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Smoking and involvement of right coronary artery in ischemic heart disease

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Abstract: *Introduction:* Smoking is an independent risk factor for ischemic heart disease and acute myocardial infarction. Smoking raise both heart rate and blood pressure, thus increasing myocardial oxygen demand, moreover it also decreases the dimension of coronary vessel and coronary blood flow. Inferior wall Myocardial Infarction is consequence of disease in usually Right coronary artery, whereas anterior wall Myocardial Infarction is usually disease in left coronary artery. The aim of the study is to evaluate whether smoking influence the incidence of inferior wall MI (Right coronary artery). *Objective:* To find out whether there is an association between smoking and inferior wall Myocardial Infarction and an early association of atherosclerosis and ischemic heart disease with smoking. *Methods:* 126 patients of STElevation Myocardial Infarction admitted from the outdoor patient department/ emergency department/ Cardiology OPD in MMIMSR, Mullana, Ambala, considered for study. Those who are willing to participate and fulfilling the inclusion and exclusion criteria. *Result:* In our study there was a high proportion of smoker in patient with inferior wall MI than other location of MI. Smokers were prone to get myocardial infarction at a younger age as compared to others. Mortality was higher in anterior wall MI as compared to Inferior wall MI. Anterior wall MI presented with more complications i.e. cardiogenic shock and arrhythmias. *Conclusion:* Smoking enhance the risk of inferior wall MI more than other MI. Smoking thus appear to adversely affect the Right coronary artery to greater extent than left coronary arterial circulation by mechanism yet to be explored. Smoking leads to ischemic heart disease at early age.

Keywords: Smoking, Inferior Wall Myocardial Infarction, Right Coronary Artery.

Introduction

Myocardial infarction is the major cause of mortality and morbidity from coronary artery disease. In various developed countries it accounts for 10-25% of all deaths. In approximately 50% of patients the condition is lethal, and many of the survivors suffer from impaired cardiac function. Myocardial infarction occurs when blood supply to a part of the heart is occluded causing some heart cells to die. This is most commonly due to occlusion of coronary arteries either by an acute thrombus or with an atherosclerotic plaque which is caused by chronic inflammatory response in the walls of arteries, caused largely by the accumulation of macrophage white blood cells and promoted by low-density lipoproteins (plasma proteins that carry cholesterol and triglycerides). The classic symptoms include sudden severe chest pain or compression (typically radiating to the left arm), shortness of breath, nausea, vomiting, palpitations and sweating.

Smoking is an independent risk factor for ischemic heart disease and acute myocardial infarction [1]. How the smoking effect on coronary artery circulation is complex and multifactorial. Smoking raise both heart rate and blood pressure, thus increasing myocardial oxygen demand, moreover it also decreases the dimension of coronary vessel and coronary blood flow [2]. Whether nicotine smoking causing coronary vasoconstriction

and decrease blood flow, affect Right and left coronary arterial system equally and whether smoking constrict normal and diseased arteries to a similar degree uncertain [3]. Inferior wall MI is consequence of disease in usually Right coronary artery, whereas anterior wall MI is usually disease in left coronary artery.

There is consistent difference in smoking prevalence by infarct location or culprit artery and supports a hypothesis that smoking may adversely affect the right coronary arterial circulation to a greater extent than the left coronary circulation. However, the mechanism(s) responsible for this so evident selective effect of smoking in ischemic heart disease remain a puzzle. The aim of the study is to evaluate whether smoking influence the inferior wall Myocardial Infarction (Right coronary artery).

Material and Methods

The study was done at MMIMSR, Ambala which is a tertiary level rural hospital. The study was an Institution based observational cross-sectional study. This study involved Department of General Medicine and Cardiology Unit for patient's selection.

Sample Design: We examined 126 patients from our institution, all of them undergone ECG, Cardiac biomarkers, 2D ECHO and coronary angiography who fulfilled the exclusion criteria. They were diagnosed as ST elevation Myocardial Infarction by clinical and relevant investigations, (Electrocardiogram, Biochemical, selected as cases).

Inclusion Criteria: Patient age more than 18 years.

Exclusion Criteria:

1. Previous history of MI
2. Patient who had associated Cardiovascular disease i.e. IHD, RHD, CHD, Systemic arterial Hypertension.
3. Cerebrovascular accidents
4. Renal and renovascular disease.
5. Endocrine disorders (Type 2 Diabetes Mellitus, Hypothyroidism)
6. Respiratory Diseases (COPD/ ILD/ PAH)
7. Anemia and other nutritional disorders.

Data Collection:

Electrocardiography and Echocardiography: Electrocardiography and Transthoracic Echocardiography using (PHILIPS ECHO) were done in all the patients at the time of hospitalization and at the time of discharge. Regional wall motion abnormalities and Ejection Fraction was noted by a qualified Cardiologist.

Smoking: Patients were classified as non-smoker if they responded that they had smoked fewer than 100 cigarettes or 5 packs of cigarettes during their lifetime.

Cardiac Injury Markers: To confirm the diagnosis of AMI, cardiac injury markers were sent in form of troponin T/I (trop T/I kit) and CPK-MB.

