e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com

International Journal of Pharmaceutical Quality Assurance 2026; 17(1); 47-53

Original Research Article

Cytological, Functional and pH Alteration of Nasal Mucosa in Smokers: A Case Control Study

Anju Singh[1], Uma Garg[2], Surender Kumar[3], Anand Krishnan[4], Swaran Kaur[5]

[1,2,3,4,5]BPS Government Medical College for Women, Khanpur Kalan, Haryana, India

Received: 25-10-2025 / Revised: 23-11-2025 / Accepted: 26-12-2025
Corresponding Author: Dr. Anand Krishnan
Conflict of interest: Nil

Abstract:

Objective: To study the effect of smoking on the cytology by analyzing the cytopathological changes and function by determining MCC time and pH of nasal mucosa of smokers.

Methods: A tertiary care hospital based case control study done in 140 patients in the age group of 18-60 years with no symptoms pertaining primarily to nose over one year. 70 patients each were selected as smokers and non-smokers. pH of the nasal mucosa nasal mucosal clearance time were measured followed by procuring the nasal mucosa sample using a sterile small nylon brush for measurement of ciliated cells. All the tests were done in the same nasal cavity which was comparatively wider than the other.

Results: The study observed a statistically significant prolongation in the mucociliary clearance time and reduction in the ciliated cell count in smokers. But no significant difference in pH of the nasal mucosa was observed between the two groups. A positive correlation was found between the pack years and the mucociliary clearance time. There was a significant difference in the mean MCC time among different types of smokers with persons smoking both beedi and hookah showing the maximum MCC time.

Conclusion: Chronic exposure to tobacco smoke causes structural and functional changes in the nasal mucociliary system. These changes were found to be more in persons with high amount of exposure to smoke in terms of pack years. Bidi and hookah smokers are at much higher risk since they show maximum functional changes.

Keywords: Ciliated Cell Count, Mucociliary Clearance, Nasal Mucosa, Smoking, Tobacco.

DOI: 10.25258/Ijpqa.17.1.9

Introduction

Tobacco use, either in smokeless or smoking forms, is a serious contributor to global morbidity and mortality. Smoking tobacco for a long period leads to alterations in the structure or the functioning of the airway system [1]. Tobacco smoke is a composite of many substances which destroys cilia in vitro and can cause ciliostasis in nasal or lower respiratory pathways [2,3].

Chronic exposure of tobacco may lead to a decrease both ciliated cells (CCs) and cilia, mucosal metaplastic changes like enlargement and increase in goblet cell count, other cells involved with inflammatory mechanism such as eosinophils, polymorphonuclear leukocytes and cytotoxic T lymphocytes and consequent increase in upper airway secretion [4]. According to data from the global adult tobacco survey (2016-17), 10.7% of adult Indian population smoke tobacco daily [5]. Inspite of the awareness campaigns in media and dreadful pictures on the pack about the detrimental effects of smoking, very little portion of people are aware about smoking and its harmful effects. Cytology has proven to be a significant tool with scientific and clinical application over the past years. Nasal mucosal scrapings are examined under microscope to analyse the characteristics and is now utilised in diagnosing and in follow up of both allergic and non-allergic nasal disorders [6]. Pathologic examinations of the nasal mucosa by brush cytology has received little attention by ENT specialists but is a practical method that is simply executed, relatively inexpensive to process compared with other techniques and reproducible [7]. Nasal Mucosa is rich in goblet cells, ciliated cells and mucus and serous secretory glands.

The secretions from these forms continuous sheet called mucous blanket is then transported along with the trapped matter by the ciliated cells beating at a frequency of 12-15hz to the pharynx where it is swallowed [8]. A noticeable decline in the ciliary beat frequency was noticed in several studies of tracheobronchial and nasal mucosal samples exposed to tobacco smoke [9,10,11]. Inhaled particles are cleared of the human airways and lungs by a defence mechanism called Mucociliary Clearance or MCC [12]. Chronic exposure to tobacco smoke

