The Indian Journal of Dermatology, Venereology and Leprology (IJDVL) is a bimonthly publication of the Indian Association of Dermatologists, Venereologists and Leprologists (IADVL) and is published for IADVL by Medknow Publications.

The Journal is indexed/listed with Science Citation Index Expanded, PUBMED, EMBASE, Bioline International, CAB Abstracts, Global Health, DOAJ, Health and Wellness Research Center, SCOPUS, Health Reference Center Academic, InfoTrac One File, Expanded Academic ASAP, NIWI, INIST, Uncover, JADE (Journal Article Database), IndMed, Indian Science Abstract’s and PubList.

All the rights are reserved. Apart from any fair dealing for the purposes of research or private study, or criticism or review, no part of the publication can be reproduced, stored, or transmitted, in any form or by any means, without the prior permission of the Editor, IJDVL.

The information and opinions presented in the Journal reflect the views of the authors and not of the IADVL or its Editorial Board or the IADVL. Publication does not constitute endorsement by the journal.

The IJDVL and/or its publisher cannot be held responsible for errors or for any consequences arising from the use of the information contained in this journal. The Journal is printed on acid free paper.

Indian Journal of Dermatology, Venereology & Leprology

Vol 74 | Issue 1 | Jan-Feb 2008

EDITOR
Uday Khopkar

ASSOCIATE EDITORS
Ameet Valia Sangeeta Amladi

ASSISTANT EDITORS
K. C. Nischal Sushil Pande Vishalakshi Viswanath

EDITORIAL BOARD
Chetan Oberai (Ex-officio) Koushik Lahiri (Ex-officio) Sanjeev Handa
Arun Inamdar Joseph Sundharam S. L. Wadhwa
Binod Khaitan Kanthraj GR Sharad Mutalik
D. A. Satish M. Ramam Shruthakirti Shenoi
D. M. Thappa Manas Chatterjee Susmit Haldar
H. R. Jerajani Rajeev Sharma Venkatram Mysore
Sandipan Dhar

EDITORIAL ADVISORY BOARD
Aditya Gupta, Canada Jag Bhawan, USA
C. R. Srinivas, India John McGrath, UK
Celia Moss, UK K. Pavithran, India
Giam Yoke Chin, Singapore R. G. Valia, India
Gurmohan Singh, India Robert A. Schwartz, USA
Howard Libman, USA Robin Graham-Brown, UK
J. S. Pasricha, India V. N. Sehgal, India
Rodney Sinclair, Australia

STATISTICAL EDITOR
S. R. Suryawanshi

OMBUDSMAN
A. K. Bajaj

IADVL NATIONAL EXECUTIVE 2006 – 2007

President
Chetan M. Oberai

Immediate Past President
Suresh Joshipura

President (Elect)
S. Sacchidanand

Vice-Presidents
Amrinder Jit Kanwar Dilip Shah

Secretary
Koushik Lahiri Arijit Coondoo

Treasurer
Rakesh Bansal Manas Chatterjee

Jt. Secretaries
Rakesh Bansal Manas Chatterjee

EDITORIAL OFFICE
Dr. Uday Khopkar
Editor, IJDVL, Department of Dermatology, 117, 1st Floor, Old OPD Building, K.E.M. Hospital, Parel, Mumbai - 400012, India. E-mail: editor@ijdvl.com

Published for IADVL by
MEdKNOW PUBLICATIONS
A-109, Kanara Business Centre, Off Link Road, Ghatkopar (E), Mumbai - 400075, India. Tel: 91-22-6649 1818 / 1816 Website: www.medknow.com

www.ijdvl.com
www.journalonweb.com/ijdvl
www.bioline.org.br/dv
EDITORIAL REPORT - 2007

