Lymphocytic infiltration of Jessner-Kanof (LIJK) usually presents with asymptomatic, erythematous, discoid lesions that occur on the face, central chest and upper back of middle-aged adults. The disorder has a prolonged course and resolves without residual scarring (1–5). LIJK nosology is still a point of debate, due to clinical and histological overlaps with other conditions, such as lupus erythematosus tumidus (LET), reticular erythematosus mucinosis (REM), polymorphic light eruption and pseudolymphoma (2–6). The aetiology is poorly understood and to date only a few cases of LIJK caused by drug administration have been described.

CASE REPORT

A 55-year-old woman presented a 4-month history of an asymptomatic, non-scaly, slightly elevated plaque, pinkish to reddish in colour, starting as small multiple coalescing papules expanding peripherally and clearing in the centre, configuring a circinate lesion on her back (Fig. 1a). The skin lesion was about 7 cm in diameter and was reported to have arisen about 6 weeks after starting therapy with duloxetine 60 mg/day for fibromyalgia. A few similar annular erythematous lesions, 2–4 cm in diameter, appeared on her upper right arm 2 weeks after our first observation. For several years the patient had been taking chlorthalidone, amlodipine and ramipril for blood hypertension, atorvastatine for hypercholesterolaemia and acetysalicylic acid for chronic vascular encephalopathy. She was furthermore affected with vulvar lichen sclerosus. The patient denied having known drug allergies and history of photosensitivity. The skin lesions were treated with both topical and systemic glucocorticoids without improvement.

Findings from a punch-biopsy specimen from the lesion of the back revealed a moderately dense lymphocytic infiltrate of the superficial and mid-dermis with perivascular and perifollicular distribution (Fig. 1b, c). The infiltrate was predominantly composed of mature T lymphocytes (Fig. 1d); some B lymphocytes and rare plasma cells were present. Staining for mucin was negative. The epidermis was unaffected and only slightly flattened. Direct immunofluorescence was negative.

All routine blood tests including differential blood count, erythrocyte sedimentation rate, C-reactive protein, Borrelia serology, complement levels, liver and renal function tests were normal or negative. A complete screening for autoantibodies panel revealed positive anti-nuclear antibodies (ANA) with a titre of 1:320 and a speckled pattern. Extractable nuclear antigens (ENA), anti-double-stranded DNA antibodies, anti-histone antibodies, lupus anticoagulant and anti-cardiolipin antibodies were negative. There was neither clinical nor instrumental (chest X-ray, abdomen and regional lymph node sonography, capillaroscopy) evidence of any systemic involvement.

Suspecting a drug-induced pseudolymphoma with LIJK features, duloxetine was discontinued and substituted with sertraline 50 mg/day. The skin lesions spontaneously vanished within 3 weeks. Clinical remission is still persisting after 6 months of duloxetine withdrawal, while the patient is continuing with the other treatments.

DISCUSSION

LIJK is a relatively uncommon and benign disorder first described in 1953 (1). It consists of solitary or multiple, erythematous discoid papules and plaques, which can frequently develop a clear centre that results in an annular and arciform appearance (2–5). The disease is localised mainly on the face, neck, upper trunk and proximal extremities without systemic involvement. Exacerbation by UV light has been reported, although with a delayed appearance, but is not the rule and the
The case reported seems to indicate that LIJK, hitherto considered a disease of unknown aetiology (7), might be included among the cutaneous patterns of drug-induced reactions in susceptible patients.

REFERENCES


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