

The effect of smoking on ocular surface and tear film based on clinical examination and optical coherence tomography

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Purpose: Smoking is a global health problem with a high burden of smokers in India and the world as well. Smoking is a known eye irritant that may have a significant effect on the anterior ocular surface. Hence, the present study was aimed to investigate the effect of smoking on dry eye parameters, ocular surface, and central corneal thickness (CCT) and to compare the results with nonsmokers. **Methods:** In this cross-sectional study, 40 (80 eyes) smokers and 40 (80 eyes) age-matched nonsmokers were included. The severity of ocular surface disease symptoms was assessed by the Ocular Surface Disease Index (OSDI) tool. All participants underwent a series of measurements, that is, the Schirmer's II test score and tear breakup time (TBUT). Tear meniscus height (TMH) and CCT were measured using anterior segment optical coherence tomography (OCT). **Results:** The mean value of all dry eye parameters was statistically different among the group of smokers and nonsmokers. The mean score of symptomatic assessment (OSDI) was observed to be significantly higher in smokers than in nonsmokers ($P < 0.0001$), and dry eye parameters, that is, Schirmer's score ($P = 0.0127$), TMH ($P < 0.0001$), and TBUT ($P < 0.0001$) were significantly lower among smokers than nonsmokers. There was no significant difference in CCT between smokers and nonsmokers ($P = 0.06$). **Conclusion:** Cigarette smoking has deteriorating effects on the ocular surface. Exposure to smoking was found to be associated with dryness of the eye. Further studies are needed on a molecular basis.

Key words: Cigarette smoking, dry eye, ocular surface

Cigarette smoking is a global problem. According to the 2018 World Health Organization report, there are approximately 120 million smokers in India, which is home to 12% of the world's smokers, and by 2015, the number of men smoking tobacco in India rose to 108 million, which is an increase of 36% between 1998 and 2015.^[1]

Dry eye is a multifactorial disease of the tears and ocular surface, which results in symptoms of discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface.^[2] Dry eye is one of the most common ophthalmological problems,^[3] and it is estimated that up to one third of the population worldwide may be affected.^[4,5] The effect on the quality of life is substantial because of symptoms such as pain and irritation, which have a negative effect on ocular health, general health, and well-being.^[3,6]

As tobacco smoke is also a known eye irritant, it worsens dry eye, which is defined as insufficient tears on the eye's surface, which are needed to keep the eye lubricated and healthy.^[2] In dry eye, reduced goblet cell density and changes in mucin amount, distribution, and glycosylation have been reported.^[7,8]

There is less evidence on the effects of tobacco smoke on the eye, especially regarding anterior ocular surface-related pathology, especially in the Indian context.^[9] The purpose of this study was to find out the effect of smoking on dry eye parameters, ocular surface, and central corneal thickness (CCT) and also to compare the results with nonsmokers.

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Methods

The cross-sectional observational study was conducted among smokers and nonsmokers presenting at the tertiary care hospital from March 2017 to October 2018. A total of 40 patients (80 eyes) who had smoked for at least one continuous year and were active smokers at the time of the study were selected for the study. Smokers were classified by the magnitude of smoking as light smokers (<20 cigarettes in a day) and heavy smokers (>20 cigarettes in a day). Age-matched 40 nonsmokers with no history of passive smoking were selected as the control group. Approval for the study was granted by the ethical committee of S. N. Medical College, and informed consent was obtained from all the participants. Patients using contact lenses; having a history of any ocular surgery; having specific occupation associated with dry eye and refractive errors (ametropia of more than 2 diopters); on medications causing dry eye such as antiglaucoma drugs, vasoconstrictors, and antihistamines; having ocular surface disorders such as herpes simplex virus infection, varicella zoster virus infection, and Stevens-Johnson syndrome; on artificial tears; and having chronic allergic eye disease were excluded from the study.

A detailed medical and ocular history was taken for smokers, including duration and number of cigarettes smoked per day, and for nonsmokers, including a history of passive smoking. The history taking also included a questionnaire