IJDVL gets into the Science Citation Index Expanded!
Uday Khopkar

EDITORIAL

Registration and reporting of clinical trials
Uday Khopkar, Sushil Pande

SPECIALTY INTERFACE

Preventing steroid induced osteoporosis
Jyotsna Oak

REVIEW ARTICLE

Molecular diagnostics in genodermatoses - simplified
Ravi N. Hiremagalore, Nagendrachary Nizamabad, Vijayaraghavan Kamasamudram

ORIGINAL ARTICLES

A clinicoepidemiological study of polymorphic light eruption
Lata Sharma, A. Basnet

A clinico-epidemiological study of PLE was done for a period of one year to include 220 cases of PLE of skin type between IV and VI. The manifestation of PLE was most common in housewives on sun exposed areas. Most of the patients of PLE presented with mild symptoms and rash around neck, lower forearms and arms which was aggravated on exposure to sunlight. PLE was more prevalent in the months of March and September and the disease was recurrent in 31.36% of cases.

Comparative study of efficacy and safety of hydroxychloroquine and chloroquine in polymorphic light eruption: A randomized, double-blind, multicentric study
Anil Pareek, Uday Khopkar, S. Sacchidanand, Nitin Chandurkar, Geeta S. Naik

In a double-blind randomized, comparative multicentric study evaluating efficacy of antimalarials in polymorphic light eruption, a total of 117 patients of PLE were randomized to receive hydroxychloroquine and chloroquine tablets for a period of 2 months (initial twice daily dose was reduced to once daily after 1 month). A significant reduction in severity scores for burning, itching, and erythema was observed in patients treated with hydroxychloroquine as compared to chloroquine. Hydroxychloroquine was found to be a safe antimalarial in the dosage studied with lesser risk of ocular toxicity.
Many faces of cutaneous leishmaniasis
Arfan Ul Bari, Simeen Ber Rahman ............................................................................................................................ 23

Symptomatic cutaneous leishmaniasis is diverse in its presentation and outcome in a tropical country like Pakistan where the disease is endemic. The study describes the clinical profile and atypical presentations in 41 cases among 718 patients of cutaneous leishmaniasis. Extremity was the most common site of involvement and lupoid cutaneous leishmaniasis was the most common atypical form observed. Authors suggest that clustering of atypical cases in a geographically restricted region could possibly be due to emergence of a new parasite strain.

Forehead plaque: A cutaneous marker of CNS involvement in tuberous sclerosis
G. Raghu Rama Rao, P. V. Krishna Rao, K. V. T. Gopal, Y. Hari Kishan Kumar, B. V. Ramachandra ....................................................................................................................................................... 28

In a retrospective study of 15 patients of tuberous sclerosis, eight patients had central nervous system involvement. Among these 8 cases, 7 cases had forehead plaque. This small study suggests that presence of forehead plaque is significantly associated with CNS involvement.

Ligand-binding prediction for ErbB2, a key molecule in the pathogenesis of leprosy
Viroj Wiwanitkit ........................................................................................................................................................... 32

SCORTEN: Does it need modification?

Universal acquired melanosis (Carbon baby)
P. K. Kaviarasan, P. V. S. Prasad, J. M. Joe, N. Nandana, P. Viswanathan ................................................................. 38

