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Comparative study of E-cadherin expression between reticular, erosive oral lichen planus and lichenoid lesions

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ABSTRACT

Background: Accurate and early diagnosis of dysplastic lesions is crucial for successful treatment. A decrease in E-cadherin expression has been observed in dysplastic lesions and tumors. Therefore, the aim of this study was to investigate the expression of E-cadherin, a cell membrane adhesive protein involved in tissue structure and differentiation, in oral reticular lichen planus, erosive lichen planus, and lichenoid lesions.

Materials and Methods: This descriptive cross-sectional study was conducted on 65 oral samples (20 reticular lichen planus, 20 erosive lichen planus, and 20 lichenoid lesions, with 5 samples of healthy mucosa), to evaluate the expression of E-cadherin using immunohistochemical methods. Data were analyzed using SPSS software (version 25), descriptive statistics, Chi-square tests, and Fisher's exact tests, with a significance threshold set at $P < 0.05$.

Results: The majority of patients were female (72.3%) and primarily in the sixth and seventh decades of life (49.2%). A significant difference was observed between the studied groups regarding staining status ($P = 0.038$), with erosive lichen planus showing the highest frequency of alterations in E-cadherin expression (45%). In addition, a significant difference was noted between staining status and lesion location ($P = 0.004$), with the highest frequency of E-cadherin expression changes occurring in buccal mucosal samples (30%).

Conclusion: E-cadherin expression in erosive lichen planus is significantly lower than in healthy tissue, reticular lichen planus, and lichenoid lesions. Given the similar reduction observed in squamous cell carcinoma samples, evaluating E-cadherin expression may aid in the early recognition of malignant changes.

Key Words: Cadherins, immunohistochemistry, lichen planus, oral mucosa

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INTRODUCTION

Considering the importance of the prevention and early diagnosis of malignancies, many studies have been conducted on various types of oral premalignant lesions, which pose a potential risk for the development of oral squamous carcinomas and their associated risk factors.[1] Oral lichen planus (OLP) is a type of chronic mucocutaneous disease primarily mediated by T-lymphocytes, often presenting clinically as white reticular networks on the cheek.[2] The World Health Organization classifies this lesion among those with potential for malignancy, with

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reported probabilities of malignant transformation ranging from 0 to 10%.[3,4]

Among the various forms of OLP (reticular, plaque-like, erosive, atrophic, etc.), erosive lesions appear to have a higher risk of malignancy, with the most significant malignant changes reported in lesions located on the cheeks.[5,6] Epithelial cell dysplasia is known as a range of cytological and structural cell changes, caused by the accumulation of genetic alterations in cells, which can ultimately lead to malignant transformation.[7]

In a review study conducted by Tampa *et al*. in 2018 on factors affecting the malignant changes in OLP, several markers were examined, including those related to apoptosis, cell cycle regulation, inflammatory factors, and cell junction proteins. Among these, studies on the level of E-cadherin protein expression in lichen planus lesions have reported conflicting results.[8,9] In this context, some studies have indicated that there is no relationship between E-cadherin expression and the potential for malignant changes in OLP.[10] In contrast, other studies have suggested that such a relationship does exist.[11]

Most studies on E-cadherin expression in premalignant lesions and oral squamous cell carcinoma (OSCC) have found that the expression of this membrane protein decreases as malignant changes develop or as the degree of malignancy increases. They suggest that E-cadherin may serve as a marker for investigating the development and progression of malignant changes.[12-14] Therefore, further research is needed to clarify the role of E-cadherin in the potential malignancy of lichen planus.

Cadherins are calcium-dependent cell adhesion molecules (CAMs) that play a crucial role in regulating various biological processes, including intercellular connections, cell polarity, and morphogenesis.[15] CAMs are present on the surface of all cells and facilitate movement processes during tissue morphogenesis, as well as the development and maintenance of mature tissues. These molecules are essential for maintaining squamous epithelial structures, as they participate in cell renewal and motility, and their presence is crucial for cell connections and interactions with the extracellular matrix (ECM). Cadherins are a specific type of CAM located at adhesion junctions in cell membranes, enabling communication between different cells. Over 50 subtypes of CAMs have been identified, forming a large and diverse family.[16] In addition, a reduction in E-cadherin expression has been observed in invasive breast cancer, prostate cancer, and gastric carcinoma in humans.[15] Most proteins connect the extracellular and intracellular regions, and they also include structures that establish connections between the cytoskeleton and the ECM or between cells. Through these structures, cells are able to regulate and generate signals and transmit them and processes such as cell division, migration, and differentiation.[16] The aim of this study was to compare the expression of E-cadherin protein in erosive, reticular lichen planus, and lichenoid lesions of the oral mucosa using immunohistochemistry.

