advocated as treatment for exfoliative cheilitis. However the disease tends to persist.

Since my patients had not responded to any treatment, I intiated an empirical treatment with twice a day application of vitamin A oil (obtained by opening a vitamin A capsule containing 50,000 IU). Both patients responded well after 2 months. The applications were gradually tapered and stopped in 4 months. The Indian patient has had no recurrences. The Lebanese patient was lost for follow-up after 1 year. He had no recurrence at that time.

It is difficult to postulate the role of vitamin A in these cases. Vitamin A may have had some effect on keratinization.

Jayadev Betkerur
Mysore

References

1. Domonkos AN, Anrold HL, Odom RB. Diseases of the mucous membranes. In: Andrew's diseases of the skin. Tokyo: Igaku-Shoin/Saunders, 1982:985.
2. Pindborg JJ. Disorders of oral cavity and lip. In : Rook A, Wilkinson DS, Ebling FJG,eds. Textbook of dermatology. Oxford : Blackwell , 1979:1905-6.

BALANOPOSTHITIS IN CHILDREN WITH SCABIES

To the Editor,

Scabies is a common parasitic disorder which at times can be of venereal origin. Usually erythema, papules, papulovesicles, urticaria, pustules, folliculitis, vesicles, nodules, bullae and burrows are seen. Some cases of scabies have associated balanoposthitis which has not received due importance and therefore we are reporting balanoposthitis in children with scabies.

Ten cases of balanoposthitis in children below 10 years of age suffering from scabies were selected for this study from dermatovenereology out patients. Detailed history, general physical, systemic and dermopathological examinations were carried out to rule out venereal diseases. Urine examination and urethral smears were prepared in all cases.

Seven cases had nodular scabies, that is nodules on penis and external genitalia, and 3 had routine scabies. Balanoposthitis was observed in the form of oedema, erythema of urethral meatus and prepuce associated with mucoid urethral discharge. Culture and sensitivity test of urine in all 10 cases ruled out UTI. Urethral smears revealed Gram +ve cocci in 8/10 cases and all 10 cases had polymorphs and occasional squamous cells. Scabies was treated with 1% GBHC for 12 hours and balanoposthitis by topical soframycin twice/day and both conditions were cured by 2 weeks.

Balanoposthitis in association with scabies in adults can be venereal/non-venereal. Balanoposthitis in children with scabies was non-venereal in this study. Balanoposthitis was more common in cases of nodular scabies where persistent pruritus and colonization by Gram +ve cocci could predispose to balanoposthitis. Cure with disappearance of residual post scabetic pruritus was better if balanoposthitis was treated simultaneously with scabies.

R R Mittal, Rita Dutt, S P Singh
Patiala

CUTIS VERTICIS GYRATA WITH EPILEPSY

To the Editor,

A 28-year-old man presented with corrugated scalp, abnormal physical growth, poor mental development and epileptic seizures since early childhood. Family and

personal history did not suggest genetic and systemic disease.

On physical examination, there were multiple furrows and folds of 2-4 cm depth and 20-30 cm length, which were arranged sagitally over the scalp. Psychometry suggested mental age of 5 years and IQ was 35. EEG showed generalised paroxysmal epileptiform activity. Other routine haematological, biochemical and radiological investigations were normal. He was provided anti-epileptic treatment and suggested for plastic surgery of the scalp.

Though cutis verticis gyrata was coined by Unna in 1907 for cerebriform appearance of the skin, Fisher (1922) provided aetiopathological details of the disorder. Various systemic and cutaneous inflammatory disorders may result into cutis verticis gyrata. Nevertheless autosomal recessive or dominant inheritance may be found in some cases. The male predominance in cutis verticis gyrata may be because of androgenic hormones and a lethal factor operating in female foetuses.[1] A distinct Lennox-Gastaut syndrome has been described to show a symmetrical spike wave discharges at less than 3Hz even in sleep.[2] Rotational traction over scalp hair can produce cutis verticis gyrata in normal person.[3]

The underlying pathology responsible for thickening of the scalp lies in abnormal proliferation of collagen and reticular fibres, deposition of mucopolysaccharides in corium and epithelial hyperplasia. In the index case evidence of mental retardation and the distinct EEG pattern without any other systemic abnormality indicates primary cutis verticis gyrata.

Jeetendra Saraswat, CM Kuldeep
Jaipur

References

1. Polar S, Butterworth T. Cutis verticis gyrata: review with report of seven new cases. Am J Ment Defic 1953; 57: 613-31.
2. Paulson GW. Cutis verticis gyrata and the lennox syndrome. Develop Med Child Neurol 1974; 16: 196-200.
3. Khare AK, Singh G. Acquired cutis verticis gyrata due to rotational traction. Br J Dermatol 1984; 110: 125-6.

SYSTEMIC CORTICOSTEROIDS IN TOXIC EPIDERMAL NECROLYSIS

To the Editor,

The role of systemic corticosteroids in the management of toxic epidermal necrolysis (TEN) is a subject of controvesy. Some workers believe that the patients who receive corticosteroids experience a poorer outcome than those who do not,[1] whereas others feel that steroids reduce the morbidity and mortality when given early enough and in sufficiently high dosage (prednisolone 2-8mg/kg/day).[2] In a study of 30 patients with drug-induced TEN (DTEN), it was observed that overall prognosis was much better in those patients who were put on high dose systemic steroids within 7 days of its development.[3] Our recent experience of treating TEN in children and adolescents with systemic steroids was also quite encouraging.[4]

In an appraisal, Pasricha highlighted the usefulness of high dose corticosteroid started very early in the management of TEN.[5] A rapid tapering followed by withdrawal within 2 weeks was recommended.

It is not difficult to understand why early institution of steroid would reduce mortality in TEN. Majority of TEN cases are due to antibody dependent cell mediated cytotoxicity (ADCC) type of hypersensitivity phenomenon which is very sensitive to corticosteroids.[6]