Adult onset, hypopigmented solitary mastocytoma:
Report of two cases
D. Pandhi, A. Singal, S. Aggarwal .................................................................................................................................... 41
<table>
<thead>
<tr>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidental finding of skin deposits of corticosteroids without</td>
<td>44</td>
</tr>
<tr>
<td>associated granulomatous inflammation: Report of three cases</td>
<td></td>
</tr>
<tr>
<td>Rajiv Joshi</td>
<td></td>
</tr>
<tr>
<td>Erythromelanosis follicularis faciei et colli: Relationship with</td>
<td>47</td>
</tr>
<tr>
<td>keratosis pilaris</td>
<td></td>
</tr>
<tr>
<td>M. Augustine, E. Jayaseelan</td>
<td></td>
</tr>
<tr>
<td>Naxos disease: A rare occurrence of cardiomyopathy with woolly hair</td>
<td>50</td>
</tr>
<tr>
<td>and palmoplantar keratoderma</td>
<td></td>
</tr>
<tr>
<td>R. Rai, B. Ramachandran, V. S. Sundaram, G. Rajendren, C. R. Srinivas</td>
<td></td>
</tr>
<tr>
<td>Granular parakeratosis presenting with facial keratotic papules</td>
<td>53</td>
</tr>
<tr>
<td>R. Joshi, A. Taneja</td>
<td></td>
</tr>
<tr>
<td>Adult cutaneous myofibroma</td>
<td>56</td>
</tr>
<tr>
<td>V. Patel, V. Kharkar, U. Khopkar</td>
<td></td>
</tr>
<tr>
<td>LETTERS TO THE EDITOR</td>
<td></td>
</tr>
<tr>
<td>Extragenital lichen sclerosus of childhood presenting as erythematous</td>
<td>59</td>
</tr>
<tr>
<td>patches</td>
<td></td>
</tr>
<tr>
<td>N. G. Stavrianeas, A. C. Katoulis, A. I. Kanelleas, E. Bozi, E.</td>
<td></td>
</tr>
<tr>
<td>Toumbis-Ioannou</td>
<td></td>
</tr>
<tr>
<td>Leukocytoclastic vasculitis during pegylated interferon and ribavirin</td>
<td>60</td>
</tr>
<tr>
<td>treatment of hepatitis C virus infection</td>
<td></td>
</tr>
<tr>
<td>Esra Adisen, Murat Dizbay, Kenan Hize, Nilser Ilter</td>
<td></td>
</tr>
</tbody>
</table>
Poland’s syndrome
Saurabh Agarwal, Ajay Arya ............................................................ 62

Hereditary leiomyomatosis with renal cell carcinoma
Sachin S. Soni, Swarnalata Gowrishankar, Gopal Kishan Adikey,
Anuradha S. Raman ........................................................................... 63

Infantile onset of Cockayne syndrome in two siblings
Prerna Batra, Abhijeet Saha, Ashok Kumar ........................................... 65

Multiple xanthogranulomas in an adult
Surajit Nayak, Basanti Acharjya, Basanti Devi, Manoj Kumar Patra .......... 67

Bullous pyoderma gangrenosum associated with ulcerative colitis
Naik Chandra Lal, Singh Gurcharan, Kumar Lekshman, Lokanatha K .......... 68

Sporotrichoid pattern of malignant melanoma
Ranjan C. Rawal, Kanu Mangla ......................................................... 70

Acitretin for Papillon-Lefèvre syndrome in a five-year-old girl
Didem Didar Balci, Gamze Serarslan, Ozlem Sangun, Seydo Homan ......................... 71

Bilateral Becker’s nevi
Ramesh Bansal, Rajeev Sen .............................................................. 73

Madarosis: A dermatological marker
Silonie Sachdeva, Pawan Prasher .................................................... 74
CONTENTS (Contd.)

FOCUS

Botulinum toxin
Preeti Savardekar ......................................................................................................................................................... 77

E-IJDVL

Net Studies
A study of oxidative stress in paucibacillary and multibacillary leprosy
P. Jyothi, Najeeba Riyaz, G. Nandakumar, M. P. Binitha .................................................................................................. 80

Clinical study of cutaneous drug eruptions in 200 patients
M. Patel Raksha, Y. S. Marfatia.................................................................................................................................... 80

Net case
Porokeratosis confined to the genital area: A report of three cases
Sujata Sengupta, Jayanta Kumar Das, Asok Gangopadhyay ................................................................................... 80

Net Letters
Camisa disease: A rare variant of Vohwinkel’s syndrome
T. S. Rajashekar, Gurcharan Singh, Chandra Naik, L. Rajendra Okade ................................................................... 81

