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**Cutaneous lesions on the legs**

A 34-year-old woman had several symmetrically located, well-circumscribed, non-ulcerating, waxy, red-brown plaques on her lower limbs [Figure 1]. The first lesion appeared 13 years ago. She was concerned about the cosmetic appearance.

**Questions**

1. What is the diagnosis?
2. What is the systemic association with this dermatosis?
3. How are these lesions managed?

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These cutaneous lesions are characteristic of *necrobiosis lipoidica* (NL). They are typically multiple, bilateral, and located on the lower legs, most commonly pretilibially and occasionally on the thighs, ankles, and feet, but rarely on the trunk, upper limbs, and scalp. Early lesions appear as rounded, dull red, asymptomatic papules or plaques that progress slowly and indurate with central atrophy. The lesions have a shiny surface and a waxy, yellowish central area with prominent telangiectasias. The margins may carry comedone-like plugs. Koebner’s phenomenon occurs in some patients. The clinical and histopathological differential diagnoses include rheumatoid nodules, granuloma annulare, necrobiotic xanthogranuloma, sarcoidosis, morphea, stasis dermatitis, subacute nodular migratory panniculitis, erythema nodosum, lichen sclerosus et atrophicus, and Hansen’s disease.

An association with diabetes mellitus has been recognized for a long time. NL was originally termed *dermatitis atrophicans lipoidica diabeatica* (Oppenheim 1929) and later renamed *necrobiosis lipoidica diabetorum* (Urbach 1932). In one large series, 111 of 171 patients (65%) had diabetes mellitus at presentation; it preceded the onset of diabetes in 15% of patients. Its prevalence is 0.3% in people with diabetes, is three times commoner in women, and occurs usually before 30 years of age. In our patient, Type 1 diabetes occurred two years prior to the first lesion. Her glycaemic control was poor and she had diabetic retinopathy as well.

The aetiology remains obscure, but is not a microangiopathy and not associated with glycaemic control or chronic diabetic complications. Antibody-mediated vasculitis and abnormalities of collagen are the other chief putative mechanisms. Histologically, NL occurs as palisading or pseudotuberculoid granulomatous lesions consisting of foci of degenerate collagen bundles with a hyalinized appearance, surrounded by fibrosis, a diffuse infiltrate of histiocytes and a giant-cell granulomatous reaction. Capillary wall thickening and microvascular occlusion are often present.

Treatment of NL is unsatisfactory with cosmetic camouflage the preferred option. Regression of lesions does not correlate with improved glycaemic control. Topical or intralesional corticosteroids may improve early NL. Psoralens and ultraviolet A (PUVA) therapy can improve patients not responsive to steroids. Antiplatelet therapy with aspirin and dipyridamole has shown no benefit but anecdotal reports with pentoxifylline, tretinoin, nicotinic acid, topical tacrolimus, cyclosporine, and infliximab have all documented benefits. Excision and skin grafting may help some. Other complications include ulceration following trauma, occasionally infections and rarely squamous cell carcinoma.

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