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Successful Management of Refractory Oral Lichen Planus with Photobiomodulation: A Case Report

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Abstract: *Oral lichen planus (OLP) is a chronic inflammatory condition with limited treatment options. We report a case of refractory OLP managed successfully with photobiomodulation (PBM).A 55-year-old female presented with a 2-year history of painful OLP lesions on the tongue and buccal mucosa. After 4 sessions of PBM (980nm diode laser, 100 mW/cm², continuous wave for 5 minutes, once every two weeks, a total of 4 sittings), significant improvements were noted in pain, lesion size, and quality of life. No adverse effects were reported. This case highlights the potential of PBM as a safe and effective adjunctive treatment for OLP.*

Keywords: *Laser, Oral Lichen Planus, Photobiomodulation, Diode Laser*

I. INTRODUCTION:

Lichen planus (LP) is a chronic inflammatory disease affecting skin, mucous membranes, and skin appendages. The prevalence of LP in the general population is estimated at up to 1.27% [1]. LP can occur at any age, without sex or racial preferences [1, 2]. Mucosal LP (MLP) shows a prevalence of 0.89% and is more commonly found in the female population [1, 2]. Oral LP (OLP) represents the most common form of MLP and can be diagnosed as an isolated disease or in association with cutaneous, scalp, nail, or mucosal involvements, including the genital, gastrointestinal, and ocular mucosa. Several therapies can be used to treat the different clinical variants of LP, although a refractory clinical course characterizes some subtypes of OLP [2].

II. PATHOGENESIS OF ORAL LICHEN PLANUS:

The etiology of OLP remains poorly understood, but it is believed to involve immune-mediated mechanisms. Environmental factors such as stress, medications, and oral hygiene practices may also exacerbate the condition.

Antigen-specific and non-specific mechanisms are involved in the pathogenesis of OLP. Antigen presentation by keratinocytes and Langerhans cells to CD4+ helper and CD8+ cytotoxic T lymphocytes leads to their activation [3, 4]. The activated helper T cells produce IL-2 and interferon (IFN)-gamma and lead to the

proliferation and activation of cytotoxic T lymphocytes, which cause the apoptosis of basal keratinocytes, and the degeneration of basal epithelial cells typically found in OLP lesions [3-5].

IL-17 plays a critical role in the pathogenesis of OLP [5]. It induces chemokine production from different cells, including endothelial cells, macrophages, and keratinocytes, leading to tissue remodeling and recruitment of pro-inflammatory cells [6]. Moreover, IL-17 release activates a pro-inflammatory cascade that leads to the recruitment of T lymphocytes [5].

On the other hand, mast cell degranulation and production of tumor necrosis factor (TNF)-alpha and chymase play a role in the pathogenesis of OLP. Indeed, TNF-alpha is involved in migrating T cells from the capillaries into the surrounding extracellular matrix. In addition, chymases activate the matrix metalloproteinase-9, which subsequently destroys the basal membrane and leads to the migration of CD8+ cytotoxic T lymphocytes into the mucosal lesions [3,4].

Therefore, OLP is considered a T-lymphocyte-mediated chronic inflammatory mucosal disease [6]. However, some authors suggested that autoimmunity can play a role in OLP pathogenesis, pointing out that CD8+ cytotoxic T lymphocytes can recognize antigens associated with major histocompatibility complex (MHC) class I on lesional keratinocytes [7].

III. CASE REPORT:

A 55-year-old systemically healthy female patient presented to the dental OPD with a 2-year history of painful whitish lesions on the tongue mucosa and buccal mucosa. The patient also reported to be a diagnosed case of Oral Lichen Planus for which she had undergone treatment in the past but did not have any supportive documents with her.

The patient failed to respond to treatment with topical corticosteroid (0.05% ointment clobetasol, local application twice a day for 30 days), and topical immunosuppressant (ointment tacrolimus 0.1%, thrice a day for 7 days). No systemic corticosteroids were tried.

