Seborrhoeic dermatitis: An overview

Schwartz RA, MD MPH
Janusz CA, MD
Janniger CK, MD
University of Medicine and Dentistry at New Jersey-New Jersey Medical School, Newark, New Jersey

Abstract

Seborrhoeic dermatitis affects the scalp, central face, and anterior chest. In adolescents and adults, it often presents as scalp scaling (dandruff). Seborrhoeic dermatitis also may cause mild to marked erythema of the nasolabial fold, often with scaling. Stress can cause flare-ups. The scales are greasy, not dry, as commonly thought. An uncommon generalized form in infants may be linked to immuno-deficiencies. Topical therapy primarily consists of antifungal agents and low-potency steroids. New topical calcineurin inhibitors (immunomodulators) sometimes are administered.

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Introduction

Seborrhoeic dermatitis can affect patients from infancy to old age.[1-3] The condition most commonly occurs in infants within the first three months of life and in adults at 30 to 60 years of age. In adolescents and adults, it usually presents as scalp scaling (dandruff) or as mild to marked erythema of the nasolabial fold during times of stress or sleep deprivation. The latter type tends to affect men more often than women and often is precipitated by emotional stress. An uncommon generalised form in infants may be linked to immunodeficiencies.

Seborrhoeic dermatitis and *pityriasis capitis* (cradle cap) are common in early childhood. According to one survey of 1,116 children,[4] the overall age- and sex-adjusted prevalence of seborrhoeic dermatitis was 10 percent in boys and 9.5 percent in girls. The highest prevalence occurred in the first three months of life, decreasing rapidly by one year of age, and slowly decreasing over the next four years.[4] Most patients (72 percent) had minimal to mild seborrhoeic dermatitis. *Pityriasis capitis* occurred in 42 percent of the children examined (86 percent had a minimal to mild case).[4] Prevalence estimates for older persons are consistently higher than estimates for the general population.[5]

Aetiology

Despite the high prevalence of seborrhoeic dermatitis, little is known about its aetiology. However, several factors (e.g., hormone levels, fungal infections, nutritional deficits, neurogenic factors) are associated with the condition. The possible hormonal link may explain why the condition appears in infancy, disappears spontaneously, then reappears more prominently after puberty. A more causal link seems to exist between seborrhoeic dermatitis and the proliferation of Malassezia species (e.g., *Malassezia furfur, Malassezia ovalis*) found in normal dimorphic human flora.[6-8] Yeasts of this genus predominate and are found in seborrhoeic regions of the body that are rich in sebaceous lipids (e.g., head, trunk, upper back). A causal relationship is implied because of the ability to isolate Malassezia in patients with seborrhoeic dermatitis and by its therapeutic response to antifungal agents.[9] A similar link has been suggested in studies of patients with seborrhoeic dermatitis that is associated with acquired immunodeficiency syndrome (AIDS).[10,11] Seborrhoeic dermatitis also may be associated with nutritional deficiencies, but there is no firm linkage.

An altered essential fatty acid pattern may be important in the pathogenesis of infantile seborrhoeic dermatitis. Serum essential fatty acid patterns from 30 children with the condition suggested a transient impaired function of the delta-6 desaturase enzyme.[12] A neurogenic theory for the development of seborrhoeic dermatitis may account for its association with parkinsonism and other neurologic disorders, including post-cerebrovascular accidents, epilepsy, central nervous system trauma, facial nerve palsy, and syringomyelia induced by neuroleptic drugs with extrapyramidal effects.[7] It may be confined to the syringomyelia-affected area or to the paralyzed side in a patient with haemiplegia. However, no neurotransmitters have been identified in this context.

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	Evidence rating	References
Infants with generalized Seborrhoeic dermatitis, diarrhoea, and failure to thrive should be evaluated for immuno-deficiencies.	C	14
The first-line therapy for seborrhoeic dermatitis of the scalp should be topical steroids.	C	2, 20, 34
Topical calcineurin inhibitors (e.g., tacrolimus ointment, pimecrolimus cream [Elidel®]) are recommended for seborrhoeic dermatitis of the face and ears.	B	26-28
Once-daily ketoconazole (Nizoral®) combined with two weeks of once-daily desonide is recommended for seborrhoeic dermatitis of the face.	B	22

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, see page 17 or http://www.aafp.org/afpsort.xml.

