Acquired Zinc Deficiency in Two Breast-fed Mature Infants

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Zinc deficiency was diagnosed in a breast-fed mature infant and her sister. In both infants the characteristic dermatitis appeared on the face and buttocks around 10 weeks of age. It responded rapidly to zinc supplements. Their mother's serum zinc level was slightly low but her milk was found to be remarkably low in zinc. Oral zinc supplementation could correct only her serum zinc level but not her low breast milk zinc level. Therefore the mother's deficiency in the transfer process of zinc from serum to breast milk was suspected as a cause of the skin changes in her children. These cases indicate that even mature infants, who feed exclusively on mother's milk, run a risk to develop zinc deficiency, if the concentration of zinc in the breast milk is very low. *Key words: Acrodermatitis enteropathica; Breast milk; Zinc; Nutrition.* (Received January 27, 1986.)

It is known that zinc (Zn) deficiency in a breast-fed infant is very rare: thus, breast milk has been used to treat acrodermatitis enteropathica. To our knowledge, all of the reported cases of acquired Zn deficiency in breast-fed infants have affected premature infants (1–6).

We describe herein two siblings in whom acquired Zn deficiency developed due to breast-feeding, although they were full-term delivered and well-nourished babies. Their mother's serum Zn level was only slightly low but her breast milk showed a very low Zn content. Oral supplementation of Zn for the mother could correct her serum Zn level alone but was of no avail to her breast milk. We therefore think that the main cause of the Zn deficiency in these two siblings is present in the deficient process of Zn transfer from blood to milk in their mother.

CASE REPORT

Case 1

A Japanese girl was born spontaneously after full-term delivery on March 22, 1983, with a birth weight of 3 090 g. Her mother had been treated with ferrous sulfate for her iron deficiency anemia during pregnancy from the 5th to the 8th month of gestation. The baby was fed by mother's milk only.

At 7 weeks of age, she developed recalcitrant diaper dermatitis. Despite treatment by a dermatologist, the exanthema gradually deteriorated and spread to involve the periorbital regions, neck and fingers. She was first seen by us at the age of 10 weeks, when her weight was 6 kg.

On her admission to our hospital dermatitic lesions were present in the above-mentioned areas. The affected skin was red with crusts and with some moist areas.

Topical therapy performed under the diagnosis of contact dermatitis was ineffective. Patch tests with all the used topical agents including steroid, antifungal and antibiotic ointments were negative.

Laboratory studies demonstrated normal serum hepatic enzymes, electrolytes, and peripheral blood cells. Though she had been well nourished by mother's milk only, we examined her serum Zn level as well as her mother's milk Zn level. The baby's Zn level was 11 μg/dl (65–110 μg/dl). While her mother's serum Zn level was 52 μg/dl, Zn level in her milk proved to be 18.5 μg/dl (80–260 μg/dl). For that reason the patient was treated with supplementary Zn ($ZnSo_4$–$7H_2O$ 30 mg/day) for 2 weeks. The clinical response was dramatic and the skin cleared within 3 days. Since her serum Zn level became 94 μg/dl, she was discharged from the hospital under continuous Zn supplementation for the following 14 weeks. At the age of 6 months, with the introduction of solid foods, Zn supplementation was stopped. At two and a half years of age, she is healthy and well nourished with her serum Zn level 91 μg/dl.

Case 2

The child was born after full term delivery on 6th May, 1985, as the younger sister of case 1. Her birth weight was 3 020 g. She was also nourished with mother's milk only. Since the age of 10 weeks, a skin eruption similar to that of her sister developed on the neck and the diaper area. When her mother brought her to our hospital 1 week after the appearance of the exanthema, there were pale erythema with scales around the eyes and the neck. Her diaper sites were partially erosive and scaled, but the grade of the exanthema was lighter than that of her elder sister.

Her serum Zn level was 40 μg/dl. The Zn level of her mother's milk was found again to be 14.3 μg/dl, whereas her serum Zn levels was 102 μg/dl. The mother was given $ZnSO_4 \cdot 7H_2O$ 5 mg/kg/day for 10 days tentatively but the zinc level of the breast milk was not rectified, remaining at 16 μg/dl. Thus the child was also treated by $ZnSO_4 \cdot 7H_2O$ 40 mg/day. The clinical response of Zn supplement was very good and the lesions regressed within 3 days.

