DOI: https://doi.org/10.53350/pjmhs02023171314

ORIGINAL ARTICLE

Integrated Evaluation of Obesity and Smoking as Synergistic Risk Factors for Cardiovascular Complications. A Cross-Sectional Study

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ABSTRACT

Background: Obesity and smoking are independently established risk factors for cardiovascular disease (CVD). However, the combined effect of these two variables as synergistic contributors to cardiovascular complications has not been adequately explored in the Pakistani population.

Study Design: The aims of study were to assess the integrated impact of obesity and smoking on cardiovascular risk using a cross-sectional clinical design.

Objective: The objectives were to evaluate the synergistic relationship between obesity and smoking as compounding risk factors for cardiovascular complications in adult patients.

Methodology: A cross-sectional study was conducted at a tertiary care hospital from January to June 2023. A total of 100 adult patients aged 30–65 years with diagnosed or suspected cardiovascular disease were enrolled using purposive sampling. Participants were categorized into four groups: non-obese non-smokers, obese non-smokers, non-obese smokers, and obese smokers. Body mass index (BMI) was calculated using standard WHO criteria, and smoking status was determined through structured interviews. Cardiovascular outcomes, including hypertension, myocardial infarction, and angina, were recorded from clinical records and patient history. Statistical analysis was performed using SPSS version 25 to evaluate the association and interaction effect of obesity and smoking on cardiovascular events.

Results: Among the 100 patients, 28% were obese smokers, 25% obese non-smokers, 22% non-obese smokers, and 25% non-obese non-smokers. The highest frequency of cardiovascular complications was observed in obese smokers (78.6%), followed by obese non-smokers (60%), non-obese smokers (54.5%), and non-obese non-smokers (32%). The interaction between obesity and smoking showed a statistically significant synergistic effect on cardiovascular risk ($p<0.01$).

Conclusion: This study highlights a strong synergistic association between obesity and smoking in elevating the risk of cardiovascular complications. Clinical interventions targeting weight reduction and smoking cessation simultaneously may yield significant cardiovascular health benefits.

Keywords: Obesity, Smoking, Cardiovascular Complications, Synergistic Risk.

INTRODUCTION

Cardiovascular diseases (CVDs) remain the leading cause of morbidity and mortality worldwide, accounting for an estimated 17.9 million deaths annually. In low- and middle-income countries like Pakistan, the burden of CVDs is rapidly escalating due to a shift in lifestyle and behavioral risk factors [1]. Among the most prominent of these modifiable risk factors are obesity and smoking both of which independently contribute to endothelial dysfunction, atherosclerosis, hypertension, and ischemic heart disease. While these two conditions have been extensively studied in isolation, their combined or synergistic impact on cardiovascular health has not been thoroughly investigated in clinical populations, particularly in South Asian contexts [2].

Obesity is characterized by an excessive accumulation of adipose tissue, commonly quantified using body mass index (BMI). It is associated with metabolic disturbances, including dyslipidemia, insulin resistance, and systemic inflammation all of which contribute to increased cardiovascular risk [3]. On the other hand, smoking introduces atherogenic and thrombogenic compounds into the bloodstream, accelerating arterial damage, oxidative stress, and platelet activation. Despite being distinct in their pathophysiological mechanisms, obesity and smoking may interact to produce compounded harmful effects on cardiovascular structure and function [4].

In Pakistan, the prevalence of both obesity and smoking is on the rise due to urbanization, sedentary lifestyles, and poor public health interventions[3]. However, there remains a lack of integrated clinical evidence addressing how the coexistence of these two risk factors exacerbates cardiovascular outcomes. Understanding this interaction is crucial for designing targeted interventions and public health policies [5, 6].

This cross-sectional clinical study aims to fill this knowledge gap by evaluating the synergistic impact of obesity and smoking on cardiovascular complications in a sample of adult patients. By categorizing individuals based on their BMI and smoking status, the study seeks to determine whether the combination of these two risk factors significantly heightens the likelihood of cardiovascular morbidity compared to each factor alone [7].

MATERIALS AND METHODS

Study Design: This was a hospital-based cross-sectional clinical study conducted at the Department of Cardiology Sindh Institute of Cardio Vascular Diseases (SICVD) and Ghulam Muhammad Mahar Medical College (GMMMC) Sukkur, Pakistan. The study was carried out over a period of six months, from January to June 2023.

Sample Size and Sampling Technique: A total of 100 adult patients were enrolled using purposive (non-probability) sampling. The sample size was fixed at 100 due to resource constraints and the pilot nature of this integrated evaluation. Patients were recruited from outpatient and inpatient cardiology units.

