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Comparitive Efficacy of Natural Extract Oral Rinse as an Adjuent to 0.1%Triamcinolone Acetonide in Oral Lichen Planus

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Abstract

Introduction: Lichen planus is a common mucocutaneous disease of unknown etiology. There is no complete cure for oral lichen planus (OLP), but various drugs are commonly used for treatment. These include corticosteroids, retinoids, cyclosporine, cryotherapy, PUVA therapy, photodynamic therapy and gene therapy. **Aim:** The aim of the study is to compare efficacy of natural extract oral rinse as an adjuent to 0.1%triamcinolone ascetonide in oral lichen planus. **Materials and methods:** A total of thirty oral lichen planuspatients are rambomly divided into two groups. Group I controls are treated with 0.1%triamcinolone acetonide and Group II experimental groups are treated with 0.1%triamcinolone acetonide along with natural extract oral rinse patients were examined after two weeks for the size of the lesion using thomprason scale score burning sensation of the patient using visual anlogue score. Data obtained was analyzed using ANOVA and independent t-tests. **Results**: The use of combination of 0.1%triamcinolone acetonide alone with natural extract oral rinse was more effective in decrease in the burning sensation and size of the lesion when compared to the 0.1%triamcinolone acetonide alone with $p<0.006$ for the size of the lesion and burning sensation of the patient $p<0.000$ but in group B ie., $p<0.040$ for size of the lesion and $p<0.000$ for burning sensation of the patient. **Conclusion**: The use of combination of triamcinolone acetonide along with natural extract oral rinse is more effective than triamcinolone alone.

Keywords: oral lichen planus, natural extract oral rinse, triamcinolone acetonide, burning sensation.

Introduction:

Oral lichen planus (OLP) is a "chronic inflammatory disorder of unknown origin. It presents with distinctive white reticular lesions, which may be or may not be accompanied by atrophic, erosive, ulcerative, and plaque-type areas." Usually, OLP presents as bilateral and symmetric lesions affecting the buccal

mucosa, the borders and dorsum of the tongue and gingiva[1]. It is more commonly seen in women. Reticular type of OLP is the most common clinical form, characterized by interlacing lines known as Wickham's striae, along with hyperkeratotic papules and plaques, and is typically asymptomatic. Erosive oral lichen planus (EOLP), the second most prevalent form, frequently causes considerable pain and discomfort, significantly diminishing the quality of life for those affected. According to WHO malignant transformation rate is 3.5% .

Various theories regarding etiology of OLP have been hypothized, including the autoimmune condition:In this condition dendritic cells (Langerhans cells) processed by antigen and its presentation to the T-lymphocytes leads to activation of the CD4+and CD8+Tcells and apoptosis of the basal cells[2]. Patients with oral lichen planus experience cycles of disease flare-ups and remissions therefore, treatment focuses on decreasing the size of the lesion and burning sensation of the patient.

Oral lichen planus treatment is symptomatic and topical corticosteroids are used as first-line drugs, including clobetasol propionate, fluocinonide, dexamethasone betamethasone and triamcinolone acetonide. Oral lichen planus is most commonly treated with topical and systemic corticosteroids. Additional documented treatment options encompass retinoids and vitamin A analogues, cyclosporine rinse, the immunomodulating agents such as levamisole, dapsone, griseofulvin, azathioprine, cryotherapy, PUVA therapy, photodynamic therapy, and gene therapy[3].Topical antioxidants can also be used in the oral lichen planus.The extracts of grape seeds and grape skin contain anthocyanins[4]. Anthocyanins are frequently referred to as "The foremost antioxidants of the plant kingdom" due to their ability to prevent the spread of free radicals[4].

Methods:

It is the prospective randomized single blinded study was carried out in the Department of Oral Medicine and Radiology. Ethical committee approval was obtained from the institutional review board(IRRB/GDCH/2023/SS-01-04).Thirty patients of either sex who are diagnosed with the oral lichen planus were included in this study. Pregnant women, lactating mothers and the patients with other potentially malignant disorders are excluded in this study. This study was approved by the institutional ethical committee.

