

PREVALENCE OF PASSIVE SMOKING IN PATIENTS WITH STROKE

Original Research

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ABSTRACT

Background: Stroke is one of the leading causes of morbidity and mortality worldwide, often associated with modifiable risk factors. Passive smoking, also known as secondhand smoke exposure, has emerged as a significant contributor to cardiovascular diseases, including stroke. Despite global efforts to reduce tobacco-related harm, passive smoke exposure remains prevalent, especially in low- and middle-income countries. Understanding its impact on stroke patients is crucial to inform targeted prevention strategies.

Objective: To determine the frequency of passive smoking among patients diagnosed with stroke.

Methods: This cross-sectional study was conducted at the Neurology Department of Pak Emirates Military Hospital, Rawalpindi, over a six-month period from September 2024 to February 2025. A total of 139 patients aged 18 to 80 years, diagnosed with acute stroke and confirmed via non-contrast computed tomography (CT) of the brain, were included after obtaining informed consent. Patients with a history of active smoking or previous stroke were excluded. Demographic data, clinical characteristics, and history of passive smoking were collected through structured interviews. Stroke types were categorized as ischemic or hemorrhagic. Data were analyzed using SPSS version 25.0, with quantitative variables expressed as medians and interquartile ranges (IQR), and qualitative variables as frequencies and percentages.

Results: Among the 139 patients, the median age was 51 years (IQR: 22), median symptom duration was 36 hours (IQR: 13), and median BMI was 23.8 kg/m² (IQR: 2.1). Of the total, 78 (56.1%) were male and 61 (43.9%) were female. Ischemic stroke was diagnosed in 112 (80.6%) patients and hemorrhagic stroke in 27 (19.4%). Passive smoking was reported by 21 (15.1%) patients.

Conclusion: A significant proportion of stroke patients were found to be exposed to passive smoking, highlighting the need for public health measures to reduce secondhand smoke exposure in the community.

Keywords: Cardiovascular Diseases, Cerebrovascular Disorders, Epidemiology, Passive Smoking, Risk Factors, Stroke, Tobacco Smoke Pollution.

INTRODUCTION

Stroke remains one of the leading causes of death and disability among adult men and women globally. Defined as an acute cerebrovascular event characterized by the disruption of blood flow to the brain, stroke leads to significant neurological damage and long-term health consequences. According to the World Health Organization, approximately 15 million people experience a stroke each year, placing a heavy burden on individuals, families, and healthcare systems worldwide (1). Strokes are generally categorized into ischemic strokes, accounting for about 87% of cases, intracerebral hemorrhagic strokes comprising around 10%, and subarachnoid hemorrhages making up the remaining 3% (2). Among the many modifiable risk factors for stroke, tobacco use remains one of the most significant and preventable contributors. Smoking continues to be prevalent globally, particularly among younger populations and in low- and middle-income countries where public health regulations may be less stringent (3). Despite global public health efforts to curb smoking through education and policy interventions, the overall number of smokers has not declined substantially due to population growth (4). It is often misunderstood that low-nicotine cigarettes are less harmful, yet substantial evidence confirms a strong dose-dependent relationship between smoking and ischemic stroke (5). Moreover, smoking has been implicated in promoting systemic inflammation, a critical process involved in the pathophysiology of cerebrovascular events (6).

Equally concerning is the impact of secondhand smoke (SHS), also referred to as passive or involuntary smoking. SHS exposure remains a serious public health issue, particularly for individuals who do not smoke but live or work in environments where tobacco smoke is present. It is estimated that 37% of the global population continues to be exposed to SHS, with disproportionately higher exposure rates among women and children, especially in economically disadvantaged populations (7). Tobacco smoke contains thousands of toxic chemicals, many of which are known carcinogens and contributors to cardiovascular disease (8). Studies have linked both short- and long-term exposure to SHS with the progression of carotid artery intimal-medial thickness, a known precursor to stroke. Various biological mechanisms have been proposed to explain this association, including increased fibrinogen levels, enhanced thrombogenic potential, reduced HDL cholesterol, carboxyhemoglobin formation, autonomic and hemodynamic disturbances, oxidative stress, endothelial dysfunction, and atherosclerosis due to toxins like 1,3-butadiene present in cigarette smoke (9,10). While considerable research has explored the relationship between active and passive smoking with stroke risk in developed countries, there is a notable lack of local data in regions such as Pakistan. This gap is particularly significant considering the ongoing public health burden of stroke and the continued exposure of vulnerable populations to SHS. In this context, the present study was undertaken to determine the frequency of passive smoking among patients presenting with stroke. The findings of this study aim to contribute to the evidence base for stroke prevention efforts and inform national tobacco control policies that are urgently needed to protect at-risk populations.