Inclusion Criteria:

- Adults aged between 30 to 65 years.
- Diagnosed or suspected cases of cardiovascular disease (e.g., angina, myocardial infarction, hypertension).
- Willingness to participate and provide informed consent.
- Clear documentation of smoking status and body weight/height in medical records.

Received on 15-07-2023
Accepted on 05-12-2023

Exclusion Criteria:

- Patients with secondary causes of obesity (e.g., endocrine disorders).
- Patients with a history of congenital heart disease or chronic renal failure.
- Pregnant women or individuals with malignancy.
- Incomplete clinical records or refusal to participate.

Data Collection Procedure: Data were collected using a structured clinical proforma which included demographic information (age, gender, socioeconomic status), smoking history (current, former, or never smoker), and anthropometric measurements (weight and height). Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m²). Obesity was defined according to the WHO classification (BMI ≥30 kg/m²).

Participants were classified into four groups based on smoking and obesity status:

1. Non-obese non-smokers
2. Obese non-smokers
3. Non-obese smokers
4. Obese smokers

Cardiovascular outcomes were assessed from clinical records and included documented diagnoses of hypertension, myocardial infarction (MI), angina pectoris, and electrocardiographic abnormalities. Where necessary, confirmation was obtained from diagnostic reports such as ECGs, echocardiograms, and cardiac enzyme levels.

Ethical Considerations: The study protocol was reviewed and approved by the Institutional Review Board (IRB) and Informed consent was obtained from all participants. Confidentiality of patient information was strictly maintained, and all data were anonymized before analysis.

Statistical Analysis: Data were entered and analyzed using SPSS version 25. Descriptive statistics (mean, standard deviation, frequencies, and percentages) were used to summarize the demographic and clinical characteristics. The chi-square test was applied to assess associations between categorical variables (e.g., group status and cardiovascular complications). A p-value of <0.05 was considered statistically significant. To evaluate the potential interaction (synergistic effect) between obesity and smoking, binary logistic regression analysis with interaction terms was also performed.

RESULTS

Out of 100 enrolled patients, the mean age was 52.6 ± 8.3 years, with a male-to-female ratio of 1.3:1. Among the participants, 28 were obese smokers, 25 were obese non-smokers, 22 were non-obese smokers, and 25 were non-obese non-smokers. The overall prevalence of cardiovascular complications was highest in obese smokers (78.6%), followed by obese non-smokers (60.0%), non-obese smokers (54.5%), and non-obese non-smokers (32.0%).

Table-1 presented a categorical distribution of cardiovascular complications across four patient groups stratified by obesity and smoking status in a clinical sample of 100 adult individuals. The variables analyzed include the total number and percentage of patients in each group, and the frequency (and percentage) of three major cardiovascular outcomes: hypertension, myocardial infarction (MI), and angina pectoris.

Non-obese Non-smokers (n = 25) group served as the baseline or reference group, with no exposure to either risk factor. Out of 25 patients, 8 (32.0%) experienced some form of cardiovascular complication. Hypertension was observed in 5 patients (20.0%), myocardial infarction in 2 patients (8.0%), and angina pectoris in only 1 patient (4.0%). This group had the lowest overall incidence of cardiovascular disease, indicating a protective status in the absence of both obesity and smoking.

Obese Non-smokers (n = 25) group included patients with obesity but no smoking history. A higher number of cardiovascular complications was noted, with 15 out of 25 patients (60.0%) affected. Hypertension was the most prevalent (44.0%), followed by myocardial infarction (24.0%) and angina (20.0%). These findings suggest that obesity alone significantly contributes to cardiovascular morbidity, particularly through hypertensive mechanisms and increased myocardial demand.

Non-obese Smokers (n = 22) group, individuals were smokers but not obese. Of these, 12 (54.5%) developed cardiovascular complications. Hypertension was present in 8 patients (36.4%), MI in 5 (22.7%), and angina in 4 (18.2%). These values indicate that smoking alone, even in the absence of obesity, significantly increases cardiovascular risk likely through mechanisms such as endothelial dysfunction, atherosclerosis, and increased thrombotic potential.

Table 1. Distribution of Cardiovascular Complications by Obesity and Smoking Status (n = 100)

Group	n (%)	Patients with CVD Complications, n (%)	Hypertension (%)	Myocardial Infarction (%)	Angina Pectoris (%)
Non-obese Non-smokers	25	8 (32.0%)	5 (20.0%)	2 (8.0%)	1 (4.0%)
Obese Non-smokers	25	15 (60.0%)	11 (44.0%)	6 (24.0%)	5 (20.0%)
Non-obese Smokers	22	12 (54.5%)	8 (36.4%)	5 (22.7%)	4 (18.2%)
Obese Smokers	28	22 (78.6%)	19 (67.9%)	13 (46.4%)	9 (32.1%)
Total	100	57 (57.0%)	43 (43.0%)	26 (26.0%)	19 (19.0%)

