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Immunohistochemical study of glucose transporter protein expression in oral lichen planus

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ABSTRACT

Background: Oral lichen planus (OLP) is a chronic mucocutaneous inflammatory disease that is somewhat frequently manifested in various clinical forms: reticular OLP (ROLP) and erosive OLP (EOLP), and some cases are associated with dysplasia. Higher risk of malignant transformation has been linked to dysplastic alterations in OLP. Glucose transporter protein (GLUT1) is a transmembrane glycoprotein associated with increased glucose metabolism and proliferation of cells. This study's objective was to analyze and compare the expression patterns of GLUT1 in EOLP, ROLP, and lichen planus-related dysplasia in an attempt to acquire improved knowledge of the molecular pathways that underlie the etiology and advancement of OLP.

Materials and Methods: In this retrospective study, analysis of GLUT1 expression was done in 32 samples of OLP (16 for ROLP, 10 for EOLP, and 6 for OLP with dysplasia) with immunohistochemistry. Statistical analysis was performed using Pearson's Chi-square and F-tests, with significance set at $P < 0.05$. The immune GLUT-1 expression was evaluated semi-quantitatively and qualitatively at ×100 magnification.

Results: The mean percentage of GLUT1-positive cells in ROLP (16.53 ± 11.72) was lower than that in EOLP and OLP with dysplasia. Among the three groups, there was a significant difference in terms of staining intensity, intracellular location, and extent of GLUT1 immunoexpression within the epithelium layers (0.000, 0.034, and 0.006, respectively).

Conclusion: GLUT1 overexpression reflects increased glycolytic activity of proliferating cells in response to hypoxia and high energy requirements in EOLP and OLP-related dysplasia. GLUT1 expression may predict the malignant potential of OLP toward oral squamous cell carcinoma.

Key Words: Lichen planus, oral; glucose transporter type 1; carcinoma, squamous cell; neoplasm progression

INTRODUCTION

Oral lichen planus (OLP) is a chronic mucocutaneous inflammatory disease that is somewhat frequent, affecting 0.5%–2.2% of the population.[1,2] It is characterized by an immune-mediated reaction against epithelial cells, subepithelial band-like infiltration of T lymphocytes, basement membrane disruption, and basal cell degeneration.[1] OLP can manifest in various clinical forms, including reticular OLP (ROLP) and erosive OLP (EOLP). Both forms exhibit distinct

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clinical and histopathological features; whereas EOLP is known for its erosive and ulcerative lesions, ROLP presents as white, lacy patches.[2-4] Research indicates that LP is caused by a complex disease process, although the exact causes remain mysterious.[3-6]

Recently, altered glucose metabolism has been suggested to contribute to the pathogenesis of OLP and other chronic inflammatory conditions.[7,8] Glucose transporter protein (GLUT1), a transmembrane glycoprotein, is responsible for enabling glucose transfer into cells without the need for Na + and is associated with increased glucose metabolism and cell proliferation. Expression of GLUT1 redirects glucose metabolism toward glycolysis.[9-11]

GLUT1 has been implicated in various pathological conditions, including cancer.[8,12-14] However, research on the expression of GLUT1 in OLP and with altered glucose metabolism conditions, on the other hand, is limited. Moreover, OLP can generally progress to epithelial dysplasia, a premalignant transformation characterized by both cellular and structural changes.[1,2] The presence of dysplastic changes in OLP has been related to a higher risk of malignant transformation. Studies have suggested that lichen planus without dysplasia is not considered premalignant, whereas lichen planus with dysplasia is considered to have premalignant potential.[3,4,15-17] Nevertheless, there is still uncertainty regarding the mechanism behind this process. Herein, this study's objective was to analyze and compare the expression pattern of GLUT1 in EOLP, ROLP, and lichen planus-related dysplasia in an attempt to acquire an improved knowledge of the molecular pathways that underlie the etiology and advancement of OLP.