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for Ocular Surface Disease Index (OSDI) scoring. The OSDI is a validated, self-administered instrument for assessing the presence and severity of ocular surface disease symptoms. The OSDI questionnaire includes 12 questions about the respondent's past week experience with the ocular symptoms, vision-related function, and environmental triggers. The total OSDI^[10] score ranged from 0 to 100. The scores were classified as follows: ≤ 12 as normal, 13 to 22 as mild, 23 to 32 as moderate, and ≥ 33 as severe. Best-corrected visual acuity using Snellen's chart was taken to fulfill the inclusion criteria. After 10 minutes, Schirmer's II test was done, and the eyes were graded based on the wetting as normal if >10 mm, mild to moderate if within 6 to 10 mm, and severe if within 0 to 5 mm.^[11] Following this, tear meniscus height (TMH) was measured. It acts as an indicator of tear volume because it forms 75% to 90% of it.^[12] TMH was measured using anterior segment optical coherence tomography (OCT) RTVue software Version 2.0.4.0 and model RT 100 [Fig. 1]. All patients were asked to avoid using any eye drops at least 1 hour prior to the OCT examination. All measurements were performed three times, and the mean value was recorded by a single examiner. All tests were performed in a dim-lit room between 21°C and 25°C with regulated humidity to avoid reflex tearing. TMH was measured from the inferior tear meniscus in μm . Dry eye was graded as mild, moderate, and severe if values observed were >0.7 mm, 0.2 to 0.7 mm, and <0.2 mm, respectively. Following TMH, tear breakup time (TBUT) was assessed. For TBUT assessment, the patient was seated on a chair facing the slit lamp. Prepacked sterile fluorescein strip (manufactured by Contacare Ophthalmics & Diagnostics, Vadodara, Gujarat) impregnated with 1 mg of fluorescein sodium, moistened with a drop of preservative-free antibiotic was gently touched onto the inferior palpebral conjunctiva to stain the tear film. Care was taken to instill approximately the same amount of fluorescein into each eye. The patient was asked to blink a few times. Then he or she was asked to open the eyes on request and to keep looking straight without blinking. The tear film was then scanned under a slit lamp with a Cobalt-blue filter, while the patient was asked not to blink. The time between the last blink and the appearance of the first randomly appearing dark spot was measured. This procedure was reported three times, and the average value in seconds was recorded in each eye. This tear breakup time is a measure of tear film stability. If the dark spot appeared at the same place every time, it was considered to be due to some ocular surface irregularity and not due to dry eye per se. The eyes were classified as normal, marginal, or low grades of dry eye based on the breakup times of >10 s, 5 to 10 s, or <5 s, respectively.^[13] Even a single spot was considered and marked as clinically significant. RTVue (software Version 2.0.4.0 and model RT

100), a spectral-domain OCT, was used for calculating CCT. Five pachymetry scans were taken for each eye in both the cohorts of this study, with a gap of 5 to 10 seconds between each scan. During this gap, participants were repositioned and allowed to blink. All the pachymetry scans were taken in the central 2-mm zone, and the mean pachymetry value in this zone was taken as the CCT. The readings were classified as less, marginal, or normal, if thickness was found to be ≤ 520 μm , 521 to 570 μm , or >570 μm , respectively.

Data were entered into Microsoft Excel, and analysis was carried out using Epi Info software package Version 6.04 (Centers for Disease Control and Prevention, Atlanta, Georgia, USA). Mean values of basic profile and different dry eye parameters were compared by using paired *t* test.

Results

This study comprised 80 participants: 40 smokers and 40 age-matched nonsmokers. The mean age of the smoker group was 39.00 ± 14.95 years, and the mean age of the nonsmoker group was 35.10 ± 15.39 years [Table 1]. There was a significant preponderance of men among smokers ($n = 38$, 95%) ($P < 0.001$). In a detailed history taken among smokers, it was revealed that the duration of smoking for almost half of the smokers (50%) ranged from 1 to 10 years, and out of 40 smokers, four were heavy smokers. Best-corrected visual

Table 1: Age and sex distribution of study participants

	Smokers		Non smokers		P
	Mean	SD	Mean	SD	
Age (years)	39	14.9	35	15.4	0.24
	<i>n</i>	%	<i>n</i>	%	
Sex					
Male	38	95%	23	57.5%	0.00008
Female	2	5%	17	42.5%	

Table 2: Comparison of dry eye parameters between smokers and nonsmokers

Variable	Smokers		Nonsmokers		P
	Mean	SD	Mean	SD	
Schirmer's II Test	22.30	12.61	28.85	10.24	0.0127
Tear Meniscus Height	0.23	0.06	0.36	0.12	<0.0001
Tear Breakup Time	9.69	3.96	12.80	1.93	<0.0001

