wjpmr, 2018,4(10), 171-174

SJIF Impact Factor: 4.639

WORLD JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.wjpmr.com

Research Article

ISSN 2455-3301

WJPMR

A STUDY TO KNOW THE CORRELATION BETWEEN BRONCHIAL ASTHMA, NASAL POLYPOSIS AND ALLERGIC RHINITIS

Sushil Jha, Vikas Sinha, Jigna Savani*, Shruti Ganvit and Swati Jha

India.

***Corresponding Author: Jigna Savani**

India.

Article Received on 19/08/2018 **Article Revised on 09/09/2018** **Article Accepted on 30/09/2018**

ABSTRACT

Nasal allergy and bronchial asthma mostly coexist with each other but not all patients with nasal allergy have bronchial asthma. At the same time there are proven evidences to show that there is a relationship between allergy and nasal polyposis and asthma. In our study, we would like to find the correlation between all three entities viz- allergic rhinitis, nasal polyposis and bronchial asthma.

KEYWORDS: Nasal allergy, asthma.

INTRODUCTION

Nasal allergy, asthma and nasal polyposis share a common etiology of allergy. As per the hygiene hypothesis these allergic disease are common in developed countries than in developing countries, but this form one of the major health issue.

AIM OF THE STUDY

The aim of the study is to show direct correlation between allergic rhinitis, bronchial asthma and nasal polyposis.

MATERIALS AND METHODS

In this study, 100 subjects were taken who presented with symptoms of allergic rhinitis or asthma during a period of January 2017 to July 2018 at Sir T. Hospital and Government medical college, Bhavnagar. Then these patients were asked to undergo routine history taking and examination with nasal endoscopy to look for allergic changes and allergic Sino nasal polyposis. To confirm the asthma pulmonary function tests are done FEV1 is noted.

Inclusion criteria

a) Patients who have complains of breathlessness on exertion.
b) Patients with unilateral/bilateral nasal blockage.
c) Patients having seasonal variation with nasal symptoms (running nose, sneezing, itching and nasal blockage).

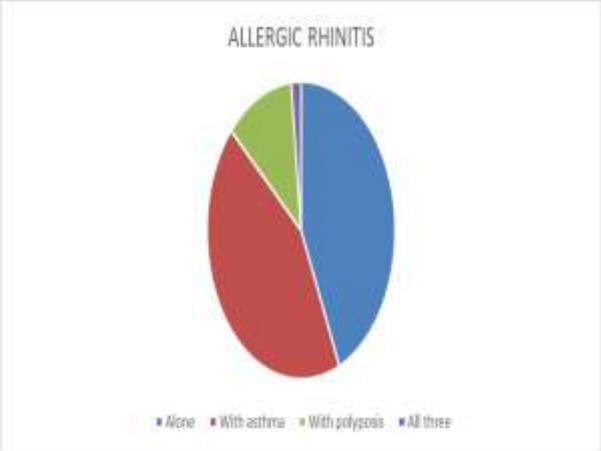
Exclusion criteria

a) Patients who are suspected cases of tuberculosis or past history of tuberculosis.
b) Pregnant women
c) COPD
d) Chronic smokers
e) Lung malignancy

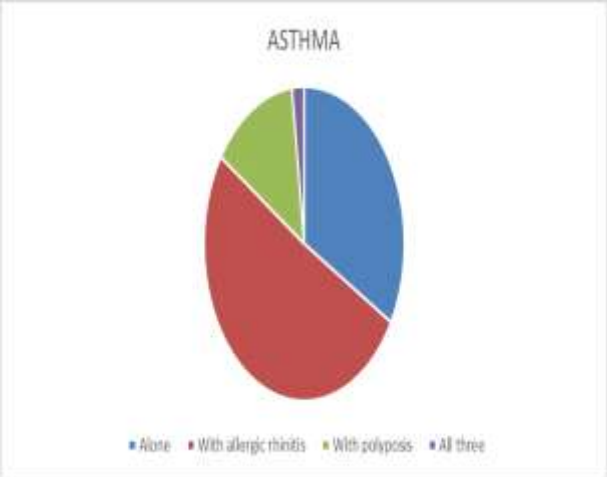
OBSERVATIONS AND RESULTS

Allergic Rhinitis + Asthma	26%
Allergic Rhinitis + Nasal Polyp	7%
Asthma + Nasal Polyp	7%
Allergic Rhinitis Alone	26%
Nasal Polyposis Alone	16%
Asthma Alone	17%
Allergic Rhinitis + Asthma + Nasal Polyp	1%

