



## Unsightly rash on shin

What started as a few small red spots was now an 8 × 6 cm well-demarcated red-brown plaque on the patient's shin. Was it linked to her diabetes?

A 60-YEAR-OLD WOMAN came into our primary care clinic and asked that we look at a rash on her left shin. The rash started as a few small red spots 8 months earlier that gradually merged and turned into one shiny irregular area with brownish discoloration. The rash was neither itchy nor painful, and remained unchanged during exposure to sunlight.

The patient indicated that she had been diagnosed with type 1 diabetes when she was 24 years old and was using a continuous subcutaneous insulin infusion device. She said that over the years she'd developed diabetic

retinopathy, polyneuropathy, and end-stage renal disease requiring hemodialysis.

Clinical exam revealed an 8 × 6 cm well-demarcated red-brown irregular plaque on her left shin. The lesion had a yellow atrophic center and extensive telangiectasias (FIGURE). There were no similar lesions anywhere else on her body.

- WHAT IS YOUR DIAGNOSIS?
- HOW WOULD YOU TREAT THIS PATIENT?

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**FIGURE**

**Red-brown plaque over left shin with yellow atrophic center and extensive telangiectasias**



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**Necrobiosis lipoidica diabetorum does not correlate with the presence or progression of vascular complications of diabetes.**

### Diagnosis: Necrobiosis lipoidica diabetorum

Necrobiosis lipoidica diabetorum (NLD) is a disorder involving subcutaneous collagen degeneration that results in thickening of blood vessel walls, fat deposition, and the formation of granulomas. It affects up to 1.2% of all patients with type 1 and type 2 diabetes, and occurs in patients without diabetes, although it is less common.<sup>1</sup> NLD mostly affects females, with a ratio of 3:1.<sup>2</sup> It's also been described in patients with rheumatoid arthritis, in whom lesions are mostly ulcerated.<sup>3</sup>

The lesions typically start as multiple shiny, painless, red-brown patches that tend to appear in the lower extremities and slowly coalesce and enlarge over months to years, forming yellow atrophic plaques. The thin overlying epidermis contains many telangiectatic vessels.<sup>4</sup> In rare cases, chronic lesions have developed into squamous cell carcinomas.<sup>5</sup>

■ **A diagnosis is established** based on the appearance of the rash and a suggestive history—as was the case with our patient. Lab tests generally aren't required, but should be performed to check for diabetes (if a diagnosis has not been established) and to explore relevant differential diagnoses.

### The differential Dx includes granuloma annulare

In its early stages, the superficial annular lesions of NLD closely resemble granuloma annulare, which is characterized by violaceous or skin-colored annular rings with firm papules or nodules. Skin manifestations of sarcoidosis may also resemble the condition, but associated systemic manifestations help distinguish the two. Rarely, paraproteinemias can develop similar lesions (known as necrobiotic xanthogranuloma) that are associated with elevated blood levels of paraproteins.

### The cause

The exact etiology of NLD is not known but a number of factors have been implicated, including microangiopathy, local trauma, metabolic changes (eg, glycoprotein deposition in the vascular endothelium, increased plate-

let aggregation), and immune-mediated deposition of immunoglobulins and fibrinogen in the vascular walls.<sup>6</sup>

■ **The histologic picture reveals** layers of subcutaneous and intradermal interstitial and palisade granulomas. These granulomas are made up of histiocytes and sometimes eosinophils. Surrounding areas show significant degeneration of collagen and nerve endings. Hence, the lesions are generally painless. Surface trauma to the lesions creates ulcerations that occasionally lead to pain. Vasculitic involvement of the traumatic plaque may demonstrate Koebner phenomenon.<sup>7</sup>

■ **No correlation.** NLD does not correlate with glycemic control or with the presence or progression of vascular (or other) complications of diabetes.<sup>8</sup> It can, however, be a clue to the presence of diabetes.

### Management: Support stockings, steroids

A lack of a clear etiology for NLD makes the treatment challenging. Leg rest may retard progression by alleviating lower extremity edema, and elastic support stockings may be used to protect against trauma (and thus, ulceration).<sup>4</sup> Topical and intralesional steroids are helpful for inflammation, but be cautious when using them in atrophic lesions, as they may worsen atrophy. (Steroids should be avoided in lesions with advanced atrophy.) Topical steroids should be started with a low potency formulation (hydrocortisone 2.5% cream) and gradually advanced to a higher potency formulation (clobetasol propionate 0.05% cream).<sup>9</sup>

Various other therapeutic interventions have been shown to be effective, including tacrolimus ointment 0.1% applied twice daily for one month to prevent T-cell activation<sup>10</sup> and antiplatelet aggregation therapy with aspirin and dipyridamole for 8 weeks.<sup>11</sup> A multicenter prospective study showed an improvement of lesions in about two-thirds of patients when topical 0.005% psoralen was applied, followed by twice weekly ultraviolet-A irradiation for a mean of 22 exposures.<sup>12</sup> Perilesional heparin injections of 5000 IU have also been shown to improve lesions by preventing micro-occlusion.<sup>13</sup> Surgical thera-

pies such as excision and grafting<sup>14</sup> and pulse dye laser<sup>15</sup> have shown promise in selected cases.

**The prognosis**

The prognosis of NLD is poor from a cosmetic point of view. Therefore, early treatment should be offered to retard its progression.

■ We advised our patient to wear elastic

support stockings to protect the affected area from trauma. We also told her to apply moisturizing lotion 4 times a day, as well as topical hydrocortisone 2.5% ointment 2 times a day for 8 weeks.

JFP

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