The Correlation between Nasal *Staphylococcus aureus* and Persistent Allergic Rhinitis in Comparison with Atopic Dermatitis

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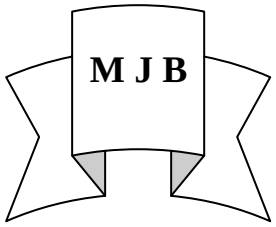

Abstract

In this study(224)nasal swabs were collected from Asthma and Allergy Center,AL-Hilla teaching Hospital and Private clinic in Hilla city and included three groups: Persistent Allergic Rhinitis Group,Atopic Dermititis Group and healthy subjects as Control Group to study the correlation between nasal carriage rate of ***staphylococcus aureus*** (***S. aureus***) and these two diseases and the development of symptoms of Persistent Allergic Rhinitis Patients .It was found that the rate of isolated ***S. aureus*** was higher in Persistent Allergic Rhinitis Group(86)% in comparison with Atopic Dermititis Group (63.7)% and the incidence of Persistent Allergic Rhinitis in (10-20) year age group was higher than other age groups (29.5)%. Isolated ***S. aureus*** was higher in male(92.9)% than female(78.7)%,as well as symptoms of Persistent Allergic Rhinitis and the rate of incedince of ***S. aureus*** were decreased with proceeding in age and symptoms showed high rate in (10-20) year age group.

Antibiotics sensitivity test studied of the isolated ***S. aureus*** showed that all isolates were highly sensitive to Vancomycin, and moderately sensitive to Oxacillin (84.1)% and Ciprofloxacin(61.4)%.

الخلاصة

تم في هذه الدراسة جمع٢٢٤ مسحة انف من مركز الحساسية والربو ومستشفى الجمهوري وعيادات خاصة في مدينة الحلة تضمنت ثلاثة مجاميع :مجموعةالتهاب الانف التحسسي المزمن ومجموعة التهاب الجلد التحسسي ومجموعة الاشخاص الاصحاء (مجموعة السيطرة) لدراسة العلاقة بين نسبة بكتريا ال ***S. aureus*** في الانف لهؤلاء المرضى مع هذين المرضين وتطور اعراض مرض التهاب الانف التحسسي المزمن.لقد وجدنا بان نسبة بكتريا ال***S. aureu*** هي اكثر لدى مرضى التهاب الانف التحسسي المزمن مقارنة بمرضى التهاب الجلد التحسسي .كذلك وجدنا بان نسبة حدوث التهاب الانف التحسسي المزمن في الفئة العمرية (١٠-٢٠) سنة هي اكثر من باقي الفئات.بكتريا ال***S. aureus*** المعزولة كانت اكثرلدى الذكور مقارنة بالاناث،بالاضافة الى ان اعراض التهاب الانف التحسسي المزمن ونسبة حدوث ال-***S. aureus*** تقل مع تقدم العمركما اظهرت الاعراض اكثر نسبة في الفئة العمرية (١٠-٢٠)سنة.

العينات المعزولة اخضعت لاختبار فحص الحساسية وقد اظهرت جميعها حساسية عالية لعقارVancomycin و كانت حساسة ايضا لعقاري oxacillin (٨٤،١)% و Ciprofloxacin (٦١،٤)%.

Introduction

S. aureus has long been recognized as an important pathogen in human disease. Due to an increasing number of infections caused by methicillin-resistant ***S. aureus*** (MRSA) strains, therapy has become problematic. Therefore, prevention of staphylococcal infections has become more important. Carriage of ***S. aureus*** appears to play a key role in the epidemiology and pathogenesis of infection. The ecological niches of ***S. aureus*** are the anterior nares. In healthy subjects, over time, three patterns of carriage can be distinguished: about 20% of people are persistent carriers, 60% are intermittent carriers, and approximately 20% almost never carry ***S. aureus.*** [1,2].

Rhinitis is a global problem and is define as the presence of at least one of the following: congestion, rhinorrhea, sneezing, nasal itching, and nasal obstruction. The two major classifications are allergic and nonallergic rhinitis. Allergic rhinitis occurs when an allergen is the trigger for the nasal symptoms. Nonallergic rhinitis is when obstruction and rhinorrhea occurs in relation to nonallergic, noninfectious triggers such as change in the weather, exposure to caustic odors or cigarette smoke, barometric pressure differences, etc [3]. In Western countries between 10–25% of people annually are affected by allergic rhinitis [4].

