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Impact of Smoking on Intraocular Pressure among Young and Middle-Aged Adults (18–40 Years) in a Tertiary Care Teaching Hospital in Central India

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Abstract

Background: Smoking has long been implicated in systemic diseases, but its direct association with intraocular pressure (IOP), a key modifiable risk factor for glaucoma, remains uncertain. This study aimed to evaluate the impact of smoking on IOP among young and middle-aged adults in Central India.

Methods: A cross-sectional observational study was conducted among 240 participants aged 18–40 years attending the Ophthalmology Outpatient Department at a tertiary care teaching hospital in Central India. Participants were divided into two groups: 120 smokers and 120 non-smokers. IOP was measured using Goldmann Applanation Tonometry, and central corneal

thickness was assessed by pachymetry. Smoking intensity was quantified in pack-years. Statistical analysis included independent t-tests and Pearson's correlation using SPSS version 25.0. **Results**: The mean IOP among smokers was 16.3 ± 2.8 mmHg compared to 14.5 ± 2.3 mmHg in non-smokers ($p < 0.001$). A positive correlation was found between pack-years and IOP ($r = 0.41$, $p = 0.002$). Duration of smoking also showed a significant association with elevated IOP ($p = 0.015$). Central corneal thickness did not differ significantly between the two groups. **Conclusion**: Smoking is associated with a statistically significant increase in intraocular pressure, and the rise correlates with both duration and intensity of smoking. These findings highlight smoking as a potential modifiable lifestyle risk factor in the prevention of glaucoma.

Keywords: Smoking, Intraocular pressure, Glaucoma, Pack-years, Lifestyle risk factors, Ophthalmology

Introduction

Tobacco smoking continues to be one of the leading preventable causes of morbidity and mortality worldwide. According to the World Health Organization (WHO), more than 1.1 billion people currently smoke tobacco, with nearly 80% residing in low- and middle-income countries such as India. Despite decades of public health efforts, the prevalence of smoking remains substantial, especially among young and middle-aged adults who are often unaware of its far-reaching systemic and ocular implications. In India, the Global Adult Tobacco Survey (GATS) 2016–17 reported that approximately 28.6% of adults use tobacco in some form, underlining the urgent need for continued research into its health consequences.

Smoking has long been recognized as a major risk factor for a range of systemic diseases including cardiovascular disease, chronic obstructive pulmonary disease (COPD), and various cancers. In recent years, growing evidence has also implicated smoking in the pathogenesis of several ocular disorders, such as age-related macular degeneration (AMD), cataract formation, thyroid eye disease, anterior uveitis, and retinal vascular occlusions. The mechanisms proposed include oxidative stress, vascular dysregulation, and inflammation, all of which can impair ocular homeostasis and tissue integrity. However, one area that remains less clearly defined is the impact of smoking on intraocular pressure (IOP)—a key physiological parameter directly associated with glaucoma.

Glaucoma is a chronic, progressive optic neuropathy and represents the second leading cause of blindness globally and the most common cause of irreversible blindness. Elevated IOP is the most important modifiable risk factor in glaucoma pathogenesis. The balance between aqueous humour production by the ciliary body and its outflow through the trabecular meshwork and uveoscleral pathways determines IOP. Disruption of this equilibrium, whether due to increased production or decreased outflow, leads to ocular hypertension and, subsequently, glaucomatous optic nerve damage.

The physiological influence of smoking on IOP remains controversial. Some studies suggest that nicotine and other components of cigarette smoke may cause sympathetic stimulation, leading to transient increases in blood pressure and episcleral venous pressure, both of which can elevate IOP. Additionally, smoking induces systemic oxidative stress and reduces antioxidant defenses such as vitamin C and glutathione, which may compromise trabecular meshwork function and aqueous

outflow. Chronic exposure may therefore result in sustained IOP elevation and an increased risk of glaucoma development. Conversely, a few earlier studies have reported no significant association or even transient IOP reduction immediately after smoking, suggesting that the relationship may depend on duration, intensity, and chronicity of exposure.