Figure 1: Nasolabial fold scaling and erythema from seborrhoeic dermatitis.

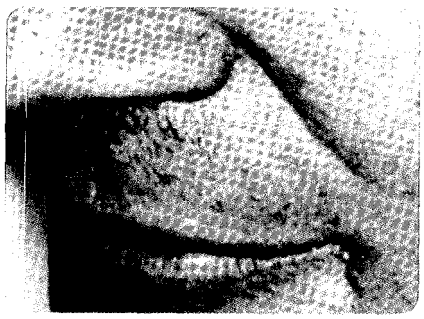


Figure 2: Severe persistent seborrhoeic dermatitis of the inframammary folds

Figure 3: Central facial erythema from seborrhoeic dermatitis.

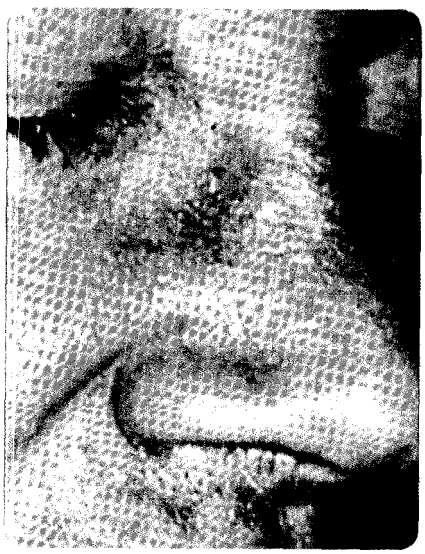
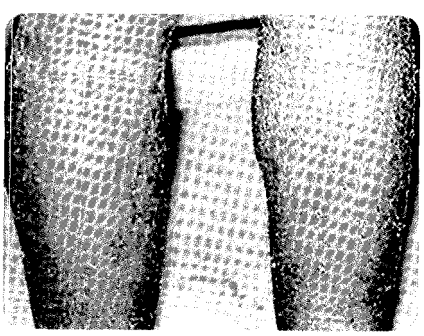


Figure 4: Generalised seborrhoeic dermatitis-like eruption associated with acquired immunodeficiency syndrome.



Two types of seborrhoeic dermatitis may appear on the chest - a common petaloid type and a rarer pityriasiform type.[2] The former starts as small, reddish-brown follicular and perifollicular papules with greasy scales. These papules become patches that resemble the shape of flower petals or a medallion (*medallion seborrhoeic dermatitis*). The pityriasiform type often has generalised macules and patches that resemble extensive ***pityriasis rosea***. These patches rarely produce an eruption so generalized that it causes erythroderma.

In infants, seborrhoeic dermatitis may present as thick, greasy scales on the vertex of the scalp (cradle cap).[2,3] The condition is not pruritic in infants, as it is in older children and adults. Typically, acute dermatitis (characterized by oozing and weeping) is absent. The scales may vary in colour, appearing white, off-white, or yellow. Infants with large, dry scales often have psoriasiform seborrhoeic dermatitis. This presentation often is the only sign of seborrhoeic dermatitis in infants and usually occurs in the third or fourth week after birth. However, the scalp, central face, forehead, and ears may have fine, widespread scaling. The dermatitis may become generalized. The flexural folds may be involved, often with a cheesy exudate that manifests as a diaper dermatitis that also may become generalized. Generalized seborrhoeic dermatitis is uncommon in otherwise healthy children and usually is associated with immunodeficiencies. Immunocompromised children with generalized seborrhoeic dermatitis often have concomitant diarrhoea and failure to thrive[5-8] (Leiner's disease); therefore, infants with these symptoms should be evaluated for immunodeficiencies.[13-15]

Differential Diagnosis

A number of disorders are similar to seborrhoeic dermatitis ***(Table 1)***. One study[11] showed that 47 percent of patients with AIDS had recalcitrant eruptions similar to seborrhoeic dermatitis that may be generalised in children and adults ***(Figure 4)***. Highly active antiretroviral therapy may reduce incidence in patients with AIDS.

