ISSN: 2581 – 3633
PubMed - National Library of Medicine - ID: 101745081

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at:www.ijmacr.com

Volume – 8, Issue – 4, August - 2025, Page No.: 05 – 10

Zinc deficiency in pregnancy Mimicking Acrodermatitis enteropathica: A Case Report

[1]Manjusha Hegde, Post Graduate Resident, Department of Obstetrics and Gynaecology, Mahadevappa Rampure Medical College, Kalaburagi

[2]Suman Umeshchandara, Professor, Department of Obstetrics and Gynaecology, Mahadevappa Rampure Medical College, Kalaburagi

[3]Meenakshi S Devarmani, Professor and Head, Department of Obstetrics and Gynaecology, Mahadevappa Rampure Medical College, Kalaburagi

Corresponding Author: Manjusha Hegde, Post Graduate Resident, Department of Obstetrics and Gynaecology, Mahadevappa Rampure Medical College, Kalaburagi.

How to citation this article: Manjusha Hegde, Suman Umeshchandara, Meenakshi S Devarmani, "Zinc deficiency in pregnancy Mimicking Acrodermatitis enteropathica**:** A Case Report", IJMACR- August - 2025, Volume – 8, Issue - 4, P. No. 05 – 10.

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Background: Zinc is a vital micronutrient involved in numerous physiological processes, including immune function, epithelial integrity, and foetal development. During pregnancy, zinc deficiency can become pronounced due to increased maternal and foetal demands, sometimes mimicking inherited dermatological disorders such as acrodermatitis enteropathica (AE).

Case Presentation: We report a rare case of a 21-year-old primigravida at 27 weeks' gestation presenting with periorificial and acral dermatitis, alopecia, and persistent diarrhoea—clinical features classically seen in AE. Laboratory evaluation revealed significantly reduced serum zinc and alkaline phosphatase levels, with histopathology supporting a diagnosis of psoriasiform dermatitis. Imaging confirmed severe intrauterine growth restriction (IUGR). The patient responded rapidly to oral zinc supplementation and dietary modifications; however, due to worsening foetal Doppler findings, a caesarean section was performed at 32 weeks, delivering a growth-restricted but viable neonate.

Conclusion: This case illustrates that acquired zinc deficiency during pregnancy can closely mimic AE, underscoring the importance of clinical awareness and timely intervention. Prompt diagnosis and supplementation not only resolve maternal symptoms but may also mitigate adverse foetal outcomes, such as IUGR and prematurity.

Keywords: Foetal, Acrodermatitis Enteropathica, IUGR,

Introduction

Zinc is an essential trace element critical for numerous biological processes, including DNA synthesis, immune function, wound healing, and epithelial integrity. Deficiency of zinc, whether inherited or acquired, can lead to a spectrum of clinical manifestations, notably affecting the skin and gastrointestinal tract. **Acrodermatitis enteropathica (AE)** is a rare dermatological condition traditionally categorized into two forms: an autosomal recessive inherited form due to mutations in the SLC39A4 gene impairing intestinal zinc absorption, and an acquired form resulting from nutritional deficiencies or malabsorption syndromes. [1,2]

Clinically, AE presents with characteristic features such as periorificial and acral dermatitis, alopecia, and diarrhoea, often accompanied by systemic symptoms including irritability and growth retardation in children. [1,3] While the inherited form typically presents in infancy following cessation of breastfeeding, acquired zinc deficiency can occur at any age and is particularly relevant during periods of increased physiological demand, such as pregnancy.[2,4] In pregnant women, altered zinc metabolism, poor dietary intake, and increased foetal requirements may precipitate zinc deficiency, occasionally mimicking the classical phenotype of AE.[4]

Accurate diagnosis relies on a combination of clinical recognition, low serum zinc levels, and prompt therapeutic response to zinc supplementation. [3,4] Misdiagnosis or delayed treatment may result in significant maternal and foetal complications. This report presents a rare case of acquired zinc deficiency in pregnancy presenting with clinical features reminiscent of acrodermatitis enteropathica, emphasizing the importance of differential diagnosis and timely management.

