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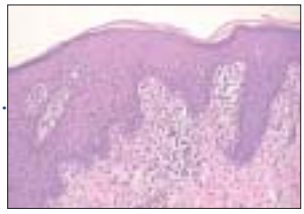

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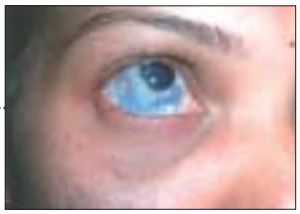

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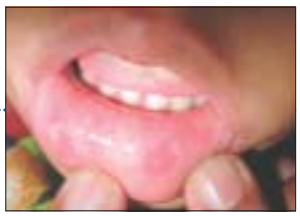

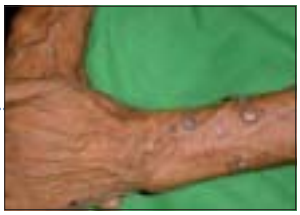

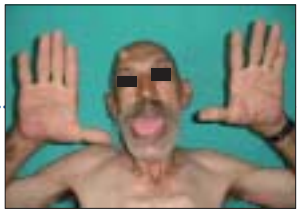

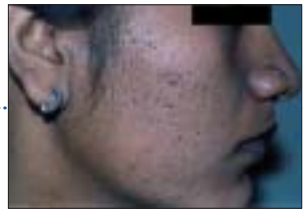

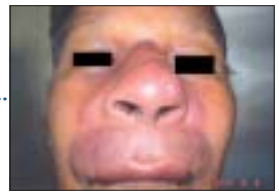

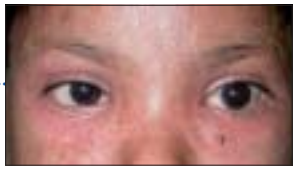

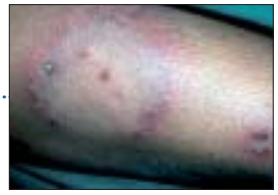

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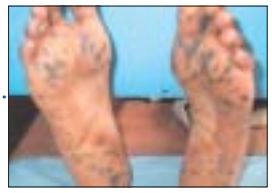

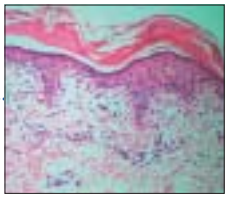

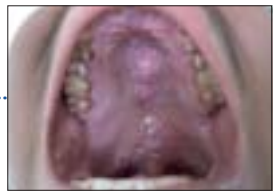

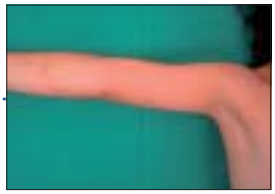

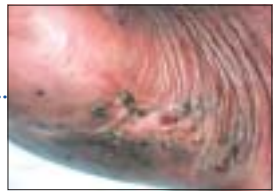

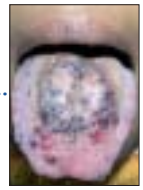

Omalizumab in severe chronic urticaria

Sir,

Urticaria patients are usually treated with oral antihistamines and 50% of them respond well to this treatment. However, the other 50% do not respond to antihistamines and need a more aggressive approach. Approximately 40-50% of patients with no apparent cause for their urticaria are believed to have an associated autoimmune profile that may play a pathogenetic role. We describe here a patient who responded to omalizumab after failure to respond to cyclosporine.

A forty-five-year old female presented with severe chronic urticaria prevalent for the last ten years and which did not respond to antihistamines and steroids. About five years ago, the patient was diagnosed to have sarcoidosis and was treated with oral steroids. She had developed osteoporosis

due to repeated courses of oral steroids in the past. She also had a history of bronchial asthma, which was controlled with a bronchodilator. She was started on cyclosporine at a dose of 3 mg/kg in December 2006. Her urticaria was well controlled with cyclosporine until June 2007. Later, her urticaria worsened in spite of regular doses of cyclosporine and antihistamines in combination (hydroxyzine 25 mg three times a day and fexofenadine 180 mg daily). Hence, the dose of cyclosporine was doubled to 6 mg/Kg per day (300 mg) but her urticaria was not controlled. The addition of montelukast also did not help.

Her blood investigations including complete blood counts, biochemistry and thyroid stimulating hormone (TSH) were within normal limits. Serum protein electrophoresis was normal. Autologous serum skin test could not be performed as antihistamines could be not stopped even for a single day. Serum immunoglobulin E (IgE) was 778 as against the normal level of 100. At this stage, she was started on omalizumab 300 mg every four weeks in consultation with a chest physician in addition to cyclosporine, antihistamines and montelukast. After the first injection, she showed more than 90% control of her urticaria, while after the second injection, she had total relief from her symptoms, which lasted for four weeks.

Omalizumab, a recombinant, humanized, monoclonal antibody against immunoglobulin IgE, represents a unique therapeutic approach for the treatment of allergic diseases. This agent acts as a neutralizing antibody by binding IgE at the same site on IgE as its high-affinity receptor, $F_c\varepsilon R_I$. Subsequently, IgE is prevented from sensitizing cells bearing high-affinity $F_c\varepsilon R_I$ receptors. Inhibition of the biological effects of IgE targets an early phase of the allergic cascade before the generation of allergic symptoms.[1] Omalizumab reduces serum levels of IgE and blocks the attachment of IgE to mast cells and other immune cells, thereby preventing IgE-mediated inflammatory changes. Omalizumab is approved for the treatment of moderate-to-severe persistent asthma in adults and adolescents older than 12 years of age who have a positive skin test to a perennial allergen.[2] Dosing is based on weight and pretreatment serum IgE levels and is administered via subcutaneous injection every 2-4 weeks. The safety profile of omalizumab is favorable with injection site reaction being the most commonly reported adverse event.

There are reports of the efficacy of omalizumab in chronic urticaria[3] and atopic dermatitis.[4] The incidence of anaphylaxis in clinical trials for omalizumab was 0.1%.[5] Boyce describes a successful treatment of cold urticaria with omalizumab.[6] Anti-IgE treatment induces the depletion of free IgE from the serum and tissue, leading ultimately to reduced binding of IgE to its high-affinity surface receptor, $F_c\varepsilon R_I$. As occupancy of $F_c\varepsilon R_I$ by IgE determines the levels of surface $F_c\varepsilon R_I$ expression, this leads to a rapid depletion of both cell-bound IgE and surface $F_c\varepsilon R_I$ expression on blood basophils.

Omalizumab may have a beneficial effect in the treatment of chronic urticaria. Further studies are needed to confirm this effect and better elucidate the mechanism for the observed improvement.

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