When the lesion failed to respond to management by use of local drug therapy, PBM (980nm, 100mW/cm^2, 0.5 W, continuous wave for 5 minutes, once every 2 weeks, a total of 4 sittings) was administered using a low-level laser therapy device.

III (a). CLINICAL EXAMINATION:

On inspection, a greyish-white region was visible on the entire dorsal surface of the tongue and adjacent buccal mucosa in region of molar teeth with no bleeding or pus discharge. Fungal superinfection was clearly evident. On palpation, all the inspector findings were confirmed. The lesion was nontender and non-scrapable. Consistency of the tongue was normal.

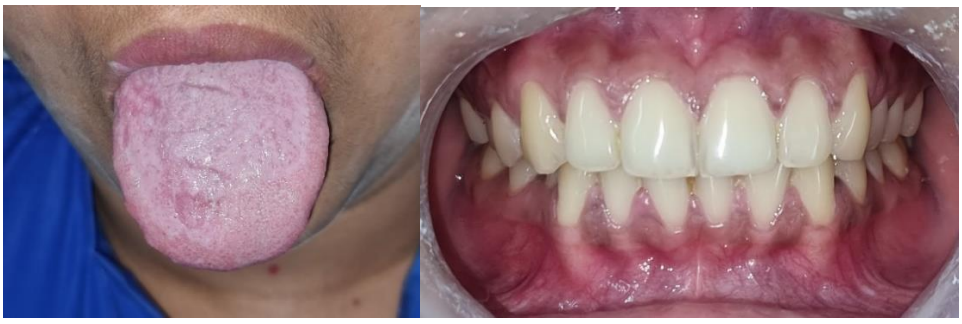

Fig 1: Baseline presentation of the tongue and the oral cavity

III (b). HISTOPATHOLOGICAL EXAMINATION:

Punch biopsy was taken from buccal mucosa and posterior part of anterior one-third of dorsal surface of the tongue. Histopathological examination showed areas of focal parakeratosis, acanthosis, liquefaction degeneration of the basal cell layer, and a thick band of juxta-epithelial infiltrate, confirming the clinical diagnosis of OLP.

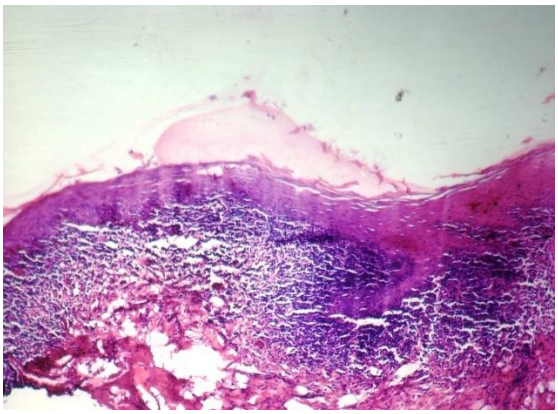

Fig 2: Histopathological examination showing areas of focal parakeratosis, acanthosis, liquefaction degeneration of the basal cell layer, and a thick band of juxta-epithelial infiltrate.

IV. TREATMENT:

A baseline evaluation of the teeth and the oral mucosa was done, and phase 1 therapy was initiated. Subsequently at 1-week interval PBM therapy was started (980nm, 100 mW/cm², 5 minutes, once every two weeks, a total of 4 sittings) using a low-level laser therapy (LLLT) device (diode laser), always by the same operator, in non-contact mode . The patient used an antimycotic solution (nystatin oral suspension 100 000 USP/mL) once a day for 4 weeks to control candidal suprainfection, if any. No antibiotics or oral antiseptics were prescribed.

