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Research Article

**ANALYSIS OF ROLE OF ALLERGIC RHINITIS (AR) AS A
COMMON IMMUNOLOGICAL DISORDER IN LOCAL
POPULATION OF PAKISTAN**Maryam Sana¹, Zafarullah², Zulfiqar Ali Soomro²¹Services institute of medical sciences, Lahore²MO at Allergy Centre NIH Centre Islamabad**Abstract:**

Introduction: In recent times, the incidence of allergic diseases, particularly bronchial asthma, has been increasing worldwide. Allergic rhinitis (AR) is the most common immunological disorder and is characterized by an immunoglobulin E (IgE)-mediated inflammation induced by allergen exposure. **Objectives of the study:** Our basic aim of the study is to find the role of Allergic rhinitis (AR) as a common immunological disorder in local population of Pakistan. **Methodology:** This study was conducted at Allergy Centre NIH Islamabad during 2017 with the permission of ethical committee and patients of the hospital. The data was collected from both genders. The sample size for this study was 50, ad those patients which was suffering from nose allergy and any other form of allergy was selected for this study. **Result:** Symptom scores were higher in the rhinitis than in control group during all of the experiments ($P < .001$) and were significantly elevated in both groups 4, 24, and 48 hours after beginning the challenge compared with baseline. Total cell count was significantly elevated in both groups. **Conclusion:** It is concluded that there is a need of more studies for the clarification of role of AR as a immunological disorder in Pakistan. IgE-mediated nasal response but have some features in common.

Key words: Immunological, disorder, asthma, nasal infection.

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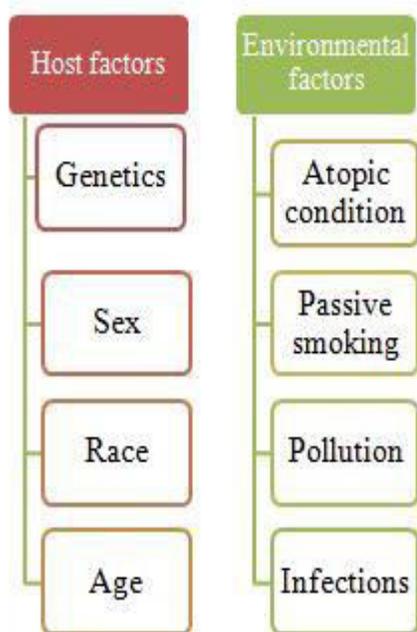
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INTRODUCTION:

In recent times, the incidence of allergic diseases, particularly bronchial asthma, has been increasing worldwide. Allergic rhinitis (AR) is the most common immunological disorder and is characterized by an immunoglobulin E (IgE)-mediated inflammation induced by allergen exposure. Infiltrating cells, including T cells, eosinophils, mast cells and basophils, release several mediators, that cause the symptoms occurrence, and cytokines, that promote and amplify the inflammatory cascade [1]. Therefore, AR triggers both a local and a systemic inflammatory process. The main symptoms are the so-called irritative ones, such as itching, sneezing and rhinorrhoea, that are histamine-dependent, and obstruction, that is inflammation-dependent [2].



AR is a risk factor for asthma. Two recent studies provided convincing confirmation that AR is an independent risk factor for asthma onset. It is reported that patients with AR are at three times the risk of developing asthma compared with those without AR [3]. In addition, a cross-sectional study of representative samples of young adults, who completed a detailed questionnaire and underwent lung function tests, bronchoprovocation challenge, IgE measurement and skin prick test, has been performed in Europe. The results demonstrate and confirm that AR is a main risk factor for asthma onset. There is a close epidemiological link between AR and asthma, as reported by several surveys. Among them, a recent European multi-centre, cross-sectional survey evaluated respiratory symptoms in the young adult population. About 60% of asthmatics

suffered from AR; on the other hand, AR patients presented an eightfold risk of having asthma compared to subjects without AR [4].

Allergic rhinitis and asthma share common immunological mechanisms characterized by Th2-dependent inflammation. As a consequence, allergic subjects have typically Th2-polarization and a reduced Th1-response. Interferon (IFN)- γ , a typical Th1-derived cytokine, is deputized for fighting infections [5]. Thus, it has been pointed out that allergic subjects could present a higher susceptibility to contracting respiratory infections (RI) than non-allergic subjects. This hypothesis could be reinforced by evidence that rhinovirus infections are the most common. The main receptor for rhinovirus is intercellular adhesion molecule-1 (ICAM-1). ICAM-1 expression on nasal epithelial cells is related strictly to allergen exposure in allergic subjects. Thus, there is a clear link between allergen exposure, ICAM-1 expression, allergic inflammation and RI. In addition, treatments with drugs able to reduce ICAM-1 expression diminish both the number and severity of RI in allergic children [6,7].

Objectives of the study

Our basic aim of the study is to find the role of Allergic rhinitis (AR) as a common immunological disorder in local population of Pakistan.