impairs mucociliary clearance by inducing remodelling of epithelium, which causes damage to the structure of cilia, promotes metaplastic changes to goblet cell, and leads to mucous cell hypertrophy, ultimately producing more mucous [13]. Certain medicines (Acetylcholine, Adrenaline, Corticosteroids) and other factors such as temperature and ageing are noted to influence mucociliary clearance time [12]. Defects to the cilial structure or mucosal function cause alterations in mucociliary clearance in nose. Elements like physical characteristics of mucous, interaction of mucous and cilia and the beat frequency of the cilia contribute to MCC [14]. Therefore, tobacco smoke serves as major risk factor respiratory airway diseases, as it leads to destruction of cilia-bearing cells or the cilia itself and disrupt the physiological mucociliary flow [15]. Measuring the nasal mucociliary transit time is a simple, cheap and reproducible method to assess NMC which can also predict the tracheobronchial clearance [16]. Upper and lower airway MCC can be measured by different techniques. The in vivo measures are done using calculation of transit time using substances like saccharin, several dyes or radiolabeled particles [12]. Researchers have shown sustained interest in nasal mucosal pH for more than fifty years, with studies dating back to 1941 reporting that the normal nasal mucosal pH ranges between 5.5 and 6.5 [17]. It is a physiological characteristic that can influence both specific and non-specific defence mechanisms of the nose. Therefore, as part of examining physiological factors that may affect nasal mucosal function, we investigated the pH of nasal nasal mucosa [18]. Most of the studies observed the consequences of exposure to tobacco smoke on either the cytological component alone or most of them have done on functional alone but in our study we have gone for the holistic approach taking all the parameters namely cytological, functional and physiological all together.

Materials and Methods

Ours is a case control study conducted in the Oto-rhinolaryngology department at a tertiary hospital over a timespan of one year from January 2020. The study was conducted in 140 patients divided in two groups of smokers and non-smokers in the age group of 18-60 years presenting to ENT outpatient department with no symptoms pertaining primarily to nose. Patients with nasal complaints, those who have been treated for nasal pathology in the last 1 month and ex-smokers (Adults who used to smoke and has smoked more than 100 cigarettes in their lifetime, but had stopped stopped it at the time of the study) were excluded. After getting a well informed and written consent from all patients, detailed history taking, careful examination of the ear, nose and throat and nasal endoscopy was done. At first, pH of the nasal mucosa was evaluated using pH paper, the pH of the nasal secretions was observed by placing the paper below the inferior turbinate for 30 secs and the reading was noted followed by NMC after 15 mins. Patient is made to sit with head fixed about 10 degrees from the horizontal and a saccharin particle of size 0.5mm was placed almost a centimetre posterior to the anterior end of inferior turbinate, without revealing nature of substance to the patient. Prior instructions were given to the patient to avoid swallowing, sniffing, coughing or sneezing if possible. The time until the the patient tastes sweetness in the posterior nasopharynx was recorded in seconds. This is the NMC time. A single person was designated to perform the test in all subjects to removed observer dependant variations. Later, anterior nasal packing using 4% lignocaine and xylometazoline nose drops was done before procuring the nasal mucosa sample. All the tests were done in the same nasal cavity which was comparatively wider than the other. Scrapings of nasal mucosa were taken from middle of inferior turbinate under direct vision. This was collected by a small sterile nylon brush with a rotatory and a translational movement. Sampling was performed by the same investigator. Samples were smeared over a slide, air dried and was handed over to the pathologist where it was stained by May Grunwald Giemsa quick stain. Later they are washed, air dried and mounted in synthetic resin and a cover glass was put to increase durability. Oil immersion analysis was done under light microscope using X100 objective lens (Olympus LM50 microscope- Olympus Corp., Tokyo, Japan). Fifty microscopic fields were examined. Ciliated cells were counted. The obtained data was noted.

Statistical Analysis: Data in the form of a spreadsheet was analysed using standard statistical software. Continuous data are reported as mean ± standard deviation (SD), whereas categorical data are summarised as counts and percentages. Assuming the data following a normal distribution comparisons between smokers and non-smokers for mucociliary clearance (MCC) time, ciliated cell (CC) count, and nasal pH were conducted using the independent-samples t-test. The Pearson correlation coefficient was employed to assess the relationship between pack-years and MCC time, CC count, and nasal pH. For comparisons across different types of smoking habits, one-way analysis of variance (ANOVA) was utilised. All analyses were two-tailed where applicable, with statistical significance set at $p < 0.05$.