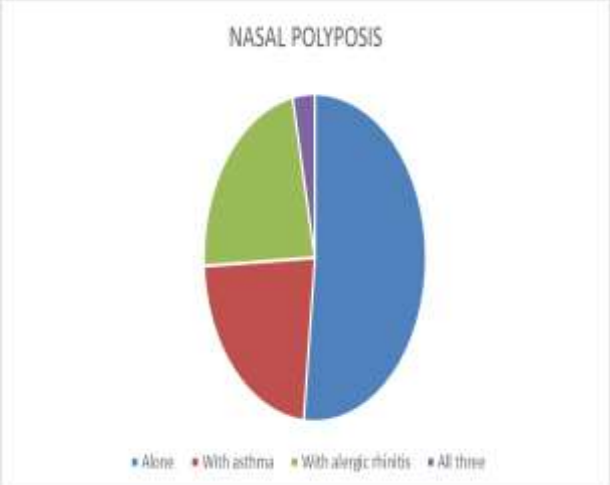
Out of the 100 patients who were examined we found that allergic rhinitis is commonest among all three. In this we observed 26 patients of allergic rhinitis alone, 26 patients had allergic rhinitis with asthma, 7 patients had allergic rhinitis with nasal polyposis 7 and 1 had all three.



Asthma is the second most common among all three and 26 patients had asthma with allergic rhinitis, 7 had asthma with polyposis, 17 had asthma alone and one had all three.



Least common among all three is nasal polyposis, 16 patients had polyp only, 7 patients had polyp with asthma, 7 had polyp with allergic rhinitis, and one had all.



DISCUSSION

In our study we had find a positive correlation with allergic rhinitis and asthma but in case of nasal polyposis both asthma and allergic rhinitis has equal incidence with polyposis.

The Copenhagen Allergy Study states that between 42 and 52 percent of patients with rhinitis had asthma and more than 99 percent of subjects with allergic asthma also had allergic rhinitis.[1]

It is also showed that the risk of getting asthma in people with allergic rhinitis is more than 300 times when compared with patients who are not having allergic rhinitis.

As mentioned earlier allergic disease are common in developed countries and urban areas than developing countries, recent study showed that allergic rhinitis is far less common among asthmatic subjects in rural china than in asthmatic subjects in industrialized countries with a western lifestyle.[2]

The age in which a child is getting atopy is also an important confounding risk factor for developing asthma. In an Australian study, it was found that atopy acquired at an early age(before the age of six years) is an important predictive factor for asthma continuing into late childhood, if atopy acquired later it was strongly associated with seasonal allergic rhinitis.[3,4]

It was shown that the allergic rhinitis patients have increased bronchial sensitivity to methacholine or histamine/ especially during and slightly after the pollen season.[5]

If we consider the causative agents for allergic rhinitis and bronchial asthma, most of the agents like pollen grain etc. commonly affect both nasal and bronchial mucosa.

The Children's Respiratory study showed that the presence of physician-diagnosed allergic rhinitis in infancy was independently associated with a doubling of the risk of developing asthma by 11 years of age.[6] In adults, allergic rhinitis as a risk factor for asthma was shown in a 23-year follow-up of college students.[7]

A Swedish study [8] and an American study,[9] showed that the onset of asthma was associated with allergic rhinitis, and in the US study, after stratification, rhinitis increased the risk of development of asthma by about three times both among atopic and Non atopic patients and by more than five times among patients in the highest IgE titre.

Although the nasal and bronchial mucosa are exposed to the same allergen epithelial shedding and inflammation is more in bronchi than in nasal mucosa.[10] In patients with moderate to severe asthma, eosinophilic

inflammation is more pronounced in the bronchi than in the nose, while in patients with mild asthma inflammation appears to be similar in both sites. Moreover, eosinophilic inflammation of the nose exists in asthmatics with or without nasal symptoms.[11]

In a study comparing mild-moderate asthmatics with corticodependent asthmatics, the proportion of patients with symptoms of rhino sinusitis was similar in both groups (74 percent in corticosteroid dependent asthma and 70 percent in mild-moderate asthma).[12]

Calhoun WJ et al done endobronchial allergen challenge in patients with seasonal rhinitis never presented with asthma before. These patients developed a bronchoconstriction, and lavage carried out serially after challenge demonstrated the occurrence of pro inflammatory mediators and cytokines, as well as the recruitment pf inflammatory cells. [13]

Robinson DS et al showed that the allergic creates a particular local 'microenvironment', promotes the differentiated and maturation of eosinophil progenitors that populate the nasal or the bronchial mucosa.[14]

Johnson SL et al showed that a large number of asthma exacerbations are due to nasal viral infections both in children and in adults. Rhinoviruses are the major cause of the common cold and trigger of acute asthma exacerbations.[15]

CONCLUSION

There are so many studies which showed direct correlation between allergic rhinitis and asthma and allergic rhinitis with nasal polyposis and nasal polyposis with asthma but asthma there are only very few studies which shows a correlation between all these three entities.