MATERIALS AND METHODS

This retrospective study involved 32 paraffin-embedded tissue samples of OLP, sourced from the archives of the Oral and Maxillofacial Pathology Department at the College of Dentistry, Baghdad University. Among these, 16 samples were from patients with ROLP, and 10 from those with EOLP. The diagnosis of OLP was confirmed both clinically and histologically, based on the modified WHO criteria proposed by Van Der Meij and Van Der Waal.[18] In addition, 6 samples were included from patients with OLP showing signs of oral epithelial dysplasia, where dysplastic changes occurred against the backdrop of OLP. This study was approved by the Research Ethics Committee of the College of Dentistry, University of Baghdad (Ref. No. 920, dated 15 July 2024; Project No. 920724).

GLUT-1 antigens were detected by immunohistochemical staining with the biotin-free Expose Mouse and Rabbit HRP/DAB Detection System (Abcam, Cambridge, UK). The primary steps involved deparaffinization, rehydration, antigen retrieval, and serial sectioning (in 4 μm sections). Following that, the sections were treated with GLUT-1 primary antibody (polyclonal; Abcam; dilution 1:200). Furthermore, the sections were exposed to a secondary antibody (complement) for 10 min at 37°C and washed in Phosphate Buffered Saline (PBS). Following a 5-min incubation period with DAB (diaminobenzidine) for visibility, the slides were washed, counter-stained with hematoxylin, and then mounted. A negative control was obtained by omitting the primary antibody. Erythrocytes in each section served as an internal positive control [Figure 1].

When the membrane, nucleus, or cytoplasm of the examined cells displayed brown staining, the sample was deemed positive for GLUT-1 expression. The immune GLUT-1 expression was evaluated semi-quantitatively and qualitatively at ×100 magnification. For semi-quantitative evaluation, the percentage of immunopositive cells was evaluated based on Remmele *et al*., 1987, criteria: 0 (negative), 1 (<10% positive cells), 2 (10%–50% positive cells), 3 (51%–80% positive cells), and 4 (more than 80%).[19] Three levels of staining intensity were assigned: 1 for mild, 2 for moderate, and 3 for high. The immunoreactive score (IRS) value, which is a number between 0 and 12, is the result of multiplying two scores, namely the proportion of positive cells and the staining intensity.[19] In qualitative analysis,

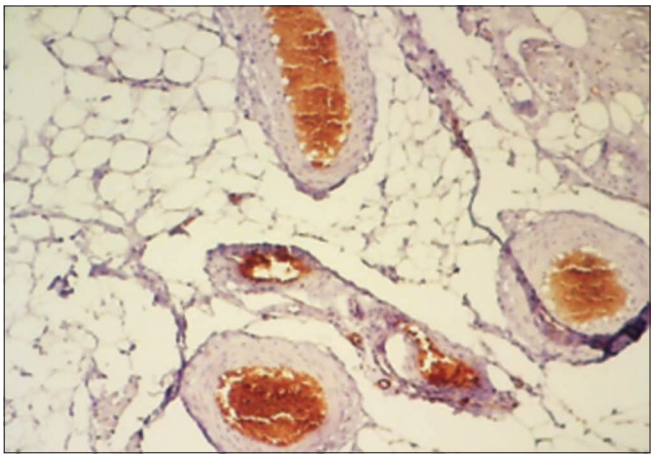

Figure 1: Internal positive control of GLUT1 expression in the vascular endothelium (x200).

the immunostaining location in cells (cytoplasmic, membrane, nuclear, or all three compartments).

Statistical analysis

Statistical analysis was carried out using Pearson's Chi-square and F-tests after immunohistochemical analysis, and the results were collated on a database using the program SPSS 24.0 (Statistical Package for the Social Sciences, SPSS Inc., Chicago, IL, USA). $P < 0.05$ was considered statistically significant.

