0.66%). Lcx showed myocardial bridging in 1 patient (0.66%) and OM branch showed bridges in 2 patients (1.3%). Of the 129 case with myocardial bridging 103 patients (79.8%) had right dominant coronary system, 15 patients (11.6%) had left dominance and 11 patients (8.5%) had co dominant system.

Mean length of the myocardial bridge was 24.45 mm in isolated myocardial bridges and 24.6 mm in bridges with CAD. Overall mean length was 24.53 mm. 18.2% of bridges were </=10 mm in length, 46.2% had length between11 to 20mm and 35.6% had length of bridge >20 mm.

Systolic lumen narrowing of more than 50% was the inclusion criterion. Mean severity of constriction among isolated bridges was 74.6%. Among 66 patients with isolated bridges 24.7% had systolic constriction >/=90% and 7.8% had 100% systolic constriction. Mean severity of systolic narrowing among 63 patients with bridge and CAD was 74.5%. 25.8% had >/=90% lumen obstruction and 12.1% had 100% lumen obstruction.

Single vessel CAD and bridge in the same vessel
11 patients in 63 patients with bridge and CAD (17.5%) had single vessel coronary disease and myocardial bridge in the same vessel. 9 patients (81.8%) had coronary atherosclerotic lesion proximal to myocardial bridge , one (9.1%) had lesion distal to bridge and the remaining one (9.1%) had lesion proximal as well as distal to bridge.

Drug treatment received for isolated bridges
Of the 66 patients with isolated bridges 45 patients(68.2%) received beta blockers and 2 patients (3.0%) received rate slowing calcium channel blockers (one received ditiazem and the other received verapamil). 1 patient (1.5%) received amiodarone. 7 patients (10.6%) received long acting nitrates and short acting nitrates were prescribed to 11 patients(16.7%)

Aspirin was given for 27 patients (40.9%), clopidogrel for 3 patients (4.5%), & aspirin plus clopidogrel was given for 17 patients (25.8%).19 patients (28.8%) received no antiplatelets

Discussion
Myocardial bridges can be an incidental finding at the time of coronary angiography. Conversely, a wide variety of clinical syndromes including unstable angina, acute myocardial infarction (AMI), life-threatening cardiac arrhythmias, and sudden cardiac death have been associated with myocardial bridging. In autopsy series, myocardial bridging was found in 5% to 86% of the cases. The incidence of myocardial bridging reported in angiographic series ranged from 0.5% to 2.5%. Of the 10492 angiograms reviewed in this study, 129 cases, (an incidence of 1.23%) had...
myocardial bridging with systolic lumen reduction more than 50%. This is in consistent with previous reports of myocardial bridging. 66 (51.2%) patients presented with isolated myocardial bridge whereas 63 (48.8%) patients had associated CAD. Bridges were more common in females which was also consistent with previous studies.

Myocardial bridging is seen with increased frequency in conditions like hypertrophic cardiomyopathy, left ventricular hypertrophy, and severe aortic stenosis. In this study there were 66 patients with hypertrophic cardiomyopathy (HCM) who underwent coronary angiography and 8 of them (12.1%) had myocardial bridging. This was also significantly higher compared to the incidence in general population. Even though the pathological series and MDCT series on myocardial bridging report an incidence of more than 50%, in almost all the angiographic studies; the incidence was found to be to be typically less than 5%. This underlies the fact that there are many bridges which may remain masked or have no clinically significant luminal narrowing.

There are reports that in stages of increased heart rate or increased contractility or increased LV wall tension some of these bridges can unmask and become clinically significant. This was demonstrated after administration of nitroprusside when angiographic incidence of myocardial bridging was found to be as high as 16%. Use of inotropes also increase the severity of myocardial bridges by increasing the contractility of heart. Another parameter which increases the severity of myocardial bridging was the systemic vascular resistance. Patients who had lower systemic blood pressure/resistance had higher incidence of myocardial bridging. An interesting finding noted in this study was the increased incidence of myocardial bridging in rheumatic heart disease. This was not previously reported in literature. Among the 120 rheumatic heart disease (RHD) patients who underwent coronary angiogram, 10 patients (8.3%) had myocardial bridging. Of these 10 patients; 6 patients had severe mitral stenosis, 3 had severe mitral stenosis and moderate MR, and 1 had severe MR and moderate MS. None had aortic involve ment of rheumatic disease.