METHODS

This study employed a cross-sectional design and was conducted over a six-month period from July 2024 to December 2024 at the Neurology Department of Pak Emirates Military Hospital, Rawalpindi. Ethical approval was obtained from the Institutional Review Board (IRB) prior to data collection, and all participants provided written informed consent. The study aimed to assess the frequency of passive smoking among patients presenting with acute stroke. A total of 139 patients were enrolled based on a sample size calculation that assumed a 95% confidence level, 7% margin of error, and an expected frequency of passive smoking in stroke patients as 23% (11). The participants were selected through non-probability consecutive sampling. Patients aged 18 to 80 years, of either gender, who were diagnosed with acute stroke and identified as non-smokers were eligible for inclusion. Exclusion criteria included any previous history of stroke, active smoking, or substance abuse. Acute stroke was defined as a sudden onset of focal neurological dysfunction lasting more than 24 hours. Strokes were classified as either ischemic or hemorrhagic. Ischemic stroke was diagnosed through non-contrast computed tomography (CT) of the brain, based on radiological signs such as hypodensity, loss of gray-white matter differentiation, cortical swelling, and sulcal effacement. Hemorrhagic stroke was confirmed by the presence of hyperdense areas indicative of intracranial bleeding on CT imaging.

To determine passive smoking exposure, patients who did not currently smoke cigarettes, cigars, cigarillos, or pipes, and had either never smoked or had quit smoking at least ten years prior, were assessed. Exposure to passive smoking was labeled if they reported

having a household member who regularly smoked in their presence or a coworker who smoked in the same indoor environment for more than a year during the last ten years, in line with the operational definition. Upon recruitment, detailed demographic data, clinical history, and physical examination findings were recorded. All patients underwent non-contrast CT brain scans to confirm stroke and classify the subtype. Information regarding exposure to passive smoking was collected through direct interviews, and stroke-related risk factors—including hypertension, diabetes, dyslipidemia, atrial fibrillation, and cardiovascular disease—were documented. Data analysis was conducted using SPSS version 25.0. The Shapiro-Wilk test was used to assess the normality of quantitative variables, which were found to be non-normally distributed. As a result, variables such as age, symptom duration, and body mass index (BMI) were reported using median and interquartile range (IQR). Categorical variables including age group, gender, stroke type, risk factors, and passive smoking exposure were summarized as frequencies and percentages. To explore associations between variables, data were stratified by age, gender, and stroke type. The Chi-square test was applied post-stratification, with a p-value ≤ 0.05 considered statistically significant.

RESULTS

A total of 139 patients with acute stroke were enrolled in the study. The median age of participants was 51 years with an interquartile range (IQR) of 22 years. The median duration of symptoms was 36 hours (IQR: 13 hours), and the median body mass index (BMI) was recorded as 23.8 kg/m² (IQR: 2.1). In terms of age distribution, 5.8% of patients were between 18 and 30 years, 31.7% were aged 31 to 45 years, 28.8% were between 46 and 60 years, and 33.8% were aged 61 to 80 years. Males represented 56.1% of the sample, while females accounted for 43.9%. Among the participants, 80.6% were diagnosed with ischemic stroke and 19.4% with hemorrhagic stroke. Assessment of cardiovascular and metabolic risk factors revealed that 49.6% of patients had no identifiable risk factor. Hypertension was observed in 18% of cases, diabetes in 13.7%, dyslipidemia in 10.8%, cardiovascular disease in 5.8%, and atrial fibrillation in 2.2% of patients. Passive smoking exposure was identified in 21 patients, accounting for 15.1% of the study population.

Stratification of passive smoking exposure across age groups revealed no cases in the 18–30 years category, while 6.8% of patients aged 31–45 years, 5% of those aged 46–60 years, and 4.3% of patients aged 61–80 years reported exposure. In terms of gender, passive smoking was reported by 9.4% of males and 5.8% of females. Regarding stroke type, 12.2% of ischemic stroke cases and 2.9% of hemorrhagic stroke cases had a history of passive smoking. Chi-square analysis indicated no statistically significant association between passive smoking and age group (p=0.546), gender (p=0.562), or type of stroke (p=0.962). Upon evaluating the relationship between individual vascular risk factors and exposure to passive smoking, it was found that among patients with hypertension, 5 (20.0%) had a history of passive smoking while 20 (80.0%) did not. Similarly, 4 (21.1%) of patients with diabetes and 3 (20.0%) of those with dyslipidemia were exposed to passive smoke. Among those without hypertension, 16 (14.0%) reported passive smoking exposure; this figure was 17 (14.2%) in patients without diabetes and 18 (14.5%) in those without dyslipidemia. However, Chi-square analysis revealed no statistically significant association between passive smoking and the presence of hypertension (p=0.656), diabetes (p=0.664), or dyslipidemia (p=0.858), suggesting that passive smoke exposure occurred across patients regardless of traditional vascular comorbidities.

