ASSOCIATION BETWEEN PSORIASIS AND ASTHMA – BOTH DISEASES WITH TH2 RESPONSE

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ABSTRACT

Introduction

Both psoriasis and asthma are chronic immune-mediated inflammatory diseases. Many studies have shown the increased risk of developing asthma in patients with psoriasis compared with controls. Myeloid dendritic cells induce Th2 responses to inhaled antigen, leading to eosinophilic airway inflammation. No study has previously explored how psoriatic inflammation affects airway inflammation. Some data suggest that systemic IL-23/STAT3 axis is responsible for enhanced airway inflammation during psoriasis. T2 interleukins IL-4, IL-5 and IL-13 are pivotal in asthmatic patients, driving the isotype switch to IgE production, eosinophilia, mast cell activation and airways
remodeling. Similar mechanisms to those in patients with psoriasis might be implicated in the pathobiology of asthmatic patients with the IL-17-high phenotype in which bronchial epithelial dysfunction and inflammatory mechanisms would be major drivers defining the clinical outcome.

Case report

We present a case of 68-years old man with a history of frequent bronchitis and allergy rhinitis in childhood. Later on he suffered from psoriasis. He is an ex-smoker (60p/y). Allergy to mites and home dust had been diagnosed and hyposensitization done. In year 2003 lobectomy was done and samples were sent to mycological, microbiological and pathohistological analysis revealed tuberculoma. Antituberculosis therapy was conducted for 6 months. At the age of 63 KOPD was diagnosed and bronchodilatator therapy was prescripted. Over next years symptoms persisted despite regular therapy- dyspnea on moderate exertion, nonproductive cough, mild to significant pulmonary function (FEV1 54%-87%) and significant eosinophilia ( 8% or 740/ periferal eosinophils). Diagnosis was changed into asthma and he became therapy with ICS/LABA/LAMA and anti-leukotrienes. Within 6 months patient showed decreased clinical symptoms and better control of disease with positive effect on quality of life.

Conclusion

Inhaled glucocorticoids suppress airway inflammation by activating anti-inflammatory genes, switching off inflammatory gene expression and inhibiting inflammatory cells. Data from clinical trials suggest that blood levels of eosinophils between 200 and 300/microL or fraction of exhaled nitric oxide (FENO) levels above 24 ppb support an underlying active type 2 immune process, which will
respond to type 2 specific therapy. We suggest that only LABA/LAMA/ICS and anti-leukotriens are often sufficient to alleviate airway inflammatory burden in asthmatics with psoriasis. It is a potential treatment for psoriasis because asthma and psoriasis appear as the diseases with the greatest overlap and significance. The success of this treatment should be followed by spirometry, fraction of exhaled nitric oxide (FENO) measurement, sputum induction and allergen skin testing.