Original Article

The Relationship of Immunoglobulin-E and Tumor necrosis factor in patients with Allergic Rhinitis in Babylon Province

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Abstract

Background: Rhinitis is defined as inflammation of the mucous membranes of the nose usually that is accompanied by swelling of the mucosa and a nasal discharge. It is caused by immunoglobulin E (IgE)-mediated reactions to inhaled allergens, smoking and alcohol use, gender, age, overweight, adoption of pets, race, educational achievement, and family history can represent a risk factor for allergic rhinitis (AR). **Objectives:** To find the effect of electrolytes tumor necrosis factor (TNF)-α and IgE on AR and study the influence of smoking, gender, and obesity (as a risk factor on AR) on the levels of TNF-α and IgE. **Materials and Methods:** Eighty subjects for this study were enrolled; 40 of those who have AR age ranged between 18 and 50 years. A control group of 40 subjects who appear to be in good health was selected. This study is a case–control study. TNF-alpha concentration and immunoglobulin-E were measured in serum by an enzyme-linked immunosorbent assay (ELISA) technique also The electrolytes concentrations were measured in the serum using an Abbott device with Integrated Chip Technology (ICT) human kit. The electrolytes were also measured in the serum using the Abbott device. Full history was taken from all patients, which include age, residence, smoking, body mass index, and family history of AR. **Results:** The results reveal a significant difference ($P < 0.05$) in levels of TNF-α, Ig-E between patients and controls, There was no significant difference between smoker and non- smoker, normal weight and overweight groups in patients and controls groups.

Keywords: Allergic rhinitis, electrolytes, enzyme-linked immunosorbent assay (ELISA), IgE, tumor necrosis factor-alpha

Introduction

Allergic rhinitis (AR) occurs due to exposure to a foreign body called allergen.[1] AR can be linked to illnesses such as weariness, headache, cognitive decline, and sleep disturbance. It is a substantial contributor covering a large area, high medical treatment expenses, productiveness, and poor quality of life. Different cytokines and adhesion receptors appear to be key players in AR. Following that, a variety of cytokines activate B-cells to secrete immunoglobulin E (IgE). The patient is exposed to a lot of allergens during the initial period. Allergens bind to IgE, which is then linked to mast cells. This process stimulates the production of several mediators, including histamine, leukotrienes, and prostaglandins, each of which has a specific role in the onset of symptoms.[2] Antigen-presenting cells are also activated by allergen-loaded Ig-E, resulting in immunomodulation of T-cell responses In allergy The symptoms the late stage, which start after 4–8 h, are significantly more severe and include nasal obstruction. The symptoms of the late stage, which start after 4–8 h, are significantly more severe and include nasal obstruction.[3]

Genetic predisposition, environmental variables, exposure to allergens, passive exposure to tobacco smoke (especially in early life),[4] diesel exhaust particles in urban areas, obesity, and the pathophysiology of AR may all have an impact on the development of disease.[5] At late stage of disease, nasal mucosal inflammation occurs with the influx and activation of a variety of inflammatory cells such as T cells, eosinophils, basophils, neutrophils, and monocytes into the nasal mucosa,[6] In normal physiological state

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intact epithelial barrier prevents allergens infiltration and hence homeostasis of immune components and functions are maintained.[7] Those belonging to the tumor necrosis factor-alpha (TNF-α) and interleukin (IL) families are thought to play a major role in the development of chronic inflammatory conditions, that is, elevated TNF-α levels are a factor in the underlying inflammation that underlies rhinitis. IgE levels in the serum of healthy people are 104 times lower than IgG concentration.[8] IgE-expressing plasma cells are relatively rare in the bone marrow, indicating that they are more prevalent in rhinitis patients.[9]

