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ANGIOGRAPHIC CHARACTERISTICS AND PATTERN OF ATHEROSCLEROTIC LESION AMONG SMOKERS PRESENTING WITH ISCHEMIC HEART DISEASE

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Abstract

Objectives: *To determine the angiographic characteristics and pattern of atherosclerotic lesion in smokers presenting with ischemic heart disease.*

Methodology: *This cross-sectional study was conducted at Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Karachi. Six months January to June, 2025 following ethical approval from CPSP. We enrolled 237 adult patients (aged 18–65 years) with a smoking history presenting with IHD. Patients underwent coronary angiography during hospital admission. Clinical, demographic, and angiographic data (culprit artery, CAD type, TIMI flow) were collected. Smokers were categorized as current or ex-smokers.*

Results: *Most patients were male (74.3%), urban residents (59.6%), with a mean age of 47.82±10.09 years. Current smokers constituted 68.4%. LAD was the most common culprit artery (51.5%), followed by RCA (30.4%) and LCx (11.1%). SVD was present in 41.5%, DVD in 28.1%, and TVD in 30.4%. Significant CAD was observed in 87.7%. TIMI grade 0 flow pre-intervention was present in 52.6%, while post-intervention TIMI grade 3 flow was achieved in 95.3%. No statistically significant differences were observed between current and ex-smokers regarding angiographic severity or lesion pattern ($p > 0.05$).*

Conclusion: *Smokers presenting with IHD predominantly exhibit LAD involvement and single-vessel disease. Angiographic patterns did not differ significantly between current and ex-smokers, emphasizing the persistent vascular impact of tobacco exposure and highlighting the need for aggressive smoking cessation and cardiovascular risk management*

INTRODUCTION

In developing countries, cardiovascular diseases (CVDs) are on the rise and represent a major cause of mortality.[1] Approximately two-thirds of the risk for acute myocardial infarction can be attributed to smoking and lipid abnormalities.[2] Smoking influences every stage of atherosclerosis, beginning with endothelial dysfunction and extending to a prothrombotic state. The key mechanisms driving the initiation and progression of atherosclerosis—vasomotor dysfunction, inflammation, and lipid oxidation—emerge even before any structural or pathological changes become evident.[3-7] Acute coronary events are more commonly linked to smoking than stable angina.[8] Additionally, research indicates that quitting smoking leads to a quicker reduction in cardiovascular risk than the gradual improvement seen with drugs that manage cholesterol and blood pressure by targeting atherosclerotic changes.[9-11] Thus, it is clear that the thrombogenic effects of smoking outweigh its role in the gradual development of atherosclerotic plaques. Smoking remains a well-recognized cardiac risk factor linked to an increased likelihood of cardiovascular mortality or acute coronary events.[12] Various studies have validated the strong and lasting effect of smoking on cardiovascular health, even after accounting for other established risk factors.[13-14] Risk of myocardial infarction (MI) and sudden cardiac death attributable to smoking is 3 to 4 times and around 10 times higher, respectively.[15] The surprising observation of improved survival and prognosis in myocardial infarction (MI) patients who smoked became known as the "smoker's paradox." However, subsequent research linked this effect to factors such as a less extensive risk profile, milder disease, and notably, younger age. Once these confounding variables were properly adjusted for, the apparent advantage of smoking largely disappeared.[16] Study by Panda et al[17] reported single vessel disease 48.1%, double vessel disease 20.3%, and multivessel disease 31.4% among smokers with ischemic heart disease. Moreover, occlusive coronary artery disease was observed in 57.6%. Another study reported pre-procedure TIMI grade 0 (55.81%), grade I (10.16%), grade 2 (19.46%) and grade 3 (14.57%) among smokers. Moreover, SVD 38.43%, DVD 26.32%, and TVD 18.97%. In addition, the most common culprit artery was LAD 54.47% followed by RCA 33.41% and LCx 10.65%. Similarly, High C lesion was found in 44.68%. In addition, post-procedure TIMI grade 0 was (0.61%), grade I (0.86%), grade 2 (1.35%) and grade 3 (97.18%).[18]

Investigating the distinct patterns of atherosclerotic lesions among smokers may contribute to the development of tailored preventive and therapeutic strategies, which could mitigate the elevated cardiovascular risk in this group. Despite the known association between smoking and atherosclerosis, there remains a paucity of data that specifically addresses angiographic patterns in this population, particularly in low- and middle-income countries where smoking prevalence and cardiovascular mortality are on the rise. This study aims to fill this gap by assessing the angiographic characteristics and pattern of atherosclerotic lesions in smokers presenting with IHD, thus providing valuable data that could improve risk stratification and treatment protocols for these high-risk individuals.

