

Angiographic Profile of Acute Coronary Syndrome in Smokers and Non Smokers: A Comparative Study

Goutam Kumar¹, Pawan Kumar Singh², Ravi Vishnu Prasad³, B.P. Singh⁴, Nirav Kumar⁵, Chandra Bhanu Chandan⁶

¹Assistant Professor, Department of Cardiology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India.

²Assistant Professor, Department of Cardiology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India.

³Additional Professor, Department of Cardiology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India.

⁴Professor, Department of Cardiology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India.

⁵Additional Professor, Department of Cardiology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India.

⁶Assistant Professor, Department of Cardiology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India.

Received: 21-02-2022 / Revised: 05-03-2022 / Accepted: 25-03-2022

Corresponding author: Dr. Pawan Kumar Singh

Conflict of interest: Nil

Abstract

Aim: To study the angiographic profile of Acute Coronary Syndrome in smokers versus non-smokers and to analyses with respect to baseline characteristics.

Material & Methods: This study comprised of a total of 200 cases. Study participants were allocated to two groups: Smoker's Group and Non Smoker's Group. Each group comprised of 100 participants. Mean age of the participants was 39:67 years. It was conducted in the Department of Cardiology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India, over a period of one year.

Results: In the smokers group, Acute coronary syndrome was more frequently encountered in males (71% versus 29%, $p < 0.001$) Non-smokers group, Acute coronary syndrome was often encountered in females (69% versus 31%).

Conclusion: (1) Acute coronary syndrome occurred seven years earlier in smokers (2) Smokers presented with more acute infarctions and less Unstable angina (3) Single vessel disease was the most common CAG diagnosis in both smokers and non-smokers.

Keywords: STEMI ST elevation myocardial infarction, unstable angina, myocardial infarction, cardiovascular disease.

This is an Open Access article that uses a fund-ing model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Cigarette smoking is a well-established risk factor for the development of coronary heart disease [1]. Several studies have

shown lower mortality after ST-segment elevation myocardial infarction in smokers compared with non-smokers [2–4]. This is

the smokers' paradox, where an individual who smokes is not only more likely than a non-smoker to suffer a heart attack but is also more likely to survive it. One explanation for this paradox is that smokers may have a larger thrombus burden, leading to a greater efficacy of intravenous thrombolysis [3–7].

The “Smoker's paradox”, typically described in studies where patients who were smoker and had acute ST-segment elevation MI (STEMI) treated with or without thrombolytic therapy [8] has been carried forward today with the primary percutaneous coronary intervention (PCI) also. [9] Young age and associated lesser co-morbidities in smokers were the proposed explanations for it. With the advent of primary PCI for acute STEMI, higher thrombus burden with less severe underlying coronary artery stenosis along with minimal or diseased free non-infarct related coronary arteries have also been added in the list to explain this “paradox”. [10]

According to World Health Organization (WHO), tobacco use has caused 100 million deaths in the 20th century worldwide, and if the current trend continues this figure will be 1 billion deaths for the 21st century. [11] Hence the aim of this study was to evaluate the angiographic profile of Acute Coronary Syndrome in smokers versus nonsmokers and to analyses with respect to baseline characteristics.

Material & Methods:

This study comprised of a total of 200 cases. Study participants were allocated to two groups: Smoker's Group and Non Smoker's Group. Each group comprised of 100 participants. Mean age of the participants was 39:67 years. It was conducted in the Department of Cardiology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India, over a period of one year.

Inclusion Criteria:

1. Patients with first episode of ACS (Unstable Angina with ECG changes, NSTEMI, STEMI) attending the ICU, Department of Cardiology, Igims, Patna, Bihar
2. Different types of ACS were diagnosed according to ECG criteria and Troponin T positivity
3. Both Smokers and non Smokers were included in the study group. Smoking history (all forms-tobacco chewing / khaini / beedi / tambaku / cigars).
4. Troponin positivity (qualitative) for NSTEMI differentiating from USA with ECG changes.
5. Coronary angiogram was done after 5 days of Heparin therapy in acute coronary syndrome.

