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.

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## 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]

Thus, steroid instituted in high doses and early in the diseases halts ADCC reaction preventing further tissue damage by the ongoing process of TEN. Once tissue damage (maximum within first 7 days) has occurred, human body systems have to face its consequences and steroids have very little role to play. Once steroids have counteracted ADCC phenomenon and further tissue damage, and the offending drug has been omitted, no more steroids are required and these should be tapered rapidly and stopped.[5] Unnecessary prolongation of steriod therapy may increase the mortality by increasing the incidence of secondary infection and sepsis.[1]

There are occasional reports of patients with TEN not responding to even very high dose(s) of corticosteroid(s) instituted from the beginning. In these patients, a mixed lymphocyte reaction with the production of killer lymphocytes, resembling acute graft versus host diseases (GVHD) is the probable underlying process.[6]

It is, therefore, felt that if the corticosteroids are used in low dosage, later in the disease and there is lengthy tapering (3 Ls), their usefulness is questioned. It is the lack of knowledge and expertise about how to use it rather than the lack of its efficacy which has made the role of corticosteroid(s) a matter of debate.

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## COWPOX

*To the Editor,*

A 20-year-old male from Bhilai presented with asymptomatic, vesicular lesion over right thumb of one week's duration. There was history of milking a cow having ulcerated teats about one week prior to the development of cutaneous lesion. There was no history of fever or malaise. Cutaneous examination showed one vesicular lesion of about 5 mm diameter having central umbilication and surrounding erythematous ring over dorsal aspect of proximal phalanx of right thumb (Fig.1). There was no regional lymphadenopathy. Biopsy of the lesion revealed massive spongiosis and reticular degeneration at several places in epidermis with marked acute inflammatory dermal infiltrate (Fig.2).

Cowpox is an occupational viral disease which affects persons who have been in contact with cows having infected teats.[1] However, half of the patients will not have such a history.[2]

Transmission of the virus from cat to man has been described.[3,4] A small wild rodent may be the reservoir of cowpox virus.[5] The lesions are characteristically found on the exposed skin, mostly on hands.[2] The incubation period varies from 2-14 days. It may affect the milk yield from inflammed teats