

4th International Scientific Conference of Alkafeel University (ISCKU 2022)

Najaf, Iraq • 20–21 December 2022

Editors • N. Aldahan and Ali J. Ramadhan



ISCKU 2022

RESEARCH ARTICLE | DECEMBER 22 2023

Evaluation of some immunological parameters in persistent allergic patients suffer nasal *Staphylococcus aureus*

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AIP Conf. Proc. 2977, 040105 (2023)

<https://doi.org/10.1063/5.0182142>

Staphylococcus aureus is a typical bacterium associated with the most common community- and hospital-acquired illnesses and has long been recognized as a significant issue in public health. *S. aureus* nasal carriage has immune-modulating effects in atopic dermatitis patients and may be a factor in allergic rhinitis patients' inflammatory airways and allergic reactions. The purpose of this study is to look at the prevalence of nasal *S. aureus* infection in individuals with chronic allergic rhinitis and its potential impact on immunological markers. Between March 2022 and November 2022, 100 patients with allergic rhinitis who visited Al-Sadder Medical City, an ENT outpatient clinic in the Najaf governorate, were included in the first group (patient groups). There were 100 healthy volunteers in the control group who weren't allergic and didn't have any respiratory issues. To isolate *S. aureus* from each group's nasal swab, sterile cotton swabs were used. Serum concentrations of IL-4, IFN- γ , and total IgE were then determined ELISA technique. In contrast to 24/100 controls, *S. aureus* was discovered in 52/100 allergic patients (P .001). The total IgE-levels of allergic *S. aureus* carriers were considerably greater as compared to patients non-suffered from allergic. Nasal *S. aureus* carriers showed greater levels of IL-4 than non-carrier individuals ($p=0.037$). IFN- γ was elevated in individuals who were not *S. aureus* carriers compared to those who were ($P=0.05$), and this was associated with elevated total IgE in allergic patients. Without a doubt, allergic rhinitis increases

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S. aureus colonization, and this might place these individuals at higher risk for more serious staphylococcal infections and found the immunological parameters(IL-4, IFN- γ , and total IgE) were significant differences between allergic *S. aureus* carriers compared to allergic non-carriers. More research is needed, such as attempts to eradicate staphylococcal infection and reassessment of illness and serological biomarkers.

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Evaluation of some Immunological parameters in persistent allergic patients suffer nasal *Staphylococcus aureus*

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Abstract *Staphylococcus aureus* is a typical bacterium associated with the most common community- and hospital-acquired illnesses and has long been recognized as a significant issue in public health. *S.aureus* nasal carriage has immune-modulating effects in atopic dermatitis patients and may be a factor in allergic rhinitis patients' inflammatory airways and allergic reactions. The purpose of this study is to look at the prevalence of nasal *S. aureus* infection in individuals with chronic allergic rhinitis and its potential impact on immunological markers. Between March 2022 and November 2022, 100 patients with allergic rhinitis who visited Al-Sadder Medical City, an ENT outpatient clinic in the Najaf governorate, were included in the first group (patient groups). There were 100 healthy volunteers in the control group who weren't allergic and didn't have any respiratory issues. To isolate *S. aureus* from each group's nasal swab, sterile cotton swabs were used. Serum concentrations of IL-4, IFN- γ , and total IgE were then determined ELISA technique. In contrast to 24/100 controls, *S. aureus* was discovered in 52/100 allergic patients (P .001). The total IgE-levels of allergic *S. aureus* carriers were considerably greater as compared to patients non-suffered from allergic. Nasal *S. aureus* carriers showed greater levels of IL-4 than non-carrier individuals (p=0.037). IFN- γ was elevated in individuals who were not *S. aureus* carriers compared to those who were (P = 0.05), and this was associated with elevated total IgE in allergic patients. Without a doubt, allergic rhinitis increases *S. aureus* colonization, and this might place these individuals at higher risk for more serious staphylococcal infections and found the immunological parameters(IL-4, IFN- γ , and total IgE) were significant differences between allergic *S. aureus* carriers compared to allergic non-carriers .More research is needed, such as attempts to eradicate staphylococcal infection and reassessment of illness and serological biomarkers.