Allergic rhinitis may also be classified as Mild-Intermittent, Moderate-Severe intermittent, Mild-Persistent, and Moderate-Severe Persistent. Intermittent is when the symptoms occur <4 days per week or <4 consecutive weeks. Persistent is when symptoms occur >4 days/week and >4 consecutive weeks [5]. In such individuals, the allergen triggers the production of the antibody immunoglobulin E (IgE) causing the release of inflammatory mediators such as histamine (and other chemicals)[6].

Atopic dermatitis (AD) is a chronic inflammatory skin disease associated with cutaneous hyperreactivity to environmental triggers that are innocuous to normal nonatopic individuals [7]. AD usually presents during early infancy and childhood, but it can persist into or start in adulthood [8]. In addition to increased colonization, 50–60% of the ***Staphylococcus aureus*** found in AD patients are toxin producing [9] Furthermore, AD patients can rapidly advance to superinfection with 10^7 organisms per cm^2 in acute lesions, as opposed to nonatopic subject who maintain a low bacterial burden [10]. About 30% of patients with AD report bacterial infections [11].

Patients and Methods

(224) nasal swabs were collected from patients attending Asthma and Allergy Center, Hilla Teaching Hospital and Private clinic in Hilla city. They were divided into three groups (78) Persistent Allergic Rhinitis Group, (66) Atopic Dermatitis Group and (80) healthy subjects as Control Group.

The nasal swabs were inoculated on culture media (Trypticase soy agar (TSA) with 5% sheep blood agar, Mannitol salt agar) and incubated at 37°C for 24hr.Traditional biochemical tests were used for final identification of bacterial isolates. The traditional method for differentiating ***S. aureus*** (coagulase positive) from coagulase-negative staphylococci in the laboratory has been the tube coagulase test (TCT). This test has been modified for direct detection of the organism from blood cultures in 2 h by using the blood culture broth [12, 13].

Some antibiotic disks were used to show the effect of them on isolated bacteria using disk diffusion method. Suspension with bacteria was swabbed over a Mueller-Hinton agar. Discs impregnated with various antibiotics are placed onto agar and incubated for 24 hours at 37 °C. Zones of inhibition of bacterial growth, measured around discs, were compared with those in the National Committee for Clinical Laboratory Standards (NCCLS) [14].

Statistical analysis

All data analyzed by using Chi-square and Student s unpaired t -test. A P- value <0.05 was considered statistically significant [15].

Results and Discussion

Nasal carriage of ***S. aureus*** exerts immunomodulatory effect in patients with Atopic Dermatitis and it

may contribute to airway inflammation and allergic response in patients with allergic rhinitis [16]. So this study was designed to investigate the frequency of nasal ***S.aureus*** carriage in Persistent Allergic Rhinitis patients and its possible influence on their symptoms in comparison with the frequency of nasal ***S.aureus*** carriage in patients with Atopic Dermatitis .It was chosed (78) non smoker patients with Persistence Allergic Rhinitis (matched for age , sex and symptoms), (66) non smoker Patients with Atopic Dermatitis and (80) non smoker healthy subjects, For all subjects rhinoscopy was done, the Persistent Allergic Rhinitis group showed high nasal carriage rate of ***S. aureus*** (86)% in comparison with Atopic Dermatitis group (63.7)% and control group (28.8)% (P<0.05 significant difference) as illustrated in Table (1).

Nasal ***S.aureus*** actively modulated the immune reaction in persistent allergic rhinitis patients by promoting local IgE production as well as patients with allergic rhinitis suffering from ulceration of nasal mucosa ,this may lead to increase colonization of staph aureus in nose [16]. The anterior nares have been shown to be the main reservoir of ***S. aureus*** in both adults and children.

The ***S. aureus*** is transmitted to nares by contaminated hands and from surfaces where it can survive for months [17] ,this may be another factor for increasing of nasal ***S. aureus*** in Atopic Dermatitis patients by contaminated hand especially after itching of the skin.

