FRANCISCO A. KERDEL
FRANCISCO JIMENEZ-ACOSTA

DERMATOLOGY

just the facts

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DERMATOLOGY

Just the Facts

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Just the Facts

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To Isabella, Christina, and Franz
(FAK)

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PREFACE

The impetus for writing this book originated when one of the editors (FAK) held the position of President of the Medical Dermatology Society (US). At the time, it was felt that a publication originating from the Society would enhance visibility and benefit the group. The concept was approved by most of the members and indeed many of the authors are current members of the Medical Dermatology Society. This book is to some degree the product of the Medical Dermatology Society and if the book is successful in promoting the Society, then we will feel that we have accomplished what we set out to do.

We had planned to publish a simple dermatology book which could be translated to many languages over a decade ago. Even though substantial effort was dedicated to the project, the publishing of a book never came to fruition. Through this book, we have been able to finish this project that had the longest gestation period on record.

We wish to acknowledge the Medical Dermatology Society for their support and to the individual authors who participated in the writing of this book. We would also like to thank Veronica A. Montoto for her efforts in reformatting and correcting the individual manuscripts. In addition, we would like to thank Leslie Baumann, MD, who was our initial contact with McGraw-Hill, and Darlene Cooke for believing that the work could be accomplished. Our gratitude is also extended to Professor Renzo Romanelli for helping review the manuscript.

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CONTENTS

1 PAPULOSQUAMOUS DISORDERS

Lela A. Lee
Theresa R. Pacheco

PSORIASIS

EPIDEMIOLOGY

- Psoriasis occurs in about 2% of the U.S. population.
- There are two peaks of onset, one in the third decade and one in the sixth decade. However, psoriasis may begin at any age.
- Onset in childhood portends more severe disease.
- Psoriatic arthritis occurs in 5% or more of patients with psoriasis.

PATHOPHYSIOLOGY

- Psoriasis is a multigenetic disease with environmental triggers. If one parent is affected, about 8% of offspring will develop psoriasis. If two parents are affected, about 40% will be affected.
- Triggers include trauma to the skin, such as a surgical incision, and infection, notably streptococcal infection.
- Considerable evidence implicates T lymphocytes as crucial to the initiation of a psoriatic lesion.
- Psoriasis has a T_H1 phenotype, with potential contributions from the T_H1-type cytokines TNF-α, IL-2, IFN-γ, IL-12, and IL-18. In addition, IL-8 may promote keratinocyte proliferation, recruit inflammatory cells to the skin, and stimulate angiogenesis.
- The importance of T cells in the pathophysiology of psoriasis is highlighted by the effectiveness of therapeutic interventions that target T-cell activation, T-cell proliferation, or migration of T cells into the skin, or that target T_H1-type cytokines.

CLINICAL FEATURES

- Untreated psoriatic lesions are typically sharply demarcated plaques with silvery scale on an erythematous base. When the scale is removed by scraping, small drops of blood appear on the erythematous base, a finding called the Auspitz sign. In some cases, lesions may follow trauma to the skin, such as a scratch or surgical incision. The occurrence of psoriasis along an area of injury to the skin is called the Koebner phenomenon.
- The lesions are not usually pruritic, but may be.
- Exacerbating factors include HIV, heavy alcohol use, use of certain medications such as lithium and beta blockers, and stress. Psoriasis is apt to be worse in climates with relatively little sunshine.
- A severe exacerbation of psoriasis may occur upon withdrawal of systemic steroids, particularly if the systemic steroids have been administered over a long period.
- Psoriasis has many phenotypes, the most common of which is chronic plaque psoriasis. Other phenotypes include guttate, erythrodermic, generalized pustular, and localized pustular psoriasis.
- In chronic plaque psoriasis, large plaques occur predominantly on the elbows, knees, scalp, retroauricular skin, umbilicus, and gluteal cleft. Lesions may occur much more widely over virtually all the skin surface, although the most sun-exposed areas are generally the least likely to be affected. Occasionally, there is a predominance of lesions in intertriginous areas, and in those areas scaling may be inapparent.
- Guttate psoriasis is often associated with a preceding streptococcal infection. Lesions are small, numerous, and widespread, particularly over the trunk and proximal extremities.
- Erythrodermic and generalized pustular psoriasis are dramatic forms of psoriasis. Erythrodermic psoriasis consists of widespread erythema and scale, with erythema over most of the body surface being the predominant finding. High-output cardiac failure may occur in susceptible individuals. In generalized pustular psoriasis, the lesions may appear acutely and be associated with fever, leukocytosis, and debility.
- Localized pustular psoriasis often occurs on hands and feet, and may produce significant functional impairment.
- Nail involvement is common. Pitting of the nails, yellowish discolorations called oil spots, diffuse onychodystrophy, or even loss of the nail may occur.
- Psoriatic arthritis may occur as asymmetric peripheral joint disease, symmetric peripheral joint disease, axial disease, tenosynovitis, and enthesopathy. The most

