Leading Article

Cutaneous manifestations of zinc deficiency in children

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In 1936, Swedish dermatologist Brandt described a form of dermatitis in children with disturbances of general condition and absorption of food[1]. Danbolt and Closs, having further investigated this entity, coined the term acrodermatitis enteropathica[2]. In 1973, Barnes and Moynahan linked acrodermatitis enteropathica to zinc deficiency[3]. At present, the term acrodermatitis enteropathica is restricted to the rare autosomal recessive form of defective zinc absorption leading to a characteristic clinical picture.

Zinc metabolism

Zinc belongs to the group of essential trace elements along with iron, iodine, fluorine and others. High concentrations of zinc are present in meat, dairy products, shellfish, legumes, nuts, whole grain and green leafy vegetables. Phytate, in plant foods, interferes with absorption and high fibre content of food also tends to decrease bioavailability of zinc. Thus, foods of animal origin are the best source of zinc as in the case of protein. Protein undernutrition invariably leads to an insufficient supply of zinc. Zinc is absorbed along the whole of small intestine and a binding ligand protein, which enhances absorption, is present in breast milk. Once absorbed, zinc is 80% bound to albumin and 20% to $ß_2$ microglobulin.

Physiological functions of zinc

Zinc is indispensable to the normal function of all human cells, cellular systems, tissues and organs. Zinc metal moiety is essential for functioning of important enzymes called metallo-enzymes. These include alkaline phosphatase, alcohol dehydrogenase, DNA and RNA polymerase. Some of these enzymes are important in maintaining epidermal and epithelial surface intergrity. Zinc also plays an important role in immunological functions.

Zinc deficiency

Zinc deficiency can be primary (undernutrition and inherited absorptive defect) or secondary to diseases of gastrointestinal tract. Undernutrition is linked to poverty and food habits. In certain physiological situations, like pregnancy, there is an increased demand. Similarly, an intercurrent illness, by reducing intake and increasing demand, may precipitate acute zinc deficiency.

Zinc deficiency and skin changes

Zinc deficiency causes characteristic skin lesions and alterations in nail and hair growth. Findings are identical whether cause is primary or secondary. Skin lesions occur mainly at sites with rapid epidermal turnover and areas subjected to wear and tear.

Cutaneous manifestations

Clinically a distinction can often be made of acute, subacute and chronic forms, based on duration and morphology of lesions. However considerable overlap occurs.

Acute zinc deficiency

This often occurs when a child develops an intercurrent illness associated with vomiting. General symptoms include photophobia and irritability. Skin changes develop over a few days and are mainly periorificial with brown red erythematous papules progressing to scaling and crusting of the edges; underlying skin is erythematous and may show fissuring (Figures 1 and 2). The scald like erythema and periorificial distribution can mimic staphylococcal scalded skin syndrome (Figure 3). In the neonate, lesions can rapidly progress to erythroderma. The finger flexural areas show flat grey bullae surrounded by red brown erythema. There may be associated paronychial inflammation. Localized lesions occur in areas subjected to constant rubbing, like side of face (Figure 4), skin in contact with NG tubes, heels in bed ridden patients etc. There is often secondary bacterial and fungal infections.

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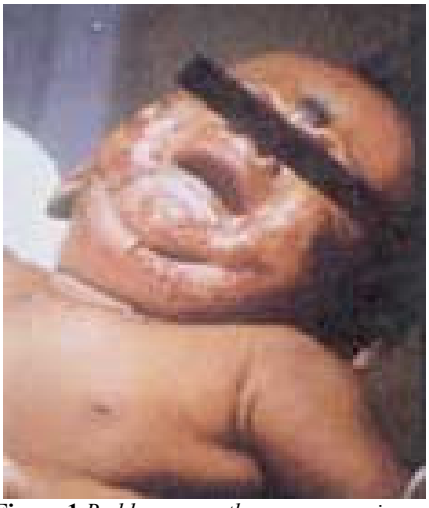


Figure 1 *Red brown erythema progressing to scaling.*

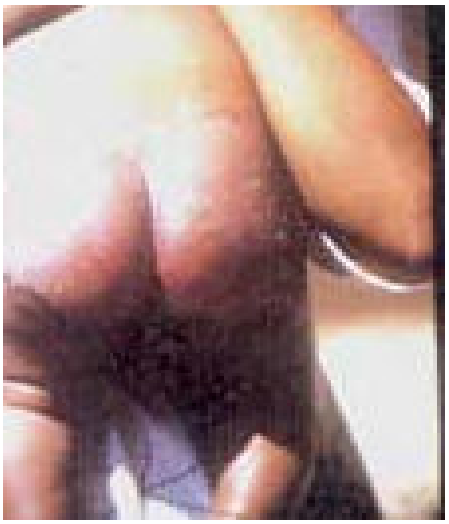


Figure 2 *Red brown scaly edge, underlying erythema & fissuring*

Figure 3 *Scald like erythema & periorificial distribution mimicking staphylococcal scalded skin syndrome.*

Figure 4 *Erythema & scaling in a neonate with continuous NG aspiration nursed in a left semiprone position.*

Subacute zinc deficiency

When lesions have been present for a few weeks, as is often the case in acrodermatitis enteropathica (seen around weaning period), the child has diarrhoea and is listless with extensive skin involvement. The skin of face, back, major folds, trunk and proximal limbs are involved. The characteristic feature is brown black crusts at the advancing edge of lesions (Figure 5). Symmetrical involvement of the areas are seen.