Results

In our study, the mean age of patient with smoking history who presented with acute myocardial infarction was 53.6 ± 7.2 years as compared to 64.2 ± 9.0 years among non-smokers ($p < 0.001$). Every patient in this study underwent conventional coronary angiography and infarct related culprit artery was identified. In 78% of the patients with inferior wall MI right coronary artery was the culprit, while the left anterior descending artery was the culprit in over 96% of the patients with anterior wall MI (Table 1).

Table-1: Culprit artery in relation to location of the acute MI

Total -126 Patients	Inferior wall MI (N=74)	Anterior wall MI (N=52)
RCA	58 (78%)	0
LCX	12 (16%)	2 (4%)
LAD	2 (3%)	50 (96%)
RAMUS	2 (3%)	0

Table-2: Smoking status in relation to location of acute MI

	Smokers (N= 88)	Non Smokers (N=38)
Inferior Wall MI (N-74)	60 (68%)	14 (36%)
Anterior Wall MI (N-52)	28 (32%)	24 (64%)
P Value – 0.0021		

In 68% of smokers localization of MI was in inferior wall and while 36% only nonsmokers had inferior wall MI (Table 2).

80% of patients less than 50 years with no other predisposing cardiovascular risk factor had inferior wall MI, while 49% of patients with age > 50 years had inferior wall MI (Table 3). In age less than 50 years 90% had risk factor of smoking which predisposed them to early atherogenesis and myocardial infarction (Table 4). In our study mortality was 10% in inferior wall MI while anterior wall MI had mortality of 18%. Cardiogenic shock and rhythm disturbance was observed more in anterior wall MI. In inferior wall Myocardial infarction 8 patients required temporary pacing in view of AV dissociation and bradycardia as a rescue while doing revascularization. Intra-aortic ballon pump was used in 6 patients with anterior wall MI and 2 patients with Inferior wall MI who presented with refractory cardiogenic shock.

Table-3: Location of acute mi in relation to age

	Inferior Wall MI (N-74)	Anterior Wall MI (N-52)
Age < 50 Years (N-40)	32 (80%)	8 (20%)
Age > 50 Years (N- 86)	42 (49%)	44 (51%)
P Value – 0.0019		

Table-4: Smoking as predisposing factor of acute mi in relation to age

	Smokers (N=88)	Non Smokers (N=88)
Age < 50 Years (N-40)	36 (90%)	4 (10%)
Age > 50 Years (N-86)	52 (60%)	34(40%)
P Value < 0.001		

Discussion

The purpose of the study is to establish the association of smoking and inferior wall MI. Smoking adversely affect the right coronary circulation than the left. However the mechanism responsible for this selective effect of smoking in inferior wall MI is not clear. In our study 68% of smokers localization of MI was in inferior wall and while 36% only nonsmokers had inferior wall MI. In other study groups [4-8] smoking ranged from 41.9% to 84.7% among patients with inferior MI and from 36.4% to 74.4% among patients with anterior wall MI (Table 5).

Table-5: Smokers (%) among inferior and anterior AMI patients in the five cohorts [4-8]

Reference	Inferior AMI (%)	Anterior AMI (%)	Odds Ratio (95% CI)
(4)	67.3	50.7	2.00 (1.08-3.70)
(5)	84.7	74.4	1.90 (1.30-2.78)
(6)	43.2	36.5	1.32 (1.03-1.73)
(7)	41.9	36.4	1.26 (0.92-1.74)
(8)	47.5	44.1	1.15 (0.85-1.57)
Pooled	54.85	46.86	1.38 (1.20-1.58)

AMI: acute myocardial infarction; *CI:* confidence interval

The data reflect higher incidence of inferior wall MI in smokers as compared to other location of MI irrespective of age. Contrary to this, non-smokers presented with higher incidence of anterior wall MI. Similar results have been observed in previous studies involving AMI patients [9-11]. Nicotine smoking increase the risk of inferior wall MI by multiple and complex mechanism. Smoking increase the process of atherogenesis and raise the serum LDL cholesterol and triglyceride level and lower serum HDL cholesterol level. It also promote free radical damage, smoking also attributes to vascular inflammation to coronary arterial vessels, which is characteristic of atherosclerosis [1].

Whether this atherogenic process is more prominent in Right coronary artery is uncertain. Nicotine smoking activate the sympathetic system which enhance the heart rate which eventually increases the

myocardial oxygen demand and it also leads to coronary vasoconstriction. Smoking increases the carboxyhaemoglobin level in blood, it also further reduce myocardial oxygen delivery to myocardial tissue. Smoking alters endothelial function, inhibiting release of tissue plasminogen activator and prostacycline which result in local hypercoaguable state. Smoking also contribute to hypercoagulability to raise tissue factor, factor VII and fibrinogen. Smoking also increase the platlet activity and interaction between platlet and endothelial cells. Whether any of these adverse endothelial and prothrombotic effect will occur to a greater extent in the Right than in left coronary circulation is matter of speculation. Smoking also reduce the nitric oxide which leads to reduce coronary flow reserve.

Conclusion

In summary, we found that cigarette smoking is associated more strongly with inferior than anterior AMI. This finding suggests that the adverse effects of tobacco oncoronary atherogenesis and/or endothelial dysfunction maybe more pronounced in the right than the left coronaryarterial circulation. This study highlights the risk of smoking in pathogenesis of both anterior wall and inferior wall MI and predisposes smokers at higher chances of ischemic heart disease at younger age group. Emphasis should be laid on cessation of smoking for primary and secondary prevention of acute myocardial infarction [12].

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