Table 1: Differential diagnosis of seborrhoeic dermatitis

Atopic dermatitis
Candidiasis
Dermatophytosis
Langerhans cell histiocytosis
Psoriasis
Rosacea
Systemic lupus erythematosus
Tinea infection

Psoriasis vulgaris may be difficult to distinguish from seborrhoeic dermatitis. ***Psoriasis vulgaris*** of the scalp presents as sharply demarcated scalp plaques. Other signs of psoriasis, such as nail pitting or distal onycholysis, also may facilitate distinction.[16,17]

Seborrhoeic dermatitis also may resemble atopic dermatitis, ***tinea capitis***, and, rarely, cutaneous lymphoma or Langerhans cell histiocytosis. Atopic dermatitis in adults characteristically appears in antecubital and popliteal fossae. ***Tinea capitis***, ***tinea faciei***, and ***tinea corporis*** may have hyphae on potassium hydroxide cytologic examination; candidiasis produces pseudohyphae. Seborrhoeic dermatitis of the groin may resemble dermatophytosis, psoriasis, candidiasis, and, sometimes, Langerhans cell histiocytosis. Rosacea may produce a facial erythema resembling seborrheic dermatitis. Although rosacea tends to include central facial erythema, it may involve only the forehead.

Infants may have atopic dermatitis that is prevalent in certain body areas (e.g., scalp, face, diaper areas, extensor limb surfaces), suggesting seborrhoeic dermatitis.[18] However, in infants, seborrhoeic dermatitis has axillary patches, lacks oozing and weeping, and lacks pruritus. The distinction is a clinical one because elevated immunoglobulin E (IgE) levels associated with atopic dermatitis are a non-specific finding. Rarely, infants are affected by histologic-specific scaling, seborrhoeic dermatitis-like eruptions on the scalp with fever, and other systemic signs of acute Langerhans cell histiocytosis (Letterer-Siwe disease). Scabetic eczema occasionally resembles widespread seborrhoeic dermatitis. Riboflavin, biotin, and pyridoxine deficiencies have been associated with seborrhoeic dermatitis-like eruptions in infants.[19] Concomitant disorders (e.g., psoriasis, scabetic eczema, superficial fungal infection) may complicate seborrhoeic dermatitis, especially in patients with AIDS.

Histology

Skin biopsies may effectively distinguish seborrhoeic dermatitis from similar disorders. Seborrhoeic dermatitis should have neutrophils in the scale crust at the margins of follicular ostia. AIDS-associated seborrhoeic dermatitis more commonly presents as parakeratosis, a few individually necrotic

keratinocytes within the epidermis, and plasma cells in the dermis. Yeast cells sometimes are visible within keratinocytes on special stains. If hyphae are present, dermatomycosis is the diagnosis. Shorter hyphae with spores ("spaghetti and meatball pattern") are present with *tinea versicolor*.[8]

Treatment

Effective therapies for seborrhoeic dermatitis include anti-inflammatory (immunomodulatory) agents, keratolytic agents, antifungals, and alternative medications *(Table 2)*.[1-3,20-37]

ANTI-INFLAMMATORY (IMMUNOMODULATORY) AGENTS

The conventional treatment for adult seborrhoeic dermatitis of the scalp starts with topical steroids or a calcineurin inhibitor. These therapies may be administered as a shampoo, such as fluocinolone, topical steroid solutions, lotions applied to the scalp, or creams applied to the skin.[30,31,34-36] Adults with seborrhoeic dermatitis typically use topical steroids once or twice daily, often in addition to a shampoo. Low-potency topical steroids may effectively treat infantile or adult seborrhoeic dermatitis of the flexural areas or persistent recalcitrant seborrhoeic dermatitis in adults.[2,20] A topical azole preparation may be combined with a desonide-type regimen (one dose daily for two weeks) for facial seborrhoeic dermatitis.[22]

Topical calcineurin inhibitors (e.g. pimecrolimus cream [Elidel®]) have fungicidal and anti-inflammatory properties without the risk of cutaneous atrophy, which is associated with topical steroids.[26-28] Calcineurin inhibitors also are good therapies when the face and ears are affected. However, one week of daily use is necessary before benefits are apparent.

