



SKIN MANIFESTATIONS OF INTERNAL DISEASE

UNILATERAL PRESENTATION OF NECROBIOSIS LIPOIDICA IN A NON-DIABETIC PATIENT

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Background: Necrobiosis lipoidica (NL) is rare, chronic, idiopathic granulomatous disease strongly associated with diabetes mellitus (DM), usually, type 1. Characteristically it affects bilateral lower legs of middle-aged women. Clinical presentation may vary from red-brown papules to sharply demarcated yellow-brown plaque with atrophic centre. Ulceration, usually following a mild trauma, is not rare, especially in individuals suffering from DM. Although there are several different theories of the etiology and pathogenesis of NL, most of them emphasize an important role of microangiopathy.

Observation: Here we report a female 57-years old patient with a solitary indurated, egg-sized plaque with brownish-red margins on her left lower extremity, diagnosed as NL upon biopsy and histological evaluation. Before she had developed NL, she was followed-up at our clinic for relapsing pigmented purpuric dermatosis on her lower extremities. At that time she had no history of DM or disrupted glycemic control. Blood test showed increased levels of total IgE, however, PRICK skin test and epicutaneous test remained negative. Doppler ultrasound imaging of lower-extremity veins and arteries showed no abnormalities. Further examination revealed repeatedly slightly increased levels of d-dimers (605, ref value 0-500 ug/L FEU) and fibrinogen 4.1 (ref. values 1.8-3.5 g/L), while coagulation test remained normal, and a positive ANF (1:320).

Key message: NL can present unilaterally and in patients with no history of DM or disrupted glycemic control. Although NL occurs in only 0.3% to 1.2% of DM patients, and there is no proven connection between the level of glycemic control and the likelihood of developing NL, it should be noted that NL can precede DM in up to 14% of the cases. Thus patients with manifested NL should be regularly checked for glycemic control. Additionally, the case of our patient adds to the theory implicating antibody-mediated vasculitis along with enhanced platelet aggregation and coagulation in the development of NL.