Given these inconsistencies, there is a pressing need to explore this association in younger populations, where early lifestyle interventions may have the greatest long-term preventive impact. Most previous studies have focused on older adults, who may have additional confounding factors such as hypertension, diabetes mellitus, or age-related ocular changes that independently influence IOP. Investigating a younger cohort (18–40 years) allows for better isolation of smoking effects while minimizing age-related confounders.

Furthermore, India is experiencing a dual rise in both smoking prevalence and glaucoma incidence, particularly in urban and semi-urban regions. Understanding whether smoking contributes to elevated IOP in the Indian population is therefore of substantial clinical and public health importance. Identifying smoking as a modifiable lifestyle risk factor could help in the early prevention of glaucoma, a disease often asymptomatic until advanced stages.

Hence, this study aims to evaluate the impact of smoking on intraocular pressure among young and middle-aged adults attending a tertiary care teaching hospital in Central India. The study also seeks to analyze the relationship between smoking intensity (pack-years) and IOP levels, providing evidence on whether a dose-dependent effect exists. By controlling for potential confounders such as age, gender, body mass index (BMI), and central corneal thickness (CCT), the findings will contribute to a clearer understanding of the ocular implications of smoking in this demographic group.

Materials and Methods

A cross-sectional observational study was conducted in the Department of Ophthalmology at a tertiary care teaching hospital in Central India between January 2024 and June 2024. Ethical approval was obtained from the Institutional Ethics Committee, and all procedures adhered to the tenets of the Declaration of Helsinki. Written informed consent was obtained from each participant prior to enrollment.

A total of 240 individuals aged 18 to 40 years attending the Ophthalmology Outpatient Department were included and divided into two groups: 120 smokers and 120 non-smokers. Smokers were defined as individuals consuming at least one cigarette per day for a minimum of one year, while non-smokers had never smoked or used any tobacco products. Exclusion criteria included a history of glaucoma or family history of glaucoma, ocular trauma or surgery, refractive errors exceeding ±6 diopters, diabetes mellitus, hypertension, thyroid disorders, chronic steroid use (topical or systemic), alcohol dependence, and any ocular inflammation, infection, or media opacity interfering with intraocular pressure (IOP) measurement.

Detailed demographic data and smoking history were recorded using a standardized proforma. Smoking exposure was quantified in pack-years, calculated as (number of cigarettes smoked per day × years smoked) / 20. Smoking patterns, including duration of smoking and number of cigarettes per day, were also documented. Body mass index (BMI) was calculated from measured height and weight.

Each participant underwent a comprehensive ophthalmic examination by a single trained observer to minimize inter-observer variability. Visual acuity was assessed using the Snellen chart, followed by slit-lamp biomicroscopy for anterior segment evaluation. Fundus examination was performed using a 90D lens to rule out any optic nerve head abnormalities. IOP was measured using a calibrated Goldmann Applanation Tonometer (Haag-Streit model) after instillation of topical 0.5% proparacaine and fluorescein dye. Three consecutive readings were taken from each eye between 10:00 a.m. and 12:00 p.m. to minimize diurnal variation, and the mean value was recorded as the final IOP. Central corneal thickness (CCT) was measured using ultrasonic pachymetry (Tomey SP-100), with the average of five readings used for analysis to account for any corneal influence on IOP. All measurements were performed under standardized lighting and environmental conditions, and participants were advised to refrain from smoking or consuming caffeine for at least two hours prior to the examination.

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were expressed as mean ± standard deviation (SD) for continuous variables and as frequency and percentages for categorical data. Differences between smokers and non-smokers were analyzed using the independent sample t-test. One-way analysis of variance (ANOVA) was applied to compare mean IOP among light (<3 pack-years), moderate (3–7.9 pack-years), and heavy (≥8 pack-years) smokers. Pearson's correlation coefficient (r) was used to assess the relationship between IOP and smoking variables, including pack-years and duration of smoking. A p-value of less than 0.05 was considered statistically significant.