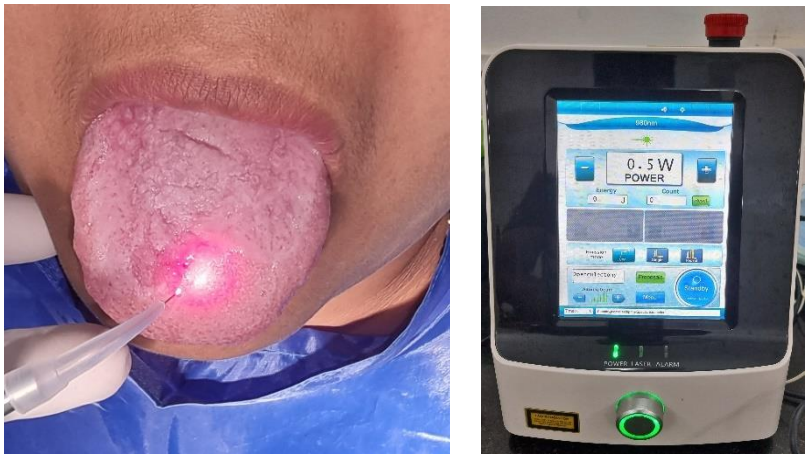

Fig 3: PBM therapy in progress using diode laser

V. TREATMENT OUTCOMES:

V(a). PRIMARY OUTCOME MEASURES:

(A) PAIN:

Pain was assessed by a visual analogue scale (VAS), ranging from '0' to '10'. One end is labeled'0' and the other '10', meaning no pain and extreme pain, respectively. The patient was evaluated at baseline, once every two weeks during treatment, and 30 days and 90 days after the end of treatment (follow-up). Significant reduction in pain was achieved (VAS 7/10 to 2/10) [8].

V (b). SECONDARY OUTCOME MEASURES:

(A) ASSESSMENT OF CLINICAL PRESENTATION OF OLP:

Clinical data was evaluated by photos and scores according to Thongprasom *et al* and the OLP lesions received a score of 0 (no lesions), 1 (hyperkeratotic lesions), 2 (atrophic area $\leq$1 cm^2), 3 (atrophic area >1 cm^2), 4 (erosive area $\leq$1 cm^2) and 5 (erosive area >1 cm^2). The patient was evaluated at baseline (day 0), once every 2 weeks during treatment, as well as 30 days and 90 days after the discontinuation of treatment (follow-up period). Photographs were taken during all periods of evaluation. The patient scored a value of 1 throughout the assessment period [9].

(B) FUNCTION:

Functional scores were assessed to evaluate chewing function, swallowing, fluid intake, and altered sense of taste, according to Libelly *et al*. Each function evaluated received one of the following scores: 0 (no difficulty), 1 (mild difficulty), 2 (moderate difficulty), 3 (severe difficulty) and 4 (impossible to perform specific function). The patient was evaluated at baseline (day 0) with a score of 2, once every 2 weeks during treatment when the scores fluctuated between 2 &1, as well as 30 days (score of 1) and 90 days (score of 1) after the discontinuation of treatment (follow-up period) [10].

(C) CLINICAL RESOLUTION :

Clinical resolution was evaluated at the end of treatment (day 60) according to Corozzo *et al*. **Complete resolution** was considered when the patient presentedan absence of symptoms and remission of atrophic/erosive lesions regardless of the presence of any persisting hyperkeratotic lesions. **Partial resolution** when a decrease, but not a complete remission of atrophic/erosive areas and symptoms, is observed. **No response** to treatment was considered when OLP lesions present the same clinical, or worse, presentation to the baseline condition. In our case only a partial resolution was achieved [11].

(D) QUALITY OF LIFE :

Quality of life was measured using the Oral Health Impact Profile (OHIP-14).The patient completed the questionnaire at baseline, at the end of treatment, and 30 days and 90 days after the end of treatment (follow-up). Improved quality of life (OHIP-14 score 24/56 at baseline to 10/56 at 3 months follow-up period was noted [12].