common type of arthritis is peripheral asymmetric oligoarthritis involving small joints of the hands and feet, large joints of the legs, or both. Although there is some correlation between severity of psoriatic skin lesions and arthritis, arthritis may occur in patients who have minimal skin findings, and the courses of the arthritis and the skin disease are not parallel.

HISTOPATHOLOGY

- The histologic findings vary depending on the stage and phenotype of the lesion. In typical plaque psoriasis, the early findings consist of dilated capillaries and perivascular mononuclear cells. As the lesion matures, there is dramatic epidermal hyperplasia, which is associated with elongation of the rete ridges and thinning of the suprapapillary plates. This brings the congested blood vessels close to the surface and accounts for the Auspitz sign when the scale is removed. Because of the rapid turnover of cells, there is no stratum granulosum, but nuclei may be observed in the stratum corneum (parakeratosis). Characteristically, there are neutrophils in the superficial epidermis.

DIAGNOSIS AND DIFFERENTIAL

- The diagnosis of chronic plaque psoriasis is usually not difficult and is typically made on clinical grounds alone. In difficult cases, and in particular in patients with the erythrodermic form, skin biopsy may be helpful in narrowing the diagnostic possibilities.
- Lesions of nummular eczema may be difficult to distinguish from those of chronic plaque psoriasis. In some instances, the differential diagnosis of chronic plaque psoriasis includes seborrheic dermatitis, cutaneous lupus, pityriasis rubra pilaris, dermatophytosis, or cutaneous T-cell lymphoma.
- The differential diagnosis of guttate psoriasis may include pityriasis rosea, secondary syphilis, and parapsoriasis.
- Erythrodermic psoriasis may be difficult to distinguish clinically from drug eruption, pityriasis rubra pilaris, cutaneous T-cell lymphoma, and severe atopic dermatitis.
- Psoriatic lesions of the nail may be mistaken for fungal infection. Close attention to the nail morphology, and in some cases sampling of the nail for microscopic examination and culture, can help differentiate.
- If the patient has a single psoriasis-like lesion, particularly if it is in an atypical location, the diagnosis of squamous cell carcinoma-in-situ should be considered.

TREATMENT

- Psoriasis is a condition with many, quite distinct treatment options available. Patients who have persistent, disabling, and/or extensive disease are best managed by a physician familiar with the full range of treatment options, including their benefits and side effects.
- Topical therapy or phototherapy is preferred for most patients, as side effects are minimized. Both topical and phototherapy may be quite helpful, even for patients with extensive skin involvement.
- Combination therapies are frequently used.
- Topical medications in frequent use are corticosteroids, tar, anthralin, calcipotriene, and tazarotene. Salicylic acid is sometimes employed to decrease scaling.
- Topical medications differ in ease of use, expense, and duration of remission. Periods of remission may refer either to periods during which there are no psoriatic lesions visible or periods where the psoriasis is still present but of minor severity by comparison to the pre-treatment severity.
- Among topical therapies administered as monotherapy, the duration of remission appears to be better for anthralin and tazarotene than steroids and calcipotriene.
- Topical steroids are easy to use, but cutaneous side effects may be limiting in some patients.
- Tar is often effective but may stain clothing and have an unpleasant odor.
- Anthralin is convenient to use for patients who follow directions closely. Short-contact (from a few minutes up to an hour) anthralin is preferred, as the short contact regimen decreases the likelihood of irritation.
- Calcipotriene is convenient to use and has efficacy comparable to a high-potency topical steroid.
- Tazarotene is convenient to use. It may be irritating to the skin, but the cream preparations are better tolerated than the gels.
- Phototherapy may be administered using natural sunlight, UVB, narrowband UVB, or PUVA (psoralen plus UVA). Many factors are considered when determining which type of phototherapy to use. Natural sunlight is obviously the least expensive.
- Systemic therapy is reserved for patients with extensive or disabling disease. The most commonly used systemic therapies are methotrexate, retinoids (particularly acitretin), and cyclosporine. A limitation of methotrexate and retinoids is their potential for hepatotoxicity. In addition, retinoids are teratogenic, and

