Steroid Interference

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Cellular immune responses are regulated by white blood cells called T cells (or T lymphocytes), circulating in the blood. Developing in the thymus, T cells are vital to effective immune responses. They directly attack intracellular viral pathogens, and cells that have transformed into tumor cells. Many T cell subtypes have been described, each with distinct functions[1]. There are two T helper cell subsets: T_h1 cells direct immune responses against pathogens and foreign proteins. T_h2 cells regulate allergic responses.

Humoral immune responses are driven by antibodies (immunoglobulins), which are specialized proteins circulating in the blood ("humor" is a medieval term for body fluid). Antibodies are produced by B cells after activation from antigens presented by T cells. Originating from bone marrow, each B cell recognizes a unique protein sequence, differentiates into a plasma cell and produces antibodies specifically directed against a single antigen determinant.

In allergic responses, T cells present allergenic proteins to B cells, directing them to make allergen-specific IgE antibodies. IgE has a unique function among immunoglobulins: it is the only antibody class that binds to mast cells. This property is central to IgE-induced allergic inflammation. Allergy symptoms arise when IgE-allergen complexes bind to mast cell surfaces, inducing degranulation and causing the release of histamine and other inflammatory proteins into the circulation. IgE-driven responses are immediate, acute phase inflammatory reactions, often occurring within minutes after allergen exposure[2].

Glucocorticosteroids are given to an allergic patient because they act in the body to reduce inflammation, in several ways:

1. Depletion of tissue-based mast cells, the predominant cell type causing allergic inflammation[3]. Mast cells are a major target of steroids.
2. Suppression of function in other inflammatory cell types: eosinophils, basophils, neutrophils and macrophages[4, 5].

Steroids have long been thought to cause generalized immunosuppression of cellular responses, while not directly affecting B cells and humoral immunity[6]. Recent evidence suggests that glucocorticoids act more selectively, causing suppression of the T_h1 axis of cellular immune responses, while moving toward enhancement of T_h2-directed (allergic) responses[7, 8]. Supporting these conclusions are studies showing that glucocorticoid treatment in allergic patients improves symptoms, while having either no effect on serum IgE levels, or causing them to rise[9-11].

Current experimental and clinical evidence indicates that steroids do not directly affect or suppress IgE levels. It is thus not necessary to withdraw your patient from steroid treatment, prior to submitting a serum sample for ACTT allergy testing.

References

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