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ACRODERMATITIS ENTEROPATHICA PRESENTING IN AN EXCLUSIVELY BREASTFED 3-MONTH-OLD INFANT: AN EXPANDED CASE REPORT WITH PRETREATMENT AND POST-TREATMENT CLINICAL IMAGING

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ABSTRACT **Background:** Acrodermatitis enteropathica is a rare but highly treatable disorder of zinc availability. It may occur as a hereditary defect of intestinal zinc absorption or as an acquired/transient zinc deficiency state. The disease is clinically important because the skin often provides the earliest and most visible clue to a systemic micronutrient deficiency. The classical triad includes periorificial dermatitis, alopecia, and diarrhea; however, infants may present with incomplete, evolving, or infection-mimicking lesions. Zinc is essential for epithelial integrity, cellular proliferation, immune regulation, wound healing, taste, appetite, and growth, and delayed recognition may result in persistent dermatitis, poor feeding, growth failure, recurrent infections, and avoidable morbidity.1,2,3**Case presentation:** We report a 3-month-old exclusively breastfed male infant who presented with a 20-day history of progressive erythematous scaly lesions around the mouth, genital/perianal region, hands, feet, and lower limbs. The lesions gradually evolved into crusted, erosive, excoriated, and hyperpigmented plaques and were associated with intermittent loose stools, irritability, decreased feeding, and poor weight gain. He was born at term by normal vaginal delivery, with no significant antenatal or perinatal complications. On examination, he weighed 4.5 kg and appeared irritable. Symmetrical periorificial, perianal, genital/perineal, acral, and lower-limb plaques were present, along with sparse scalp hair and mild alopecia. Systemic examination was otherwise normal. Laboratory evaluation showed hemoglobin 10.2 g/dL, total leukocyte count 8,500/mm3, serum zinc 35 microg/dL (reference range 70-120 microg/dL), and reduced serum alkaline phosphatase.**Intervention and outcome:** A clinicobiochemical diagnosis of zinc deficiency dermatitis consistent with acrodermatitis enteropathica phenotype was made. The infant was started on oral elemental zinc supplementation at 3 mg/kg/day along with supportive skin care and maternal nutritional counseling. A clinically evident response was observed within one week, with reduced erythema, healing of erosions, improved feeding, and decreased irritability. Complete resolution of active skin lesions occurred over four weeks, leaving only residual post-inflammatory pigmentation in some areas.**Conclusion:** This case highlights the need to consider zinc deficiency in infants with periorificial, acral, genital, and perianal dermatitis, particularly when diarrhea, alopecia, irritability, feeding difficulty, or poor weight gain coexist. Early recognition, serum zinc estimation, and timely zinc replacement can produce rapid clinical recovery and prevent complications.

KEYWORDS : Acrodermatitis enteropathica; zinc deficiency; infantile dermatitis; exclusively breastfed infant; periorificial dermatitis; acral dermatitis; alopecia; diarrhea; oral zinc supplementation; clinical imaging.

INTRODUCTION

Acrodermatitis enteropathica occupies a distinctive place among pediatric nutritional and dermatologic disorders because it links a characteristic skin phenotype with a correctable systemic deficiency. The term is traditionally used for an autosomal recessive disorder of zinc absorption, but in clinical practice an acrodermatitis-enteropathica-like phenotype may also be produced by acquired or transient zinc deficiency. This broader clinical concept is especially useful in infancy, where nutritional requirements are high, cutaneous signs may be dramatic, and rapid therapeutic response can be achieved if the diagnosis is considered early.1,4

Zinc is a trace element with extensive biological roles. It functions as a catalytic, structural, and regulatory cofactor for numerous enzymes and transcription factors and participates in DNA and RNA synthesis, cell division, protein metabolism, antioxidant defense, immune function, epithelial repair, and wound healing. The skin, gastrointestinal tract, immune system, and growing tissues are therefore particularly vulnerable when zinc supply becomes inadequate.2,3,5,6

