

steroids has played a large part in the development of Norwegian scabies in this case. There was no evidence of any other known contributory factor. In this context it is particularly notable that this patient always suffered intractable itching in the presence of the disease. In the other cases mentioned above, itching was absent or minimal, and the failure to remove acari by scratching is thought to be a major factor in the development of Norwegian scabies (6).

Thus, this case represents yet another side effect of topical fluorinated steroids and demonstrates how the abuse of one of these preparations has changed the character, extent and natural history of an eruption. It has also helped to show that, in the absence of any other known contributory factor, Norwegian scabies will develop when the interaction between skin and *Sarcoptes scabiei* is altered by topical steroids.

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REFERENCES

1. Burgess, I.: Unusual features of scabies associated with topical fluorinated steroids. *Br J Dermatol* 88: 519, 1973.
2. Clayton, R. & Farrow, S.: Norwegian scabies following topical steroid therapy. *Postgrad Med J* 51: 657, 1975.
3. Espy, P. D. & Jolly, H. W.: Norwegian scabies. *Arch Dermatol* 112: 193, 1976.
4. Logan, J. C. P., Grant, P. W. & Keczkcs, K.: Norwegian scabies and lymphatic leukaemia. *Br J Dermatol* 79: 303, 1967.
5. Macmillan, A. L.: Unusual features of scabies associated with topical fluorinated steroids. *Br J Dermatol* 87: 496, 1972.
6. Mellanby, K.: *In Scabies*. E. W. Classey Ltd., Middlesex, England, 1972.
7. Paterson, W. D., Allen, B. R. & Beveridge, G. W.: Norwegian scabies during immunosuppressive therapy. *Br Med J* 4: 211, 1973.
8. Snell, E. S.: The pharmacological properties of corticosteroids in relation to clinical efficiency. *Br J Dermatol* 94: Suppl. 12: 15, 1976.
9. Stoughton, R. C. D. & August, P. J.: Cushing's syndrome and pituitary-adrenal suppression due to clobetasol propionate. *Br Med J* 11: 419, 1975.

Lines of Beau: Possible Markers of Zinc Deficiency

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Key words: Acrodermatitis enteropathica; Beau's lines; Leukocyte endogenous mediator; Zinc depletion Syndrome; Zinc deficiency

In 1846 Beau described transverse lines and depressions on nails following severe acute diseases (1). In the following communication, evidence is presented that alterations in zinc metabolism might be an etiological factor involved in this anomaly.

Case 1. A 10½-year-old boy is the subject of an earlier report on acrodermatitis enteropathica (AE) (8). Treatment with 8-hydroxyquinoline was discontinued when an ophthalmological examination revealed damage of the visual function. Before zinc therapy was started, the serum zinc level had fallen from 9.9 to 5.4 $\mu\text{mol/l}$ (normal range in men: 11.4–18.9), and he developed a bullous dermatitis on his hands, feet and knees. Oral zinc therapy normalized the serum zinc level in 2 weeks, cleared the skin lesions and activated the growth of hair and nails. About 4 weeks after zinc therapy was started, marked lines of Beau appeared on the finger nails (Fig. 1).

Case 2. A 16-year-old female had suffered from AE, which had been completely controlled with 8-hydroxyquinolines. When this treatment was discontinued,

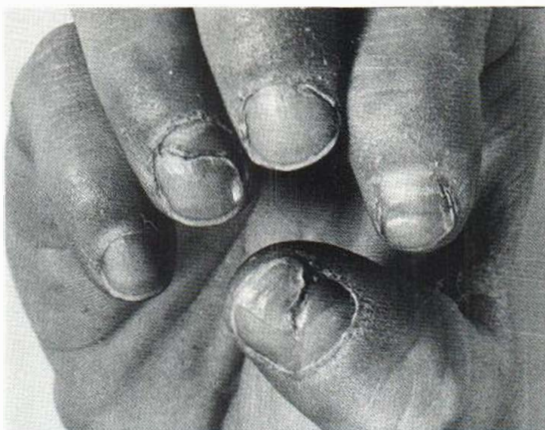


Fig. 1. Acrodermatitis enteropathica with marked lines of Beau on fingernails, photographed 10 weeks after initiation of oral zinc therapy.

