

Zinc Spray for Treatment of Acrodermatitis Enteropathica

Akrodermatitis Enteropatika Tedavisinde Çinko Sprey

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ABSTRACT

Acrodermatitis enteropathica is a rare disorder caused by zinc deficiency. The classic triad of symptoms includes perioral dermatitis, diarrhea, and alopecia. A rare cause of the acquired form is zinc deficiency due to total parenteral nutrition. Diagnosis can be challenging due to nonspecific symptoms. This paper presents a case of acrodermatitis enteropathica in a 5-month-old infant who had been on total parenteral nutrition since birth. Unlike the literature, the patient's clinical improvement was observed with zinc spray and zinc cream treatment.

Key Words: Acrodermatitis enteropathica, Zinc deficiency, Zinc spray

ÖZ

Akrodermatitis enteropatika çinko eksikliğinin neden olduğu nadir görülen bir hastalıktır. Klasik triadı perioral dermatit, diyare ve alopesidir. Kazanılmış formunun nadir bir nedeni total parenteral beslenmeye bağlı eksik çinko alımıdır. Belirti ve bulguları spesifik olmadığı için tanı koymak zordur. Biz bu makalede doğduğundan beri total parenteral beslenen 5 aylık bir hastada gelişen akrodermatitis enteropatika olgusunun literatürden farklı olarak çinko sprej ve çinko krem tedavisi ile görülen klinik iyileşmesini sunuyoruz.

Anahtar Kelimeler: Akrodermatitis enteropatika, Çinko eksikliği, Çinko sprej

INTRODUCTION

Zinc, the body's second-most abundant trace element, is critical for tissue development, differentiation, and growth (1,2). Zinc deficiency presents as acrodermatitis enteropathica (AE), a condition marked by eczema, pustular lesions in perioral and acral regions, diarrhea, and alopecia (3). This deficiency can be inherited or acquired, with acquired cases stemming from inadequate dietary intake, malabsorption syndromes (e.g., celiac disease), and short bowel syndrome (4,5). This paper presents a case of acrodermatitis enteropathica arising from zinc deficiency in a patient with short bowel syndrome who relies on long-term total parenteral nutrition (TPN).

CASE REPORT

A female infant born at 36 weeks via cesarean section with a weight of 2540 grams to a 24-year-old mother was admitted to the neonatal intensive care unit (NICU) with a diagnosis of hypoxic-ischemic encephalopathy. Due to respiratory distress, the patient was intubated and placed on mechanical ventilation. Therapeutic hypothermia was initiated. After completing 72 hours of therapeutic hypothermia, the patient underwent surgery on the third day of hospitalization for perforated necrotizing enterocolitis. The patient had multiple perforations in the jejunum, terminal ileum, and transverse colon. Approximately 50 cm of necrotic bowel was resected, and a colostomy was created. Following surgery, the

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Figure 1: Diffuse hair loss due to zinc and trace element deficiency.



Figure 3: Improvement after zinc therapy.



Figure 2: Skin lesions at the initial presentation of zinc deficiency.

patient developed short bowel syndrome and was initiated on TPN. Intermittent enteral feeding was attempted, but enteral feeding was never possible due to feeding intolerance and abdominal distension. The patient received TPN for 5 months. In the fourth month of follow-up, the patient developed perioral lesions and alopecia of the scalp (Figure 1). Physical examination revealed sharply demarcated, yellow-brown erythematous, scaly, erosive, and occasionally dirty yellow crusted lesions around the mouth, face, neck, arms, and genital area (Figure 2). Laboratory tests revealed leukocytes $10.51 \times 10^3/\mu\text{L}$, hemoglobin 10.1 g/dL, platelets $343 \times 10^3/\mu\text{L}$, albumin 3.4 g/dL, and alkaline phosphatase 33 U/L. Serum zinc level was 54.16 $\mu\text{g}/\text{dL}$ (normal range 70-114 $\mu\text{g}/\text{dL}$). Acrodermatitis enteropathica secondary to zinc deficiency was suspected in our patient. Due to the unavailability of an intravenous form of zinc-containing trace element preparation in our country for the past year, it could not be added to TPN. Oral zinc suspension could not be administered to the patient with short bowel syndrome who was unable to tolerate enteral feeding. Therefore, the patient was given 1.1 mg zinc spray (Costus Root Spray, Nutraxin, Germany) twice a day intraorally and wound care was performed with zinc cream. Significant improvement in skin lesions was observed within a week of initiating treatment. The lesions completely regressed on the 15th day of treatment (Figure 3). The patient is currently being followed up in the NICU and continues to receive zinc therapy.

DISCUSSION

Zinc is the third most abundant trace element in the human body, with the highest concentrations found in skeletal muscle, bone, and skin (6). Acrodermatitis enteropathica (AE) is a rare disorder characterized by dermatitis, diarrhea, and alopecia caused by zinc deficiency (7). The classic triad of symptoms is present in only about 20% of cases (8). The disease typically presents with erythematous, scaly, eczematous lesions in perioral, genital, and acral areas. If left untreated, the lesions may become erosive (9). Our patient also had erythematous, scaly, erosive lesions of yellowish-brown color around the mouth, neck, and genital area, as well as alopecia. Since she had diarrhea throughout her enteral feeding period, it was more likely associated with short bowel syndrome.

Inherited AE results from mutations in the SLC39A4 gene, which encodes the zinc transporter protein ZIP4 (5). The acquired form can be due to multiple causes, including inadequate intake, malabsorption syndromes, excessive intestinal loss, renal loss, and, rarely, iatrogenic zinc deficiency due to TPN (10). Patients on long-term TPN are at risk of trace element deficiencies (4). In our country, since trace elements and intravenous forms of zinc have not been added to TPN for the past year, careful attention should be paid to zinc deficiency in patients receiving TPN.

Zinc is a cofactor for numerous enzymes, including alkaline phosphatase and RNA polymerase (9). The diagnosis of acrodermatitis enteropathica is primarily based on clinical findings and confirmed by plasma zinc deficiency (10,11). Low alkaline phosphatase levels, a zinc-dependent enzyme, can also aid in diagnosis (8). Even if the serum zinc level is normal, clinical symptoms may be present if the zinc-bound form of albumin is low (9). Our patient was diagnosed with acquired zinc deficiency-related AE based on clinical findings, a serum zinc level of 54.16 $\mu\text{g}/\text{dL}$, and an alkaline phosphatase level of 33 U/L. After treatment, her alkaline phosphatase level was found to be 371 U/L. In patients suspected of zinc deficiency, low alkaline phosphatase levels can be a helpful diagnostic clue if zinc levels cannot be measured.

Zinc replacement is the standard treatment for AE stemming from dietary deficiency. Elemental zinc is often initiated at a dosage of 0.5-1 mg/kg/day. In acquired cases, response to zinc supplementation is typically rapid (12). Previous work in the literature demonstrates the use of oral or intravenous zinc to treat iatrogenic AE in patients receiving TPN (3,4,10). Our patient's inability to tolerate oral intake precluded the use of zinc suspension. Additionally, an intravenous zinc formulation was unavailable in our country for inclusion in the TPN regimen. In a departure from standard protocols, we administered zinc spray orally twice daily and applied zinc cream for wound care. The patient's lesions showed improvement on the 7th day of treatment and fully resolved by the 15th day.

In conclusion, careful monitoring for trace element deficiencies is essential in patients receiving TPN. This case highlights the potential effectiveness of topical zinc spray and cream formulations as alternative treatment options for individuals with zinc deficiency who cannot receive oral or intravenous replacement.

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