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C O N T E N T S

EDITORIAL

Management of autoimmune urticaria

Arun C. Inamadar, Aparna Palit 89

VIEW POINT

Cosmetic dermatology versus cosmetology: A misnomer in need of urgent correction

Shyam B. Verma, Zoe D. Draelos 92

REVIEW ARTICLE

Psoriasiform dermatoses

Virendra N. Sehgal, Sunil Dogra, Govind Srivastava, Ashok K. Aggarwal 94



ORIGINAL ARTICLES

A study of allergen-specific IgE antibodies in Indian patients of atopic dermatitis

V. K. Somani 100

Chronic idiopathic urticaria: Comparison of clinical features with positive autologous serum skin test

George Mamatha, C. Balachandran, Prabhu Smitha 105



Autologous serum therapy in chronic urticaria: Old wine in a new bottle

A. K. Bajaj, Abir Saraswat, Amitabh Upadhyay, Rajetha Damisetty, Sandipan Dhar 109

Use of patch testing for identifying allergen causing chronic urticaria

Ashimav Deb Sharma 114

Vitiligoid lichen sclerosis: A reappraisal

Venkat Ratnam Attali, Sasi Kiran Attali 118



BRIEF REPORTS

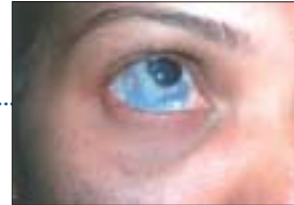
Activated charcoal and baking soda to reduce odor associated with extensive blistering disorders

Arun Chakravarthi, C. R. Srinivas, Anil C. Mathew 122



Nevus of Ota: A series of 15 cases

Shanmuga Sekar, Maria Kuruvila, Harsha S. Pai 125



Premature ovarian failure due to cyclophosphamide: A report of four cases in dermatology practice

Vikrant A. Saoji 128

CASE REPORTS

Hand, foot and mouth disease in Nagpur

Vikrant A. Saoji 133



Non-familial multiple keratoacanthomas in a 70 year-old long-term non-progressor HIV-seropositive man

Hemanta Kumar Kar, Sunil T. Sabhnani, R. K. Gautam, P. K. Sharma, Kalpana Solanki, Meenakshi Bhardwaj 136



Late onset isotretinoin resistant acne conglobata in a patient with acromegaly

Kapil Jain, V. K. Jain, Kamal Aggarwal, Anu Bansal 139



Familial dyskeratotic comedones

M. Sendhil Kumaran, Divya Appachu, Elizabeth Jayaseelan 142



Nasal NK/T cell lymphoma presenting as a lethal midline granuloma

Vandana Mehta, C. Balachandran, Sudha Bhat, V. Geetha, Donald Fernandes



145

Childhood sclerodermatomyositis with generalized morphea

Girishkumar R. Ambade, Rachita S. Dhurat, Nitin Lade, Hemangi R. Jerajani.....



148

Subcutaneous panniculitis-like T-cell cutaneous lymphoma

Avninder Singh, Joginder Kumar, Sujala Kapur, V. Ramesh.....



151

LETTERS TO EDITOR

Using a submersible pump to clean large areas of the body with antiseptics

C. R. Srinivas



154

Peutz-Jeghers syndrome with prominent palmoplantar pigmentation

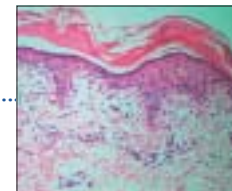
K. N. Shivaswamy, A. L. Shyamprasad, T. K. Sumathi, C. Ranganathan



154

Stratum corneum findings as clues to histological diagnosis of pityriasis lichenoides chronica

Rajiv Joshi



156

Author's reply

S. Pradeep Nair

157

Omalizumab in severe chronic urticaria

K. V. Godse.....

157

Hypothesis: The potential utility of topical eflornithine against cutaneous leishmaniasis

M. R. Namazi

158

Nodular melanoma in a skin graft site scar

A. Gnaneshwar Rao, Kamal K. Jhamnani, Chandana Konda



159

Palatal involvement in lepromatous leprosy

A. Gnaneshwar Rao, Chandana Konda, Kamal Jhamnani.....



161

Unilateral nevoid telangiectasia with no estrogen and progesterone receptors in a pediatric patient

F. Sule Afsar, Ragip Ortac, Gulden Diniz.....



163

Eruptive lichen planus in a child with celiac disease

Dipankar De, Amrinder J. Kanwar.....



