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# Severe, Rapidly Progressive Seborrheic Dermatitis in an Infant: Recognizing Atopic Overlap and Management Challenges

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**Title:** Severe, Rapidly Progressive Seborrheic Dermatitis in an Infant: Recognizing Atopic Overlap and Management Challenges

**Abstract:**

Infantile seborrheic dermatitis is typically a benign, localized condition; however, severe and widespread presentations are uncommon, especially if presentation may overlap with atopic dermatitis. This is the case of a 3-month-old Filipino male with rapidly progressive seborrheic dermatitis with strong family history of atopy. Initially limited to the scalp at 2 months, the eruption progressed to diffuse facial involvement by 3 months, with extensive erythema, scaling, crusting, and auricular involvement. This was accompanied by excoriations and significant pruritus disrupting sleep. Despite treatment with topical antifungal therapy, symptoms worsened. The child also demonstrated generalized dry, eczematous-appearing skin in flexural areas and dry, flaky skin on extremities. Family history was notable for a childhood-onset, self-resolving dermatologic condition in first-degree relatives, father and paternal aunt, raising concern for underlying atopy.

We hypothesize that impaired skin barrier function associated with atopic predisposition, characterized by increased transepidermal water loss and enhanced susceptibility to microbial colonization, facilitated an exaggerated inflammatory response to *Malassezia*, contributing to the rapid spread and severity of disease. This case highlights the importance of recognizing atypical or extensive seborrheic dermatitis in infants with possible barrier dysfunction. Although disease severity in this case warranted the use of topical corticosteroids, clinicians should remain mindful of associated risks in young infants. Early pediatric referral is recommended for close

monitoring and timely evaluation of steroid-sparing therapies, such as topical calcineurin inhibitors, in managing widespread inflammation.

**Key Words:** atopic dermatitis, seborrheic dermatitis, skin barrier, infantile seborrheic dermatitis

**Introduction:**

Infantile seborrheic dermatitis is a common chronic inflammatory skin condition that typically presents within the first few months of life. It is characterized by symmetric, poorly demarcated erythematous patches with yellow, greasy scales and fine desquamation, most often involving sebaceous gland–rich areas such as the scalp (cradle cap), face, and intertriginous regions[1]

Although generally benign and self-limited, with most cases resolving by 6 months of age, more severe presentations can lead to significant parental distress and may overlap clinically with atopic dermatitis, complicating both diagnosis and management[1]. Additionally, Severe, rapidly progressive, and widespread seborrheic dermatitis in early infancy is uncommon and may signal underlying skin barrier dysfunction or atopic predisposition.

The pathophysiology is thought to involve an inflammatory response to *Malassezia* species colonization, although the precise mechanisms remain incompletely understood[2].

Management typically includes gentle skin care, emollients, and topical antifungal agents, with low-potency topical corticosteroids reserved for cases with significant inflammation[1]

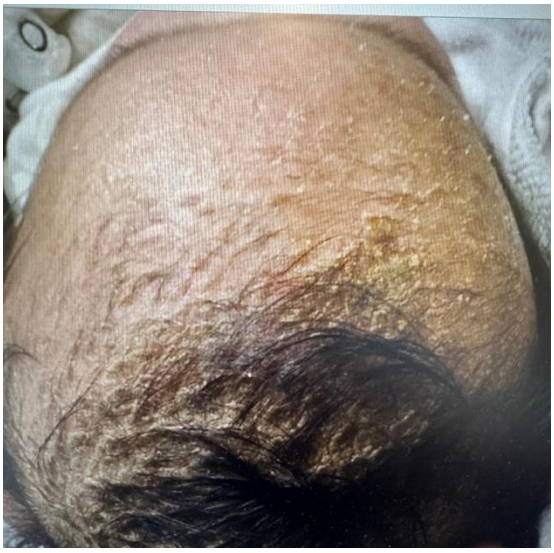
However, when involvement is extensive, concerns regarding systemic absorption and potential adverse effects of topical corticosteroids become particularly important, especially in infants due to their increased body surface area–to–weight ratio[3,4].