Patients were randomly allocated into group A and group B of 15 each, for the group A 0.1%triamcinolone acetonide was given and for the group B 0.1%triamcinolone acetonide was given along with natural extract oral rinse which contains Thymus vulgariso.07%, Punica granatum0.2%, Citrus paradisio.6%, Xylitol7.0%, Citric acid anhydrouso.16%, Methyl salicylateo.03%, Glycerin12.5%, Sodium benzoate. patients were asked to apply

0.1%triamcinolone acetonide three times daily for 2weeks and for group B they were asked to rinse the oral cavity with natural extract oral rinse three times daily by slightly tilting the head towards the lesion for 30 secs after the rinsing patients were advised to apply the 0.1%triamcinolone acetonide for 2 weeks.size of the lesion was measured with the thongprason size score and burning sensation was measured using visual analogue scale (VAS) of score 0-10. The overall treatment response was recorded at baseline (0day) and after 2 weeks (14^{th} day).

Data obtained was analyzed using ANOVA and independent t-tests to assess the efficacy of natural extract oral rinse and triamcinolone acetonide.

Results:

In this study there are total 16 females and 12 males are included ,showing slight female predilection with a male to female ratio of 3:4. The mean age of the patients enrolled in the study was 26-55 years. The use of medications in both groups resulted in a reduction in size of the lesion and the severity of burning sensation in oral lichen planus, which was statistically significant ie.,P<0.040 and P<0.00 for the size and pain of the lesion in group I [see the table I] and <0.006 and <0.000 for the size and pain of the lesion in group II[see the table II] and although there is significant reduction in Pvalue in both the groups there is reduction of VAS score in group B when compared to group A. Hence, this study revealed that 0.1%topical triamcinolone acetonide and natural extract oral rinse showed reduction in VAS score compared to Triamcinalone acetonide alone.

Table I

Intra Group Analayis Of Evaluation Of Pain & Size Of Lesion During Pre And Post, Treated With 0.1%Topical Triamcinolone Acetonide By Visual Analogue Scale & Thongprasom Sign Score (Group 1)

Parameter	Measuring Periods	MEAN	SD	SE	95% CI		T value	P value
					LOWER	UPPER		
Size of Lesion	VISIT 1 TS - VISIT 2 TS	.28571	.46881	.12529	.01503	.55640	2.280	.040*
Pain of Lesion	VISIT 1 VAS - VISIT 2 VAS	2.14286	1.09945	.29384	1.50805	2.77766	7.293	.000*

In total of 15 subjects in group 1 treated with 0.1%TOPICAL TRIAMCINOLONE ACETONIDE, the cumulative mean size of lesion during pre and post was 0.28 and cumulative mean pain score of lesion during pre and post was 2.14 respectively, resulted as statistical significant difference is observed in both the size of lesion (P<0.040*) and pain of lesion (P<0.000**).

Table -II

Parameter	Measuring Periods	MEAN	SD	SE	95% CI		T value	P value
					Lower	Upper		
Size of Lesion	VISIT 1 TS - VISIT 2 TS	.71429	.82542	.22060	.23770	1.19087	3.238	.006*
Pain of Lesion	VISIT 1 VAS - VISIT 2 VAS	3.78571	1.76193	.47090	2.76841	4.80302	8.039	.000*

Intra Group Analayis of Evaluation of Pain & Size of Lesion During Pre and Post Treated With 0.1%Topical Triamcinolone Acetonide & Natural Extract Oral Rinse by Visual Analogue Scale and Thongprasom Sign Score (Group 2)

In total of 15 subjects in group 2 treated with 0.1%topical triamcinolone acetonide, &natural extract oral rinse, the cumulative mean size of lesion during pre and post was 0.71 and cumulative mean pain score of lesion during pre and post was 3.78 respectively, resulted as more precise statistical significant difference is observed in both the size of lesion (<0.006*) and pain of lesion (<0.000**).