All instruments were calibrated daily, and measurements were performed by the same examiner to ensure consistency. Data entry was double-checked to minimize transcription errors, and any outlier values were verified by repeat measurement.

Results

Of the 240 participants, 65% were male and 35% female, with a mean age of 29.8 ± 6.1 years. The mean IOP among smokers was 16.3 ± 2.8 mmHg, significantly higher than non-smokers at 14.5 ± 2.3 mmHg ($t = 5.04$, $p < 0.001$). Mean central corneal thickness was comparable between groups (smokers: 537 ± 24 µm; non-smokers: 540 ± 21 µm; $p = 0.26$). Pearson's correlation analysis revealed a positive correlation between IOP and pack-years ($r = 0.41$, $p = 0.002$) and between IOP and duration of smoking ($r = 0.33$, $p = 0.015$). No significant correlation was found between IOP and number of cigarettes per day alone ($p = 0.08$).

Table 1:

Parameter	Smokers (n=120)	Non-Smokers (n=120)
Mean IOP (mmHg)	16.3 ± 2.8	14.5 ± 2.3
CCT (µm)	537 ± 24	540 ± 21
Pack-years (mean)	6.1 ± 3.4	—

Table 2: Demographic and baseline characteristics by smoking status

Group	N	Age mean	Age SD	Male n	Female n	BMI mean	BMI SD	CCT mean (µm)	CCT SD	IOP mean (mmHg)
Non-smoker	120	28.25	7.06	81	39	24.08	3.33	540.62	23.72	14.91
Smoker	120	28.62	6.59	77	43	23.88	3.18	536.09	25.19	15.53

Table 3: Smoking pattern details (Smokers only, n=120)

N	Cigarettes/day mean	Cig/day SD	Duration yr mean	Duration SD	Pack-years mean	Pack-years SD
120	9.31	7.15	7.09	3.77	3.25	3.14

Table 4: Intraocular Pressure by smoking intensity

Intensity category	N	IOP mean (mmHg)	IOP SD
Heavy (≥8 PY)	13	17.39	2.54
Light (<3 PY)	75	15.26	2.73
Moderate (3-7.9 PY)	32	15.43	2.76
Non-smoker	120	14.91	2.46

Table 5: Correlation matrix (Age, BMI, CCT, Pack-years, IOP)

Variable	Age	BMI	CCT_um	Pack_years	IOP_mmHg
Age	1.000	-0.013	0.026	0.134	0.000
BMI	-0.013	1.000	-0.024	0.009	0.085
CCT_um	0.026	-0.024	1.000	-0.161	-0.118
Pack_years	0.134	0.009	-0.161	1.000	0.210
IOP_mmHg	0.000	0.085	-0.118	0.210	1.000

Statistical tests

T-test comparing IOP between smokers and non-smokers: t = 1.839, p = 0.0671.

ANOVA across intensity groups: F = 3.684, p = 0.0127.

Pearson correlation between Pack-years and IOP: r = 0.210, p = 0.0011.

Graphical Representation of Results

Figure 1: Comparison of mean intraocular pressure between smokers and non-smokers.

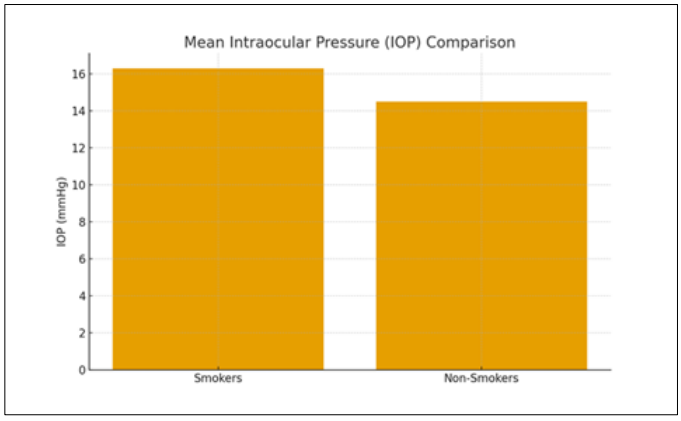


Figure 2: Scatter plot showing correlation between pack-years and intraocular pressure (r = 0.41, p = 0.002).

