CHAPTER 22

Seborrheic Dermatitis

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Seborrheic dermatitis is a common chronic papulosquamous dermatosis that is usually easily recognized. It affects infants and adults and is often associated with increased sebum production (seborrhea) of the scalp and the sebaceous follicle–rich areas of the face and trunk. The affected skin is pink, edematous, and covered with yellow-brown scales and crusts. The disease varies from mild to severe, including psoriasiform or pityriasiform patterns and erythroderma.[1] Seborrheic dermatitis is one of the most common skin manifestations in patients with human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS).[2] Consequently, it is included in the spectrum of premonitory lesions and should be carefully evaluated in high-risk patients.

EPIDEMIOLOGY

Seborrheic dermatitis has two age peaks, one in infancy within the first 3 months of life and the second around the fourth to the seventh decades of life. No data are available on the exact incidence of seborrheic dermatitis in infants, but the disorder is common. The disease in adults is believed to be more common than psoriasis, for example, affecting at least 3 percent to 5 percent of the population in the United States.[3] Men are affected more often than women in all age groups. There does not appear to be any racial predilection. Seborrheic dermatitis is found in up to 85 percent of patients with HIV infection and AIDS.[2]

ETIOLOGY AND PATHOGENESIS

Although many theories abound, the cause of seborrheic dermatitis remains unknown.

SEBORRHEA

Seborrhea is associated with oily-looking skin (seborrhea oleosa), although an increased sebum production cannot always be detected in these patients.[4] Even if seborrhea does provide a predisposition, seborrheic dermatitis is not a disease of the sebaceous glands. The high incidence of seborrheic dermatitis in newborns parallels the size and activity of the sebaceous glands at this age. It has been shown that newborns have large sebaceous glands with high sebum secretion rates similar to adults.[5] In childhood, sebum production and seborrheic dermatitis are closely connected. In adulthood, however, they are not, as the sebaceous gland activity peaks in early puberty and decades later seborrheic dermatitis may occur.

The sites of predilection—face, ears, scalp, and upper part of the trunk—are particularly rich in sebaceous follicles. Two diseases are prevalent in these regions: seborrheic dermatitis and acne. In patients with seborrheic dermatitis, the sebaceous glands are often particularly large on cross-sectional histologic specimens. In one study, skin surface lipids were not elevated but the lipid composition was characterized by an increased proportion of cholesterol, triglycerides, and paraffin, and a decrease in squalene, free fatty acids, and wax esters.[6] However, mild abnormalities in the skin surface lipids could well result from the ineffective keratinization, which is often demonstrable histopathologically. Seborrheic dermatitis seems to be more frequent in patients with parkinsonism and other neurologic disorders, in whom sebum secretion is increased. Similarly, after reduction of sebum production induced by levodopa and by promestriene, seborrheic dermatitis may improve.

The synonym *eczéma flannelaire* stems from the idea that a retention of skin surface lipids by clothing and rubbing of rough textiles on the skin (flannel), or synthetic underwear promotes or aggravates seborrheic dermatitis.

MICROBIAL EFFECTS

Unna and Sabouraud, who were among the first to describe the disease, favored an etiology involving bacteria, yeasts, or both. This hypothesis has remained unsupported, although bacteria and yeasts can be isolated in great quantities from affected skin sites.

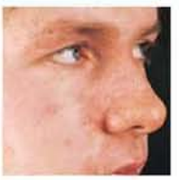
In infancy, *Candida albicans* is often found in dermatitic skin lesions and in stool specimens. Although intracutaneous tests with candidin, positive agglutinating antibodies in serum, and positive lymphocyte-transformation tests in affected infants revealed sensitization to *C. albicans*, this yeast cannot be convincingly linked to the pathogenesis.

Aerobic bacteria were recovered from the scalp of patients with seborrheic dermatitis (140,000 bacteria/cm^2 vs. 280,000 in unaffected individuals and 250,000 in persons with dandruff). In contrast, *Staphylococcus aureus* was rarely seen in unaffected persons or those with dandruff. *Staphylococcus* was recovered in approximately 20 percent of patients with seborrheic dermatitis, accounting for an average of approximately 32 percent of the total skin flora.[7]

Propionibacterium acnes counts were low in patients with seborrheic dermatitis (7550 bacteria/cm^2 in those without dandruff). The small quantities of *P. acnes* in patients with seborrheic dermatitis may explain the low yield of free fatty acids from their skin surfaces.

