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Acquired zinc deficiency: A case report

Maruška Marovt[1]✉, Vlasta Dragoš[2]

Abstract

Zinc deficiency can be an autosomal recessive inherited or acquired disorder and is classically characterized by acral and periorificial dermatitis, alopecia, and diarrhea. We report a case of acquired zinc deficiency in a premature 6-week-old boy with typical skin manifestation of zinc deficiency and decreased plasma zinc level. After starting zinc replacement therapy, the skin lesions completely disappeared in the first few weeks.

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Introduction

Zinc deficiency can be an autosomal recessive inherited or acquired disorder and is classically characterized by acral and periorificial dermatitis, alopecia, and diarrhea. We report a case of acquired zinc deficiency in a premature 6-week-old boy with typical skin manifestation of zinc deficiency and decreased plasma zinc level.

Case report

A 6-week-old boy presented with a 2-week history of confluent erythematous, scaly, crusted patches on his cheeks and in the diaper area (Fig. 1). The boy was born in the 26th week of gestation, but was otherwise doing well. Before coming to our outpatient clinic, he was treated for presumed milk hypersensitivity reaction with diet and topical corticosteroids, but no improvement was observed. Physical examination did not reveal any abnormalities. In a laboratory test to determine the serum zinc level, the value was profoundly decreased (4.1 µmol/l, normal 10.0–19.7 µmol/l). Acquired zinc deficiency due to prematurity was diagnosed based on clinical and laboratory findings. After starting oral zinc replacement therapy (0.5 mg/kg/day), the skin lesions completely cleared within the first few weeks (Fig. 2). After 3 months, zinc replacement therapy was stopped and we did not observe further relapses in follow-up examinations.

Discussion

Zinc deficiency can be divided into two forms: congenital, also known as acrodermatitis enteropathica (first identified by Danbolt and Closs in 1942 (1)), and acquired form. Acrodermatitis enteropathica is believed to be caused by defective gene SLC39A4, which encodes the Zip4 zinc transporter (2). Skin manifestation and clinical findings can be similar in both types. It is of crucial importance to differentiate between congenital and acquired forms mainly because of the duration of zinc replacement therapy. In the acquired forms it is variable, but always limited, and there is no recurrence when oral replacement is discontinued (3).

Zinc is an essential mineral for humans and is quantitatively the second most important after iron (4). The functions of zinc have been divided into three categories: catalytic, structural, and regulatory (2). Zinc is an essential component of the catalytic site of hundreds of different metalloenzymes; it is an important structural component of gene regulatory proteins and is also known to regulate gene expression by binding to the metal response element transcription factor (2).

Major causes of zinc deficiency are summarized in Table 1.

The clinical presentation of acrodermatitis enteropathica is similar to deficiency dermatitis caused by low dietary zinc (2).

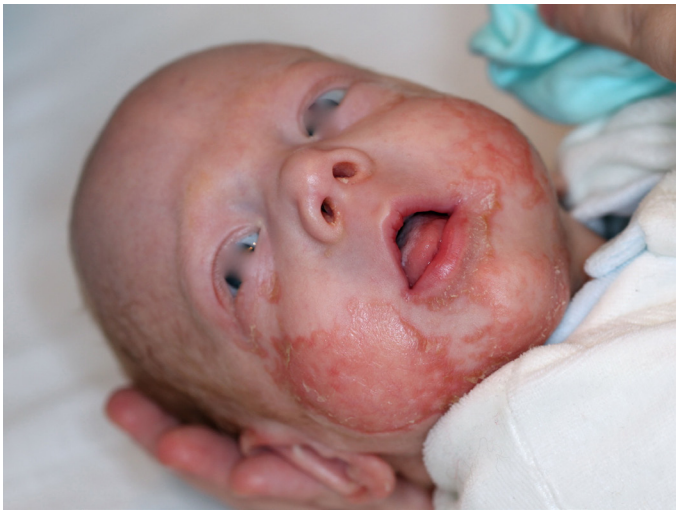

Figure 1 | Confluent erythematous, scaly, crusted patches on the cheeks.

Figure 2 | After treatment.