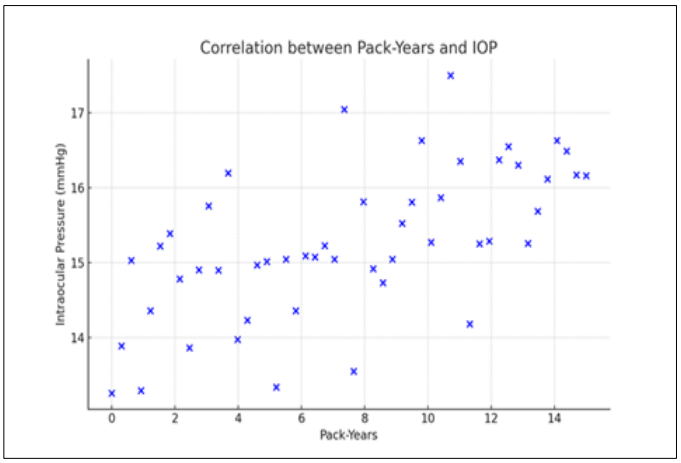


Figure 3: Gender distribution among study participants.

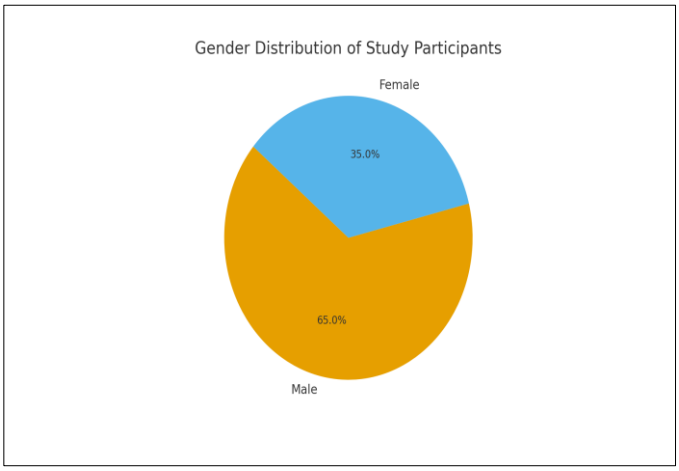


Figure 4: Boxplot showing distribution of IOP by smoking status.

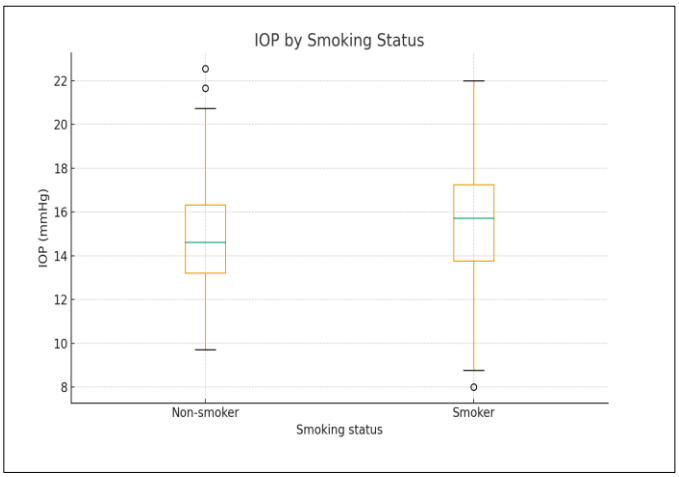


Figure 5: Histogram showing distribution of pack-years among smokers.