Chronic skin colonization with ***S. aureus*** is a characteristic feature of Atopic Dermatitis (AD) [18].About 60-90% of ***S. aureus*** strains isolated from the skin AD patients (19).Bacterial counts on unaffected skin are lower than on affected a topic skin [20].

Table (2) shows the rate of nasal ***S. aureus*** which was higher in (10-20) year age group(31.3%) in comparison with other age groups. Persistent carriage of ***S. aureus*** is more common in children than in adults [21].

The symptoms also were higher in (10-20) year age group (29.5) % in comparison with other age groups. Total immunoglobulin E levels increased with age until (14-15) years, and declined thereafter [22]. Allergic rhinitis (AR) is characterized by one or more symptoms including sneezing, itching, nasal congestion, and rhinorrhea [23],so the symptoms in table (2) represent one or more of sneezing, itching, nasal congestion, and rhinorrhea. Allergic diseases of the airways, including asthma and allergic rhinitis, remain highly prevalent with associated symptoms placing considerable restrictions on the physical, emotional, and social functioning of the affected individual. It is now recognized that immunoglobulin E (IgE) plays a central role in mediating the allergic response that follows exposure to allergens after an initial process of sensitization [24].

Both of nasal ***S. aureus*** carriage rate and symptoms in Persistent Allergic Rhinitis patients showed high prevalence in (10-20) year age group and decrease as the age proceed. Table (3) shows the nasal carriage rate of ***S. aureus*** in male (92.9)% which was higher than female (77.8)%(P<0.05 significant difference). So we recommend that ***S. aureus*** carriage must be screened on regular intervals in Persistent Allergic Rhinitis and Atopic Dermatitis patients. Nasal ***S. aureus*** carriage follow-up and treatment is a process that will protect patients from more severe clinical pictures. Antibiotics sensitivity tests showed high sensitivity (100%) of ***S.***

aureus to Vancomycin and in lesser degree of other antibiotics as showed in Table (4).

References

1-Kluytmans J.,van Belkum A., and Verbrugh H.(1997). Nasal carriage of ***Staphylococcus aureus***: epidemiology, underlying mechanisms, and associated risks. Clin Microbiol Rev. July; 10(3): 505–520.

2-Von Eiff C, Becker K, Machka K, Stammer H. and Peters G.(2001). Nasal carriage as a source of ***Staphylococcus aureus*** bacteremia. Study Group. N Engl J Med. Jan 4;344(1):11-6.

3-Tran N., Vickery J.and Blaiss M.(2011). Management of rhinitis: allergic and non-allergic. Jul;3(3):148-56.

4-Bousquet J, Reid J and Van Weel C.(2008). Allergic rhinitis management pocket reference . Allergy Aug; 63 (8):990-996.

5-Dykewicz M. and Hamilos D.(2010). "Rhinitis and sinusitis". J. Allergy Clin. Immunol, 125 (2 Suppl 2): S103–15.

6-May J. and Smith P.(2008), Allergic Rhinitis. In: Dipiro JT, Talbert RL, Yee GC, et al eds.Pharmacotherapy: A Pathophysiologic Approach. 7th ed. New York, NY: The McGraw-Hill Companies, Inc; :1565-1575).

7-Leung D. and Bieber T.(2003). Atopic dermatitis. Lancet. 361**:**151-160.

8-Spergel, J.and Paller A. (2003) .Atopic dermatitis and the atopic march. J. Allergy Clin. Immunol. 2003. **112:**S128-S139.

9-Akiyama H., Toi Y., Kanzaki H., Tada J.and Arata J .(1996). Prevalence of producers of enterotoxins and toxic shock syndrome toxin-1 among ***Staphylococcus aureus*** strains isolated from atopic dermatitis lesions. Arch Dermatol Res 288:418–420.

10-Leung D.and Bieber T. (2003).Atopic dermatitis.Lancet 361: 151–160.

11-Christophers E.and Henseler T. (1987), Contrasting disease patterns in psoriasis and atopic dermatitis. Arch Dermatol Res 279(Suppl):S48–S51.

12-Garrity G.(2001). Bergy's Manual of systematic Bacteriology. 2nd ed. Springer-Verlage , N.Y. vol.3., Group-17.

