

Discoid Lupus Erythematosus-Like Lesions in Lyme Disease

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SUMMARY

Lesions of discoid lupus erythematosus (DLE) have not been reported as presenting findings in Lyme disease. Our patient had atrophic alopecia and somewhat erythematous scalp lesions which, on clinical and histopathologic examinations, were highly suggestive of DLE. Serum immunofluorescence test for *Borrelia burgdorferi* was positive with high titers, and the patient's lesions subsided after antibiotic treatment. Isolated lesions of discoid lupus erythematosus should be considered in the differential diagnosis of dermatologic presentations for Lyme disease.

We report a case of discoid lupus erythematosus (DLE) of the scalp in a 49 year-old woman who after serologic examination proved to have high titers of antibodies to *Borrelia burgdorferi*; the causative agent for Lyme Disease (LD). Up to now, DLE had not been considered as a presenting sign for LD; and in discussions on DLE no reference could be found to LD. Erythematous skin eruptions have been seen in LD^{1,2} but no mention of DLE lesions has been made in description of skin lesions in LD.

Case Report:

A forty-nine-year-old healthy white woman was seen by one of us (HN) because of developing four small and non-pruritic bald

patches on her scalp. These had been discovered by her hairdresser about two weeks prior to our examination. The patient had not been aware of these lesions and this was the first time that they had been noticed although she had been going to her hairdresser for some time on a regular basis.

History:

Her past medical history showed no prior scalp lesions and the patient denied having any other skin lesions. Her family history was not contributory to DLE or other connective tissue disease. On further questioning, she denied recent ticks or other insect bites, and she did not have house pets.

Physical Examination

The patient's general appearance was normal and she did not show any facial or other exposed skin lesions besides the four isolated small and rather round bald patches on the scalp (Figs. 1,2) which had minimal scaling with slight depression and induration.

Three of the four lesions were surrounded by two or three millimeters of erythema and some of them showed also few follicular plugging. The lesions ranged from 0.5cm to 1.5cm in diameter. The examination of her fingers showed mild onychotillomania, but there was no cuticular telangiectasia. The review of the other organs did not reveal any abnormalities.



Fig.1: Somewhat atrophic scalp bald patches with slight erythema and minimal scaling.

