Zinc deficiency in a preterm neonate with necrotizing enterocolitis

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A case is reported of a preterm neonate with necrotizing enterocolitis, who developed zinc deficiency while receiving zinc-supplemented parenteral nutrition.

Case report

KS was born at 28 weeks' gestation, the sole survivor of twins. She developed hyaline membrane disease and required positive pressure ventilation for 4 weeks. Nasojejunal feeding with expressed breast milk was given from the second day of life. At 8 weeks she developed necrotizing enterocolitis; total parenteral nutrition and intravenous antibiotics were commenced. A standard protocol of amino acid solution (Vamin), dextrose and intravenous lipids (Intralipid), with vitamin and trace element supplements (Solivito and Pedel, KabiVitrum) was used. The mineral supplement contained 0.6 μmol/kg/day of zinc.

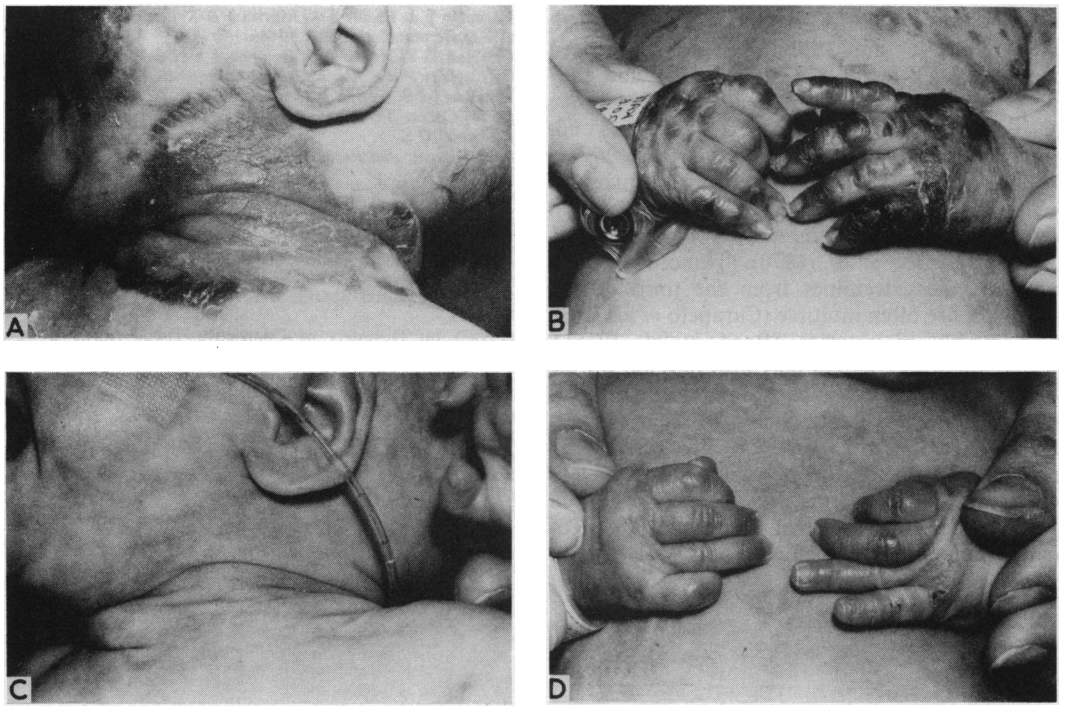

Figure 1. Acquired zinc deficiency. A: desquamation of skin around the neck. B: the hands are severely affected with areas of necrosis. C and D: One week after starting zinc therapy, showing dramatic improvement

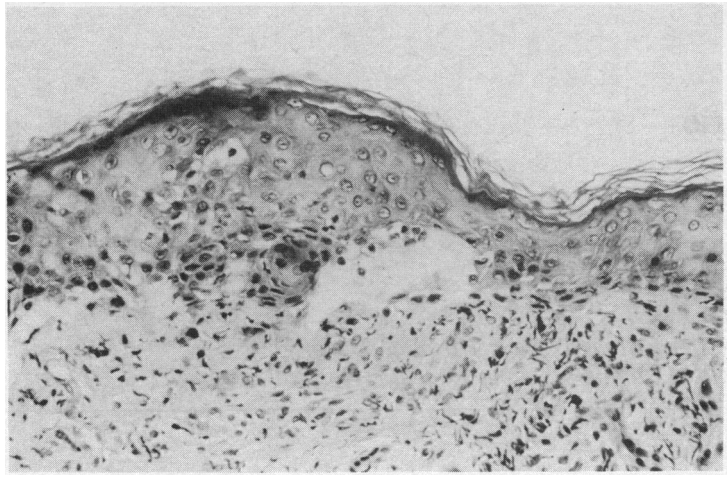

Figure 2. Histopathology of the skin: intraepidermal and subepidermal vesicles with an upper dermal mixed inflammatory infiltrate

One week after starting parenteral nutrition she developed an extensive vesiculobullous eruption of the scalp, face, especially around the mouth, neck (Figure 1A), hands (Figure 1B), feet, knees and elbows, which worsened over the next 3 days. The clinical diagnosis of zinc deficiency was confirmed by the finding of a plasma zinc concentration of 2.5 μmol/l (normal range 9–29 μmol/l).

A skin biopsy from the right knee showed epidermal necrosis, spongiosis and acantholytic intraepidermal vesicles; also subepidermal vesicles and an upper dermal mixed inflammatory infiltrate (Figure 2).

Oral zinc sulphate solution (30 mg per day) was commenced and feeding with mother's expressed breast milk was re-established. The skin recovery was dramatic, with healing evident by 48 hours and a near normal skin appearance one week later (Figure 1C,D), by which time the plasma zinc had increased to 10.5 μmol/l. At 14 weeks of age, zinc supplementation was discontinued with no further recurrence of skin lesions. Breast-milk feeding was continued until 17 weeks of age, with no clinical evidence of nutritional or trace element deficiency on subsequent follow up.

Discussion

Skin changes associated with zinc deficiency are well recognized in the hereditary condition of acrodermatitis enteropathica. Acquired zinc deficiency has been described in a breast-fed preterm infant (Aggett *et al.* 1980) and when total parenteral nutrition is administered without zinc supplementation. Acquired zinc deficiency can result from reduced zinc absorption or increased losses due to gastrointestinal pathology or surgery (Arlette 1983). The zinc deficiency seen in our patient presumably occurred as a result of trace element loss from the inflamed gut mucosa at a time of increased requirement. As 60% of the total body zinc at birth is transferred to the fetus during the last trimester, the preterm neonate is more liable to develop zinc deficiency than is the term neonate.

Recommendations for zinc supplementation of total parenteral nutrition are based on the requirements of full-term babies and vary by an order of magnitude from 0.6–7.5 μmol/kg/day; our patient received the lower limit of 0.6 μmol/kg/day. Most studies rely on plasma zinc concentrations as a measure of deficiency and these may be an inadequate reflection of body zinc status.

We believe that it is important to recognize that zinc deficiency may be a complication of necrotizing enterocolitis and that current recommended total parenteral nutrition regimens would seem to provide inadequate zinc supplementation for the ill, preterm neonate.

References

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