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Transient symptomatic zinc deficiency in a preterm exclusively breast-fed infant

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Abstract

A 5-month-old female infant, preterm, exclusively breast-fed, presented with a 2-month history of erythematous, erosive, and crusted patches and plaques in a peri-oral, scalp, genital, and peri-anal distribution. A clinical diagnosis of zinc deficiency was confirmed by a low serum zinc level in the infant and decreased maternal breast milk zinc. Complete resolution occurred within two weeks of oral zinc supplementation. Acquired zinc deficiency is a rare nutritional disorder of infants. Early diagnosis and adequate treatment will prevent associated morbidity and complications.

Keywords: Human milk, prematurity, symptomatic zinc deficiency

Introduction

Transient symptomatic zinc deficiency (TSZD) is a rare, self-limited disease mainly seen in breast-fed infants [1]. Its diagnosis is frequently delayed.

Case synopsis

A 5-month-old-female infant, born at the 34th week of gestation, with low birth-weight, presented with a 2-month history of an erythematous rash on the face with a peri-orificial distribution including nasal, oral, scalp, genital, and perianal areas. She had been exclusively breast-fed. She was not irritable and had no diarrhea. There were no similar family cases. On physical examination, multiple, symmetrical, sharply demarcated, erythematous, erosive and exudative patches and crusted plaques were found (Figures 1,2,3,4).



Figure 1. Erythematous, erosive patches and crusted plaques on the face

Figure 2. Erythematous, erosive patches and crusted plaques on the face



Figure 3. Erythematous, erosive patches and crusted plaques on the scalp

Figure 4. Erythematous, erosive patches and crusted plaques on the peri-anal and genital areas

The clinical diagnosis of zinc deficiency was confirmed by a low serum zinc level in the infant (0.35 mg/L, normal: 0.7-1.20 mg/L). The mother had normal serum zinc level (1.40 mg/L, normal: 0.7-1.50 mg/L), but maternal breast milk zinc was decreased (0.02, normal: 0.17-3.02 mg/L). The remaining laboratory investigations were normal, including alkaline phosphatase.

Treatment with zinc sulphate was started, at a dosage of 3 mg/kg daily, and a dramatic improvement was seen after two weeks (Figure 5).



Figure 5. Clinical improvement within two weeks of oral zinc treatment

Zinc supplementation was maintained for two months as food diversification was started. After withdrawal of zinc she had no recurrence of lesions and remained with normal serum zinc levels.



Figure 6. Clinical improvement sustained four months after weaning from breastfeeding and withdrawal of supplementation

Discussion

The diagnosis of TSZD was established on the basis of several features. The infant was exclusively breast-fed, premature, and without family history of zinc deficiency. The infant exhibited typical skin lesions, decreased serum zinc level in the infant and maternal milk zinc, and fast clinical improvement after zinc supplementation. There was no recurrence after weaning from breastfeeding and withdrawal of oral zinc. This acquired deficiency differs from acrodermatitis enteropathica, an autosomal recessive disorder of enteric zinc absorption.

As seen in our patient, prematurity and a low zinc level in maternal milk are important risk factors. A negative zinc balance with inadequate stores in premature infants results from increased urinary and intestinal zinc secretion and fast growth. In addition, most of the mother-fetus zinc transfer occurs in the last ten weeks of gestation [2,3]. A low zinc level in maternal milk is probably the result of a rare deficiency or malfunction of a zinc binding ligand, decreasing the uptake from plasma by the mammary gland [2,3].

Recently, Corbo and Lam [4] proposed a revised classification based on the etiology of the zinc deficiency that may facilitate its diagnosis and management in children: type I for inadequate intake, type II for excessive losses, type III for malabsorption, type IV for increased demand, and type V for other possible factors.

In our patient, the combination of many factors, mostly type I (inadequate intake from low breast milk levels), type III, and IV (increased losses and demands in preterm infants, respectively), may explain this transitory deficiency with clinical manifestations of the integumentary system. These symptoms are related to the anti-inflammatory and antioxidant properties of this mineral. Complete and sustained clinical and laboratory normalization is observed after oral zinc supplementation.

Conclusion

Early recognition of this rare disorder and introduction of zinc supplementation rapidly reverses TSZD, avoiding inadequate treatments when misdiagnosed as seborrheic dermatitis or impetigo.

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