Case

A 21-year-old primigravida at 27 weeks and 6 days of gestation presented with a five-month history of painful, erythematous, scaly, and crusted lesions localized around the mouth, perineum, and extremities. She also reported progressive hair loss and persistent diarrhoea. Her medical history was unremarkable, with no prior gastrointestinal or dermatologic disorders.

On physical examination, she exhibited well-demarcated, erythematous, scaly plaques with crusting in the periorificial regions (perioral, perinasal, perianal) and acral areas (dorsum of hands and feet). Diffuse alopecia was noted, along with signs of delayed wound healing. Neurologically, she was mildly irritable and fatigued, but no focal deficits were observed. Abdominal examination revealed a uterus corresponding to a gestational age of approximately 24 weeks, and foetal heart rate was 142 beats per minute.

Laboratory investigations showed a significantly reduced serum zinc level of 30.4 µg/dL (reference range: 60–120 µg/dL) and a low alkaline phosphatase (ALP) level of 44 IU/L. C-reactive protein (CRP) was elevated at 19.1 mg/L. Other investigations, including complete blood count with peripheral smear, liver and renal function tests, and viral markers, were within normal limits. Nutritional deficiencies such as iron, folate, and vitamin B12 were ruled out. Stool studies did not reveal any evidence of malabsorption.

A skin biopsy demonstrated features consistent with psoriasiform dermatitis. An antenatal ultrasound revealed a single live intrauterine foetus in a changing lie, corresponding to a gestational age of 24 weeks and 3

days, with evidence of severe intrauterine growth restriction (BPD, HC, AC, FL all < 3rd percentile). The cerebroplacental ratio was 1.4 (11th percentile), and Doppler studies were within normal range but raised concerns about foetal compromise.

Based on the constellation of clinical findings and laboratory evidence, a diagnosis of acquired zinc deficiency mimicking acrodermatitis enteropathica, complicated by severe intrauterine growth restriction (IUGR), was established.

The patient was commenced on oral zinc supplementation with 50 mg of elemental zinc daily, along with dietary modifications emphasizing zinc-rich foods such as lean meats, dairy products, and legumes. Within a few days, her skin lesions began to heal, and gastrointestinal symptoms improved markedly.

Despite maternal improvement, foetal growth remained severely restricted. Due to deteriorating Doppler findings, she underwent caesarean delivery at 32 weeks of gestation. She delivered a 1.05 kg neonate with no congenital anomalies. The newborn required admission to the neonatal intensive care unit (NICU) for management of prematurity-related complications.

Figure 1A: Lateral view of scalp showing patchy alopecia, follicular hyperkeratosis, and scaly plaques involving the temporal region.

Figure 1B: Posterior view of scalp demonstrating diffuse scaling and lichenified plaques suggestive of zinc deficiency-related dermatitis.

Figure 2A: Palmar view showing erythematous, fissured plaques with crusting and scaling over the fingers and palms, indicative of acral dermatitis in zinc deficiency.

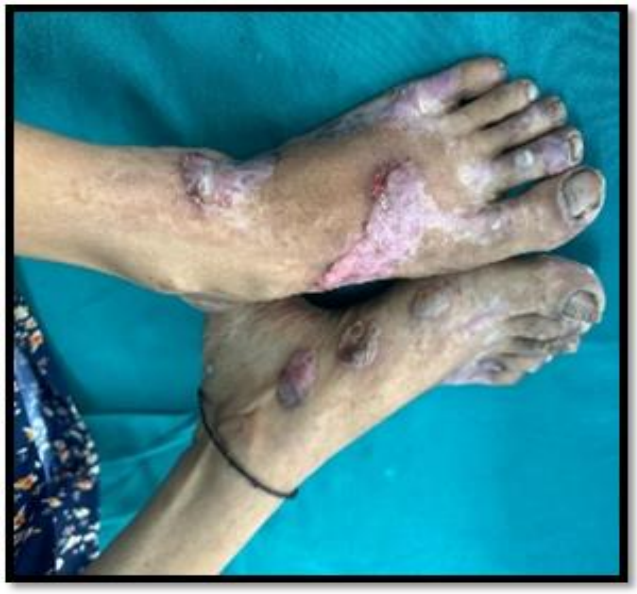

Figure 2B: Dorsal aspect of feet displaying erythematous, scaly, and crusted plaques with erosions and secondary infection—typical acral involvement in acquired acrodermatitis enteropathica.