MATERIALS AND METHODS

In this descriptive and analytical cross-sectional study, which was approved by the ethical committee (approval number IR.MUI.MED.REC.1399.923), a total of 65 samples were selected from the archives of the Pathology Department at the Faculty of Dentistry, Isfahan University of Medical Sciences. The samples included 20 with a microscopic diagnosis of reticular OLP, 20 with erosive OLP, 20 with lichenoid lesions of the oral mucosa, and 5 normal mucosal tissue samples.

The inclusion criteria for this study required that the selected samples clinically and microscopically exhibited the characteristics of erosive lichen planus, reticular lichen planus, or lichenoid lesions and that they had adequate and satisfactory tissue fixation. In addition, the samples needed to be free of necrosis, inflammation, and bleeding, and relevant patient information had to be available in their files. Samples lacking sufficient tissue in the paraffin blocks were excluded from the study.

To detect specific antigens in the target tissues, the immunohistochemical staining technique was used, which is based on the reaction between antibodies and specific antigens. The markers used in this research included E cadherin (lyophilized monoclonal antibody, clone 36B5, NCL E Cad) at a dilution of 1:50. The Biotin-Streptavidin Novolink Polymer Detection System was chosen for its high sensitivity and accuracy compared to other methods. First, a 3–4 μm thick section was prepared from the paraffin blocks of each sample. The sections were then placed on slides coated with poly-l-lysine to prevent tissue tearing. The prepared slides were subsequently placed in an oven at 58–60ºC for 40 min.

Afterward, the samples on the slides were deparaffinized by immersing them in three changes of xylene for 5 minutes each, followed by rehydration in distilled water and a series of five graded alcohol solutions. In this step, the samples were immersed in a citrate buffer solution with a pH of 6 to preserve the antigens. This setup was then placed in a microwave (W = 750) for 15–20 min until the molecular structure of the antigen was restored through controlled heating. Following this, the samples were allowed to cool at room temperature for 20 min.

All samples were then transferred to a phosphate-buffered saline (PBS) solution and incubated for 5 min in 3% hydrogen peroxide to inhibit endogenous peroxidase activity. To prevent false staining, a protein-blocking solution (RE7102) was applied for 5 min. The samples were then washed with distilled water and PBS, followed by incubation with trypsin (1% trypsin, pH = 7.3) for 10 min. This enzyme enhances staining by facilitating and accelerating the reactions. After washing the samples with PBS, the slides were incubated in the antibody solution for 1 h. Following this incubation, the slides were washed again with PBS for 5 min and then treated with postprimary block solution (RE7111) for 30 min. This solution contains antibodies against the primary antibody of rabbit or mouse origin. At this stage, the primary antibody and the postprimary block solution form a complex. The slides were then washed with PBS for 5 min before being placed in Novolink Polymer (RE7112) solution for another 30 min, followed by a final wash with PBS for 5 min.

Finally, the samples were incubated in diluted diaminobenzidine chromogen for 5 min and then washed with distilled water. For the evaluation of stained samples, the presence of the target antigen in the tissue will be indicated by a brown color.

Subsequently, all samples were stained with hematoxylin to enhance the background coloration. The samples were then dehydrated in graded alcohol and cleared in xylene before being mounted. The stained slides were examined simultaneously by two oral and maxillofacial pathologists in six fields, with approximately 100 cells evaluated in each field using a light microscope (Olympus BX41, Tokyo, Japan) at ×400.[17]

In normal squamous epithelium, E-cadherin is uniformly expressed in the cell membrane. The stained epithelial cells were evaluated based on the location of the marker staining within the cell, the presence or absence of staining uniformity, and the percentage of stained cells. To compare these findings, the data were converted from qualitative to semi-quantitative measures. Cells with membrane staining received a score of 3, cells with membrane-cytoplasmic staining received a score of 2, cells with cytoplasmic staining received a score of 1, and nonstaining cells received a score of 0.

For staining uniformity, homogeneous expression of this protein was assigned a score of 2, nonhomogeneous expression received a score of 1, and absence of expression was given a score of 0.

Ultimately, the percentage of stained cells was scored as follows: score 0 = below 25% of cells were stained, score 1 = 25 to below 50% of cells were positive for staining, score 2 = 50%–75% of cells show positive cell staining, and score 3 = more than 75% of cells were stained.

