

Original Research Article

Nasal flora in allergic rhinitis

Valli Rajasekaran*, Sharath Kumar B. V., Gurumani Sriraman

Department of Otorhinolaryngology, Shri Sathya Sai Medical College and Research Institute, Thirupurur-Chennai, Tamil Nadu, India

Received: 22 August 2017

Accepted: 14 September 2017

*Correspondence:

Dr. Valli Rajasekaran,

E-mail: valli_rajasekaran@yahoo.co.in

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Allergic rhinitis is a common cause for recurrent rhinosinusitis. The microbiology in allergic nasal mucosa has not been much documented. The aim of the study is to identify the microbes in the middle meatus in patients with allergic rhinitis and to compare with the normal nasal flora.

Methods: A cross sectional study was conducted in our institute to study the nasal microbial pattern in 50 patients with allergic rhinitis and was compared with 50 normal healthy nasal flora. Nasal swabs were taken from middle meatus under endoscopic guidance in both the groups and sent for microbial analysis.

Results: Organisms like *Klebsiella*, *E.coli*, and *Staphylococcus aureus* were predominant isolates in patients with allergic rhinitis, whereas Staph epidermidis were predominant in controls.

Conclusions: This alteration in microbial flora could possibly explain recurrent sinonasal infections in patients with allergic rhinitis.

Keywords: Allergic rhinitis, Nasal flora, Commensals, Nasal swab

INTRODUCTION

Allergic rhinitis is an IgE mediated hypersensitivity of nasal mucosa characterised by sneezing, nasal itching, watery nasal discharge and nasal obstruction. Allergic rhinitis can be associated with other atopic conditions like bronchial asthma and atopic dermatitis.

Allergic rhinitis is one of the most common diseases affecting all age groups. Allergic rhinitis is a global health problem affecting social life, sleep, school and work with a significant economic impact.¹ The worldwide prevalence of allergic rhinitis is estimated to be 10-30% in adults and about 40% in children.² This increasing incidence can be explained by “hygiene hypothesis” according to which decreasing rate of infection in early life explains increase in allergy.³ According to “microbial hypothesis” change in microbes

due to changes in lifestyle plays a significant role in development of immune system.⁴⁻⁶

Allergic rhinitis is a major risk factor for acute bacterial rhinosinusitis.^{7,8} Allergic rhinitis creates an environment susceptible for bacterial infections either by disruption of immune defence or by causing a th2 type of inflammatory reaction in the nasal mucosa.⁹ Recently airway “superantigen hypothesis” has explained production of local mucosal IgE in nasal mucosa due to local bacterial superantigens which is acting as a potent airway inflammatory cofactor.¹⁰ Allergy can cause inflammation of nasal mucosa and impair ciliary function. This can block the drainage of paranasal sinuses, thereby predisposing to stasis of secretions and consequently infection of sinuses.

Normal microbial flora denotes the population of microorganisms that inhabit the skin and mucous

membrane of normal persons. On the mucous membrane, the resident flora prevents colonisation by pathogens and possible disease through bacterial interference. Disruption of the normal flora can predispose to other pathogenic microbes.

In this study we have identified the microbes in nasal smears of patients with allergic rhinitis and compared it with the flora in normal individuals.

Objectives

1. To identify the nasal flora in patients with allergic rhinitis.
2. To compare the nasal flora in allergic rhinitis patients with the nasal flora in normal individuals.

METHODS

The study was conducted in ENT outpatient department of Shri Sathya Sai Medical College and Research Institute. The study was carried out after obtaining clearance from Institutional Ethical Committee.

Study type: Case control study

Study duration: December 2015 – December 2016.

Sample size: 50 cases and 50 controls

Inclusion criteria

Inclusion criteria were patients presenting to ENT OPD with symptoms (sneezing / watery nasal discharge/ nasal obstruction) and signs (pale nasal mucosa / hypertrophy of turbinates) of allergic rhinitis with elevated serum IgE; all ages and both sexes.

Exclusion criteria

Exclusion criteria were patients having purulent nasal discharge or post nasal drip; patients with congested nasal mucosa; patients using steroid nasal sprays.

Controls

Normal subjects, without any nasal complaints, who were willing to participate in the study, all ages and both sex.