are generally not used to treat psoriasis in women of childbearing potential. Cyclosporine has a potential for nephrotoxicity.
- There are a host of other therapies that have been used successfully for psoriasis, including 6-thioguanine, hydroxyurea, tacrolimus, mycophenolate mofetil, and topical 5-fluorouracil.
- Newer or experimental therapies for psoriasis include immune-modulating agents such as etanercept (which binds the TNF-α receptor), infliximab (which binds TNF-α), IL-10 (which inhibits T_H1-type cytokine production), and molecules that block the interactions of B7 and CD28, LFA1 and ICAM-1, or CD2 and LFA3.
- The National Psoriasis Foundation is an excellent resource for patient education.

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SEBORRHEIC DERMATITIS

EPIDEMIOLOGY

- Seborrheic dermatitis is an exceedingly common condition and is usually not an indicator of underlying internal disease.
- Patients with HIV or with neurologic disease such as Parkinson's disease have an increased prevalence and severity.

PATHOPHYSIOLOGY

- The cause is unknown.
- Some evidence implicates *Malassezia furfur*, but there is not a definitive link.

CLINICAL FEATURES

- There is scaling, which is often greasy in appearance, and erythema, which ranges widely in severity from imperceptible to severe.
- Lesions have a symmetric distribution and a predilection for scalp, retroauricular skin, nasolabial area, eyebrows, eyelids, beard area, midchest, and intertriginous skin.
- Tinea amiantacea is a name given to describe thick scaling adhering to the proximal portions of hairs and often binding them together; tinea amiantacea is not a dermatophyte infection.

HISTOPATHOLOGY

- Findings include moderate acanthosis, a mild perivascular mononuclear cell infiltrate, mild spongiosis, parakeratosis, and often some neutrophils at the follicular ostia.

DIAGNOSIS AND DIFFERENTIAL

- Diagnosis is made by clinical examination; skin biopsy is usually unnecessary and nondiagnostic.
- Consider checking HIV in a patient with otherwise unexplained, severe seborrheic dermatitis.
- Differential diagnosis may include psoriasis, atopic dermatitis, rosacea, and perioral dermatitis.
- Scalp psoriasis and seborrheic dermatitis may be indistinguishable. Features more suggestive of psoriasis are discrete, sharply marginated, erythematous, scaly plaques with intervening areas of normal-appearing scalp.
- Although dermatophyte infection of the scalp is common in children, tinea capitis is uncommon in immunocompetent adults and is usually not a likely diagnostic consideration.

TREATMENT

- Medicated shampoo is sufficient for most individuals with the condition limited to the scalp. Shampoo may also be used for scaling in eyebrows and other hairy sites such as the moustache and midchest.
- Seborrhea shampoos may contain ketoconazole, tar, sulfur, selenium sulfide, or zinc pyrithione. Currently, these shampoos are available in the United States without a prescription.
- Shampoos or solutions containing a keratolytic agent such as salicylic acid may be helpful to decrease thick scale in the scalp.
- Shampooing daily to every other day is beneficial, as infrequent shampooing may contribute to seborrhea.

- Because medicated shampoos tend to be more expensive than nonmedicated shampoos, and some medicated shampoos may have a somewhat unpleasant odor, it is often acceptable to alternate the use of nonmedicated shampoos with medicated shampoos. The frequency of use of medicated shampoo necessary to keep the condition under control may be determined empirically.
- For inflamed skin, topical steroids are often used. In the scalp, steroid solutions such as fluocinolone or fluocinonide topical solution used once daily after shampooing may be helpful. On the facial skin or intertriginous areas, twice to thrice daily nonprescription-strength hydrocortisone cream may suffice. If prescription strength steroid is needed, a steroid should be chosen from the low-potency category in order to minimize the likelihood of adverse cutaneous reactions on the face or intertriginous skin.
- Other topical treatments that have been used include imidazole creams, metronidazole, and ciclopirox.

