**e - ISSN - 2348 - 2168**
**Print ISSN - 2348 - 215X**

**Acta Biomedica Scientia**

Journal homepage: www.mcmed.us/journal/abs

# DISCOID LUPUS ERYTHEMATOSUS: A CASE REPORT

**Arun Subramaniam*[1], Tulsi Subramaniam[2], Asha Chowdhery[3] and Abhignini Gavarraju[4]**

[1]Professor & Head, Dept. of Oral Medicine & Radiology, [2]Professor, Dept. of Prosthodontics & Crown & Bridge, [3]Professor, Dept. of Oral Medicine & Radiology, [4]PG student, Dept. of Oral Medicine & Radiology, Dr.D.Y.Patil Dental College & Hospital, DPU, Pune, Maharashtra,India.

**Article Info**

*Received 29/08/2014*
*Revised 16/09/2014*
*Accepted 19/09/2014*

**Keywords**:-
Discoid lupus Erythematous, hyperpigmentation.

**ABSTRACT**

A 20-year-old male patient presented with non-healing ulcers of the mouth since 6 months and skin lesions since 2 months. Examination revealed erythematous, disc-like, scaly plaques over the face, nose and scalp, showing signs of healing accompanied by scarring and hypopigmentation. Histopathologic examination verified a diagnosis of Discoid Lupus Erythematosus (DLE). Topical steroids and antifungals were applied to the lesions twice daily for 2 weeks. After 2 weeks of application, all lesions regressed significantly. Presentation of this case could contribute to a better understanding of DLE in clinical practice.

## INTRODUCTION

Discoid Lupus Erythematosus (DLE) is a chronic, scarring, atrophy producing photosensitive dermatosis. DLE may occur in patients with systemic lupus erythematosus (SLE); some patients with DLE (<5%), progress on to SLE [1]. Lupus erythematosus is a classic example of an immunologically mediated condition and is one of the most common of the so called "collagen vascular" or connective tissue diseases. Lupus erythematosus was first described by Biett in1828 and Kaposi in 1872 [2]. Worldwide, the prevalence of lupus erythematosus (LE) ranges from 17 to 48 cases per 100,000 populations. The highest prevalence of LE occurs in persons aged 40–60 years and is approximately 10 times higher in women than in men [3].

## CASE REPORT

A 68-year-old male patient presented with a complaint of non - healing ulcers in the mouth since 6-months associated with pain and burning sensation.

Corresponding Author

**Arun V Subramaniam**
Email: - arunsubramaniam66@gmail.com

There was a gradual onset associated with initial erythema and burning sensation followed by blister formation that ruptured within 2–3 days of formation, leading to ulceration of the region.

There was no history of prodromal symptoms like fever or malaise. Initially, lesions were noted over the dorsum of the tongue followed by involvement of the palate and buccal mucosa. Two months later, the patient noticed similar lesions over the skin, predominantly on the trunk and other regions (Figure 7, 8, 9), which healed within 7–14 days.

The patient consulted a skin specialist 3 months ago and was under treatment with topical corticosteroids and anti candidal mouth rinses. Skin lesions showed signs of healing with hypopigmentation, but the intraoral lesions persisted. Family history was non-contributory.

Examination of the trunk, hands and legs revealed multiple, well-defined, roughly round erosive lesions measuring approximately 1 × 2 cm in dimension (Figure 7-9). Discrete erosive lesions over the cheek, nose and ear measuring approximately 0.5 × 1 cm in diameter (Figure 1-3). Intraoral examination revealed multiple superficial discrete ulcerations along the buccal mucosa, lateral border of tongue and vestibule (Figure 4-6). Diffuse, irregular ulcers covered by a pseudo membranous slough were also observed.

Based on history and clinical examination, a provisional diagnosis of DLE was made.

Histological examination showed moderately dense superficial and deep perivascular and periappendegeal infiltrate of lymphocytes with focal interface vacuolar change. The papillary dermis is edematous and shows scattered colloid bodies. The epidermis is focally thinned and the dermoepidermal junction is smudged with occasional necrotic keratinocytes/colloid bodies. Moderate amount of mucin is present within the reticular dermis (Figure 10-12). With the following features the final diagnosis of discoid lupus erythematosus was done.

