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Skin lesions in a 16-month-old toddler with impaired zinc absorption

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ABSTRACT

Acrodermatitis enteropathica is a rare, autosomal recessive condition which results from impaired zinc absorption in the gastrointestinal tract. We report a 16-month-old female patient with a 7-month history of periorificial and acral skin lesions. Diagnosing of Acrodermatitis enteropathica was established on the basis of the patient's history as well as clinical and laboratory findings (a lowered zinc level in the child's serum: 17.2μg/dL (N 70-160 μg/dL)). Rapid clinical improvement was observed right after launching zinc supplementation.

Key words: Acrodermatitis enteropathica, zinc deficiency, skin lesions

INTRODUCTION

Skin lesions may be suggestive of various gastrointestinal disorders, some of them result from impaired absorption of trace elements essential for proper functioning of the bodily organs. Zinc (Zn), the second after iron rudimentary element quantity-wise, is responsible for catalytic, structural and regulatory functions in the body. It is supplied in the diet rich in meat and dairy products [1]. Since it is a co-factor in at least 100 metalloenzymes, forming "zinc fingers", zinc plays a vital role in protein, carbohydrate and vitamin metabolism, growth and development, cell proliferation, healing and tissue repair [2, 3]. Moreover, as Zn is essential for cells mediating innate immunity, the growth and functioning of T and B cells, neutrophils, NK (natural killer) cells and macrophages, its deficiency has a negative effect on the immune system leaving the patient prone to infections [4]. Due to its anti-oxidant properties, zinc is also an anti-inflammatory agent, thus its deficiency will invariably cause inflammation [5].

Zinc deficiency may have a congenital background or it may develop as a consequence of improper diet (vegetarians, malnourishment, eating disorders, parenteral feeding, alcoholism), increased Zn demand (premature infants, pregnancy, lactation, serious major diseases) or decreased absorption from the gastrointestinal tract (chronic inflammatory intestinal diseases, diarrhoea, coeliac disease, short bowel syndrome) [3]. Hereditary zinc deficiency is extremely rare and its incidence is connected with major systemic disorders where more often than not skin lesions are the heralds of their development [1, 3]. In the light of all this, we find it interesting to present and discuss the case of a 16-month-old toddler with skin lesions indicative of Acrodrmatitis enteropathica.

CASE PRESENTATION

In April 2010, a 16-month-old female patient was referred to the Outpatients' Clinic at the Department of Dermatology, Venereology and Paediatric Dermatology, the Medical University of Lublin because of a 7-month history of periorificial and acral skin lesions.

The full-term born girl weighed 3.250 g, was 54 cm long and her Apgar scores were 9 at the 1st minute and 10 at the 5th minute. On delivery nothing abnormal was detected. Prenatal and natal histories were normal. The mother denied infections during pregnancy. She had had no miscarriages and given

Figure 1. **Erythematous and erosive plaques partially covered in yellow crusts in the perioral region.**

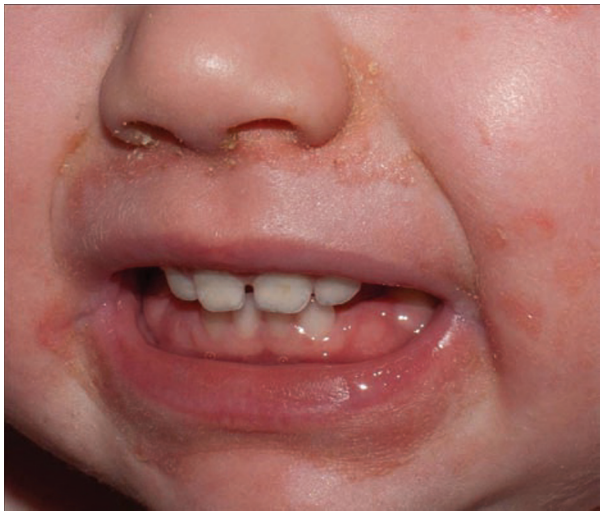
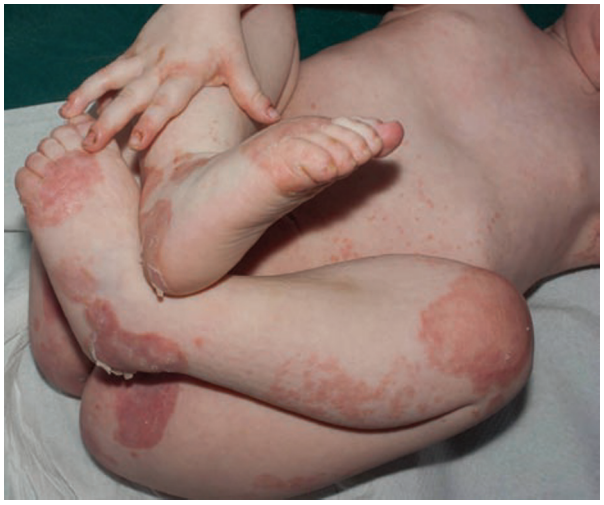