RESULTS

Thirty-two cases of OLP were used in the current research (16 reticular, 10 erosive, and 6 cases of lichen planus-related dysplasia). Out of 32 cases, 24 patients were females (75%), and the rest were males (25%). The expression of GLUT-1 was readily visible as brown staining and was seen in the cytoplasm or cell membrane or both of them of epithelial cells. All three types of lichen planus (reticular, erosive, and lichen planus-related dysplasia) showed positive expression of GLUT-1.

Percentage of immunoreactivity

In ROLP, the average proportion of GLUT1-positive cells (16.53 ± 11.72) was lower than that in EOLP (57.1 ± 24.62) and lichen planus with dysplasia (56.42 ± 12.73). Regarding the expression of GLUT1, the number of immunostained cells revealed highly significant differences among the groups [Table 1].

Intensity of immunoreactivity

In ROLP, 8 of the sixteen cases (or 50%) were mild, and 8 (or 50%) were moderate. Out of the 10 cases of EOLP, 4 (40%) were moderate and 6 (60%) were intensely stained. In OLP with dysplasia, one was moderate (16.7%), and five were intense (83.3%). The *P* value was found to be statistically highly significant [Table 2 and Figures 2-4]. The Chi-square test was used to compare the IRS classifications of the groups. A statistically significant difference was shown by the IRS categorization, with a $P = 0.008$ [Table 3].

Table 1: Mean percentage of glucose transporter protein 1-positive epithelial cells by oral lichen planus subtypes

Group	Mean±SD	*F*-test	*P*
Reticular OLP (*n*=16)	16.53±11.72	28.678	<0.001
Erosive OLP (*n*=10)	57.10±24.62		
OLP with dysplasia (*n*=6)	56.42±12.73		

P<0.001 considered highly significant. Statistical analysis performed using one-way ANOVA. SD: Standard deviation; OLP: Oral lichen planus

Intracellular location of immunoreactivity

The intercellular location of GLUT1 immunoexpression was compared among the groups. In ROLP, most cases demonstrated predominantly combined membranous and cytoplasmic expression (11 cases, 68.75%), whereas the majority of OLP-related dysplasia cases showed membranous expression. The immunoexpression in EOLP cases was 4 cases (40%) for each membranous, membranous, and cytoplasmic location. This difference in intercellular GLUT1 expression was statistically significant with a $P = 0.034$ [Table 4 and Figures 5, 6].

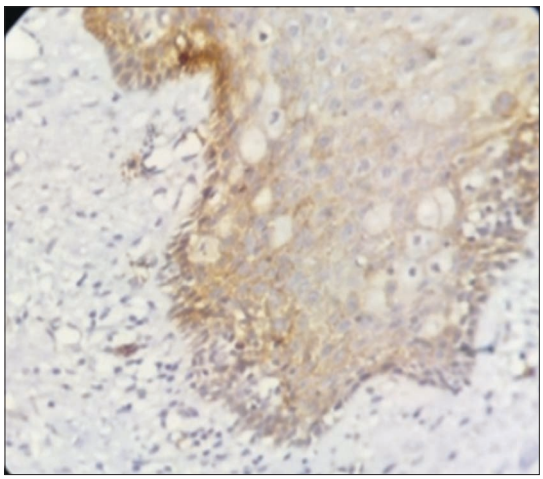

Figure 2: Photomicrograph showing mild glucose transporter protein 1 expression in reticular oral lichen planus ×400.

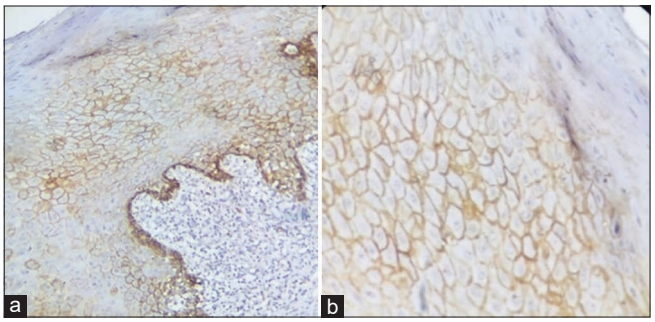

Figure 3: Photomicrograph showing moderate glucose transporter protein 1 expression oral lichen planus. (a) ×100, (b) ×400.