METHODOLOGY

This cross-sectional study was conducted at the Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Karachi, over a duration of six months January to June, 2025 following approval from the College of Physicians and Surgeons Pakistan (CPSP). The primary objective was to assess the angiographic characteristics and atherosclerotic lesion patterns in smokers presenting with ischemic heart disease (IHD). The sample size was calculated using the WHO sample size calculator, based on a 19.87% frequency of triple vessel disease among smokers, with a 5% margin of error and 95% confidence level. A minimum of 237 patients was required. A non-probability consecutive sampling technique was employed to enroll eligible patients. **Inclusion criteria** included adult patients aged 18–65 years of either gender who presented with ischemic heart disease and had a documented history of smoking, classified as per the operational definition (current smoker or ex-smoker). All participants underwent coronary angiography during their hospital stay. **Exclusion criteria** were: patients with a history of recurrent myocardial infarction, previous coronary interventions such as PCI or CABG, renal failure, chronic liver disease, and pregnancy. Ethical approval was obtained from the institutional review

board and CPSP before initiation of the study. Written informed consent was obtained from all participants prior to enrollment. Demographic and clinical data were recorded on a predesigned structured proforma. Variables collected included age, gender, place of residence, BMI, history of diabetes mellitus, hypertension, dyslipidemia, smoking status, number of pack years, and duration of smoking. Electrocardiography (ECG) was used to determine the specific type of IHD (Unstable Angina, NSTEMI, STEMI).

All patients underwent percutaneous coronary intervention (PCI) with coronary angiography. Angiographic parameters recorded included infarct-related (culprit) artery, type of coronary artery disease (single, double, or triple vessel disease), presence of significant CAD (defined as ≥70% stenosis in major coronary arteries or ≥50% in the left main artery), and TIMI flow grade both before and after angioplasty. Data entry and analysis were conducted using SPSS version 26. Quantitative variables such as age, BMI, pack years, and duration of smoking were summarized as mean ± standard deviation or median with interquartile range, depending on distribution. Qualitative variables such as gender, residence, diabetes, hypertension, dyslipidemia, smoking status, and angiographic findings were reported as frequencies and percentages. Stratification was performed based on age, gender, residence, duration of symptoms, diabetes, hypertension, dyslipidemia, and smoking status to assess their association with angiographic findings. Post-stratification comparisons were carried out using Chi-square or Fisher's exact test, as appropriate. A p-value less than 0.05 was considered statistically significant.

RESULTS:

Table 1: Demographic Profile of the Patients (n = 237)

This table outlines the baseline demographic characteristics of patients with ischemic heart disease. The majority of the participants (57.9%) were in the first age category, while 42.1% belonged to the second category. Male patients constituted a larger portion of the sample (74.3%) compared to females (25.7%). Regarding residence, 59.6% of patients were from urban areas and 40.4% were from rural settings. The mean age of the patients was 47.82 years (±10.09), with an average BMI of 26.23 (±4.10). In terms of smoking-related parameters, the mean number of pack years was 27.15 (±13.19), and the mean duration of smoking was 19.91 years (±10.07).

Table 2: Clinical Risk Factors of the Patients (n = 237)

This table presents the distribution of clinical risk factors among the patients. Diabetes mellitus was present in 36.8% of the individuals, whereas 63.2% were non-diabetic. Hypertension was observed in 43.9% of the patients, with the remaining 56.1% not having a history of elevated blood pressure. Dyslipidemia was noted in 29.8% of the participants. A family history of cardiovascular disease (CVD) was reported by 22.2% of the cases. Smoking status revealed that a significant proportion, 68.4%, were current smokers, while 31.6% were ex-smokers.

Table 3: Cardiac Diagnosis of the Patients (n = 237)

This table summarizes the clinical cardiac diagnoses among the patients. The most common type of ischemic heart disease was unstable angina (36.3%), followed by NSTEMI (32.2%) and STEMI (31.6%). The most frequently involved culprit artery was the left anterior descending artery (LAD), accounting for 51.5% of the cases. Other affected arteries included the right coronary artery (RCA) in 30.4%, the left circumflex artery (LCx) in 11.1%, the ramus in 2.9%, and the left main (LM) coronary artery in 4.1%. When categorized by the extent of coronary artery disease (CAD), single-vessel disease (SVD) was most common at 41.5%, followed by triple-vessel disease (TVD) at 30.4% and double-vessel disease (DVD) at 28.1%. A significant majority (87.7%) had angiographically confirmed significant CAD.

Table 4: Angiographic Findings of the Patients (n = 237)

This table focuses on the angiographic assessment of TIMI (Thrombolysis In Myocardial Infarction) flow pre- and post-intervention. Prior to intervention, 52.6% of the patients had no flow (Grade 0), while 12.9%, 13.5%, and 21.1% had Grade 1, 2, and 3 flow, respectively. Post-intervention results showed a marked improvement, with 95.3% achieving optimal reperfusion (Grade 3). Only a small number of patients had Grade 0 (1.2%), Grade 1 (1.8%), or

Grade 2 (1.8%) flow after intervention, indicating the overall success of revascularization procedures.

