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ORIGINAL ARTICLE

An analytic study of biomarkers in allergic diseases and asthma

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ABSTRACT:
Background: Asthma is a heterogeneous disease diagnosed by the presence of intermittent symptoms of wheeze, cough and chest tightness, typically related to a reversible airflow obstruction, usually resolves spontaneously or with asthma treatment. The present study was conducted to assess biomarkers in asthma and allergic diseases. **Materials & Methods:** 56 patients of asthma and allergic diseases was included. Submucosal or transbronchial bronchial biopsies were obtained from all patients. Assessment of Eosinophil, Eosinophil cationic protein (ECP) and Lipoxins A4 was performed. **Results:** out of 56 patients, males were 36 and females were 20. The mean eosinophil level in group I was 300.6 cells/µL and in group II was 4.5 cells/µL, ECP level was 24.6 in group I µg/L and 114.5 µg/L in group II and lipoxins A4 level was 1.8 ng/mL in group I and 0.4 in group II ng/mL. The difference was significant (P< 0.05). **Conclusion:** The level of biomarkers was increased in patients with asthma and allergic diseases as compared to healthy subjects.
Key words: Asthma, biomarkers, ECP.

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INTRODUCTION

Asthma is a heterogeneous disease diagnosed by the presence of intermittent symptoms of wheeze, cough and chest tightness, typically related to a reversible airflow obstruction, usually resolves spontaneously or with asthma treatment.[1] Over the years, clinicians have defined several phenotypes based on the presentation and age of onset of symptoms, the severity of the disease, and the presence of other conditions such as allergy and eosinophilia with different long-terms outcomes and response to therapy with corticosteroids.[2] Despite the recognition of these phenotypes of asthma, the approach to the management of asthma recommended by the international Global Initiative for Asthma (GINA) guidelines continues to be based on the severity of the condition, with drugs added on the basis of asthma control.[3]

Atopy is the key factor predisposing for the development of allergic airways disease. Despite modern technologies enabling to unravel several inflammatory mechanisms of allergic airway disease, presently, still many etiological and pathophysiological questions remain unanswered.[4] Overall, the allergic inflammation within the bronchial and nasal tissues shows many similarities with some local differences. Exposure to a new allergen results in uptake and processing by dendritic cells (DCs).[5] Subsequent presentation of the processed allergen by DCs to naïve T helper (Th) cells induces the development of Th2 cells in genetically predisposed individuals. The Th2 cells then release interleukins (IL)-4 and IL-13, causing the differentiation of B cells into allergen-specific immunoglobulin (Ig)-E-producing plasma cells.[6] The present study was conducted to assess biomarkers in asthma and allergic diseases.

MATERIALS & METHODS

The present study comprised of 56 patients of asthma and allergic diseases of both genders. Equal number of healthy subjects were also recruited. All patients were informed regarding the study and their consent was obtained.

Demographic profile such as name, age, gender etc. was recorded. Submucosal or transbronchial bronchial biopsies were obtained from all patients. Assessment of Eosinophil, Eosinophil cationic protein (ECP) and Lipoxins A4 was performed. Results thus obtained were tabulated and presented for statistical interferences, where p value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 56		
Gender	**Males**	**Females**
Number	36	20