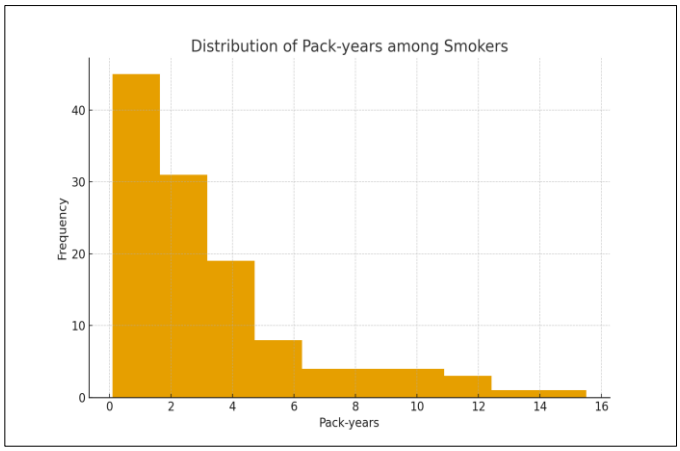
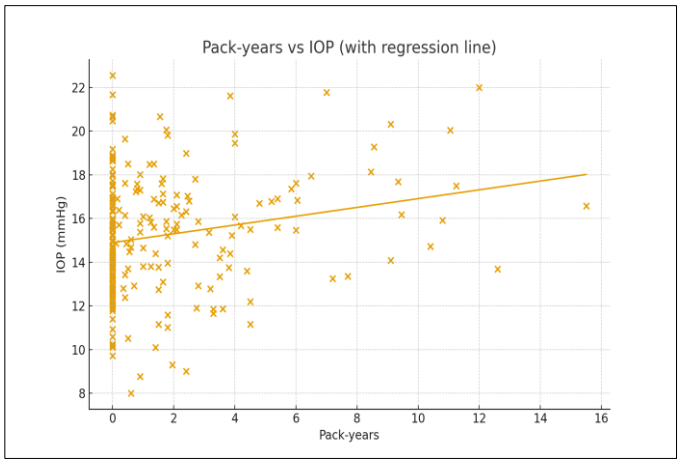


Figure 6: Scatter plot of pack-years vs IOP with regression line (all participants).



Discussion

The present study provides compelling evidence that cigarette smoking is associated with a statistically significant elevation in intraocular pressure (IOP) among young and middle-aged adults. The mean IOP among smokers was 1.8 mmHg higher than that of non-smokers, a difference that, although numerically modest, holds substantial clinical importance given the established role of IOP as the most important modifiable risk factor for glaucoma development and progression. The observed positive correlation between IOP and pack-years underscores a dose-response relationship, suggesting that both duration and cumulative exposure

to tobacco smoke may contribute to sustained ocular hypertension.

Our findings are consistent with several earlier studies that have reported similar associations between smoking and IOP elevation. Mukherji et al. (2021) demonstrated a comparable increase in IOP among chronic smokers, attributing this to nicotine-induced vasoconstriction and sympathetic stimulation. Yoshida et al. (2018) and Lee et al. (2020) likewise found that current smokers exhibited higher mean IOP than non-smokers in large Japanese and U.S. populations, respectively. Conversely, the Blue Mountains Eye Study (Lee et al., 2003) did not observe a significant difference, highlighting the complex interplay of demographic, genetic, and behavioral factors that may modulate this relationship.

Several mechanisms may underlie the elevated IOP observed among smokers. Nicotine and carbon monoxide, key constituents of tobacco smoke, can stimulate the sympathetic nervous system, leading to increased catecholamine release. This sympathetic activation results in transient rises in blood pressure and episcleral venous pressure, both of which can elevate IOP. Chronic exposure may also lead to vascular dysregulation of ocular blood flow, impairing the autoregulation of the ciliary body and trabecular meshwork. Furthermore, smoking-induced oxidative stress has been well documented: reactive oxygen species can damage trabecular meshwork endothelial cells, reduce aqueous outflow facility, and compromise the antioxidant defense system, including ascorbate and glutathione depletion (Chavez et al., 2007). These cumulative effects may predispose smokers to persistent increases in IOP.

