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 disturbances. 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 zinc level was 16.2 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].

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 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](image1)

![Figure 2: (a) Biopsy from the lesion revealed hyperkeratosis, parakeratosis, subcorneal vesicles, spongiosis, psoriasiform acanthosis, and perivascular infiltrate consisting of mononuclear cells (Hematoxylin and eosin; x10). (b) Biopsy from the lesion showed subcorneal vesicles. (Hematoxylin and eosin; x40)](image2)
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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Sturge–Weber Syndrome with Choroidal Hemangioma
Sir,

A 13‑year‑old girl presented to the Department of Ophthalmology, with dimness of vision in the left eye. She had a port‑wine stain on the face bilaterally, along with the involvement of left upper lid [Figure 1]. The left eye had an elevated round orange subretinal mass with ill‑defined margins, suggestive of circumscribed choroidal hemangioma [Figure 2]. The left eye showed prominent red reflex due to the red color of the elevated hemangioma and induced hypermetropia. Intraocular pressure was raised in the left eye. She did not give a history of seizures. Based on the history and examination findings, a diagnosis of Sturge–Weber syndrome (SWS) was made. She is presently being managed under a multidisciplinary approach for her ophthalmic condition and cutaneous lesion.

The manifestations of SWS include port‑wine stain along the distribution of the trigeminal nerve, ipsilateral choroidal hemangioma, glaucoma, seizures, and leptomeningeal angioma.[1,2] The choroidal hemangioma may be circumscribed or diffuse, and may be isolated or associated with SWS.[3] Treatment options for choroidal hemangioma causing vision loss include laser, photodynamic therapy, and radiotherapy.[3] In 15% of SWS (similar to our patient), port‑wine stain may be bilateral.[2] Pulsed dye laser is the treatment of choice for port‑wine stains.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and/or personal information to be reported in the journal. The authors state that the patient(s) does/don’t wish his/her/their image/s to be published and this has been confirmed by patient(s).

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