The lipophilic yeast *Malassezia furfur* (also known as *Pityrosporum*) is abundant in normal skin (504,000 organisms/cm^2 vs. 922,000 in individuals with dandruff and 665,000 in patients with seborrheic dermatitis).[7] Some authors claim strong evidence in favor of a pathogenic role for these microbes, whereas others do not share this view. Their argument is that *M. furfur* is not the causative organism, but is merely present in large numbers. In patients with pityriasis versicolor[8] and *Malassezia* folliculitis,[9] seborrheic dermatitis has been found in a higher percent-

SEBORRHEIC DERMATITIS AT A GLANCE



- Infantile and adult forms exist.
- Characterized by erythema and greasy scaling.
- Lesions favor scalp, ears, face, chest, and intertriginous areas.
- Generalized and even erythrodermic forms may occur.
- Etiology unknown but may be related to increased sebum secretion, abnormal sebum composition, certain drugs, or *Malassezia* yeasts.
- May be a cutaneous marker of human immunodeficiency virus infection and acquired immunodeficiency syndrome, especially when severe, atypical, and therapy resistant.

age than expected. Clearing of seborrheic dermatitis by selenium sulfide and continued suppression of *M. furfur* with topical amphotericin B caused a relapse of the disease on inflamed scalp skin.[10] In seborrheic dermatitis, both normal and high levels of serum antibodies against *M. furfur* have been demonstrated. A cell-mediated immune response to *M. furfur* has been found in normal individuals using *Malassezia* extracts in lymphocyte-transformation studies.[11] Overgrowth of *M. furfur* may lead to inflammation, either through introduction of yeast-derived metabolic products into the epidermis or as a result of the presence of yeast cells on the skin surface. The mechanism of production of inflammation would likely then be through Langerhans cell and T lymphocyte activation by *Malassezia* or its by-products. When *M. furfur* comes into contact with serum, it can activate complement via the direct and alternative pathways and this may play some part in the introduction of inflammation.[12] A possible role for this yeast in the pathogenesis of seborrheic dermatitis is supported by the fact that seborrheic dermatitis–like lesions have been shown to be reproducible in animal models by inoculation of *M. furfur*.[13]

MISCELLANEOUS

Drugs

Several drugs have been reported to produce seborrheic dermatitis–like lesions, including arsenic, gold, methyldopa, cimetidine, and neuroleptics.

Neurotransmitter Abnormalities

Seborrheic dermatitis is often associated with a variety of neurologic abnormalities, pointing to a possible influence of the nervous system. These neurologic conditions include postencephalitic parkinsonism, epilepsy, supraorbital injury, facial paralysis, unilateral injury to the trigeminal ganglion, poliomyelitis, syringomyelia, and quadriplegia. Emotional stress seems to aggravate the disease; a high rate of seborrhea is reported among combat troops in times of war.

Physical Factors

It has been suggested that cutaneous blood flow and skin temperature may be responsible for the distribution of seborrheic dermatitis.[14] Seasonal variations in temperature and humidity are related to the course of the disease. Low fall and winter temperatures and low humidity in centrally heated rooms are known to worsen the condition. Seborrheic dermatitis of the face was observed in 8 percent of 347 patients receiving psoralen and ultraviolet A light therapy for psoriasis and occurred within a few days to 2 weeks after the beginning of treatment;[15] the patients had no previous history of facial psoriasis or seborrheic dermatitis. Lesions were avoided by masking the face during irradiation.

Aberrant Epidermal Proliferation

Epidermal proliferation is increased in seborrheic dermatitis, similar to psoriasis, explaining why cytostatic therapeutic modalities may improve the condition.[16]

Nutritional Disorders

Zinc deficiency in patients with acrodermatitis enteropathica and acrodermatitis enteropathica-like conditions may be accompanied by dermatitis mimicking seborrheic dermatitis of the face. Seborrheic dermatitis is, however, not associated with zinc deficiency nor does it respond to supplementary zinc therapy.

Seborrheic dermatitis in infancy may have a different pathogenesis. Biotin deficiency, whether secondary to holocarboxylase or biotinidase deficiency, and abnormal metabolism of essential fatty acids have been proposed as possible mechanisms.[17] However, biotin has been subsequently shown to have no more than placebo effect when studied in a double-blind manner.[18] Although one study suggested the possible role of food allergy in seborrheic dermatitis of infancy,[19] this has not been confirmed.

Genetic Factors

Recently, a condition considered to be seborrheic dermatitis with a gene defect in a zinc finger protein has been described.[20]

IMMUNODEFICIENCY AND SEBORRHEIC DERMATITIS

The development of seborrheic dermatitis either de novo or as a flare of preexisting disease also may serve as a clue to the presence of HIV infection and AIDS. The first report of this association in 1984 was followed by observations from all parts of the world.[2]

The increased incidence and severity of seborrheic dermatitis in HIV seropositive individuals has led to speculation that unchecked growth of *Malassezia* in immunosuppressed patients is responsible. However, a study that compared quantitative *Malassezia* cultures in AIDS patients with and without seborrheic dermatitis did not demonstrate increased yeast colonization in patients with seborrheic dermatitis.[21]

PSORIASIS AND SEBORRHEIC DERMATITIS

In patients with a psoriatic predisposition, particularly adults, seborrheic dermatitis is said to evolve into psoriasis. The term *sebopsoriasis* is sometimes used for these overlapping conditions. It should be used with caution because psoriasis, especially of the scalp, is clinically and histopathologically almost indistinguishable from seborrheic dermatitis.