**Figure 1. Discrete erosions on cheek and nose**

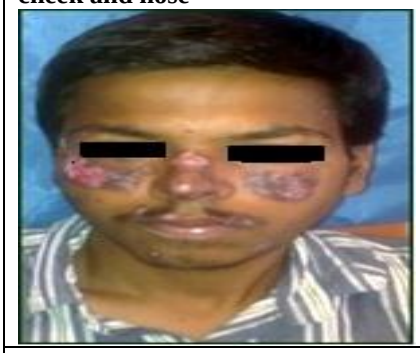


**Figure 2. Discrete erosions on cheek**

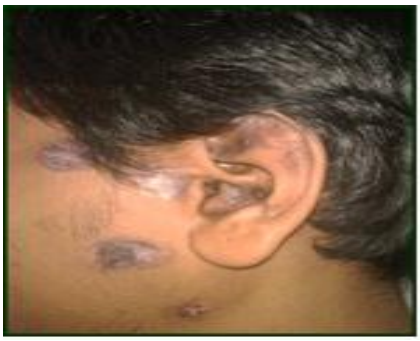


**Figure 3. Discrete erosions cheek and ear**

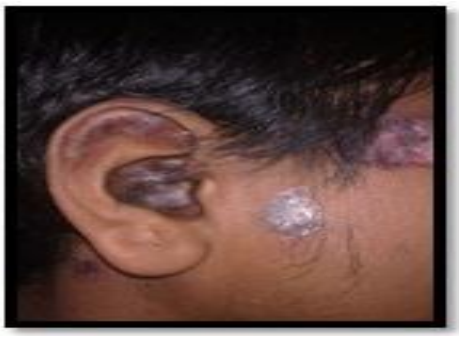


**Figure 4. Discrete ulcerations on buccal mucosa**

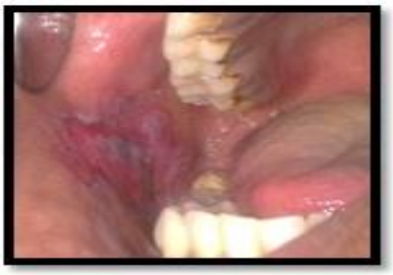


**Figure 5. Discrete ulcerations on buccal mucosa left side**

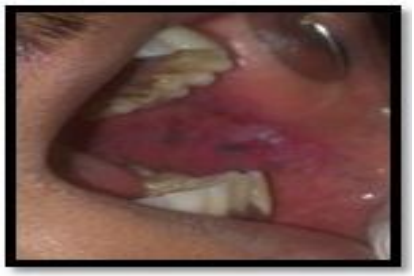


**Figure 6. Discrete ulceration on lateral border of tongue**

**Figure 7. Discrete erosions on trunk**

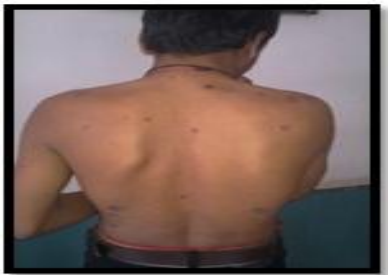


**Figure 8. Discrete erosions on chest**

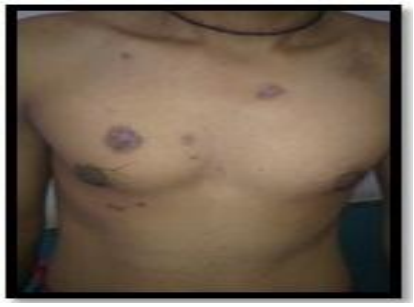
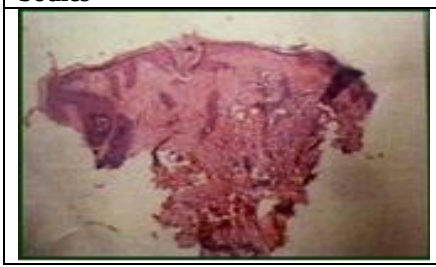
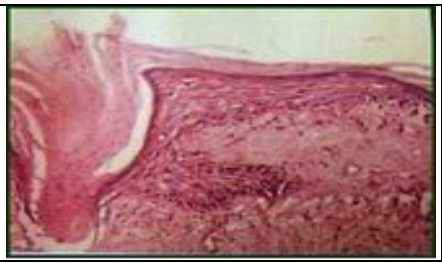
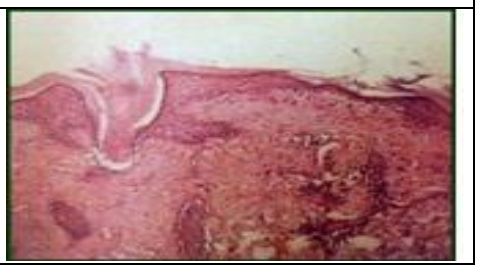


**Figure 9. Discrete erosions on hand**

**Figure 10, 11, 12. Perivascular and periappendegeal infiltrate of lymphocytes, papillary dermis is edematous and shows scattered colloid bodies. Dermoepidermal junction is smudged with occasional necrotic keratinocytes/colloid bodies**







## DISCUSSION

Lupus erythematosus (LE) is thought to be an autoimmune disease among other connective tissue diseases like scleroderma, rheumatoid arthritis, polymyositis, and mixed connective tissue disease. Within the spectrum of diseases included in LE, at one end is a disease confined mainly to the skin and referred to as discoid lupus erythematosus (DLE) and at the other end is a florid disease with systemic involvement of heart, lungs, brain, kidneys and other organs called systemic lupus erythematosus (SLE). In between the 2 ends of the spectrum are disorders like subacute cutaneous lupus. Subacute cutaneous lupus erythematosus (SCLE) has a rather sudden onset with annular or psoriasis form plaques erupting on the upper trunk, arms, and/or dorsa of hands, usually after exposure to sunlight [4]. Although at the benign end of the spectrum, 1% to 5% of patients with discoid lupus may develop SLE1 and 25% of patients with SLE may develop typical chronic discoid lesions at some time during the course of their illness [5].

