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Original Research Article

Cardiometabolic risk and treatment patterns in smokers versus non-smokers with coronary artery disease: Comparative insights from Telangana

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Abstract

Background: Coronary artery disease (CAD) continues to be a leading cause of morbidity and mortality all over the world, with smoking being a major risk factor that is modifiable.
Materials and Methods: A six-month observational study was carried out at a tertiary care hospital in Telangana. A total of 110 patients with diagnostically confirmed coronary artery disease were enrolled. These patients were split into two groups: smokers (n=55) and non-smokers (n=55). Demographics, comorbidities, lifestyle, lab parameters, and medications were recorded. Statistical analysis was done by using Student's t-test, considering a p-value of <0.05 indicating significance.
Results: Smokers developed coronary artery disease (CAD) at an earlier age (41–50 years) compared to non-smokers (51–60 years). Smokers were more likely to have Hypertension (74.5% vs. 61.8%) and diabetes (67.3% vs. 36.9%). Smokers had significant lipid abnormalities, with higher levels of LDL (29.09%), VLDL (18.1%), triglycerides (25.4%), and total cholesterol (23.6%), and lower levels of HDL (40%). The difference in lipid profile between smokers and non- smokers was statistically significant (p = 0.01). Furthermore, smokers exhibited higher rates of alcohol consumption, suboptimal sleep quality, obesity, elevated serum creatinine levels, and increased blood pressure. Medication use was more intensive in smokers, particularly clopidogrel (96.3%) and rosuvastatin (63.64%).
Conclusion: Smoking leads to hypertension, diabetes, dyslipidaemia, and renal dysfunction by increasing the onset and progression of coronary artery disease. Risk management, early screening, cessation techniques, and intensive pharmacological management are essential.

Keywords: Cardiovascular risk, Smoking, Dyslipidemia, Hypertension

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1. Introduction

Coronary artery disease (CAD) occurs when the blood vessels that deliver oxygen- rich blood to the heart become narrowed. Tackling individual risk factors can significantly boost cardiovascular health.[1] Since CAD arises from a mix of factors, both genetic and lifestyle elements come into play, including age, sex, family history, hypertension, diabetes, and high cholesterol levels. Among these, smoking stands out as a major risk factor because it speeds up atherosclerosis by oxidizing LDL and damaging the endothelium, which can lead to early vascular issues. The extent and pattern of blood vessel blockage are key in deciding treatment options and overall outcomes.[2]

This study is designed to examine and compare the clinical traits, risk factors, and biochemical indicators of smokers versus non-smokers with coronary artery disease (CAD) at a tertiary hospital in Telangana, India. The insights gained from this research will help in developing targeted strategies for the prevention and management of CAD.

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2. Materials and Methods

2.1. Study design

This research employed a prospective observational study at Prathima Institute of Medical Sciences in Karimnagar, Telangana, during a six- month period.

2.2. Study population

A total of 110 patients diagnosed with coronary artery disease (CAD) were recruited into the study. Patients were included regardless of smoking status. Smoking status was determined based on the patient's history of tobacco exposure as follows: smokers (individuals with current or past tobacco exposure), and non-smokers (individuals with no history of tobacco exposure).

2.3. Inclusion criteria

Patients meeting the inclusion criteria were above the age of 18 years and were clinically confirmed diagnosis of CAD on clinical, biochemical and imaging studies.

2.4. Exclusion criteria

Patients with congenital heart diseases, patients with severe systemic illnesses, and patients with renal impairment were not included in the study.

2.5. Data collection

Information was collected related to sociodemographic variables, medical history, lifestyle (diet/exercise/sleep), body mass index (BMI), blood pressure, lipid profile analyses, fasting blood glucose, Serum creatinine and clinical outcomes.

3. Statistical Analysis

Descriptive statistics were used to describe demographic data by smoking status. Differences between smoking status and risk factors, included in this study, were analyzed using Student's t-test for continuous variables. Statistical significance was defined as a p value of <0.05.

4. Ethical Approval

The research was carried out in compliance with ethical guidelines, and a document of informed consent was obtained and signed by all participants in the study.