KERATOLYTICS

Older modalities for treating seborrhoeic dermatitis may have had keratolytic but not specific antifungal properties.[1,2] Keratolytics that are widely used to treat seborrhoeic dermatitis include tar, salicylic acid, and zinc pyrithione shampoos. Pyrithione zinc has non-specific keratolytic and antifungal properties[3,21] and can be applied two or three times per week. Patients should leave these shampoos on the hair for at least five minutes to ensure that it reaches the scalp. Patients also may use it on other affected sites, such as the face. Infantile seborrhoeic dermatitis of the scalp requires a gentle approach[3] (e.g., a mild, non-medicated shampoo).

ANTIFUNGALS

Most antifungal agents attack Malassezia associated with seborrhoeic dermatitis.[1,2] A once-daily ketoconazole gel preparation (Nizoral®) combined with a two-week, once-daily regimen of desonide (Not in RSA), may be useful for facial seborrhoeic dermatitis.[22] Shampoos containing selenium sulfide (Selsun®) or an azole often are used.[1,2,20,21] These shampoos can be applied two or three times per week. Ketoconazole (cream or foaming gel)[31,32] and oral terbinafine (Lamisil®) also may be beneficial.[23] Other topical antifungal agents include ciclopirox (Not in RSA)[33,36] and fluconazole (Diflucan®).[29] Patients also may use a 2 % ketoconazole or a fluconazole shampoo.[29,30,35] Some azoles (e.g., itraconazole [Sporanox®], ketoconazole) also have anti-inflammatory properties.[37]

ALTERNATIVE MEDICATIONS

Natural therapies are becoming increasingly popular. Tea tree oil (Melaleuca oil) is an essential oil from a shrub native to Australia. The therapy appears to be effective and well tolerated when used daily as a 5 % shampoo.[25]

See CPD Questionnaire, page 48

This article has been peer reviewed

Table 2: Therapies for treating seborrhoeic dermatitis

Therapy	Usage
Anti-inflammatory (immunomodulatory) agents	
Steroid shampoo	
Fluocinolone	Two times per week
Topical steroids	
Fluocinolone	Daily
Betamethasone valerate lotion	Daily
Desonide cream (Not in RSA)	Daily
Topical calcineurin inhibitors	
Tacrolimus ointment (Not in RSA)*	Daily
Pimecrolimus cream (Elidel)*	Daily
Keratolytics	
Salicylic acid shampoo	Two times per week
Tar shampoo	Three times per week
Zinc pyrithione shampoo (also has antifungal properties)	Two times per week
Antifungals	
Ketoconazole shampoo (Nizoral	Three times per week
Selenium sulfide shampoo (Selsun®)	Two times per week
Alternative medication	
Tea tree oil shampoo	Daily

**-Off-label use.*
Information from references 1 through 3 and 20 through 37.

The Authors

ROBERT A. SCHWARTZ M.D., M.P.H., is professor and head of dermatology and professor of medicine, pediatrics, pathology, and preventive medicine and community health at the University of Medicine and Dentistry of New Jersey (UMDNJ)-New Jersey Medical School, Newark. He received a medical degree from New York Medical College in Manhattan and completed a residency at the University of Cincinnati College of Medicine, Ohio, and at Roswell Park Cancer Institute, Buffalo, N.Y. He is a member of *American Family Physician*'s editorial advisory board.

CHRISTOPHER A. JANUSZ, M.D., is resident physician at New York Medical College. He received a medical degree from Jagiellonian University School of Medicine in Cracow, Poland.

CAMILA K. JANNIGER, M.D., is clinical professor and chief of pediatric dermatology and geriatric dermatology at the UMDNJ-New Jersey Medical School. She received a medical degree from the Medical Academy of Warsaw in Poland. Dr. Janniger completed an internship at the Albert Einstein College of Medicine of the Yeshiva University's Montefiore Medical Center, Bronx, N.Y., and a dermatol-

ogy residency at the UMDNJ-New Jersey Medical School.
Address correspondence to Robert A. Schwartz, M.D., M.P.H., UMDNJ-New Jersey Medical School, Dept. of Dermatology, 185 S. Orange Ave., Newark, NJ 07103 (e-mail: roschwar@cal.berkeley.edu). Reprints are not available from the authors.
The authors dedicate this paper to Decio Cerimele, M.D., professor and chair of dermatology at the University of Sassari in Italy, and to Francesca Cerimele, M.D., University of Alabama Huntsville Family Medicine Residency.
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Infantile Colic

Roberts DM, MD
Ostapchuk M, MD
O'Brien JG, MD
University of Louisville, School of Medicine, Louisville, K*entucky*