The inherited form of acrodermatitis enteropathica is caused by impaired intestinal zinc uptake, most commonly due to pathogenic variants in SLC39A4, which encodes the ZIP4 zinc transporter. ZIP4 is critical for zinc absorption at the intestinal epithelial surface, and its dysfunction produces a lifelong tendency to severe zinc deficiency unless supplementation is provided. The molecular identification of SLC39A4 explained the classical autosomal recessive disease phenotype and highlighted the importance of zinc transporters in human nutrition.7,8,9

Zinc homeostasis is maintained through coordinated regulation of uptake, efflux, intracellular sequestration, and protein binding. ZIP transporters generally increase cytosolic zinc by promoting influx into the cytoplasm, whereas ZnT transporters help lower cytosolic zinc by efflux or sequestration. In infants, this finely balanced system can be stressed by rapid growth, inadequate intake, low stores, increased losses, malabsorption, or inadequate zinc transfer in breast milk.10,11,21

Clinically, the classical triad of acrodermatitis enteropathica includes periorificial dermatitis, alopecia, and diarrhea. Nevertheless, the triad may not be complete at first presentation. Cutaneous lesions may be erythematous, eczematous, psoriasiform, vesiculobullous, pustular, erosive, crusted, or hyperpigmented, and they often involve perioral, perianal, genital, acral, and intertriginous sites. Because such lesions can resemble common conditions such as atopic dermatitis, seborrheic dermatitis, candidiasis, irritant diaper dermatitis, and bacterial infection, diagnostic delay is possible.1,12,13,17,18

The present case is noteworthy because the infant was exclusively breastfed and developed a classical zinc-deficiency dermatitis pattern with biochemical confirmation and rapid response to oral zinc. The serial clinical images provide a clear visual correlation between disease activity and therapeutic recovery, reinforcing the teaching value of this report for pediatricians, dermatologists, and primary care clinicians.14,15,16

CASE REPORT

Patient information and presenting complaints

A 3-month-old male infant was brought for evaluation of progressive skin lesions of approximately 20 days duration. The eruption began as erythematous and scaly lesions around the mouth and subsequently involved the genital and perianal areas, hands, feet, lower limbs, and perineal region. Over time, the lesions became more extensive and developed crusting, erosions, excoriation, and post-inflammatory hyperpigmentation. The infant also had intermittent loose stools, irritability, reduced feeding, and poor weight gain, creating concern for a systemic process rather than an isolated localized dermatitis.

The infant was born at term by normal vaginal delivery. No significant antenatal, intrapartum, or perinatal complications were documented. He was exclusively breastfed at the time of presentation. There was no documented systemic abnormality outside the dermatologic, gastrointestinal, feeding, and growth-related concerns described in the supplied case record.

Clinical examination

On examination, the infant weighed 4.5 kg and appeared irritable. The cutaneous eruption showed a strikingly symmetrical and characteristic distribution. Erythematous crusted plaques involved the perioral region, with extension to other periorificial and acral sites. The perianal and diaper area showed severe erosive dermatitis with excoriation. The lower limbs and feet showed erosive erythematous plaques with surrounding hyperpigmentation. Symmetrical hyperpigmented scaly lesions were noted over the lower limbs and perineal region. Sparse scalp hair and mild alopecia were present. Systemic examination was otherwise normal.

The distribution of lesions was clinically important. Perioral involvement, acral extension, and severe perianal dermatitis together form a highly suggestive pattern for zinc deficiency dermatitis. The association with intermittent diarrhea, feeding difficulty, irritability, poor weight gain, and alopecia further strengthened the clinical impression of acrodermatitis enteropathica phenotype rather than isolated eczema or diaper dermatitis.

Laboratory investigations

Initial laboratory investigations showed hemoglobin 10.2 g/dL and total leukocyte count 8,500/mm3. Serum zinc was markedly reduced at 35 microg/dL against a stated reference range of 70-120 microg/dL. Serum alkaline phosphatase level was reduced. In the setting of a compatible cutaneous distribution and systemic symptoms, the low serum zinc level provided strong biochemical support for the diagnosis. Reduced alkaline phosphatase was also supportive because alkaline phosphatase is a zinc-dependent metalloenzyme and may be low in zinc deficiency.