[1]Department of Dermatovenerology, Maribor University Medical Center, Ljubljanska cesta 5, 2000 Maribor, Slovenia. [2]Dermatovenerology Clinic, Ljubljana University Medical Center, Zaloška cesta 2, 1000 Ljubljana, Slovenia. ✉**Corresponding author: marovtmaruska@hotmail.com**

Table 1 | Distribution of BCC lesions and patient characteristics.

1. Inadequate intake	**3. Excessive loss**
1) Low-zinc-containing diets: foods poor in animal protein (vegetarians)	1) Loss into digestive fluid: child intractable diarrhea, intestinal fistula, gastrointestinal disease associated with diarrhea
2) Loss of zinc during food processing (desalting during production of artificial milk)	2) Increased urinary elimination: liver cirrhosis, diabetes mellitus, renal disease, hemolytic anemia, intravenous alimentation, enhanced catabolism (surgery, trauma, infection, etc.), diuretics, sodium polyphosphate
3) Prolonged intravenous alimentation, enteral alimentation	3) Others: burns, hemodialysis
4) Shortage of nutrient intake	
2. Malabsorption	**4. Increased demand**
1) Congenital: acrodermatitis enteropathica (very rare)	Pregnancy, neonates (premature babies), enhanced anabolism (during intravenous alimentation, etc.)
2) Acquired	
(1) Ingestion of absorption inhibitors: phytic acid, edible fibers	**5. Unexplained**
(2) Malabsorption syndrome: liver dysfunction, inflammatory bowel disease, short bowel syndrome	Congenital thymus defect, Down syndrome
(3) Drugs, chelating agents: EDTA, penicillamine	

Although the symptoms of zinc deficiency are common irrespective of the causative factors, they may vary depending on the severity of zinc deficiency (5). The three main clinical signs are lesions with localization around the body orifices and on the extremities, total alopecia, and diarrhea (1). Skin lesions are eczematous pink scaly plaques and may evolve into vesicular, bullous, pustular, or desquamative lesions. Angular cheilitis is a common early manifestation followed by paronychia (2). Patients with advanced disease will experience growth delay, mental slowing, poor wound healing, anemia, photophobia, hypogeusia, anorexia, delayed puberty, and hypogonadism in boys and men (2). The most serious complication observed in acrodermatitis enteropathica is associated immunodeficiency and related secondary infections. Such infections are commonly caused by *Candida albicans* and gram-positive bacteria (3), but gram-negative infections by *Pseudomonas aeruginosa* (7–6) and *Klebsiella* sp. (3) have also been reported. Initial assessment of a patient's zinc status is determination of plasma or serum zinc concentrations (plasma zinc concentrations are preferred) (2). Blood should be drawn before breakfast and the patient should not receive zinc supplements on the day of the test (2). A second approach to determining body zinc status is to measure the activity or concentration of zinc-dependent enzymes, but there is yet no consensus on which zinc-dependent enzyme is most appropriate to measure (2). Histopathological findings are similar to those in other deficiency diseases and vary with the age of the skin lesion. Early lesions show pale keratinocytes in the uppermost part of the spinous zone and in the granular zone, and are covered by a broad thin band of parakeratosis. Fully developed lesions show pale acanthotic epidermis and beneath the original cornified layer, recognizable by its basket-wave appearance, the very beginning of formation of a scale-crust can be seen (7). Late lesions show clear swollen keratinocytes of the acanthotic epidermis and the lesion is topped by a scale-crust (7). Differential diagnosis includes epidermolysis bullosa, cystic fibrosis, glucagonoma syndrome, widespread candidiasis, pellagra, seborrheic dermatitis, hypovitaminoses, atopic dermatitis, celiac disease, and congenital periorificial and palmoplantar keratoderma (3). Acquired types of zinc deficiency should be treated with 0.5–1 mg/kg/day elemental zinc (2).

The cause of acquired zinc deficiency in our case was prematurity. The patient was checked by pediatricians and no other abnormalities were observed.

Conclusion

When dealing with premature babies presenting with periorificial dermatitis, we always have to keep in mind zinc deficiency as a possible cause because after starting zinc replacement therapy skin lesions completely clear in the next few weeks.

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