CLINICAL FINDINGS

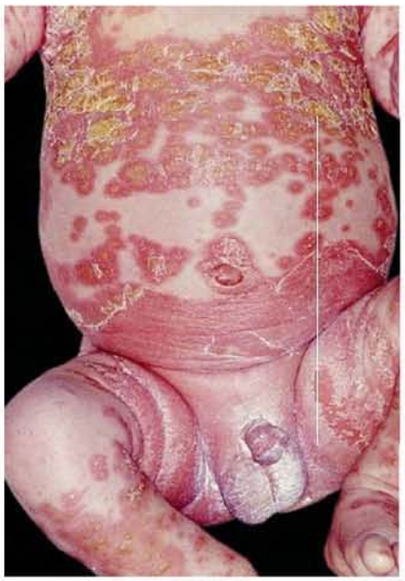
In all patients with seborrheic dermatitis there is a so-called *seborrheic stage*, which is often combined with a gray-white or yellow-red skin discoloration, prominent follicular openings, and mild to severe pityriasiform scales. Several forms can be distinguished (Table 22-1).

SEBORRHEIC DERMATITIS IN INFANTS

The disease occurs in infants predominantly within the first months of life as an inflammatory disease mainly affecting the hairy scalp and intertriginous folds with greasy-looking scales and crusts (Fig. 22-1). Other regions such as the center of the face, chest, and neck may also be affected. Scalp involvement

TABLE 22-1
Clinical Patterns of Seborrheic Dermatitis

- Infantile
  - Scalp (cradle cap)
  - Trunk (including flexures and napkin area)
  - Leiner's disease
    - Nonfamilial
    - Familial C5 dysfunction
- Adult
  - Scalp
  - Face (may include blepharitis)
  - Trunk
    - Petaloid
    - Pityriasiform
    - Flexural
    - Eczematous plaques
    - Follicular
  - Generalized (may be erythroderma)



▲ **FIGURE 22-1** Seborrheic dermatitis in an infant. Widespread pattern of seborrheic dermatitis with psoriasiform lesions on the trunk and groin.

is fairly characteristic. The frontal and parietal scalp regions are covered with an oily-looking, thick, often fissured crust (*crusta lactea, milk crust,* or *cradle cap*). Hair loss does not occur and inflammation is sparse. In the course of the disease, the redness increases and the scaled areas form clearly outlined erythematous patches topped by a greasy scale. Extension beyond the frontal hairline occurs. The retroauricular folds, the pinna of the ear, and the neck may also be involved. Otitis externa is often a complicating factor. Semiocclusive clothing and diapers favor moisture, maceration, and intertriginous dermatitis, particularly in the folds of the neck, axillae, anogenital area, and groin. Opportunistic infection with *C. albicans, S. aureus,* and other bacteria occurs. The clinical aspect reminds one of psoriasis, hence the expressions *psoriasoid* or *napkin psoriasis.*

Prognosis and Clinical Course

The disease is usually protracted over weeks to months. Exacerbation and, rarely, generalized exfoliating dermatitis may occur. The prognosis is good. There is no indication that infants with seborrheic dermatitis are more likely to suffer from the adult form of the disease.

Differential Diagnosis (See Box 22-1)

The most useful distinguishing feature between atopic dermatitis and seborrheic dermatitis is the increased number of lesions on the forearms and shins in the former and in the axillae in the latter. The development of skin lesions solely in the diaper area favors a diagnosis of infantile seborrheic dermatitis. Radioallergosorbent assay test screening to egg white and milk antibodies or other geographically or ethnically relevant allergens (e.g., soybean), and, to a lesser extent, total immunoglobulin E levels, may be useful in diagnosing atopic dermatitis at an early stage and distinguishing it from infantile seborrheic dermatitis. Absent to mild pruritus is considered a significant feature of infantile seborrheic dermatitis. Some authors believe that infantile seborrheic dermatitis is a clinical variant of atopic dermatitis rather than a separate entity.[22]

Box 22-1

Differential Diagnosis of Infantile Seborrheic Dermatitis

Most Likely
- Atopic dermatitis

Consider
- Scabies, psoriasis

Rule Out
- Langerhans cell histiocytosis

ERYTHRODERMA DESQUAMATIVUM (LEINER DISEASE)

Erythroderma desquamativum is a complication of seborrheic dermatitis in infants (dermatitis seborrhoides infantum) and was described in 1908 by Leiner.[23] There is usually a sudden confluence of lesions, leading to a universal scaling redness of the skin (erythroderma). The young patients are severely ill with anemia, diarrhea, and vomiting. Secondary bacterial infection is common. The prognosis is very good unless proper intensive care and skin care are not provided. The disease is both a familial and a nonfamilial form. The former is noted for having a functional deficiency of C5 complement, resulting in defective opsonization. These patients respond to antibiotics and infusions of fresh frozen plasma or whole blood. The true nature of this disease remains obscure.