Key words: *Staphylococcus aureus*, Allergic rhinitis, IgE, IFN- γ , IL-4.

INTRODUCTION

Staphylococcal infections are significantly influenced by nasal *S. aureus* carriage [1], which may potentially increase the likelihood of illness aggravation in extremely uncommon cases like Wegener's granulomatosis [2].

Methicillin - resistant bacterial disease is the main cause of several conditions, ranging from mild skin infections to severe toxic shock syndrome [3]. Additionally, Staphylococcal colonization could control allergy illness. Following this organism's dermal overgrowth, individuals with atopic dermatitis experience illness relapses, and the majority of these patients produce IgE antibodies to Staphylococcal antigens [4].

Enterotoxins, a series of proteins with various serological types but similar structures and functions, are produced by *Staphylococcus aureus* and serve as superantigens [5] In people who are genetically susceptible, allergic rhinitis (AR) is a persistent inflammatory disorder of the airways caused by the eosinophilic cell [6].

In an Iraqi study it was found that allergic rhinitis and asthma contributed to poor performance at work for adult and school absence for children and adolescents ,also to decreasing the quality of life for those who are affected and their families, moreover both allergic rhinitis and asthma shown to be strongly associated with each other ,so that when we think to treat each of them better for us , to think about the entire airway rather than any isolated part [7].

METHODOLOGY

Two groups were involved in the study:

First group: This group consisted of 100 people who had allergic rhinitis. From March to November 2022, These individuals went to the ENT outpatient center Al-Sadder Medical City in the Najaf governorate.

1. Persons who had taken oral corticosteroids or had upper respiratory infections in the four weeks before to enrollment are excluded from the study.

2. Individuals who have started treatment or have just ended it.

The healthy groups involve of 100 non-allergic person participants without respiratory isolation symptoms. Nasal

swab taken from both patients and controls to test for *S. aureus* using a sterile cotton swab. Age, gender, and address data were collated for both the sick group and the healthy control. Venipuncture was used to obtain five milliliters of each person's venous blood. A blood sample was given a few minutes to properly coagulate. To separate the serum for use in various immunological assays, it was centrifuged at 1500 rpm for 5 min., split into three tubes, and kept at -20°C. finally, *S. aureus* can be detected by VITEK-2 system.

RESULTS

Comparing the rate of *S. aureus* isolated from patients with allergic rhinitis (AR) to that from control subjects' nasal cavities, the table (1) shows the rate of *S. aureus* isolated from AR patients' nasal cavities.

Table (1): Patients with allergic rhinitis and healthy controls were enrolled in the research.

Study groups	Number	<i>S. aureus</i> Positive		<i>S. aureus</i> Negative		P-value
			%		%	
Allergic rhinitis patients	100	52	52%	48	48.0%	Odds=3.4306
Control	100	24	24.0%	76	76%	
Total	200	76	38%	124	62%	

Table (2) shows AR patients expressed cytokine IL-4 mean level (327.56369pg/ml) higher than the mean level of Control carry *S. aureus*(137.94304pg/ml) Present study showed a significant difference between AR patients and control groups (p< 0.001) . IFN γ (44.19817 Pg/ml) in AR patients and (201.31092 Pg/ml) in the control group the difference was significant.

Table (2): Mean levels of the cytokines (IL-4 and IFN- γ) in patients and healthy controls who were *S. aureus* carriers

Interleukins	type	No.	Mean Pg/ml	Std. Error Mean	P-value
IL-4	Patient carry <i>S. aureus</i>	52	327.56369	25.079888	<0.001
	Control carry <i>S. aureus</i>	24	137.94304	19.204989	
IFN γ	Patient carry <i>S. aureus</i>	52	44.19817	7.021542	<0.001
	Control carry <i>S. aureus</i>	24	201.31092	19.327231	

Table (3) shows the mean IL-4 of Patient carry *S. aureus* group was (327.56369 pg/ml), for Patient non carrier it was (277.24077pg/ml) and it was statistically significant. Regarding the mean of IFN- γ it was significantly higher in Patient non carrier group than Patient carry *S. aureus* group.

