LETTERS TO THE EDITOR

POST KALA AZAR DERMAL LEISHMANIASIS

To the Editor

The article *Post-kala-azar Dermal Leishmaniasis by Padhiar* et al. in the latest issue of the Ind J Dermatol Venereol Leprol 1998; 64:243-244, highlights the fact that uncommon dermatosis prevalent in a particular area can be seen elsewhere due to migration of people to earn their livelihood. What rather confuses the reader short of distorting the facts are certain unusual observations that are conveniently left unexplained.

1. The opening word in the introductory part starts with "Post-kala azar dermal leishmaniasis....." and what follows does not make sense at all. However sense would be restored if the word is read as "kala-azar" instead of "Post-kala-azar dermal leishmaniasis".

2. The most typical presentation of post-kala-azar dermal leishmaniasis (PKDL) pertains to the face which is nearly always involved. No mention has been made of this unusual feature.

3. Peripheral smear (which one would understand as a peripheral blood smear) has been reported to be positive for Leishmania donovan (LD) bodies, the diagnostic feature of PKDL. This is very surprising for there is not a single report to my knowledge in the literature which mentions this observation. More surprising is it for the reader to learn that repeated slit-skin smears were done for AFB, but not once for L.D. body!

4. The combination of splenomegaly, lymphadenopathy, atypical clinical features and presence of L.D. bodies in the peripheral smear would make the diagnosis of PKDL a remote possibility.

5. Rifampicin and ketoconazole were given as treatment. This is again a deviation from the standard procedure which recommends sodium antimony gluconate injections since these drugs have been found to be ineffective in PKDL. The authors have not at all explained the rationale nor has the follow up of the patient been mentioned (since it has to be assumed by the reader that this combination was effective in this report).

These lapses cannot simply be ignored without proper explanation. Further the authors have not resorted to the large number of good clinical descriptions of this interesting dematosis in the Indian literature. The discussion is again a carry over of the major lapses in the report. Without even mentioning the presence of LD bodies in the histopathologic description the authors seem to have confirmed PKDL when writing the discussion! It would be informative to know the authors view on the points raised above.

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NAIL CHANGES SECONDARY TO SYSTEMIC DRUGS

To the Editor

Systemic drugs can affect nails and nail changes range from pain, colour change, shedding or loss of the nail plate. Nail changes after drugs can be due to toxicity to the matrix, nail bed or hyponychium and to the periungual structures.[1] Red lunulae were described after

systemic corticosteroid therapy in cases of alopecia areata.[2] Pigmentary changes in nails were reported after antimalarials.[3] Results of observation of nail changes induced by drugs in our study are given in table.

Table I. Nail changes induced by drugs

Sr. No.	Age and Sex	Name of drug	Dose and duration	Type of nail changes	Recovery Period
1	20 M	Ketoconazole	200mg BDx7days	Blue lunulae of thumb nails	2 months
2	35 F	Ciprofloxacin	500mg BDx7days	Blue lunulae of thumb nails	3 months
3	19F	Cefadroxyl	500mg BDx7 days	Blue lunulae of finger nails	2 months
4	46F	Cyclophos-phamide Dexamethasone Pulse therapy	500mg IV-Ist day and 50mg OD 1000mgx3 days IV in 5% Dextrose		
5	50M	Carbamazepine Phenytoin Lonazepam	200mg BD-5 Yrs 200mg-BD 4 Yr. 0.5mg BD-3 Yr.	Lunula dystrophy, Pain	Persisting
6	21M	Phenytoin	200mg BD-15 day	Blue discolouration of all finger and toenails	Persisting
7	35M	Chloroquin	200mg BD	Black pigmented bands on finger nails	6 months
8	36M	Mefloquin	250 mg BDx3 days	Photoonycholysis	Lost to follow up

Age range was 19 to 50 years. There were 5 males and 3 females. Bluish discolouration[1] of lunulae[2] was the commonest change seen in 4/8 cases after ketoconazole, ciprofloxacin, cefadroxyl, and cyclophosphamide plus corticosteroid combination. Painful dystrophy of lunulae was observed after combination of carbamazepine, phenytoin and lonazepam. Prominent and persistent bluish grey discolouration of all twenty nails was observed after phenytoin. Black longitudinal bands were observed in a case of DLE after chloroquin intake. Mefloquin resulted in phototoxic drug eruptions, was withdrawn, and even after 2 weeks of withdrawal resulted in photoonycholysis. Discolouration disappeared in 4 after 2 to 6 months of discontinuation of the offending drugs and persisted in 3 who were unable to stop the drugs and one patient was lost to follow up.

Nail changes, secondary to systemic drugs are rare as only 8 cases were collected during 21/2 yrs.

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References

1. Daniel CR, Scher RK. Nail changes secondary to systemic drug ingestants. J Am Acad Dermatol 1984; 2: 250-255.

2. Wilkerson MG, Wilkin JK. Red lunulae revisited. A clinical and histopathological examination. J Am Acad Dermatol, 1989; 3: 453-457.

3. Fenton D, Wilkinson JD. The nail in systemic diseases and drug induced changes. In: Disease of the Nails and Their Management. Eds Baran R., Dawber RPR. Blackwell Scientific Publications London. 1984; 248-258.

FOOT WEAR DERMATITIS

To the Editor

Footwear dermatitis is a common disorder with an overall prevalence of 3 to 11%.[1-4] The common sensitizers are potassium dichromate, colophony and rubber accelerators.[4] Many patients with sensitivity to potassium dichromate still prefer leather shoes. Rubber glues are used in the manufacture of leather footwear thus making leather footwear unsafe for patients with sensitivity to rubber.

We recently had a patient who had sensitivity to potassium dichromate and rubber accelerators. As finding a suitable footwear posed a challenge we wrote to central leather research institute. The institute (Shoe design and