Cross reaction between two azoles used for different indications
Arika Bansal, Rashmi Kumari, M. Ramam ................................................................................................................. 81

Net Quiz
Asymptomatic erythematous plaque on eyelid
Neeraj Srivastava, Lakhan Singh Solanki, Sanjay Singh .......................................................................................... 82

QUIZ
A bluish nodule on the arm
Ragunatha S., Arun C. Inamadar, Vamseedhar Annam, B. R. Yelikar ..................................................................... 83

REFEREE INDEX-2007

INSTRUCTIONS FOR AUTHORS

The copies of the journal to members of the association are sent by ordinary post. The editorial board, association or publisher will not be responsible for non-receipt of copies. If any of the members wish to receive the copies by registered post or courier, kindly contact the journal’s / publisher’s office. If a copy returns due to incomplete, incorrect or changed address of a member on two consecutive occasions, the names of such members will be deleted from the mailing list of the journal. Providing complete, correct and up-to-date address is the responsibility of the members. Copies are sent to subscribers and members directly from the publisher’s address; it is illegal to acquire copies from any other source. If a copy is received for personal use as a member of the association/society, one cannot resale or give-away the copy for commercial or library use.
INTRODUCTION

Madarosis is derived from the ancient Greek word “madaros” meaning “bald” and is defined as hair loss of the eyebrows (superciliary madarosis) or loss of eyelashes (ciliary madarosis). Loss of eyelashes is also known as milphosis. In addition to the obvious cosmetic blemish for which the patient usually presents to dermatologists or ophthalmologists, madarosis may be the presenting sign of many systemic diseases and warrants detailed systemic examination and in some cases, consultation with an internist or endocrinologist for further management. This article focuses on the various causes of madarosis.

ETIOLOGY AND ASSOCIATIONS OF MADAROSIS

1. Inflammation

Inflammation of the eyelids (blepharitis) can cause loss of eyelashes. It can be due to infection, seborrhea, trauma or allergy.[1-2]

a) Infections: Infection due to Staphylococcus aureus results in thin, honey-colored flakes (collarettes) among the eyelashes. Long-standing staphylococcal infection is associated with loss (madarosis), whitening (poliosis) and misdirection (trichiasis) of eyelashes. Madarosis has been reported as the most common ocular lesion (76%) in leprosy patients.[3] The ocular involvement is higher in lepromatous leprosy followed by borderline and tuberculoid leprosy and shows increased incidence with the age of the patient and duration of the disease.[4] Parasitic infestation of eyelids with the mite Demodex folliculorum commonly found in the pilosebaceous components of the eyelid can also result in the loss of eyelashes.[5] Mites have been found to be more abundant in older persons, diabetics, and those with S. aureus infection of the eyelid. These are characterized by the presence of waxy, cylindrical cuffs (hypertrophic follicular epithelium) around the bases of the eyelashes. The mite consumes epithelial cells, produces follicular distention and hyperplasia and increases keratinization leading (in eyelashes) to cuffing, which consists of keratin and lipid moieties. Follicular inflammation produces edema and results in easier epilation of the eyelashes. It also affects cilia construction so that lashes become brittle and fall.

b) Trauma from rubbing or plucking may be the cause of unilateral or bilateral lash loss.

c) Allergy: The loss of lashes may be secondary to allergy to the use of eye cosmetics such as mascara. Waterproof ‘mascaras’ are the most difficult to remove and can take too many lashes with them.