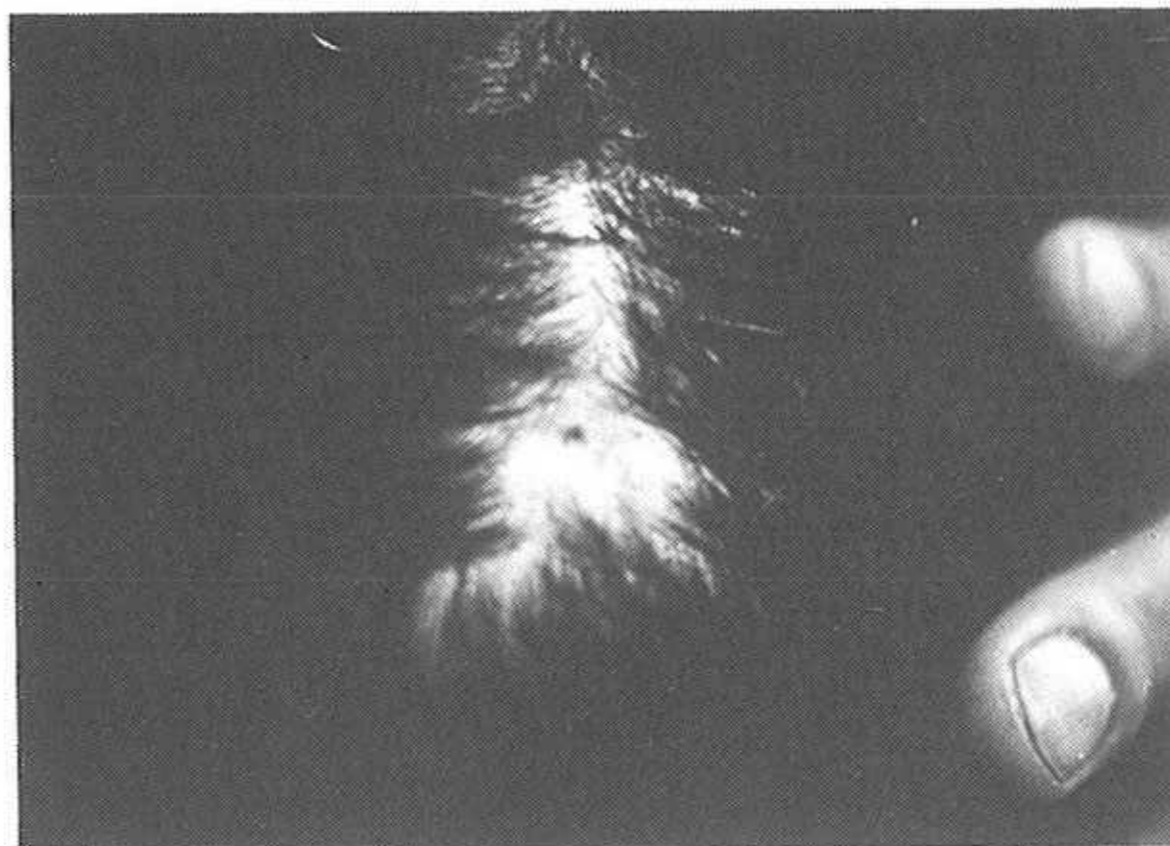


Fig.3: Atrophic discoid lupus erythematosus-like presentation (H&E x 40 original magnification).

