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Original Research

Evaluation of salivary interleukin levels in oral lichen planus patients

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ABSTRACT

Background: This study aimed to assess the salivary levels of interleukin-8 (IL-8) in oral lichen planus (OLP) subjects.
Material and methods: This descriptive cross-sectional study was conducted on 100 patients, out of which 80 had oral lichen planus while 20 subjects were chosen as controls. The salivary levels of IL-8 measured in all the subjects. Data was analyzed with one-way ANOVA and post hoc least significant difference tests.
Results: The mean salivary level of IL-8 was higher among subjects with oral lichen planus than the control group.
Conclusion: The increasing salivary level of IL-8 in OLP group indicates the role of this inflammatory cytokine in the pathogenesis of OLP.

Introduction:

Lichen planus is a chronic inflammatory disease that affects the skin, hair follicles, nails, and mucosa.[1] Mucosal surfaces affected include the oral, genital, ocular, otic, esophageal surfaces, and in rarer instances, the bladder, nasal, laryngeal, and anal surfaces. The skin and oral mucosa are the major sites that are affected.[2]

The oral variant, termed oral lichen planus (OLP), is a chronic condition with periods of relapses and remissions, requiring long-term symptomatic treatment and surveillance monitoring. About 15% of patients with oral lichen planus (OLP) develop cutaneous lesions, and 20% develop genital lesions.[3]

Oral lichen planus is a known T-cell mediated chronic inflammatory response affecting the oral mucosa. Evidence suggests that other factors such as trauma, dental plaque, and stress may play a role in exacerbating OLP symptoms.[4,5] The estimated global prevalence of oral lichen planus is about 2%.[6,7] It is twice as common in women and is often diagnosed between the fifth and sixth decades of life, although it may also occur in children and young adults.[8,9,10]

Interleukin-8 (IL-8) is an important mediator of host response to injury and inflammation. Its role is to activate neutrophils, neutrophil chemotactic factor, T cells and basophils. It is produced by different cells, including monocytes/macrophages, T cells, neutrophils, endothelial cells, fibroblasts and keratinocytes during the inflammatory and pathological processes.[11]

The concentration of IL-8 is insignificant in healthy tissues; however, its level rapidly reaches 10–100 times its baseline value in response to pro-inflammatory cytokines, namely tumor necrosis factor-alpha (TNF-α) and IL-1 as well as bacterial or viral products and cellular stress. In patients with OLP, keratinocytes also produce IL-1 and TNF-α.[12] Moreover, mononuclear cells infiltrating around the OLP mucosal lesions can produce TNF-α.[13] Keratinocytes, macrophages, T-cells, endothelial cells and fibroblasts stimulated by IL-1 and TNF-α in OLP lesions can release significant amounts of IL-8. This cytokine further enhances the infiltration of T cells, particularly cytotoxic T cells, around OLP lesions; therefore, it plays a role in the pathogenesis of OLP.[14]Hence, this study was

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carried out to evaluate salivary interleukin levels in oral lichen planus patients.

Material and methods:

On subjects having OLP, a cross-sectional investigation was carried out. An oral medicine specialist examined the red and white areas, took a history, and then took a biopsy. Patients who were taking medicine, had systemic illnesses other than OLP, had inflammation in other body parts, had periodontal disease, or were taking any other medications were all disqualified from the study. Alcohol intake, drug usage, and cigarette smoking were also prohibited behaviours for the two groups. Each subject had 3 mL of saliva taken. The procedure of spitting was employed to collect the salivary samples. The blood samples were kept at 20°C until the IL-8 concentration could be measured using the ELISA kit.

Results:

