

A Case of Discoid Lupus Erythematosus Masquerading as Acne

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Sir,

We describe here a case of discoid lupus erythematosus (DLE) masquerading as acne vulgaris. Cutaneous manifestations of lupus erythematosus (LE) are usually characteristic enough to permit straightforward diagnosis. However, occasionally they may be variable and mimic other dermatological conditions.

Acneiform presentation is one of the most rarely reported and one of the most confusing, as it resembles a very common inflammatory skin disease and therefore can be easily missed clinically. Only 5 cases have been reported in the literature (1–4). The patient described here presented with a widespread pruritic acneiform rash, which was initially diagnosed and treated as acne vulgaris with no response. Subsequently, when the diagnosis of DLE was established, the patient was treated with hydroxychloroquine and showed complete response. This case illustrates that it is important to consider LE in patients who present with an acneiform rash who fail to respond to conventional acne treatment.

CASE REPORT

A 38-year-old man from Afghanistan presented with a 7-year history of a pruritic and photo-aggravated rash affecting his face, chest and back. He was otherwise well apart from intermittent neck pain following an injury 2 years previously, but had recently also developed backache and arthralgia of his left shoulder. His only medication was a non-steroidal anti-inflammatory drug (NSAID).

On examination he had a widespread acneiform eruption, which was distributed on his face, pre-sternal area and back, particularly down the length of his spine. He had multiple brown-red follicular papules and open comedones, especially on his back, and hypopigmented atrophic scars. There were no pustules or nodulocystic lesions (Fig. 1).

Treatment was started with erythromycin 500 mg bid and adapalene cream once daily to treat a presumed diagnosis of acne vulgaris. He was seen 3 months later with a deterioration of his clinical appearance and increased pruritus. This was attributed by the patient to increased sun exposure. The photo-aggravation, the intense pruritus and the absence of pustules and nodulocystic lesions broadened our differential diagnosis and therefore diagnostic biopsies were obtained. A biopsy from the back where the rash was most suggestive clinically of acne vulgaris, showed hyperkeratosis with orthokeratosis, epidermal atrophy and extensive vacuolar degeneration of the basal layer of the epidermis. There was also prominent interface dermatitis, accumulation of cytooid bodies and plugging of the follicles by keratin and deep periadnexal and pericapillary chronic inflammation. These findings were consistent with a diagnosis of DLE. Facial biopsies showed periadnexal inflammation with no interface changes. No obvious features of acne vulgaris such as prominent folliculitis or foreign body reaction were seen in either biopsies.

Direct immunofluorescence from lesional skin was negative. Blood inflammatory markers and antibody profile including extractable nuclear antibody and radiological investigations were normal. Rheumatological evaluation for arthralgic symptoms showed no evidence of systemic lupus or an inflammatory arthritis.

In view of the histological findings a diagnosis of DLE was made. Acne treatment was discontinued. The patient was commenced on hydroxychloroquine 200 mg bid and was advised to use sunscreen with sun protection factor of 60 meticulously. Four months later there was marked improvement with minimal inflammation, significantly fewer numbers of open comedones and less pruritus. One year later his skin was completely clear with minimal pruritus.

DISCUSSION

Cutaneous lupus erythematosus (CLE) may present with atypical manifestations, which can cause diagnostic difficulty. These include LE profundus, LE tumidus and chilblain lupus, which are well known and recognizable. Hypertrophic lupus represents 2% of the total cutaneous lesions of LE and presents with hyperkeratotic lesions on the face, extensor surfaces of the limbs, palms and soles, resembling ostraceous



Fig. 1. (a) Multiple erythematous papules and atrophic scars over the right cheek and chin. (b) Distant view of the back.

Table I. Profiles of patients reported to have acneiform lupus, including our case

Case	Age	Sex	Sites of involvement	Pruritus	DIF	Evolution to SLE
Haroon & Fleming (1)	32	F	Back, shoulders, upper arm	Yes	+	No
Motel et al. (2)	29	F	Face, shoulders, upper back	Yes	-	Yes
Motel et al. (2)	24	F	Face and neck	Yes	+	Yes
Deruelle-Khazaal et al. (3)	30	F	Face	No	+	Yes
Chang et al. (4)	32	M	Face	No	+	No
Our case	38	M	Face, back and chest	Yes	-	No

DIF: direct immunofluorescence of lesional skin (1–4); SLE: systemic lupus erythematosus

psoriasis or hypertrophic lichen planus. LE telangiectoides is, on the other hand, a very rare form, which presents with persistent blotchy reticulate telangiectasia that heal with prominent atrophic scarring (5). Acneiform lesions are another rare and misleading presentation of CLE. Five cases have been reported previously in the literature (1–4). Their ages ranged from 25 to 35 years and 4 out of 5 were females. Three of the 5 patients presented with pruritus, as in our case, and a similar distribution affecting mainly the central back, face and chest. Photoaggravation was also noted in 3 of these cases as in our patient. Three of them subsequently developed systemic lupus erythematosus (SLE) (Table I).

The differential diagnosis in our patient included follicular lichen planus, acneiform drug eruption and Koebner reaction to pre-existing acne vulgaris. In lichen planus, scarring and photosensitivity are not usual and deep periadnexal inflammation is not a prominent histological pattern. Acneiform drug eruption due to NSAID ingestion was a possibility, but the rash started prior to NSAID use and improved with hydroxychloroquine, despite the patient continuing to take an NSAID. A Koebner response to previously existing sites of acne vulgaris was ruled out on histological grounds.

The pathogenesis of this phenotypic expression of CLE is unknown and the prognosis is uncertain as 3 out of 5 of the previous cases developed SLE. We will therefore closely monitor our patient in the future.

This case highlights the importance of considering this unusual presentation of CLE in patients with acneiform rashes that fail to respond to conventional acne treatment.

REFERENCES

1. Haroon TS, Fleming KA. An unusual presentation of discoid lupus erythematosus. *Br J Dermatol* 1972; 87: 642–645.
2. Motel PJ, Bernstein EF, Fazio M, Humeniuk H, Kauh YC. Systemic lupus erythematosus in patients diagnosed with treatment-resistant acne. *Int J Dermatol* 1995; 34: 338–340.
3. Deruelle-Khazaal R, Segard M, Cottencin-Charriere A-C, Carotte-Lefebvre I, Thomas P. Chronic lupus erythematosus presenting as acneiform lesions. *Ann Dermatol Venereol* 2002; 129: 883–885.
4. Chang YH, Wang SH, Chi CC. Discoid lupus erythematosus presenting as acneiform pitting scars. *Int J Dermatol* 2006; 45: 944–945.
5. Mascaro JM, Herrero C, Hausmann G. Uncommon cutaneous manifestations of lupus erythematosus. *Lupus* 1997; 6: 122–131.