

TABLE 72-3 ERYTHRODERMA (PRIMARY CUTANEOUS DISORDERS)

	Initial Lesions	Location of Initial Lesions	Other Findings	Diagnostic Aids	Treatment
Psoriasis ^a	Pink-red, silvery scale, sharply demarcated	Elbows, knees, scalp, presacral area, intergluteal fold	Nail dystrophy, arthritis, pustules; SAPHO syndrome, especially with palmoplantar pustulosis	Skin biopsy	Topical glucocorticoids, vitamin D; UV-B (narrowband) > PUVA; oral retinoid; MTX, cyclosporine, anti-TNF agents, anti-IL-12/23 Ab
Dermatitis^a					
Atopic	Acute: Erythema, fine scale, crust, indistinct borders, excoriations Chronic: Lichenification (increased skin markings), excoriations	Antecubital and popliteal fossae, neck, hands, eyelids	Pruritus Personal and/or family history of atopy, including asthma, allergic rhinitis or conjunctivitis, and atopic dermatitis Exclude secondary infection with <i>Staphylococcus aureus</i> or HSV Exclude superimposed irritant or allergic contact dermatitis	Skin biopsy	Topical glucocorticoids, tacrolimus, pimecrolimus, tar, and antipruritics; oral antihistamines; open wet dressings; UV-B ± UV-A > PUVA; oral/IM glucocorticoids (short-term); MTX; mycophenolate mofetil; azathioprine; cyclosporine Topical or oral antibiotics
Contact	Local: Erythema, crusting, vesicles, and bullae Systemic: Erythema, fine scale, crust	Depends on offending agent Generalized vs major intertriginous zones (especially groin)	Irritant—onset often within hours Allergic—delayed-type hypersensitivity; lag time of 48 h Patient has history of allergic contact dermatitis to topical agent and then receives systemic medication that is structurally related, e.g., thiuram (skin), disulfiram (oral)	Patch testing; open use test Patch testing	Remove irritant or allergen; topical glucocorticoids; oral antihistamines; oral/IM glucocorticoids (short-term) Same as local
Seborrheic (rare in adults)	Pink-red to pink-orange, greasy scale	Scalp, nasolabial folds, eyebrows, intertriginous zones	Flares with stress, HIV infection Associated with Parkinson's disease	Skin biopsy	Topical glucocorticoids and imidazoles
Stasis (with autosensitization)	Erythema, crusting, excoriations	Lower extremities	Pruritus, lower extremity edema, varicosities, hemosiderin deposits History of venous ulcers, thrombophlebitis, and/or cellulitis Exclude cellulitis Exclude superimposed contact dermatitis, e.g., topical neomycin	Skin biopsy	Topical glucocorticoids; open wet dressings; leg elevation; pressure stockings; pressure wraps if associated ulcers
Pityriasis rubra pilaris	Orange-red (salmon-colored), perifollicular papules	Generalized, but characteristic "skip" areas of normal skin	Wax-like palmoplantar keratoderma Exclude cutaneous T cell lymphoma	Skin biopsy	Isotretinoin or acitretin; MTX; perhaps anti-IL-12/23 Ab, IV anti-TNF agent

^aDiscussed in detail in [Chap. 71](#).

Abbreviations: Ab, antibody; HSV, herpes simplex virus; IL, interleukin; IM, intramuscular; IV, intravenous; MTX, methotrexate; PUVA, psoralens + ultraviolet A irradiation; SAPHO, synovitis, acne, pustulosis, hyperostosis, and osteitis (also referred to as chronic recurrent multifocal osteomyelitis); TNF, tumor necrosis factor; UV-A, ultraviolet A irradiation; UV-B, ultraviolet B irradiation.

circulating atypical lymphocytes), while reactions to allopurinol may be accompanied by gastrointestinal bleeding.

The most common malignancy that is associated with erythroderma is CTCL; in some series, up to 25% of the cases of erythroderma were due to CTCL. The patient may progress from isolated plaques and tumors, but more commonly, the erythroderma is present throughout the course of the disease (Sézary syndrome). In the Sézary syndrome, there are circulating clonal atypical T lymphocytes, pruritus, and lymphadenopathy. In cases of erythroderma where there is no apparent cause (idiopathic), longitudinal evaluation is mandatory to monitor for the possible development of CTCL. There have been isolated case reports of erythroderma secondary to some solid tumors—lung, liver, prostate, thyroid, and colon—but it is primarily during a late stage of the disease.

ALOPECIA

([Table 72-4](#)) The two major forms of alopecia are scarring and non-scarring. *Scarring alopecia* is associated with fibrosis, inflammation, and loss of hair follicles. A smooth scalp with a decreased number of follicular openings is usually observed clinically, but in some patients, the changes are seen only in biopsy specimens from affected areas. In *nonscarring alopecia*, the hair shafts are absent or miniaturized, but the hair follicles are preserved, explaining the reversible nature of nonscarring alopecia.

The most common causes of nonscarring alopecia include *androgenetic alopecia*, *telogen effluvium*, *alopecia areata*, *tinea capitis*, and the early phase of *traumatic alopecia* ([Table 72-5](#)). In women with androgenetic alopecia, an elevation in circulating levels of androgens may be seen as a result of ovarian or adrenal gland dysfunction or neoplasm. When there are signs of virilization, such as a deepened voice and enlarged clitoris, the possibility of an ovarian or adrenal gland tumor should be considered.

Exposure to various drugs can also cause diffuse hair loss, usually by inducing a telogen effluvium. An exception is the anagen effluvium observed with antimetabolic agents such as daunorubicin. Alopecia is a side effect of the following drugs: warfarin, heparin, propylthiouracil, carbimazole, isotretinoin, acitretin, lithium, beta blockers, interferons, colchicine, and amphetamines. Fortunately, spontaneous regrowth usually follows discontinuation of the offending agent.

Less commonly, nonscarring alopecia is associated with *lupus erythematosus* and *secondary syphilis*. In systemic lupus there are two forms of alopecia—one is scarring secondary to discoid lesions (see below), and the other is nonscarring. The latter form coincides with flares of systemic disease and may involve the entire scalp or just the frontal scalp, with the appearance of multiple short hairs ("lupus hairs") as a sign of initial regrowth. Scattered, poorly circumscribed patches of alopecia with a "moth-eaten" appearance are a manifestation