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CASE REPORT

Zinc Supplementation for Acrodermatitis Enteropathica Overlapped with Psoriasiform Lesions

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Abstract: Acrodermatitis enteropathica (AE) can be caused by inherited or acquired zinc deficiency, among which site-specific skin lesions or even psoriasiform skin manifestations are present. Few cases exist in the literature involving the diagnosis and treatment of AE overlapped with psoriasiform lesions. In this case, we reported a teenage boy presented characteristic site-specific skin lesions of AE with low serum zinc level, subsequently progressed into generalized pustular psoriasiform manifestations under a genetic background, while a rapid recovery was observed after monotherapy of zinc supplementation. Since zinc malabsorption was suspend to be the trigger of both AE and psoriasiform lesions, simply maintaining balanced zinc homeostasis might be a safe and effective treatment for the overlapped manifestations.

Keywords: acrodermatitis enteropathica, psoriasis, zinc

Zinc is important for skin metabolism and health.[1] Acquired or inherited zinc deficiency can induce acrodermatitis enteropathica (AE) as well as psoriasiform skin lesions, making the diagnosis challenging. We here reported a case of zinc deficiency-triggered AE overlapped with psoriasiform lesions with useful and safe zinc supplementation for treatment.

A 13-year-old boy complained of generalized erythema, desquamation and pustules for 4 weeks with significant pain and mild itching. A history of intermittent erythema, erosion, and exudation in the perianal and perineal regions for almost 2 years was reported. He was initially diagnosed with perianal eczema and treated with topical corticosteroids and antibiotics at a local hospital, but no improvement was observed and the eruption of erythema and pustules was subsequently generalized. He was then diagnosed with acute generalized exanthematous pustulosis, and oral acitretin (30 mg/d) was administered for over 4 weeks, but minimal improvement was achieved. No family history of skin diseases was reported.

Notable erythema and desquamation were present around the anus and genitalia, as well as on the lower legs and scalp (Figure 1A and B). And the symptoms of geographic tongue were apparent (Figure 1C). Blood tests revealed an elevated white blood cell count, increased serum alkaline phosphatase levels, and decreased serum zinc levels. We prescribed an oral zinc supplement (30 mg, twice a day) and continued acitretin treatment (30 mg/d). Dramatic improvement was observed after 1-week treatment; oral acitretin was withdrawn by tapering, and the zinc supplement dosage was maintained for the next 4 weeks. The skin eruptions completely disappeared at the 3rd week. Serum zinc concentration increased to a normal level at the 4th week. On the basis of the patient's clinical

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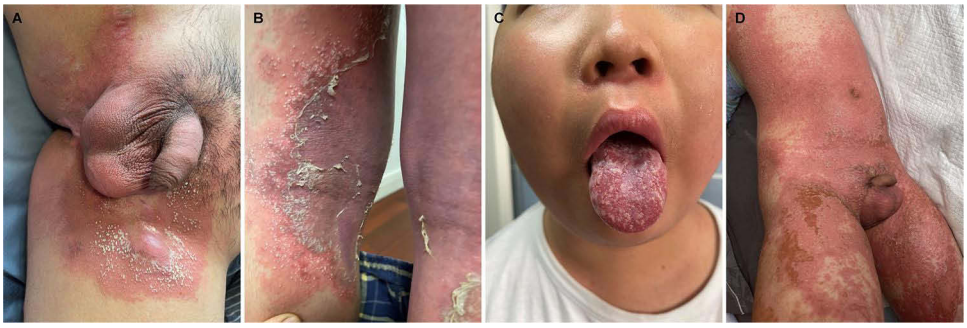

Figure 1 Physical examination images. (**A**) Perianal lesions with erythema. (**B**) Lower limb erythema with desquamation. (**C**) Oral lesions with a "geographic tongue". (**D**) Erythrodermic manifestations with generalized erythema.

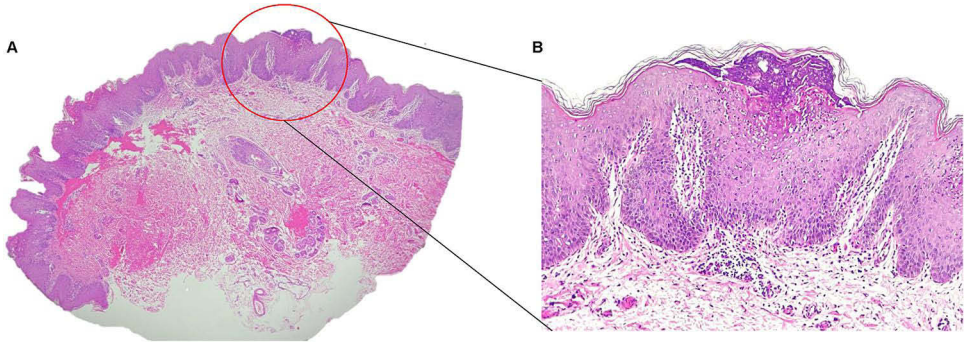

Figure 2 Representative histopathological images. (**A**) The biopsy of an inguinal skin lesion exhibits eruptive pustular psoriasis with mild to moderate acanthosis and multifocal subcorneal pustules, with mounds of neutrophils, dilated vessels in the superficial dermis and lymphocytic perivascular infiltrates (low power ×4). (**B**) A typical Kogoj spongiform subcorneal pustule filled with neutrophils can be observed, with dilated capillaries in the dermal papillae (high power ×10).

manifestations, blood test, rapid treatment response, and histopathological results (Figure 2), a high suspicion of AE was initially diagnosed.