Results: As of any study involving a population of smokers, we could also observe a strict male predilection of the habit. Out of the 140 patients 118 (84.3%) were males, with a ratio of 5.3:1 males to females. Figure 1 shows that the age of the study population varied between 21 and 60 with a mean age of 43 years.

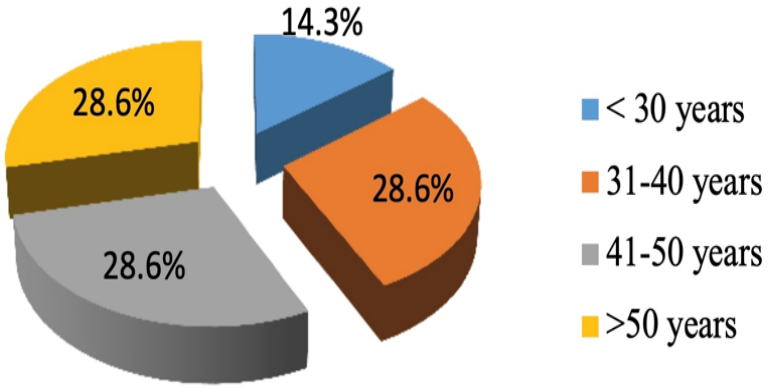

Figure 1: Pie chart showing the age distribution of the study population.

In group S of our study, various types of smoking habits were included namely hookah smoking, cigarette smoking and bidi smoking either alone or in combination with the others. Bidi smokers were 22 (31.4%), bidi and hookah smokers were 23 (32.9%), bidi and cigarette smokers were 2 (2.9%), cigarette smokers were 14 (20%), hookah and cigarette smokers were 2 (2.9%), and hookah smokers were 7 (10%). The lowest pack year amongst the smokers was 0.75 and the highest was 40 with a mean of 18.22 ±11.55 pack years. 21 smokers (30%) had pack years less than 10 years while only 13 (18.6%) had pack years above 30 as depicted in Figure 2.

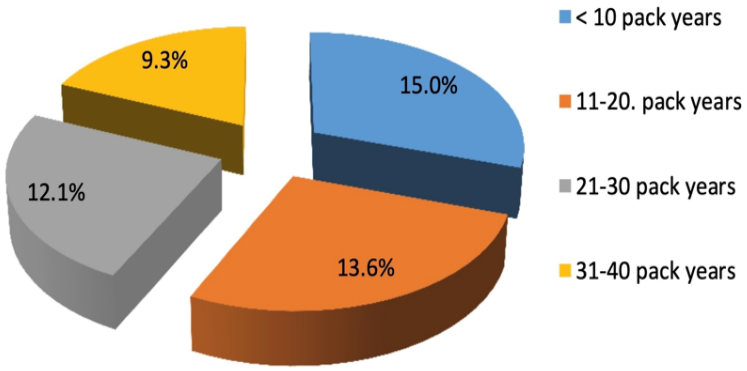

Figure 2: Pie chart showing the distribution of smoking exposure (pack years) among the study population.

Comparison of MCC time, CC count and pH in smokers and non-smokers: Table 1 shows the minimum MCC time recorded in a non-smoker was 7 mins while it was 8 mins for smoker and the maximum time recorded was 14 mins in a non-smoker and 22 mins in a smoker. Minimum CC count observed was 5 cells in a non-smoker whereas it was 2 cells in a smoker. The minimum pH noted was 4.0 in both the smoker and non-smoker while the maximum pH noted was 6.5 in a non-smoker and 7 in a smoker.

Table 1: Distribution of MCC time, CC count and pH in smokers and non-smokers

	Non smokers		Smokers	
	Minimum	**Maximum**	**Minimum**	**Maximum**
MCC time (Mins)	7	14	8	22
CC count/50hpf	5	65	2	25
Nasal pH	4.0	6.5	4.0	7.0

Table 2 shows the mean mcc time in smokers was 15.20 ±4.02 minutes versus in non- smokers which was 9.56 ±2.01 minutes.