Exclusion Criteria:

1. Age <18yrs/>80yr
2. Patients with recurrent episode of ACS and Cardiogenic shock
3. Patients with Valvular heart diseases/cardiomyopathies/pericardial diseases/conduction diseases other than ischemia
4. Contradictions to CAG (pts with serum creatinine >1.5mg/dl, lack of consent for CAG) and prior coronary angiogram report.
5. Unstable Angina without ECG changes. Disease (TVD), Left main coronary artery (LMCA) involvement, Proximal LAD involvement.

Statistical Analyses:

The observations were recorded in a proforma created for this purpose and entered in the master chart. Statistical Analysis was done using SPSS software. Mean, median, standard deviation and Chi Squares were calculated wherever applicable. A value of $p < 0.05$ was considered statistically significant.

Results:

This study comprised of a total of 200 cases. Study participants were allocated to two groups: Smoker’s Group and Non Smoker’s Group. Each group comprised of 100 participants. Mean age of the participants was 39:67 years.

In the smokers group, Acute coronary syndrome was more frequently encountered in males (71% versus 29%, $p < 0.05$) Nonsmokers group, Acute coronary syndrome was often encountered in females (69% versus 31%). [Figure 1]

Out of 100 study subjects in the smokers group, Diabetes mellitus was present in 33% subjects and in the smokers group (57%) ($P < 0.05$). Hypertension was commonly encountered in both study groups (52% versus 59%). Smokers group, Overweight/Obesity was present in 70% subjects. Nonsmokers group, Overweight/Obesity was present in 30% subjects. Acute myocardial infarction (STEMI+NSTEMI) was the most common presentation in both smokers (89%) and nonsmokers (81%)

($p < 0.05$) Unstable angina was often encountered as initial presentation in Nonsmokers (35% versus 15%, $p < 0.05$). Single vessel disease was the most common CAG diagnosis in both the smokers and nonsmokers (45% versus 48%). Double vessel disease, even though not statistically significant, was more commonly encountered in smokers (25% versus 14%, $p > 0.05$, Not significant). The smokers group, LMCA stenosis (>30% diameter stenosis) was present in 9% subjects and in the nonsmoker group, LMCA stenosis was present in 11% subjects. No statistically significance difference. [Table 1]

The mean EF was $54.2 \pm 10.2\%$. The median EF was 56.4%. 50% patients presented with Anterior wall STEMI which was the most common presentation of ACS in Smokers followed by inferior wall MI 39%. Only 3% patients were presented with high lateral / true posterior wall STEMI.

