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Letters to the Editor

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 not be 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, FcεRI. Subsequently, IgE is prevented from sensitizing cells bearing high-affinity FcεRI receptors. Inhibition of the biological effects of IgE targets an early phase of the allergic cascade before the generation of allergic symptoms. 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. 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 and atopic dermatitis. The incidence of anaphylaxis in clinical trials for omalizumab was 0.1%. Boyce describes a successful treatment of cold urticaria with omalizumab. 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, FcεRI. As occupancy of FcεRI by IgE determines the levels of surface FcεRI expression, this leads to a rapid depletion of both cell-bound IgE and surface FcεRI 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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Hypothesis: The potential utility of topical eflornithine against cutaneous leishmaniasis

Sir,
The trypanothione biosynthetic pathway is common to the trypanosomatid family of protozoa, which includes Leishmania and Trypanosoma, and is absent in the host systems. This pathway constitutes an important target for
chemotherapy against leishmaniasis. The trypanothione pathway combines two metabolic pathways: the glutathione and the polyamine biosynthetic pathways, to produce trypanothione, a glutathione-spermidine conjugate.\[1\]

The levels of trypanothione are increased in the Leishmania parasite selected for resistance to the heavy metal, arsenic. The levels of putrescine and spermidine were increased in resistant mutants. This increase is mediated by overexpression of ornithine decarboxylase, the rate-limiting enzyme in polyamine biosynthesis.\[2\] Fluorinated analogues of L-ornithine are powerful inhibitors of ornithine decarboxylase and inhibit the cell growth of L. infantum promastigotes.\[3\]

Eflornithine was originally used orally in the treatment of childhood hyperactivity.\[4\] It was used as an anti-cancer drug in 1970\[5\] and was later used intravenously in the treatment of African sleeping sickness.\[3,6\] Interestingly, hair loss was observed as an adverse effect of this treatment.\[5,7\]

Eflornithine hydrochloride cream (13.9%) is the first topical preparation approved by the FDA in August 2000 for the reduction of facial hirsutism in women.\[5\] It is a potent inhibitor of ornithine decarboxylase. A topical formulation of this agent has been used for treatment of hirsute women as inhibition of ornithine decarboxylase delays the initiation of anagen and keeps hair in telogen. Therefore, eflornithine does not remove the excess hair but it causes slowing of excessive hair growth.

Given the important role of ornithine decarboxylase in the trypanothione biosynthetic pathway, eflornithine could prove to be effective against leishmaniasis. Combining this agent with glaucantime could potentiate the therapeutic response of the latter and break the resistance of the resistant strains against it. Clinical studies on this subject are warranted.

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REFERENCES


Sir,

Nodular melanoma is the second most common subtype of cutaneous melanomas with a frequency of 15-30%.\[1\] Most frequently it presents in midlife with a median age at presentation of 53 years, it is more common in males than in females. Recognition of nodular melanomas can be problematic as they lack many of the conventional clinical features.

A 47 year-old man was referred to the Dermatology Department with complaints of a nonhealing ulcer over the right thigh and popliteal fossa prevalent since the last two years and also, nodules over the right lower limb extending up to the ankles observed for the last eight months. The patient was apparently asymptomatic two years before when he met with an accident leading to a nonhealing ulcer over the right thigh. There was no history of any burn injury. The ulcer was treated with skin grafting from the left thigh. The graft site healed well at that time. Gradually over the span of a few months, the patient developed small swellings over the scar of the grafted site. This was followed by ulceration and rapid growth of a fungating mass extending up to the popliteal fossa. This was associated with a serosanguineous discharge. Over the next year, the patient started developing skin-colored nodules over the right lower limb below the growth extending up to the dorsum of the foot. The growth was painless and asymptomatic to start with but...