Serum IgE level: Serum IgE level was estimated using immunocap method for IgE specific test. Patients with serum IgE level more than 100Ku/L were considered to have elevated serum IgE levels.

Sterile swab was taken from the middle meatus using 0°endoscope and sent for Gram stain and culture sensitivity, where in the microbiologist was not aware of the cases and controls, thereby making the study single blinded. The swab was transported in Stuart’s medium, charcoal based medium. Gram staining was done. The

culture was done in blood agar, chocolate agar and Mc conkey’s agar. The culture plates were incubated at 37° c for 24-48 hrs. The organisms were identified and tabulated.

The organisms identified in both cases and controls were tabulated and the differences were compared using Fischer exact test.

RESULTS

Patients of all age groups were included in the study, the range being 8 years to 62 years (Figure 1). The maximum number of patients sampled were between 16 years to 45 years. Among the patients sampled 60% (n=30) were females, rest 40% (n=20) were males (Figure 2). The age and sex of the controls were matched to that of the cases.

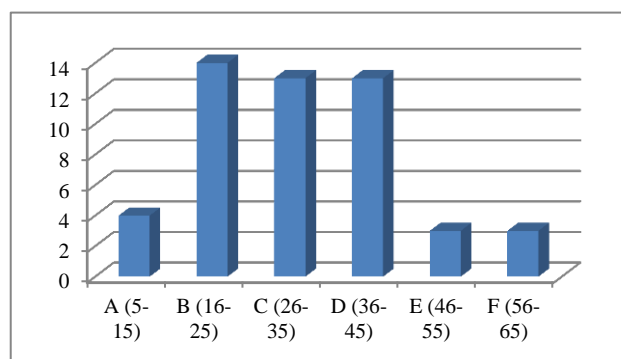


Figure 1: Age wise distribution of cases.

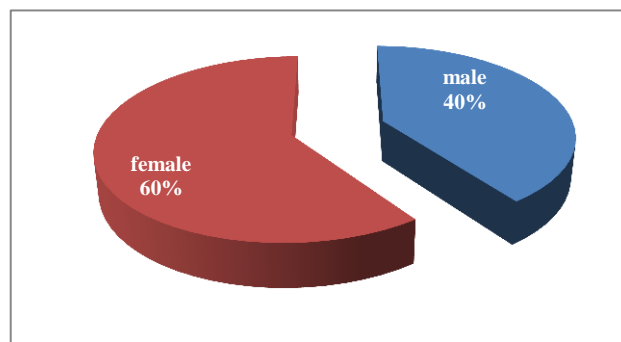


Figure 2: Sex distributions of cases.

Of the 50 patients with allergic rhinitis, the predominant isolate was *Staphylococcus epidermidis*, 80% (n=40). Other organisms were diptheroids 70% (n=35), *S. aureus* 40% (n=20), *Klebsiella pneumoniae* 30% (n=15), *Pneumococcus* 20% (n=10), *Hemophilus influenzae* 10% (n=5), *Escherichia coli* 2% (n=1) and *Micrococci* 2% (n=1).

Control group was 50 in number. Predominant isolate was *Staphylococcus epidermidis* 96% (n=48) (Figure 3).

Pathogenic organisms like *Klebsiella pneumoniae* and *E. coli* were isolated only in cases, in spite of the patients

having no signs of active infection when the swab was taken. In addition, *S. aureus* was more in allergic rhinitis patients which is usually a normal commensal in the

nose. The predominant normal commensal, *Staphylococcus epidermidis* was reduced in patients with allergic rhinitis.