SEBORRHEIC DERMATITIS IN ADULTS

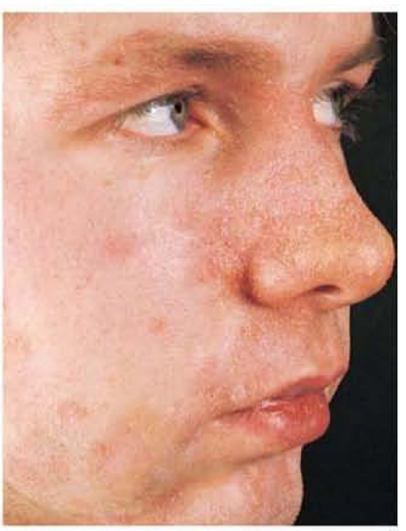
The clinical picture and course of seborrheic dermatitis in adults differs from that of infants.

Seborrheic eczematid is the mildest form of the disease (eczematid = eczema-like, dermatitis-like). It is associated with seborrhea, scaling, mild redness, and often pruritus of the scalp, eyebrows, nasolabial folds, and retroauricular area, as well as over the sternum and the shoulder blades. Asymptomatic fluffy, white dandruff of the scalp represents the mild end of the spectrum of seborrheic dermatitis and has been referred to as *pityriasis sicca*. Nasolabial erythema, more common in young women than men, may be part of this disease spectrum.

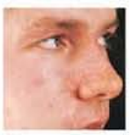
Patchy seborrheic dermatitis is the classic, well-known disease with chronic recurrent lesions. Lesions have a predilection for scalp, temples, inner parts of the eyebrows and glabella with nasolabial folds (Figs. 22-2 and 22-3), retroauricular folds and external ear canal (Fig. 22-4) and V-shaped areas of the chest and back (Fig. 22-5). Less frequently, intertriginous areas, such as the side of the neck, axillae, submammary region, umbilicus, and genitocrural folds, are involved.

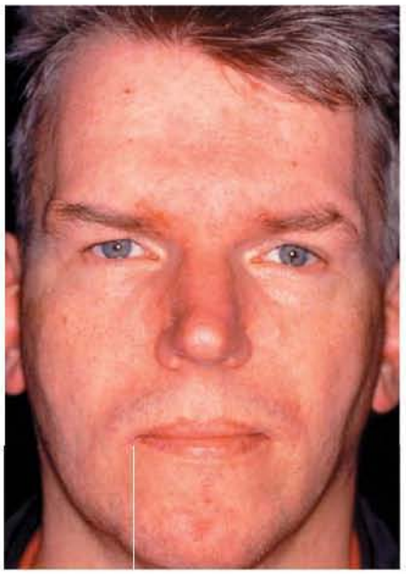
Clinical Findings

Skin lesions are characterized by a yellow color, mild to severe erythema, mild inflammatory infiltrate, and oily, thick scales and crusts. This has occasionally been referred to as *pityriasis steatoides*. Patients report pruritus, particularly on the scalp and in the ear canal. The lesions start with follicular and perifollicular redness and mounds; they spread until they form clearly outlined, round to circinate (petaloid) patches (Greek *petalon,* a thin plate or leaf). The pityriasiform type of seborrheic dermatitis is seen on the trunk and mimics the



▲ **FIGURE 22-2** Seborrheic dermatitis with involvement of nasolabial folds, cheeks, eyebrows, and nose.



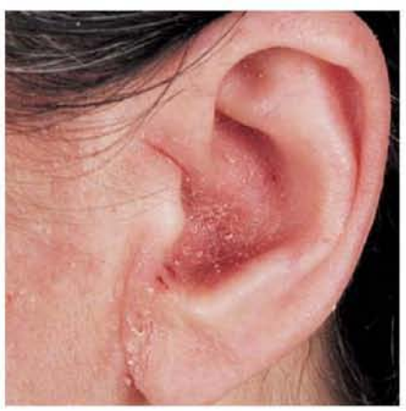


▲ **FIGURE 22-3** Seborrheic dermatitis of the forehead and centrofacial region.

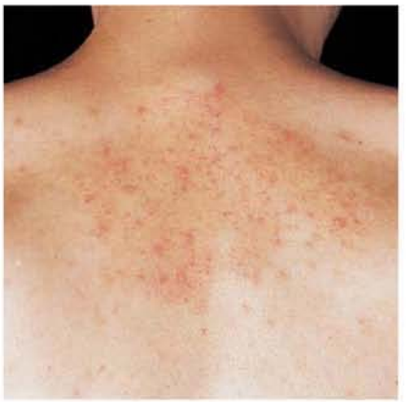
lesions of pityriasis rosea, producing oval scaly lesions whose long axes tend to parallel the ribs. In some individuals, only one or two sites are involved. Chronic dermatitis of the ear canal may be the sole manifestation of seborrheic dermatitis, often mistaken for fungal infections. Another possible manifestation is blepharitis with honey-colored crusts along the rim of the eyelid and casts of horny cell debris around the eyelashes. In men, a more follicular type of seborrheic dermatitis may extend over large parts of the back, flanks, and abdomen.