The mean ciliated cell count in smokers was 8.71 ± 4.44 cells versus in non-smokers which was 26.89 ±12.58 cells. The mean pH in smokers was 5.40 ± 0.78 versus in non-smokers which was 5.31 ±0.69.

There was statistically significant difference ($p<0.05$) observed in MCC time and ciliated cell count of smokers.

Table 2: Comparison of MCC time, CC count and pH in smokers and non-smokers

	Non smokers		Smokers		
	Mean	**SD**	**Mean**	**SD**	**P value**
MCC time (Mins)	9.56	2.01	15.20	4.02	**0.000**
CC count/50hpf	26.89	12.58	8.71	4.44	**0.000**
Nasal pH	5.31	0.69	5.40	0.78	0.477

Correlation of pack years with MCC time, CC count and nasal pH: Table 3 shows correlation of pack years with MCC time, CC count and the nasal pH. This shows a statistically significant correlation between pack years and MCC time. Pearson correlation test checks the possibility of relationship in both ways (2 tailed) and the p-value obtained is significant ($p<0.05$). As pack years increase there is tendency to prolong the MCC time.

Table 3: Correlation of pack years with with MCC time, CC count and pH in smokers

		MMC time (min)	CC Count/50hpf	Nasal pH
	Pearson correlation	0.945	0.058	0.117
	Sig. (2 tailed)	0.000	0.635	0.334

Comparison of MCC time, CC count and pH in different types of smokers: Table 4 shows the comparison of MCC time, CC count and pH of nasal mucosa in people with different types of smoking habits. There is a significant difference in the mean MCC time among these types and the longest is found to be in patients who smoke both Beedi and Hookah. There is no statistically significant difference in CC count or nasal pH between different type of smokers.

Table 4: Comparison of MCC time, CC count and pH among different types of smokers

	Type of smoking	Number of individuals	Mean	Standard deviation	P value
MCC time (Mins)	Beedi + Hookah	23	17.78	2.798	**0.0001**
	Beedi only	22	15.78	2.454	
	Beedi + Cigarette	2	14.00	2.828	
	Hookah only	7	12.86	5.669	
	Cigarette only	14	12.36	4.034	
	Cigarette + Hookah	2	8.50	0.707	
	Total	**70**	**15.20**	**4.022**	
CC count (Per 50hpf)	Beedi + Hookah	23	8.82	4.067	0.646
	Beedi only	22	9.87	5.173	
	Beedi + Cigarette	2	6.50	2.121	
	Hookah only	7	7.50	4.879	
	Cigarette only	14	7.50	3.536	
	Cigarette + Hookah	2	8.00	2.236	
	Total	**70**	**8.71**	**4.440**	
Nasal pH	Beedi + Hookah	23	5.39	0.822	0.958
	Beedi only	22	5.37	0.786	
	Beedi + Cigarette	2	5.75	1.061	
	Hookah only	7	5.47	0.830	
	Cigarette only	14	5.00	0.707	
	Cigarette + Hookah	2	5.36	0.690	
	Total	**70**	**5.40**	**0.779**	

Discussion

The use of tobacco, whether smoked or smokeless, remains a leading global driver of illness and premature death. With an enormous number of deaths over a million per year, India holds an ample share in the international toll of tobacco consumption related deaths [19]. After china, India holds second position in world's tobacco production. According to Global Adult Tobacco Survey India 2 (GATS), India has a population 99.5 million adults who smokes currently out of which 90.6 million are men. Almost 73.0 million out of these are from rural India and urban areas contribute to the remaining 26.6 million [5].