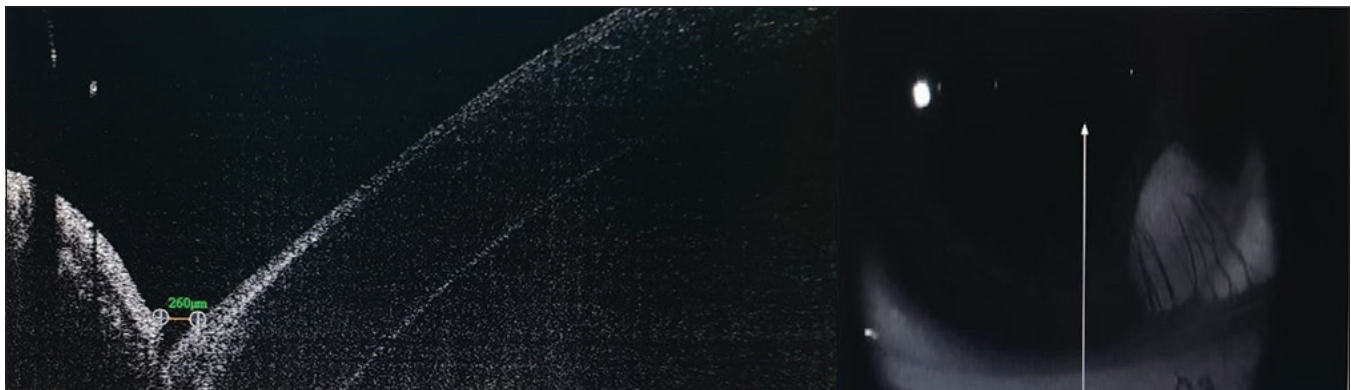


Figure 1: OCT image showing tear meniscus height

acuity was taken, and none of the participants had ametropia of more than 2 diopters.

Out of 80 eyes of smokers, 24 eyes (30%) had Schirmer's scores <10 mm, whereas 10 eyes (12.5%) of nonsmokers had Schirmer's scores less than <10 mm. Mean Schirmer's scores were lesser in smokers (22.3 ± 12.61 mm) compared with nonsmokers (28.8 ± 10.24 mm) [Table 2]. The difference in Schirmer's scores was found to be statistically significant ($P = 0.0127$).

Out of 80 eyes of smokers, 34 eyes (42.5%) had TBUT <10 s, whereas 18 eyes (22.5%) of nonsmokers had TBUT <10 s. Mean TBUT was less in the smokers (9.69 ± 3.96 s) than in nonsmokers (12.8 ± 1.93 s) [Table 2]. The difference was found to be statistically significant ($P < 0.0001$).

Out of 80 eyes of smokers, 38 eyes (47.5%) had TMH less than 0.2 mm, whereas 12 eyes (15%) of nonsmokers had TMH less than 0.2 mm. Mean TMH was lower among smokers (0.23 ± 0.06 mm) than in nonsmokers (0.36 ± 0.12 mm) [Table 2]. The difference was found to be statistically significant ($P < 0.0001$).

Severe OSDI score of >32 was seen among 10 smokers (26.32%), whereas none of the nonsmokers had OSDI score >32. Mean OSDI was almost five times higher in smokers (20.8 ± 15.2) than in nonsmokers (4.03 ± 2.6) [Table 3]. The difference was found to be statistically significant ($P < 0.0001$).

Out of 80 smokers' eyes, 48 eyes (60%) had CCT in the range of 521 to 570 μm. Similarly, for the nonsmokers too, maximum patients, that is, 54 eyes (67.5%) had CCT in the range of 521 to 570 μm. Mean CCT was lower in smokers than in nonsmokers, but the difference found was statistically insignificant ($P = 0.06$) [Table 3].

Discussion

Dry eye disease (DED) is one of the commonest ocular problems characterized by a vicious cycle of tear film instability, hyperosmolarity, chronic inflammation, and neurosensory abnormalities, which subsequently leads to ocular surface damage.^[14] Dry eye is associated with many risk factors among which lifestyle factors play an important role.^[15] Smoking is already known as an important risk factor for many chronic diseases; however, it is still an unclear risk factor for dry eye.^[15] The Blue Mountains Eye Study reported that cigarette smoking could be a significant risk factor for dry eye.^[16] Similarly, there are studies that also showed an association between smoking and dry eye risk.^[17,18]

In the present study, it was found that smoking was less prevalent among females in the study area; this might be a possible reason for the huge difference among male and female smokers. The study also showed that smoking has statistically significant detrimental effects on the precorneal tear film and ocular surface. Similar to the present study, some authors observed that cigarette smoking had a harmful effect on tear film stability and ocular surface.^[19-21] Schirmer's score and TBUT were found lower among smokers than nonsmoker in the present study. Similarly, previous studies also found values lower among smokers.^[9,22,23]