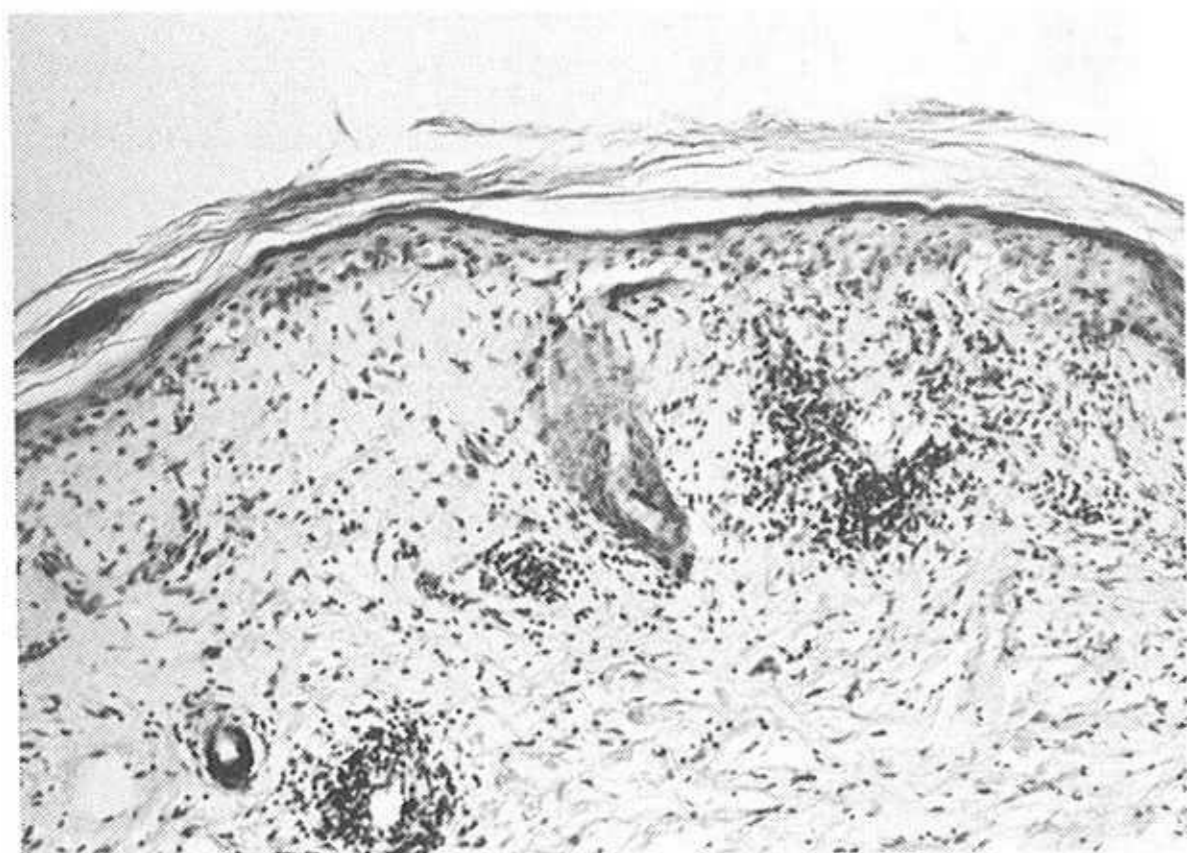


Fig.2: Alopeciac plaque with atrophy.

Laboratory Findings

The blood and urine studies including complete blood count and differential, erythrocyte sedimentation rate, SMA12 and urinalysis were within normal limits, and ANA was negative. Serum immunofluorescence test for *Borrelia burgdorferi* was positive with titer of 1/1024 (non-reactive if less than 1/256). Search for cryoglobulinemia was negative.

Histopathologic Examination

Four millimeter punch biopsy specimen was taken from the largest erythematous scalp lesion. The hematoxylin-eosin stained sections (Fig.3) show irregular epidermal atrophy,

covered with slight hyperkeratosis. There are extensive areas of erosion of the epidermal basal cell layer and impingement of inflammatory cells (Fig.4). In the corium