Prognosis and Clinical Course

Usually, the disease lasts for years to decades with periods of improvement in warmer seasons and periods of exacerbation in the colder months. Widespread lesions may occur as a result of improper topical treatment or sun exposure. The extreme variant of the disease is generalized exfoliative erythroderma (seborrheic erythroderma). Onychodystrophy, electrolyte imbalance, and thermal dysregulation are additional features sometimes found in these patients.



▲ **FIGURE 22-4** Seborrheic dermatitis of the earlobe and ear.



▲ **FIGURE 22-5** Seborrheic dermatitis of the upper back.

PITYRIASIS AMIANTACEA

Pityriasis amiantacea (synonyms: tinea amiantacea, asbestos scalp, porrigo amiantacea, tinea asbestina, keratosis follicularis amiantacea) is the name given to a disease of the scalp in which heavy scales extend onto the hairs and separate and bind together their proximal portions. See Box 22-2 for site-specific differential diagnosis of seborrheic dermatitis.

Pityriasis amiantacea is a reaction of the scalp, often without evident cause, that may occur at any age. It may be observed as a complication or sequel of streptococcal infection, seborrheic dermatitis, atopic dermatitis, lichen simplex, and it also occurs in psoriasis, of which it may be the first clinical manifestation.[24]

The process may be circumscribed or diffuse. It is only slightly inflammatory with dry, micaceous scales, or markedly inflammatory with admixture of a crust (Fig. 22-6). Removal of the scales reveals normal or erythematous edematous epidermis. The process is not followed by atrophy, scarring, or alopecia. If scarring alopecia occurs, it may be related to secondary infection. A common form complicates chronic or recurrent fissuring behind one or both ears, mostly in young girls. The sticky scales extend several centimeters into the neighboring scalp. Another form extends from patches of lichen simplex and is seen mainly in middle-aged women.

▲ **FIGURE 22-6** Pityriasis amiantacea. Masses of sticky silvery scales adhere to the scalp and are attached in layers to the shafts of the hairs that they surround.

SEBORRHEIC DERMATITIS IN HUMAN IMMUNODEFICIENCY VIRUS–INFECTED INDIVIDUALS

The expression of seborrheic dermatitis differs in several aspects from its classic form seen in HIV seronegative individuals: The distribution is extensive, severity is marked (Fig. 22-7), and treatment often difficult. Even the histopathologic changes differ somewhat from those seen in commonly encountered seborrheic dermatitis (Table 22-2).

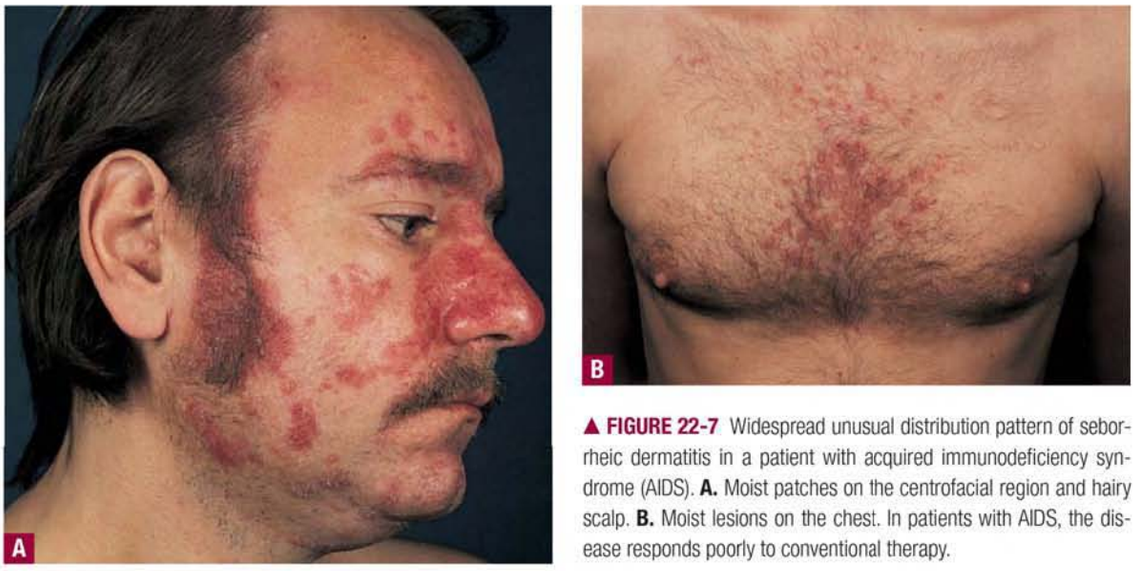
HISTOPATHOLOGY

The histopathologic picture varies according to the stage of the disease: acute, subacute, or chronic.[25,26] In acute and subacute seborrheic dermatitis,