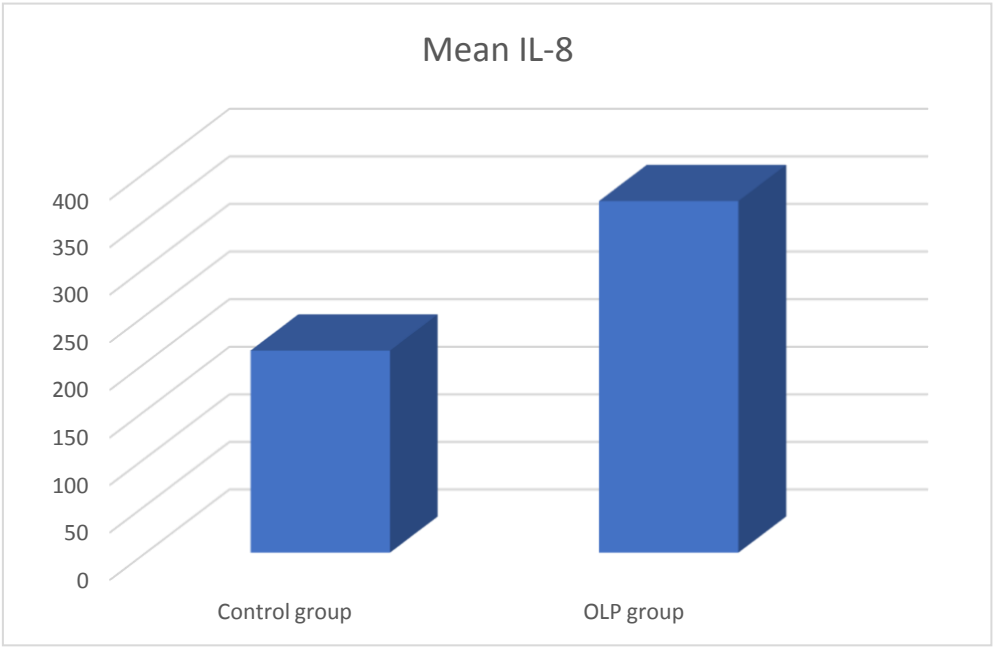
20 healthy controls and 80 OLP patients were evaluated. The mean salivary level of IL-8 was high in patients with OLP (369.57 ± 93.66 pg/mL) in comparison to healthy controls. It was found that there was a significant difference in the salivary levels of IL-8 between the OLP and control group (P = 0.017).

Groups	Number of subjects
Control group	20
OLP group	80
Total	100

Table 1: Prevalence of oral lichen planus.

Groups	Mean IL-8	SD	p-value
Control group	212.5	58.4	0.017 (Significant)
OLP group	369.57	93.66	

Table 2: Comparison of mean salivary IL-8 levels



Graph 1: Comparison of mean salivary IL-8 levels

Discussion:

While the pathophysiology of oral lichen planus is not entirely understood, the two main proposed mechanisms are antigen-specific and non-specific mechanisms.[15]The antigen-specific mechanism suggests that antigen presentation by Langerhans cells or basal keratinocytes leads to the activation of CD4+ helper T cells, stimulating the release of pro-inflammatory T-helper 1 (Th1) cytokines such as tumor necrosis factor-alpha (TNF-α) and interferon-gamma (IFNγ).[16] This induces a CD8+ T cell-mediated cytotoxic reaction against the epidermal basal cell layer resulting in keratinocyte apoptosis via TNF-α, granzyme, or Fas-Fas ligand-mediated mechanisms.The non-specific mechanism suggests that the activation of mast cells releases pro-inflammatory mediators such as proteases and the upregulation of matrix metalloproteinases. This results in T cell infiltration of the superficial lamina propria, disruption of the basement membrane, and eventual keratinocyte apoptosis. The chronic nature of OLP has been postulated to be due to the activation of nuclear factor kappa B (NF-kB) and the inhibition of the transforming growth factor control pathway (TGF-β/Smad), leading to hyperkeratosis and the appearance of distinct white lesions.[17,18] Genetic polymorphisms of the first intron of the promoter gene of IFNγ have also been postulated to be risk factors for developing OLP.[19,20]

In our study it was found that the levels of IL-8 were elevated in the subjects having oral lichen planus.Rhodus et al.[21] stated that salivary and serum levels of IL-8 were significantly higher in patients with lichen planus than the control group.Zhang et al.[22] reported that IL-8 level in the oral fluids of OLP patients was significantly higher than that in healthy controls. They also mentioned that salivary IL-8 is a reliable biomarker for the assessment of the severity of OLP. Tavangar et al.[23] showed that serum level of IL-8 in OLP patients was significantly higher than that in healthy individuals (P = 0.002).

Conclusion:

It was concluded that the levels of IL-8 were elevated in subjects having oral lichen planus. The increasing salivary level of IL-8 in OLP group indicates the role of inflammatory cytokine in the pathogenesis of OLP.

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