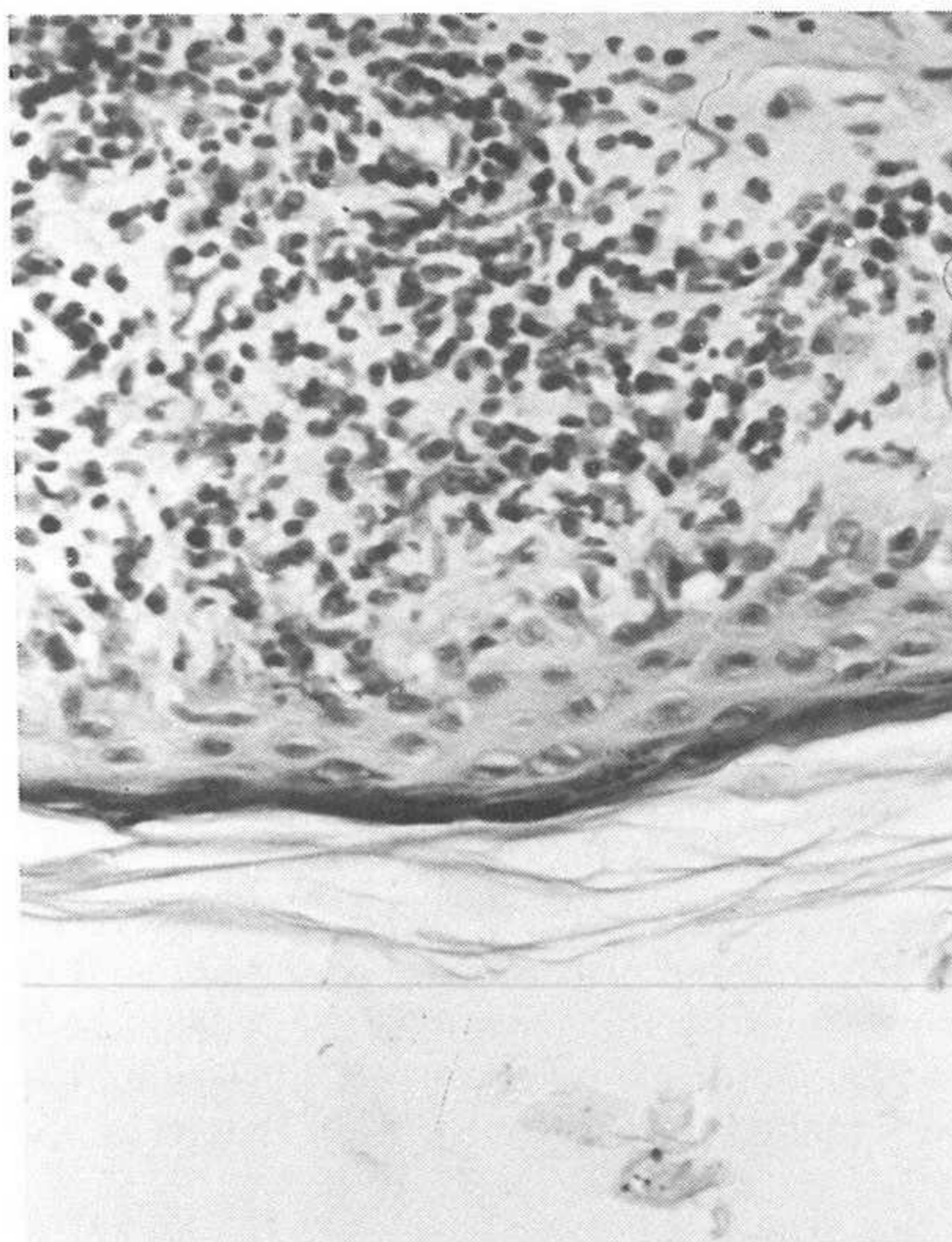


Fig. 4: Lichenoid changes of the epidermis with heavy lymphocytic infiltrate. (H&E x 100 original magnification).

(Fig.5) there is diffuse edema and rather heavy lymphocytic inflammatory infiltrate extending around atrophic and dilated hair follicles, some of which contain keratinous plugs (Fig.6). The inflammation in the corium extends deeply down to the level of subcutaneous fat tissue in some sections. The sections stained with PAS technique show no evidence of fungal bodies. The combination of these findings are highly suggestive of inflammatory lesion of DLE. Silver stains for *Borrelia* were negative in two sets of sections attempted at two laboratories.

Treatment and Clinical Course:

The patient was treated with Doxycycline monohydrate (Monodox) 100mg twice a day for six weeks. The erythematous background of the scalp lesions disappeared and a few new

hairs were growing on last examination. The patient has been doing well for the last six months and has not developed any new lesions.

Discussion

Appearance of similar or indistinguishable skin lesions due to unrelated underlying