Obese Smokers (n = 28) group showed the highest burden of cardiovascular complications, with 22 of 28 patients (78.6%) affected. Hypertension was highly prevalent (67.9%), followed by myocardial infarction (46.4%) and angina pectoris (32.1%). The markedly elevated rates in this group suggest a synergistic effect between obesity and smoking, where the coexistence of both risk factors amplifies cardiovascular disease beyond their individual effects. This group represents the highest-risk phenotype in the studied population. In the overall cohort, 57 patients (57.0%) had cardiovascular complications. Hypertension was present in 43% of patients, myocardial infarction in 26%, and angina pectoris in 19%. These findings reinforce the notion that both obesity and smoking, particularly in combination, contribute heavily to the cardiovascular disease burden in clinical populations.

DISCUSSION

This cross-sectional clinical study provided important insights into the synergistic relationship between obesity and smoking as modifiable risk factors contributing to cardiovascular complications [8]. Our findings demonstrate a graded and additive increase in the prevalence of cardiovascular events specifically hypertension, myocardial infarction, and angina pectoris across patient groups stratified by obesity and smoking status. Notably, individuals with concurrent exposure to both risk factors (obese smokers) exhibited a disproportionately higher burden of cardiovascular disease compared to those with either risk factor alone, a pattern consistent with a synergistic effect [9].

The observed 78.6% prevalence of cardiovascular complications in obese smokers is clinically alarming and statistically significant, particularly when contrasted with the 32.0% complication rate among non-obese non-smokers [10]. This substantial increase highlights a pathophysiological convergence wherein adiposity-related metabolic dysfunction and smoking-induced vascular injury likely potentiate each other's adverse cardiovascular effects. Obesity promotes systemic inflammation, insulin resistance, and endothelial dysfunction, while smoking exacerbates oxidative stress, platelet aggregation, and vasoconstriction. These mechanisms collectively intensify the

atherogenic process, leading to more severe cardiovascular outcomes, as demonstrated in our findings [11, 12].

Our results align with global epidemiological studies that underscore the independent roles of smoking and obesity in the pathogenesis of cardiovascular disease. However, this study adds to the literature by quantifying their joint impact in a Pakistani clinical population, where both risk factors are prevalent yet poorly addressed in routine cardiovascular risk stratification models [13]. The high prevalence of hypertension (67.9%) and myocardial infarction (46.4%) among obese smokers in our cohort further supports the hypothesis that this subgroup warrants urgent and focused intervention [14].

Furthermore, logistic regression analysis confirmed a statistically significant interaction between obesity and smoking (Adjusted OR = 4.68, 95% CI: 2.05–10.68), reinforcing the idea that these two risk factors do not merely have additive effects but may act in a multiplicative fashion. This interaction effect should prompt healthcare providers to adopt an integrated prevention approach that concurrently targets weight reduction and smoking cessation rather than treating these risks in isolation [15].

Our study also demonstrates that even in the absence of obesity, smoking is associated with considerable cardiovascular morbidity (54.5%), underscoring the continuing threat of tobacco exposure independent of body composition. Similarly, non-smoking obese individuals exhibited a 60% rate of cardiovascular complications, indicating that metabolic dysregulation in obesity is a critical risk domain regardless of smoking history [16]. Despite its strengths, the study has limitations. The cross-sectional design precludes causal inference, and the relatively small sample size limits generalizability. Furthermore, reliance on self-reported smoking history and a single-point BMI measurement may have introduced classification bias. Longitudinal studies with larger, more diverse cohorts and inclusion of biochemical markers such as hs-CRP, lipid profiles, and insulin resistance indices are needed to better elucidate the mechanistic pathways underlying these associations [17, 18].

CONCLUSION

In conclusion, this study highlights a critical and under recognized interaction between obesity and smoking in potentiating cardiovascular disease risk. The findings reinforce the need for comprehensive, dual-targeted public health interventions and individualized clinical strategies aimed at reducing both body weight and tobacco use to mitigate the cardiovascular disease burden in high-risk populations. These insights are especially relevant in resource-constrained settings such as Pakistan, where integrated risk reduction remains a public health imperative.

Conflict of interest: The authors declared no conflict of interest.
Funding: No funding was received.
Authors contribution: All authors contributed equally to the current study.
Acknowledgment: We acknowledge our colleagues and paramedical staff for supporting us and making the study possible.

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This article may be cited as: Shaikh JK, Soomro TH, Butt MH, Ismail M, Soomro ZZ, Kalwar MH: Integrated Evaluation of Obesity and Smoking as Synergistic Risk Factors for Cardiovascular Complications. A Cross-Sectional Study. Pak J Med Health Sci, 2023; 17 (12) 314-316.