13- Forbes B., Sahm D. and Weiss feld, A.(1998). Baily & Scotts. Diagnostic Microbiology . 10th ed. Vol.1 , Mosby co. St Louis., pp:68-280.

14-Bauer R.,Kirby M.,Sherris J. and Turck M.(1966).Antibiotics by standardized single disk method. Amercan Journal of Clinical Pathology.45:493-496.

15-Daniel W. and Mugge. (1995).Transesophageal echocardi-ography. N Engl J Med. 332:1268-1279.

16- Maged M., Tarek M., Zeinab A., Mohamed Y.(2008). Immunological role of nasal ***staphylococcus aureus*** carriage in patients with persistent allergic rhinitis.The Pan African Medical Journal;1:3.

17-Van Belkum A., Verkaik N., Vogel C., Boelens H., Verveer J.and Nouwen J.(2009). Reclassification of Staphylococcus aureus Nasal Carriage Types. J Infect Dis. Jun 15 ;199(12):1820-6.

18-Breuer K., HAussler S., Kapp A. and Werfel T. (2002). ***Staphylococcus aureus***: colonizing features and influence of an antibacterial treatment in adults with atopic dermatitis. Br J Dermatol 147:55–61.

19-Guzik T., Bzowska M., Kasprowicz A., Czerniawska-Mysik G., Wojcik K. and Szmyd D. (2005). Persistent skin colonization with ***Staphylococcus aureus*** in atopic dermatitis: relationship to clinical and

immunological parameters. Clin Exp Allergy 35:448–455.

20-Leung D., Nicklas R., Li J., Bernstein I., Blessing-Moore J.,and Boguniewicz M. (2004). Disease management of atopic dermatitis: an updated practice parameter. Joint task force on practice parameters. Ann Allergy Asthma Immunol 93:S1–S21.

21-Kluytmans J, Van Belkum A and Verbrugh H.(1997). Nasal carriage of ***Staphylococcus aureus***: epidemiology, underlying mechanisms, and associated risks. Clin Microbiol Rev , 10(3):505-520.

22-Bousquet J, Reid J.and Van Weel C.(2008). Allergic rhinitis management pocket reference . Allergy Aug; 63 (8):990-996.

23-Skoner D.(2001). Allergic rhinitis: definition, epidemiology, pathophysiology, detection, and diagnosis. J Allergy Clin Immunol Jul;108(1 Suppl):S2-8

24-Berger W.(2006).Treatment of allergic rhinitis and other immunoglobulin E-mediated diseases with anti-immunoglobulin E antibody. 27(2):29-32.

Table 1 Distribution of Nasal *S.aureus* incidence in patients groups and Control Group.

S. aureus	Atopic Dermatitis		Persistence Allergic Rhinitis		Control Group		Total	
	No.	%	No.	%	No.	%	No.	%
+	42	63.7	67	86	23	28.8	132	58.9
–	24	36.3	11	14	57	71.2	92	41.1
Total No.	66	100	78	100	80	100	224	100

P<0.05

Table 2 shows the distribution of Nasal *S.aureus* incidence in Persistence Allergic Rhinitis according to age and symptoms.

Persistent Allergic Rhinitis	Age(year)										Total	
	10-20		21-30		31-40		41-50		51-60			
	No.	%	No	%	No	%	No	%	No	%	No.	%
+ s.aureus isolates	21	31.3	17	25.3	15	22.4	9	13.5	5	7.5	67	100
symptoms	23	29.5	19	24.3	16	20.5	12	15.4	8	10.3	78	100

P<0.05

Table 3 Distribution of Nasal *S.aureus* in Persistent Allergic Rhinitis according to sex.

Study group	Sex						Total	
	Male			Female				
	Total No	No.+	%	Total No	No.+	%	Total No	Total No+
Persistent Allergic Rhinitis	42	39	92.9	36	28	77.8	78	67

P<0.05

Table 4 Susceptibility of isolated *S. aureus* to different antibiotics.

Antibiotics	Result				Total	
	Sensitive		Resistance			
	No.	%	No.	%	No.	%
Vancomycin	132	100	0	0	132	100
Oxacillin	111	84.1	21	15.9	132	100
Ciprofloxacin	81	61.4	51	38.6	132	100