Figure 5 *Extensive involvement of back with characteristic brown black crusts at the advancing edge of lesions*

The centre often shows scaling. There may be excoriations in infected areas but diffuse erythema, seen in acute form, is not seen. Major flexures and genital

area show candidal superinfection. Involvement of back of head is common with patchy alopecia. The author has seen a number of boys with lesions confined to scrotum (Figure 6).

Figure 6 *Localized involvement of scrotum & penile skin.*

Chronic zinc deficiency

The characteristic feature is lack of inflammation. Lesions can be either dry, scaly, psoriasiform in appearance and distribution or lichenified, pigmented and annular (Figure 7). An abundance of lesions are seen on the limbs. Scalp hair growth is poor and sparse. Hairs are brown and sometimes white (Figure 8). Structural changes of hair like breakage, spearhead like appearance, transverse striations, longitudinal splits, pseudomonilithrix and bayonet hairs may occur. Nail changes consist of poor growth, thinning and deep transverse ridges (Beau's lines). These are seen 3 to 4 weeks after starting zinc supplements. White transverse bands also occur.

Figure 7 *Psoriasiform lesion on left knee & pigmented lichenified lesions on right thigh.*

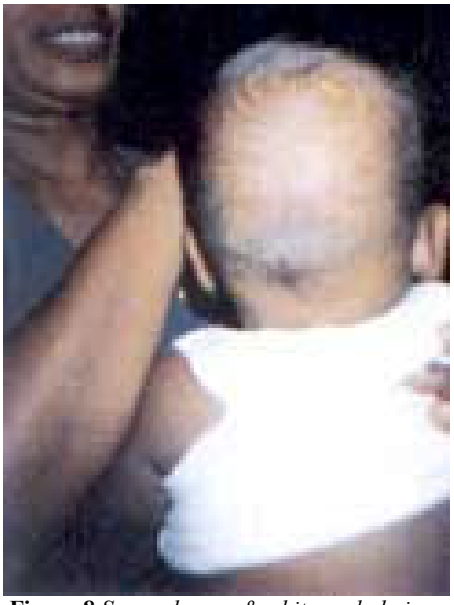


Figure 8 *Sparse brown & white scalp hairs.*

Diagnosis

Zinc deficiency should be suspected by clinical findings. Serum zinc and alkaline phosphatase are low and should rise during zinc therapy. The parallel course of the two parameters can be used diagnostically and to control treatment. It is important to consider the plasma level of albumin which binds 80% of circulating zinc. Severe hypoalbuminaemia is always associated with low serum zinc which does not necessarily reflect zinc deficiency. In clinical practice, a therapeutic trial with oral or parenteral zinc should be undertaken. If no clinical improvement occurs within 4 to 5 days and if serum alkaline phosphatase remains unaltered or even decreases, despite a rise in serum zinc, the patient is not zinc deficient.

Differential diagnosis

Zinc deficiency should be suspected in atypical or persistent napkin rash. Biotin responsive multiple carboxylase deficiency can present in infancy with a periorificial eruption similar to zinc deficiency. Cystic fibrosis is another disease that can cause zinc and essential fatty acid deficiency. In essential fatty acid deficiency lichenification, a fine branny desquamation and intertriginous and perioral dermatitis, is seen. Rarely a rash resembling seborrhoeic dermatitis can occur in zinc deficiency (Figures 9 and 10).

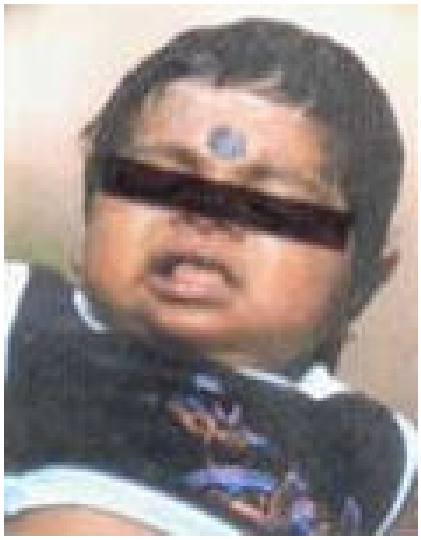
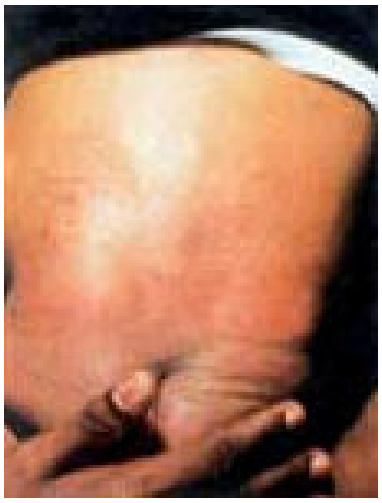


Figure 9 *Seborrhoeic dermatitis like lesions.*



. **Figure 10** *Involvement of back with seborrhoeic dermatitis like lesions & brown scaly edge*

References

1. Brandt T. Dermatitis in children with disturbances of general condition and absorption of food. *Acta Derm Venereol* 1936; **17**: 513-46.

2. Danbolt N, Closs K. Acrodermatitis enteropathica. *Acta Derm Venereol*1942; **23**: 127.

3. Zinc deficiency in Acrodermatitis enteropathica: multiple dietary intolerance treated with synthetic diet. *Proc Roy Soc Med* 1973; **66**: 327-9.