

Infantile Seborrheic Dermatitis

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GOAL

To understand infantile seborrheic dermatitis (ISD) to better treat patients with the condition

OBJECTIVES

Upon completion of this activity, dermatologists and general practitioners should be able to:

1. Describe the etiology of SD.
2. Recognize and diagnose SD.
3. Effectively treat ISD.

CME Test on page 292.

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Dr. Elish reports no conflict of interest. Dr. Silverberg is an investigator and speaker for Astellas Pharma Inc, and Novartis Pharmaceuticals Corporation. The authors discuss off-label use of ketoconazole 2% shampoo, sodium sulfacetamide 10% solution, topical calcineurin inhibitors (pimecrolimus and tacrolimus), and topical corticosteroid lotions. Dr. Fisher reports no conflict of interest.

Seborrheic dermatitis (SD) is one of the most common dermatoses of infancy. SD is an inflammatory process that presents as tiny papules covered by scales typically localized to the seborrheic region. We report a case of a 2-month-old infant with SD who went on to develop atopic dermatitis (AD). Additionally, we discuss epidemiology, etiology, diagnosis, differential diagnosis, and treatment modalities for SD, as well as an association of SD and AD.

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Case Report

A 2-month-old white infant presented with diffuse hyperkeratosis of the scalp of 2 weeks' duration. He also had fine macerated erythema of the retroauricular area, neck, axillae, and groin. These lesions were consistent with a clinical diagnosis of infantile seborrheic dermatitis (ISD). Application of mineral oil to the scalp resulted in softening and improvement of scalp lesions. The body lesions were ameliorated by the application of a mixture of hydrocortisone and nystatin creams to the neck, axillae, and groin. The lesions recurred, requiring periodic reapplication of the medicaments. Eventually, the lesions occurred less frequently and the scalp lesions resolved completely over the next 2 months. However, the patient developed typical atopic dermatitis (AD) as a 6-month-old, typified by erythematous excoriated plaques in the antecubital and popliteal regions.

Comment

SD was first described by Unna in 1887.¹ SD is a common chronic inflammatory disease characterized by erythema accompanied by greasy scales in the so-called seborrheic region, which includes the scalp, forehead/glabella, eyebrows, malar eminences, paranasal and nasolabial folds, retroauricular area, chest, and axillae. SD occurs most frequently in infants and adults aged 30 to 60 years. Its prevalence in immunocompetent adults is estimated to be between 1% and 3%.² The incidence of SD is unusually high among patients with AIDS, ranging from 30% to 83%.²⁻⁴ There also is an increased incidence of SD in patients with tinea versicolor, depression, spinal cord injuries, and parkinsonism, and in patients receiving psoralen and UVA therapy.⁵⁻⁹

SD usually develops in neonates within the first 3 to 4 weeks of life. Spontaneous recovery generally occurs at about 6 to 7 months of age, though persistence until 2 years of age can be seen. SD in adults affects men more often than women; ISD shows no gender predilection. The occurrence of SD in prepubertal children (aged 2–5 years) is uncommon.

The etiology of SD is poorly understood. SD may be hormonally dependent, which could explain why the condition appears briefly in infancy and recurs in puberty. The role of sebum excretion in the pathogenesis of SD is controversial. In fact, sebum excretion has been shown to be either normal or subnormal in many patients with SD.^{10,11} Commensal yeast *Malassezia* also has been thought to be causative.¹² The response of SD to topical antifungal agents such as ketoconazole and selenium sulfide indicates that *Malassezia* yeast may be pathogenic. Research suggests that SD is not caused by an overgrowth of *Malassezia* but an abnormal host response.¹² The evidence supporting this theory lies in the increased incidence of SD in immunocompromised patients. In a study of fatty acids in the serum of infants with ISD, Tolleson et al¹³ demonstrated evidence of impaired function of the enzyme δ -6-desaturase, which desaturates linoleic acid to dihomogammalinolenic and arachidonic acids. The study indicated the function of the enzyme appeared to normalize in the infants by about 6 to 7 months of age, the age at which spontaneous recovery from ISD usually occurs.¹³

ISD is a self-limited process that usually involves the scalp. The scalp lesions can present as small dry patches of hyperkeratosis overlying mildly erythematous skin that may become so thickened that it forms a cap, meriting its description as cradle cap (Figure).¹⁴ Scalp hyperkeratosis often is the only manifestation of ISD and usually appears 3 or 4 weeks after birth.^{15,16} The scales may be white,



Cradle cap-type scale in a 2-month-old infant.

off-white, or yellowish. The central part of the face; forehead; neck; ears; and intertriginous areas such as the axillae, groin, and inner thigh folds, also may be involved. SD begins as erythematous macules and papules that gradually become confluent to form scaly patches and slightly elevated plaques.^{15,16} In adolescents, SD has a clinical picture similar to ISD but is focused in the head and neck region.

The diagnosis of ISD usually is straightforward and is based on clinical findings about the distribution and appearance of the lesions. However, failure to respond to therapy must lead clinicians to reconsider the diagnosis.¹⁵ ISD must be differentiated from AD, psoriasis, and tinea capitis. ISD and AD have similar sites of predilection including the face, scalp, retroauricular area, diaper area, and extensor limb surfaces. The distinction is made on clinical grounds. Axillary and anterior neck involvement favors the diagnosis of ISD, as do the lack of evidence of pruritus and the absence of oozing and weeping. Infants with AD tend to be aged 3 to 12 months and usually have at least one parent or sibling with a positive history of atopy. Sometimes, however, overlap of ISD and AD can be seen, particularly in infants aged 2 to 6 months.^{15,16}

Our patient went on to develop AD. The relationship between ISD and infantile AD (IAD) is controversial. According to some authors, more than 50% of children with widespread ISD have or will develop AD.¹⁷ Conversely, Moises-Alfaro et al¹⁸ conducted a small and not as convincing study that led them to conclude that there is no relationship between ISD and IAD. Our patient supports the association of IAD and ISD. Recent studies have demonstrated that patients with head and neck AD have immunoglobulin E antibodies to *Malassezia furfur*, the yeast causative of ISD. This supports the overlap and possible progression between IAD and ISD through sensitization to cutaneous *Malassezia*.