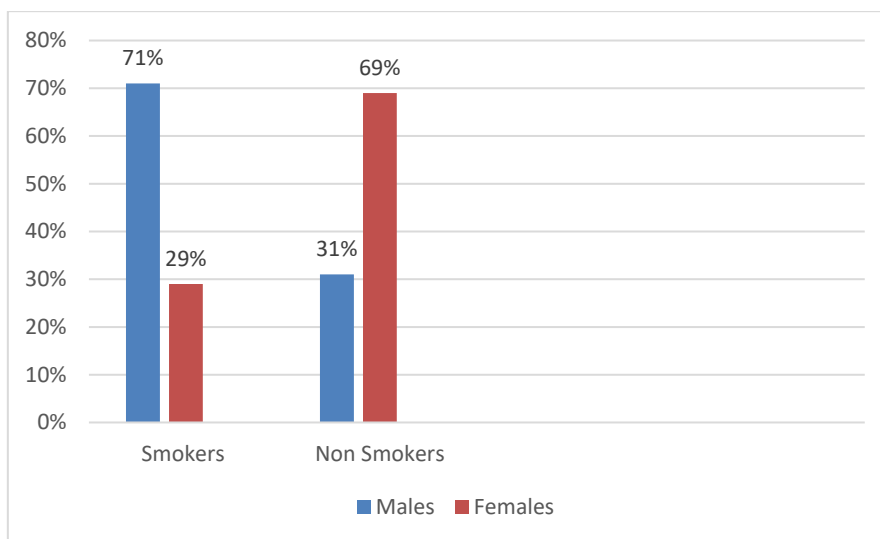


Figure 1: distribution of acute coronary syndrome among study participants

Table 1: Comparison of smoker’s and nonsmoker’s group with various variables

Variables	Smoker’s Group	Non Smoker’s Group	P value
Diabetes Mellitus	33	57	0.05*
Hypertension	52	59	0.271
Obesity	70	30	0.233

Acute myocardial infarction	89	81	0.05*
Unstable Angina	15	35	0.05*
Single Vessel Disease	45	48	0.738
Double Vessel Disease	25	14	0.374
LMCA Stenosis	9	11	0.111

Discussion:

Significantly less prevalence of hypertension and DM in our study among smokers was in accordance with most of the studies and this can be because of younger mean age of the smokers. Anterior wall MI with Left anterior descending (LAD) coronary artery as culprit vessel was the most frequent presentation in both the groups. However, in few studies inferior wall MI was the more frequent presentation in smokers. [12]

LAD coronary artery was also the most common culprit vessel in the previous data from EUROTRANSFER Registry. [13] Another important aspect regarding the finding of a higher incidence of thrombosis rather than atherosclerosis in smokers, was also evaluated in our study. Both the groups were profiled with coronary angiography at the time of STEMI. It was seen that the smokers group had higher thrombotic burden which, however, was not statistically significant. Besides this, thrombo suction, which is an indirect marker of higher thrombus burden was used more frequently in our study in smokers group. Higher use of thrombo- suction in smokers in our study is in contrast to previous data from EUROTRANSFER Registry. [14]

A consistent finding in the GRACE population is the lower risk profile of current smokers compared with former smokers and non-smokers. This was also observed in most previous large-scale studies [15], in which the lower risk profile accounted for much of the seemingly improved outcome of smokers compared with former smokers and non-smokers. However, the clinical profile of former smokers does not appear to be

simply intermediate between those of current smokers and non-smokers. As already noted in the Platelet glycoprotein IIb/IIIa in unstable angina: Receptor Suppression Using Integrilin Therapy (PURSUIT) trial [16], former smokers were more likely to have had prior cardiac events than current smokers and non-smokers. This difference may be related to the encouragement of smokers to quit smoking after a first cardiac event.

In coronary angiogram, single vessel disease was seen in 42% of patients and multi vessel disease in 37% of patients. Recanalised coronary arteries with no significant flow limiting disease were seen in 21% of patients. Multi vessel disease was seen in patients with diabetes or newly detected diabetics. Multi vessel disease was higher in patients with LV dysfunction (68%) whereas recanalised coronaries after thrombolytic therapy was 9 %.

Overall around 94% of patients were prescribed aspirin during hospitalization, which is high in comparison with previous studies involving patients with myocardial infarction [17-18]. However, a lower percentage of former smokers and non-smokers were given aspirin compared with current smokers. Statin therapy was prescribed in around half of the patients. [19] Again, former smokers and nonsmokers were less likely to receive statin therapy than current smokers. The age and the potentially high prevalence of comorbidities encountered in former smokers and non-smokers may explain why these patients were less likely to receive evidence-based therapies than current smokers.

Conclusion:

Acute coronary syndrome occurred seven years earlier in smokers. Smokers presented with more acute infarctions and less unstable angina. Single vessel disease was the most common CAG diagnosis in both smokers and nonsmokers.