The main constituents of cigarettes are heavy metals and toxic minerals, which are known to contain 4,000 chemicals.^[24] The toxic components in cigarettes lead to damage primarily in the respiratory and circulatory systems, and in several other organs, including the eyes.^[25,26]

Smoke particles, nicotine, and harmful gases such as carbon monoxide damage the tissues in the eye by causing vasospasm and platelet aggregation. Nicotine triggers inflammation by stimulating macrophages, and ocular surface inflammation causes apoptosis. It has been previously determined that in smokers, inflammation is triggered on the ocular surface and the lipid layer of the tear film that is expressed from the meibomian glands.^[23,27] In a study by Altinors *et al.*,^[23] the stability of the tear lipid layer was examined with DR-1 lipid layer interferometry, and it was found that there was no lipid distribution on the ocular surface in the cigarette-smoking group. This impairment of the lipid layer in smokers could be a marker of meibomian gland destruction, which could lead to evaporative DED.^[28]

In reaction to these agents, the lipid layer on the surface tries to increase tear film stability by preventing evaporation of the aqueous component of tears.^[29]

Cigarette smoking has also been shown to increase the production of proinflammatory cytokines such as tumor necrosis factor alpha and interleukin (IL)-1, IL-6, and IL-8 and suppress the production of anti-inflammatory cytokines such as IL-10.^[30] It has been suggested that by triggering inflammatory reactions in the meibomian glands and ocular surface, cigarette smoking can lead to meibomian gland disorder, thereby leading to DED.

In the present study, TMH was found less among smokers than nonsmokers. Uchino *et al.*^[31] concluded that tobacco smoking might decrease tear secretion and goblet cell density in the conjunctiva of office workers. It was also determined in another study that in chronic cigarette smokers, there is a significant decrease in goblet cell density on the ocular surface and an increase in squamous metaplasia.^[19,27]

The current findings could also be explained based on two main theories: ischemic theory and antioxidant theory.^[32] The toxins associated with smoking induce ischemic changes, including decrease in blood flow or formation of a clot within ocular capillaries. These ultimately lead to a lack of nutrition essential for eye cell physiology.^[26,33] The lack of antioxidants emerging from peroxidation can lead to a disturbance in the normal functionality of the ocular tissue. Free radicals that are produced because of cigarette smoking cause oxidative stress in proteins, lipids, and cell DNA.^[34] Consequently, the cells cannot function normally and ocular diseases develop.^[35]

Among study participants, the severity of ocular surface disease symptoms was assessed by using the OSDI score. Similar to the present study, a comparative study conducted among smokers and nonsmokers by Mohidin and Jaafar^[36] also found that a higher proportion of smokers had severe OSDI scores than nonsmokers.

Conjunctival mucosa is highly sensitive to chemicals, smoke, and irritative gases in the air originating from cigarette smoking. The free nerve ends on the ocular surface are stimulated by these gases, causing stinging, conjunctival redness, and excessive lacrimation.^[17,35,37] Cigarette smoking causes irritative eye complaints such as itching, burning, and stinging. These could explain the higher OSDI score among smokers. In the present study, mean CCT was lower among smokers than nonsmokers, but the difference was statistically not significant. The findings of the present study are comparable with a study conducted by Sayin *et al.*^[38] in which smoking was found not to be related to CCT. Wang *et al.*^[39] found evidence that smoking could be correlated with decreased CCT in primary open-angle

Table 3: Comparison of ocular symptoms and central corneal thickness between smokers and nonsmokers

Variable	Smokers		Nonsmokers		P
	Mean	SD	Mean	SD	
OSDI	20.81	15.18	4.03	2.59	<0.0001
Central corneal thickness	509.3	44.48	525.15	28.15	0.0606

OSDI=Ocular Surface Disease Index.

glaucoma. They hypothesize that cigarette smoking may exert this effect through hypoxia and collagen in the cornea, so smoking probably affects the biosynthesis of collagen and extracellular matrix turnover, which could explain a decreased corneal thickness. However, in our study, we did not notice a significant difference in baseline ocular pachymetry readings between chronic smokers and nonsmokers.

The limitations of our study were a small sample size, not using lissamine staining, and lack of other tests to assess inflammatory cytokines of the tears and ocular surface, which could have given a more detailed result.

Conclusion

In conclusion, cigarette smoking has deteriorating effects on dry eye parameters such as Schirmer's score, TBUT, and TMH. However, CCT was found not related to smoking. Further studies are needed to determine the correlation between smoking magnitude and effect on the ocular surface and corneal endothelium on a molecular basis in terms of free radicals.

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Conflicts of interest

There are no conflicts of interest.

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