Case Report

A case report on discoid lupus erythematosus

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ABSTRACT

Inflammatory plaques are a hallmark of discoid lupus erythematosus (DLE), a chronic cutaneous illness that can cause skin shrinkage and disfiguring scarring if left untreated. The best treatments, however, are not well supported by the available data. As of right now, no drugs have been authorized expressly to treat DLE. This uncommon ailment manifests as a crimson telangiectatic lesion in the oral cavity, surrounded by white patches in the mucous membrane. Occasionally, thick or thin white papillary radiating patterns are visible around the lesion as extremely thin lines. Here, we present a 24-year-old male patient with oral DLE.

Key words: Cutaneous illness, Discoid lupus erythematosus, Lesions

Discoid lupus erythematosus (DLE) is a persistent photosensitive dermatosis that causes atrophy and scarring. Circular whitish buccal mucosa lesions and erythematous rashes of sun-exposed skin are signs of a chronic inflammatory condition of the skin, connective tissue, and certain internal organs that have circulating autoantibodies to DNA and other nuclear and RNA proteins. The center areas may appear lighter in color with a rim darker than normal skin [1]. Systemic lupus erythematosus (SLE) individuals may develop it, however, the estimated progression rate from DLE to SLE is <5% [2]. The majority of DLE patients do not have a serious systemic illness. About 20% of patients may also develop DLE as a symptom of SLE [3]. The multisystem condition known as lupus erythematosus primarily affects the skin. Skin lupus comes in a variety of forms. Acute cutaneous lupus, subacute cutaneous lupus, and discoid lupus (DLE) are the three most prevalent forms [4]. DLE represents the most prevalent subtype of chronic cutaneous lupus erythematosus. Although lesions are often photo-distributed and tend to have subsequent atrophy or scarring, these patients may or may not experience photosensitivity. Topical/intralesional corticosteroids and antimalarial medications have been the mainstays of DLE management. In situations of resistant DLE, more recent research has supported the use of anifrolumab in conjunction with mycophenolate mofetil, methotrexate, and thalidomide/lenalidomide as second and/or third-line treatments.

Oral DLE is a rare illness with uncommon and infrequent or rare clinical presentations which often leads to diagnostic challenges or misdiagnoses due to its resemblance with other oral lesions such as lichen planus, oral candidiasis, and leukoplakia. Notifying others about a case raises awareness, facilitates early detection, and emphasizes the significance of histopathological confirmation. Timely diagnosis and management are essential due to the possibility of malignant transformation. Especially, in cases those are refractory, recording treatment response, and offering insights into therapeutic efficacy. By emphasizing clinical presentations, diagnostic techniques, and management strategies, this case report adds to the body of medical literature while also ultimately improving patient outcomes. It acts as a teaching tool for medical professionals, stressing the importance of careful monitoring and ongoing follow-up to avoid complications.

CASE REPORT

A 24-year-old male patient presented to the general medicine department with complaints of pain in the left side of the check region with painful lesions for 1 year. The patient was having a history of chest pain, shortness of breath, weight loss, flank pain, burning micturition, joint pains (elbow and ankle), and fever (on and off) for 2 months.

The patient's blood pressure was 130/80 mmHg, body temperature was elevated which was 99.3°F, pulse rate was 90 beats/min, and respiration rate was found to be 18 counts/min. On examination, an erythematous patch surrounded by white lines was found in the right buccal mucosa, and the surface was intermixed with post-inflammatory pigmentation and lichenoid reaction at the right buccal mucosa. In view of the oral examination, the provisional diagnosis might be a lichenoid reaction at the right

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buccal mucosa and the differential diagnosis might be lupus erythematosus or oral lichen planus according to the physician.