Minerals are the fundamental chemical components needed by living organisms. The seven bulk elements are calcium, magnesium, sodium, potassium, phosphorus, potassium, sulfur, and chloride. They make up between 60% and 80% of the body's total inorganic matter.[10] Sodium is the principal extracellular cation fluid, and bones contain about 50% of the body's sodium, 40% of extracellular fluid, and 10% of soft tissues. Biochemical processes play an essential role in the preservation of osmotic pressure in compartments of extracellular fluid. It is essential for maintaining healthy cell permeability and irritable muscle. Sodium aids in the intestinal absorption of amino acids, glucose, and galactose. It is essential for starting and keeping heartbeat tissues.[11] Potassium is the main intracellular cation. The concentration of potassium in the erythrocytes is about 23 times the concentration in plasma. For this reason, only an unhemolyzed sample must be used. Potassium is required for equilibrium within the cells. For optimal action, the glycolysis enzyme pyruvate kinase needs K+. The propagation of nerve impulses requires potassium. The right amount inside the cell K+ is required for ribosomes to properly biosynthesize proteins. Cardiac muscle activity is influenced by extracellular K+.[12] More than 90% of the plasma cation concentration in healthy subjects comes from sodium, and modest levels of potassium and calcium are also present.

Calcium is the most abundant among the minerals in the body Biochemical functions : Development of bones and teeth, Muscle contraction, Blood coagulation, Nerve transmission, Membrane integrity and permeability Activation of enzyme, second messenger for such hormonal action on heart : Ca2+ acts on myocardium and prolongs systole.[13]

Chloride is the major extracellular substance found in sodium chloride. As a result, the metabolic processes of sodium and chlorine metabolism are closely connected. Chloride regulates fluid balance, osmotic pressure, and acid-base equilibrium. Chloride interacts with Na+ and K+ to carry out these duties. It is also required for the gastric juice to produce HCl, and it shifts chloride with the help of Cl− and K+. Chloride activates the salivary amylase enzyme.[14]

In this study, we investigated the effect of the four most important essential elements in the body, namely sodium, potassium, calcium, and chloride, and their effect on patients with AR. Additionally, this study aimed to study the effect of some risk factors such as sex, body mass index (BMI), and smoking on patients with AR.

Materials and Methods

Eighty subjects for this study were enrolled; 40 of those who have AR age ranged between 18 and 50 years. A control group of 40 subjects who appear to be in good health was selected. Samples were collected from. Imam Sadiq Hospital. Hilla Teaching Hospital. Allergy and Asthma Center to measure TNF-alpha concentration and Ig-E in serum by an enzyme-linked immunosorbent assay (ELISA) technique also The electrolytes concentrations were measured in the serum using an Abbott device with Integrated Chip Technology (ICT) human kit. Full history was taken from all patients, which include age, residence, smoking, BMI, family history of AR, medical history drug history, and surgical history. No drugs were prescribed to those patients that may interfere with the measured parameters. Statistical analyses were performed with country. Mean ± standard deviation was used to express the data. The Student's test and Pearson correlation analysis, which were employed to establish the significance of the difference between the two groups, were used to evaluate the normality of the distribution of all variables. A *P* value of below 0.05 is regarded as significant.

Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patients' verbal and

Table 1: Serum levels of TNF-α, IgE, sodium, potassium, calcium, and chloride in patients with allergic rhinitis and control

Variable	AR	Control	*P* value
TNF-α (pg/mL) (mean ± SD)	78.84 ± 5.1	18.88 ± 7.2	<0.01
IgE (IU/mL) (mean ± SD)	297.1 ± 15.2	93.2 ± 12.3	<0.01
Sodium (mEq/L) (mean ± SD)	139.2 ± 2.1	140.5 ± 1.5	>0.01
Potassium (mEq/L) (mean ± SD)	4.49 ± 0.2	4.38 ± 0.15	>0.01
Calcium (mEq/L) (mean ± SD)	2.1 ± 0.05	2.2 ± 0.15	>0.01
Chloride (mEq/L) (mean ± SD)	103.05 ± 1.2	105.2 ± 1.11	>0.01

SD: standard deviation

analytical approval before the sample was taken. The study protocol and the subject information and consent form were reviewed and approved by a localethics committee according to the document number 4360 on September 12, 2022.

