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An Analytical Study of the Relation between Smoking and Allergy in Sinonasal Polyposis

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ABSTRACT
Introduction: Nasal polyposis (NP) is a disease in which abnormal hyperplastic inflammatory lesions originate from the sinus and paranasal sinus mucosa.
Objectives of the study: The main objective of the study is to find the relation between smoking and allergy in sinonasal polyposis.
Material and methods: This cross-sectional study was conducted in Sharif medical and dental college, Lahore during 2020 to 2021. The data was collected from both genders. The sample size for this study was 50 (smokers and nonsmokers), ad those patients which was suffering from nose allergy was selected for this study.
Result: The data was collected from 50 patients, out of which 22 were smokers and 28 were nin-smokers. The mean age for smokers was 46.4 ± 5.3 years and 42.5 ± 5.4 for non-smokers. There were 14 (69.1%) male and 8 (30.9%) females in smokers group and 16 (57%) males and 12 (42.85%) females in non-smokers group. The mean duration of sinonasal polyposis for smokers group was 2.3 ± 2.8 years and for non-smokers was 2.5 ± 1.3 years.
Conclusion: It is concluded that tobacco smoke may significantly affect in patients with sinonasal polyposis. Consequently, careful evaluation and management of smokers should be performed.

KEYWORDS: Allergy, Polyposis, Smoking.

INTRODUCTION

Nasal polyposis (NP) is a disease in which abnormal hyperplastic inflammatory lesions originate from the sinus and paranasal sinus mucosa. Historically, nasal polyps have not been believed to either represent a precancerous condition or to confer additional risk for the development of nasal cavity/paranasal sinus (NCPS) or nasopharyngeal cancers[1]. There have been studies that have suggested such a relationship; however, these have been primarily case-control studies, and their small sample size has limited the ability to assess future risk for head and neck cancers. Also in the recent literature are two larger database analyses, one looking at the relationship between chronic sinusitis and head and neck cancer and another looking at nasopharyngeal cancer in a Taiwanese population[2].

These prior studies were limited by having only a small number of patients with nasal polyps or from being unable to control for smoking history as a risk factor.5,6 A key feature in understanding NP as it might pertain to the risk for neoplasia is the nature of the underlying inflammatory pathology. NP, especially in Western countries and North America, is more often an eosinophilic disease associated with type 2 inflammation and occurring in the setting of asthma, allergic rhinitis, and concomitant atopic disease[3]. However, in a smaller subset of subjects in these countries (but most subjects in China and other Asian countries), NP is characterized by neutrophilic inflammation in the setting of robust innate immunity with or without type 1 (IFN-g)/type 3 (IL-17) inflammation but not eosinophila[4]. Several of these recent studies regarding neoplastic development in patients with NP draw from populations that are enriched for tobacco abuse/exposure and/or in which there is a predominance of neutrophilic inflammatory polyposis. Properly determining the risk for the development of neoplasia in patients with NP is extremely important because this will both guide physicians in performing appropriate clinical investigations regarding the basis of risk and will determine how best to counsel and treat patients with NP[5-6].

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OBJECTIVES OF THE STUDY

The main objective of the study is to find the relation between smoking and allergy in sinonasal polyposis.

MATERIAL AND METHODS

This cross-sectional study was conducted in Sharif medical and dental college, Lahore during 2020 to 2021. The data was collected from both genders. The sample size for this study was 50 (smokers and nonsmokers), ad those patients which was suffering from nose allergy was selected for this study.

- **Inclusion criteria**

All the patients who have sinonasal polyposis and history of smoking was selected for this study.

- **Exclusion criteria**

Those who have the history of cardiac issue and any other deformity were excluded from this study.

Data collection

Environmental and behavioural factors and medical history with a focus on allergies, including specific months when rhinitis symptoms without a cold occurred, were assessed at 9 time points by using face-to-face, paper, telephone, and online questionnaires. The data was divided into two parts, one was smokers group and one was non-smokers group. Specific IgE levels were measured at 9 time points. The study was approved by local institutional review boards in all study centers. Parents and participants provided written informed consent. Status of current smoking was assessed measuring the salivary cotinine levels. Cotinine is a biomarker of nicotine exposure and can be measured in blood, urine, and saliva; in all three matrices, it has a half-life of approximately 17 h, allowing time for detection of recent nicotine exposure.