Table(3):Mean concentration of cytokines (IL-4 and IFN- γ) in patients carriers for *S. aureus* and patient's non carrier

Interleukins	Case	No.	Mean	Std. Error Mean	P –value
IL4	Patients carry <i>S. aureus</i>	52	327.56369	25.079888	0.037
	Patients non carry <i>S. aureus</i>	48	277.24077	24.414725	
IFN- γ	Patients carry <i>S. aureus</i>	52	190.32096	28.717107	0.05
	Patients non carry <i>S. aureus</i>	48	201.31092	19.327231	

Table (4) shows mean values of the IgE in patient carry *S. aureus* (274.05773pg/ml) higher than mean level of Control carry *S. aureus*(65.25567pg/ml) Present study showed a significant difference between IgE patients and control groups ($p < 0.001$).

Table (4) : Mean level of total IgE in in patients and controls carriers for *S. aureus*

Immunoglobulin	Patient	No.	Mean	Std. Error Mean	P-value
IgE	Patient carry <i>S. aureus</i>	52	274.05773pg/ml	20.382739	<0.001
	Control carry <i>S. aureus</i>	24	65.25567pg/ml	7.834469	

Table (5) shows mean values of the IgE in patients (261.93594 pg/ml) higher than mean level of Control (54.80353 pg/ml), present study showed a significant difference between IgE patients and control groups (p< 0.001).

Table (5): Mean level of total IgE in in patients and controls

Immunoglobulin	Patient	No.	Mean	Std. Error Mean	P-value
IgE	Patient	100	261.93594 pg/ml	19.047552	<0.001
	control	32	54.80353 pg/ml	6.799404	

Table (6) a significant difference in mean values of the IgE between patients carry *S.aureus* (274.05773 pg/ml) and control non carrier *S. aureu* (23.44713 pg/ml).

Table(6): Mean level of total IgE in patients carriers for *S. aureus* and control non carrier

Immunoglobulin	case	No.	Mean	Std. Error mean	P-value
IgE	Patient carry <i>S.aureus</i>	52	274.05773	20.382739	<0.002
	control non carrier	8	23.44713	5.16366	

Table (7) shows a significant difference in mean values of the IgE between patients carry *S. aureus* (and Patient non carrier *S. aureus*).

Immunoglobulin	case	No.	Mean	Std. Error mean	P-value
IgE	Patients without <i>S. aureus</i>	48	248.80400	33.116117	<0.001
	control without <i>S. aureus</i>	8	23.44713	5.163669	

Table(8): Mean level of total IgE in patients carriers for *S. aureus* and patient non carrier

Immunoglobulin	case	No.	Mean	Std. Error mean	P-value
IgE	Patient carry <i>S.aureus</i>	52	274.05773	20.382739	0.023
	Patient non carrier	48	248.80400	33.116117	