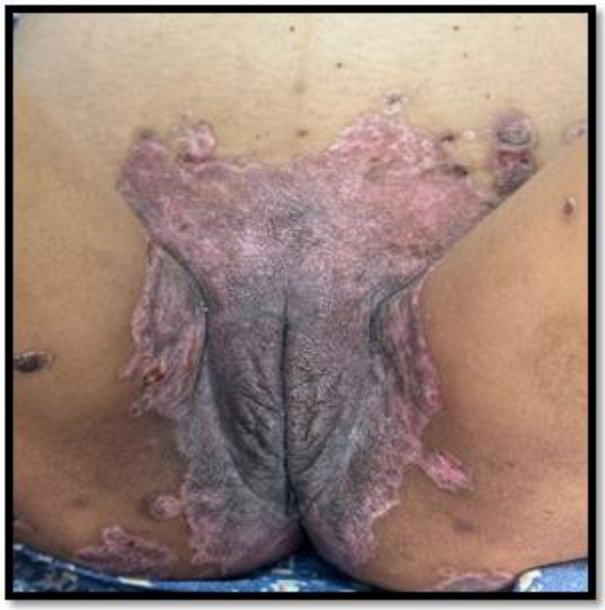

Figure 3A: Perianal region displaying sharply demarcated, erythematous to violaceous plaques with scaling and maceration—characteristic periorificial involvement seen in zinc deficiency.

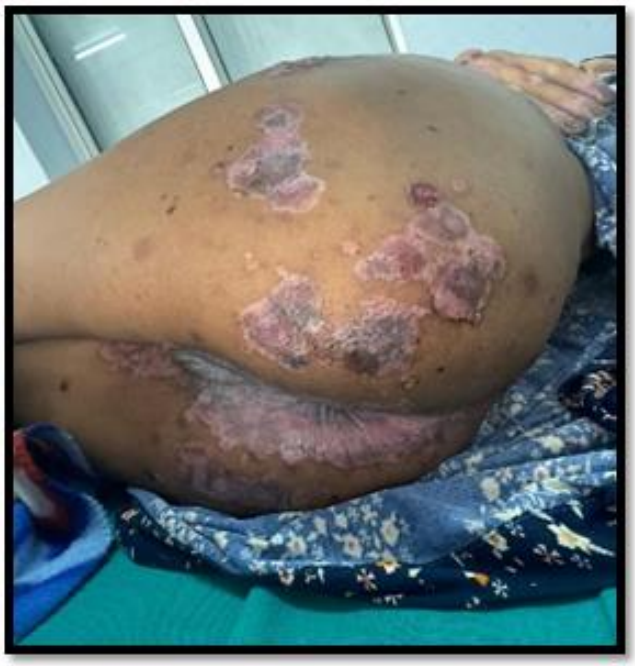

Figure 3B: Gluteal region with multiple well-defined, hyperpigmented and scaly plaques showing erosions and crusting—consistent with chronic zinc-deficiency dermatitis.

Discussion

Zinc deficiency, although often underrecognized, plays a critical role in a wide range of dermatological, gastrointestinal, immunological, and obstetric manifestations. In this case, the patient—a young primigravida—presented with classic dermatological signs and systemic symptoms that closely mimicked acrodermatitis enteropathica (AE), a condition primarily known for its inherited form but also seen in acquired settings.

Acrodermatitis enteropathica is classically described as a triad of periorificial and acral dermatitis, alopecia, and diarrhoea, all of which were present in this patient. These features are consistent with both inherited and acquired zinc deficiency, though the latter is more commonly associated with adults and states of increased physiological demand, such as pregnancy.[4,5] Notably,

pregnancy increases maternal zinc requirements significantly, often resulting in marginal or deficient zinc status, particularly in women from low-resource settings or with poor nutritional intake.[9]

In this case, the patient had no history of malabsorption syndromes or chronic illnesses, and common nutritional deficiencies such as iron, folate, and B12 were ruled out. These findings aligned well with the case by Saritha et al., where acquired zinc deficiency occurred in an adult female without identifiable gastrointestinal pathology. [4]

Skin findings such as erythematous, scaly plaques with crusting in periorificial and acral regions, and psoriasiform changes on biopsy, have been previously documented in the literature and are highly characteristic of AE.[5–7] The pathognomonic nature of these dermatological lesions, along with supporting low serum zinc and ALP levels, guided the clinical diagnosis in this case.

