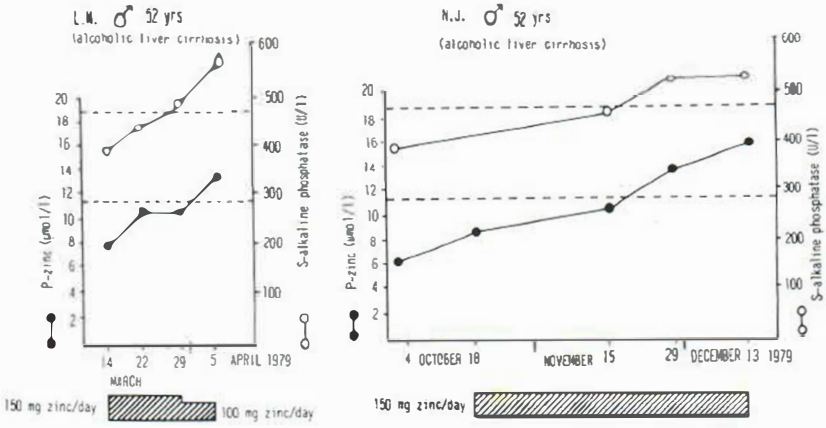

Fig. 4. Plasma zinc and serum alkaline phosphatase levels in Case 1 (L. M.) and Case 2 (N. J.) during oral zinc therapy. Serum alkaline phosphatase, normal range 50–250 U/l. Plasma zinc, normal range (males) 11.4–18.9 μmol/l indicated by dotted lines.

phosphatase level and cause a decrease in the serum bilirubin concentration (6), as also demonstrated here. It is conceivable that this effect of zinc is somehow related to restoration of pre-existing low zinc levels in the cirrhotic liver, which was first demonstrated by Vallee et al. (4).

It remains speculative whether the bumetanid therapy in Case 2 was involved in the onset of the zinc deficiency symptoms. Wester (9) has shown that diuretics (furosemide, chlorthalidone and thiazides) which cause sustained hyperzincuria with maintained or increased serum zinc levels, reduce the zinc content of normal livers. Were a depletion of zinc also to take place in patients with liver cirrhosis receiving diuretic treatment, it might explain the sequence of symptoms observed in Case 2.

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Acquired Dermatitis Due to Zinc Deficiency in a Premature Infant

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Abstract. A case of dermatitis with lesions reminiscent of enteropathic acrodermatitis of the premature of low gestational age is presented. The condition responded dramatically to oral zinc sulphate therapy.

Key words: Zinc; Acquired dermatitis; Premature infant

While the importance of zinc in the nutrition of the premature infant and the occurrence of skin lesions in the zinc deficiency syndrome are well known, the pertinent literature so far seems to have brought no reports of enteropathic dermatitis-like lesions in the bottle-fed premature infant.

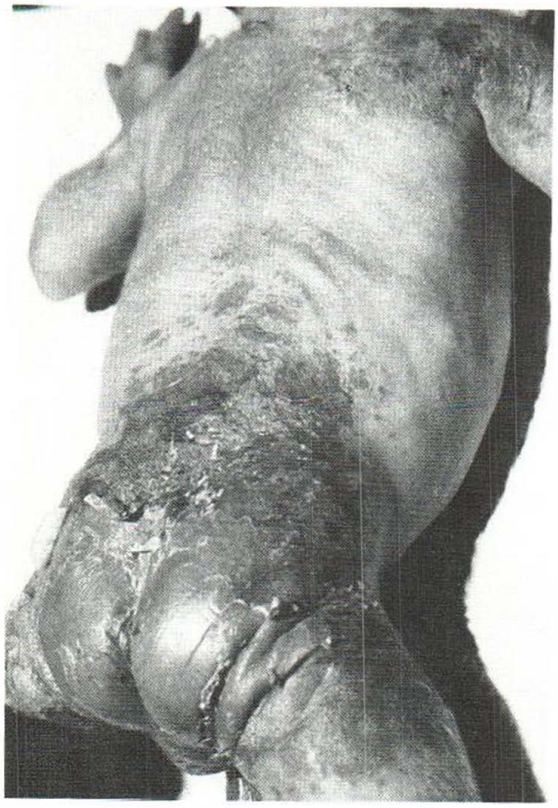

Fig. 1. Case 1. Burn-like skin lesions in the nappy area.

CASE REPORT

V.G. was born after 29 weeks' gestation, weighing 1280 g and was bottle-fed with zinc-enriched milk containing no added vitamins. At the age of 55 days he developed burn-like skin lesions in the nappy area. Routine tests demonstrated iron deficiency anaemia but the results of bacteriological and mycological examinations of the skin were negative. Systemic treatment with multivitamin and iron preparations was instituted, with local hypochlorite dressings. After 10 days the lesions spread into the buttocks and on to the thighs towards the knees. They presented well defined borders (Fig.1), as seen in burns. Similar lesions developed on the extensor aspects of the elbows and knees, at the corners of the mouth and eyes and, later on the dorsal aspects of fingers and toes. There was no alopecia and the stools were normal, but the baby was extremely fractious.

The spectrofluorometrically determined plasma zinc was 80 μg/100 ml (normal value 108±15 μg/100 ml). Zinc sulphate supplementation 15 mg three times a day was prescribed, with dramatic improvement; the lesions disappeared after 2 days.

Subsequently, 5 similar cases were observed (3 males, 2 females, with gestational ages below 32 weeks). All fed with the same milk and all presented a very similar clinical picture. In all these cases the lesions developed between the 45th and 60th day of life, were never associated with either alopecia or diarrhoea, failed to respond to routine treatments, but responded dramatically to oral administration of zinc. The plasma levels of zinc ranged from 80 to 95 μg/100 ml and, with treatment, increased to normal values. The treatment was continued for 10 days after complete clinical recovery. No relapses were observed during the 1 to 6 months follow-up period.

