Case Report

Patient on allergen immunotherapy developed systemic lupus erythematosus?– A clinico-pharmacological look out

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ABSTRACT

Drug induced lupus is an autoimmune condition secondary to drug exposure which leads to development of systemic lupus erythematosus (SLE). However, labelling the culprit drug needs a prudent insight into the pharmacological plausibility of each of the offending drugs in suspicion. Here we present a report where allergen immunotherapy was suspected to cause SLE and a deeper clinico-pharmacological evaluation cleared the air.

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1. Introduction

Allergen Immunotherapy (AIT) has seen widespread advancements in the arena of atopic diseases over the past decades. However, its use has been linked and has regarded contraindicated in patients with rheumatologic complications. Evidence to support the idea that AIT can trigger autoimmune disease is considerably weak and is mainly based on few case reports highlighting causation of vasculitis.¹ However, no report of patients developing autoimmune diseases has been reported from randomised controlled trials probing the effect of AIT. Even, there exist pharmaco-epidemiological studies which have failed to detect any increased risk of autoimmune disease development during AIT compared with other conventional allergy procedures.

Nevertheless, due to lack of available data, researchers propose there is relative contraindication in autoimmune disorders in remission and absolute contraindications in active forms with AIT. As a principle of caution, it is prudent to undergo a detailed risk-benefit assessment in auto-immune disease candidates in need of AIT. A detailed clinical examination of the patient status along with careful consideration of all related factors may show up the flip side of the story. Here we present a similar instance of clinical dilemma in a 24-year-old male.

2. Case Report

MK, 24 year old male, patient initially presented with moderate to severe persistent allergic rhinitis for 1 year and asthma for 2 years on step 3 management strategy. His initial laboratory work-up demonstrated eosinophilia (14%) in peripheral blood smear with total leucocyte count (7800/µL), elevated IgE total (2045 IU/ml), high FeNO (78 ppb). Skin prick test report suggested hypersensitivity with dust mites, pollens like Cocos nucifera, Azadirachta indica and Cynodon dactylon. He had also skin prick test positivity with foods like milk, wheat, cauliflower, prawn,
All anti-TNF agents have been associated with DIL, with hydralazine are associated with the highest incidence of DIL, acting on B cells. Treg cells increase IgG4 and IgA and decrease IgE. There would be decrease in release of proinflammatory cytokine from mast cells, eosinophils. Decrease IgE. There would be decrease in release of cytokines (interferon gamma, IL-2) without any effect on CD4 + cell proliferation and changes of cytokines produced by T-cell at local allergen challenge sites by allergen immunotherapy. Following allergen immunotherapy there would be generation of autoantibodies. By virtue of AIT there would be high Th1 response and decrease in Th2 response and pharmacological plausibility is not explaining this reaction.

Allergen immunotherapy should be used with caution when benefits outweigh potential risks in an individual patient in case of patient with systemic autoimmune disease. Impaired balance of T-helper-cell (TH) subsets (TH1/TH2/TH17) and regulatory T-cells (Tregs) is contributing to the pathophysiology of Systemic lupus erythematosus (SLE). One study had demonstrated reduction in IFN-γ (marker of TH1 activity) and TGF-β1 (marker of Treg activity) with the elevation in IL-6 and IL-17 (marker of TH17). Derangement of TH17/Treg balance in blood would be responsible for development of SLE. This causes an increased pro-inflammatory response especially in the active form of the disease.

| Allergen | Immunotherapy causes reduction in the production of histamine releasing factors from mononuclear cells and had decreased immediate and late-phase allergen challenged nasal reactions. Eosinophil counts in nasal secretions after nasal reactions. Eosinophil counts in nasal secretions after nasal challenge was decreased in allergen immunotherapy treated groups. There is decrement in allergen-specific CD4 + cell proliferation and changes of cytokines produced by T-cell at local allergen challenge sites by allergen immunotherapy. Following allergen immunotherapy there would be upregulation of T-helper (Th) 1 CD4 + cell-type cytokines (interferon gamma, IL-2) without any effect on CD4 + cell Th2 cytokine (IL-4, IL-5) expression. Regulatory T (Treg) cells and related cytokines like IL-10 and transforming growth factor beta suppress Th2-type immune responses. This would control allergic diseases. Acting on B cells Treg cells increase IgG4 and IgA and decrease IgE. There would be decrease in release of proinflammatory cytokine from mast cells, eosinophils. Allergen induced lymphoproliferative responses have been decreased after immunotherapy. Allergen specific immune response to generation of autoantibodies. By virtue of AIT there would be high Th1 response and decrease in Th2 response and pharmacological plausibility is not explaining this reaction. |
deviation from a Th2 to a Th1 is the key mechanism by which allergen immunotherapy works. There are no studies which suggest increase in Th17 responses by allergen immunotherapy. It is not right to state allergen immunotherapy has causal relationship with development of SLE.

4. Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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References


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