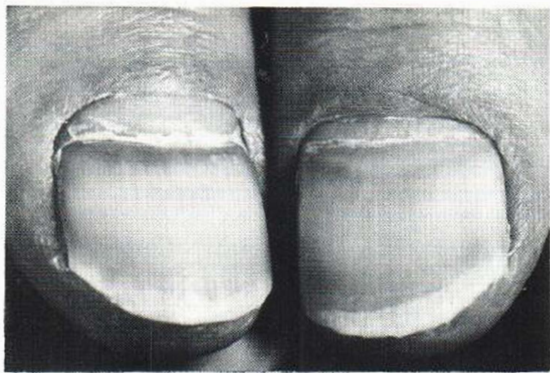


Fig. 2. Zinc depletion syndrome. Lines of Beau on thumb nails 5 weeks after i.v. zinc therapy was started. Note faster growth of right nail plate.

tinued and replaced by zinc sulfate therapy, the serum zinc level was low, 4.4 and 4.9 $\mu\text{mol/l}$ (normal range in females 10.6–17.7). The patient's general condition was perfect, and she had no skin symptoms. Four weeks later, transverse depressions were seen proximally on her finger nails. The bands involved the entire width of the nail, but did not show discontinuation of the nail plate. The serum zinc level returned to normal within a month.

Case 3. A 49-year-old female developed a zinc depletion syndrome (ZDS) during total parenteral nutrition. Her case is reported elsewhere (9). The serum zinc level was very low, 3.8 $\mu\text{mol/l}$, when i.v. treatment with zinc sulfate was started. In a few days her general condition and the integrity of skin and mucous membranes was restored. Four weeks later, lines of Beau appeared on her finger nails (Fig. 2). The serum zinc level returned to normal in 2 weeks.

DISCUSSION

These case reports, and one additional case of ZDS reported by Kay et al. (4), show that severe zinc deficiency, or recovery from a state of deficiency, might cause lines of Beau.

Experiments in rats have shown that polymorphonuclear leukocytes release a humoral factor, "leukocyte endogenous mediator" (LEM) during infection and after injection of endotoxin of *Escherichia coli* (3, 5). The factor causes a significant fall in the serum zinc and iron levels within a few hours, with a concomitant rise in liver zinc. At the same time, significant amounts of zinc are removed from muscles and skin, to be stored in the liver (7).

Halsted & Smith (2) reported a rapid fall in serum zinc level in volunteers receiving injections of endotoxin. Doses too low to produce fever and leukocytosis did not affect the serum zinc level. Pekarek et al. (6) showed that exposure of vol-

unteers to Venezuelan equine encephalomyelitis virus vaccine caused a rapid fall in serum zinc. The decrease was most pronounced in those who developed febrile illness.

Diseases known to cause lines of Beau include primarily severe acute febrile diseases (1) in which significantly depressed serum zinc levels seem to be characteristic (2). A sudden change in the serum zinc level suggests a redistribution of zinc mediated by LEM and/or an acute stress reaction (2, 7). Thus, zinc deficiency might develop and be responsible for Beau's lines in acute febrile disorders, such as in the general zinc deficiency states of AE and ZDS.

The fact that lines of Beau did not appear until shortly after treatment with zinc was started, suggests that activation of nail growth caused them; or it merely shows that the lines, being formed at the height of the illness, were not pushed forward until cure occurred and nail growth was reactivated.

Though zinc deficiency might cause lines of Beau, it does not necessarily mean that they are always caused by zinc deficiency, nor that zinc deficiency inevitably causes them. The severity of the deficiency, and the mode of development and recovery probably play a role too. Besides, factors other than zinc deficiency interfering with normal nail formation might be present in the patient at the same time.

REFERENCES

1. Beau, J. H. S.: Note sur certains caractères de sémiologie rétrospective présentés par les ongles. *Arch Général Méd* 11: 447, 1846.
2. Halsted, J. A. & Smith, J. C.: Plasma-zinc in health and disease. *Lancet* i: 322, 1970.
3. Kampschmidt, R. F. & Upchurch, H. F.: The effect of endogenous pyrogen on plasma zinc concentration. *Proc Soc Exp Biol Med* 134: 1150, 1970.
4. Kay, R. G., Tasman-Jones, C., Pybus, J., Whiting, R. & Black, H.: A syndrome of acute zinc deficiency during total parenteral alimentation in man. *Ann Surg* 183: 331, 1976.
5. Pekarek, R. S. & Beisel, W. R.: Characterisation of the endogenous mediator(s) of serum zinc and iron depression during infection and other stresses. *Proc Soc Exp Biol Med* 138: 728, 1971.
6. Pekarek, R. S., Burghen, G. A., Bartelloni, P. J., Calia, F. M., Bostian, K. A. & Beisel, W. R.: The effect of live attenuated Venezuelan equine encephalomyelitis virus vaccine on serum iron, zinc, and copper concentrations in man. *J Lab Clin Med* 76: 293, 1970.
7. Pekarek, R. S., Wannemacher, R. W. & Beisel, W. R.: The effect of leukocytic endogenous mediator (LEM) on the tissue distribution of zinc and iron. *Proc Soc Exp Biol Med* 140: 685, 1972.