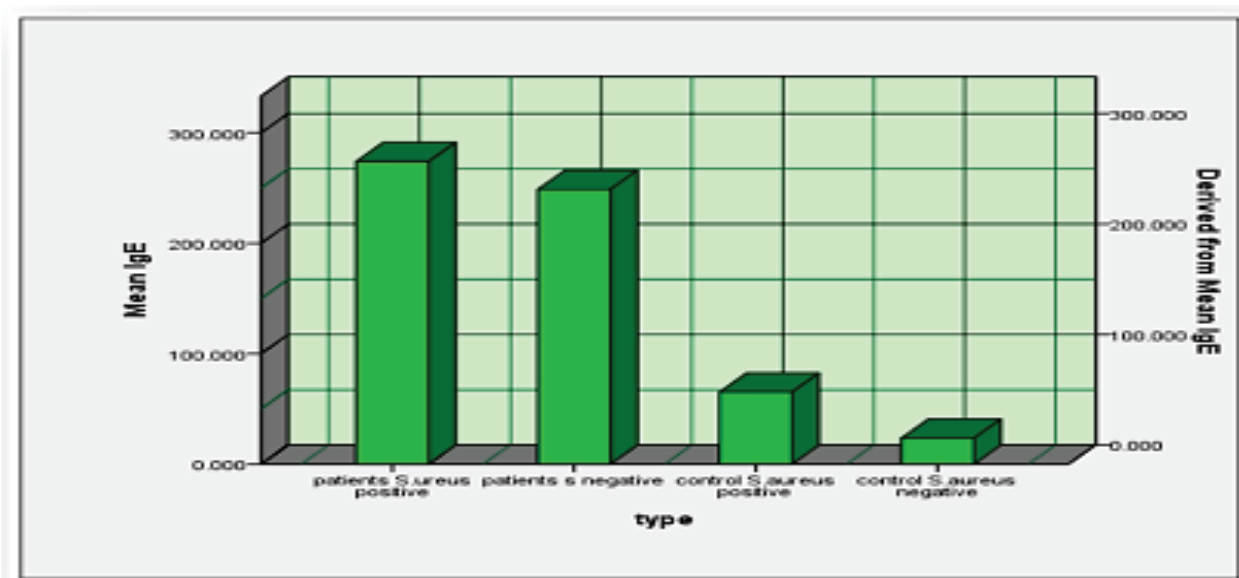


Fig (1):Distribution of IgE in different groups covered in the study

DISCUSSION

In this research, we concentrated on a typical nasal cavity bacterial flora. Table 1 examined the frequency of *S. aureus* isolation from the nasal cavity and showed that patients with AR carried more *S. aureus* (52% vs. 24% in controls). There was a big discrepancy between the two results (P 0.001, Odds 3.43336). *S. aureus* was more common in the nostrils of patients with allergic rhinitis. This table's outcome is consistent with [8] and he attributed a role in this *S aureus* occurrence in the nasal passages to the pathogenesis of the allergic rhinitis disease .also the result agree with [9 ; 10 ; 11].

Serum Levels of Cytokines among allergic rhinitis Cases and control group:

Serum Levels of IL-4 in patients and controls carriers for *S. aureus*,from the table (2) cytokine IL-4 mean level (327.56369 pg/ml) higher than mean level of control positive group (137.94304pg/ml) the present study showed a significant difference between AR patients and control groups (p<0.001) this finding was similar to those reported by [10] who found that patient with AR produced large amount of IL-4 after stimulation. The finding was in agreement with those of [12] who showed significantly higher concentrations of Th2 cytokines (IL-4, IL-5 and IL-6)

in patients with AR than in non-allergic patient, regarding IFN- γ the increment in the level of this cytokine the result of present study is agreement with a previous study that done by [13] which showed that AR patients formed increase significant differences IFN- γ than that from healthy subjects.

An explanation can be put for present result of this table (3) is that a comparison between the cytokine levels was done between two groups who were both carriers for *S.aureus* in their nasal cavities, the mean IL-4 of Patient carry *S.aureus* group was (327.56369 pg/ml) for Patient non carrier it was (277.24077pg/ml) and it was statistically significant ($0.037 < 0.05$), the consequence of *S. aureus* nasal carriage the nasal cytokine situation was seen by [14].

IL-4 is a significant contributor to airway allergic illness [26] These findings revealed that *S. aureus* superantigens activate T cells and release TH2 type cytokines in individuals with allergic rhinitis, which might enhance local inflammation and contribute to allergic rhinitis lesions. IL-4 is known to stimulate B cell IgE switching and eosinophilia [9]. The mean of IFN- was significantly greater in the noncarrier group compared to the carrier group (201.31092 pg/ml vs (190.32096 pg/ml) ($P=0.05$).

When exposed to staphylococcal superantigen, patients with allergic rhinitis and atopic dermatitis released more IL-4 and less IFN from their mononuclear cells. Theorizing that staphylococcal superantigens increase the TH2- and IgE-bias of the immune response in allergy sufferers [15].