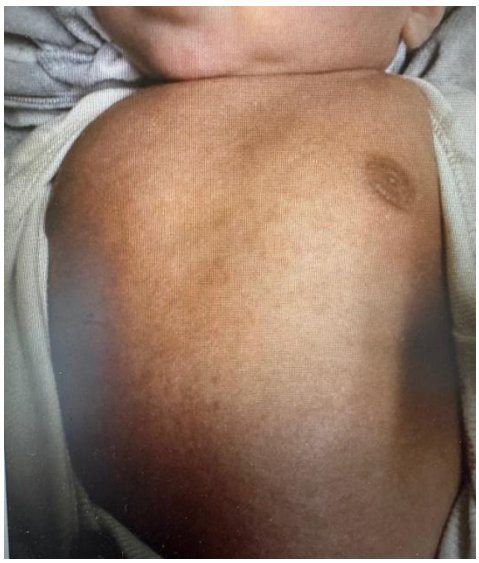
In addition, severe pruritus may significantly disrupt sleep and negatively impact caregiver quality of life, even when the infant appears clinically well during evaluation. This case report describes an infant with severe, widespread seborrheic dermatitis complicated by underlying eczema and significant pruritus. It highlights the diagnostic challenges, the broader impact on patient and family well-being, and the therapeutic considerations required when extensive skin involvement necessitates a multimodal treatment approach.

**Case Description:**

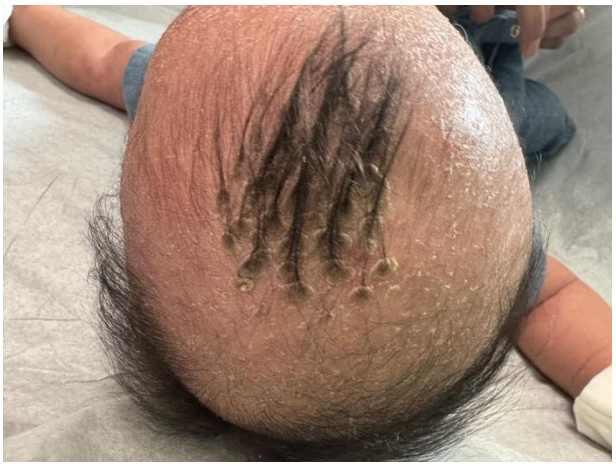
Treatment in order:

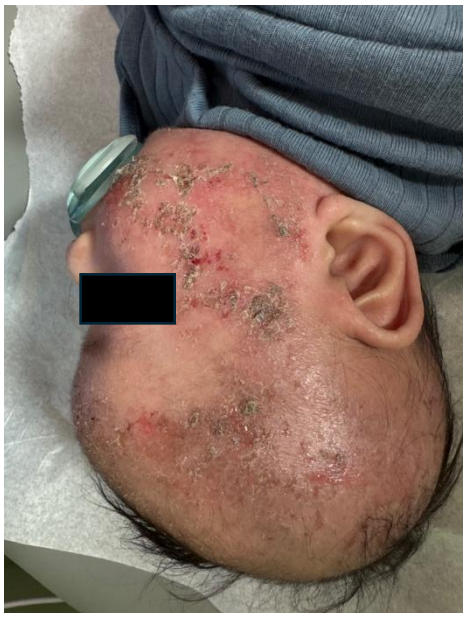
1. Initial visit: Ketoconazole topical shampoo?

