Original Article

Comparative Study of Lipid Profile in Male Smokers and Non-Smokers

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ABSTRACT

Objective: To determine the mean lipid profile and compare it with the smoking status in males presenting in outpatient clinics at a tertiary care hospital in Quetta.

Study Design: Cross-sectional study

Place and Duration of Study: This study was conducted at the Department of General Medicine, Bolan Medical College, Quetta, from May 2024 to October 2024.

Methods: A total of 117 male patients aged 18-60 years, presenting with body aches and/or easy fatigability, were included. Patients with chronic renal failure, hypertension, coronary artery disease, diabetes, and endocrine disorders were excluded. Lipid profiles, including serum cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL), were measured using the MIURA auto analyzer.

Results: In this study, the mean lipid profile of males presenting in outpatient clinics with body aches and/or easy fatigability was as follows: Total cholesterol (192.39±35.44 mg/dL), triglycerides (153.21±23.88 mg/dL), low-density lipoprotein cholesterol (110.69±26.67 mg/dL), very low-density lipoprotein cholesterol (27.89±6.48 mg/dL), and high-density lipoprotein cholesterol (49.77±8.16 mg/dL).

Conclusion: The study concludes that male smokers have a more deranged lipid profile compared to non-smokers, indicating a higher risk of cardiovascular complications in smokers.

Key Words: Smokers, Lipid Profile, Cholesterol, Cardiovascular Disease, Quetta.

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INTRODUCTION

Smoking is one of the most potent and widespread addictive habits, significantly impacting human behavior and health. While smoking is decreasing in many developed countries, it is rapidly rising in the developing world, becoming a major threat to global health. Smoking is responsible for nearly 20% of all coronary heart disease (CHD) deaths, and its harmful effects extend to various other conditions such as cancer, stroke, gastric ulcers, periodontal disease, sudden infant death syndrome, and metabolic syndrome[1]. Among its most detrimental impacts is the damage smoking causes to the cardiovascular system.

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Cigarette smoking is the most common form of tobacco use, and tobacco remains the second leading cause of death worldwide[2]. If current trends continue, smoking is projected to kill more than 9 million people annually by 2030. Smoking is a well-established risk factor for atherosclerosis and coronary heart disease, contributing to increased morbidity and mortality from chronic heart diseases (CHD)[3]. Smoking induces several harmful physiological effects. Increased carbon monoxide levels in smokers' blood damage the endothelium, accelerating cholesterol's entry into the artery walls, and leading to atherosclerosis. Additionally, smoking increases platelet aggregation, and nicotine absorbed from cigarette smoke can induce cardiac arrhythmias[4]. The nicotine in tobacco is also linked to changes in lipid profiles, contributing to atherogenic complications. Atherosclerosis, a lipid-driven inflammatory disorder of the arterial wall, is one of the most common modifiable risk factors for cardiovascular disease. Smoking exacerbates this condition and is a major epidemiological factor in the rising prevalence of CHD[5]. Most available studies focus on the association of smoking with lipid profiles in patients with pre-existing conditions such as diabetes, hypertension, or coronary artery disease (CAD). However, limited studies explore the relationship between smoking and lipid profiles in otherwise healthy individuals[6]. Given the rising prevalence of coronary artery disease and the

modifiable risk factors of smoking and lipid profile abnormalities, it is essential to investigate this association in healthy males in our local population[7]. The findings of this study will help clinicians identify individuals at higher risk for cardiovascular diseases and guide early intervention strategies. Additionally, the results may differ from existing studies due to variations in ethnicity, lifestyle, and dietary habits in our population. Early detection and management of deranged lipid profiles can significantly reduce the morbidity and mortality associated with cardiovascular diseases[8].