However, during follow-up, the patient exhibited psoriasiform manifestations other than acrodermatitis, such as generalized erythema and desquamation on the thighs and trunk (Figure 1D), when the dosage of the oral zinc supplement was reduced by half (30 mg/d) or the patient suffered from infections. Specific mutations in the gene encoding the IL-36 receptor antagonist (*IL36RN*) were detected (Figure 3). Notably, treatment to normalize the serum zinc levels also improved these manifestations. This teenage patient is undergoing regular follow-up every 3 months, including clinical examination and serum zinc level estimation. The patient was maintained at 30 mg of zinc twice a day for remission during the 2 years of follow-up.

In this case, zinc deficiency may be viewed as a trigger for dermatosis caused by bowel inflammation, since gastrointestinal endoscopy indicated bowel inflammation in other hospitals. Low zinc level and site-specific skin lesions, predominantly in extremity skin regions, with a rapid clinical response to zinc supplementation, strongly supported a diagnosis of acquired AE.[2] Moreover, the family gene test profile and the pathological finding of skin biopsy co-confirmed the genetic background of psoriasis,[3] especially the generalized pustular psoriasis. Actually, zinc deficiency also plays an important role in the pathogenesis of psoriasis.[4] Inflammatory bowel disease is also a common comorbidity of psoriasis inducing zinc deficiency.[5] And the clinical presentations of psoriasis and AE may overlap, and even on histopathological examination.[6] Impressively, maintaining balanced zinc homeostasis via monotherapy of zinc supplement may be a safe and effective treatment, which provides significant clinical experience for the treatment of overlapping manifestations of AE and psoriasis.

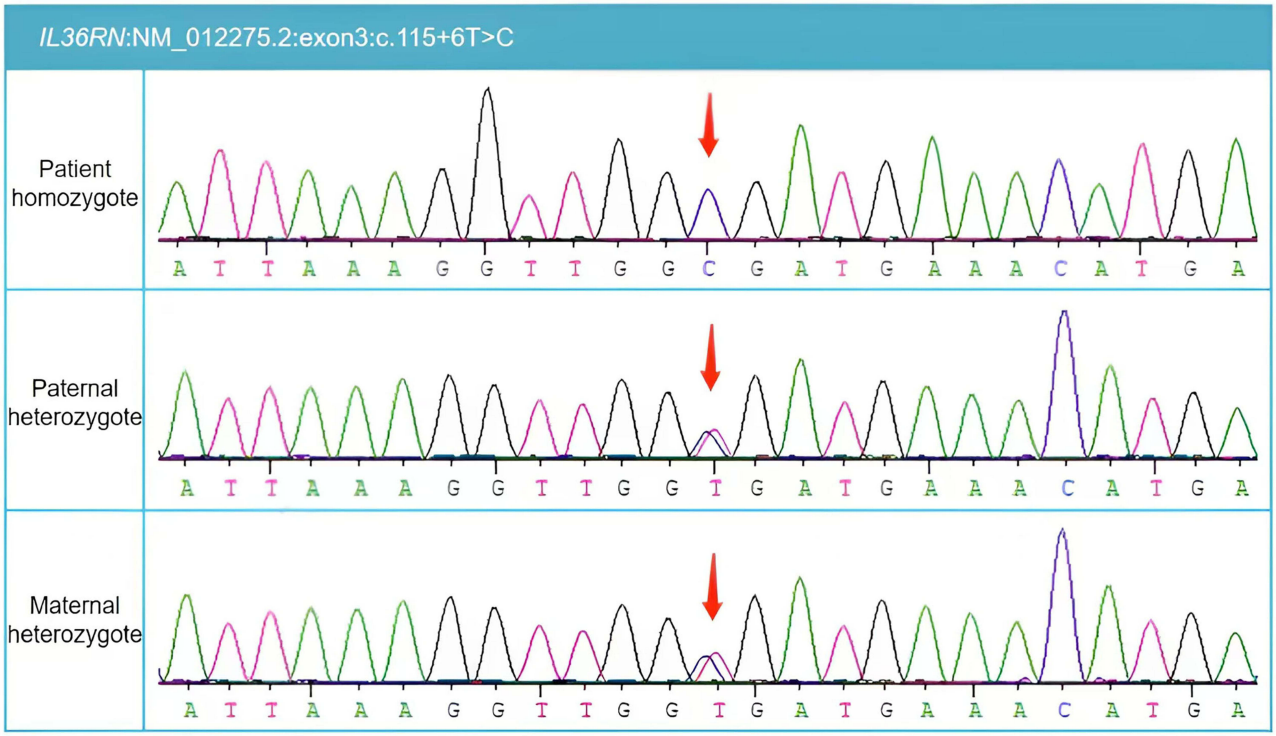

Figure 3 Gene sequencing results of the patient and his parents. The patient was homozygous for a novel missense mutation (c.115+6T>C) in exon 3 of gene *IL36RN* on chromosome 2, which is related to GPP. The c.115+6T>C mutation was inherited from his parents who were compound heterozygous.

Ethics and Consent

The patient reportedly and his parents gave written consent to the authors for the publication of the clinical history and photographs. It was not required the institutional approval to publish the case details.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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