164

Xerosis and pityriasis alba-like changes associated with zonisamide

Feroze Kaliyadan, Jayasree Manoj, S. Venkitakrishnan.....

165

Treatment of actinomycetoma with combination of rifampicin and co-trimoxazole

Rajiv Joshi.....



166

Author's reply

M. Ramam, Radhakrishna Bhat, Taru Garg, Vinod K. Sharma, R. Ray, M. K. Singh, U. Banerjee, C. Rajendran.....

168

Vitiligo, psoriasis and imiquimod: Fitting all into the same pathway

Bell Raj Eapen.....

169

Author's reply

Engin Şenel, Deniz Seçkin.....

169

Multiple dermatofibromas on face treated with carbon dioxide laser: The importance of laser parameters

Kabir Sardana, Vijay K. Garg.....

170

Author's reply

D. S. Krupa Shankar, A. Kushalappa, K. S. Uma, Anjay A. Pai.....

170

Alopecia areata progressing to totalis/universalis in non-insulin dependent diabetes mellitus (type II): Failure of dexamethasone-cyclophosphamide pulse therapy

Virendra N. Sehgal, Sambit N. Bhattacharya, Sonal Sharma, Govind Srivastava, Ashok K. Aggarwal.....



171

Subungual exostosis

Kamal Aggarwal, Sanjeev Gupta, Vijay Kumar Jain, Amit Mital, Sunita Gupta.....

173

Clinicohistopathological correlation of leprosy

Amrish N. Pandya, Hemali J. Tailor 174

RESIDENT'S PAGE

Dermatographism

Dipti Bhute, Bhavana Doshi, Sushil Pande, Sunanda Mahajan, Vidya Kharkar 177

FOCUS

Mycophenolate mofetil

Amar Surjushe, D. G. Saple 180

QUIZ

Multiple papules on the vulva

G. Raghu Rama Rao, R. Radha Rani, A. Amareswar, P. V. Krishnam
Raju, P. Raja Kumari, Y. Hari Kishan Kumar 185



E-IDVL

Net Study

Oral isotretinoin is as effective as a combination of oral isotretinoin and topical anti-acne agents in nodulocystic acne

Rajeev Dhir, Neetu P. Gehi, Reetu Agarwal, Yuvraj E. More 187

Net Case

Cutaneous diphtheria masquerading as a sexually transmitted disease

T. P. Vetrichevvel, Gajanan A. Pise, Kishan Kumar Agrawal,
Devinder Mohan Thappa 187



Net Letters

Patch test in Behcet's disease

Ülker Gül, Müzeyyen Gönül, Seray Külcü Çakmak, Arzu Kılıç 187

Cerebriform elephantiasis of the vulva following tuberculous lymphadenitis

Surajit Nayak, Basanti Acharjya, Basanti Devi, Satyadarshi Pattnaik,
Manoj Kumar Patra 188



Net Quiz

Vesicles on the tongue

Saurabh Agarwal, Krishna Gopal, Binay Kumar 188



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, $F_{\epsilon}R_1$. Subsequently, IgE is prevented from sensitizing cells bearing high-affinity $F_{\epsilon}R_1$ 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, $F_{\epsilon}R_1$. As occupancy of $F_{\epsilon}R_1$ by IgE determines the levels of surface $F_{\epsilon}R_1$ expression, this leads to a rapid depletion of both cell-bound IgE and surface $F_{\epsilon}R_1$ 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.

K. V. Godse

Consultant Dermatologist, Mumbai, India

Address for correspondence: Dr. Kiran Godse,
Shree Skin Centre, 22, L market, Sector 8, Nerul,
Navi Mumbai - 400 706 India.
E-mail: drgodse@yahoo.co.in

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