The systemic manifestations of zinc deficiency, including alopecia, fatigue, irritability, and delayed wound healing, stem from zinc's critical role in enzymatic activity, immune modulation, and epithelial maintenance.[6,8] These signs, although nonspecific, often co-occur with the dermatologic findings and support a diagnosis when considered in the clinical context.

One of the most important and concerning aspects of this case was the impact of maternal zinc deficiency on foetal well-being. The foetus exhibited severe intrauterine growth restriction (IUGR), likely due to compromised placental zinc transfer, which has been previously associated with adverse perinatal outcomes including low birth weight and prematurity.[9] Zinc plays a key role in DNA synthesis, cell division, and angiogenesis, all vital for foetal development. In line with this, Karimi et al. demonstrated that zinc supplementation during pregnancy improved foetal outcomes, emphasizing its importance in prenatal care.[9]

Although the patient responded favourably to oral zinc supplementation and dietary interventions—with notable improvement in her skin and gastrointestinal symptoms—the persistent IUGR and abnormal Doppler findings necessitated early delivery. Such foetal compromise despite maternal clinical improvement has been noted in prior case reports, where the timing of supplementation relative to gestational age appeared to play a pivotal role in outcomes.[9]

This case underscores the need for early recognition of zinc deficiency in pregnant women, especially when presenting with suggestive dermatologic or gastrointestinal symptoms. Given the rapid therapeutic response to zinc, a high index of suspicion can facilitate timely intervention and potentially prevent foetal morbidity.

Conclusion: Acquired zinc deficiency in pregnancy can mimic acrodermatitis enteropathica, presenting with characteristic cutaneous, gastrointestinal, and neuropsychiatric symptoms. This case highlights the potential for severe IUGR as a consequence of prolonged zinc deficiency. Early recognition and supplementation are crucial in improving maternal health and optimizing fetal outcomes. Close fetal monitoring is essential in such cases to ensure timely intervention and delivery when necessary.

References

1. Acrodermatitis enteropathica, J Am Acad Dermatol, 1983 Jul;9(1):140-4.
2. Maverakis E, Fung MA, Lynch PJ, Draznin M, Michael DJ, Ruben B, et al. Acrodermatitis enteropathica and an overview of zinc metabolism. J Am Acad Dermatol 2007;56:116-24.

3. Perafán-Riveros C, França LF, Alves AC, Sanches JA Jr.Acrodermatitis enteropathica: Case report and review of the literature. Pediatr Dermatol 2002;19:426-31.
4. Saritha M, Gupta D, Chandrashekar L, Thappa DM, Rajesh NG. Acquired zinc deficiency in an adult female. Indian J Dermatol 2012;57:492-4 .
5. Habib S, Moin A, Jeelani SM. Acrodermatitis Enteropathica: A Rare Case Report and Review of Literature. Int J Contemp Med Res. 2016;3(6):1755–1756.
6. Kumar M, Raghunath D, Mathew A. Acrodermatitis Enteropathica: A Case Report. Indian J Clin Pract. 2014;25(6):613–615.
7. Mittal R, Sudha R, Murugan S, Adikrishnan, Shobana S, Anandan S. Acrodermatitis enteropathica – A case report. Sri Ramachandra J Med. 2007;1(2):57–59.
8. Crowley N, Lai-Cheong J. A case of a rapid skin eruption caused by zinc deficiency. Infant. 2014;10(6):194–195
9. Karimi A, Bagheri S, Nematy M, Saeidi M. Zinc deficiency in pregnancy and fetal-neonatal outcomes and impact of the supplements on pregnancy outcomes. Iranian Journal of neonatology. 2012;3(2):77-83.