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Lichen Planus and Lichen Nitidus

EPIDEMIOLOGY

- The incidence of lichen planus is not known precisely, but may be about 0.5%.
- Most cases occur in the fourth to sixth decades, but all ages may be affected.

PATHOPHYSIOLOGY

- The histologic findings of a lymphocytic infiltrate with damage to basal cells of the epidermis suggest the possibility of cytotoxicity of basal cells mediated by lymphocytes. There is no evidence that lichen planus is mediated by autoantibodies.
- An association with hepatitis C has been reported in many studies, most commonly in patients with erosive mucosal lesions.

CLINICAL FEATURES

- The characteristic skin lesions are flat-topped, violaceous, polygonal papules, typically rather small in diameter and with a thin, transparent scale.
- Pruritus is extremely common and often intense.
- The above characteristics may be remembered as *p*lanar (flat-topped), *p*urple, *p*olygonal, *p*ruritic *p*apules.
- Lesions are symmetric and occur predominantly on the extremities, with the wrists and flexural areas of arms and legs most likely to be affected, and the trunk and neck occasionally affected. The shaft of the penis is commonly involved.
- The Koebner phenomenon may be observed (see the section on psoriasis earlier in this chapter).
- Oral lesions may occur either coincident with skin lesions or as an isolated finding. Oral lesions often consist of lacy white papules or erosions, although other phenotypes may be observed. The buccal and gingival mucosa and the tongue are often involved. Other mucosal surfaces, particularly genital mucosa, may be affected.
- A small but increased risk for oral squamous cell carcinoma has been reported in patients who have oral lichen planus.
- Nails are affected in a minority of patients. Typical findings are ridging, thinning, distal splitting, and dorsal pterygium. Permanent loss of the nails may occur.
- There are several clinical variants of lichen planus. These include actinic, follicular, erosive, ulcerative, bullous, atrophic, hypertrophic, palmoplantar, annular, and linear lichen planus, and lichen planus-lupus overlap syndrome.
- Lichen planus usually resolves in 1 or 2 years, but relapses may occur. Oral lesions appear to have a longer duration.
- Lichen-planus-like, or lichenoid, eruptions may result from exposure to certain medications or metals. The lesions tend to be somewhat larger in diameter than those of classic lichen planus and the distribution is more often on sun-exposed skin, but it may not be possible to distinguish a lichenoid drug eruption from lichen planus in every case. Some of the drugs that have been reported to induce a lichenoid eruption are gold, beta blockers, captopril, penicillamine, antimalarials, thiazide diuretics, furosemide, and spironolactone. Certain dental materials have been noted to induce oral lichenoid eruptions.
- Lichen nitidus is a condition consisting of numerous pinpoint, flesh-colored or pink, round papules with minimal scale. Most common sites of involvement are the flexural surfaces of the arms and wrists, abdomen, breasts, and genital area. Lesions are usually

asymptomatic. Histology is distinctive. A relationship between lichen nitidus and lichen planus has been proposed, as some patients with lichen planus also have lesions of lichen nitidus, but the relationship of these two conditions has not been definitively established.

HISTOPATHOLOGY

- Lichen planus is characterized by apoptotic damage of basal keratinocytes, a dense band-like infiltrate of mononuclear cells at the dermal–epidermal interface, wedge-shaped hypergranulosis, orthokeratosis, and elongation of rete ridges in a saw-toothed pattern.
- Immunofluorescent examination of lichen planus lesions shows the apoptotic keratinocytes staining heavily with IgM and often IgG, IgA, C3, and fibrin. Fibrinogen deposits in a shaggy pattern at the dermal-epidermal junction are characteristic.
- Lichen nitidus has a well-circumscribed, dense infiltrate of mononuclear cells in a widened dermal papilla. Above the infiltrate there is epidermal thinning, basal cell damage, loss of the granular layer, and parakeratosis. At the lateral aspects of the infiltrate, the rete ridges point toward the infiltrate, giving the appearance of arms enveloping the infiltrate.

