Acquired Acrodermatitis Enteropathica Secondary to Alcoholism

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Acrodermatitis enteropathica is a zinc deficiency disorder characterized by well-demarcated, erythematous, eczematous plaques in a periorificial and acral distribution. Hereditary and acquired forms have been described. We report a case of acquired acrodermatitis enteropathica secondary to alcoholism. Treatment of the underlying disorder and zinc replacement therapy resulted in rapid resolution of the condition.

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Case Report

A 61-year-old woman with a history of alcoholic cirrhosis was admitted to the hospital for detoxification after a weeklong alcohol binge. She complained of a diffuse, pruritic, prickly rash of 1 week's duration. Total body skin examination revealed scaly erythematous plaques in a periorbital, perioral, and perineal distribution, as well as in the nasolabial and inguinal folds (Figure). She also reported intermittent diarrhea, which she thought was exacerbating her perineal rash that had been treated using topical zinc oxide. Initial laboratory test results revealed a critically elevated serum alcohol level of 0.24 g/dL (reference range, <0.02 g/dL), as well as elevated levels of aspartate aminotransferase, lactate dehydrogenase, and total bilirubin. A nutritional deficiency was suspected. Her serum zinc level was decreased (55 μg/dL; reference range, 70–150 μg/dL). The clinical findings and laboratory test results indicated acquired acrodermatitis enteropathica, and the patient was started on zinc replacement therapy. She completed inpatient detoxification and underwent outpatient treatment for alcoholism. At a 3-month follow-up visit, she reported sobriety since her discharge from the hospital and there was near resolution of her cutaneous lesions. Additionally, a serum zinc level within reference range (71 μg/dL) was associated with her improved clinical and nutritional status.

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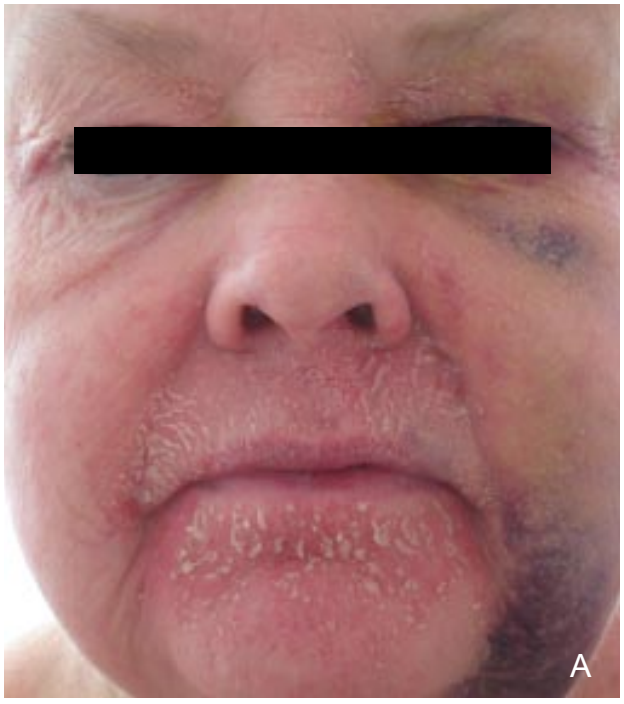
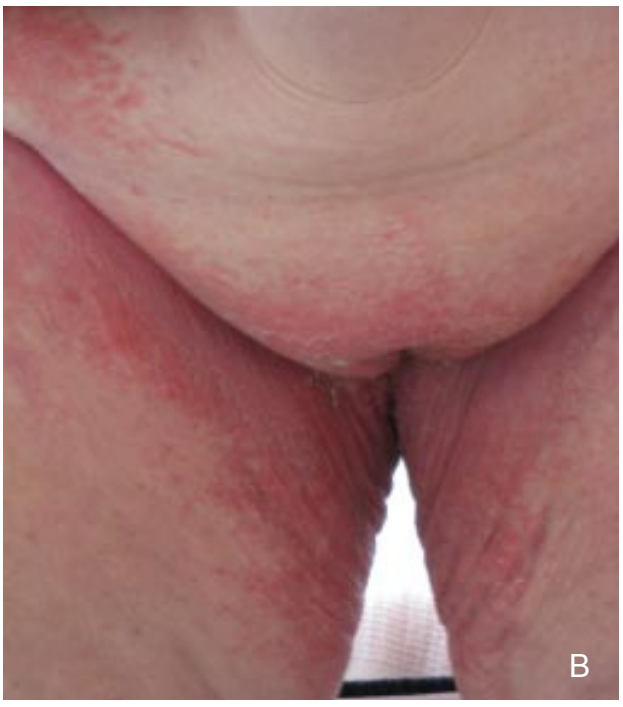
Comment