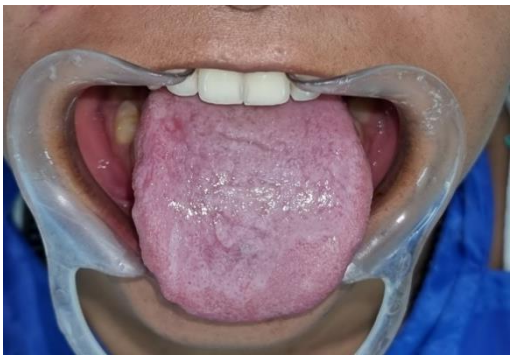

Fig 4: 3 months follow up with increased epithelisation of dorsum of tongue

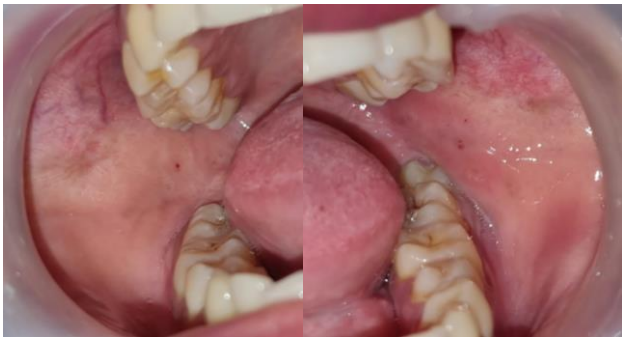

Fig 5: 3 months follow up with complete resolution of the lesions from the buccal mucosa.

PATIENT SATISFACTION:

The patient expressed high satisfaction with the treatment outcome. She highlighted the significant improvement in her quality of life, with a restored ability to eat and speak without discomfort. Importantly, she reported no adverse effects during or after the treatment.

VI. DISCUSSION:

Photobiomodulation (PBM) has been proposed as a non-invasive clinical tool that utilizes low-level laser therapy (LLLT) or light-emitting diodes (LEDs) to treat OLP, with the advantage over current therapies of not being associated with any side effects [13].

The use of PBM in different inflammatory conditions has potential analgesic, biostimulatory and immunomodulatory effects, as well as for improving healing[14,15,16].

The mechanisms through which PBM exerts its effects include:

- **Mitochondrial Activation**: PBM stimulates mitochondrial function, increasing adenosine triphosphate (ATP) production, which enhances cellular metabolism.

- **Cytokine Modulation**: PBM influences the production of inflammatory cytokines, promoting a shift towards an anti-inflammatory response.

- **Enhanced Tissue Repair**: PBM has been shown to increase collagen synthesis and facilitate angiogenesis, contributing to tissue regeneration.

In OLP, PBM has been used to treat symptomatic lesions with controversial results. Dillenburg *et al* showed a significant improvement in signs, symptoms and reduced recurrence rates in patients treated with PBM in relation to standard treatment with clobetasol propionate [12]. In the study performed by Jajram *et al*, PBM showed comparable results with clobetasol propionate [13]. However, El-Shenawy *et al*, Othaman *et al* and Kazancioglu *et al* showed that corticosteroid therapy was associated with significant improvement of OLP when compared with PBM [13,14,15].

It is noteworthy that all of these studies used different PBM parameters, with wavelengths ranging between 630 nm and 970 nm, power density from 10 mW/cm^2 to 1000 mW/cm^2 and radiant exposure from 1.5 J/cm^2 to 120 J/cm^2.

Treatment protocols also varied. These studies were recently included in two systematic reviews to access the efficacy of PBM in OLP [17,18]. However, except the study performed by Dillenburg *et al* [18] the included studies were associated with a high risk of bias due to the lack of sample size calculation, methods of randomisation and treatment masking. In addition, a wide range of laser parameters and treatment outcomes were observed, and no effective dose or protocol could be established. Thus, both reviews have concluded that there is an urgent need for rigorous clinical studies to better understand the efficacy of PBM in OLP.

Until now, due to the lack of well-designed randomised controlled trials evaluating the efficacy of PBM in OLP, it remains unclear if PBM is a viable alternative option for treating this chronic disease.

This case demonstrates the potential of PBM in managing refractory OLP. The exact mechanisms are unclear, but PBM may modulate inflammation, promote wound healing ,reduce oxidative stress, regulate immune responses, and enhance tissue repair[19, 20, 21, 22].

VII. CONCLUSION:

PBM shows promise as a safe and effective adjunctive treatment for OLP. Further research is needed to establish standardized protocols and confirm these findings.

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