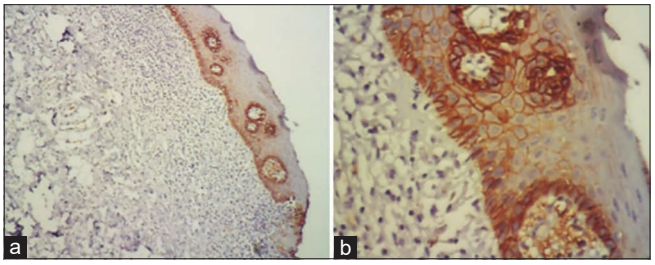

Figure 4: Photomicrograph showing intense GLUT1 expression in OLP related dysplasia (a) ×100 (b) ×400.

Table 2: Distribution of glucose transporter protein 1 immunostaining intensity across oral lichen planus subtypes

Group	Mild, *n* (%)	Moderate, *n* (%)	Intense, *n* (%)	χ^2	*P*
Reticular OLP (*n*=16)	8 (50.0)	8 (50.0)	0	43.08	<0.001
Erosive OLP (*n*=10)	0	4 (40.0)	6 (60.0)		
OLP with dysplasia (*n*=6)	0	1 (16.7)	5 (83.3)		

P<0.001 considered highly significant. OLP: Oral lichen planus

Table 3: Immunoreactive score classification among oral lichen planus subtypes

Group	Weak, *n* (%)	Mild, *n* (%)	Strong, *n* (%)	Total (*n*)	χ^2	*P*
Reticular OLP	16 (100.0)	0	0	16	8.97	0.008
Erosive OLP	1 (10.0)	5 (50.0)	4 (40.0)	10		
OLP with dysplasia	0	3 (50.0)	3 (50.0)	6		

P<0.05 considered significant. IRS calculated as product of proportion score (0–4) and intensity score (1–3); categorized as: Weak (IRS 0–3), Mild (IRS 4–6), Strong (IRS 7–12). OLP: Oral lichen planus; IRS: Immunoreactive score

Table 4: Intracellular localization of glucose transporter protein 1 immunoreactivity in oral lichen planus subtypes

Group	Membranous, *n* (%)	Membranous + cytoplasmic, *n* (%)	Cytoplasmic, *n* (%)	χ^2	*P*
Reticular OLP (*n*=16)	3 (18.8)	11 (68.8)	2 (12.5)	7.12	0.034
Erosive OLP (*n*=10)	4 (40.0)	4 (40.0)	2 (20.0)		
OLP with dysplasia (*n*=6)	4 (66.7)	2 (33.3)	0		

P<0.05 considered significant. OLP: Oral lichen planus

Table 5: Epithelial layer involvement of glucose transporter protein 1 expression by oral lichen planus subtype

Group	Basal only, *n* (%)	Basal + suprabasal, *n* (%)	Full-thickness, *n* (%)	χ^2	*P*
Reticular OLP (*n*=16)	11 (68.8)	4 (25.0)	1 (6.3)	8.029	0.006
Erosive OLP (*n*=10)	0	4 (40.0)	6 (60.0)		
OLP with dysplasia (*n*=6)	0	4 (66.7)	2 (33.3)		

P<0.05 considered significant. Full-thickness: Expression in basal, suprabasal, and superficial layers. OLP: Oral lichen planus

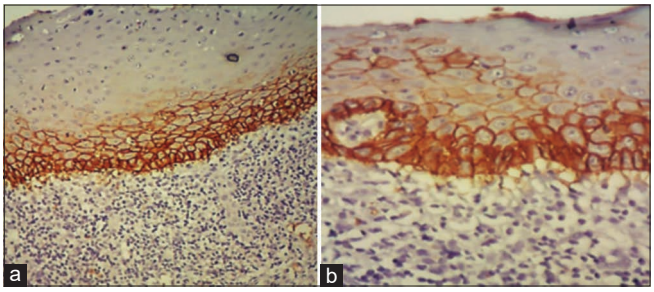

Figure 5: Combination membranous and cytoplasmic GLUT1 expression in OLP (a) ×100 (b) ×200.