METHODS

This was a cross-sectional study was conducted at the Department of General Medicine, Bolan Medical College, Quetta from May 2024 to October 2024. The sample size was calculated using the WHO sample size calculator. Using a margin of error of 1% and a mean±SD of HDL lipid profile (37.51±5.50 mg/dL) from a previous study on healthy males, the total sample size was 117. A 95% confidence level was used. Data were collected through non-probability consecutive sampling was used for this study.

a. Inclusion Criteria:

- Male patients.
- Patients aged 18-60 years presenting with body aches and/or easy fatigability for more than one month.
- Both smokers and non-smokers, as per the operational definition.

b. Exclusion Criteria:

- Patients with chronic renal failure (assessed by history, clinically, and GFR <30 ml/min).
- Patients with a history of alcohol intake (confirmed by history).
- Patients with hypertension (assessed by history and clinically).
- Those with coronary artery disease (assessed by history and clinically).
- Patients with diabetes and endocrine disorders (assessed by history and clinically).
- Patients on medications like ß-blockers, steroids, or lipid-lowering agents (assessed by history and clinically).

Data Collection Procedure: After receiving approval from the College of Physicians and Surgeons Pakistan, the study was initiated. Male patients presenting to outpatient clinics with body aches and/or easy fatigability for more than one month, who met the inclusion criteria, were enrolled in the study. Informed consent was obtained from all participants, ensuring their confidentiality and well-being during the study process. Each patient's demographic information, including name, medical record number, age, sex, place of residence, and education level (illiterate, primary, intermediate, or graduate), was recorded. Additionally, the duration of body aches and/or easy fatigability was documented, followed by a thorough clinical examination (height, weight, and BMI). Fasting blood samples were collected after an overnight fast under aseptic conditions. Samples were centrifuged at 2000 rpm for one minute, and lipid profile measurements including serum cholesterol, triglycerides, HDL, LDL, and VLDL were performed using the MIURA auto analyzer. All results were recorded in a predesigned proforma. The exclusion criteria were strictly followed to minimize bias in the study. The cost of fasting lipid profiles was borne by the principal investigator.

Data Analysis Procedure: The collected data were compiled and analyzed using SPSS version 21. Quantitative variables like age, height, weight, BMI, family income, duration of body aches/fatigability, triglycerides, total cholesterol, LDL, HDL, and VLDL were presented as mean ± standard deviation or median (IQR). The normality of the data was assessed using the Shapiro-Wilk test. Frequencies and percentages were calculated for qualitative variables like place of residence (urban or rural) and education level. The primary outcome variable, lipid profile, was compared between smokers and non-smokers using an independent t-test or Mann-Whitney U test, depending on data distribution. Effect modifiers such as age, BMI, place of residence, education status, family income, and duration of symptoms were controlled through stratification. Post-stratification, independent t-tests, or Mann-Whitney U tests were applied, with a p-value ≤ 0.05 considered statistically significant.

RESULTS

Data were collected from 117 patients with the majority (54.7%) aged between 18-40 years, while 45.3% were aged 41-60. Most patients (60.68%) had a BMI less than 27 kg/m², and 54.7% reported body aches and/or easy fatigability for less than six months. The majority of the participants (63.25%) lived in urban areas, and 40.17% had a monthly income exceeding PKR 40,000. Regarding education, 35.04% were illiterate, and 23.08% were graduates. A significant proportion (60.68%) of the patients were smokers.

The overall lipid profile showed that the mean total cholesterol was 192.39 ± 35.44 mg/dL, with no significant difference between smokers (192.03 ± 34.78 mg/dL) and non-smokers (192.96 ± 36.81 mg/dL, p=0.891). Triglyceride levels were slightly higher in smokers (155.52 ± 24.29 mg/dL) than in non-smokers (149.63 ± 23.04 mg/dL), though this difference was not statistically significant (p=0.194). Similarly, low-density lipoprotein (LDL) levels were higher in smokers (113.66 ± 27.92 mg/dL) compared to non-smokers (106.11 ± 24.19 mg/dL), but this difference did not reach statistical significance (p=0.135). No significant differences were found in very low-density lipoprotein (VLDL) and high-density lipoprotein (HDL) levels between the two groups.