Clinical and laboratory summary

Differential diagnosis	Clinical overlap	Features favoring zinc deficiency in this case
Irritant diaper dermatitis	Erythema and erosions in diaper area	Extensive perioral/acral lesions, alopecia, diarrhea, low serum zinc, rapid zinc response
Candidal dermatitis	Perianal erythema and erosions; may involve folds	Acral-periorificial distribution and biochemical zinc deficiency
Atopic dermatitis	Eczematous lesions and irritability	Periorificial, acral, and perianal pattern with diarrhea and alopecia
Seborrheic dermatitis	Infantile scaling and erythema	Erosive acral/perianal disease and low serum zinc
Biotin deficiency	Periorificial dermatitis and alopecia	Documented low zinc with reduced alkaline phosphatase and response to zinc
Essential fatty acid deficiency	Scaly dermatitis and growth concerns	Typical zinc distribution with low serum zinc
Langerhans cell histiocytosis	Persistent seborrheic/diaper-like rash	Clinical improvement with zinc and absence of systemic red flags in record
Immunodeficiency-associated dermatitis	Recurrent/persistent infection-like lesions	Classic zinc deficiency pattern and zinc response

Diagnostic reasoning

The diagnosis was made on clinicobiochemical grounds. The morphology and distribution of the eruption were highly suggestive: periorificial dermatitis, acral involvement, severe diaper/perianal dermatitis, and genital/perineal lesions. The accompanying symptoms - diarrhea, irritability, feeding difficulty, poor weight gain, sparse scalp hair, and alopecia - supported a nutritional/systemic etiology. The markedly low serum zinc level and reduced alkaline phosphatase further consolidated the diagnosis of zinc deficiency dermatitis/acrodermatitis enteropathica phenotype.1,13,16

The diagnosis was not based on one isolated observation. Instead, it emerged from the convergence of distribution, morphology, associated gastrointestinal symptoms, hair changes, biochemical zinc deficiency, and therapeutic response. This integrated approach is important because serum zinc levels may be affected by timing of sampling, inflammation, albumin status, and laboratory technique, while cutaneous morphology alone can overlap with several common pediatric dermatoses.1,10,17

Differential diagnosis

Parameter	Case finding
Age/sex	3-month-old male infant
Feeding status	Exclusively breastfed
Birth history	Term infant, normal vaginal delivery; no significant antenatal or perinatal complications documented
Duration of lesions	Approximately 20 days
Initial morphology	Erythematous and scaly lesions
Progression	Crusting, erosions, excoriation, and hyperpigmentation
Distribution	Perioral, genital/perineal, perianal/diaper area, hands, feet, lower limbs
Associated symptoms	Intermittent loose stools, irritability, decreased feeding, and poor weight gain
Hair finding	Sparse scalp hair and mild alopecia
Weight at presentation	4.5 kg
Hemoglobin	10.2 g/dL
Total leukocyte count	8,500/mm3
Serum zinc	35 microg/dL; reference range 70-120 microg/dL
Serum alkaline phosphatase	Reduced
Working diagnosis	Zinc deficiency dermatitis consistent with acrodermatitis enteropathica phenotype
Treatment	Oral elemental zinc 3 mg/kg/day with supportive skin care and maternal nutritional counseling
Initial response	Clinical improvement within one week
Final response	Complete resolution of active lesions over four weeks

Several conditions may mimic zinc deficiency dermatitis in early infancy. The following differential diagnosis framework was considered clinically relevant for this presentation:

Final diagnosis

Zinc deficiency dermatitis consistent with acrodermatitis enteropathica phenotype in a 3-month-old exclusively breastfed infant, presenting with periorificial, acral, genital/perineal, and perianal dermatitis, diarrhea, irritability, feeding difficulty, poor weight gain, alopecia, low serum zinc, reduced alkaline phosphatase, and rapid response to oral zinc supplementation.