Table 5: Angiographic Characteristics and Atherosclerotic Lesion Pattern in Smokers with IHD (n = 237)

This table compares angiographic and diagnostic features between current smokers and ex-smokers. The LAD was the most common culprit artery in both groups, observed in 47.9% of current smokers and 59.3% of ex-smokers (p = 0.234). RCA and LCx involvement followed, with LCx being more frequent in current smokers. There was no statistically significant difference between groups regarding the type of CAD (p = 0.262), though single-vessel disease was slightly more common in ex-smokers. Significant CAD was almost equally prevalent in both groups—88.0% in current smokers and 87.0% in ex-smokers (p = 0.853). TIMI flow grades before intervention showed no significant variation (p = 0.942), with Grade 0 being predominant in both groups. Similarly, post-intervention TIMI flow achieved Grade 3 in over 94% of both groups, with no significant difference (p = 0.816). These findings suggest that smoking status (current vs ex-smoker) did not significantly impact the angiographic severity or intervention outcomes in this cohort.

Table 1: Demographic profile of the patients(n=237)

Variables	Group	Count	Percent
Age(years)	Category 1	99	57.9%
	Category 2	72	42.1%
Gender	Male	127	74.3%
	Female	44	25.7%
Residence	Urban	102	59.6%
	Rural	69	40.4%
Age	Mean+SD	47.82±10.09	
BMI		26.23±4.10	
Pack Years		27.15±13.19	
Smoking Years		19.91±10.07	

Table 2: Clinical risk factors of the patients(n=237)

Clinical risk factors	Group	Count	Percent
Diabetes	Yes	63	36.8%
	No	108	63.2%
Hypertension	Yes	75	43.9%
	No	96	56.1%
Dyslipidemia	Yes	51	29.8%
	No	120	70.2%
Family History of CVD	Yes	38	22.2%
	No	133	77.8%
Smoking Status	Current Smoker	117	68.4%
	Ex-Smoker	54	31.6%

Table 3: Cardiac diagnosis of the patients(n=237)

Cardiac Diagnosis	Group	Count	Percent
IHD Type	Unstable Angina	62	36.3%
	NSTEMI	55	32.2%
	STEMI	54	31.6%

Culprit Artery	LAD	88	51.5%
	RCA	52	30.4%
	LCx	19	11.1%
	Ramus	5	2.9%
	LM	7	4.1%
Type of CAD	SVD	71	41.5%
	DVD	48	28.1%
	TVD	52	30.4%
Significant CAD	Yes	150	87.7%
	No	21	12.3%

Table 4: Angiographic findings of the patients(n=237)

Angiographic Findings	Group	Count	Percent
TIMI Flow Pre	Grade 0	90	52.6%
	Grade 1	22	12.9%
	Grade 2	23	13.5%
	Grade 3	36	21.1%
TIMI Flow Post	Grade 0	2	1.2%
	Grade 1	3	1.8%
	Grade 2	3	1.8%
	Grade 3	163	95.3%

Table 5: Angiographic characteristics and pattern of atherosclerotic lesion in smokers presenting with ischemic heart disease (n=237)

Variable	Group	Current Smoker	Ex-Smoker	Total	Chi-Square p-value
Culprit Artery	LAD	56 (47.9%)	32 (59.3%)	88 (51.5%)	.234
	RCA	36 (30.8%)	16 (29.6%)	52 (30.4%)	
	LCx	17 (14.5%)	2 (3.7%)	19 (11.1%)	
	Ramus	4 (3.4%)	1 (1.9%)	5 (2.9%)	
	LM	4 (3.4%)	3 (5.6%)	7 (4.1%)	
Type of CAD	SVD	47 (40.2%)	24 (44.4%)	71 (41.5%)	.262
	DVD	30 (25.6%)	18 (33.3%)	48 (28.1%)	
	TVD	40 (34.2%)	12 (22.2%)	52 (30.4%)	
Significant CAD	Yes	103 (88.0%)	47 (87.0%)	150 (87.7%)	.853
	No	14 (12.0%)	7 (13.0%)	21 (12.3%)	
TIMI Flow Pre	Grade 0	61 (52.1%)	29 (53.7%)	90 (52.6%)	.942
	Grade 1	15 (12.8%)	7 (13.0%)	22 (12.9%)	
	Grade 2	17 (14.5%)	6 (11.1%)	23 (13.5%)	
	Grade 3	24 (20.5%)	12 (22.2%)	36 (21.1%)	
TIMI Flow Post	Grade 0	2 (1.7%)	0 (0.0%)	2 (1.2%)	.816
	Grade 1	2 (1.7%)	1 (1.9%)	3 (1.8%)	
	Grade 2	2 (1.7%)	1 (1.9%)	3 (1.8%)	
	Grade 3	111 (94.9%)	52 (96.3%)	163 (95.3%)	