Data were analyzed by using the Statistical Package for Social Sciences program (SPSS for Windows 20.0 Chicago, USA). Groups were compared using the Student t test for normally distributed quantitative data.

Table 01: Baseline characteristics of study participants

Characteristics	Current smokers N=22	Non-smokers N=28	p-value
Age			
Mean ± SD	46.4 ± 5.3	42.5 ± 5.4	0.102
Range	20-60	20-65	-
Gender			
Male	14 (69.1%)	16 (57%)	0.484
Female	8 (30.9%)	12 (42.85%)	
Duration of sinonasal polyposis, (years)			
Mean ± SD	2.3 ± 2.8	2.5 ± 1.3	0.357
Range	1-5	1-5	-

The values of nasal and serum immuno-inflammatory markers in both smokers and non-smokers were presented in table 02. The mean value of IgE in nasal was 3.5 ± 0.6 (kU/L) in smokers and 3.6 ± 0.4 (kU/L) in non-smokers.

Table 02: Nasal and serum immuno-inflammatory markers in both groups

Characteristics	Smokers	Non-smokers	p-value
Nasal immuno-inflammatory biomarkers, mean ± SD			
IgE, (kU/L)	3.5 ± 0.6	3.6 ± 0.4	0.318
IL-4, (pg/mL)	13.9 ± 2.1	12.7 ± 1.4	0.287
IL-5, (pg/mL)	No detected	No detected	
Serum immuno-inflammatory biomarkers, mean ± SD			
IgE, (kU/L)	178.4 ± 22.6	187.6 ± 29.5	0.281

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IL-4, (pg/mL)	32.2 ± 8.3	22.3 ± 10.2	0.400
IL-5, (pg/mL)	0.4 ± 0.3	0.3 ± 0.2	0.621

Current smoker patients presented a significantly higher concentration of cotinine in saliva than non-smokers. The mean values of cotinine were presented in table 03.

Table 03: Salivary cotinine levels in both groups

Characteristics	Smokers	Non-smokers	p-value
Salivary cotinine levels (ng/mL), mean ± SD	285.7 ± 52.3	1.9 ± 0.6	< 0.001*

DISCUSSION

Cigarette smoke is probably the most common environmental factor that has been associated with various airway diseases, including rhinosinusitis, bronchitis, and pneumonia in children. However, previous population-based studies have provided conflicting information regarding the potential correlation between tobacco smoke and AR[7]. A study recruiting 200 patients demonstrated that both past and current SHS exposure were significant risk factor for AR. Contrariwise, other studies showed a negative association between cigarette smoke and AR. Allergic rhinitis is characterized by a Th2-polarized inflammation[8]. Th2-derived cytokines, such as IL-4 and IL-13, are the primary pathogenic factors in inducing, maintaining and amplifying inflammatory allergic inflammation[8]. IL-4 and IL-13 orchestrate allergic inflammation promoting IgEsynthesis, up-regulating adhesion molecules selective for eosinophil recruitment and causing increased mucus production and airway hyperreactivity[9].

On the other hand, there is accumulating evidence that Th1-related cytokines, such as IFN-γ and IL-12, may suppress and counteract this Th2 response, and vice versa, as there is a functional dichotomy between Th1 and Th2 cells[10].

Challenges with non-allergenic stimuli on target organs of allergic diseases (eg, the nose, lung, eye, and skin) have shown that the degree of nonspecific tissue reactivity contributes significantly to the clinical picture of allergic diseases, and the heterogeneity of allergic phenotypes is best approached taking into account a wider variety of symptom triggers[11-15].

CONCLUSION

It is concluded that tobacco smoke may significantly affect in patients with sinonasal polyposis. Consequently, careful evaluation and management of smokers should be performed.

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