Zinc Deficiency in Total Parenteral Nutrition

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SUMMARY
A patient with multiple enterocutaneous fistulae who developed the skin manifestations of acrodermatitis enteropathica while on total parenteral nutrition is described. The value of monitoring the serum alkaline phosphatase to demonstrate zinc deficiency is shown. The skin manifestations responded rapidly to the re-introduction of zinc to the diet.

Zinc is an essential element of more than 70 metalloenzymes (e.g. alcohol dehydrogenase, carboxypeptidase and DNA polymerase). These are involved in almost all aspects of cellular metabolism (metabolism of lipids, proteins, carbohydrates and nucleic acids). Hence, the element is crucial to growth, development and normal function of all living matter. Zinc is widely distributed in foods, and most diets provide the minimal daily requirement (about 15 mg) for human beings. Abnormalities of zinc metabolism have been shown in humans with cirrhosis, with acute myocardial infarction, in hypogonadal dwarfism, and in those with idiopathic loss of taste and smell. Recently, an association between zinc deficiency and acrodermatitis enteropathica has been found.

Congenital acrodermatitis enteropathica is an autosomal recessive disorder characterized by bullous exfoliating lesions, situated peri-orificially and distally on the extremities, alopecia, and chronic diarrhoea. Other features include secondary fungal or bacterial infections, diarrhoea, photophobia, failure to thrive, nail dystrophy, stomatitis and emotional disturbances. Moynahan reported that zinc deficiency in patients with acrodermatitis enteropathica was probably due to diminished intestinal zinc absorption. The administration of zinc corrected the major defect. There have been several reported cases of acquired acrodermatitis enteropathica secondary to severe limitation of zinc intake in patients on total parenteral nutrition.

In this article, we describe a malnourished patient with multiple enterocutaneous fistulae who developed an acute zinc deficiency syndrome while on total parenteral nutrition. The alkaline phosphatase level showed a concomitant precipitous drop.

CASE REPORT
A Black man aged 25 years was admitted to hospital with multiple enterocutaneous fistulae, intestinal malabsorption, hepatic dysfunction, and general deterioration following drainage of a pelvic abscess. Two surgical attempts to repair the fistulae had failed and he was maintained on intravenous alimentation, blood transfusion, nutritional supplements and antibiotics. On admission, he was grossly emaciated with a mass of 32 kg. Sinographic examination showed three enterocutaneous fistulae, communicating with the small bowel, transverse colon and sigmoid colon, which drained 1 litre per day.

Abnormalities in blood chemistry were: creatinine 65 mmol/l (normal 75 - 115 mmol/l), total protein 56 g/l (normal 60 - 80 g/l), albumin 19 g/l (normal 35 - 50 g/l), calcium 1,85 mmol/l (normal 2,10 - 2,6 mmol/l), cholesterol 2,8 mmol/l (normal 3,9 - 7,8 mmol/l), alkaline phosphatase 111 units (normal 30 - 85 units).

The patient was treated conservatively and managed on intravenous alimentation (Travasol and Intralipid) and a precision diet which contributed, among other nutrients, 15 mg of zinc per day.

The patient improved on this regimen and his mass increased to 37 kg. Drainage from the fistulae decreased to 20 ml per day. He developed a chest infection, which responded rapidly to cloxacillin, and renal dysfunction, with a raised urea and creatinine level and a decrease in creatinine clearance, thought to be related to marked hypo-albuminaemia causing hypovolaemia, which was managed by increasing his fluid intake. Egg flips, and later a soft ward diet, were introduced.

During February 1978 there was a recurrence of fistulae with significant drainage, and at this stage, it was decided to discontinue oral intake including the precision diet and to manage the patient on intravenous alimentation and antibiotics, i.e. Travasol, Intralipid, gentamicin and penicillin.

Fig. 1. Facial rash showing acne-like pustules and crusted erythematous lesions.
March 1978 51 Oral zinc

(1977):


DISCUSSION

Zinc trace element deficiency should be suspected where the predisposing factors exist, such as malabsorption, marked proteinuria, severe burns and severe infections, chronic alcoholism, pancreatitis and diabetes, and possibly, myocardial infarction. Zinc deficiency, therefore, is to be expected in the severely ill postoperative or badly injured patient, or the patient with multiple enterocutaneous fistulae, particularly if parenteral feeding is required. The total zinc loss from all sources such as faeces, urine, gastro-intestinal tract fluids, blood, wound exudate and sweat can be as much as 1 mmol per day. Apart from this intestinal loss, it is known that in a catabolic state, large amounts of zinc are lost via the urine. When predisposed patients receive adequate parenteral nutrition in terms of energy and protein, their metabolism becomes anabolic. In this situation, the zinc metallo-enzymes, of which alkaline phosphatase is just one, are needed for the synthesis of new proteins. Large amounts of zinc are therefore required. Kay and Tasman-Jones suggested that the diminished zinc intake, together with the change in the metabolic state, is responsible for the acute occurrence of this deficiency syndrome. Therefore, a decrease of the serum alkaline phosphatase level during total parenteral nutrition may indicate an impending acute zinc deficiency. This is well demonstrated in our patient (Table I). A low serum zinc level is a late sign of zinc deficiency and not an index of marginal deficiency.

Zinc deficiency develops more frequently during long-term parenteral nutrition than has been thought, and routine zinc administration has been advised by Wretland, and Michie et al. Zinc sulphate appears to be the preparation of choice. Where the symptoms of acrodermatitis enteropathica already exist, administration of zinc sulphate either intravenously or orally in doses of 40 - 220 mg daily will rapidly reverse the skin lesions, diarrhoea and mental depression.

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REFERENCES


TABLE I. SERUM ALKALINE PHOSPHATASE LEVELS CHARTED AGAINST THE PATIENT'S CLINICAL COURSE

<table>
<thead>
<tr>
<th>Date</th>
<th>Clinical course</th>
<th>Alk. phos. (U)</th>
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<tr>
<td>28 Dec. 1977</td>
<td>Patient admitted to hospital</td>
<td>111</td>
<td>Precision diet, Travasol and Intralipid</td>
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<tr>
<td>10 Feb. 1978</td>
<td>Fistulae recurred with marked drainage — all oral intake discontinued</td>
<td>149</td>
<td>Precision diet</td>
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<td>20 Feb. 1978</td>
<td>Lung consolidation</td>
<td>120</td>
<td>Total parenteral nutrition (TPN)</td>
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<td>23 Feb. 1978</td>
<td>Facial rash</td>
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<td>TPN</td>
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<td>24 Feb. 1978</td>
<td>Skin lesions more marked</td>
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<td>TPN</td>
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<tr>
<td>26 Feb. 1978</td>
<td>Skin clear</td>
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<td>3 March 1978</td>
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<td>20</td>
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