Based on these numerical criteria, the scores for each sample were summed. A total score of 6 or higher indicated preservation (consistent with normal staining or expression), while a score of 5 or lower indicated altered expression or mutation.[17]

The data were analyzed using SPSS software (version 25) (IBM, Chicago, USA), descriptive statistics, Chi-square, and Fisher's exact tests (significant level was considered 0.05).

RESULTS

Of the participants, 47 (72.3%) were women and 18 (27.7%) were men. The average age of the patients was 52.51 ± 14.30 years, with most falling within their sixth and seventh decades of life. The buccal mucosa was the most common site of involvement, with 40 samples, while the labial mucosa had the fewest, with 6 samples. Most samples exhibited E-cadherin expression in a membrane-cytoplasmic form [Table 1]. In addition, the expression of E-cadherin was predominantly homogeneous, observed in 55.4% of cases [Table 2]. In reticular lichen planus, lichenoid lesions, and healthy mucosa, over 75% of the cells were stained in the majority of samples [Table 3 and Figure 1]. Regarding the expression status of E-cadherin (normal versus altered), the frequency of E-cadherin protein expression changes was 25% in reticular lichen planus, 45% in erosive lichen planus, and 10% in lichenoid lesions.

The Chi-square test revealed a significant difference in E-cadherin expression (normal versus altered) between

reticular and erosive lichen planus (P = 0.046), as well as between erosive lichen planus and lichenoid lesions (P = 0.014). However, no significant difference was found in E-cadherin expression between reticular lichen planus and lichenoid lesions (P = 0.158).

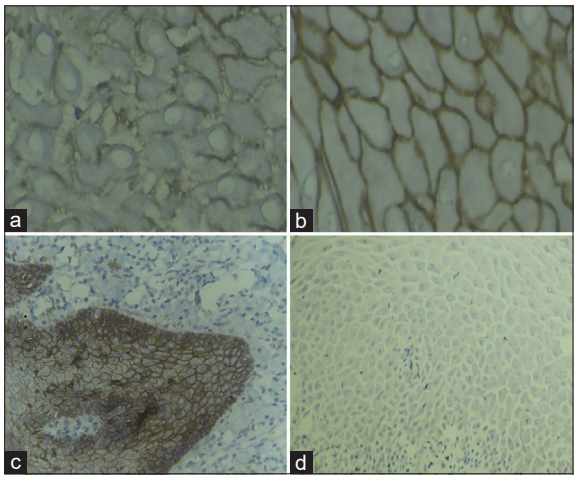

Figure 1: E-cadherin staining: (a) uniform (homogeneous) cell membrane staining in reticular lichen planus (×400), (b) nonuniform (heterogeneous) cell membrane staining in erosive lichen planus (×400), (c) no staining of cells with E-cadherin in erosive lichen planus (×100), and (d) staining with E-cadherin in more than 75% of cells (×100).

In most sites, lesions exhibited a normal expression status (75.4%), while only 16 (24.6%) cases showed altered E-cadherin protein expression. The Chi-square test indicated a statistically significant difference between the staining status and the location of the lesions (P = 0.004).

The highest frequency of changes in E-cadherin expression was found in buccal mucosal samples (30%). A significant difference in expression status was observed among the various groups (P = 0.038), with erosive lichen planus exhibiting the highest frequency of altered expression compared to the other groups [Table 4].

DISCUSSION

The present study revealed a significant difference in E-cadherin expression between reticular and erosive lichen planus (P = 0.046) and between erosive lichen planus and lichenoid lesions (P = 0.014). However,

Table 1: Frequency distribution of E-cadherin expression in types of lesions based on expression area (membrane-cytoplasmic)

Groups	Nonstaining, *n* (%)	Cytoplasmic, *n* (%)	Membranous and cytoplasmic, *n* (%)	Membranous, *n* (%)	Total, *n* (%)	*P*
Reticular lichen planus	2 (3.1)	0	10 (15.4)	8 (12.3)	20 (30.8)	0.385
Erosive lichen planus	0	1 (1.5)	8 (12.3)	11 (16.9)	20 (30.8)	
Lichenoid lesions	1 (1.5)	1 (1.5)	9 (13.8)	9 (13.8)	20 (30.8)	
Normal tissue	0	0	5 (7.5)	0	5 (7.7)	
Total	3 (4.6)	2 (3.1)	32 (49.2)	28 (43.1)	65 (100)	

Table 2: Frequency distribution of E-cadherin expression in types of lesions based on uniformity of staining (heterogeneous-homogeneous)