5. Results

This study included 110 CAD patients, 88 were men (80%) and 22 were women (20%) as shown in **Table 1**. Family history for CAD was noted in 20% of the study population .Most CAD cases were found in the 51–60 age group, accounting for 34.54%. However, smokers tend to develop CAD earlier, with the highest incidence in the 41–50 age range (32.72%), while non-smokers peaked at 51–60 years (38.18%). (**Table 2**)

Table 1: Demographic and lifestyle characteristics of participants

Characteristics	Frequency	percentage
Male	88	80%
Female	22	20%
Alcoholic	53	48.18%
Non-alcoholic	57	51.81%
Smokers	55	50%
Non-smokers	55	50%
Family History	20	22%

Data are presented as frequency (n) and percentage (%). "Family History" refers to participants with a first-degree relative affected by the coronary artery disease

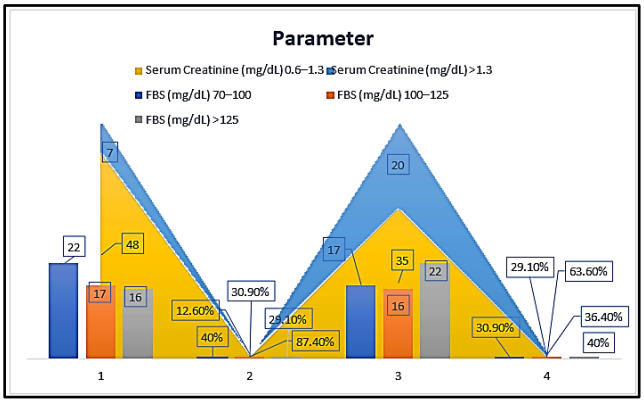

Figure 1: Fasting blood sugar and serum creatinine levels among participants

Data are presented as frequency (n) and percentage (%) for smokers and non- smokers. FBS: fasting blood sugar.

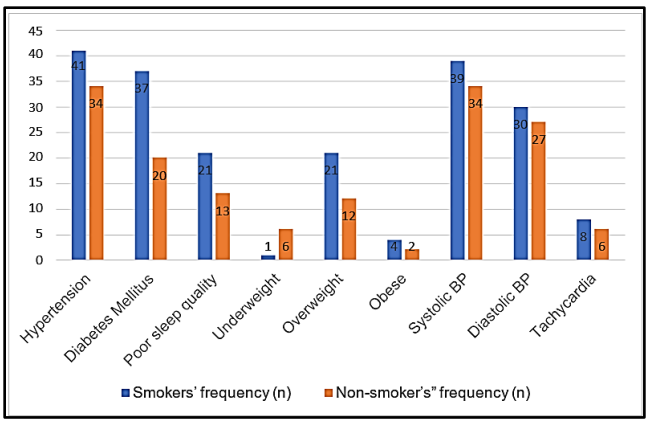

Figure 2: Distribution of risk factors among smokers and non-smokers

Data are presented as number of participants (n). BP: blood pressure.

About 48.18% of patients reported consuming alcohol, with no significant difference between smokers and non-smokers.(**Table 1**). Smokers had a notably higher rate of being overweight (38.18%) compared to non-smokers (21.81%), and obesity rates were also elevated among smokers (7.27% vs. 3.63%). (**Table 3**), Hypertension stands out as the most prevalent risk factor, affecting 68.18% of individuals, and it's notably higher among smokers at 74.54%

compared to 61.81% in non-smokers as shown in **Figure 1**. Smokers showed elevated blood pressure levels, with 66.36% having systolic readings over 120 mmHg and 51.8% with diastolic readings above 80 mmHg. Tachycardia was also more prevalent among smokers (14.54%) compared to non-smokers (10.90%). (**Table 3**)

Diabetes mellitus was found in 51.81% of patients, with a greater occurrence in smokers (67.27%) versus non-smokers (36.36%). (**Table 3**)

Smokers showed elevated blood pressure levels, with 66.36% having systolic readings over 120 mmHg and 51.8% with diastolic readings above 80 mmHg. Tachycardia was also more prevalent among smokers (14.54%) compared to non-smokers (10.90%). (**Table 3**)

Table 2: Age distribution of participants by smoking status

Age group	Overall Frequency (n)	Overall Percentage (%)	Smokers Frequency (n)	Smokers Percentage (%)	Non- Smokers Frequency (n)	Non- Smokers Percentage (%)
30–40	7	6.36%	3	5.45%	4	7.27%
41–50	27	24.54%	18	32.72%	9	16.36%
51–60	38	34.54%	17	30.90%	21	38.18%
61–70	23	20.90%	9	16.36%	14	25.45%
71–80	14	12.72%	7	12.72%	7	12.72%
81–90	1	0.90%	1	1.81%	0	0%

Data are shown as number of participants (n) and corresponding percentage (%). Smokers and non-smokers percentages reflect proportions within each subgroup.