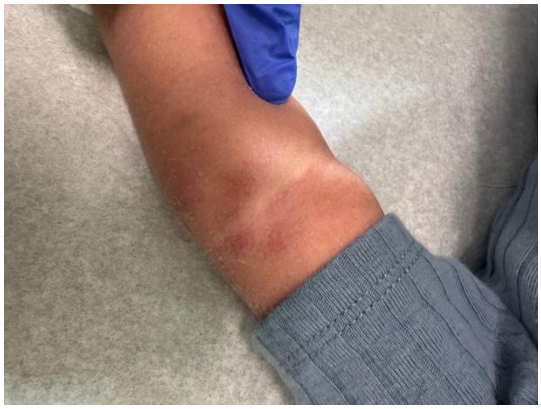


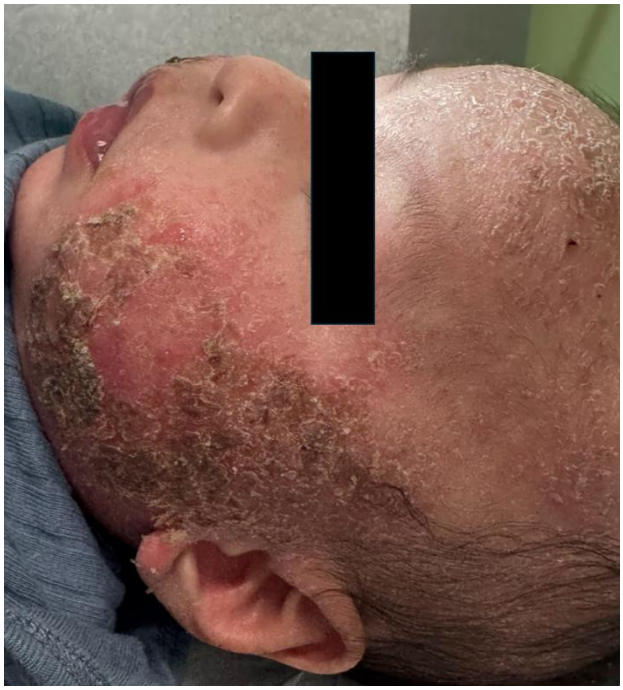


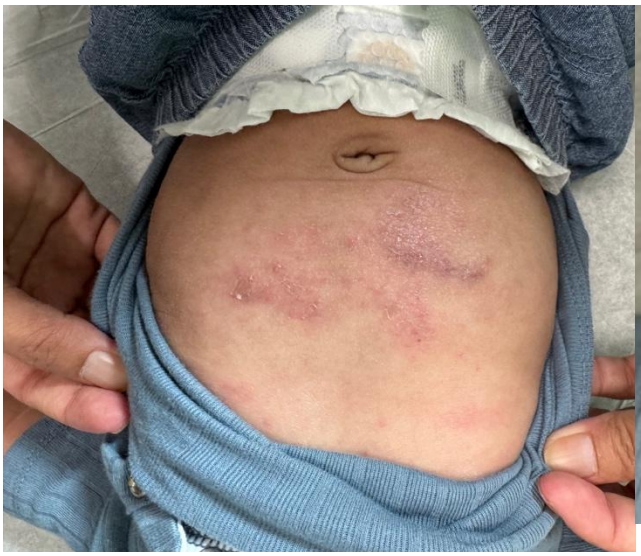
2. 3 month visit: Muporicin given to address open lesions either from scratches or inflamed lesions.