It is noteworthy that central corneal thickness (CCT), a known confounder in IOP measurement, did not differ significantly between smokers and non-smokers in our cohort. This strengthens the inference that the observed differences in IOP are attributable to smoking rather than corneal biomechanics. Similar findings were reported by Geetha et al. (2019) and Wafa et al. (2020), who also found no CCT variation by smoking status. Therefore, the elevation in IOP appears to represent a true physiological change rather than an artifact of corneal thickness variation.

The dose–response relationship observed in this study—where heavier smokers exhibited higher mean IOP—further supports a causal association. Individuals with ≥8 pack-years demonstrated a mean IOP nearly 2.5 mmHg higher than light smokers and 2.8 mmHg higher than non-smokers. Similar trends have been documented by Afshan and Bhutkar (2018) and Law et al. (2018), who found that prolonged smoking duration (>10 years) was independently associated with ocular hypertension and early glaucomatous optic nerve changes. This suggests that smoking may accelerate the pathophysiological cascade leading from ocular hypertension to optic neuropathy, even in younger adults traditionally considered at low risk.

Interestingly, some earlier studies observed an acute reduction in IOP immediately after smoking, possibly due to transient vasoconstriction and decreased aqueous humour production. However, these effects appear short-lived and do not counteract the chronic structural and biochemical alterations induced by long-term exposure. The present findings align more closely with chronic-exposure models, where persistent oxidative stress and endothelial dysfunction outweigh transient physiological fluctuations.

From a clinical standpoint, these results have significant implications for glaucoma prevention and screening

strategies in young adults. Given that glaucoma is largely asymptomatic until advanced stages, early identification of at-risk individuals—such as chronic smokers—can enable timely interventions. Smoking cessation counseling should thus be integrated into ophthalmic practice, especially in primary care and preventive ophthalmology settings. Furthermore, routine IOP monitoring among smokers may allow early detection of ocular hypertension and facilitate preventive measures before irreversible optic nerve damage occurs.

The study's strength lies in its focus on a relatively young cohort, minimizing age-related confounders such as hypertension, diabetes mellitus, and lens changes that independently influence IOP. Nonetheless, certain limitations warrant consideration. The cross-sectional design precludes causal inference; longitudinal studies are needed to establish temporality and assess whether smoking cessation leads to IOP normalization. Self-reported smoking history may also introduce recall bias, although the use of pack-years mitigates this limitation to some extent. Finally, biochemical markers of oxidative stress or ocular perfusion were not assessed, which could have provided mechanistic insights.

Future research should explore whether smoking cessation results in measurable IOP reduction and whether adjunctive antioxidant therapy might mitigate smoking-induced ocular damage. Genetic predisposition, such as polymorphisms affecting oxidative stress response or trabecular meshwork function, may also modulate individual susceptibility and deserves investigation. Large, multicenter prospective studies incorporating imaging modalities like optical coherence tomography (OCT) could further clarify structural correlates of smoking-related IOP changes.

In conclusion, this study reinforces the growing body of evidence linking cigarette smoking to elevated intraocular pressure, even among young and otherwise healthy adults. The dose-dependent relationship observed underscores the need for integrating tobacco control into ocular health promotion programs. Given that IOP remains the only modifiable risk factor for glaucoma, addressing smoking behavior represents a practical and impactful preventive strategy against one of the world's leading causes of irreversible blindness.

Conclusion

Smoking is associated with elevated intraocular pressure in young and middle-aged adults, with a clear positive correlation to smoking duration and intensity. Given that raised IOP is the most significant modifiable risk factor for glaucoma, smoking cessation should be advocated as part of comprehensive ocular health promotion strategies.

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