LETTERS TO THE EDITOR

POST-KALA-AZAR DERMAL LEISHMANIASIS WITH ATYPICAL PRESENTATION.

To the Editor,

Post-kala-azar dermal leishmaniasis (PKDL) is a distinct clinical entity which occurs 1-6 years after an attack of visceral leishmaniasis. Three main types of lesions have been described: hypopigmented macules, which may be pin point to 1 cm and may sometimes coalesce; erythematous butterfly rash on the face; and infiltrative nodules.[1] We present a case of PKDL who had extensive hypopigmented macules.

An 18-year-old boy presented with infiltrated skin coloured nodules of varying sizes over central part of face trunk, upper and lower extremities. Many of the lesions on the lower trunk and thighs had coalesced to form large hypopigmented macules and in lower extremities the lesions covered the whole limb. There was history suggestive of kala-azar 8 years back for which the patient had been adequately treated. Skin smear from infiltrated nodule was positive for *Leishmania donovani* bodies on staining with giemsa. Histology from skin lesion, was consistent with post-kala-azar dermal leishmaniasis. Routine hematological investigations, the liver, renal function tests and ultrasonography of abdomen were within normal limits.

The hypopigmented lesions of PKDL are usually pinpoint to 1 cm in size and may coalesce. We have seen several cases of PKDL with hypopigmented macules, but in none of the patients were the lesions so extensive. On reviewing the literature also we did not come across any reference of presence of extensive hypopigmented lesions in PKDL.

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Reference

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AUTOHAEMOTHERAPY IN CHRONIC URTICARIA

To the Editor,

Urticaria is characterised by transient erythematous or oedematous swelling of dermis or subcutaneous tissue.[1] It can be acute when it is of less than 2 months duration or it can be chronic if it lasts for more than 2 months.

Besides genetic predisposition many other factors are responsible for urticaria e.g., foods and preservatives, drugs like penicillin, salicylates etc, insect bites, emotional stress and internal diseases like anaemia, worm infestation etc. Many physical factors like heat, cold, water, sun exposure also promote urticaria like reactions. Some defects in the immune regulatory system can also lead to urticaria.

Different modalities are used for treatment of urticaria. Main line of treatment consists of treatment of the cause, if detectable, alongwith antihistaminics. But combination of both H_1 and H_2 blockers are helpful as they block the release of histamine at both H_1 and H_2 receptor sites. Corticosteroids are used in acute cases only. Mast cell stabilizing agents like sodium

cromoglycate, terbutaline and ketotifen are also used. Tranquilizers are of help in cases with psychogenic component. Topically soothing agents like calamine are used. But none of these modalities has given satisfactory results. Autohaemotherapy is being evaluated for treatment in chronic urticaria.

We have tried autohaemotherapy in 50 cases of chronic urticaria. Every possible cause was ruled out in all the cases. Investigations were within normal limits except for more than 7% eosinophils in every case. 50 patients were given autohaemotherapy and 50 were treated by other modalities. The procedure consisted of taking blood from patient's cubital vein and directly injecting it into gluteus muscle of same patient without mixing the blood with any anticoagulant. During first week 2 ml blood was injected biweekly and then 5 ml biweekly in second week and in the third week 10 ml blood was given biweekly.[2]

We found that patients responded better to autohaemotherapy than to any other modalities. Itching subsided quickly, duration of weal was also decreased and interval between two episodes of the disease was increased from days to weeks.

Due to lack of satisfactory treatment and good results of autohaemotherapy it should be considered in the treatment of chronic urticaria but it needs further trials.

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SCLEREDEMA

To the Editor,

An 8-year-old boy presented with sudden onset of thickened, taut skin over the neck, shoulders rapidly spreading to involve the face, upper trunk, arms and upper abdomen preceded with mild prodrome. Increased pigmentation was seen over affected areas with puffiness of face and inability to open the mouth. The skin was bound down, non-tender with non-pitting oedema. No clear line of demarcation could be detected between affected and unaffected skin. Distal upper extremities and lower limbs were remarkably free. The patient had pyoderma one month ago. There was no history of fever, joint pain or Raynaud's phenomenon in the past or present. Other systems were not involved. Routine haematological examination and urinalysis were within normal limits except ESR which was raised. ASO titre was high (250 Todd units/ml). Tests for Rheumatoid factor and LE cell phenomenon were negative. ECG and chest X-ray did not reveal any abnormality. Histopathology of biopsy specimen from scapular region stained with H and E showed characteristic findings[1] of scleredema. No specific treatment was given but the child showed spontaneous improvement from 3 weeks onwards and after 6 months there were no residual sign of disease. The characteristic clinical course, histopathological findings were somewhat similar to other documented cases of scleredema.[1,2] In view of the sudden onset, preceding history of pyoderma and raised ASO titre a streptococcal hypersensitivity reaction is suggested.

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