DIAGNOSIS AND DIFFERENTIAL

- The differential may include other papulosquamous diseases such as dermatitis, lichen simplex, and psoriasis, but these are usually distinguishable by clinical examination.
- Skin biopsy may be quite helpful in establishing a definitive diagnosis, as the histologic findings are distinctive. In cases of typical cutaneous lichen planus, a biopsy may not be necessary.
- Findings on immunofluorescent examination of tissue are non-diagnostic, but may nevertheless provide supportive information and may help distinguish lichen planus from autoantibody-associated diseases. This is particularly the case in oral lichen planus, where the differential diagnosis may include pemphigus or pemphigoid syndromes.
- The histologic findings of lichenoid graft-versus-host disease, lichenoid keratosis, and lichenoid drug eruption may resemble those of lichen planus.
- Lichen planus lesions of the nail may be mistaken for fungal infection. Close attention to the nail morphology, and in some cases sampling of the nail for microscopic examination and culture, can help distinguish.
- Lesions of lichen nitidus may resemble flat warts, follicular eczema, or keratosis pilaris.

TREATMENT

- Lichen planus is often poorly responsive to therapy. Anecdotal reports of responses to specific therapies are difficult to evaluate, given that the disease spontaneously remits.
- In selecting therapy, potential side effects must be weighed against potential benefits. Topical corticosteroids are often the first agent to be used for cutaneous lichen planus and are usually well tolerated. The potential side effects of systemic steroids usually outweigh potential benefits. Presently, topical tacrolimus has proven useful in oral lichen planus.
- Phototherapy with UVB or PUVA has been used for cutaneous lesions.
- Oral antihistamines may be helpful for decreasing pruritus.
- For disabling oral lesions, systemic retinoids have been used, as has cyclosporine.
- If lichenoid drug eruption is in the differential, consider discontinuing the possible inciting agent.
- Lichen nitidus usually does not require therapy.

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PITYRIASIS ROSEA

EPIDEMIOLOGY

- The incidence of pityriasis rosea (PR) has been reported to be 0.75 per 100 dermatologic patients.
- The peak incidence is in the 20- to 24-year-old age group, with a majority of cases occurring between the ages of 10 and 35.
- The disease is more common in the spring and fall.

PATHOPHYSIOLOGY

- PR is a common, acute, self-limited papulosquamous eruption of unknown cause.
- An infectious, possibly viral, etiology has been proposed and extensively investigated. To date, no specific virus has been conclusively associated with PR.

CLINICAL FEATURES

- The eruption has a characteristic pattern. A single, sharply-defined thin oval plaque (herald patch) first appears, followed by numerous similar-appearing, smaller lesions.
- Most cases have a truncal distribution with sparing of the face, palms, and soles. In approximately 10% of cases, an inverse distribution involving mainly the extremities is seen.
- The rash lasts from 1 to 8 weeks in 80% of the patients with most patients having the rash for about 5 weeks.
- The eruption is usually asymptomatic, but pruritus can occur.

HISTOPATHOLOGY

- There is focal spongiosis with mounds of parakeratosis and a superficial perivascular lymphocytic infiltrate.

DIAGNOSIS AND DIFFERENTIAL

- Differential diagnoses to consider include tinea and secondary syphilis. In selected cases, other papulosquamous disorders may be in the differential diagnosis. Tinea is more likely to be considered when PR is in the isolated, herald patch stage. A scraping of scale to examine for fungus can be helpful.
- Secondary syphilis is more likely to be considered when the eruption is extensive. Secondary syphilis is characterized by discrete pink macules or pink papules with a fine scale distributed over the trunk, and is associated with lymphadenopathy, low-grade fever, malaise, and arthralgias. Skin lesions erupt 3- to 6 weeks after the appearance of the primary chancre. Clinically, secondary syphilis can be differentiated from PR by documenting involvement of the palms and soles, lymphadenopathy, systemic symptoms, and a positive serologic test for syphilis (RPR or VDRL).
- A biopsy specimen is helpful to confirm the diagnosis of PR in atypical cases.

TREATMENT

- Treatment of PR is only required if the lesions are symptomatic. Topical steroids and antihistamines may help relieve pruritus.
- Ultraviolet radiation treatment (UVB or sun exposure) may be helpful in some patients.

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PITYRIASIS RUBRA PILARIS

EPIDEMIOLOGY

- The incidence of pityriasis rubra pilaris (PRP) is not known precisely, but it is quite uncommon.
- PRP occurs equally among male and female patients.
- There are two peaks of onset of the acquired form of PRP, one in the first decade and one in the fifth decade. However, PRP may begin at any age.
- Additionally, there is a familial autosomal dominant form of PRP, which begins in early childhood.