METHODOLOGY:

This study was conducted at Allergy Centre NIH Islamabad during 2017 with the permission of ethical committee and patients of the hospital. The data was collected from both genders. The sample size for this study was 50, and those patients which were suffering from nose allergy and any other form of allergy were selected for this study. Environmental and behavioral 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. 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.

Statistical analysis

The influence of early-life exposures and behavior on AR was assessed by using time-to-event analysis. Disease onset was estimated at the midpoint between the first follow-up meeting case definition criteria and the prior negative (or missing) follow-up coded in days.

RESULT:

Symptom scores were higher in the rhinitis than in control group during all of the experiments ($P < .001$) and were significantly elevated in both groups 4, 24, and 48 hours after beginning the challenge compared with baseline. Total cell count was significantly elevated in both groups. Table 1 summarizes the status of some SNPs associations with asthma in

Pakistan and in some other populations as well as their allele frequency comparison with global MAF. Participants from both rich and poor backgrounds (grandparents and parents) had the same risk of AR as those from average-income families. Low parental education showed a weak association with lower AR incidence. Other aspects of the family's background were not linked to AR.

Gene ID	SNP ID	Asthma Phenotype	Association in Pakistani Population	Populations Already Associated	Global MAF	Allele Frequency in Pakistan
CD14	rs2569190	Allergic rhinitis	Significant[12]	Turkish[13], Chinese Han children[14], Korea[15], Norway[16]	T=0.47	0.557
	rs2569191	Atopic asthma	Significant[12]	Norway[16]	G= 0.47	0.557
IL-4	rs2070874	Atopic asthma	Non-significant [17]	China[18,19], Netherlands [20]	T=0.40	0.198
	rs2243250	Atopic asthma	Significant [17]	Turkish[13], Netherlands [20]	T= 0.47	0.198
	rs2227284	Atopic asthma	Significant [17]	Polish[21], China[22]	G= 0.39	0.625
IL-13	rs1881457	Atopic asthma	Significant [23]	UK[24],	C= 0.20	0.245
	rs847	Atopic asthma	Non-significant [23]	-	T= 0.25	0.323
	rs20541	Atopic asthma	Non-significant [23]	Korea[15,25], UK[24], Portugal[26], Japan[27], China[28]	A= 0.27	0.328
	rs1800925	Asthma	Significant [29]	UK[24], Caucasians[30]	T= 0.25	0.25
TBXA2R	rs1131882	Asthma	'A allele' protective factor [29]	Japan[31]	A= 0.23	0.203
IL10	rs1800896	Asthma	Significant [29]	South India[32]	G= 0.27	0.318
TNF- α	rs1800629	Asthma	Non-significant [33]	West Asians[34], South Asians[34]	A= 0.09	0.057
IKZF3	rs3816470	Asthma	Significant [35]	southern Chinese children[36]	G= 0.47	0.411

Table 01: Genetical analysis of Allergy factors

Table 2. Association of Participant General Characteristics and Symptom-Based and Allergy Test Result-Based Allergic Rhinitis

Characteristic	OR (95% CI)	P Value
Symptom-based allergic rhinitis, KNHANES 2008-2012		
Male sex	1.04 (0.98-1.11)	.18
Aging	0.99 (0.98-0.99)	<.001
Urban residence	1.21 (1.06-1.38)	.005
Family income greater than average income	1.05 (0.98-1.13)	.19
Allergy test result-based allergic rhinitis, ^a KNHANES 2010		
Male sex	1.19 (0.88-1.62)	.26
Aging	0.98 (0.97-0.99)	<.001
Urban residence	1.22 (0.85-1.75)	.27

DISCUSSION:

Allergic rhinitis is characterized by a Th2-polarized inflammation. Th2-derived cytokines, such as IL-4 and IL-13, are the primary pathogenic factors in inducing, maintaining and amplifying inflammatory allergic inflammation⁸. IL-4 and IL-13 orchestrate allergic inflammation promoting IgE synthesis, up-regulating adhesion molecules selective for eosinophil recruitment and causing increased mucus production and airway hyperreactivity.

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 [9].

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 [10]. Considering that none of the tested individuals had nasal eosinophilic syndromes, this study contributes to the understanding of the mechanism of physical stimuli leading to inflammatory responses in the allergic population [11]. This is in contrast with the nonallergic noneosinophilic rhinitis that, in spite of nasal hyperresponsiveness to nonallergenic stimuli, showed no demonstrable inflammation, and it has been considered a misnomer because of the absence of inflammatory alterations [12].

CONCLUSION:

It is concluded that there is a need of more studies for the clarification of role of AR as a immunological disorder in Pakistan. IgE-mediated nasal response but have some features in common. They suggest the occurrence of hyperreactivity of the nasal mucosa in the rhinitis group with immediate epithelial shedding followed by an inflammatory response with the recruitment and activation of eosinophils, resulting in higher symptom scores in response to artificially induced sudden temperature changes [13,14].

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