DISCUSSION:

This study aimed to evaluate angiographic characteristics and the pattern of atherosclerotic lesions in smokers presenting with ischemic heart disease (IHD) at a tertiary cardiac care center in Pakistan. The findings demonstrated that the left anterior descending (LAD) artery was the most frequently involved culprit vessel, followed by the right coronary artery (RCA) and left circumflex (LCx), with single-vessel disease (SVD) being the most common pattern. These observations are in alignment with a growing body of regional and global evidence suggesting a unique coronary profile among smokers and younger patients with CAD.

In our cohort, LAD was the culprit artery in 51.5% of cases, which closely mirrors the findings reported in several studies. For instance, Agrawal et al. (2023),[19] in a systematic review of South Asian patients under 45 years, also identified LAD as the most commonly affected artery and SVD as the predominant angiographic pattern. Similar trends were seen in a local study by Khan et al. (2022),[20] where LAD involvement was noted in 40% of very young acute myocardial infarction (AMI) patients, and 60% had SVD. These findings reinforce the notion that younger smokers, particularly in South Asia, tend to present with discrete coronary involvement, often limited to a single vessel, most frequently the LAD. Our results also reflect findings from Ahmed et al (2003),[21] who observed significantly higher LAD (71%), RCA (59%), and LCx (56%) involvement among smokers compared to non-smokers. Notably, their study also reported a higher frequency of triple-vessel disease (42%) in smokers, which is slightly higher than the 30.4% noted in our study. These variations may be attributed to differences in study populations and the proportion of chronic versus acute presentations.

In terms of lesion severity, our study showed that 87.7% of smokers had significant CAD (defined as ≥70% stenosis or ≥50% in left main), with a high proportion exhibiting complete occlusion (TIMI 0) on initial angiography. This aligns with Samaan et al. (2023)[22] who reported that smokers exhibited more obstructive and mixed-type plaques, particularly in the middle RCA and proximal LAD segments, compared to non-smokers. Their findings underscore the morphological distinction of plaques in smokers, often more vulnerable and thrombogenic, contributing to acute presentations. Interestingly, our findings revealed no statistically significant difference in lesion patterns, culprit artery involvement, or TIMI flow between current and ex-smokers, though current smokers exhibited slightly more LCx involvement. These results resonate with findings from Kanher et al (2021),[23] who noted no major difference in the extent of coronary involvement among subgroups of young ACS patients, suggesting that prior smoking exposure may sustain long-term vascular changes even after cessation.

Our cohort had a predominantly male population (74.3%), echoing findings from Khan et al. (2022)[20] and Yousif et al. (2025),[24] where CAD and left main disease were notably more common in males. Gender-specific disparities in angiographic profiles are also supported by Mehta et al. (2014)[25] and Sharma et al,[26] who emphasized that although LAD remains the most commonly affected artery in women, non-obstructive CAD and symptom underreporting are more prevalent, potentially complicating diagnosis and management in female patients. In terms of clinical diagnoses, the predominance of unstable angina and NSTEMI over STEMI (68.5% vs. 31.6%) in our study reflects similar trends reported in Adil et al. (2023)[27] who found that NSTEMI and unstable angina often accompanied varying CAD severities. Additionally, the overall success of percutaneous interventions was evident in our post-intervention TIMI grade 3 flow achievement in 95.3% of cases, consistent with the FFR and CT angiography study by Park et al. (2015)[28] which concluded that plaque burden and composition (particularly low-attenuation volume and positive remodeling) were strongly predictive of ischemia, emphasizing the importance of lesion characteristics beyond simple anatomical narrowing.

The analysis also emphasizes the critical need for aggressive risk factor modification in smokers. Our data showed current smokers accounted for 68.4%, and the average smoking duration exceeded 19 years, supporting Sharma et al. (2022)[26] and Agrawal et al. (2023),[19] both of whom highlighted smoking as the leading modifiable risk factor for premature MI. Importantly, the "smoker's paradox" — an apparent survival advantage in smokers post-MI — was not supported in our study, aligning with updated interpretations by Ahmed et al[21] and subsequent literature which attribute early recovery to younger age

and fewer comorbidities rather than any protective mechanism of smoking itself.

CONCLUSION:

In light of our findings and the corroborative evidence from global and local literature, there is a clear need to develop smoking-specific cardiovascular prevention strategies. These may include early screening for atherosclerosis in high-risk smokers, public health interventions aimed at tobacco cessation, and targeted clinical management pathways to address unique angiographic patterns observed in this subgroup.

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