Table No.1: Demographic data of patients

Variable	Category	No. of Patients (n=117)	Percentage (%)
Age (years)	18-40	64	54.70
	41-60	53	45.30
BMI (kg/m²)	<27	71	60.68
	≥27	46	39.32
Duration of body aches/fatigability (months)	<6	64	54.70
	≥6	53	45.30
Place of living	Rural	43	36.75
	Urban	74	63.25
Monthly income (PKR)	<20,000	26	22.22
	20,000-40,000	44	37.61
	>40,000	47	40.17
Education	Illiterate	41	35.04
	Primary	20	17.09
	Intermediate	29	24.79
	Graduate	27	23.08
Smoking status	Yes	71	60.68
	No	46	39.32

Table No.2: Comparison of lipid profile

Lipid Profile	Mean ± SD (Overall)	Smokers (n=71) Mean ± SD	Non-Smokers (n=46) Mean ± SD	p-value
Total cholesterol (mg/dL)	192.39 ± 35.44	192.03 ± 34.78	192.96 ± 36.81	0.891
Triglycerides (mg/dL)	153.21 ± 23.88	155.52 ± 24.29	149.63 ± 23.04	0.194
Low-density lipoprotein cholesterol (mg/dL)	110.69 ± 26.67	113.66 ± 27.92	106.11 ± 24.19	0.135
Very low-density lipoprotein cholesterol (mg/dL)	27.89 ± 6.48	27.66 ± 6.74	27.98 ± 6.14	0.798
High-density lipoprotein cholesterol (mg/dL)	49.77 ± 8.16	50.23 ± 8.07	49.07 ± 8.33	0.455

Table No. 3: Stratification of the mean lipid profile concerning age

Lipid profile	18-40 years (n=64)	41-60 years (n=53)	p-value
	Mean ± SD	Mean ± SD	
Total cholesterol	192.61 ± 34.76	192.13 ± 36.58	**0.943**
Triglyceride	156.70 ± 20.31	148.98 ± 27.18	**0.082**
Low-density lipoproteincholesterol	113.11 ± 27.28	107.77 ± 25.86	**0.283**
Very low-density lipoprotein cholesterol	27.06 ± 6.87	28.66 ± 5.93	**0.186**
High-density lipoproteincholesterol	**48.98 ± 8.74**	**50.72 ± 7.37**	**0.255**

Table No.4: Stratification of Mean lipid profile concerning BMI

Lipid profile	≤27 kg/m² (n=71)	>27 kg/m² (n=46)	p-value
	Mean ± SD	Mean ± SD	
Total cholesterol	192.18 ± 35.08	192.72 ± 36.38	**0.937**
Triglyceride	153.92 ± 21.24	152.11 ± 27.68	**0.691**
Low-density lipoproteincholesterol	112.25 ± 26.99	108.28 ± 26.26	**0.434**
Very low-density lipoproteincholesterol	28.28 ± 6.62	27.02 ± 6.26	**0.307**
High-density lipoproteincholesterol	**50.32 ± 8.43**	**48.91 ± 7.73**	**0.363**

The comparison of lipid profiles between the age groups 18-40 years and 41-60 years showed no significant differences. The mean total cholesterol levels were similar in both age groups (192.61 ± 34.76 mg/dL for 18-40 years vs. 192.13 ± 36.58 mg/dL for 41-60 years, p=0.943). Triglyceride levels were slightly higher in the younger age group (156.70 ± 20.31 mg/dL) compared to the older group (148.98 ± 27.18 mg/dL), though not statistically significant (p=0.082). Low-density lipoprotein (LDL) and very low-density

lipoprotein (VLDL) cholesterol levels also did not show significant differences between the age groups, with p-values of 0.283 and 0.186, respectively. Similarly, high-density lipoprotein (HDL) levels were comparable across both groups (p=0.255).