Box 22-2

Site-Specific Differential Diagnosis of Seborrheic Dermatitis

Scalp	Dandruff, psoriasis, atopic dermatitis, impetigo
Face	Psoriasis, rosacea, contact dermatitis, impetigo
Ear canal	Psoriasis, contact dermatitis
Eyelids	Atopic dermatitis, psoriasis, *Demodex folliculorum* infestation
Chest and back	Pityriasis rosea, pityriasis versicolor
Intertriginous areas	Psoriasis, candidiasis
All sites, rule out	Secondary syphilis, pemphigus foliaceus



▲ **FIGURE 22-7** Widespread unusual distribution pattern of seborrheic dermatitis in a patient with acquired immunodeficiency syndrome (AIDS). **A.** Moist patches on the centrofacial region and hairy scalp. **B.** Moist lesions on the chest. In patients with AIDS, the disease responds poorly to conventional therapy.

there is a sparse superficial perivascular infiltrate of lymphocytes and histiocytes, slight to moderate spongiosis, slight psoriasiform hyperplasia, follicular plugging by orthokeratosis and parakeratosis, and scale-crusts containing neutrophils at the tips of the follicular ostia (see Table 22-2). In chronic seborrheic dermatitis, there are markedly dilated capillaries and venules in the superficial plexus, in addition to the above-mentioned features.

Clinically and histopathologically the lesions of chronic seborrheic dermatitis are psoriasiform and often difficult to distinguish from those of psoriasis.[25] Abortive forms of psoriasis share many features with seborrheic dermatitis. There are lesions that resemble psoriasis and may persist over many years before they finally turn into overt psoriasis. The most important diagnostic signs of seborrheic dermatitis are mounds of scale-crust–containing neutrophils at the tips of the dilated horn-filled follicular infundibula. Acrosyringia and acroinfundibula may be plugged by corneocyte casts.

The most consistent findings in pityriasis amiantacea are spongiosis, parakeratosis, migration of lymphocytes into the epidermis, and a variable degree of acanthosis.[27] The essential features responsible for the asbestosis-like scaling are diffuse hyperkeratosis and parakeratosis together with follicular keratosis in which each hair is surrounded by a sheath of corneocytes and debris.

Cytologic abnormalities of superficial horny cells (corneocytes) including ortho- and parakeratotic (nucleated) cells, horny cells in different stages of nuclear decomposition (halo cells), and masses of leukocytes can be evaluated by exfoliative cytology. Seborrheic dermatitis and psoriasis, however, present similar findings compared with other conditions of the dermatitis-eczema group.[28] The histopathology of AIDS-associated seborrheic dermatitis is more severe and differs in some respects from the classical form (see Table 22-2).

TREATMENT

General Considerations

In general, therapy is directed toward loosening and removal of scales and crusts, inhibition of yeast colonization, control of secondary infection, and reduction of erythema and itching. Adult patients should be informed about the chronic nature of the disease and understand that therapy works by controlling the disease rather than by curing it. The prognosis of infantile seborrheic dermatitis is excellent because the condition is benign and self-limited.

Infants

SCALP Treatment consists of the following measures: removal of crusts with 3 percent salicylic acid in olive oil or a water-soluble base; warm olive oil compresses; application of low-potency glucocorticosteroids (e.g., 1 percent hydrocortisone) in a cream or lotion for a few days; topical antifungal agents such as imidazoles (in a shampoo); mild baby shampoos; proper skin care with emollients, creams, and soft pastes.

INTERTRIGINOUS AREAS Treatment measures include drying lotions, such as 0.2 percent to 0.5 percent clioquinol in zinc lotion or zinc oil. In cases of candidiasis, nystatin or amphotericin B lotion or cream can be applied followed by soft and stiff pastes. In cases of oozing der-

TABLE 22-2
Histopathologic Differences Between Acquired Immunodeficiency Syndrome–Associated Seborrheic Dermatitis and Classic Seborrheic Dermatitis

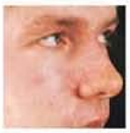
Classic Seborrheic Dermatitis	Acquired Immunodeficiency Syndrome–Associated Seborrheic Dermatitis
Epidermis	
Limited parakeratosis	Widespread parakeratosis
Rare necrotic keratinocytes	Many necrotic keratinocytes
No interface obliteration	Focal interface obliteration with clusters of lymphocytes
Prominent spongiosis	Sparse spongiosis
Dermis	
Thin-walled vessels	Many thick-walled vessels
Rare plasma cells	Increased plasma cells
No leukocytoclasis	Focal leukocytoclasis

From Soeprono FF et al: Seborrheic-like dermatitis of acquired immunodeficiency syndrome: A clinicopathologic study. *J Am Acad Dermatol* **14**:242, 1986, with permission.

matitis, application of 0.1 percent to 0.25 percent gentian violet where still available in combination with cotton or muslin diapers is often helpful. Imidazole preparations (e.g., 2 percent ketoconazole in soft pastes, creams, or lotions) may also be effective.