Groups	Nonstaining, *n* (%)	Heterogeneous, *n* (%)	Homogeneous, *n* (%)	Total, *n* (%)	*P*
Reticular lichen planus	2 (3.1)	9 (13.8)	9 (13.8)	20 (30.8)	0.274
Erosive lichen planus	0	10 (15.4)	10 (15.4)	20 (30.8)	
Lichenoid lesions	1 (1.5)	7 (10.8)	12 (18.5)	20 (30.8)	
Normal tissue	0	0	5 (7.7)	5 (7.7)	
Total	3 (4.6)	26 (40)	36 (55.4)	65 (100)	

Table 3: Frequency distribution of E-cadherin expression in types of lesions based on the percentage of stained cells

Groups	<25%, *n* (%)	25%–<50%, *n* (%)	50%–75%, *n* (%)	>75%, *n* (%)	Total, *n* (%)	*P*
Reticular lichen planus	2 (3.1)	0	5 (7.7)	13 (20)	20 (30.8)	0.385
Erosive lichen planus	4 (6.2)	3 (4.6)	4 (6.2)	9 (13.8)	20 (30.8)	
Lichenoid lesions	1 (1.5)	1 (1.5)	2 (3.1)	16 (24/6)	20 (30.8)	
Normal tissue	0	0	0	5 (7.7)	5 (7.7)	
Total	7 (10.8)	4 (6.2)	11 (16.9)	43 (66.2)	65 (100)	

Table 4: Frequency distribution of E-cadherin expression status (normal-altered) based on the types of groups

Groups	Altered expression, *n* (%)	Normal expression, *n* (%)	Total, *n* (%)	*P*
Reticular lichen planus	5 (7.7)	15 (23.1)	20 (30.8)	0.038
Erosive lichen planus	9 (13.8)	11 (16.9)	20 (30.8)	
Lichenoid lesions	2 (3.1)	18 (27.7)	20 (30.8)	
Normal tissue	0	5 (7.7)	5 (7.7)	
Total	16 (24.6)	49 (75.4)	65 (100)	

no significant difference was observed in E-cadherin expression between reticular lichen planus and lichenoid lesions ($P = 0.158$). In addition, there was no statistically significant difference between the groups of reticular lichen planus ($P = 0.292$) and lichenoid lesions ($P = 0.633$) compared to healthy oral mucosa. In contrast, the alteration or abnormal expression of E-cadherin in the erosive OLP group was significantly greater than in healthy oral mucosa ($P = 0.038$).

In the healthy mucosa group, E-cadherin protein exhibited normal staining, in contrast to the other groups. The frequency of E-cadherin expression changes in buccal mucosa lesions was statistically significantly higher than in other locations ($P = 0.004$). This finding may be attributed to the much higher prevalence of lesions in the buccal mucosa, which accounted for 30% of the samples.

The change in E-cadherin expression in erosive lichen planus lesions, when compared to reticular lichen planus, lichenoid lesions, and healthy tissue, underscores the importance of accurately diagnosing these lesions and maintaining regular follow-up for patients diagnosed with erosive lichen planus. The decrease in E-cadherin protein expression observed in erosive OLP compared to healthy mucosa in this study aligns with the findings of Hämäläinen *et al*. in 2019. In their research, Hämäläinen *et al*. examined 54 samples of lichen planus alongside 22 samples of healthy oral mucosa using immunohistochemical techniques. They reported a decrease in E-cadherin protein expression in lichen planus samples compared to healthy mucosa.[18]

In line with this study, Du and Li noted a significant increase in the abnormal expression of E-cadherin in lichen planus samples, suggesting that E-cadherin may play a role in the malignant changes associated with the condition.[11] However, their research did not differentiate between the various clinicopathological forms of the lesions, which is an aspect considered in this study.

Akhtar *et al*. similarly demonstrated that as the severity of dysplastic changes increases, E-cadherin expression levels decrease. They suggested that assessing E-cadherin levels could be useful in estimating the likelihood of malignant change development and progression.[12] The results of the present study align with the findings of Hämäläinen *et al*., Akhtar *et al*., Chujo *et al*., and Du and Li.[11,12,18,19]

In the research conducted by Chujo *et al*., analysis of 25 samples of OLP revealed that staining in lichen planus lesions primarily occurs in the intercellular areas of the basal and spinous layers of the epithelium, while the stratum corneum exhibited different staining patterns. The study found that the methylation levels of E-cadherin and p16ink4a in OLP were significantly higher than in normal tissues, suggesting that hypermethylation of these genes may be associated with the pathogenesis and progression of OLP.[19]

The present study observed a decrease in E-cadherin staining and abnormal expression in lichen planus compared to healthy tissue. While the frequency of normal E-cadherin expression was lower in lichen planus cases–particularly in erosive lichen planus, there were no statistically significant differences between the other groups and healthy tissues. This suggests that although changes in E-cadherin expression are evident, they may not be sufficient to differentiate those groups statistically. The findings indicate that alterations in E-cadherin may be more pronounced in erosive lichen planus, warranting further investigation into the underlying mechanisms and their potential implications.

