Chronic Cutaneous (Discoid) Lupus Erythematosus

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Discoid lupus erythematosus is the most common form of cutaneous lupus erythematosus. It is a chronic, indolent skin disease with a low incidence of systemic involvement. Chronic cutaneous lupus erythematosus, or simply discoid lupus, is an indolent skin disease usually occurring between age 20 and 45 years. It is more common in females, and may be more common in African-Americans.

Figure 1. Chronic cutaneous lupus. The face is the most commonly affected area. Epidermal atrophy occurs early and gives the surface either a smooth and white or a wrinkled appearance. From Habif. Copyright 2001. With permission from Elsevier.

Figure 2. Chronic cutaneous lupus. Follicular plugs may be prominent. These lesions progress, ultimately forming smooth and white, or hyperpigmented depressed scars with telangiectasia and scarring alopecia. From Habif. Copyright 2001. With permission from Elsevier.
Discoid lupus typically presents with bright red papules, which evolve into sharply margined plaques having adherent scaling. Plaques are round or oval, annular or polycyclic, with irregular borders. Often they expand in the periphery and regress in the center, resulting in depression of lesions, atrophy and eventually scarring. Follicular plugs are prominent; peeling the scale back reveals an undersurface resembling a carpet penetrated by tacks. While active lesions are bright red, “burned-out” lesions may be pink or white, smooth, atrophic and scarred. Often, a still active inflammatory and raised border surrounds smooth whitish-scarred lesions (Figures 1 and 2). Persons with brown or black skin may actually develop hyperpigmentation in more advanced, atrophied lesions.

Discoid lupus may occur on any body surface, may be localized or generalized, and most often involves the scalp, face and ears. Mucous membrane involvement occurs less than 5% of the time. It begins asymptotically and endures for months or years. Lesions occasionally regress, but usually evolve with atrophy and scarring, which destroys hair follicles and results in a haphazard irreversible alopecia. Lesions may be initiated and exacerbated by trauma and ultraviolet B light.

Diagnosis is clinical and confirmed by histology. Dermatopathology shows hyperkeratosis, atrophy of the epidermis, follicular plugging and liquefaction degeneration of the basal cell layer. Immunofluorescence is positive in active lesions by 6 weeks. The presence of granular deposits of IgG and IgM at the dermal-epidermal junction is known as a positive lupus band test and can be found in 90% of active lesions. The differential diagnosis includes actinic keratosis, plaque psoriasis, polymorphic light eruption, and lichen planus.

Treatment involves routine use of sunscreens, topical and intralesional fluorinated glucocorticoids, oral hydroxychloroquine occasionally supplemented with quinacrine and oral retinoids.

CAVEAT

Discoid lupus is a chronic indolent skin disorder with no significant excess mortality. There is a low incidence of systemic disease; only 1% to 5% of cases progress to systemic lupus erythematosus. With localized skin involvement, complete remissions occur in 50%, but with more generalized involvement, remissions are less frequent (less than 10%). In some cases, however, discoid lupus can be the presenting cutaneous sign of systemic lupus erythematosus.

REFERENCES