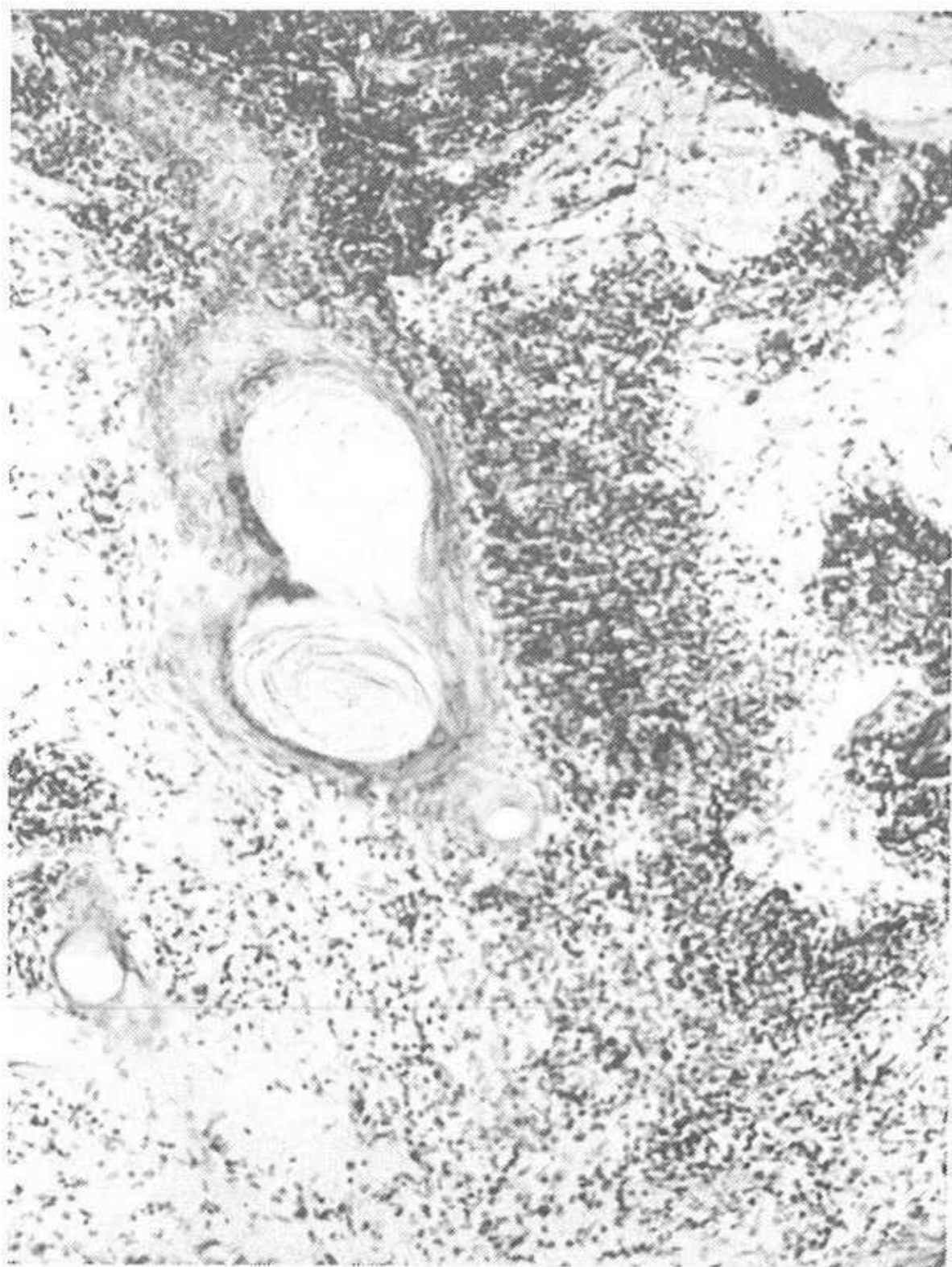


Fig.5: Atrophic hair follicles with dermal diffuse infiltrate of lymphocytes. (H&E x 40 original magnification).



Fig.6: Dilated hair follicle with atrophic walls and keratin plug showing perifollicular infiltrate. (H&E x 100 original magnification).

conditions have been reported, such as a case of tinea capitis due to *trichophyton tonsurans* presenting as discoid lupus erythematosus³ There could also be cases with two separate conditions such as *Borrelia* infection and systemic lupus erythematosus with similar clinical and laboratory findings causing confusion and resulting in misdiagnosis.⁴ On the other hand, there are disease that show some common pathways in the production of

skin lesions or laboratory findings,⁵⁻⁷ but no definite overlap. There could also be cases where one disease is a very good imitator and has laboratory or clinical presentations reminiscent of several other diseases. Such is the case of Lyme disease⁸ which shows a variety of clinical and pathological conditions and lesions having histological features in common with collagen-vascular diseases. Our case is a good instance of this last group and it can be assumed that the serologic and cellular changes seen in this patient are produced in reaction to *B. burgdorferi* in the tissues.

Although we could not show positive staining for *B. burgdorferi* in the tissue samples in our patient, the histopathologic changes resembling or indistinguishable from atrophic DLE that are seen in our patient's lesions must have resulted from the interaction of *B. burgdorferi* and the skin constituents of the patient. In a case of benign lymphocytic infiltrate, polymerase chain reaction assay of *B. burgdorferi* DNA was positive⁹ and in another case, stimulation of cutaneous T-cell lymphoma cells with superantigenic staphylococcal toxins has been reported.¹⁰ It is reasonable to consider a parallel manner of stimulation of the skin lymphocytes in some cases of Lyme disease to affect DLE-like lesions such as seen in our case.

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