Results

The data show that concentration of TNF- and IgE in serum were increased significantly in patients with AR in ($P < 0.01$) compared with controls group, and no significant difference ($P > 0.01$) in the sera level of sodium, potassium, calcium, and chloride, between patients and control group

Discussion

Activation of Th cells leads to the production of various cytokines, such as IL-3, IL-4, IL-5, IL-6, IL-13, interferon, and TNF-α. Leads to a latent recruitment phase that ushers in the inflammatory late-phase response. Eosinophils are the predominant cell, and they generate vasoactive mediators and cytotoxic proteins.[15]

Histamine and leukotriene syntheses are both enhanced by cytokines, which are created when mast cells and T-cells are activated. They stimulate gland and goblet cell secretion, vasodilation, and blood vessel leakage, which results in the recognizable watery rhinorrhea. At this early stage, nasal congestion is already present because of cavernous sinusoids blood pooling and vessel dilatation. Sneezing reflexes and itchy sensations are brought on by sensory nerve activation.[16]

TNF-α alters the flow of ionized Ca+ inside smooth muscle, which, in turn, encourages the formation of IgE. IgE causes the release of mediators that cause mucous secretions, itching, rhinorrhea, developed vascular permeability, and arteriolar dilatation.[17] As the results in Table 1.

Numerous studies have found risk factors for some types of rhinitis, including AR. But an additional study is still needed to fully comprehend nonallergic (noninfectious) rhinitis and its related risk factor. The following are risk factors for AR: the existence of additional allergic conditions including asthma, eczema,[18] rhinitis in parents typical home aeroallergens causing allergic sensitivity, being overweight or obese, increased nitric oxide exhalation, high total serum IgE, exposure to smoking by parents, exposure to animals genetic propensity, exposure to contaminants related to traffic and fossil fuels, increased moisture and inadequate ventilation from modern dwellings' tight insulation, and infants are introduced to food and formula early.[19]

The results in Table 2 reveal a significant difference ($P < 0.05$) in levels of TNF-α, IgE between males and females, smoking and nonsmoking, and normal weight and overweight groups in AR.

Table 2: Levels of TNF-α, IgE between male and female, smoking and nonsmoking, and normal weight and overweight groups in allergic rhinitis

Variable	AR	*P* value
TNF-α (pg/mL) (mean ± SD)		
Male	72.2 ± 3.4	<0.05
Female	67.4 ± 1.2	
Smoker	68.91 ± 3.2	<0.05
Nonsmoker	44.3 ± 2.1	
Normal weight	77.7 ± 9.3	<0.05
Overweight	79.5 ± 8.2	
IgE (IU/mL) (mean ± SD)		
Male	283.5 ± 10.3	<0.05
Female	220.2 ± 6.7	
Smoker	291.2 ± 4.2	<0.05
Non-somker	202.4 ±1.5	
Normal weight	299.2 ± 13.1	
Overweight	295.2 ± 17.3	

SD: standard deviation

Conclusion

The increase in those two parameters and its interfering effects can be explained by the following . Allergen is mediated by sensitized mast cells whose high-affinity IgE receptors (FceRI receptors) are occupied by IgE directed against specific allergens Activation of Th cells leads to the production of various cytokines, such as interleukin, and TNF-α All these events amplify the allergic inflammatory response, leading to a real cascade of reactions. TNF-α causes changes in the ionized calcium flux within smooth muscle. which lead consequently to the promotion of IgE production. IgE triggers the release of mediators that are responsible for arteriolar dilation, increased vascular permeability, itching, rhinorrhea and mucous secretions.

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Conflicts of interest

There are no conflicts of interest.

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