8. Weismann, K.: Acrodermatitis enteropathica treated with oral zinc sulfate. *Dan Med Bull*, unpubl. data.
9. Weismann, K., Hjorth, N. & Fischer, A.: Zinc depletion syndrome with acrodermatitis during longterm intravenous feeding. *Clin Exp Derm* 1: 237, 1976.

Lymphogranuloma Venereum with Hepatic Involvement

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Key words: Lymphogranuloma venereum; Hepatitis; Doxycycline

Whether or not the liver may be affected in patients suffering from lymphogranuloma venereum (LGV) is a subject of controversy, and very few communications have been published on this topic (1, 2, 3, 5, 7). The purpose of the present paper is to further elucidate this subject.

CASE REPORT

A 36-year-old unmarried Norwegian seaman attended the clinic in September 1975 with a moderately tender enlargement in the right groin.

In 1959 he had chancroid, and since 1962 gonorrhoea 8 times and, in the last 2 months, a steadily growing lump in the right groin. He had sailed in tropical and subtropical waters for several years. Last sexual contact was in Thailand 2 months prior to admittance.

The patient presented an enlarged lymph node in the right groin, measuring 5 cm in diameter, moderately tender, fluctuating and adherent to the skin and underlying tissues. A small crust was seen on the shaft of the penis. There was no fever. The findings of the general physical examination were otherwise normal.

Laboratory investigations. Hb 14.1 g/100 ml, white blood count 15800, with a moderate shift to the left, E.S.R. 98 mm/hour, serum proteins 8.3 g/100 ml (albumin 3.9 g/100 ml, globulin 4.4 g/100 ml), gamma globulin 1.82 g/100 ml (normal 0.6–1.5 g/100 ml), otherwise normal serum electrophoresis.

Liver investigations. Total bilirubin 0.6 mg/100 ml, alkaline phosphatase 1118 U/l (normal <270 U/l), gamma-glutamyl transpeptidase 174 U/l (normal <50 U/l), SGPT 405 U/l (normal <40 U/l). OCT (4 weeks later) 173 U/l (normal <45 U/l), sulphobromophthalein retention (4 weeks later) 3.0% (normal <5%). Liver scan showed hepatomegaly with no filling defects. Needle biopsy of the liver (5 weeks later). The biopsy (P. 13776/75) specimen showed some capsular, portal and septal fibrosis with slight infiltration of lymphocytes, histiocytes and some eosinophilic granulocytes. The radiary structure of the liver cords was preserved, and the liver cells contained some bile pigment. Focal hyperplasia of Kupffer cells could be seen (E. Blück, The Gade Institute) (Fig. 1).

Serological tests. Immune electrophoresis (4 weeks later): IgA 6.1 mg/100 ml (normal 0.5–3.3 mg/100 ml), IgG 14.5 mg/100 ml (normal 7–18 mg/100 ml), IgM 2.6 mg/100 ml (normal 0.3–2.5 mg/100 ml). The complement fixation test for lymphogranuloma venereum (LGVCF) was positive at a serum dilution 1:512 on admission and showed a fourfold rise in the titre during the observation period, 1:1024 (1 week later) and 1:2048 (16 weeks later). The LGV specific immunofluorescence test was not available.

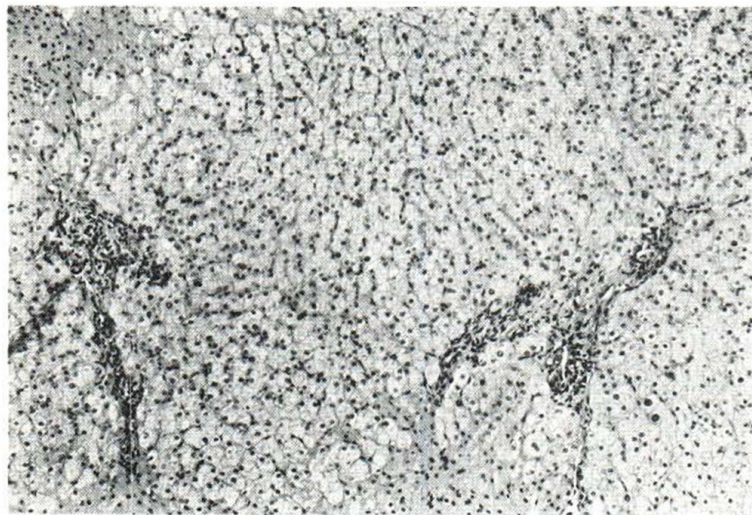


Fig. 1. Periportal and septal cell infiltrates with slight fibrosis. $\times 110$.