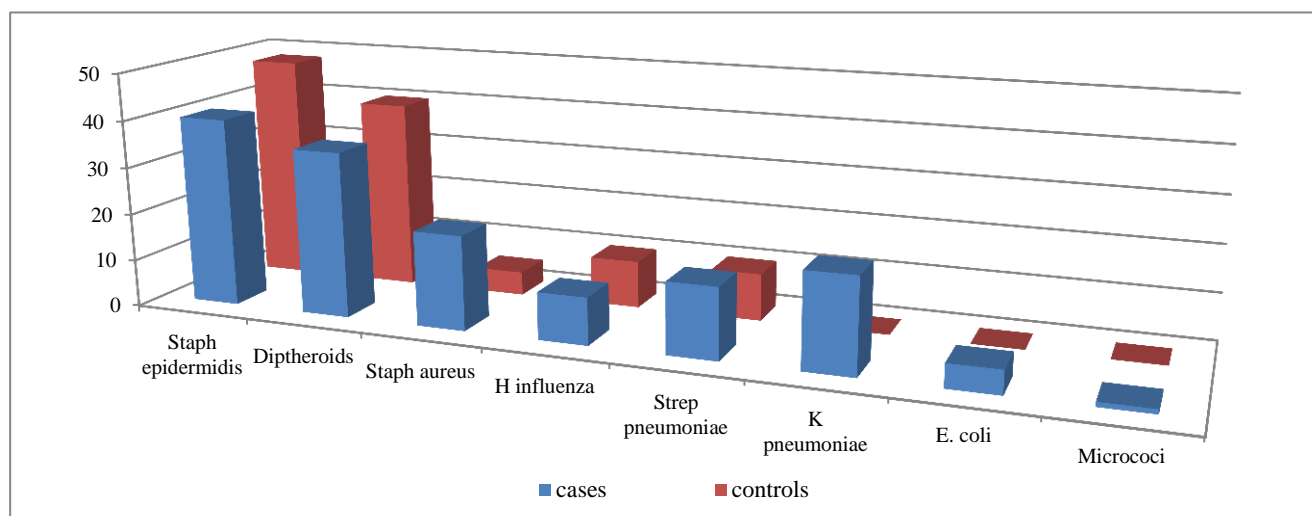


Figure 3: Graph representing the growth of organisms in cases and controls.

Table 1: Comparison between organisms in cases and controls.

Organisms	Cases	Controls	P value
Gram positive:			
<i>Staph epidermidis</i>	40	48	0.028 (significant)
<i>Diptheroids</i>	35	40	0.248
<i>Staph aureus</i>	20	05	0.0005 (significant)
<i>Strep pneumoniae</i>	15	10	0.248
<i>H influenza</i>	10	10	1.00
<i>Micrococci</i>	01	00	1
Gram negative:			
<i>K.pneumoniae</i>	20	00	<0.000 (significant)
<i>E.coli</i>	05	00	<0.05 (significant)

The organisms identified were tabulated and the differences in the profile of the organisms were studied. Statistical analysis was done using Fischer exact test. The p value for gram negative organisms like *Klebsiella pneumoniae* and *E.coli* was <0.000 and <0.05 respectively which is statistically significant. The p value for gram positive organisms like *Staphylococcus epidermidis* and *S. aureus* was <0.028 and <0.0005 respectively which is statistically significant (Table 1). *Klebsiella*, *E.coli* and *S. aureus* were more in patients with allergic rhinitis and *Staphylococcus epidermidis* was more in controls.

DISCUSSION

Our study shows an alteration in nasal microbial flora in patients with allergic rhinitis. Growths of organisms like *E.coli*, *Klebsiella pneumoniae* were seen only in patients with allergic rhinitis. *Staphylococcus aureus* which is a normal commensal was seen more in patients with

allergic rhinitis. On the other hand, *Staphylococcus epidermidis* was down regulated in patients with allergic rhinitis.

Identification of organisms like *Klebsiella* and *E. coli* can explain increase in episodes of suppurative infections in patients with allergic rhinitis. In addition, there is a decrease in normal microbes which can interfere with the normal protective functions of normal flora and can create a favourable environment for pathogenic organisms, thereby predisposing to recurrent infections.

The findings in our study is supported by Choi et al, in which swab from middle meatus when subjected to restricted fragment length polymerase, which showed an increase in bacterial number and diversity in swabs from middle meatus during seasonal allergic rhinitis.¹¹ This study provides evidence that certain microbes can be characteristically identified in allergic rhinitis.

Presence of microbes in pollen was demonstrated in study by Heydenreich et al.¹² These microbes in pollen can act as adjuvant and initiate an allergic immune response. The microbes in pollen could possibly explain the change in flora and recurrent infection in allergic rhinitis patients.

S. aureus is found in abundance in skin of individuals with atopic eczema.¹³ This supports the finding in our study where *Staphylococcus aureus* is found more in middle meatus in patients with allergic rhinitis than in controls.