PATHOPHYSIOLOGY

- PRP is a rare disease in which the primary abnormality may be hyperproliferation of the epidermis.
- Vitamin A deficiency or abnormal vitamin A metabolism has been postulated to contribute to the disease, but evidence to support this hypothesis is lacking.

CLINICAL FEATURES

- Five variants have been described. The five categories are the classical adult type, atypical adult type, classical juvenile type, circumscribed juvenile type, and atypical juvenile type. The classical adult type is the most common.
- Orange-red or salmon-colored scaling plaques with sharp borders characterize PRP. There are often areas of uninvolved skin referred to as "islands of sparing."
- The eruption begins on the head and neck and may expand to involve virtually the entire body, resulting in erythroderma.
- The palms and soles become thickened and yellow, resulting in a well-demarcated palmoplantar keratoderma called the "PRP sandal."
- Follicular hyperkeratosis is commonly seen on the dorsal aspects of the proximal phalanges, elbows, and wrists.
- Nail changes include distal yellow-brown discoloration with subungual hyperkeratosis. Complete loss of the nail can occur in severe cases.

- The eruption is usually asymptomatic, but pruritus can occur.
- Prognosis is variable, but about 80% of patients clear spontaneously in several years.

HISTOPATHOLOGY

- There is mild acanthosis without thinning of the suprapapillary plates. This latter feature explains the absence of the Auspitz sign clinically. The stratum corneum shows parakeratosis, which characteristically alternates in both horizontal and vertical directions.

DIAGNOSIS AND DIFFERENTIAL

- There are no specific lab tests to confirm the diagnosis of PRP. The diagnosis is usually made based on a correlation between clinical and histologic findings.
- A biopsy can be useful to rule out other possible papulosquamous and erythrodermic disorders.
- Erythroderma is a reaction pattern of the skin that can occur in the setting of several different skin disorders, most commonly psoriasis, eczema, cutaneous T-cell lymphoma, and drug reactions.

TREATMENT

- Topical care with hydration and emollients reduces fissuring and dryness, providing some patient comfort. Petroleum jelly or equivalent emollients may be used.
- Topical steroids are not helpful.
- The treatment of choice is oral retinoids. The majority of patients experience a significant benefit from the use of oral retinoids. Clinical improvement can be expected within 4 to 6 months. High doses of vitamin A were used before synthetic retinoids became available.
- Immunosuppressive drugs such as methotrexate and cyclosporine have been used in retinoid-resistant cases.

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PARAPSORIASIS

PITYRIASIS LICHENOIDES ET VARIOLIFORMIS ACUTA (PLEVA, ACUTE PARAPSORIASIS, MUCHA–HABERMANN DISEASE)

PITYRIASIS LICHENOIDES CHRONICA (CHRONIC PARAPSORIASIS)

EPIDEMIOLOGY

- The nomenclature of parapsoriasis, pityriasis lichenoides et varioliformis acuta (PLEVA), and pityriasis lichenoides chronica (PLC) has been complicated and confusing. The different forms of parapsoriasis have been classified as large-plaque parapsoriasis, small-plaque parapsoriasis, and pityriasis lichenoides. Of these, only pityriasis lichenoides is discussed further in this chapter. Pityriasis lichenoides can be viewed as a single entity with a spectrum of clinical disease, with PLEVA at one end of the spectrum and PLC at the other end.
- No accurate statistics on the incidence and frequency of pityriasis lichenoides (PLEVA or PLC) exist.
- These dermatoses occur in both children and adults.

PATHOPHYSIOLOGY

- The etiology of pityriasis lichenoides (PLEVA or PLC) is unknown.
- Various infections have been proposed as causative agents. Based on the current state of knowledge, no known infectious agents have been conclusively associated with PLEVA or PLC.

CLINICAL FEATURES

- PLEVA begins with the appearance of numerous erythematous, edematous papules that may vesiculate. Some lesions develop central necrosis and heal with varioliform scars. At any point in time, the lesions are in multiple stages.
- The papules are predominantly present on the trunk, arms, and legs, sparing the palms and soles. The face is usually spared.
- Lesions are often pruritic.
- PLC is characterized by erythematous, tan or red-brown, scaly papules. Lesions are typically about 3 to 4 mm in diameter, although lesions up to 1 cm in