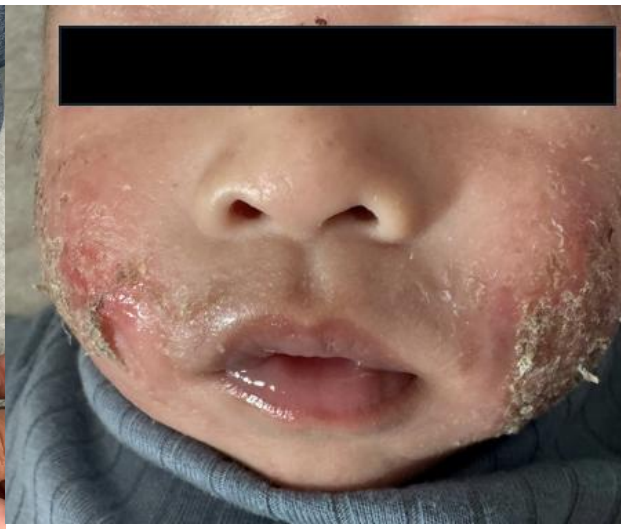




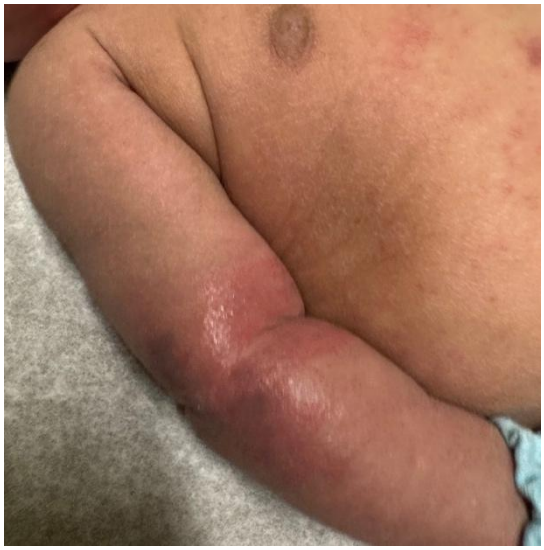


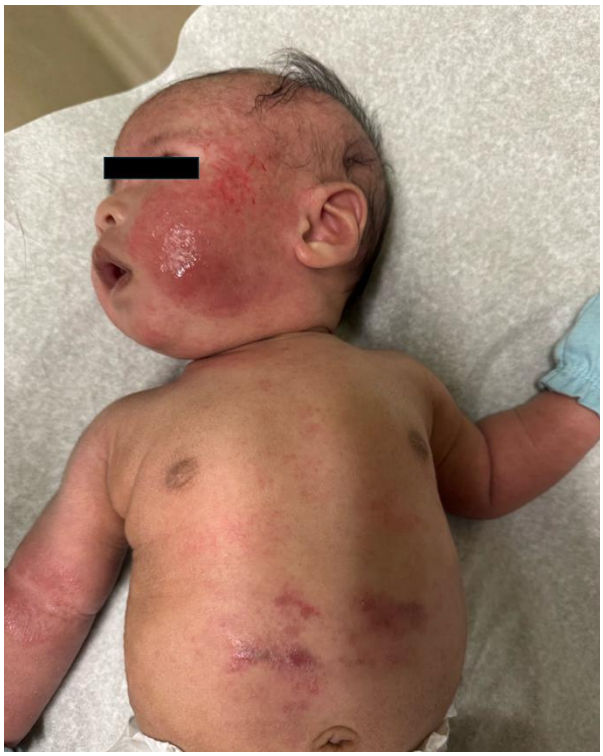


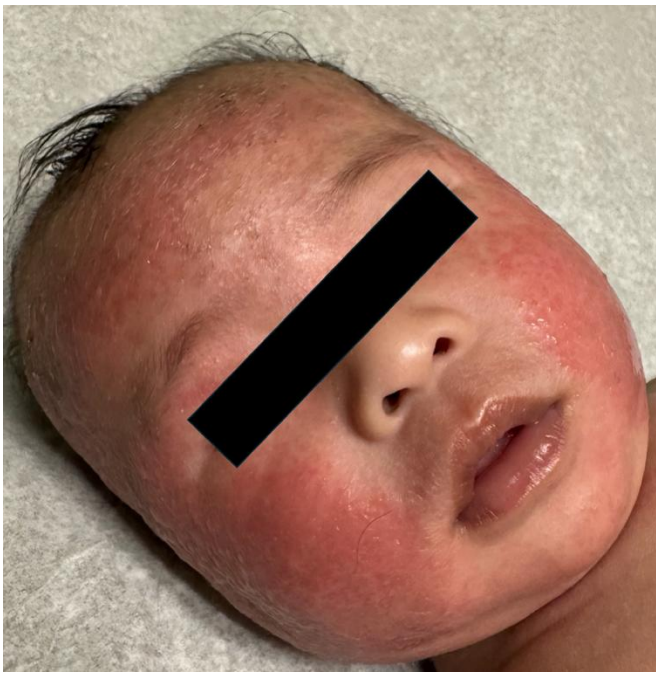




3. 3 day f/u after 3 month visit:
    a. Triamcinolone 0.025% for below the neck BID for 3 weeks
    b. Desonite 0.05% for head and neck BID for 3 weeks
    c. Urgent referral to pediatric dermatology to evaluate calcineurin inhibitors