In a similar study by *S. Berrettini* the predominant organism isolated was *S. aureus* followed by *Klebsiella* and *Pseudomonas*. Anaerobic organisms were not detected.¹⁴ Pathogenic organisms like *Klebsiella* were identified in middle meatus as in our study.

Patients with allergic rhinitis are at risk of developing recurrent episodes of bacterial rhinosinusitis. Our findings go in favour of this. The alteration of microbial flora could be a reason for recurrent infections in patients with allergic rhinitis.

Studying the sensitivity pattern of these organisms, can help us in choosing and initiating treatment with an appropriate antibiotic.

CONCLUSION

The nasal mucosa when exposed to allergens can create a favourable environment for growth of certain organisms like *Klebsiella* and *E. coli* which are not identified as normal nasal commensals. These organisms can derange the mucosal immune mechanisms. These organisms with these altered immune responses can predispose to infections when the conditions become conducive.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards

REFERENCES

1. Bousquet J, Khaltaev N, Cruz AA, Denburg J, Fokkens WJ, Togias A, et al. Allergic Rhinitis and its Impact on Asthma (ARIA) 2008 update (in collaboration with the World Health Organization, GA(2)LEN and AllerGen). *Allergy*. 2008;63(86):8-160.
2. Pawankar R, Canonica GW, ST Holgate ST, Lockett RF, Blaiss M. The WAO White Book on Allergy. Update. 2013: 27.
3. Okada H, Kuhn C, Feillet H, Bach JF. The 'hygiene hypothesis' for autoimmune and allergic diseases: an update. *Clin Experimental Immunol*. 2010;160(1):1-9.
4. Orrhage K, Nord CE. Factors controlling the bacterial colonization of the intestine in breastfed infants. *Acta Paediatr Suppl*. 1999;88:47-57.
5. Dominguez-Bello MG, Costello EK, Contreras M, Magris M, Hidalgo G, Fierer N, et al. Delivery mode shapes the acquisition and structure of the initial microbiota across multiple body habitats in newborns. *Proc Natl Acad Sci USA* 2010;107:11971-5.
6. Penders J, Thijs C, Vink C, Stelma FF, Snijders B, Kummeling I, et al. Factors influencing the composition of the intestinal microbiota in early infancy. *Pediatrics*. 2006;118:511-21.
7. Clin N, Kirtsreesakul V, Naclerio RM. Role of allergy in rhinosinusitis. *Current Opinion Allergy Clin Immunol*. 2004;4(1):17-23.
8. Dykewicz MS, Hamilos DL. Rhinitis and sinusitis. *J Allergy Clin Immunol*. 2010;125(2):103-15.
9. Barnes PJ. Pathophysiology of allergic inflammation. *Immunol Rev*. 2011;242(1):31-50.
10. Bernstein JM, Kansal R. Superantigen hypothesis for the early development of chronic hyperplastic sinusitis with massive nasal polyposis. *Current Opinion Otolaryngol Head Neck Surg*. 2005;13(1):39-44.
11. Choi CH, Poroyko V, Watanabe S, Jiang D, Lane J, deTineo M, et al. Seasonal allergic rhinitis affects sinonasal microbiota. *Am J Rhinol Allergy*. 2014;28(4):281-6.
12. Heydenreich B, Bellinghausen I, König B, Becker WM, Grabbe S, Petersen A, et al. Gram-positive bacteria on grass pollen exhibit adjuvant activity inducing inflammatory T cell responses. *Clin Experimental Allergy*. 2012;42(1):76-84.
13. Rosenthal M, Goldberg D, Aiello A, Larson E, Foxman B. Skin microbiota: microbial community structure and its potential association with health and disease. *Infection, Genetics Evol*. 2011;11(5):839-48.
14. Berrettini S, Carabelli A, Sellari-Franceschini S, Bruschini L, Quartieri F, Sconosciuto F, et al. Perennial allergic rhinitis and chronic sinusitis: correlation with rhinologic risk factors. *Allergy*. 1999;54(3):242-8.

Cite this article as: Rajasekaran V, Sharath KBV, Sriraman G. Nasal flora in allergic rhinitis. *Int J Otorhinolaryngol Head Neck Surg* 2017;3:904-7.