The compounds like phenols, acrolein, formaldehyde, hydrogen cyanide and ammonia are present in tobacco smoke and are provenly toxic to cause increased oxidative stress, inflammatory changes in

airways and lungs, impaired anion transport, abnormal cilia ultrastructure and cilia genesis [14,20]. Ciliated cells of the airways play a crucial role in mucociliary clearance, helping to move secretions and harmful inhaled particles out of the respiratory tract [21]. Exposure to tobacco smoke causes a major reduction in the host defence resulting from impaired mucociliary clearance of upper airways [20]. The current study is a case control study between smoker and non-smoker group which aims at assessing the effect of tobacco smoke on the cytology, mucociliary clearance and the pH of the nasal mucosa. The smoker group in this study smoke tobacco in form of cigarettes, bidi and hookah. The prevalence of smoking bidi is common among people of lower socioeconomic status especially in rural areas as was seen in this study conducted in our institute located in rural area of the country [19]. In the study, the mean age of smokers was 42.8±9.82 years and in non-smokers this was 43.2±10.67 years. The upper limit of the age was taken 60 years to rule out ageing as confounding factor.

In this study, there were 118 (84.3%) males in which 57 (81.4%) were smokers and 61 (87.1%) were non-smokers and 22 (15.7%) females where 13 (18.6%) were smokers and 9 (12.9%) were non-smokers. As per GATS India Report 2016-2017, the proportion of smokers is considerably higher in men (19%) than in women (2%) [5]. One possible explanation for the lower proportion of female smokers is that male participants were more willing to disclose their tobacco use history.

Calculation of pack years was done for cigarette, bidi and hookah. To calculate pack-years for bidi and cigarettes, the number of packs smoked per day was multiplied by the years of smoking history. Cigarette smokers normally take 8-12 puffs over about 5-7 minutes while hookah sessions typically last for 20-80 minutes during which the smoker may take 50-200 puffs [22]. Taking history of hookah smoking in terms of puffs/day, 40puffs/day was considered as 5 cigarettes. It was observed that smokers in bidi + hookah category had high pack years as compared to rest. Frequent and long sessions of hookah smoking and bidis being affordable and easily available contribute to the observation made in the study. Altogether this combination has a comparatively higher exposure rate and high prevalence in the population.

In the current study, a strong correlation of pack years was seen with MCC time which was statistically significant ($p<0.05$), although no correlation was found with the ciliated cells and the pH. The reason being chronic and continuous exposure of nasal epithelium and mucosa to the noxious fumes present in the tobacco smoke leading to increased number of goblet cells, thickened epithelium as described in a study conducted by Hadar et al [23]. Nasal mucosa is subjected to environmental infectious agents, allergens and other substances like dusts or gases [7]. Therefore, the study has aimed to find out the changes in the cytophysiology of mucosa of smokers by counting the ciliated cells using brush cytology and the effect of smoking on them which in turn affect the mucociliary clearance. In the present study, ciliated cells were found significantly low in smokers (mean value- 8.7) as compared to non-smokers (mean value- 26.8) which is statistically significant. This is attributed to the fact that several chemicals in tobacco smoke like potassium cyanide, phenols, formaldehyde, acrolein are extremely toxic for airway cilia [15]. In the study conducted by Elwany S et al, examination of the electron microscopic sections of nasal mucosa of smokers sdecrease in number of cilia and columnar cells, intercellular oedema, fewer goblet cell count, overgrowth of mixed serous and mucous glands and vascular congestion [24]. The study conducted by Pagliuca et al also concluded that tobacco exposure decreases the count of ciliated cells [15].

In the present study, the MCC time of the smokers was compared with the non- smokers and the results were found to be statistically significant ($p<0.05$). The smoker group had an average MCC time of 15.20 ±4.02 minutes and non-smoker had time of 9.56 ±2.01 minutes. Most of the previous studies in this field also found results similar to the present study. In the study conducted by Aricigil et al in Turkey, MCC was markedly higher hookah smokers than the control group ($p<0.05$) [25]. In a similar study done by Baby et al in India, NMC was markedly longer in smokers (481.2 ±29.83 sec; $p < 0.01$) relative to non-smokers (300.32 ± 17.42 sec). Longer smoking history was linked to a significant prolongation of MCC which corroborates with our study. The delayed clearance seen in smokers may result from a decrease in frequency of beating of cilia or count of cilia and changes in the viscoelastic characteristics of mucus [12].