A 3-month-old Filipino male, born full-term via vaginal delivery to a first-time mother, presented with progressive skin changes. The infant had been formula-fed since 3 weeks of age following treatment for neonatal hyperbilirubinemia with phototherapy. His medical history was otherwise unremarkable. Mongolian spots were noted on the back and lower extremities. Growth parameters, including weight and length, were appropriate for age and followed normal percentiles. There were no associated systemic symptoms, including fever, feeding difficulties, or changes in bowel or urinary habits. The infant remained playful and well-appearing during examinations, although the mother reported increased nighttime irritability and scratching suggestive of pruritus that disrupted sleep.

At 2 months of age, the patient initially presented with a mild cradle cap and small areas of dry skin on the face. These findings were consistent with mild infantile seborrheic dermatitis and xerosis. He was treated with topical ketoconazole and advised to use an emollient (CeraVe) for generalized dry skin.

Over the subsequent four weeks, the rash progressed significantly. At follow-up at 3 months of age, the dermatitis had become widespread, involving the face, cheeks, and auricles. The affected areas demonstrated yellow, flaky, and crusted plaques with underlying excoriations, particularly over the cheeks and temporal scalp. The mother reported significant pruritus, with frequent scratching and sleep disruption, noting that symptoms appeared worse at night. She stated that the rash had been more severe approximately two weeks prior but had shown slight partial improvement.

A bacterial swab was obtained from the open lesions, which grew gram-positive cocci in clusters, consistent with *Staphylococcus aureus*. Topical mupirocin was initiated for localized treatment of the excoriated areas to prevent further secondary infection. Cultures returned many gram-positive clusters however due to lack of systemic symptoms and signs of infection in addition to the expectancy of staph aureus as part of the normal skin flora, further treatment for this concern was not pursued.

At follow-up three days later, there was improvement in flaking and healing of open lesions; however, the skin appeared diffusely erythematous, increasingly inflamed, and raw, with persistent plaques on the scalp. Given the extent of inflammation and ongoing pruritus, treatment was escalated to include topical corticosteroids: triamcinolone 0.025% applied twice daily to affected areas below the neck and desonide 0.05% applied twice daily to the head and neck for a planned duration of three weeks. An urgent referral to pediatric dermatology was placed for further evaluation and management.

At the time of this report, clinical resolution remains ongoing.

**Discussion:**

Infantile seborrheic dermatitis (ISD) is a common condition in early infancy that is typically mild, localized, and self-limited[5]. It most often presents as cradle cap with occasional extension to the face or intertriginous areas and usually responds well to conservative measures such as emollients and topical antifungals[5]. In contrast, this case illustrates an atypical and severe presentation, with rapid progression from localized scalp involvement to widespread disease

affecting the face, ears, and trunk, accompanied by significant inflammation, excoriation, and secondary bacterial infection.

A key feature in this case is the presence of generalized dry, eczematous-appearing skin along with a family history suggestive of childhood skin disease, raising concern for underlying atopy. While ISD and atopic dermatitis are classically described as distinct conditions, overlap in infancy is increasingly recognized[5]. In patients with impaired skin barrier function, increased trans epidermal water loss and altered microbial colonization may predispose to both inflammation and disease spread[5]. In this setting, the inflammatory response to *Malassezia*—implicated in seborrheic dermatitis—may be amplified, contributing to the unusually extensive and severe presentation observed[2].

