Clinical Immunology in Dermatology

Clinical Immunology is a widely diverse subject, relating the achievements of basic immunological research to clinical practice. It is generally accepted that Clinical Immunology should include immunodeficiency, autoimmune, allergic and lymphoproliferative diseases. These diseases cover a wide range of organ-based medical specialties, but as Clinical Immunology itself is not organ-based, it is somehow difficult to define a Clinical Immunology specialty. This problem is recognised world-wide. Even in those countries where Clinical Immunology exists, different people have different views on what it is.

On the laboratory side, the picture is clearer. Clinical Laboratory Immunology is a well-defined discipline in Pathology covering tests related to immunochemistry (for example, IgG, IgA, IgM, C3, C4, paraproteins), autoantibodies (for example, ANA, anti-dsDNA, anti-mitochondria, anti-smooth muscle) and immune cell functions (for example, lymphocyte functions, neutrophil functions). These tests support the diagnosis and management of a wide variety of diseases. Dermatological diseases, including dermatological manifestations of other systemic diseases, are among the most frequent disease groups which utilise immunological tests.

A growing number of dermatological diseases are autoimmune in nature and are characterised by blistering. The diagnosis and management of some of these are aided by testing relevant autoantibodies, such as antibodies against the dermo-epidermal junction or the epidermal intercellular substance. The titre of the latter is shown to correlate with disease activity while the former is not.

Recently, new lights on the pathophysiological mechanisms of the so-called chronic 'idiopathic' urticaria have concluded it might also be autoimmune in nature (for review - please see *J Allergy Clin Immunol* 105(4):664-672, 2000). Approximately a quarter to one half of these patients have functional antibodies against the high-affinity IgE receptor (FceRI). These autoantibodies are IgG in nature and bind the alpha chain of the FceRI. A further 5% have antibodies against the Fc part of the IgE molecules. Both autoantibodies (anti-FceRI and anti-IgE) functionally cross-linked the FceRI on mast cells resulting in degranulation. Patients with these autoantibodies can be diagnosed by autologous serum/plasma skin test. Patient serum (or plasma) is injected intradermally into uninvolved skin. A positive result will appear as a wheal 30 minutes later. This test is not diagnostic and a positive result should be confirmed by *in vitro* testing, either histamine release assay or immunoblotting. Unfortunately, due to the lack of appropriate facilities for histamine measurement and the unavailability of the blotting antigen, neither of these tests is available locally at present. These findings have important implications to treatment of severe cases recalcitrant to conventional measures. Cyclosporin A was proved to be of value in autoantibody-positive patients and is probably also effective in most autoantibody-negative patients. In the Queen Mary Hospital Allergy Clinic, there has not yet been a patient whose disease is severe enough for this expensive drug. With our limited number of patients, so far we can manage with H_1 antihistamines and doxepin, occasionally supplemented by H_2 antihistamine, hydroxychloroquine and short course of systemic steroid.

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