Inflammatory reaction to *Malassezia* (ie, ISD) may be the inciting event in the development of IAD, though this has not been proven so far in children.^{19,20}

Occasionally, psoriasis has predilection for seborrheic areas (inverse psoriasis), making it difficult to clinically decide whether the patient has psoriasis or SD; however, psoriasis is more sharply demarcated.²¹ Rarely, both appear concurrently. In rare cases, infants are affected with a scaling eruption resembling ISD on the scalp in association with fever and other systemic signs of acute disseminated Langerhans cell histiocytosis.¹⁵ Persistent erythematous scaling (especially if hemorrhagic and therapy resistant) in an infant who is doing poorly or has hepatosplenomegaly requires a biopsy to exclude Langerhans cell histiocytosis.

Severe treatment-resistant SD may be associated with human immunodeficiency virus infection and is common in infants who develop human immunodeficiency virus–related immune suppression in the first year of life.²²⁻²⁴ As the immune deficiency in these patients becomes progressively worse, so does SD. SD occasionally progresses to erythroderma, a cradle cap of scales sometimes associated with non-scarring alopecia or postinflammatory hyperpigmentation or hypopigmentation.¹⁵

In prepubertal children, AD or tinea capitis are more likely diagnoses for hyperkeratotic scalp lesions than SD; therefore, tinea capitis must be excluded by a fungal culture of the scalp.^{25,26} When SD is diagnosed in a prepubertal child, precocious puberty should be suspected. However, AD is a more likely diagnosis for scalp hyperkeratosis, but it is not impossible to see SD in prepubertal children.²⁷

In most instances, the diagnosis of SD is clinically obvious. When the diagnosis is not so obvious, a biopsy may be necessary to differentiate SD from other skin diseases by histologic examination. Sections of tissue of the biopsy specimens show characteristic changes, namely superficial perivascular and interstitial infiltrates of lymphocytes, slight spongiosis, scale crusts and mounds of parakeratosis that reside at the lips of infundibular ostia and at interinfundibular sites, markedly dilated venules and capillaries of the superficial plexus, and psoriasiform hyperplasia in more long-standing lesions of SD.^{21,28}

Therapy

Therapy for SD is based on the age of the patient and the extent of the disease. The usual therapeutic approach for ISD of the scalp is conservative. In mild cases, an emollient such as white petrolatum or mineral oil may be used to soften the cradle cap so that it can be gently removed by brushing off the scales.^{14,15} Crusts are soaked overnight with slightly warmed oil

and washed off in the morning. A mild nonmedicated shampoo should be used at the start of therapy in conjunction with brushing off scales with a baby's toothbrush. If a mild shampoo is not helpful, a shampoo containing ketoconazole 2% can be used.^{14,29} Coal tar–based shampoos must be avoided because of the carcinogenicity of coal tar.³⁰ Mild topical corticosteroid lotions can be used adjunctively to reduce erythema of the scalp. Salicylic acid shampoos are contraindicated in ISD because of concerns about percutaneous absorption of the substance and the risk of metabolic acidosis and salicylism.³¹

ISD involving intertriginous areas is treated with gentle skin care and topical medicaments. Topical ketoconazole or nystatin are safe and effective therapies, particularly when combined with a mild topical corticosteroid.³² Topical tacrolimus ointment or pimecrolimus cream can be substituted for a topical corticosteroid; however, the use of tacrolimus and pimecrolimus is off-label and should not be used in children younger than 2 years, according to the US Food and Drug Administration.³³ Calcineurin inhibitors are used in topical corticosteroid–resistant AD patients 2 years and older. Similar guidelines are prudent for SD therapy. Recently, the US Food and Drug Administration issued a warning regarding a biologic potential for skin cancers and lymphomas with the use of topical calcineurin inhibitors; however, human data have not supported these risks.³³

Adolescents with SD should be treated similar to adults. Because SD is chronic, the initial therapy for the condition should be followed by a maintenance regimen. Conventional therapy for SD of the scalp is the use of a medicated shampoo 2 to 3 times per week. Shampoos containing salicylic acid, selenium sulfide, an antifungal agent, or zinc pyrithione are effective.¹⁵ In more severe cases, a topical corticosteroid in a lotion, oil, or solution base may be used once or twice daily, often in addition to a medicated shampoo.

Seborrheic blepharitis is managed by the gentle removal of scales and crusts using a cotton ball dipped in diluted baby shampoo.¹⁵ In severe cases involving the eyelids, the eyelids may be covered with sodium sulfacetamide 10% solution or ketoconazole 2% cream.^{14,15} In our experience, nonsteroidal anti-inflammatory preparations, such as tacrolimus ointment and pimecrolimus cream, also can be used safely on the eyelids in children under the same guidelines as other cutaneous application sites.

Conclusion

In summary, a number of factors such as immune function and heredity are important in the pathogenesis

of SD. The role of *Malassezia* in SD needs to be clarified. In most instances, SD is easily diagnosed on clinical grounds alone. Safe and effective treatment modalities are available. More studies are needed to determine whether a relationship between SD and AD exists; however, our clinical experience supports this associator.

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