Table 3: Distribution of risk factors among participants

Risk Factor	Overall Frequency (n)	Overall percentage (%)	Smokers' frequency (n)	Smokers' percentage (%)	Non- smoker's" frequency (n)	Non-smokers' percentage (%)
Hypertension	75	68.18%	41	74.54%	34	61.81%
Diabetes Mellitus	57	51.81%	37	67.27%	20	36.36%
Poor sleep quality	34	30.90%	21	38.18%	13	23.63%
Underweight	7	6.36%	1	1.81%	6	10.90%
Overweight	33	30%	21	38.18%	12	21.81%
Obese	6	5.45%	4	7.27%	2	3.63%
Systolic BP	73	66.36%	39	70.90%	34	61.81%
Diastolic BP	57	51.81%	30	54.54%	27	49.09%
Tachycardia	14	12.72%	8	14.54%	6	10.90%

BMI categories are defined as underweight (<18.5 kg/m²), overweight (25–29.9 kg/m²), and obese (≥30 kg/m²). Systolic BP (>120 mm/hg), Diastolic BP (>80 mm/hg) and Pulse (>100 bpm). Values are presented as frequency (n) and percentage (%).

Table 4: Lipid profile of participants

Parameter	Category (mg/dL)	Overall Frequency	Non-smoker's Frequency	Smokers Frequency
HDL	<35	33 (30%)	11 (20%)	22 (40%)
	35–60	77 (70%)	44 (80%)	33 (60%)
LDL	<70	88 (80%)	49(89.09%)	39 (70.9%)
	70–190	22 (20%)	6 (10.9%)	16(29.09%)
TG	<150	87(79.09%)	46 (83.6%)	41 (74.5%)
	150–500	23 (20.9%)	9 (16.3%)	14 (25.4%)
VLDL	<40	96 (87.2%)	51 (92.7%)	45 (81.8%)
	40–100	13 (11.8%)	3 (5.45%)	10 (18.1%)
	>100	1 (0.90%)	1 (1.8%)	0 (0%)
Serum Cholesterol	<200	91 (82.7%)	49(89.09%)	42 (76.3%)
	200–280	19 (17.2%)	6 (10.9%)	13 (23.6%)

Values are presented as frequency (percentage). HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein; TG: Triglycerides; VLDL: Very Low-Density Lipoprotein; mg/dL: milligrams per decilitre.

Table 5: Lipid profile differences between smokers and non-smokers

Lipid levels	Smokers	Nonsmokers	P Value
HDL	22	11	0.01
LDL	16	6	0.01
TG	14	9	0.01
VLDL	10	4	0.01
Serum Cholesterol	13	6	0.01

$p = 0.01$ indicates a significant difference between smokers and non-smokers.

Table 6: Prescribed medications among participants

Drugs	Overall Frequency	Smokers Frequency	Smokers Percentage	Nonsmokers Frequency	Nonsmokers Percentage
Tab Torsemide+ Spironolactone	26(23.64%)	13	23.64%	13	23.64%
Tab Bisoprolol fumarate	37(33.64%)	25	45.45%	12	21.82%
Tab Telmisartan +hydrochlorothiazide	19(17.27%)	12	21.82%	7	12.73%
Tab Carvedilol	23(20.91%)	14	25.45%	9	16.36%
Tab Nitro-glycerin	20(18.18%)	14	25.45%	6	10.91%
Tab Atorvastatin	48(43.64%)	33	60%	15	27.27%
Tab Aspirin	43(39.09%)	30	54.5%	13	23.64%
Tab Rosuvastatin	50(45.45%)	35	63.64%	15	27.27%
Tab Dapagliflozin+ Metformin+Sitagliptin	6(5.4%)	3	5.45%	3	5.45%
Tab clopidogrel	78(70.91%)	53	96.3%	25	45.45%
Tab Dapagliflozin	16(14.55%)	9	16.3%	7	12.73%
Tab Glicazide	15(13.64%)	7	12.7%	8	14.55%

Values are presented as number of patients (n) with corresponding percentages (%). Multiple drugs may have been prescribed to the same patient; therefore, the cumulative frequency may exceed 100%

Smokers exhibited significantly worse lipid profiles, characterized by higher levels of low-density lipoprotein (LDL, 29.09%), very-low-density lipoprotein (VLDL, 18.1%), triglycerides (25.4%), and total serum cholesterol (23.6%) than non- smokers, alongside lower HDL levels (40%).(**Table 4**) The disparity in lipid profiles between smokers and non- smokers was statistically significant (p = 0.01).(**Table 6**). Furthermore, fasting blood glucose levels were elevated in smokers (69.1% vs. 60%).Renal dysfunction was also more common, with 36.36% of smokers showing increased serum creatinine levels compared to 12.6% of non-smokers as shown in **Figure 2**. Smokers were prescribed antiplatelets like clopidogrel much more often (96.36% compared to 45.45%) and statins such as rosuvastatin (63.64% versus 27.27%), reflecting their higher burden of risk factors (**Table 6**). The more enhanced pharmacological approach taken with smokers underscores the critical need for robust secondary prevention in this group.