As already shown allergic rhinitis mostly co-exist with either of the two but at times, all these three may co-exist.

Our limitations are as it is developing country the allergic disorder are less common than the western countries and we didn't do any test to confirm the atopy and CT of PNS is done only in some cases.

REFERENCES

1. Linneberg A, Nielsen NH, Madsen F, Frolund L, Dirksen A, Jorgensen T, Secular trends of allergic asthma in Danish adults. The Copenhagen Allergy study. Respiratory Medicine, 2001; 95: 258-64.
2. Celedon JC, Palmer U, Weiss ST, Wang B, Fang Z, Xu X. Asthma, rhinitis and skin test reactivity to aeroallergens in families of asthmatic subjects in Anqing, China. American Journal of Respiratory and critical care medicine, 2001; 163: 1108-12.
3. Peat JK, Salome CM, Woodcock AJ. Longitudinal changes in atopy during a 4-year period: relation to bronchial hyperresponsiveness and respiratory symptoms in a population sample of Australian schoolchildren. Journal of allergy an d Clinical Immunology, 1990; 85: 65-74.
4. Von Mutius E, Weiland SK, Fritzsch C, Duhme H, Keil U. Increasing prevalence of hay fever and atopy among children in Leipzig, East Germany. Lancet, 1998; 351: 862-6.
5. Scotomayor H, Badier M, Vervolet O, Orehek J. Seasonal increase of carbachol airway responsiveness in patients allergic to grass pollen. Reversal by corticosteroids. American review of Respiratory disease, 1984; 130: 56-8.
6. Wright AL, Holberg CJ, Martinez FD, Halonen M, Morgan W, Traussig LM. Epidemiology of physician-diagnosed allergic rhinitis in childhood. Pediatrics, 1994; 947: 895-901.
7. Settipane RJ, Hagy GW, Settipane GA. Long term risk factors for developing asthma and allergic rhinitis in childhood. Pediatrics, 1994; 15: 21-5.
8. Plaschke PP, Janson C, Norrman E, Bjornsson E, Ellbjar S, Jarvholm B. Onset and remission of allergic rhinitis and asthma and the relationship with atopic sensitization and smoking. American journal of Respiratory and Critical Care Medicine, 2000; 162: 920-4
9. Guerra S, Sherrill DL, Martinez FD, Barbee RA. Rhinitis as an independent risk factor for adult onset asthma. Journal of Allergy and Clinical Immunology, 2002; 109: 419-25.
10. Channez P, Vignola AM, Vic P, Guddo F, Bonsignore G, R ey al. Mechanism of grass pole induced asthma. Godard P et al. Comparison between nasal and bronchial inflammation in asthmatic and control subjects. American journal of respiratory and critical care medicine, 1999; 159: 588-95.
11. Gaga M, Lambrou P, Papageorgeiou N, Kouloris NG, demonstrated for the first time that allergen trigger of the upper and lower airway pathology in non-atopic asthma, site of the airways. Irrespective of the presence of rhinitis. Clinical and Experimental Allergy, 2000; 30: 663-9.
12. Bresciani M, Paradis L, Des Roaches A, Vernhelt H, Vachier I, Godard P et al. Rhinosinusitis in severe asthma. American journal of respiratory cell allergy and clinical immunology, 2001; 107: 73-80.
13. Calhoun WJ, Jarjour NN, Gleich GJ, Stevens CA, Busse WW, Increased airway inflammation with segmental Campbell MJ, Joshephs LK et al. American review of upper respiratory infections and hospital admissions for respiratory diseases, 1993; 147: 1465-71.
14. Robinson DS, Damia R, Zeibecoglou K, Molet S, North J, Yamada T, et al. CD34(+)/interleukin-5R alpha messenger RNA+cells in bronchial mucosa in asthma: potential airway eosinophil progenitors.

American journal of 2000; 106: 904-10. Respiratory cell and Molecular Biology, 1999; 20: 9-13.

15. Johnson SL, Pattemore PK, Sanderson G, Smith S, Campbell MJ, Josephs LK et al. the relationship between upper respiratory infection and hospital admission for asthma: a time –trend analysis. American journal of Respiratory and Critical Care Medicine, 1996; 154: 654-60.