Extent of glucose transporter protein immunoexpression

Out of 16 cases of ROLP, 11 cases (68.75%) showed expression in basal cells only, and 4 cases showed basal and suprabasal cells. In EOLP, 6 cases (60%) showed full-thickness expression [Figure 3], followed by 4 cases (40%) showing expression in basal and suprabasal layers. The majority of OLP with dysplasia demonstrated combined basal and suprabasal expression [Figure 5]. The extent of GLUT1 immunoexpression was statistically significant with $P < 0.001$ [Table 5].

DISCUSSION

OLP is a chronic inflammatory mucocutaneous disorder with an unknown cause,[20] which is considered a precancerous lesion with the potential ability to undergo malignant transformation.[21] Standardized screening and better follow-up for patients with oral precancerous lesions are made possible by the discovery of trustworthy biomarkers for the detection of malignant transformation. There is hope for the practical integration of these methods with other approaches, including multimodal cell analysis for brush biopsies in the early identification of lesions that may be premalignant.[22] Carcinogenesis is a multistep

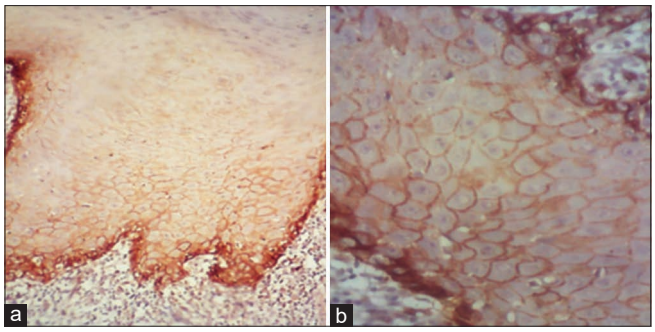

Figure 6: Membranous GLUT1 expression in OLP (a) ×200 (b) ×400.

process that relies on the breakdown of general barriers imposed by cells, including senescence, programmed cell death, and antiproliferative response. The first mechanism that is impacted when normal tissue gives way to malignant cells is the cellular energy metabolism; in particular, tumor cells usually have disruptions in the metabolism of glucose. One of the seven characteristics of cancer is thought to be an increase in glucose metabolism.[23] There are 14 members in the GLUT family, with GLUT-1 being the earliest member and serving as the primary mechanism for glucose uptake. The activity of the glucose transporter increases in response to an increase in glucose metabolism.[24,25]

Although few studies have focused on comparing the different types of OLP in terms of metabolic activity, this study is the first to specifically investigate GLUT1 expression across ROLP, EOLP, and OLP with dysplasia. The primary aim of this research was to determine whether variations in metabolic activity, as indicated by GLUT1 expression, exist between these OLP subtypes. By examining differences in GLUT1 immunoexpression, we sought to assess the potential role of metabolic alterations in the progression and severity of these clinical forms of OLP.

It appears that GLUT1 is crucial for carcinogenesis and may have several functions in the malignant transformation of OLP.[7] According to certain research, poor prognosis was found to be correlated with GLUT1 overexpression in many cancer types.[26] In the present study, GLUT1 expression of OLP with dysplasia, reticular, and erosive specimens was examined, with all cases showing positivity. These results were in agreement with Wang *et al*., who reported that the expression of GLUT1 in patients with OLP was noticeably higher than that in normal oral mucosal tissue.[7] the mean percentage of cells positive for GLUT 1 in both EOLP and OLP with dysplasia was significantly higher compared with ROLP. According to this finding, the overexpression of GLUT-1 is thought to be an early step in a malignant transformation and indicates that the likelihood of a malignant transformation in EOLP and lichen planus-related dysplasia is higher than that of ROLP.

