Zinc Responsive Acrodermatitis in Nephrotic Syndrome

Sir,

A 13-year-old boy presented with dark-colored, itchy skin lesions over both the extremities and back since 6 months. He was previously diagnosed as a case of lichen planus, dermatophytosis, and scabies elsewhere, and was treated unsuccessfully. He was a known case of idiopathic nephrotic syndrome since the age of 7 years and had received oral corticosteroids and cyclosporin. There was no history of aerodigestive distubances. General examination showed cushingoid features. Dermatological examination revealed symmetrical, well-defined, scaly, hyperpigmented papules, and plaques over the lower extremities, gluteal region, groin, genitalia, elbows, dorsum of hands, and lower trunk, admixed with hypopigmentation and depigmentation [Figure 1a and b], which resolved following treatment with oral zinc [Figure 1c and d]. Face, hair, nails, palms, soles, and mucosae were normal. Provisional diagnosis of acrodermatitis in nephrotic syndrome due to zinc deficiency was made.

His serum zinclevel was16.2 μg/dl (normal: 70-150 μg/dl) and serum alkaline phosphatase was 52 IU/L (normal: 70-390 IU/L), which were low in addition to hypoproteinemia, hypoalbuminemia, and proteinuria. Renal parameters were normal and hepatitis B/C serology were negative. Renal biopsy showed focal and segmental glomerulosclerosis (not otherwise specified), with clusters of foam cells in the interstitium (Alports syndrome to be ruled out). However, on audiogram, hearing sensitivity was normal in both ears and ophthalmic examination was normal. Urine protein was 3+. Biopsy from the skin lesion showed hyperkeratosis, parakeratosis, subcorneal vesicles, spongiosis, psoriasiform acanthosis, and perivascular infiltrate consisting of mononuclear cells [Figure 2a and b].

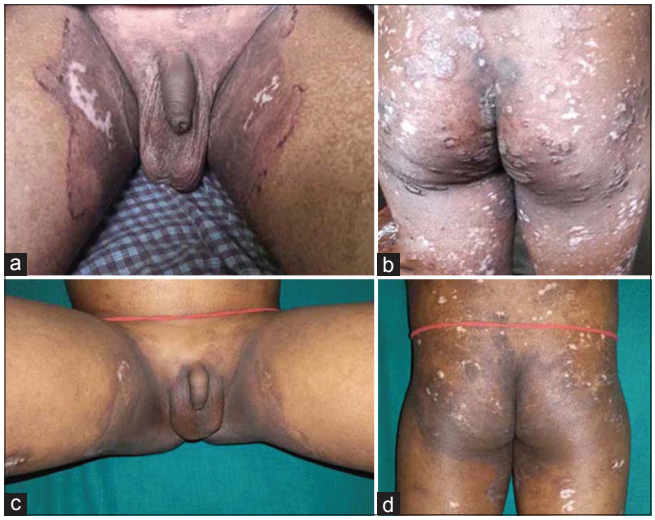

Figure 1: (a and b) Psoriasiform and lichenoid papules and plaques involving groin and both gluteal regions. (c and d) Complete resolution of the lesions following treatment, with residual pigmentation

Based on the above mentioned findings, a diagnosis of acrodermatitis due to acquired zinc deficiency was confirmed. Patient was started on oral zinc at a dose of 1 mg/kg/day. Lesions started resolving in 3 weeks, and complete resolution was observed at the end of 2 months with residual pigmentation and without any change in the course of renal disease.

The dermatological manifestations of zinc deficiency may be acute or insidious in nature. Acute deficiency can manifest as vesiculobullous, erosive, or as a scaling eruption involving the periorificial areas, hands and feet, whereas chronic deficiency presents as lichenified and psoriasiform plaques on the dorsa of hands and feet.[1]

The probable mechanisms for zinc deficiency in nephrotic syndrome are decreased intestinal absorption

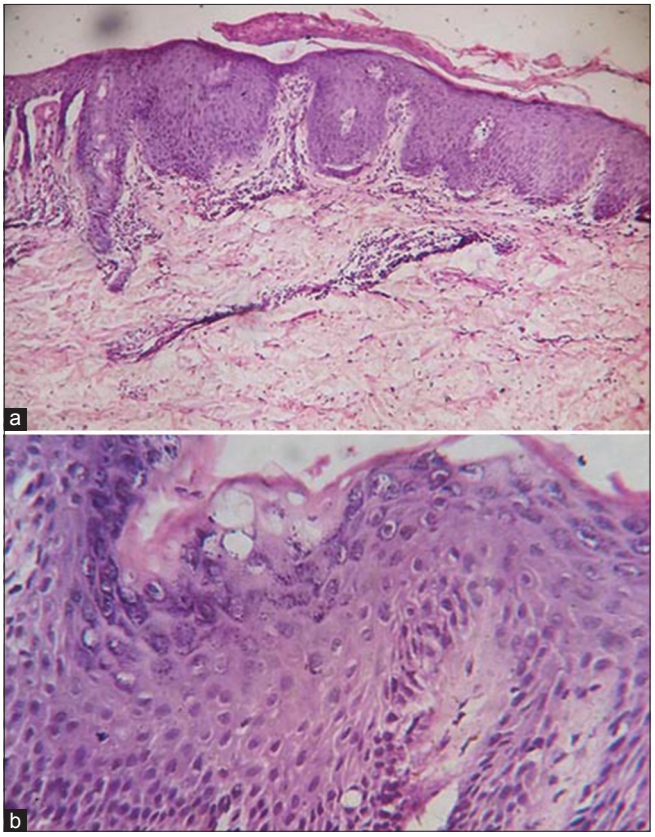

Figure 2: (a) Biopsy from the lesion revealed hyperkeratosis, parakeratosis, subcorneal vesicles, spongiosis, psoriasiform acanthosis, and perivasular infiltrate consisting of mononuclear cells (Hematoxylin and eosin; x10). (b) Biopsy from the lesion showed subcorneal vesicles. (Hematoxylin and eosin; x40)

due to gut edema, increased intestinal secretion, and proteinuria.[2]

The exact pathophysiologic mechanisms underlying the dermatological manifestations in zinc deficiency are still an enigma. Not all patients with zinc deficiency in nephrotic syndrome develop skin manifestations. Either concomitant immunosuppression may prevent development of overt skin manifestations or zinc deficiency in these patients is not severe enough to produce symptoms.[3]

Zinc deficiency can be diagnosed by decreased serum zinc and alkaline phosphatase levels, the latter should be monitored along with serum zinc levels during the evaluation and treatment of zinc deficiency.

Treatment consists of zinc supplementation at 1mg/kg in divided doses, and higher doses are recommended if continuing renal or gastrointestinal loss is suspected.[4] Maintanence dose should be administered after clinical resolution.

This case is being reported for its rarity, unusual extensive involvement of lower trunk, and gluteal areas, which has not been reported in previous literature,[5] as well as to highlight the significance of early diagnosis and prompt treatment which will help to reduce the morbidity and improve the quality of life.

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Conflicts of interest

There are no conflicts of interest.

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