DIET Milk-free and high-protein, low-fat diets have not been shown to be of value, nor has the efficacy of oral or intramuscular biotin, vitamin B complex, or essential fatty acids been established.

Adults



Because the disease runs an unpredictably long course, careful and mild treatment regimens are recommended. Anti-inflammatory agents and, when indicated, antimicrobial or antifungal agents have to be used.

SCALP Frequent shampooing with shampoos containing 1.0 percent to 2.5 percent selenium sulfide, imidazoles (e.g., 2 percent ketoconazole), zinc pyrithione, benzoyl peroxide, salicylic acid, coal or juniper tar where still available, or detergents is recommended. Crusts or scales can be removed by overnight application of glucocorticosteroids or salicylic acid in water-soluble bases or, when necessary, under occlusive dressings. Tinctures, alcoholic solutions, hair tonics, and similar products usually aggravate the inflammatory state and should be avoided.

In pityriasis amiantacea, scales should be removed by the use of cade oil ointment or a topical tar/salicylic ointment. Either preparation should be washed out of the scalp after 4 to 6 hours with a suitable shampoo (e.g., tar or imidazole shampoo). Potent topical corticosteroid scalp creams or liquids may be beneficial in some cases, preferably under plastic occlusion in the initial phase. If topical treatment does not work, systemic glucocorticosteroids (e.g., 0.5 mg prednisolone/kg body weight/day for approximately 1 week) in combination with topical treatment (steroid under occlusion, followed by open application) should be considered. Concomitant antimicrobial treatment (e.g., macrolides, sulfonamides) is reserved for stubborn cases, especially if bacterial coinfection of the scalp is proven or suspected. Of course, the underlying condition must be treated. Treatment remains difficult, and relapses occur frequently.

FACE AND TRUNK Patients should avoid greasy ointments and reduce or omit the use of soaps. Alcoholic solutions or pre- or aftershave lotions should not be recommended. Low-potency glucocorticosteroids (1 percent hydrocortisone is usually sufficient) are helpful early in the course of the disease. Uncontrolled long-term applications lead to side effects such as steroid dermatitis, steroid rebound phenomenon, steroid rosacea, and perioral dermatitis.

SEBORRHEIC OTITIS EXTERNA Seborrheic otitis externa can be best treated with a low-potency glucocorticosteroid cream or ointment. Many otic preparations (solutions) that contain neomycin and other antibiotics, often in combination, are strong sensitizers and should be avoided. Once dermatitis is under control, the glucocorticosteroid should be discontinued and a solution containing aluminum acetate should be applied once or twice daily to maintain control. This acts as a drying agent and reduces the microbial flora. Basic ointments or plain petroleum jelly, gently applied into the ear canal (without cotton tips), are often helpful to maintain satisfaction of the patient. Topical pimecrolimus is effective.

SEBORRHEIC BLEPHARITIS Special consideration is given to the treatment of seborrheic blepharitis. The use of hot compresses with gentle débridement with a cotton-tipped applicator and baby shampoo one or more times daily is recommended. Stubborn cases may require the use of a topical antibiotic such as sodium sulfacetamide ophthalmic ointment. The use of ocular preparations containing glucocorticosteroids should be deferred to an ophthalmologist. If *Demodex folliculorum* mites occur in large numbers, a trial with antiparasitic drugs such as crotamiton, permethrin, or benzyl benzoate is worthwhile.

Antifungals

Good results are achieved with topical application of antifungal agents, especially imidazoles. Clinical studies have reported response rates ranging from 63 percent[29] up to 90 percent[30] after 4 weeks. In these trials, imidazoles such as itraconazole, miconazole, fluconazole, econazole, bifonazole, climbazole, ciclopirox, and ciclopiroxolamine were studied. The imidazole compound that has been mostly used is ketoconazole. In several clinical studies, 2 percent ketoconazole cream has been found as effective as glucocorticosteroid creams, and this often results in more prolonged remissions.[29,31] Comparative studies of topical antifungal agents, however, are lacking. Personal experience, though based on open uncontrolled studies only, favors 2 percent ketoconazole cream. Other antifungal agents may also be effective. In a limited trial, 1 percent butenafine cream, a benzylamine derivative, demonstrated efficacy in the topical treatment of seborrheic dermatitis.

Oral antifungal agents such as ketoconazole, itraconazole, and terbinafine are also effective, but because of potential side effects and pharmacoeconomic considerations, should probably be limited to severe or refractory cases. Antifungal agents have a wide spectrum of effects, including anti-inflammatory properties and inhibition of cell wall lipid synthesis. This efficacy is not proof of a causal relationship between *M. furfur* and seborrheic dermatitis.

