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Acrodermatitis Enteropathica: A Classical case responsive to Zinc

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Abstract

Abstract: Acrodermatitis Enteropathica is an uncommon zinc deficiency disorder being known as basically of two types genetic or primary and secondary or acquired. It usually manifests as psoriasiform and/or vesicobullous dermatitis of distal extremities and periorificial areas associated with alopecia and intractable diarrhea. The response to systemic zinc is very quick. Herein, we report a case of acrodermatitis enteropathica in a 2 year old girl having classical features of zinc deficiency.

Keywords: Acrodermatitis enteropathica, zinc transporter, psoriasiform

Introduction

Acrodermatitis enteropathica (AE) is a rare disorder of zinc deficiency manifesting as acral and periorificial dermatitis, alopecia, intractable diarrhea, and failure to thrive. It has been classified as primary or genetically based deficiency, and acquired/secondary deficiency.[1] Herein, we report an uncommon case of AE in a 2 year old girl having classical features of zinc deficiency.

Case Report

A 2-year old female child came to the outpatient door of skin department presenting with skin lesions on periorificial and acral areas, and recurrent diarrhoea for past 6 months. The lesions were initially vesicobullous in nature which turned into eczematous form over the same sites.

Local cutaneous examination demonstrated erythematous, eczematous and scaly lesions distributed symmetrically in the perioral, acral and perianal areas **[Figure 1a-c].** The lesions were also crusted at some sites such as buttock. The child was very irritable too and increased frequency of stool with altered consistency were noted.

Systemic examination was unremarkable. Laboratory investigations revealed microcytic anemia (Hb-9.6%, Haematocrit -30%, mean corpuscular volume – 68 fL) and serum zinc level were very low, 28 microgram/dl.

The histopathologic examination could not be performed due to denial of the patient's parents.

Based on the above clinical and laboratory findings, diagnosis of acrodermatitis enteropathica was made and

patient was put on oral zinc therapy and antibiotics for diarrhoea. Oral rehydration was also done to prevent water loss. After 4 weeks of treatment, significant improvement was seen in all the cutaneous lesions [**Figure 2a-c].**

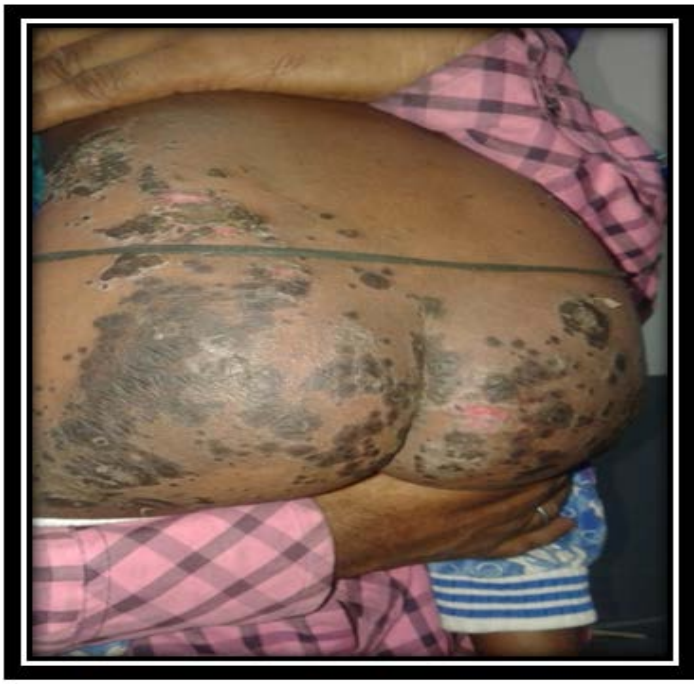

Figure 1a: Crusted lichenified plaques with erosion

Figure 1b: Eczematous lesions on cheeks and perioral areas

Figure 1b: Eczematous lesions on cheeks and perioral areas

Figure 1c: Eczematous lesions on extremities

Figure 2a: Significantly improved lesions on face

Figure 2b: Similar improvement on limbs

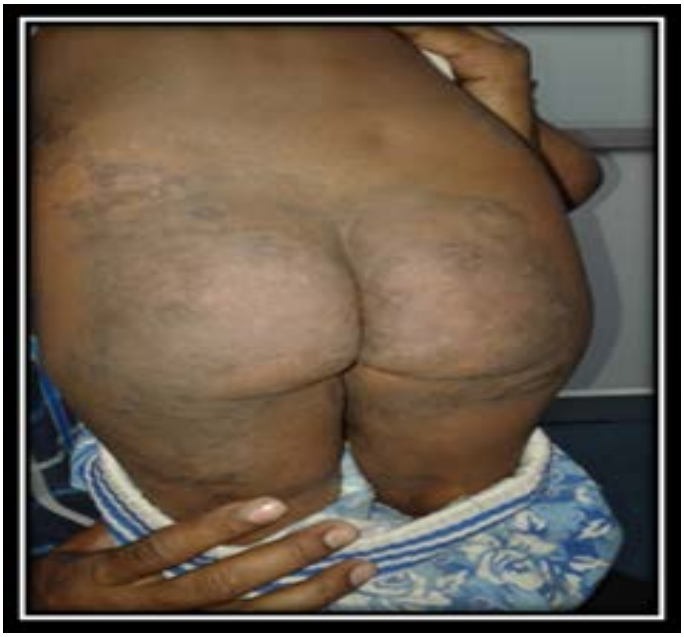

Figure 2c: Resolution of crusted lesions lesions on buttock

Discussion

Acrodermatitis Enteropethica is a broad spectrum of zinc deficiency disorders, classically caused by the defects in zinc transporters involved in intestinal zinc uptake Zrt- and Irt-like protein-4 (ZIP), causing classical AE.[2],[3] The other form of genetic AE is of lactogenic origin due to defective breast milk zinc transporter-2 (ZnT) resulting in impaired zinc secretion in breast milk. Acquired form of AE develop due to inadequate zinc intake, usually after weaning of child.[3] Besides, other variants of AE also exist in the condition of low serum albumin level or low serum alkaline phosphatase.

Whatever be the underlying aetiology, the Initial presenting symptoms are similar and very characteristics in the child. It starts with the development of scaly, erythematous, psoriasiform plaques with vesicobullous eruption and erosion.[4] The sites of predilection are skin around orifices such as perioral and perianal, and pressure or friction-prone areas of extremities such as elbow, knee and buttocks. Over the time, lesions may get hyperkeratotic and/or lichenified. The child becomes irritable and developmental milestones get affected. The reversible alopecia is another clinical hallmark of AE.[4]

Therapeutically, the response to zinc therapy is very quick and surprising. The standard dose of oral zinc for AE is 2-3 mg/kg of body weight as loading dose till the serum level becomes normal. Then, dose is reduced to 1-2 mg/kg for maintenance.[5] Of note, for classical AE in which intestinal zinc transported is defective, the lifelong zinc supplementation is required for the child. In case of acquired AE, once the serum zinc level normalizes, adequacy of zinc intake is sufficient enough to prevent the further episode of AE occurrence. [5]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent form. In the form, the patient's parents have given their consent for the images and other clinical information to be reported in the journal. They understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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