COMMENT

In contrast to the previously reported cases of Zn deficiency due to breast-feeding, which were all noted in premature infants with gestation periods ranging from 27 to 32 weeks (1–6), it is to be noticed that our two cases occurred in babies, born after full-term delivery. Thus the zinc deficiency can be induced in a full-term baby by inadequate dietary supplies even in the abscence of prematurity.

The onset of the skin lesions in the reported cases ranged from 8 to 10 weeks as was also the case in our patients. These periods are consistent with the periods when babies require a large amount of Zn for the rapid growth.

All of the mother's breast milk investigated in this study contained only a very low amount of Zn, which could not be corrected by oral Zn supplements of 5 mg/kg/day of $ZnSO_4 \cdot 7H_2O$. The symptoms of acrodermatitis decreased rapidly when the infants were directly given Zn orally. They did not require further Zn supplementation, when solid foods were added to their diet; both children showed normal development afterwards.

Both findings, i.e. that the Zn deficiency in our two patients, which developed during the period of breast-feeding, was successfully treated with Zn supplement and that the skin changes did not relapse after they had started weaning diet, suggest that an abnormality might exist in their mother. She showed low Zn concentrations in the breast milk consistently despite her apparently normal nutritional status and slightly decreased serum Zn level. In addition, the low Zn level of the breast milk could not be corrected by the administration of a relatively large dose of Zn supplements that could achieve elevation of serum Zn concentration. It is likely, therefore, that there is a defect in the process of transfer of Zn from serum into milk which is a main cause of the skin changes in the cases reported here. Zimmerman (1) postulated that such a disorder is inherited rather than acquired and that it does not result from an abnormality in the mammary secretory cell itself but from a deficiency or abnormality of a Zn ligand that is involved in its transfer from the serum to the milk.

ADDENDUM IN PROOF

Since submission of this article for publication, Bye AME et al. reported "Transient zinc deficiency in a full-term breast-fed infant of normal birth weight" in Pediatric Dermatology 2: 308–311, 1985.

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Pyrogallol in the Tumour Stage of Mycosis fungoides: A Case Report

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van de Kerkhof PCM. Pyrogallol in the tumour stage of mycosis fungoides: A case report. Acta Derm Venereol (Stockh) 1986; 66: 361–363.

The case history of a patient with mycosis fungoides (tumour stage) is reported. As ultimum refugium pyrogallol 5% in petrolatum proved to be remarkably effective. (Received January 30, 1986.)

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CASE REPORT

The patient was a 60-year-old woman, who at the age of 53 experienced erythemato-squamous skin lesions on the face, neck, presternal region and lower arm. After 4 years the clinical and histological picture had developed to a classical mycosis fungoides. One year later tumours were seen on her face. Physical examination, laboratory investigations, X-ray of thorax, CT scan of the abdomen, sternum aspirate and crista biopsy did not reveal any sign of systemic involvement.

Topical application of nitrogen mustard had been partially effective. However, contact sensitization complicated this therapy. PUVA and PUVA + Etretinate had no effect at all. Radiotherapy (electron beam and orthovoltage X-ray) resulted in remissions of short duration (a few weeks). Cumulative dosages on the forehead had reached 5400 Rad.

Therefore we tried an alternative therapy; pyrogallol 5% in petrolatum was applied daily on the tumours. The clinically uninvolved skin surrounding the lesions was protected by zinc paste. The effectivity of this treatment is shown in Fig. 1. The tumours resolved via crust formation. After 2 months of therapy only a residual erythema and some hyperpigmentation remained. Therapy was discontinued and up to the time of writing this communication (5 months of observation) the treated areas remained clinically clear.

DISCUSSION

Pyrogallol has been used with success in the treatment of psoriasis (1). As far as we know this is the first report of a beneficial effect of this substance in mycosis fungoides. In this respect it is of interest that many other therapies are effective in both mycosis fungoides and psoriasis. Topical corticosteroids, tar, PUVA, radiotherapy and nitrogen mustard are well known therapies for both diseases. Methotrexate and retinoids, classical therapies for psoriasis, are also effective in mycosis fungoides (2–4).

The working mechanism of pyrogallol is not clear. It is a potent reducing agent. An inhibitory effect of pyrogallol on catechol-O-methyltransferase has been described (5). Theoretically this biochemical effect could account for the effectivity in psoriasis and mycosis fungoides, both diseases being characterized by a hyperproliferative cell system (epidermal hyperproliferation and T-cell proliferation respectively). Hyperproliferative