Therapeutic intervention

The infant was started on oral elemental zinc supplementation at a dose of 3 mg/kg/day. Supportive skin care was provided to promote healing of erosions, reduce irritation, and prevent secondary infection. Maternal nutritional counseling was also provided because the infant was exclusively breastfed. Oral zinc therapy is the cornerstone of treatment for both classical acrodermatitis enteropathica and severe zinc-deficiency dermatitis, and clinical response is often rapid when the diagnosis is correct.12,13,19

The management plan emphasized correction of the underlying deficiency rather than repeated topical treatment alone. This is a key clinical message: while skin care is important for comfort, barrier repair, and prevention of superinfection, definitive improvement requires zinc repletion when zinc deficiency is the primary driver of disease.

Outcome and follow-up

Significant clinical improvement was observed within one week of starting zinc supplementation. Erythema decreased, erosions began to heal, feeding improved, and irritability reduced. Over the following weeks, the cutaneous lesions continued to improve. Complete resolution of active lesions occurred over four weeks. Post-treatment images demonstrated marked healing of perioral and perianal dermatitis and significant improvement of lower-limb lesions, with residual post-inflammatory pigmentation in some areas.

The speed of improvement was diagnostically meaningful. In zinc deficiency dermatitis, visible improvement can begin within days of

treatment, and the dramatic response helps distinguish the disease from many mimics. Continued follow-up is important to monitor growth, feeding, recurrence of lesions, adherence, and the need for ongoing supplementation, especially when the underlying mechanism - inherited, acquired, or transient - has not been definitively established.12,13,21

Clinical Figures and Figure Legends

The clinical photographs are arranged into pretreatment and post-treatment sequences. Each image has been directly labeled for clarity, and individual figure legends are provided below each panel. The sequence demonstrates the characteristic distribution of zinc deficiency dermatitis before therapy and the marked clinical recovery after zinc supplementation.

Pretreatment clinical images

Figure 1A. Pretreatment periorificial dermatitis showing erythematous, crusted, and hyperpigmented lesions involving the perioral region.

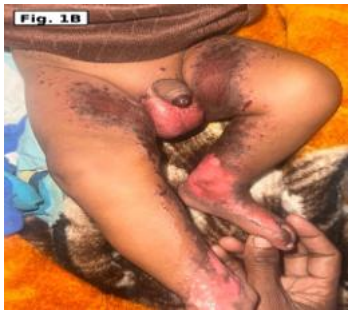

Figure 1B. Pretreatment acral and lower-limb involvement with erosive erythematous plaques and surrounding hyperpigmentation.

Figure 1C. Pretreatment severe perianal dermatitis with erosions and excoriation involving the diaper area.

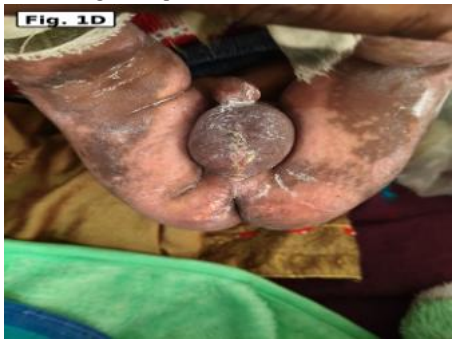

Figure 1D. Pretreatment symmetrical hyperpigmented and scaly lesions involving the lower limbs and perineal region.

Post-treatment clinical images

Figure 2A. Post-treatment complete resolution of perioral lesions after oral zinc supplementation.

Figure 2B. Post-treatment marked healing of perianal dermatitis with residual post-inflammatory pigmentation.

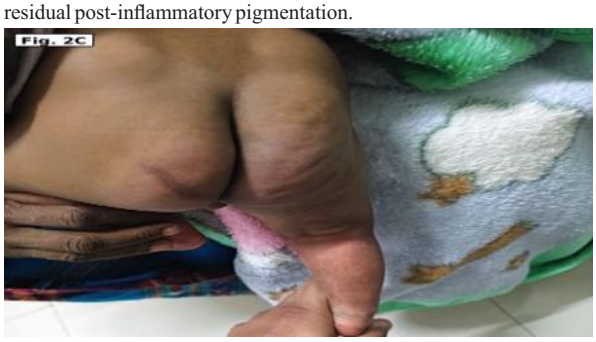

Figure 2C. Post-treatment significant improvement of lower-limb lesions after zinc supplementation.