Occurrence of myocardial bridging among rheumatic heart disease patients was significantly higher compared to general population and reasons for these findings are not clear. Most of the patients had severe heart disease and majority of the patients had advanced heart failure. A combination of higher heart rate due to heart failure, increased LV contractility and use of positive inotropic drugs like digoxin would have accounted for this finding. All RHD patients with myocardial bridging in this study had bridging in the LAD territory. Hence right ventricular hypertrophy secondary to pulmonary artery hypertension could not be attributed to myocardial bridging.

Analysis of patients with isolated bridges (without STEMI) showed a mean ejection fraction of 72%±10% which was significantly higher than general population. Even patients presented with STEMI had preserved mean EF. (60%±15.2%). This finding is probably due to the fact that all these patients had either thrombosis or spasm of the coronary which responded well to medical therapy including thrombolysis. Unlike atherosclerotic plaque, these lesions produced only transient occlusion of the coronary and myocardial loss was significantly lower. But indeed; there was one patient who presented with LV dysfunction with an EF of 40%.

Patients with isolated myocardial bridging without STEMI had significantly higher EF than the general population. Whether this increased contractility is the etiology for increased myocardial bridging in this subset of patients is not clear. If this data is extrapolated into valvular heart disease or left ventricular hypertrophy or even to any condition with increased contractility, whenever the patient develops a coronary event or cardiac arrhythmia; myocardial bridging should also be considered among the possible etiologies. Some of the previous reports suggested a correlation with length of the bridge and clinical presentation where as others report no such relation with length or severity of bridge and clinical events. In this study no relation could be found between severity of systolic narrowing or length of bridge and cardiac events.

Of the 129 case with myocardial bridging 103 patients (79.8%) had right dominant coronary system. There are pathological reports that myocardial bridges are more common with a left coronary dominant system. However there was no such relation observed in the current study. 11 patients (17.5%) in 63 patients with bridge and CAD had single vessel coronary disease and myocardial bridge in the same vessel. 9 patients (81.8%) had coronary atherosclerotic lesion proximal to myocardial bridge, one (9.1%) had lesion distal to bridge and the remaining one (9.1%) had lesion proximal as well as distal to bridge. There are multiple reports of accelerated atherosclerosis in segments of coronary
artery proximal and distal to a myocardial bridge and a surprising absence of the same in the segment underlying the bridge. This may also be related to the compression of the tunneled segment during systole, enhancing the lymph drainage of the vessel wall that is important for the prevention of lipid accumulation and disease development.

Current study which included 129 cases is one of the largest ever reported data in literature on myocardial bridging. Previous largest series reported from India was by Harikrishnan et al28 which included 21 cases.

Even though considered as a relatively benign condition, 10.6% patients in this study with isolated bridges presented with acute myocardial infarction. Of the 66 patients with isolated myocardial bridges; presentation of 4.5% of patients with cardiac arrhythmias—one had ventricular tachycardia and another had complete heart block—further underlines the importance of this anatomic variant. Whether such patients should undergo PCI is a matter of debate. Of the 63 patients with significant coronary disease, 17.5% patients had single vessel disease and they had the culprit lesion and myocardial bridge seen in the same vessel. Whether myocardial bridging predisposed for coronary disease or not is difficult to prove. Another interesting observation noted in this study was the increased incidence of myocardial bridges in rheumatic heart disease especially mitral valve disease. This is not reported previously in literature and the explanation for this occurrence is not clear.

**Conclusions**

Myocardial bridging can be lethal—can accelerate atherosclerosis, can precipitate acute myocardial infarctions and life threatening arrhythmias. Myocardial bridging must be considered especially in patients at low risk for coronary atherosclerosis but with angina-like chest pain or established myocardial ischemia. Medical management is the main stay and requires more awareness. PCI/Surgical management is still a matter of debate. It may be considered if there are recurrent events despite optimal medical therapy. HCM patients have increased incidence of myocardial bridging. RHD patients have increased incidence of myocardial bridging. TMT positivity is very frequent in myocardial bridging.