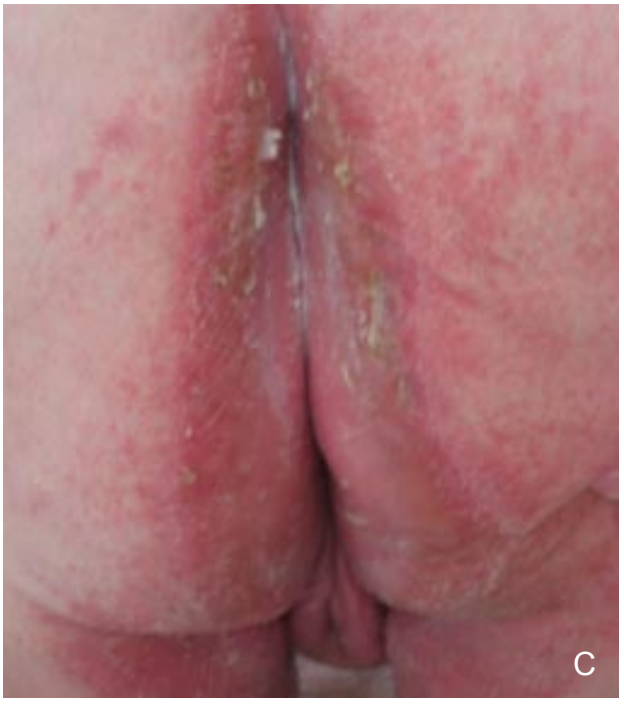
Acrodermatitis enteropathica was first described in 1936 by Brandt,[1] though the term was not coined until 1942 when Danbolt and Closs[2] identified it as a definitive disease. They described a dermatitis "located around the natural openings of the body and on protruding parts of the head, trunk, and extremities."[2] They also noted alopecia, pustulous paronychia, atrophy of the nails, photophobia, and oral mucosal involvement.[2] Initially thought to be uniformly fatal, Moynahan[3] linked the clinical entity with zinc deficiency and demonstrated clinical improvement with zinc replacement therapy. Since this crucial link was established, hereditary and acquired deficiency states have been described. The hereditary form typically becomes apparent in infancy, within days for bottle-fed infants and within days to weeks after weaning breastfed infants.[4] The acquired form can present at any age.

Acrodermatitis enteropathica can be inherited as a rare autosomal recessive disorder. The solute carrier family 39 (zinc transporter) member 4 gene, *SLC39A4*, located on band 8q24.3, has been implicated in the pathophysiology of hereditary acrodermatitis enteropathica. The gene encodes a histidine-rich protein hZIP4, which is believed to play a role in zinc uptake.[5] Several mutations of this gene have been described.[6,7]

Acrodermatitis enteropathica also can be acquired by inadequate dietary consumption, impaired intestinal absorption, or increased loss of zinc. The condition has been described in a variety of malnutrition and malabsorption states, including anorexia nervosa, parenteral hyperalimentation, cystic fibrosis, malignancy, pancreatitis, intestinal bypass surgery, and burns.[8-18] Several cases of acquired zinc deficiency associated with alcoholic liver disease







Erythematous eczematous periorbital and perioral plaques (A). Well-demarcated erythematous plaques in inguinal folds (B). Perineal involvement with sharply demarcated erythema and scale (C).

have been described,[9-13] which may be attributable to several different mechanisms. Poor eating habits; atrophic gastritis; malabsorption; and steatorrhea, which leads to the formation of insoluble alkaline zinc soaps, may all be contributing factors.[11] In addition, excess consumption of alcohol has been associated with hyperzincuria.[19]

Clinically, acrodermatitis enteropathica is characterized by well-demarcated, erythematous, eczematous plaques in a periorificial and acral distribution. Chronicity, alopecia, nail dystrophy, angular stomatitis, and chronic paronychia may develop.[9] As in our patient, diarrhea is a common complaint and, in the past, was thought to be essential in diagnosing acrodermatitis enteropathica, along with total alopecia and the periorificial distribution of the skin lesions.[20] Poor wound healing may be evident for several reasons. Cellular immunity and delayed-type hypersensitivity is reduced in alcohol abuse, and zinc deficiency itself has been shown to reduce both cellular and humoral immunity.[21,22]

The histopathology of acrodermatitis enteropathica varies with the clinical stage of the lesions. Early lesions show focal parakeratosis, loss of the granular layer, and mild pallor of the keratinocytes in the upper epidermis. With progression, the parakeratosis becomes more confluent, the epidermis becomes more psoriasiform, and the pallor of the upper epidermis becomes more prominent. Although the pallor later disappears, the psoriasiform hyperplasia persists. With involution, only parakeratosis remains.[23]

Clinical presentation and patient history should be indicative of acrodermatitis enteropathica. Decreased serum zinc levels and rapid resolution of skin lesions with zinc replacement therapy should confirm the diagnosis. Treatment of the underlying

cause is paramount. After laboratory test results are obtained, zinc replacement therapy should be initiated early to prevent the serious complications of secondary infections.[18] Zinc replacement therapy should be started at 3 mg/kg per day, with monitoring of serum or plasma zinc levels every 3 to 6 months. Typically, clinical improvement is seen before a substantial change in serum zinc levels.[4]

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