DISCUSSION

This report illustrates a classic but easily missed presentation of zinc deficiency dermatitis in early infancy. The major teaching value lies in the pattern recognition: periorificial dermatitis, acral involvement, severe perianal/diaper-area disease, gastrointestinal symptoms, sparse hair/alopecia, poor feeding, and biochemical zinc deficiency. Each feature alone may be nonspecific, but their combination creates a characteristic clinical signature. The response to zinc supplementation then completes the clinicobiochemical narrative.1,13,16

The skin manifestations of zinc deficiency are biologically plausible. Zinc is required for keratinocyte proliferation, epithelial repair, nucleic acid synthesis, protein metabolism, and immune defense. When zinc availability falls, rapidly renewing tissues such as skin and intestinal mucosa are affected early. This explains why infants may present with dermatitis, diarrhea, feeding difficulty, impaired growth, poor wound healing, and susceptibility to infection.2,3,5,22

The distribution of lesions in this infant was particularly characteristic. Perioral involvement reflects periorificial predilection; severe perianal and diaper-area dermatitis reflects vulnerability of moist, irritated, high-turnover skin; and acral/lower-limb disease reflects the acral component of the syndrome. Hyperpigmentation and residual pigmentation after healing are common in inflammatory dermatoses and were evident in both pretreatment and post-treatment images.1,17,18,20

Alopecia and sparse scalp hair are valuable diagnostic clues. Hair follicles are rapidly proliferating structures and are vulnerable to

micronutrient deficiency. Similarly, diarrhea may be both a manifestation and an aggravating factor: zinc deficiency can impair intestinal epithelial integrity and immune function, while diarrhea can increase losses and further worsen deficiency. The coexistence of dermatitis, diarrhea, and alopecia should therefore trigger early zinc estimation.1,3,13

An important aspect of this case is exclusive breastfeeding. Breast milk is usually adequate for most infants, yet zinc deficiency phenotypes can occur in exclusively breastfed infants under certain circumstances. Potential mechanisms include low maternal zinc status, reduced mammary zinc secretion, transient neonatal zinc deficiency, increased infant requirement, prematurity-related low stores, or unrecognized malabsorption. In this case, the available history documented exclusive breastfeeding and term delivery, but detailed maternal zinc studies, breast-milk zinc concentration, and genetic testing were not included in the supplied record. Therefore, the most precise wording is zinc deficiency dermatitis consistent with an acrodermatitis enteropathica phenotype.14,15,21

Classical hereditary acrodermatitis enteropathica is a lifelong disorder due to impaired zinc absorption, usually linked to pathogenic variants in SLC39A4. The affected ZIP4 transporter impairs intestinal zinc uptake and produces a tendency toward severe deficiency after zinc supply becomes insufficient. However, acquired and transient forms can produce very similar clinical pictures. Differentiating these possibilities is relevant for long-term management because hereditary disease generally requires lifelong zinc supplementation, whereas acquired or transient deficiency may improve once the underlying factor is corrected.7,8,9,13

The diagnosis of zinc deficiency dermatitis should be practical, prompt, and integrated. Serum zinc is useful but not perfect; values may vary with infection, inflammation, fasting status, albumin concentration, and sampling technique. Reduced alkaline phosphatase may support the diagnosis because the enzyme is zinc dependent. Nevertheless, the most persuasive evidence is often the combination of compatible clinical pattern, low zinc level, supportive laboratory findings, and rapid therapeutic response.1,10,11,16