Figure 2. **Erythematous and erosive plaques with scales in their borders located in diaper area and extremities.**



birth to one healthy male child. There was no consanguinity between the parents nor was there any known family history of zinc deficiency. Before the skin lesions characteristic of AE first appeared in the baby girl, she had been regularly seen by her paediatrician who had observed no abnormalities in her psycho-physical development and assessed her state normal for this age group. No reasons for acquired zinc deficiency were also found.

Up to 5 months the child was only breast fed and then, from 5-8 months it was gradually switched to bottle feeding. Skin lesions appeared in the 9th month of the infant's life, about 2 weeks after breast feeding had been stopped altogether, and they were mainly located in the perioral, periorbital, perianal regions, diaper area and spreading to the extremities. The first lesions to have occurred were found in the angles of the mouth and they assumed the form of perleche.

Large, well-demarcated, erythematous and erosive plaques with vesicles and scales in their borders were observed on skin examination. The lesions were also partially exudative and covered in yellow crusts (impetiginisation). They were distributed on the face, diaper area and extremities (*Fig. 1*, *Fig. 2*).

A scaly papular erythematous rash was observed on the trunk. The patient had also thin, brittle, soft, distrophical hair with a tendency for alopecia. In the distal parts of the fingers paronychia with following nail plate changes, such as longitudinal rows, and dystrophical changes was seen. On physical examination the child's weight and height were 10.5kg and 80 cm, respectively (about 50th percentile). She had no diarrhoea nor other systemic disorders. The patient had been previously treated with topical medications and was unresponsive to corticosteroids, antibiotics and emollient preparations.

Diagnostic tests carried out by the referring doctor, i.e. blood and urine tests, liver enzymes, glucose and protein levels, were within normal limits, whereas the laboratory tests conducted by the Outpatients' Clinic at the Department of Dermatology, Venereology and Paediatric Dermatology, the Medical University of Lublin, revealed a decreased zinc level in the child's serum: 17.2μg/dL (N 70-160 μg/dL). The serum alkaline phosphatase level, however was within normal limits: 102.0IU/L (N 96-360 IU/L).

After the AE diagnosis , the child was initially given 3mg/kg/day of oral zinc sulfate and since then the dose has been continuously adjusted depending on the patient's currant condition and Zn demand. Rapid clinical improvement was seen in a week's time, the skin lesions healed completely and hair growth was resumed within one month. The patient is still being followed-up and regularly seen at 1-month or 2-month intervals.

DISCUSSION

When all acquired causes of impaired zinc absorption have been ruled out, it appears quite plausible to diagnose Acrodermatitis enteropathica (AE) (Danbolt disease) especially in those babies who have just been switched from breast- to bottle- feeding. AE is a rare autosomal recessive form of zinc deficiency which affects 1 in 500,000 babies [6] and results from zinc uptake impairment in the gastrointestinal tract (duodenum and jejunum) [7]. It is caused by a defect in the production, structure or function of the specific zinc transporter families, i.e. ZnT and Zip (zinc/iron-regulated transporter-like protein), encoded by SLC30 and SLC 39, respectively. Both ZnT and Zip transporter families are known to exhibit tissue specific expression and have differential responsiveness to dietary zinc deficiency or excess, controlling cellular Zn homeostasis [3, 8]. It has been proven that a mutation in the human SLC39A4 gene, which encodes Zip4 zinc transporter, is responsible