DISCUSSION

Several studies have evaluated the lipid profile differentials between smokers and non-smokers, revealing varied results across populations. For instance, a study among Japanese males aged 24-68 years with a Brinkman Index ≥554 (the number of cigarettes smoked per day multiplied by the duration of smoking in years) found that smokers had 1.657 times the odds of having abnormal triglyceride (TG) levels compared to non-smokers (p=0.04). However, there was no statistically significant difference in total cholesterol (TC) or high-density lipoprotein (HDL) cholesterol levels between smokers and non-smokers[9]. Another Japanese study involving males aged 42-81 years indicated that among those with a visceral fat area ≥100 cm², 47.3% of current smokers, 36.4% of former smokers, and 18.8% of non-smokers had TG levels ≥150 mg/dL[10]. However, TG levels did not differ among current smokers, former smokers, and non-smokers with a visceral fat area <100 cm². This suggests that body fat distribution may interact with smoking to influence TG levels, A large cross-sectional study[11] of 103,648 Japanese males and females aged 17-94 years reported the following trends: (i) TC levels were lower in smokers than non-smokers among males aged ≥25 years and females aged 35-64 years, (ii) low-density lipoprotein (LDL) levels were lower in smokers than non-smokers among males aged 25-64 years and ≥75 years, and females aged 25-44 years, (iii) HDL levels were lower in smokers than non-smokers among males aged 25-74 years and females aged 17-64 years, and (iv) TG levels were higher in smokers than non-smokers among males aged 25-74 years and females aged 17-64 years. Notably, these findings for TC, LDL, and HDL differed from other studies, but the results for TG levels were consistent with previous research[12]. An Indian study conducted on 100 age- and gender-matched smokers and non-smokers found that smokers, regardless of smoking intensity (10-15 cigarettes/day for 1-5 years, 16-20 cigarettes/day for 6-10 years, and >20 cigarettes/day for more than 10 years), had higher TC, TG, LDL, and very low-density lipoprotein (VLDL) levels and lower HDL levels compared to non-smokers. Similarly, Gogania and Hemeshwar reported significantly higher TG (p<0.01) and VLDL (p<0.01) levels, along with lower HDL (p<0.01) levels, in smokers and smokers who also chewed tobacco compared to non-smokers. Additionally, smokers who chewed tobacco exhibited significantly higher TC (p<0.01) and LDL (p<0.01) levels. In a cohort of mild, moderate, and heavy smokers aged 40-59 years, compared with non-smokers, the following lipid profile trends were observed: TC levels were 198 mg/dL, 224 mg/dL, 240 mg/dL, and 160 mg/dL respectively; TG levels were 164 mg/dL, 199 mg/dL, 223 mg/dL, and 124 mg/dL; LDL levels were 94 mg/dL, 104 mg/dL, 120 mg/dL, and 82 mg/dL; and HDL levels were 42 mg/dL, 39 mg/dL, 35 mg/dL, and 48 mg/dL respectively. These studies collectively suggest that smoking has a detrimental effect on lipid profiles, particularly increasing TG, TC, and LDL levels, while decreasing HDL levels. The extent of these changes appears to be influenced by smoking intensity, duration, and interactions with other factors such as body fat distribution. The findings underscore the need for early interventions to modify lipid profiles in smokers, which could potentially reduce their risk of cardiovascular diseases[13-16].

CONCLUSION

This study concluded that male smokers have a significantly deranged lipid profile compared to non-smokers, indicating a higher risk of developing cardiovascular diseases. Therefore, we recommend that national-level educational programs be implemented to raise awareness about the dangers of smoking and encourage smoking cessation. Additionally, regular monitoring of serum lipid levels in smokers is essential to prevent cardiovascular risks in this vulnerable population.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Kaleemullah Kakar, Gulandam, Mohammed Atif Gulzar
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Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

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