References:

1. U.S. Department of Health and Human Services. Smoking and health in the americas. Atlanta, Georgia: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health 1992:1–211.
2. Barbash GI, Reiner J, White HD, et al. Evaluation of paradoxical beneficial effects of smoking in patients receiving thrombolytic therapy for acute myocardial infarction: mechanism of the smoker's paradox from the gusto-i trial, with angiographic insights. Global utilization of streptokinase and tissue-plasminogen activator for occluded coronary arteries. *J Am Coll Cardiol* 1995;26:1222–9.
3. Grines CL, Topol EJ, O'Neill WW, et al. Effect of cigarette smoking on outcome after thrombolytic therapy for myocardial infarction. *Circulation* 1995;91:298–303.
4. Zahger D, Cercek B, Cannon CP, et al. How do smokers differ from nonsmokers in their response to thrombolysis? (the timi-4 trial). *Am J Cardiol* 1995;75:232–6.
5. Lundergan CF, Reiner JS, McCarthy WF, et al. Clinical predictors of early infarct-related artery patency following thrombolytic therapy: importance of body weight, smoking history, infarct-related artery and choice of thrombolytic regimen: the gusto-i experience. Global utilization of streptokinase and t-pa for occluded coronary arteries. *J Am Coll Cardiol* 1998;32:641–7.
6. de Chillou C, Riff P, Sadoul N, et al. Influence of cigarette smoking on rate of reopening of the infarct-related coronary artery after myocardial infarction: a multivariate analysis. *J Am Coll Cardiol* 1996;27:1662–8.
7. Hasdai D, Rihal CS, Lerman A, Grill DE, Holmes Jr DR. Smokers undergoing percutaneous coronary revascularization present with fewer narrowings in the target coronary artery. *Am J Cardiol* 1997;80:1212–4.
8. Gourlay SG, Rundle C, Barron HV, et al. Smoking and mortality following acute myocardial infarction: results from national registry of myocardial infarction 2 (NRM12). *Nicotine Tob Res.* 2002;4:101–107.
9. Weisz G, Cox DA, Gracia E, et al. Impact of smoking on status on outcomes of primary coronary intervention for acute myocardial infarction- the smoker's paradox revisited. *Am Heart J.* 2005;150(2):358–364.
10. Rakowski T, Siudak Z, Dziewierz A, et al. Impact of smoking status on outcome in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. *J Thromb Thrombolysis.* 2012;34:397–403.
11. World Health Organization WHO report on the global tobacco epidemic, 2008 The MPOWER package Tobacco Free Initiative. (http://www.who.int/tobacco/mpower/gtcr_download/en/).
12. Weisz G, Cox DA, Gracia E, et al. Impact of smoking on status on outcomes of primary coronary intervention for acute myocardial infarction- the smoker's paradox revisited. *Am Heart J.* 2005;150(2):358–364.
13. Grines CL, Topol EJ, O'Neill WW, et al. Effect of cigarette smoking on outcome after thrombolytic therapy after myocardial infarction. *Circulation.* 1995;91:298–303.

14. Rakowski T, Siudak Z, Dziewierz A, et al. Impact of smoking status on outcome in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. *J Thromb Thrombolysis*. 2012;34:397–403.
15. Hansen EF, Andersen LT, Von Eyben FE. Cigarette smoking and age at first acute myocardial infarction, and influence of gender and extent of smoking. *Am J Cardiol* 1993;71:1439–42.
16. Hasdai D, Holmes Jr DR, Criger DA, et al. Cigarette smoking status and outcome among patients with acute coronary syndromes without persistent st-segment elevation: effect of inhibition of platelet glycoprotein iib/iiia with eptifibatide. The pursuit trial investigators. *Am Heart J* 2000;139:454–60.
17. Khan, A. ., & Tidman, D. M. M. . . (2022). Causes of Medication Error in Nursing . *Journal of Medical Research and Health Sciences*, 5(1), 1753–1764.
18. Mehta RH, Montoye CK, Gallogly M, et al. Improving quality of care for acute myocardial infarction: the guidelines applied in practice (gap) initiative. *JAMA* 2002;287:1269 –76.
19. Allison JJ, Kiefe CI, Weissman NW, et al. Relationship of hospital teaching status with quality of care and mortality for medicare patients with acute mi. *JAMA* 2000;284:1256 –62.