Acute Eczematous Dermatitis

The Greek word *eczema*, meaning "to boil over," vividly describes the appearance of acute eczematous dermatitis one of the most common skin disorders. Based on initiating factors, eczematous dermatitis can be subdivided into the following categories: (1) allergic contact dermatitis, (2) atopic dermatitis, (3) drug-related eczematous dermatitis, (4) photoeczematous dermatitis, and (5) primary irritant dermatitis.

The causes of eczema are sometimes broadly separated into "inside" and "outside" types: disease resulting from external application of an antigen (e.g., poison ivy) or a reaction to an internal circulating antigen (which may be derived from ingested food or a drug). Treatment involves a search for offending substances that can be removed from the environment. Topical steroids nonspecifically block the inflammatory response. While such treatments are only palliative and do not cure, they are nevertheless helpful in interrupting acute exacerbations of eczema that can become self-perpetuating if unchecked.

Pathogenesis. **Eczematous dermatitis typically results from T cell-mediated inflammatory reactions (type IV hypersensitivity).** This has been well studied in dermatitis triggered by contact antigens (e.g., uroshiol from poison ivy). It is believed that reactive chemicals introduced at the epidermal surface modify self proteins, acting as "haptens", and these proteins become neoantigens. The antigens are taken up by Langerhans cells, which then migrate by way of dermal lymphatics to draining lymph nodes. Here the antigens are presented to naive CD4+ T cells, which are activated and develop into effector and memory cells (Chapter 6). On antigen reexposure, memory T cells expressing homing molecules such as common lymphocyte antigen and particular chemokine receptors migrate to skin sites of antigen localization. Here they release the cytokines and chemokines that recruit the numerous inflammatory cells characteristic of eczema. This process occurs within 24 hours and accounts for the initial erythema and pruritus that characterize cutaneous delayed hypersensitivity in the acute, spongiotic phase.

Langerhans cells within the epidermis play a central role in contact dermatitis, and understandably factors that affect Langerhans cell function impact the inflammatory reaction. Chronic exposure to UV light is injurious to epidermal Langerhans cells and can prevent sensitization to contact antigens, although UV light can also alter antigens and generate forms that are more likely to induce sensitivity reactions.

MORPHOLOGY

All types of eczematous dermatitis are characterized by red, papulovesicular, oozing, and crusted lesions that, if persistent, develop reactive **acanthosis** and **hyperkeratosis** that produce raised scaling plaques (Fig. 25-22). A striking example of

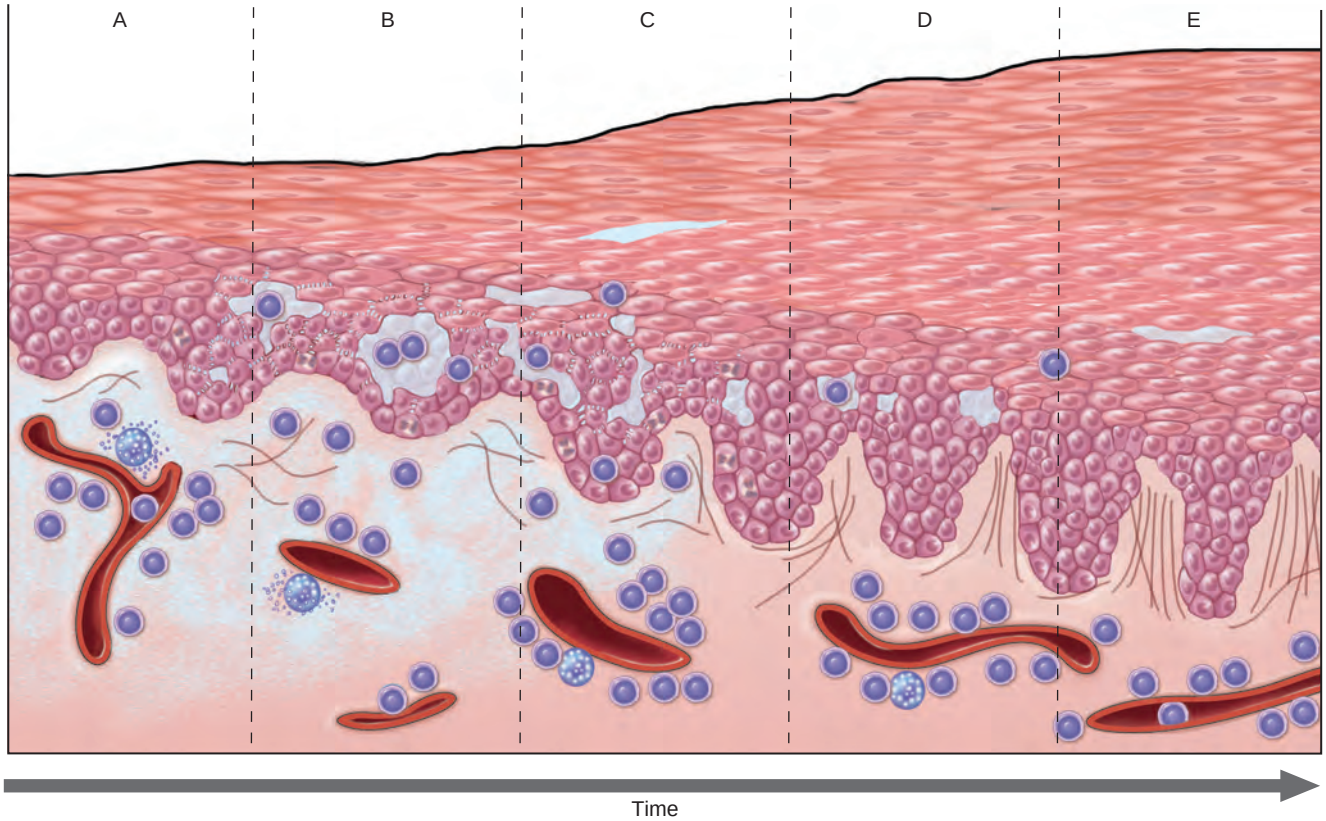

Figure 25-22 Stages of eczema development. **A,** Initial dermal edema and perivascular infiltration by inflammatory cells is followed within 24 to 48 hours by **(B)** epidermal spongiosis and microvesicle formation. **C,** Abnormal scale, including parakeratosis, along with progressive acanthosis **(D)** and hyperkeratosis **(E)** appear as the lesion becomes chronic.