Correspondence to: Dr Donna Roberts, E-mail: dmrobe01@gwise.louisville.edu

Abstract

Infantile colic can be distressing to parents whose infant is inconsolable during crying episodes. Colic is often defined by the "rule of three": crying for more than three hours per day, for more than three days per week, and for longer than three weeks in an infant who is well-fed and otherwise healthy. The physician's role is to ensure that there is no organic cause for the crying, offer balanced advice on treatments, and provide support to the family. Colic is a diagnosis of exclusion that is made after performing a careful history and physical examination to rule out less common organic causes. Treatment is limited. Feeding changes usually are not advised. Medications available in the United States have not been proved effective in the treatment of colic, and most behaviour interventions have not been proved to be more effective than placebo. Families may turn to untested resources for help, and the physician should offer sound advice about these treatments. Above all, parents need reassurance that their baby is healthy and that colic is self-limited with no long-term adverse effects. Physicians should watch for signs of continuing distress in the child and family, particularly in families whose resources are strained already.

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Introduction

Excessive crying or colic in an infant during the first few months of life can be alarming for physicians and parents. Estimates of the occurrence of infantile colic in community-based samples vary from 5 to 25 percent of infants, depending on study design, definition of colic, and method of data collection.[1,2] Fussing and crying are normal aspects of development during the first three months of life. During this time, infants cry an average of 2.2 hours per day, peaking at six weeks of age and gradually decreasing.[3] Parents who think their infant cries excessively may seek a physician's help.

Physicians and parents use the term colic to describe an infant with excessive crying, irritability, or fussiness. The most commonly accepted definition of colic, which originated in 1954,[4] describes using the "rule of three": crying for more than three hours per day, for more than three days per week, and for more than three weeks in an infant that is well-fed and otherwise healthy. This definition has been used repeatedly in clinical studies of colic. The motor behaviours of infants with colic also were first described in 1954.[4] Colicky infants have attacks of screaming in the evening with associated motor behaviours such as flushed face, furrowed brow, and clenched fists. The legs are pulled up to the abdomen, and the infants emit a piercing, high-pitched scream.[5]

Behaviour characteristics usually are classified by the timing of the event, paroxysmal crying, and associated behaviors.[6] Colic typically begins at two weeks of age and usually resolves by four months of age. Crying is concentrated in the late afternoon and evening, occurs in prolonged bouts, and is unpredictable and spontaneous. It appears to be unrelated to environmental events. The child cannot be soothed, even by feeding.

Table 1: Organic Causes of Excessive Crying in Infants*

CNS
CNS abnormality (Chiari type I malformation)
Infantile migraine
Subdural haematoma
Gastrointestinal
Constipation
Cow's milk protein intolerance
Gastroesophageal reflux
Lactose intolerance
Rectal fissure
Infection
Meningitis
Otitis media
Urinary tract infection
Viral illness
Trauma
Abuse
Corneal abrasions
Foreign body in the eye
Fractured bone
Hair tourniquet syndrome

CNS = central nervous system
*-Organic causes account for less than 5 percent of infants with colic.
Adapted with permission from Barr RG. Colic and crying syndromes in infants. Pediatrics 1998;102(5 suppl E):1283, and Poole SR. The infant with acute, unexplained, excessive crying. Pediatrics 1991;88:452.

Aetiology

The cause of infantile colic remains unclear. Underlying organic causes of excessive crying must be considered during the evaluation. Organic causes account for less than 5 percent of infants presenting with excessive crying *(Table 1).*[6,7] Gastrointestinal, psychosocial, and neuro-developmental disorders have been suggested as the cause of colic.

GASTROINTESTINAL

Gastrointestinal disorders have been implicated in colic because of the infant's leg position and grimacing during a crying spell. Excessive crying or increased gas production from colon function can result in intraluminal gas formation and aerophagia. This mechanism does not appear to be the cause of colic, however, because radiographic images taken during a crying episode have shown a normal gastric outline.[8] There is conflicting evidence showing that colic is caused by allergy to human and cow's milk protein. It also has been speculated that abdominal cramping and colic may be a result of hyperperistalsis. The latter theory is supported by evidence that the use of anticholinergic agents decreases colic symptoms. Gut hormones such as motilin also may play a causative role in