This amplified inflammatory response in susceptible infants may be further driven by several factors, including underlying skin barrier dysfunction associated with atopy[7], secondary bacterial colonization or infection (such as *Staphylococcus aureus*)[6], and mechanical irritation from scratching in the setting of pruritus. Together, these factors may create a cycle of inflammation, barrier disruption, and disease propagation, distinguishing more severe presentations from typical, self-limited seborrheic dermatitis.

Barrier dysfunction may also increase susceptibility to secondary infection[6]. The presence of excoriations and significant pruritus, severe enough to disrupt sleep, further supports a component of atopic overlap, as pruritus is typically less prominent in isolated seborrheic dermatitis[4].

This case also highlights important management considerations for primary care providers. Initial treatment with topical antifungal therapy alone may be insufficient in more severe or mixed presentations[7]. Although the degree of inflammation in this case ultimately warranted the use of topical corticosteroids, clinicians must carefully weigh the risks of systemic absorption in infants, particularly when large surface areas are involved[8]. Infants have an increased body surface area–to–weight ratio, which increases the risk of systemic corticosteroid exposure and potential adverse effects such as hypothalamic-pituitary-adrenal axis suppression and skin atrophy[8].

Early recognition of atypical or rapidly progressive seborrheic dermatitis, especially in infants with evidence of dry skin or possible atopy, is crucial. Prompt referral to pediatrics or dermatology may allow for closer monitoring and consideration of steroid-sparing therapies, such as topical calcineurin inhibitors[9], which may be more appropriate for managing widespread inflammation while minimizing potential adverse effects.

In patients with persistent or severe disease and features suggestive of atopic predisposition, further evaluation for underlying atopic dermatitis may be warranted[4]. Referral to an allergist for assessment of potential environmental or contact triggers may be considered, particularly in cases with recurrent or refractory symptoms.

Overall, this case underscores the need for increased vigilance in infants presenting with seborrheic dermatitis that is extensive, inflammatory, or refractory to standard therapy, as early intervention may help prevent complications and improve patient and caregiver outcomes.

**Conclusion/Clinical Takeaway:**

This case highlights an atypical presentation of infantile seborrheic dermatitis characterized by rapid progression, widespread involvement, significant inflammation, and secondary bacterial infection. While infantile seborrheic dermatitis is typically mild and self-limited, presentations that are widespread, progressive, or highly inflammatory should prompt reconsideration of the diagnosis and closer clinical follow-up. The presence of generalized dry or eczematous-appearing skin in this patient suggests underlying skin barrier dysfunction and possible overlap with atopic dermatitis, which may contribute to an amplified inflammatory response and increased disease severity. Additionally, marked pruritus, excoriations, and crusted lesions should raise concern for secondary bacterial infection, as seen in this case with *Staphylococcus aureus*.

Management in such cases may require escalation beyond topical antifungal therapy alone. Although topical corticosteroids may be necessary to control significant inflammation, clinicians must carefully weigh the risks of systemic absorption and adverse effects in infants, particularly when treating large surface areas. Early referral to pediatric dermatology is important in atypical or refractory cases to guide management and evaluate the use of steroid-sparing therapies such as topical calcineurin inhibitors.

Finally, in infants with persistent or severe disease and features suggestive of atopy, further evaluation for underlying atopic dermatitis and potential triggers may be warranted, including referral to an allergist when appropriate. Recognition of this clinical pattern is important, as it may signal an increased risk for chronic skin conditions into childhood and adolescence,

underscoring the need for ongoing monitoring, anticipatory guidance, and early intervention strategies.

**Long-term implication**

Severe or atypical presentations of infantile seborrheic dermatitis, particularly in the setting of suspected skin barrier dysfunction, may represent an early manifestation of underlying atopic disease. Recognition of this pattern is important, as it may signal an increased risk for chronic skin conditions into childhood and adolescence, underscoring the need for ongoing monitoring, anticipatory guidance, and early intervention strategies.

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