The differential diagnosis is broad and clinically important. Diaper dermatitis, candidiasis, atopic dermatitis, seborrheic dermatitis, bacterial infection, psoriasis-like dermatitis, nutritional dermatoses such as biotin or essential fatty acid deficiency, Langerhans cell histiocytosis, and immunodeficiency can all produce overlapping features. However, the acral-periorificial distribution, gastrointestinal symptoms, alopecia, low zinc, and zinc responsiveness make zinc deficiency a unifying diagnosis in this infant.17,18,20

Treatment is simple, inexpensive, and highly effective when instituted early. Oral elemental zinc at therapeutic doses can lead to visible improvement within days to one week, with progressive healing over subsequent weeks. Supportive skin care remains important because erosions and excoriations may be painful and can become secondarily infected. Nutritional counseling and follow-up are equally important to ensure adherence, monitor growth, and determine whether supplementation needs to be continued long term.12,13,19

Follow-up should be individualized. In a suspected transient or acquired case, clinicians may monitor clinical recovery, growth, recurrence, serum zinc, alkaline phosphatase, and nutritional status. If lesions recur when supplementation is stopped, if there is a family history or consanguinity, if symptoms are severe or persistent, or if there is no clear acquired explanation, evaluation for hereditary acrodermatitis enteropathica should be considered. When available, genetic testing and assessment of maternal/breast-milk zinc can help define etiology.7,8,14,15

The case also highlights the value of clinical imaging. Pretreatment photographs capture the severity and distribution of disease, while post-treatment photographs document the therapeutic response. In educational and publication settings, such visual sequences are powerful because they transform a rare diagnosis into a recognizable clinical pattern. However, because the patient is an infant, written informed consent from the parent or legal guardian is essential before publication of clinical images.18,20

The main limitation of this case report is that advanced etiological work-up was not included in the available material. Genetic testing for SLC39A4, maternal serum zinc, breast-milk zinc concentration, albumin level, inflammatory markers at the time of sampling, and long-term recurrence data were not provided. Despite these limitations, the diagnosis is strongly supported by the classical clinical distribution, low serum zinc, reduced alkaline phosphatase, and dramatic response to oral zinc supplementation.1,7,13

CONCLUSION

Acrodermatitis enteropathica/zinc deficiency dermatitis should be suspected in infants presenting with periorificial, acral, genital/perineal, and perianal dermatitis, especially when accompanied by diarrhea, alopecia, irritability, decreased feeding, or poor weight gain. This case demonstrates that a careful dermatologic examination, recognition of the typical lesion distribution, serum zinc estimation, and early therapeutic zinc supplementation can lead to rapid and complete recovery. The dramatic pretreatment-to-post-treatment clinical change emphasizes that zinc deficiency is not only diagnosable but also highly treatable when identified promptly.

For clinicians, the practical message is clear: do not repeatedly treat extensive periorificial-acral-diaper dermatitis as routine eczema or diaper rash when systemic clues are present. Zinc deficiency should be actively considered, investigated, and treated. Early diagnosis prevents prolonged discomfort, feeding impairment, growth compromise, recurrent infection, and unnecessary therapeutic delay.

Learning Points

- Periorificial, acral, genital, and perianal dermatitis in infancy should prompt consideration of zinc deficiency, particularly when diarrhea, alopecia, irritability, decreased feeding, or growth faltering coexist.
- The full triad of dermatitis, diarrhea, and alopecia may not be complete at presentation; clinicians should act on a suggestive pattern rather than wait for all features to appear.
- Low serum zinc with reduced alkaline phosphatase supports the diagnosis in a compatible clinical context.
- A rapid response to oral zinc supplementation is both therapeutic and diagnostically supportive.
- Exclusively breastfed infants can rarely present with zinc deficiency phenotypes; maternal, breast-milk, transient neonatal, and genetic mechanisms may need consideration during follow-up.

DECLARATIONS

Patient consent for publication: Clinical photographs of an infant are included in this manuscript. Written informed consent for publication of clinical details and images should be obtained from the parent/legal guardian before journal submission.

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Ethical approval: Case reports may not require formal institutional ethics committee approval; however, local institutional and journal policy should be followed.

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