



ANALYSIS OF INFLAMMATORY BIOMARKER EXPRESSION IN ADULTS WITH SEVERE ASTHMA

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Objective:

To analyze biomarker expression in adult patients diagnosed with severe asthma.



Methods:

The data was collected on adult patients (>18 years old) diagnosed with severe asthma, on their first visit to the Department of Allergic and Obstructive Pulmonary Diseases, Clinic for Lung Diseases Jordanovac, Clinical Hospital Center Zagreb. All the participants were also enrolled in Severe Heterogenous Asthma Research collaboration registry (SHARP). The thresholds for inflammatory biomarker positivity were prespecified (total serum IgE ≥ 75 kU/L, blood eosinophils ≥ 300 cells/mcL, FeNO ≥ 25 ppb). Eosinophilic phenotype was defined as a blood eosinophils cutoff point of ≥ 300 cells/mcL. Allergic phenotype was defined as having positive allergen specific IgE for at least one common aeroallergen or positive skin prick test.

Result:

Of 135 patients in our registry, 82 had data on all of the aforementioned biomarkers and were included in further analysis.

69.51% were positive for either 2 or more biomarkers, suggesting the significant inflammatory biomarker overlap.



Among patients with:

- positive total IgE (83.67%), 66.13% were also eosinophil positive and 70.97% were FeNO positive,
- positive blood eosinophils (54.88%), 91.11% were also total IgE positive and 73.33% FeNO positive,
- positive FeNO (65.85%), 81.48% were also total IgE positive and 61.11% eosinophil positive.

40.24% patients were both FeNO and eosinophil positive, while 53.66% were both total serum IgE and FeNO positive. Furthermore, 34.15% were specific IgE and/or skin prick test positive in addition to being both FeNO and total IgE positive.

52.44% were characterized as allergic phenotype and 54.88% as eosinophilic. However, a large proportion of the latter group was atopic as well - only 13.81% were "strictly" eosinophilic (total serum IgE of ≤ 188 kU/L in addition to positive blood eosinophils).

Conclusion:



Today, inflammatory biomarkers play a key role in severe asthma management. We use them to distinguish between the two predominant phenotypes, the T2 high and the T2 low phenotype, as well as to determine if T2 high phenotype is allergic or eosinophilic. All of the above affects the final decision on the best therapeutic option for each patient. However, our results are showing a notable inflammatory biomarker overlap, implying the need for a more comprehensive approach, rather than relying on a simple biomarker threshold positivity.