The idea [16] that IL-4 is essential for the improvement of *S. aureus* adhesion to the skin, also took into account the fact that normal and atopic patients produced different amounts of IL-4 and IFN- γ [17].

Major Histocompatibility Complex class II expression is reported to be induced by interferon-gamma (IFN-) on bronchial epithelial cells in vitro [18]. Bronchial epithelial cells can behave as antigen-presenting cells and communicate with T cells since MHC class II molecule expression is increased in vivo in lung cancer and asthma [19]. Although MHC class II's primary function is to deliver antigens to T lymphocytes, activation of the class II receptor by superantigens and other bacterial products can also cause class II-expressing cells to secrete more cytokines and undergo apoptosis [20]. The cytokine network imbalances may play a major role in the pathogenesis of allergic rhinitis [21] *S. aureus* superantigen responses toward TH1 during severe disease, but toward Th2 response during atopic disease [21; 22].

A significant difference was found, between the two groups and this could be attributed to the both initiation factors ~ presence of *S.aureus* in the nasal cavity mucous and the allergy state ~ which when all together are present could make the cells of the nasal mucosa to excrete a higher levels of cytokines and initiate more allergic state, a fact that is an agreement with what was found by [8 ; 14].

Serum Levels of IgE in patients and controls in different state of carrier

As shown in table (4) a significant difference in mean values of the IgE between patients (+) carry *S. aureus* (274.05773 pg/ml) and control carrier *S. aureus* (65.25567 pg/ml). The results goes well with a study oof previous study [25] who revealed that he discovered a strong association ($r = 0.78, p0.01$) between nasal *S. aureus* numbers and serum total IgE. This study's finding concurs with those of [13], who found that the mean value of IgE levels in allergic versus non-allergic groups was statistically substantially different.

As shown in table (5) a significant difference in mean values of the IgE between patients (261.93594 pg/ml) and control (54.80353 pg/ml) the results goes well with a study of [18] who found that a important association between serum total IgE and count of bacteria.

As shown in table (6) a significant difference in mean values of the IgE between patients carry *S. aureus* (274.05773 pg/ml) and control non carrier *S. aureus* (23.44713 pg/ml) the results goes well with a study of [11].

As shown in table (7) a significant difference in mean values of the IgE between patients (+) carry *S. aureus* (274.05773 pg/ml) and patient non carrier *S. aureus* (248.80400 pg/ml). These results are in agreement with a research by [23], which discovered that allergic patients with nasal SA had blood serum total IgE levels that were greater than allergic non-carriers (101.90.3 kU/l; mean SD) but that did not achieve statistical significance ($P = 0.09$).

In this table (8) reported that the mean level of IgE in patients without *Staph aureus* higher than in Control without *S. aureus* and the difference was significant our finding were in agreement with those of others like [24] who found that the IgE levels were elevated in more than 90% of patients with allergic rhinitis.

The result of the present study in the line of previous study [27] who reported that a total serum IgE level was considered to be higher in allergic subjects than in healthy subjects.

According to several investigations, the existence of super antigen specific IgE (ssIgE) to *S. aureus* superantigens defined the exposure to staphylococcal superantigens. High serum total IgE levels were consistently correlated with ssIgE to SA-superantigens [14].

CONCLUSION

We were able to show that patients with allergic rhinitis had a much greater rate of *Staphylococcus aureus* nasal carriage than did control subjects.

Indicating an immunomodulatory function, *Staphylococcus aureus* was associated with high IL-4 and low IFN- levels. Additionally, the imbalance in the cytokine network may be a key factor in the development of allergic rhinitis.

The outcome of this investigation supports the idea that in people with allergic rhinitis, Staphylococcal superantigens augment the Th2 and IgE bias of the immune response.

There is no question that allergic rhinitis increases *Staphylococcus aureus* colonization, which may increase the risk of more serious staphylococcal infections in these patients.

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Acceptance Letter

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Dear (author) We are pleased to inform you that your manuscript

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