Table 1: Median (IQR) of Quantitative Variables (n=139)

Variables	Median (IQR)
Age (in years)	51 (22)
Duration of symptoms (in hours)	36 (13)
BMI (in Kg/m ²)	23.8 (2.1)

Table 2: Frequency of qualitative variables (n=139)

Variables	Frequency (percentage)
Age group:	
18 to 30 years	8 (5.8%)
31 to 45 years	44 (31.7%)
46 to 60 years	40 (28.8%)
61 to 80 years	47 (33.8%)
Gender:	
Male	78 (56.1%)
Female	61 (43.9%)
Type of stroke:	
Ischemic	112 (80.6%)
Hemorrhagic	27 (19.4%)
Presence of risk factors:	
None	69 (49.6%)
Hypertension	25 (18%)
Diabetes	19 (13.7%)
Dyslipidemia	15 (10.8%)
Cardiovascular disease	8 (5.8%)
Atrial fibrillation	3 (2.2%)
Passive smoking:	
Yes	21 (15.1%)
No	118 (84.9%)

Table 3: Stratification of passive smoking with respect to age, gender and type of stroke (n=139)

Variables	Passive smoking		p value
	Yes	No	
Age group:			0.546
18 to 30 years	0 (0%)	8 (5.8%)	
31 to 45 years	8 (5.8%)	36 (25.9%)	
46 to 60 years	7 (5%)	33 (23.7%)	
61 to 80 years	6 (4.3%)	41 (29.5%)	
Gender:			0.562
Male	13 (9.4%)	65 (46.8%)	
Female	8 (5.8%)	53 (38.1%)	
Type of stroke:			0.962
Ischemic	17 (12.2%)	95 (68.3%)	
Hemorrhagic	4 (2.9%)	23 (16.5%)	

Table 4: Subgroup Analysis of Risk Factors and Passive Smoking

Risk Factor	Present Passive Smoking	& Present Smoking	& No Passive Smoking	Absent Smoking	& Passive Smoking	Absent Smoking	& No Passive Smoking	p-value
Hypertension	5	20		16		98		0.656
Diabetes	4	15		17		103		0.664
Dyslipidemia	3	12		18		106		0.858