Table Iii: Inter Group Analayis of Evaluation of Pain & Size of Lesion During Pre and Post Treated with 0.1%Topical Triamcinolone Acetonide Group and 0.1% Topical Triamcinolone Acetonide Plus Natural Extract Oral Rinse Group by Visual Analogue Scale and Thongprasom Sign Score

Groups	Measuring Priods	N	MEAN	SD	SE	95% CI		T value	P value
						Lower	Upper		
GROUP 1	Visit 1 TS	15	2.2143	.89258	.23855	-0.82	0.75	-0.417	0.04*
	Visit 2	15	1.928	.91687	.2450				

	TS		6		5				
GROUP 2	Visit 1 TS	15	2.3571	1.15073	.30755	-0.420	0.80	0.879	0.007*
	Visit 2 TS	15	1.6429	.63332	.16926				
GROUP 1	Visit 1 VAS	15	6.5000	1.65250	.44165	-1.96	0.67	-1.287	0.000*
	Visit 2 VAS	15	4.3571	1.39268	.37221				
GROUP 2	Visit 1 VAS	15	7.1429	1.16732	.31198	-0.23	2.19	1.744	0.000*
	Visit 2 VAS	15	3.3571	1.54955	.41413				

The treatment outcomes for two groups shows that in Group 1, the mean lesion size decreased from 2.2 during the first visit to 1.9 during the second visit, indicating a slight improvement. Additionally, the mean pain score in Group 1 was reduced from 6.5 in the first visit to 4.3 in the second visit, reflecting a significant reduction in pain. In Group 2, the mean lesion size showed a more substantial decrease, from 2.3 in the first visit to 1.6 in the second visit. The mean pain score in Group 2 also dropped notably from 7.1 during the first visit to 3.3 during the second visit. These findings suggest that both groups experienced a reduction in lesion size and pain over time, with Group 2 showing a slightly greater overall improvement in both parameters.

Discussion

Lichen planus (LP) is a persistent inflammatory mucocutaneous disorder that affects the skin and/or mucosa, affecting 0.2-2.4% of the overall population.[1] The exact etiology of oral lichen planus is unknown. It is now thought that immunological, genetic, and mental variables have a role in the etiology of OLP[2,5]. Several scholars have discovered that oxidative stress contributes to the development and incidence of oral lichen planus.The pathogenesis of oral lichen planus includes antigen specific and nonspecific mechanisms[2].

Antigen specific mechanism: The infiltration of T lymphocytes beneath the epithelium leads to an elevation in cytokines, which in turn stimulate keratinocytes to generate reactive oxygen species (ROS). These reactive oxygen species leads to lipid peroxidation, resulting in significant damage to integrity of the cell. This damage impacts membrane structure, crosslinking, fluidity, and function, ultimately leading to apoptosis. The apoptosis of keratinocytes and the

liquefaction of epithelial basal cells are key pathological characteristics of Oral Lichen Planus (OLP), indicating that ROS may be crucial in its development[6].
Antigen non-specific mechanism:
Antigen-nonspecific mechanisms in Oral Lichen Planus (OLP) lesions include degranulation of the mast cell and the matrix metalloproteinases (MMPs) activation. Reactive oxygen species (ROS) play a role in the nonspecific pathogenesis of OLP, influencing factors such as p53, Bcl-2 family proteins, TNF-α, the granzyme B, Fas/FasL pathway, and MMP-9. These elements are associated with apoptosis and lymphocyte infiltration in OLP lesions[7].The p53 protein can regulate directly around 500 target genes, thereby overseeing a range of processes, including cell aging, cell cycle arrest, metabolic adaptation, DNA repair, and cell death. Under minimal stress conditions, p53 may be involved in antioxidant reactions to lower ROS levels or engage in DNA damage repair, inducing a brief cell cycle arrest to ensure cell survival and facilitate tissue repair[8].It can inhibit the interaction between autophagy and Bcl-2 family members, thereby preventing cell apoptosis and necrosis by altering mitochondrial permeability.
Bcl-2 anti-apoptotic proteins play a crucial role in preserving the integrity of the mitochondrial outer membrane by inhibiting the activity of pro-apoptotic proteins. This inhibition is essential for maintaining cellular homeostasis and preventing the initiation of apoptosis. The ultimate fate of cells is determined by the balance between the various members of the Bcl-2 protein family, with this equilibrium dictating whether a cell will survive or undergo programmed cell death.
Cell signaling can modulate the expression of pro-apoptotic and anti-apoptotic proteins, tipping the balance towards either survival or apoptosis. When the balance favors cell death, oligomers of pro-apoptotic proteins on the mitochondrial outer membrane increase its permeability, leading to the release of cytochrome c.This event initiates a cascade of apoptotic proteases that sequentially cleave downstream substrates, ultimately culminating in cell death[9].Therefore, p53 has the ability to downregulate apoptosis inhibitors such as PIG-3, Bcl-2, and Apaf-1. Furthermore, p53 directly stimulates mitochondria to release ROS, thereby facilitating the induction of apoptosis.Elevated intracellular ROS levels induce cell death by activating pathways linked to Fas-dependent signaling and pro-apoptotic responses to TNF-α. The presence of tumor necrosis factor receptor-1 (TNFR-1) and Fas receptor (FasR) within cells amplifies ROS production, thereby accelerating cellular demise. TNF-α, a cytokine released by activated macrophages, not only inhibits osteoblast activity but also promotes osteoclast function, leading to significant alterations in bone metabolism.