Upon histopathological examination, the given H and E-stained soft-tissue sections showed hyperkeratotic stratified squamous epithelium exhibiting areas of acanthosis. There was evidence of diffuse lymphocytic infiltrate along with melanin incontinence predominantly in the superficial connective tissue stroma. The deeper stroma was fibrous with thick collagen fiber bundles and perivascular inflammation. Furthermore, there was evidence of adipose tissue and minor mucous salivary glands. Meanwhile, the antinuclear antibody (ANA) blot test showed SS-A/Ro60 positive. Liver function tests and renal function tests showed elevated serum glutamic pyruvic transaminase (51 Unit/L), elevated alkaline phosphatase (160 Units/L), elevated protein (9.67 g/dL), and finally elevated albumin (6.24 g/dL). However, the complete blood picture showed that mean corpuscular volume – 73fl (Low) and mean corpuscular hemoglobin – 26 pg (Low).

The physician diagnosed the case as DLE based on the ANA blot test which showed SS-A/Ro60 and Ku proteins positive and a histopathology report.

The patient was treated with an intralesional steroid injection of triamcinolone acetonide, (once a week), capsule amoxiclav 625 mg, (twice a day), tablet zincovit, (once a day), and tablet paracetamol 500 mg, (twice a day).

DISCUSSION

Along with other connective tissue conditions such as polymyositis, rheumatoid arthritis, scleroderma, and mixed connective tissue disease, LE is an autoimmune disease. At one extreme of the spectrum of conditions that fall under LE is a condition known as DLE that primarily affects skin and mucosa [5].

The pathogenesis of DLE is mostly poorly known. Recent research has shown that type 1 interferons and possibly autoreactive cytotoxic lymphocytes that target adnexal structures are strongly linked to scarring in this disorder, which is thought to be largely caused by infiltrating T lymphocytes. Both novel and previously recognized genetic variations have been found to provide a high risk for SLE by genome-wide association studies. It is currently unknown if these or other genetic variations play a significant role in the onset of DLE [1].

DLE is a chronic inflammatory condition that frequently results in increased morbidity, diminished quality of life, scarring, and disfigurement. Disease management relies heavily on early diagnosis and treatment, as well as preventing lesion formation and exacerbation [6].

Hough and Rothfield *et al*. said that the age range for disease onset was 21–40 years old. The age group of 26–45 years old accounted for the largest percentage of patients (66.66%). The female-to-male ratio was 4:1 according to Davis and Marks (1977), and it was 4:1 and 50:1 according to Anderson (1980) and Schiodt *et al*.'s study of oral DLE. Nonetheless, some research, such as our case study, found a preference for men [7].

Lesions on photo-exposed skin are a common feature of the autoimmune disease DLE. Up to 15% of patients will fit the criteria for SLE, which is a subset of cutaneous lupus spectrum conditions [8]. All age groups are susceptible to DLE, although women and African Americans are more likely to experience it. It typically strikes between the second and fourth decades of life. According to estimates, there are four cases for every 100,000 people in the US and Europe [9].

Although DLE has more distinct histopathological characteristics, it can initially be mistaken for lichen planus, and the best way to diagnose it is to combine histopathological and clinical findings [10]. According to five case reports on oral manifestations of lupus published by Daniel Finn and Bijaya *et al*., oral manifestations can occur in both DLE and SLE. The clinical and histological characteristics may resemble those of oral lichen planus or other oral lichenoid lesions, as in some of the cases reported, which could result in an inaccurate or postponed diagnosis. In our case, the patient's symptoms were similar to those of lichen planus, but the diagnosis was correct [11]. In a case report by Sreejan *et al*., the patient had oral lesions in addition to lesions on the trunk and scalp; however, in our case, the patient only had oral lesions [7].

As demonstrated in the case of Zhou *et al.* who reported a case of DLE of the palms, not all cases of DLE present oral symptoms. The patient's primary complaint over the previous few years was a progressive, mildly itchy eruption on his face, ears, and bilateral upper extremities, which led to a diagnosis of DLE of the palms [12].

CONCLUSION

Due to its many clinical characteristics and resemblance to lichen planus, DLE requires a thorough diagnosis and research. At the same time, patients must be treated with caution regarding the potential adverse effects of steroid medications. This helps patients feel less uncomfortable while also encouraging the healing of existing lesions and preventing skin lesions from becoming scarred.

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