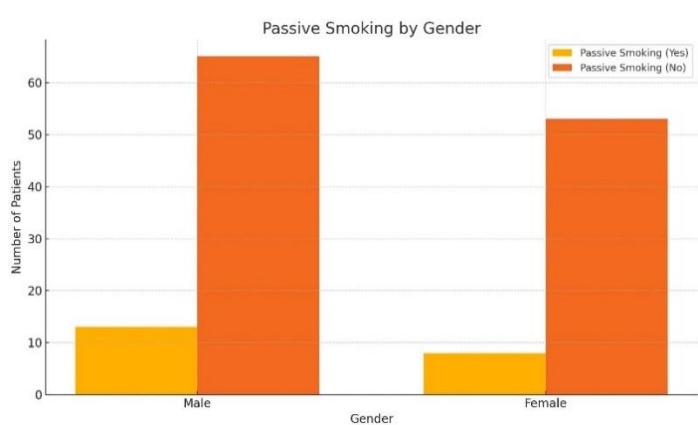


Figure 1 Passive Smoke by Gender

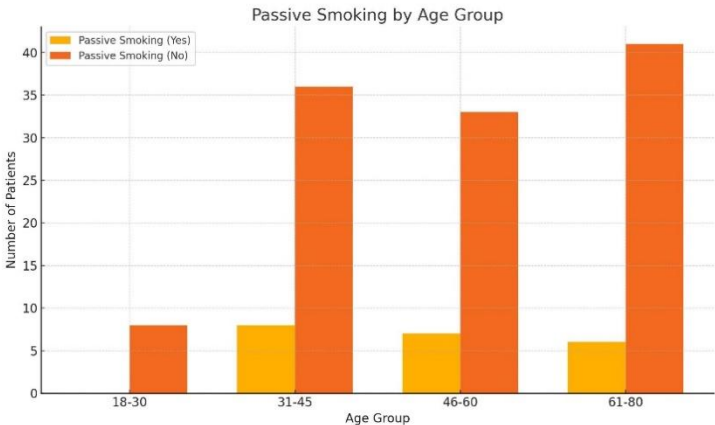


Figure 2 Passive Smoke by Age Groups

DISCUSSION

The present study identified that 15.1% of patients diagnosed with acute stroke were exposed to passive smoking. The majority of these patients were males, predominantly aged between 61 and 80 years, and most commonly presented with ischemic stroke. Hypertension emerged as the most prevalent underlying risk factor, followed by diabetes mellitus. These findings support the growing body of evidence indicating that passive smoking remains an important, yet often overlooked, contributor to stroke burden, particularly in settings where tobacco exposure within domestic environments is still prevalent. Tobacco use continues to rank among the leading preventable causes of premature mortality and disability globally, particularly in the domain of cardiovascular disease. While global smoking prevalence has declined in some regions, several countries have seen rising rates in both men and women, often attributed to lax tobacco control policies and socio-cultural acceptance of smoking indoors (12). Although passive smoke contains different proportions of combustion products compared to active smoke, it retains comparable toxicity and carcinogenic potential, with multiple studies associating it with significant cardiovascular morbidity, including stroke (13–15). Despite the well-established role of smoking in atherosclerosis, thrombosis, and vascular inflammation, there remains limited data on the isolated impact of secondhand smoke exposure among non-smokers in stroke populations (16,17).

Passive smoke exposure can originate from household, occupational, and public environments, with domestic exposure being particularly challenging to control due to the influence of household smoking habits and cultural dynamics (18–20). The current study’s findings are aligned with previously reported frequencies of passive smoking in stroke patients, which have ranged from as low as 6.6% to over 40% in various contexts. Reported figures have varied due to differences in study populations, operational definitions of exposure, and cultural smoking behaviors. While the present study recorded a 15.1% frequency, previous research has reported exposure rates ranging from 6.6% to 42% among stroke patients, reinforcing the variability in exposure patterns and underlining the need for localized data (10–11,19–20). This study's findings underscore the need for robust public health interventions focused on minimizing exposure to secondhand smoke, particularly within households. Public awareness campaigns, community-based behavioral interventions, and stricter enforcement of smoke-free environments may play a pivotal role in lowering this avoidable risk factor. Since

stroke prevention strategies have traditionally focused on direct risk modifiers such as hypertension and diabetes, the inclusion of passive smoking as a targeted domain may yield further reductions in stroke incidence, especially in vulnerable populations.

A major strength of this study lies in its focus on a relatively under-researched yet clinically relevant risk factor—passive smoking—within a defined patient population. The study also benefits from its clear operational definitions and consistent methodology. However, the findings must be interpreted in light of certain limitations. The study was conducted at a single tertiary care center, limiting the generalizability of the results to broader or more diverse populations. The cross-sectional design precludes causal inference, and the reliance on patient-reported exposure to passive smoking introduces potential recall and reporting bias. Furthermore, the absence of biomarker validation (e.g., serum cotinine levels) limits the objectivity of passive smoke exposure assessment. To strengthen future research, larger multicenter studies with diverse geographic representation and more precise quantification of smoke exposure—both in terms of duration and intensity—are warranted. Incorporating objective measures and controlling for additional confounders such as socioeconomic status, environmental pollution, and dietary habits may offer a more comprehensive understanding of how passive smoking influences stroke risk. Overall, the current findings contribute valuable local evidence to an emerging global concern and provide a basis for policy dialogue and preventive strategies aimed at reducing the burden of stroke linked to involuntary tobacco smoke exposure.

CONCLUSION

This study concluded that passive smoking remains a notable concern among patients with stroke, reinforcing its relevance as a preventable yet often underestimated risk factor. Despite the ongoing prevalence of smoking in Pakistan, efforts to shield vulnerable populations—particularly non-smokers—from secondhand smoke are critical. The findings highlight the urgent need for public health strategies that prioritize smoke-free environments through family-focused and community-driven awareness campaigns. By addressing passive smoke exposure alongside traditional stroke risk factors, such initiatives have the potential to significantly reduce the burden of stroke and improve long-term cardiovascular outcomes across the population.

AUTHOR CONTRIBUTION

Author	Contribution
Aida Younis	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Khurram Haq Nawaz	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Syeda Anosha Tariq	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Asif Hashmat	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Anusha Pervaiz	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Munawar Khan	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Inayat Ullah*	Contributed to study concept and Data collection Has given Final Approval of the version to be published

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