When tumor necrosis factor-alpha (TNF-α) binds to tumor necrosis factor receptor 1 (TNFR-1), it triggers the TNFR-1 signaling pathway. This process involves the participation of TNFR-1-associated death domain protein (TRADD) and TNF-α receptor-associated factor 2 (TRAF2). As a consequence of this interaction, a signaling cascade is activated, which subsequently leads to the activation of Fas-associated death domain protein (FADD) and caspase-8. In a similar manner, when the Fas receptor (FasR) is engaged, it recruits FADD to initiate the activation of caspase-8, ultimately resulting in the induction of apoptosis[10].Granzyme B (GB) induces reactive oxygen species (ROS)-dependent apoptosis during mitosis by initiating a complex biochemical pathway that disrupts the mitochondrial respiratory chain. Specifically, GB directly targets the mitochondria and cleaves the NDUFV1, NDUFS1, and NDUFS2 subunits of NADH dehydrogenase. This cleavage results in a significant increase in ROS levels within the targeted cells. The elevated ROS levels contribute to cellular damage and stress, thereby promoting the apoptotic pathway triggered by Granzyme B. This entire process underscores the crucial role of GB in mediating cell death through mitochondrial dysfunction and oxidative stress. As a result of this process, ROS production occurs specifically at mitotic centers. Furthermore, there is a loss of activities in complex I and III of the respiratory chain, leading to its disruption. This disruption impairs mitochondrial respiratory function and contributes to the The loss of mitochondrial ridge connections leads to the disruption of the structural integrity and functional efficiency of the mitochondria, which can result in impaired cellular energy production and a range of metabolic dysfunctions[10,3].Research has demonstrated heightened expression of MMP-1, MMP-2, and MMP-3 in epithelial cells of OLP. Additionally, there is increased MMP-9 expression observed in inflammatory infiltrating cells within oral lichen planus. Therefore, MMPs are believed to contribute to the rupture of the basal membrane in OLP, facilitating the migration of inflammatory cells into the epithelium[9].The interaction of various nonspecific mechanisms significantly contributes to the exacerbation of both the onset and progression of oral lichen planus..

Corticosteroid agents like triamcinolone acetonide are commonly used in oral lichen planus.It crosses the cell membrane and binds to DNA, altering the transcription of messenger RNA (mRNA). This process stimulates or inhibits the synthesis of lipocortin, which then inhibits phospholipase A2 activity. Consequently, the expression of prostaglandins and leukotrienes decreases, producing anti-inflammatory and immunosuppressive effects.
In this study natural extract oral rinse is used and the main key ingredient was anthocyanin. The antioxidant properties of anthocyanins are mainly due to their capability to neutralize harmful free radicals in the body is vital, as these free

radicals are highly reactive molecules capable of damaging cells.. Anthocyanins can donate electrons to these unstable free radicals, effectively neutralizing them and preventing damage to cellular structures. The results in this study comparitive efficacy of natural extract oral rinse and 0.1%triamcinolone acetonide in management of oral lichen planus are similar to the study conducted by the Gutierrez et al[4]

Conclusion

From this study we conclude that Adjunctive use of natural extract oral rinse with triamcinolone showed improved clinical outcomes. It allowed reduction in steroid usage without compromising therapeutic efficacy. This approach offers a safer and more sustainable management option for oral lesions.

Limitations

Taking large sample size would be helpful in future to assess the efficacy of natural extract oral rinse in oral lichen planus cases. follow up for longterm is necessery to assess the efficacy of natural extract oral rinse in oral lichen planus cases

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