In this context, Sridevi *et al*. investigated OLP, squamous cell carcinoma (SCC), and oral leukoplakia and found a reduction in E-cadherin protein expression in these lesions compared to healthy tissue. However, they observed no significant correlation between the severity of dysplasia or the degree of malignancy and E-cadherin expression. As a result, they concluded that the utility of E-cadherin as a marker for assessing malignant changes remains uncertain.[20]

Tampa *et al*. noted in their systematic review that studies have reported contradictory results regarding

the role of E-cadherin in the malignant changes associated with lichen planus. This underscores the need for further research to clarify its significance in this context.[8]

Neppelberg and Johannessen studied 56 samples of OLP and concluded that despite a decrease in E-cadherin expression in the basal layer keratinocytes, E-cadherin cannot be considered a reliable marker for determining the occurrence of malignant changes. They showed that the decrease in expression occurred as small foci in the basal and spinous layers and was not statistically significant.[21]

In contrast, the present study found a more extensive and widespread decrease in E-cadherin expression in the erosive lichen planus group. Furthermore, this study considered not only the location, number of stained cells, and uniformity of staining but also employed a specific scoring scale to evaluate E-cadherin staining more comprehensively.

In fact, the difference of this study from other research, conducted in this field, was the use of three criteria (percentage of stained cells, uniformity of staining, location of membrane or cytoplasmic staining, or both) to determine the state of E-cadherin protein expression. The majority of studies had only investigated the percentage of stained cells. The difference in the results of these studies may be attributed to the various evaluation systems for the state of staining in addition to the sample size.

In their review, Sagari *et al*. showed no relationship between the decrease in E-cadherin expression and malignant changes in OLP. They highlighted that few studies have specifically focused on lichen planus in this context, with most research primarily evaluating SCC.[22]

Other studies have indicated that the severity of malignancy in lesions can influence E-cadherin expression. Specifically, an increase in E-cadherin expression has been observed in the early lesions of OSCC; however, as the lesions progress, the expression level of this protein decreases.[23]

In studies examining E-cadherin expression in oral lesions, particularly among Iranian populations, majority of research have focused on SCC, with a lack of simultaneous studies assessing E-cadherin expression in OLP and lichenoid lesions. Therefore, it would be impossible to compare the present results with the previous research in some cases.

Kalbasi *et al*. demonstrated a relationship between the histopathological grading of tumors and the expression of CAMs, suggesting that E-cadherin functions as a tumor suppressor that helps prevent the spread of lesions. This research focused on OSCC and highlighted the necessity for further studies on premalignant lesions.[24]

Based on the results of this study, it can be concluded that the site of the lesions may influence E-cadherin expression. Specifically, lesions on the buccal mucosa exhibited greater changes in E-cadherin expression, followed by lesions on the tongue. This finding aligns with research suggesting that the potential for malignant changes is higher in the tongue area.[5,6]

In Saberi *et al*.'s study, dysplastic changes were reported in 3 out of 416 patients with lichen planus and in 26 out of 450 patients with lichenoid lesions. The study concluded that the potential for malignant changes in lichenoid lesions is higher than that in lichen planus.[25]

Overall, E-cadherin expression in erosive lichen planus was significantly lower than in healthy tissue, demonstrating more pronounced changes compared to reticular lichen planus and lichenoid lesions. Although both reticular lichen planus and lichenoid lesions also exhibited a decrease in E-cadherin expression in comparison to healthy tissue, this reduction was not statistically significant.

CONCLUSION

Based on the findings of this research and the observed decrease in E-cadherin protein expression in erosive lichen planus samples, along with numerous studies on OSCC, it can be probably suggested that reduced E-cadherin expression may be associated with an increase in the severity of malignant changes. E-cadherin could potentially serve as an auxiliary marker, alongside other factors, for predicting the progression of premalignant lesions and for the early detection of malignant changes in their initial stages.

Eventually, E-cadherin protein's use could aid in the prevention and early detection of malignancies, facilitate regular follow-ups, and support targeted therapy planning.

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Conflicts of interest
The authors of this manuscript declare that they have

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