6. Discussion

Demographic profile-In our study male predominance aligns with global trends that show men are more likely to develop CAD, largely due to higher rates of smoking and other risky behaviors, along with hormonal factors.[3] Interestingly, recent studies indicate that female smokers might actually face an even higher relative risk of CAD, which makes it crucial to focus on preventive measures for women.[4]

1. **Age distribution:** Smokers developed CAD a decade earlier than nonsmokers. This supports the idea that smoking speeds up vascular aging, disrupts endothelial function, and accelerates atherosclerosis.[5,6]

2. **Lifestyle factors:** Lifestyle factors such as alcohol intake and poor sleep were more frequent among smokers. Alcohol alone might not set the groups apart, the combination of smoking and drinking can have compounded harmful effects, increasing oxidativestress, vascular inflammation, and endothelial dysfunction.[7,8] Smokers also reported poor sleep quality (38.18% vs. 23.63%), likely due to nicotine's effects on the sympathetic nervous system and its suppression of REM sleep.[9]

3. **Anthropometric profile:** Smoking is often linked to lower body weight, recent findings suggest it actually contributes to central fat accumulation and insulin resistance, which increases the cardiometabolic risk.[10]

4. **Major risk factors:** Hypertension (74.54%) and diabetes (67.27%) were significantly higher among smokers .Smoking triggers acute spikes in blood pressure due to the release of catecholamines from nicotine, stiffening of blood vessels, and a decrease in nitric oxide availability.[11] Smoking is recognized as a contributor to insulin resistance and β-cell dysfunction, which heightens the risk of diabetes and accelerates the progression of coronary artery disease (CAD).[12]

5. **Hemodynamic and biochemical parameters:** Biochemical parameters revealed higher LDL, VLDL, triglycerides, and total cholesterol, with lower HDL in smokers ($p = 0.01$), reflecting smoking-induced dyslipidaemia and oxidative modification of lipoproteins. These observations align with the hemodynamic stress caused by smoking, which speeds up endothelial damage and vascular dysfunction.[13] This aligns with earlier research that connects smoking to dyslipidaemia, LDL oxidation, and disrupted cholesterol transport.[14,15] Furthermore, fasting blood glucose levels are elevated in smokers when compared to non-smokers[16] Renal dysfunction was also more common, in smokers showing increased serum creatinine levels compared to non-smokers. This supports findings that link smoking to a reduced glomerular filtration rate and microvascular damage.[17]

6. **Therapeutic patterns:** Smokers received more intensive pharmacotherapy—especially clopidogrel and rosuvastatin—suggesting greater disease severity and clinician awareness of their elevated risk. The treatment patterns observed in this study were closely tied to the severity of the disease. Rosuvastatin achieves the most significant reduction in LDL-C and LDL-P levels, enhances the apoA-I/apoB ratio, and maintains a favorable safety profile.[18] Data from registries like CLARIFY also underline the necessity for strict adherence to guideline-directed therapy for smokers with coronary artery disease (CAD).[19]

7. Conclusion

Smoking has a serious impact on cardio metabolic risk factors and speeds up the development of coronary artery disease (CAD). It also raises the chances of needing long-term medication. To lessen the burden of disease, it's crucial to conduct early screenings and manage modifiable risks effectively. Policymakers can bolster anti-tobacco initiatives to aid in prevention efforts. Pharmacist-led interventions are key in managing CAD. Clinical pharmacists play an important role in helping patients quit smoking, stick to their treatment plans, and keep an eye on other health issues. Teaching patients about the direct dangers smoking poses to heart health can be a powerful motivator for making lifestyle changes.

8. Author Contributions

Humera Sadaf, B. Shiva Prasad and G. Akash were the creative minds behind the concept and design of this study. The data collection and analysis were expertly handled by Nahiya Fathima and Humera Sadaf. Nahiya Fathima took on the role of supervisor, interpreting the findings and putting the finishing touches on the manuscript. All authors came together to review, edit, and give their stamp of approval on the final version of the manuscript.

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11. Conflict of Interest

The authors have no conflicts of interest to disclose.

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