Systemic fungal infection with paracoccidioidomycosis can present with eyelid involvement in rare cases.[6] Active lesions present with erythematous patches of madarosis to frank destructive ulcers indistinguishable from malignancies while inactive lesions present with loss of eyelashes. Syphilis can also cause madarosis causing lateral brow loss (Hertoghe sign). Other infectious causes include chronic ulcerative blepharitis, tuberculosis, severe acute bacterial infections such as scarlet fever, viral infections such as herpes zoster, smallpox, measles, hepatitis, and chlamydia trachomatis infection.[1-2,7]
2. Autoimmune disorders
Loss of eyebrows and eyelashes can occur in association with alopecia areata. Although loss of scalp hair is usually present, rarely madarosis may be the presenting sign. Discoid lupus erythematosus (DLE) usually presents with lesions on the sun-exposed areas. Periocular involvement occurs uncommonly and may progress from eyelid erythema to scarring and madarosis. However, madarosis may be the presenting sign of DLE in the absence of any history of preceding erythema and scarring and should therefore be considered in the differential diagnosis of chronic blepharitis that persists despite usual medical management and eyelid hygiene. Madarosis has also been reported to occur in systemic lupus erythematosus and scleroderma.

3. Tumors
Benign and malignant tumors of the eyelids such as chalazion, squamous cell carcinoma, basal cell carcinoma, sebaceous carcinoma, lymphomas and sclerosing sweat duct carcinoma of the eyelid can present with loss of eyelashes.

4. Endocrine disorders
Hair follicle activity is affected in pathologic states such as hypothyroidism or hyperthyroidism. Changes of hair growth and hair structure may be the first clinical sign of a thyroid hormonal disturbance as a result of the influence on the cell cycle kinetics of the hair follicle cells. In hyperthyroidism, hair changes include thinning, breaking off, shortening of the hair and patchy areas of hair loss. Eyelash loss has been reported as an early sign in hyperthyroidism. In hypothyroidism, the hair may become dull, brittle and coarse, with reduced diameter and may involve the eyelashes and brows. Madarosis may also be associated with hypopituitarism and hypoparathyroidism.

5. Congenital causes
Loss of eyelashes, in association with other ocular abnormalities, has been reported in congenital ichthyosiform erythroderma, lamellar ichthyosis, hereditary ectodermal dysplasia syndrome, congenital atrichia, cryptophthalmos, Ehlers Danlos syndrome and lid coloboma.

6. Drugs and toxins
Idiosyncratic reaction resulting in unilateral madarosis and facial alopecia has been reported secondary to long-term use of Botulinum A injections for orofacial dystonia. Drugs such as miotics, anticoagulants, anticholesterol drugs, antihyroid drugs, boric acid, bromocriptine, propranolol, valproic acid and chronic epinephrine therapy have been reported to cause loss of eyelashes. Ciliary madarosis has also been reported following cocaine use. Intoxication with arsenic, bismuth, thallium, gold, quinine, and vitamin A can also cause loss of eyelashes.

7. Psychiatric causes
This includes trichotillomania which refers to a rare form of hair/eyelash loss resulting from avulsion of hairs by the patient. It is characterized by compulsive pulling out of one’s hair associated with tension or an irresistible urge before pulling, followed by pleasure or relief. The hairs are broken at different levels, they may be tufted, tortuous and some hair fibers may be abnormally longer than others. The hair follicles may be prominent.

8. Miscellaneous
Dermatological conditions such as acanthosis nigricans can be associated with ectodermal defects. Familial acanthosis nigricans has been reported with madarosis. Loss of eyelashes has also been reported in association with Vogt-Koyanagi syndrome, epidermolysis bullosa, rosacea, psoriasis, metabolic diseases such as mitochondriopathy, adrenoleukodystrophy, malnutrition, Meige syndrome, sickle cell anemia, HIV infection, post-proton beam irradiation for tumors of the choroid of the eye, eyelid tattooing, thermal injury and cryotherapy.

TREATMENT
Identification of the cause and its treatment will lead to reversal of madarosis in most cases. Madarosis can be camouflaged by eyeliner, artificial lashes affixed by methacrylate-based adhesive or permanent pigment tattooing. Interlesional triamcinolone can be tried in the case of loss of brows. Surgical repair of the traumatic madarosis can be done but good thickness of the eyelashes and ideal direction of their growth are difficult to achieve.

REFERENCES