Metronidazole

Topical metronidazole is a worthwhile alternative in the treatment repertoire of seborrheic dermatitis. It has been used successfully in patients with rosacea. Extemporaneous formulations (1 percent to 2 percent in a cream base) or commercial products (0.75 percent gel, cream, or lotion; 1 percent cream) are used once or twice daily. Recently, a significant benefit of using 1 percent metronidazole gel over placebo in the treatment of seborrheic dermatitis was demonstrated.[32] Conversely, 0.75 percent metronidazole gel was shown to have similar efficacy than placebo in the treatment of seborrheic dermatitis.[33]

Lithium

Other topical agents that are effective in the treatment of seborrheic dermatitis are lithium succinate[34] and lithium gluconate,[35] which possess antifungal properties.

Calcineurin Inhibitors

Topical tacrolimus[36] and pimecrolimus[37] may be superior alternatives to corticosteroids as they both have anti-inflammatory properties but do not have long-term side effects. Tacrolimus also exhibits antifungal properties.

Vitamin D_3 Analogues

Vitamin D_3 analogues (calcipotriol cream or lotion, calcitriol ointment, or tacalcitol

ointment) are also recommended and useful in selected patients.[38] Their anti-inflammatory and antifungal properties may be responsible for their efficacy in seborrheic dermatitis.

Isotretinoin

Oral isotretinoin (13-*cis*-retinoic acid) is a useful, although not officially approved, drug for this indication. Low doses (0.05 to 0.10 mg/kg body weight daily) given for several months clear stubborn seborrheic dermatitis in many cases. In women of child-bearing age, all precautions must be met (see Chap. 229).

Phototherapy

Narrow-band ultraviolet B phototherapy appears to be an effective and safe treatment option for patients with severe and refractory seborrheic dermatitis.[39] Psoralen and ultraviolet A light therapy has been used successfully in the erythrodermic form of the disease.[40]

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CHAPTER 23

Exfoliative Dermatitis

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EPIDEMIOLOGY

The incidence of exfoliative dermatitis (ED) has varied widely in studies from 0.9 to 71.0 per 100,000 outpatients.[1–4] Males are affected more commonly than females with a 2:1 to 4:1 male-to-female ratio. The average age of disease onset varies from 41 to 61, with the majority of studies excluding children.

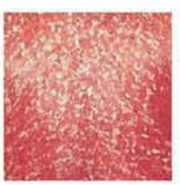
Preceding skin disease plays a role in more than one-half of the cases of ED. Psoriasis is the leading underlying skin disease identified in almost one-fourth of the cases. Recently, ED was reported in 87 of 160 cases of severe psoriasis.[5]

ETIOLOGY AND PATHOGENESIS

ED can be caused by a wide range of cutaneous and systemic diseases (Table 23-1). Data from 18 published studies on ED were compiled to assess etiology.[1,2,4,6–20] Pre-existing skin disease plays a role in approximately 52 percent (range, 27 percent to 68 percent) of ED cases. ED is most commonly caused by psoriasis (23 percent), spongiotic dermatitis (20 percent), drug hypersensitivity reactions (15 percent), and cutaneous T-cell lymphoma (CTCL) or Sézary syndrome (5 percent). In approximately 20 percent of ED cases (range, 7 percent to 33 percent), no underlying etiology is identified and these cases are classified as idiopathic. Less common causes of ED in adults include immunobullous disease; connective tissue disease; infections, including scabies and dermatophytes; pityriasis rubra pilaris (PRP) (4 percent of dermatoses); and underlying malignancy. In neonates and infants, the differential diagnosis includes dermatoses (such as psoriasis, atopic dermatitis, and seborrheic dermatitis), drugs, and infection (particularly staphylococcal scalded-skin syndrome). In addition, several congenital disorders including the ichthyoses, both bullous and non-bullous congenital ichthyosiform erythroderma, Netherton syndrome, and immunodeficiencies should be considered (Box 23-1).

Drugs are implicated in a significant percentage (mean, 15 percent; range, 4 percent to 39 percent) of ED cases, and the list is constantly expanding. Table 23-2 is a listing of drugs reported to cause ED. However, many of these drugs are reported in single case notes. The most commonly implicated drugs include calcium channel blockers, antiepileptics, antibiotics (penicillin family, sulfonamides, vancomycin), allopurinol, gold, lithium, quinidine, cimetidine, and dapsone.

EXFOLIATIVE DERMATITIS AT A GLANCE



- Exfoliative dermatitis (ED) is diffuse erythema and scaling of the skin involving more than 90 percent of the total body skin surface area.
- Common underlying etiologies are psoriasis, atopic dermatitis and other spongiotic dermatoses, drug hypersensitivity reactions, and cutaneous T-cell lymphoma.
- The cause of ED is unknown in approximately 20 percent of cases (idiopathic ED).
- Diagnostic workup includes a complete history, physical examination, dermatohistopathology, and other laboratory work up determined by clinical clues.
- Systemic complications include fluid and electrolyte imbalance, thermoregulatory disturbance, fever, tachycardia and high-output failure, hypoalbuminemia, and peripheral edema.
- Treatment addresses the underlying etiology, symptomatic relief, and potential systemic complications.