The 7th case was that of G.S., aged 2 months, who presented similar lesions and was not treated with zinc. The condition showed progressive deterioration over a period of 20 days, followed by very slow resolution.

DISCUSSION

Moynahan (1) was the first to demonstrate that the skin lesions of enteropathic acrodermatitis are related to a congenital error of zinc metabolism. Subsequently, similar skin lesions were described in several zinc-deficient patients with malabsorption, especially in connection with periods of exclusively parenteral nutrition (2).

The importance of zinc in the nutrition of infants, especially the premature infant, was recently demonstrated (3). In fact, zinc stores of premature infants are far lower than those of full-term babies. Babies on zinc-enriched diets gain weight quicker than controls. These studies led to an increase in the zinc content of bottle feeds, but whether the amounts of zinc currently provided in such feeds meet with the requirements of premature babies remains uncertain.

The development of eneteropathic acrodermatitis-like lesions of the skin in premature children and their dramatic response to treatment with zinc have thus found a full theoretical explanation.

The absence of gastroenteritis and of alopecia in the cases described could be attributed to the fact that the zinc plasma levels were not as low as those found in enteropathic acrodermatitis.

The timing of the onset of lesions, the prompt return to normal plasma zinc values in spite of cessation of the treatment and the spontaneous recovery observed in one of our cases suggest that in the bottle-fed premature infant, zinc deficiency reaches critical levels towards the end of the second month of life and is followed by spontaneous improvement.

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Fish Tank Granuloma

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Abstract. A case of fish tank granuloma is presented. A lesional biopsy sample yielded growth of *Mycobacterium marinum* on Löwenstein's medium. Treatment with co-trimoxazole was successful.

Key words: Fish tank granuloma; *Mycobacterium marinum;* Co-trimoxazole

Infection with atypical mycobacteria has been known for almost a century in cold-blooded vertebrates (see 1). Human infection was first described in 1954 when Linell and Nordén (8) reported 80 cases of cutaneous granulomas among swimming pool bathers in Sweden. Human infection acquired from tropical fish tanks was first observed by Swift and Cohen (10). This paper describes a localized infection caused by *Mycobacterium marinum* (formerly called *M. balnei*), presumably contracted from a tropical fish aquarium.

CASE REPORT

A 12-year-old Norwegian boy was admitted to the outpatient clinic, Department of Dermatology, with a 7-month history of two tender swellings on the dorsum of the right middle finger. The swellings had been intermittently discharging in spite of two 7–10 day courses of phenoxymethylpenicillin. Local treatment with clotrimazole cream (1%) for several weeks had also proved unsuccessful. The boy had been sporadically visiting a public swimming pool. He had also kept a tropical fish aquarium which he regularly cleaned without using gloves. Some of the fish had recently died. He had therefore disposed of the aquarium. Unfortunately this could not be traced.

Over the dorsum of the proximal interphalangeal and the metacarpophalangeal joint of the right middle finger he presented two isolated, elevated, slightly crusted, granulomatous lesions (Fig. 1) There was no lymphadenopathy. Physical examination otherwise revealed unremarkable findings.

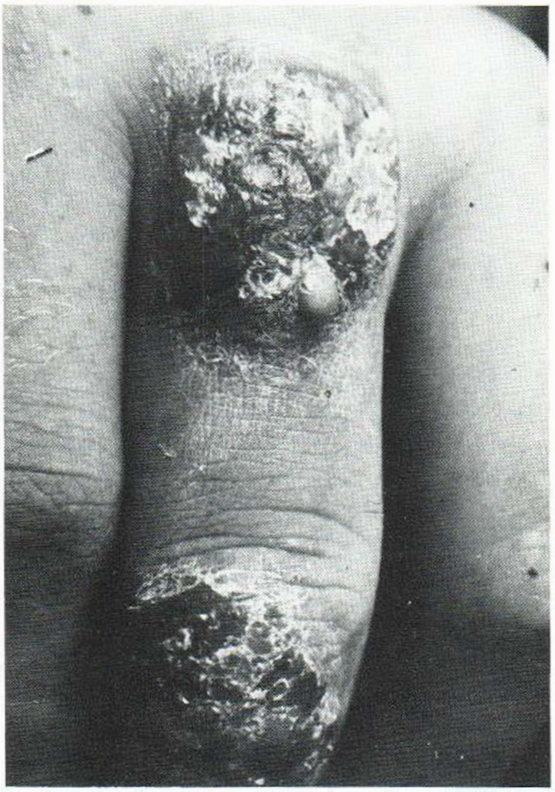

Fig. 1. Isolated granulomatous lesions caused by *M. marinum*, before treatment.

A lesional biopsy yielded growth of an acid-fast organism on Löwenstein's medium. The organism was identified as *M. marinum* by cultural characteristics and by animal inoculation. The strain was not pathogenic to guinea pigs, but produced swellings and blisters on mouse tails 4–5 days after intravenous injection as described by Fenner (5). The strain was sensitive to all the conventional antituberculous agents.

In line with recent reports (2, 3, 6), treatment was initiated with trimethoprim-sulfamethoxazole (co-trimoxazole) 2 tablets twice daily, each tablet containing 80 mg trimethoprim and 400 mg sulfamethoxazole. After 6 weeks a complete remission was achieved, leaving only slight residual scarring (Fig. 2). During a follow-up period of 10 months, there has been no sign of recurrence.

DISCUSSION

Recent reports indicate that diseased tropical fish might be the most common source of human *M.*