In the current study, the mean MCC time amongst bidi + hookah smokers was longest (17.78 ±2.7 minutes) followed by bidi smokers (15.78 ±2.4 minutes) and bidi + cigarette smokers (14.00 ±2.8 minutes). Raised MCC time in bidi smokers can be due to the fact that bidis generate roughly three times more carbon monoxide and nicotine, and about five times more tar, compared to cigarettes and it was presumed that ciliary toxicity would manifest earlier than in cigarette smokers or non-smokers [19].

Upon comparing a 45-minute session of hookah smoking with a single cigarette, it was found that in the study by Aricigil et al that the hookah smoker had higher nicotine and carbon monoxide concentrations and 20 times more PAHs than the cigarette smoker [25]. In this study, 2 smokers consuming

cigarette and hookah together were found to have least MCC time amongst the smokers this can be probably due to the fact they had less than 5 pack years and were comparatively young. The relatively younger age of the study participants may have contributed to lesser ciliary damage among smokers [19]. Although, a statistically significant difference was not seen in the ciliated cell count and nasal pH amongst these types.

The pH of nasal mucosa has been under scientific scrutiny for over fifty years (26). The normal nasal mucosal pH is between 5.5-6.5. Both ciliary function and mucus viscosity have been shown to be pH dependent as optimal pH plays an important role in normal ciliary beats function and mucociliary clearance (27). Prolonged rest and sleep promote an acidic trend in the nasal pH. The nasal pH is more acidic during the night than it is in the day (26). In the present study, 50% (70) of the study population had pH less than 5.5, 33.6% (47) had pH between 5.5 to 6.5 and 16.4% (23) had pH above 6.5. A study conducted by R J A England et al on nasal pH measurement observed that the nasal mucosal pH lies in the range of 5.5-6.5, increases in rhinitis to 7.2-8.0 and smoking did not markedly affect the pH of nasal mucosa (17). The results concurred with the current study where no significant difference was observed in smokers compared to non-smokers.

Conclusion

All these results helped us to conclude that the chronic exposure to tobacco smoke causes structural and functional changes in the nasal mucociliary system. These changes were found to be more in persons with high amount of exposure to smoke in terms of pack years. Bidi and hookah smokers are at much higher risk since they show maximum functional changes, which is a significant finding in a country like India where consumption of such products are very high.