Concerning GLUT-1 staining intensity, the majority of EOLP and OLP with dysplasia cases frequently showed strong staining. Strong GLUT-1 staining was also seen in earlier research on benign and malignant tumors.[27,28] Although the staining intensity may have an impact on the lesions' development, it doesn't appear to be related to the biological characteristics of the lesions that are being studied.

A significant shift in the intracellular location of GLUT1 expression was observed in our study, with the majority of OLP with dysplasia lesions exhibiting membranous positivity. These findings were consistent with a study by Harshani *et al*. that discovered that in all OSCC grades, GLUT1 expression was primarily membranous.[29] While the majority of ROLP showed a combination of both cytoplasmic and membranous staining and cytoplasmic positivity, these results were in disagreement with Wang *et al*., who reported that GLUT1 stained moderately in the cytoplasm of basal and spinous cell layers of epithelium.[7] Anti-GLUT-1 antibody usually finds membrane-linked proteins on epithelial cells. An alteration in GLUT-1's intrinsic activity, kinetics, and expression is part of the hypoxic induction process. The protein first undergoes "unmasking," which raises its glucose affinity. A subsequent increase in the synthesis of GLUT-1 mRNA is the consequence of further stimulation, which causes the present glucose transporters to relocate from cytoplasmic vesicles to the plasma membrane. In their investigation, Ariely *et al*. found a correlation between the amount and duration of hypoxia in various regions and the cytoplasmic and membranous expression of GLUT-1 in lesions. They proposed that GLUT-1's co-localization with the Golgi results in its simultaneous cytoplasmic and membrane expression.[30]

Regarding ROLP, in the majority of cases, GLUT1 expression was noted in the basal layers, whereas in EOLP and OLP-related dysplasia, it reached the spinous layer in some cases and involved the full epithelium in most cases. Our results are consistent with the research of Burstein *et al*., who found that

GLUT1 expression increases from basal layers to superficial layers in response to dysplasia severity. Their findings also indicate that GLUT1 enhancement is a primary change in the development of squamous cell carcinoma.[31] The overexpression of GLUT-1 in ROLP's basal layers revealed that basal cells were using more glucose to meet their metabolic requirements, which is especially connected to cell division. The relationship between the OLP types and GLUT-1 expression has been connected to the cells' glycogen content. Glycogen is linked to the squamous epithelium's cellular maturation and withdraws with loss of differentiation during neoplastic transformation; in nondysplastic epithelium, it is highly concentrated, whereas in dysplasia, it is either missing or substantially reduced.[32] When the glycogen content was examined to see if any changes in glucose absorption were connected to tumor-associated changes, it was found that nondividing cells in the superficial layers of benign cervical epithelium had higher glycogen contents. Consequently, it appears that as the degree of dysplasia grows, decreased glycogen levels are substantially linked to increasing GLUT-1 expression.[33,34] Such a finding in relation to OLP types was not described in any literature for a comparison.

CONCLUSION

The results of the study indicate variations in GLUT1 immunoexpression among ROLP, EOLP, and OLP with dysplasia. The least amount of GLUT1 was found in ROLP, while the rest of the EOLP and OLP with dysplasia had higher and more intense GLUT1 levels. Distribution of positive cells was also different, since ROLP showed mostly combined membranous and cytoplasmic expression, whereas OLP dysplasia showed mostly membranous expression. EOLP showed more mixed expression. In addition, in ROLP, GLUT1 was expressed mainly in the basal cells, while in EOLP and OLP with dysplasia, expression extended beyond the basal layer of epithelial cells. These differences suggest that GLUT1 may be involved in OLP progression, especially in more severe forms, and could serve as a marker for disease severity.

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Conflicts of interest

The authors of this manuscript declare that they have no conflicts of interest, real or perceived, financial or non-financial in this article.

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