Specific Hyposensitization in Atopic Dermatitis

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Published data on the efficacy of hyposensitization therapy in atopic dermatitis (AD) are inconsistent, above all with regard to selection criteria for the subjects to be treated. We report the clinical results of tests in specific hyposensitization therapy which were conducted in AD patients.

The patients demonstrated specific IgE against inhaled allergens, a clinical history of exacerbation of the dermatitis following contact with aggravating allergens, and they did not have clinical signs of food allergies. Our material consisted of 24 men and 39 women of ages varying from 4 to 45 years. Fifty of the 63 patients had associated asthma and/or hay fever. At the commencement of therapy, 10 were seriously affected by AD, 40 moderately and 13 mildly. We used delayed aluminium hydroxide-adsorbed vaccines. Initial administration was weekly, and was later reduced to fortnightly. Doses were lower and the build-up to maximum dose was more gradual than in normal treatment for asthma and hay fever, these measures being designed to minimize exacerbation of the dermatitis deriving from the treatment itself. 20 subjects were treated with vaccine for dermato-phagoides, 26 for grass pollen, 7 for parietaria or mug-wort, whilst 10 were submitted to two types of treatment (dermatophagoides and pollen). 17 patients underwent therapy for more than 24 months, 16 for more than 12 months, 30 for 6–12 months. In certain phases of the illness, topical emollients and antiseptics and systemic an-ti-histamines were applied. We evaluated the results by comparing the points scored at check-ups conducted every 3 months on a scoring system which took into account the following: the extent of the dermatitis, the frequency of relapses, the intensity of itching, and clinical characteristics of the dermatitis.

None of the patients suffered noteworthy collateral effects from the treatment, except in 4 cases where, in the initial months of treatment, slight exacerbation of the dermatitis was evident during the 24 h following administration of the vaccine. Those subjects who recorded an improvement did so in the first 4–5 months of

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therapy. At the last check up, no patient demonstrated a worsened condition, 22 patients showed no or slight improvement, 35 had improved significantly and 6 were totally free of skin lesions. Age distribution of those patients who responded satisfactorily to therapy was relatively uniform, although there was a slight prevalence in the 4–15 age group, which may probably be explained

by the natural evolution of AD towards a spontaneous improvement. The high percentage of positive results is, in our opinion, attributable to the careful selection of our subjects, and in particular to the inclusion of subjects with a clinical history of reaction to inhaled allergens. This preliminary open clinical study requires further confirmation. This would include a comparison with results deriving from a control group, along with an evaluation of total and specific IgE and of IgG subclasses, which would provide biological support for our findings.

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Letters to the Editor

Pemphigus vulgaris and Ulcerative Colitis

The association of pemphigus with other autoimmune diseases is infrequent. Associations of pemphigus with myasthenia gravis, SLE, pernicious anemia, and thymoma have been reported [2, 3, 5]. We report a case of pemphigus vulgaris associated with ulcerative colitis; this association, to our knowledge, has never been reported in the literature.

Two years ago the patient, a 40-year-old farmer, showed some painful erosions of oral mucosa with burning and pharyngodynia. Some weeks later he developed many flaccid bullous lesions on the whole body surface, with large areas of denuded skin after their rupture.

A cytologic and histologic examination confirmed the diagnosis of pemphigus vulgaris and the patient was treated with corticosteroid (prednisone 120 mg/day) and immunosuppressive (methotrexate 25 mg/week) drugs with regression of mucocutaneous lesions.

Eight months later, while the patient was being treated with prednisone 15 mg/day with stationary clinical healing of pemphigus, he presented with a bloody and painful diarrhoea (4–6 evacuations/day). The diagnosis of ulcerative colitis was confirmed by rectoscopy and histopathological examination of multiple intestinal biopsies. The symptomatology regressed after pure sulfa-pyridine therapy (8 g/day for 2 weeks).

Laboratory findings demonstrated: ESR 42, gamma-globulins 25%, IgG 1,924 g/l, IgA 425 g/l, serum iron 27 gamma%ml. The remaining laboratory findings and the X-ray of chest, stomach and duodenum yielded normal results. An eye examination showed conjunctivitis.

Histopathologic findings from skin biopsy revealed edema between and above the basal cells of the epidermis with the formation of a cleft above the basal cells; the cavity of the bulla contained degenerate acantholytic cells.

The etiopathogenetic role of an autoimmune mechanism in pemphigus is certain. In fact, it is known that antibodies to intercellular antigens fix to the cell surface of keratinocytes, complement is not involved primarily and proteolytic enzymes are activated or liberated at the

cell surface or from within the cells [4, 8]. An autoimmune etiology for ulcerative colitis is also invoked [1,6,7].

The frequent development of an autoimmune disease in a subject with another autoimmune disease can be explained either by the same common immune process or by a predisposition to autoimmune disease.

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Fixed Drug Eruption to Trimethoprim

A sharply demarcated erythema, oval or circular in shape, is the characteristic lesion of the fixed eruption. Numerous drugs have been implicated in its cause, the commonest being barbiturates, phenolphthalein, pyra-zolon derivatives, sulphonamides and tetracyclines [1]. We recently had the opportunity of seeing a patient who had fixed drug eruption due to trimethoprim. To our