References

1. Verra F, Escudier E, Lebargy F, Bernaudin JF, De Crémoux H, Bignon J. Ciliary abnormalities in bronchial epithelium of smokers, ex-smokers, and nonsmokers. Am J Respir Crit Care Med. 1995;151(3):630-4.
2. Ballenger JJ. Experimental effect of cigarette smoke on human respiratory cilia. N Engl J Med. 1960 Oct 27; 263:832-5.
3. Dalhamn T. Effect of cigarette smoke on ciliary activity. Am Rev Respir Dis. 1966 Mar;93(3):108-14.
4. Vachier I, Vignola AM, Chiappara G, Bruno A, Meziane H, Godard P et al. Inflammatory features of nasal mucosa in smokers with and without COPD. Thorax. 2004 Apr;59(4):303-7.
5. WHO. Tobacco Free Initiative (TFI). Global Adult Tobacco Survey. 2016. Available from: http://www.who.int/tobacco/surveillance/survey/gats/in.
6. Heffler E, Landi M, Caruso C, Fichera S, Gani F, Guida G et al. Nasal cytology: Methodology with application to clinical practice and research. Clinical & Experimental Allergy. 2018 Jun; 48(9):1092-1106
7. Glück U, and Gebbers JO. Cytopathology of the Nasal Mucosa in Smokers: A Possible Biomarker of Air Pollution? Am J Rhinol. 1996 Jan 01;10(1): 55–8.
8. Sauvalle M, Alvo A. Effect of the temperature of nasal lavages on mucociliary clearance: a randomised controlled trial. Eur Arch Otorhinolaryngol. 2018 July; 275:2403-6.
9. Agius AM, Smallman LA, Pahor AL. Age, smoking and nasal ciliary beat frequency. Clin Otolaryngol. 1998 Jun;23(3):227-30.
10. Cohen NA, Zhang S, Sharp DB, Tamashiro E, Chen B, Sorscher EJ et al. Cigarette smoke condensate inhibits transepithelial chloride transport and ciliary beat frequency. Laryngoscope. 2009 Nov;119(11):2269-74.
11. Wanner A, Salathe M, O'Riordan TG. Mucociliary clearance in the airways. Am J Respir Crit Care Med. 1996 Dec;154(6):1868-1902.
12. Baby MK, Muthu PK, Johnson P, Kannan S. Effect of cigarette smoking on nasal mucociliary clearance: A comparative analysis using saccharin test. Lung India. 2014 Jan-Mar;31(1):39-42.
13. Xavier RF, Ramos D, Ito JT, Rodrigues FM, Bertolini GN, Macchione M et al. Effects of cigarette smoking intensity on the mucociliary clearance of active smokers. Respiration. 2013 Apr 19;86(6):479-85.
14. Utiyama DM, Yoshida CT, Goto DM, Carvalho T, Santos U, Koczulla AR et al. The effects of smoking and smoking cessation on nasal mucociliary clearance, mucus properties and inflammation. Clinics Sao Paulo. 2016 Jul;71(6):344-50.
15. Pagliuca G, Rosato C, Martellucci S, de Vincentiis M, Greco A, Fusconi M et al. Cytologic and functional alterations of nasal mucosa in smokers: temporary or permanent damage? Otolaryngol Head Neck Surg. 2015 Apr;152(4):740-5.
16. Harugop AKS, Deepthi B, Hanumaiah S, Nayak SP, Ankale NR, Bellad SA. Does Endoscopic Sinus Surgery (ESS) Retrieve Mucociliary Clearance of Maxillary Sinus: Prospective Study at a Tertiary Care Hospital. Indian J Otolaryngol Head Neck Surg. 2019 Nov;71(3):2210-3.
17. England RJ, Homer JJ, Knight LC, Ell SR. Nasal pH measurement: a reliable and repeatable parameter. Clin Otolaryngol Allied Sci. 1999 Feb;24(1):67-8.

18. Nungester WJ, Atkinson AK. pH of the nasal mucosa measured in situ. Arch Otolaryngol.1944;39(4):342-3.
19. Paul B, Menon SS, Vasthare R, Balakrishnan R, Acharya S. Effect of bidi smoking on nasal mucociliary clearance: a comparative study. J Laryngol Otol. 2018 Dec;132(12):1077-1082.
20. Ramos EM, De Toledo AC, Xavier RF, Fosco LC, Vieira RP, Ramos D, Jardim JR. Reversibility of impaired nasal mucociliary clearance in smokers following a smoking cessation programme. Respirology. 2011;16:849-855
21. Zhou H, Wang X, Brighton L, Hazucha M, Jaspers I, Carson JL. Increased nasal epithelial ciliary beat frequency associated with lifestyle tobacco smoke exposure. Inhal Toxicol. 2009 Aug;21(10):875-81.
22. Shihadeh A, Azar S, Antonios C, Haddad A. Towards a topographical model of narghile water-pipe café smoking: a pilot study in a high socioeconomic status neighborhood of Beirut, Lebanon. Pharmacol Biochem Behav. 2004Sep1;79(1):75-82.
23. Hadar T, Yaniv E, Shvili Y, Koren R, Shvero J. Histopathological changes of the nasal mucosa induced by smoking. Inhal Toxicol. 2009 Nov;21(13):1119-22.
24. Elwany S, Shewel Y, Bazak R, Talaat I, Elwany M. Quitting smoking reverses nasal mucosal changes. Eur Arch Otorhinolaryngol. 2020 Jun;277(6):1691-1698.
25. Arıcıgil M, Arbağ H. Hookah smoking impairs nasal mucociliary clearance. Tob Induc Dis. 2018 Feb 28; 16:1-6.
26. Fabricant ND. Significance of the ph of nasal secretions in situ: further studies. Arch otolaryngol. 1941;34(2):297–301.
27. Amin, Hany. Nasal Mucosal pH in Different Nasal Diseases. Faculty of Benha Medical Journal. 2012.