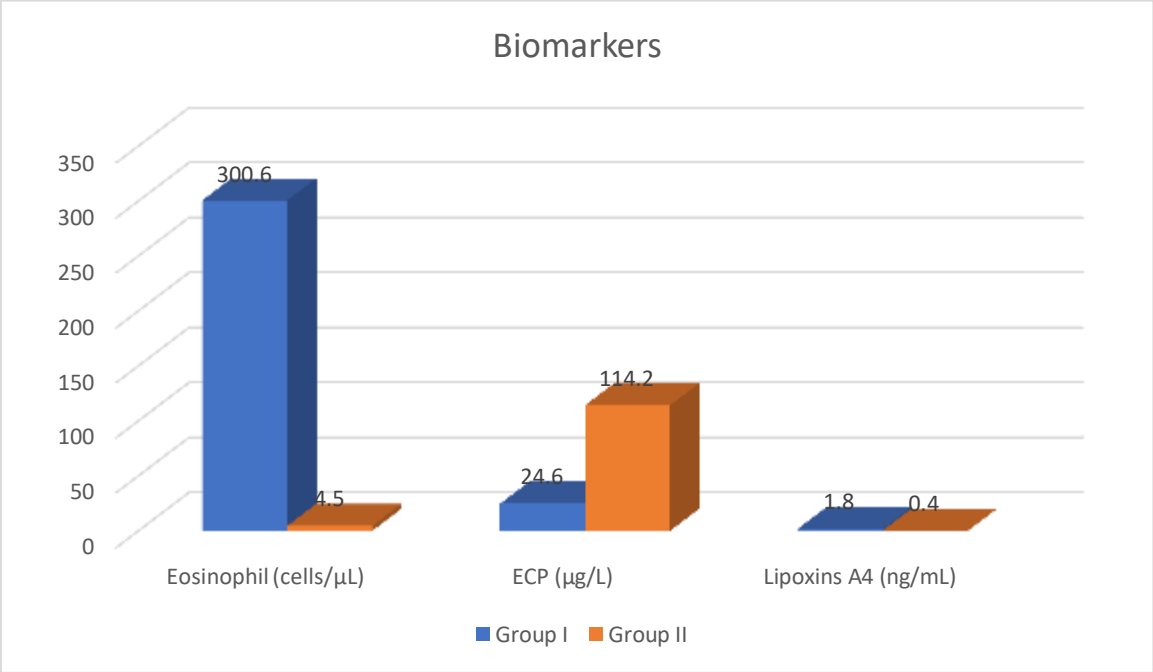
Table I shows that out of 56 patients, males were 36 and females were 20.

Table II Assessment of biomarkers

Biomarkers	Group I	Group II	P value
Eosinophil (cells/µL)	300.6	4.5	0.01
ECP (µg/L)	24.6	114.2	0.02
Lipoxins A4 (ng/mL)	1.8	0.4	0.05

Table II, graph I shows that mean eosinophil level in group I was 300.6 cells/µL and in group II was 4.5 cells/µL, ECP level was 24.6 in group I µg/L and 114.5 µg/L in group II and lipoxins A4 level was 1.8 ng/mL in group I and 0.4 in group II ng/mL. The difference was significant (P< 0.05).

Graph I Assessment of biomarkers



DISCUSSION

In the era of the personalized medicine, in order to deliver this approach for asthma, it is important to be able to phenotype the condition in an unbiased way and to define biomarkers able to predict the course of the disease and the response to therapy.[7] A biomarker is a measurable indicator that can evaluate a normal or pathological biological processes or pharmacologic response to a therapeutic intervention.[8] A valid biomarker would have several key characteristics: to distinguish between disease and health with high positive and negative predictive values, to provide information about disease prognosis and clinical outcomes, to change with disease progression and "normalize" with successful treatment, to be reliable and reproducible in the clinical setting with little or no day-to-day variation, to be easy to collect in the "real-world" setting, to be quantifiable in an analytical system with well-defined performance, and to be cost-effective.[9] The present study was conducted to assess biomarkers in asthma and allergic diseases.

In present study, we found that out of 56 patients, males were 36 and females were 20. Celik et al[10] detect LX A4 and 15-epi-LX A4 levels in asthma patients with and without AERD of comparable severity. The study groups consisted of 22 subjects with AERD, 22 subjects with ATA and 10 volunteers without asthma and aspirin sensitivity. Whole-blood samples were stimulated with calcium ionophore, A23187 and A23187, 1aspirin (10 4 M). LX A4 and 15-epi-LX A4 levels were analysed by the enzyme immune assay method. Results Severe asthma patients in both AERD [0.5 (0.8)] ng/mL and ATA [0.5 (0.45) ng/mL] groups showed diminished generation for LX

A4 to stimulation with A23187 in comparison with other severity degrees in their groups (P = 0.02 and 0.046, respectively). LX A4 generation in both severe groups was comparable with each other (P< 0.05). Although severe cases with AERD showed a diminished capacity to generate 15-epi-LX A4, this did not reach statistical significance. Conclusion This study indicated that diminished LX A4 generation was unique to severe asthma phenotype regardless of comorbid aspirin sensitivity.