The characteristic cutaneous lesions of DLE begin as erythematous, edematous, scaling papules that spread centrifugally and coalesce into plaques, the size of which varies from a few millimeters to a few centimeters. Lifting of the scales produces a carpet-tack appearance revealing dilated pilosebaceous orifices occupied by horny plugs. The healing of a lesion takes place in the center, producing atrophy, scarring, telangiectasia and pigmentary changes. Scarring alopecia is a significant finding which was not found in our case [6].

### Diagnostic criteria for DLE

- One or more cutaneous lesions in the form of an erythematous, disc-like, patch or plaque with some or all of the following features: Scales, follicular plugs, scarring, atrophy, telangiectasia, central hypopigmentation and peripheral hyperpigmentation.
- Skin biopsy of an established lesion showing features compatible with a diagnosis of LE.
- An absence of significant extracutaneous symptoms and signs at initial presentation, e.g., fever, arthralgia, Raynaud's phenomenon and diffuse alopecia.
- Normal systemic examinations except for the presence of skin and mucous membrane lesions.

### Histopathological criteria [7]

- Hyperkeratosis with keratotic plugs.
- Normal or decreased thickness of stratum granulosum.
- Irregular acanthosis alternating with atrophy of the stratum spinosum.
- Focal liquefaction degeneration of the stratum basale.
- Migration of inflammatory cells into the epithelium.
- Thickening of the basement membrane forming a focal homogenous, eosinophilic and PAS positive band.
- Perivascular accumulation of chiefly lymphocytes even deep into the connective tissue.
- Vasodilatation and edema.
- Fibrinoid degeneration of the blood vessel walls with PAS positive reactions.

In our case we could find almost all features of the clinical and histological diagnostic features which gave the perfect diagnosis of discoid lupus erythmetosus.

## TREATMENT

The treatment of DLE aims at avoiding exacerbating factors and the palliative suppression of lesions. Patients sensitive to sunlight need to wear a UVA/UVB-15 protective sunscreen daily and a hat while outdoors. Topical steroid is the first-line drug for localized cutaneous and mucosal lesions. Intralesional cortisone injections, systemic corticosteroids, calcineurin inhibitors, pimecrolimus cream or tacrolimus ointment, amino quinoline antimalarials, dapsone or imiquimod 5% may also be used along with topical antifungal therapy. Topical hydroquinone is used for the treatment of hyperpigmentated scars [8, 9].

More recently, biological therapies with agents like etanercept and tumor necrosis factor have demonstrated an overall decline in the disease activity. Efalizumab, a monoclonal antibody and a T-cell modulator has also shown a good response in patients with DLE [10].

## CONCLUSION

Early recognition and treatment improves the prognosis. The diagnosis is usually made by clinical examination. In some cases histopathology may be required to confirm the diagnosis. The histology is that of an inflammatory interface dermatosis. There is insufficient evidence for which treatment is most effective. Because lesions are induced or exacerbated by ultraviolet exposure, photoprotective measures are important. Potent topical steroids and antimalarials are the mainstay of treatment.

## REFERENCES

1. Sreejan, et al. (2009). Discoid lupus erythematosus. *Journal of Indian Academy of Oral Medicine and Radiology*, 21(1), 37-41.
2. Amena MR, Monali MC, Pritam P, Khushbu PB, Hemant MK. (2012). Oral discoid lupus erythematosus: A study of twenty-one cases. *Journal of Oral and Maxillofacial Pathology*, 16(3), 368-373.
3. Brennan MT, Valerin MA, Napeñas JJ, Lockhart PB. (2005). Oral manifestations of patients with lupus erythematosus. *Dent Clin N Am*, 49, 127-41.

4. Suresh P. (2008). Oral lesions in lupus erythematosus: correlation with cutaneous lesions, *Eur J Dermatol*, 18(4), 376-81.
5. Lahita RG. (1987). In systemic lupus erythematosus, 2nd ed. New York (NY): Churchill Livingstone, 620.
6. Rajendran R, Shivapathasundaram. (2005). Shafer's Textbook of Oral pathology. 5th ed. Philadelphia: W B Saunders Co Ltd.
7. Neville BW, Damn DD, Allen CM, Bouquot JE. (2002). Oral and maxillofacial pathology. 2nd ed. London: W B Saunders Co. Ltd.
8. Fabbri P, Cardinali C, Giomi B, Caproni M. (2003). Cutaneous lupus erythematosus: diagnosis and management. *Am J Clin Dermatol*, 4, 449–65.
9. Usmani N, Goodfield M. (2007). Efalizumab in the treatment of discoid lupus erythematosus. *Arch Dermatol*, 143, 873-7.
10. Jayakumar ND, Jaiganesh R, Padmalatha O, Sheeja V. (2006). Systemic lupus erythematous. *Indian J Dent Res*, 17, 91-3.