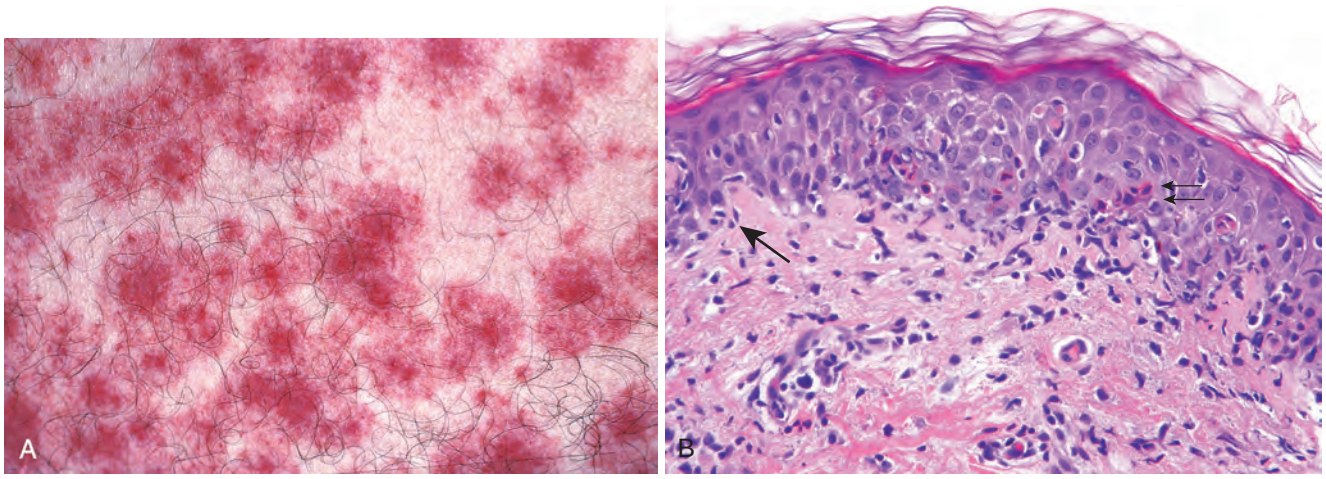

Figure 25-24 Erythema multiforme. **A,** The target-like lesions consist of a central blister or zone of epidermal necrosis surrounded by macular erythema. **B,** An early lesion shows lymphocytes accumulating along the dermoepidermal junction where basal keratinocytes have begun to become vacuolated (arrow). With time, necrotic/apoptotic keratinocytes appear in the overlying epithelium (double arrow).

Chronic Inflammatory Dermatoses

This category includes inflammatory skin disorders that persist for many months to years. The skin surface in some chronic inflammatory dermatoses is roughened as a result of excessive or abnormal scale formation and shedding. However, not all scaling lesions are inflammatory; witness the hereditary ichthyoses, described earlier, with extensive scale due to defects in desquamation.

Psoriasis

Psoriasis is a chronic inflammatory dermatosis that appears to have an autoimmune basis. It is a common disorder, affecting as many as 1% to 2% of people in the United States. Persons of all ages may develop the disease. Approximately 15% of the patients with psoriasis have associated arthritis. Psoriatic arthritis may be mild or may produce severe deformities resembling the joint changes seen in rheumatoid arthritis. It can affect any joint in the body and may be symmetrical or affect one side only. In addition, psoriasis may also be associated with myopathy, enteropathy, and AIDS.

Pathogenesis. Psoriasis results from interactions of genetic and environmental factors. As in the case of many autoimmune diseases it is linked to genes within the HLA locus. There is a strong association with HLA-C, particularly with the *HLA-Cw*0602* allele. About two thirds of affected individuals carry this allele, and homozygotes for *HLA-Cw*0602* have a 2.5-fold higher risk for developing psoriasis than do heterozygotes. Conversely, only about 10% of *HLA-Cw*0602* heterozygotes develop psoriasis, indicating that other factors interact with this MHC molecule to cause disease susceptibility. The culprit antigens remain elusive, but it appears that sensitized populations of CD4+ T_H1 and T_H17 cells and activated CD8+ cytotoxic effector T cells enter the skin and accumulate in the epidermis. These T cells may create an abnormal microenvironment by stimulating the secretion of cytokines and growth factors that induce keratinocyte proliferation, resulting in the characteristic lesions. The interactions between CD4+ T cells, CD8+ T cells, dendritic cells, and keratinocytes give rise to a cytokine "soup" dominated by T_H1-type and T_H17-type cytokines such as IL-12, interferon-γ, tumor necrosis factor (TNF), and IL-17. The importance of these factors is highlighted by the generally excellent clinical responses that are observed in patients treated with therapies that block TNF function. Lymphocytes also produce growth factors for keratinocytes that may contribute to epidermal thickening. Psoriatic lesions can be induced in susceptible individuals by local trauma, a process known as the *Koebner phenomenon*, presumably because trauma sets in motion a local inflammatory response that becomes self-perpetuating.

MORPHOLOGY

Psoriasis most frequently affects the skin of the elbows, knees, scalp, lumbosacral areas, intergluteal cleft, and glans penis. The typical lesion is a well-demarcated, **pink to salmon-colored plaque covered by loosely adherent silver-white scale** (Fig. 25-25*A*). Variations exist, with some lesions occurring in annular, linear, gyrate, or serpiginous configurations. Psoriasis is one cause of total body erythema and scaling known as erythroderma. **Nail changes** occur in 30% of cases of psoriasis and consist of yellow-brown discoloration (often likened to an oil slick), with pitting, dimpling, separation of the nail plate from the underlying bed (onycholysis), thickening, and crumbling.

Established lesions of psoriasis have a characteristic histologic picture. Increased epidermal cell proliferation results in marked epidermal thickening (acanthosis), with regular downward elongation of the rete ridges sometimes described as appearing like test tubes in a rack (Fig. 25-25*B*). Mitotic figures are easily identified well above the basal cell layer, where mitotic activity is confined in normal skin. **The stratum granulosum is thinned or absent, and extensive overlying parakeratotic scale is seen.** Typical of psoriatic plaques is thinning of the portion of the epidermal cell layer that overlies the tips of dermal papillae (suprapapillary plates) and dilated, tortuous blood vessels within these papillae. This constellation of changes results in abnormal proximity of vessels within the dermal papillae to the overlying parakeratotic scale, and accounts for