We found that mean eosinophil level in group I was 300.6 cells/µL and in group II was 4.5 cells/µL, ECP level was 24.6 in group I µg/L and 114.5 µg/L in group II and lipoxins A4 level was 1.8 ng/mL in group I and 0.4 in group II ng/mL. According to the presence of assessable biomarkers of T2 mediated airway inflammation, the cluster-analysis identified several asthma phenotypes.[11]The T2-high phenotype includes the classical allergic one (mild blood eosinophilia, high levels of FeNO, high level of serum total IgE) and the late-onset, nonallergic but highly eosinophilic one, frequently associated to chronic rhinosinusitis with nasal polyps (high FeNO but serum total IgE normal or elevated but probably with a lower etiopathogenetical importance). The eosinophilic phenotype is associated with an intense production of IL-5 and IL-13. The T2-low phenotypes are more diversified and less well defined, with predominant neutrophilic airway inflammation, higher frequency of recurrent airway infections, higher prevalence of obesity and cigarette smoking. The mechanisms implicated in these phenotypes are the TNFα and IL-17 inflammatory pathways.[12]

CONCLUSION

Authors found that level of biomarkers were increased in patients with asthma and allergic diseases as compared to healthy subjects.

REFERENCES

1. Ray A, Raundhal M, Oriss TB, Ray P, Wenzel SE. Current concepts of severe asthma. J Clin Invest. 2016;126:2394–403.
2. Chung KF. Personalised medicine in asthma: time for action. Eur Respir Rev. 2017. https://doi.org/10.1183/16000617.0064-2017.
3. Eguiluz-Gracia I, Tay TR, Hew M, Escribese MM, Barber D, O'Hehir RE, et al. Recent developments and highlights in biomarkers in allergic diseases and asthma. Allergy. 2018; doi: 10.1111.
4. Fitzpatrick AM. Biomarkers of asthma and allergic airway diseases. Ann Allergy Asthma Immunol. 2015;115:335–40.
5. Kelly RS, Dahlin A, McGeachie MJ, Qiu W, Sordillo J, Wan ES, et al. Asthma metabolomics and the potential for integrative omics in research and the clinic. Chest. 2017;151:262–77.
6. Villaseñor A, Rosace D, Obeso D, Pérez-Gordo M, Chivato T, Barbas C, et al. Allergic asthma: an overview of metabolomic strategies leading to the identification of biomarkers in the field. Clin Exp Allergy. 2017;47:442–56.
7. Price DB, Rigazio A, Campbell JD, Bleecker ER, Corrigan CJ, Thomas M, et al. Blood eosinophil count and prospective annual asthma disease burden: a UK cohort study. Lancet Respir Med. 2015;3:849–58.
8. Hancox RJ, Pavord ID, Sears MR. Associations between blood eosinophils and decline in lung function among adults with and without asthma. Eur Respir J. 2018. https://doi.org/10.1183/13993003.02536-2017.
9. Konradsen JR, Skantz E, Nordlund B, Lidegran M, James A, Ono J, et al. Predicting asthma morbidity in children using proposed markers of Th2- type inflammation. Pediatr Allergy Immunol. 2015;26:772–9.
10. Celik GE, Erkekol FO, Mısırlıgil Z, Melli M. Lipoxin A4 levels in asthma: relation with disease severity and aspirin sensitivity. Clinical & Experimental Allergy. 2007 Oct;37(10):1494-501.
11. Kato M, Yamada Y, Maruyama K, Hayashi Y. Serum eosinophil cationic protein and 27 cytokines/chemokines in acute exacerbation of childhood asthma. Int Arch Allergy Immunol. 2010;152(Suppl 1):62–6.
12. Spahn JD, Covar RA, Jain N, Gleason M, Shimamoto R, Szefler SJ, et al. Effect of montelukast on peripheral airflow obstruction in children with asthma. Ann Allergy Asthma Immunol. 2006;96:541–9.