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Assessment of E-cadherin Expression in Oral Epithelial Dysplasia and Squamous cell carcinoma

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ABSTRACT

Introduction: Oral squamous cell carcinoma (SCC) accounts for about 90% of all oral cancers and is the sixth most frequent type of cancer globally. The risk of developing SCC is high in precancerous lesions of the oral cavity. SCC undergoes a multistep carcinogenic process. Various molecular mechanisms are involved in this process, and E-cadherin is one such molecule. E-cadherin is a 120-kDa calcium-dependent cell adhesion glycoprotein. Downregulation or aberrant expression of E-cadherin has been observed in precursor lesions and invasive cancers of the cervix, esophagus, and head and neck areas.

Aim: The current study was designed to assess the immunohistochemical expression profile of E-cadherin in oral premalignant and malignant lesions and to compare the expression levels of E-cadherin in varying grades of epithelial dysplasia and carcinoma.

Materials and methods: This cross-sectional study was carried out in the Department of Pathology, Great Eastern Medical School & Hospital, Srikakulam, Andhra Pradesh. The study period was one year, from July 2023 to June 2024. Systematic random sampling was used to collect a total of 80 cases. The premalignant and malignant lesions of the oral squamous epithelium were assessed based on the 2017 World Health Organization criteria and for E-cadherin expression by immunohistochemistry. The intensity of staining was scored based on the percentage of epithelial cells showing continuous and uniform membranous staining: 0 (negative) for 0-10%, 1+ (loss) for 11-25%, 2+ (weak) for 26-50%, 3+ (strong) for 51-75%, and 4+ (intense) for >75%. The data were analyzed using SPSS version 20.0.

Results: A marked variation in E-cadherin expression was noticed among the varying grades of premalignant lesions ($p = 0.0163$). The expression varied from strong in mild dysplasia to weak in higher grades of dysplasia. Among premalignant lesions, 13 lesions showed strong (4+) immunoreactivity, 8 lesions showed strong (3+) expression, and 2 lesions showed weak (2+) expression. Likewise, the expression of E-cadherin was significantly different among the different grades of malignant lesions ($p = 0.0003$), with poorly differentiated SCC having no expression and well-differentiated lesions having intense staining. Among the malignant lesions, 2 had negative (0) expression, 16 had intense (3+) expression, 12 had weak (2+) expression, and 9 had (1+) expression. The difference in expression between premalignant and malignant lesions was also significant ($p < 0.0001$).

Conclusion: With the progressive grades of dysplasia and malignancy, the expression of E-cadherin decreases. Therefore, there is a negative correlation between the expression of E-cadherin and the grades of dysplasia and malignancy.

Keywords: *E-cadherin, Immunohistochemistry, Oral premalignant and malignant, Pattern of expression.*

INTRODUCTION

Squamous Cell Carcinoma (SCC) constitutes 90% of the malignancies of the oral cavity. Overall, oral cancer is the 6th most common cancer worldwide. The global incidence of oral cancer is 4 cases per 100,000 population, and in India, it is more than 10 per 100,000 population due to the high prevalence of tobacco chewing [1]. The mortality rate in India due to oral cancer is 3-6.7 per 100,000 population [2]. The progression of premalignant lesions into carcinoma is a multistage process involving numerous molecules, with E-cadherin being one of the key molecules [3,4]. E-cadherin is encoded by the CDH1 gene located on chromosome 16q21. It is a calcium dependent transmembrane glycoprotein which is expressed in most epithelial cells and helps in maintaining cell polarity and normal tissue architecture [5]. E-cadherin is a 120kDa glycoprotein composed of three components: extracellular, cytoplasmic, and transmembrane domains. The extracellular domain contains five tandem repeats, each consisting of a 100-residue amino acid motif. The N-terminal of these tandem repeats contains adhesive activity sites. The extracellular domain also has binding sites for calcium ions in the pockets between these tandem repeats. The calcium binding site has an amino acid sequence specific to each cadherin family and different species. Cell-cell adhesion is mediated through interactions of extracellular domains in a process of lateral dimerisation [6,7]. The cytoplasmic domain consists of the Juxtamembrane Domain (JMD) and Catenin-Binding Domain (CBD), each containing around 30-35 residues. The JMD allows the clustering of cadherins and contributes to adhesive strength through p120-catenin [8]. The CBD interacts with β-catenin and γ-catenin. The α-catenin then links the bound β-catenin to the actin cytoskeleton, promoting protein clustering at adherence junctions and stabilising cell-to-cell adhesion [7].

Cell adhesion molecules play more than a purely structural role within stratified squamous epithelia. There is a strong relationship between reduced expression of these adhesion molecules, decreased differentiation and increased invasiveness[12]. The malignant transformations are generally associated with the loss of the epithelial phenotype and differentiation[13]. The epithelial-to-mesenchymal transition (EMT) features are seen in oral epithelial dysplasia (OED) and in their progression to malignancy[14]. During the progression, there is a loss of cohesion. It is a result of a defect at the cell adhesion molecular level.[14]. Normally, E-cadherin stains the epithelial cells of the oral mucosa, sparing the connective tissue. It is expressed as strong, membranous, homogeneous staining of the basal, parabasal, and superficial cells [4,8,9]. The staining is absent or weak in the basal aspect of basal cells and in the most superficial cells [10]. E-cadherin acting as tumour suppressor protein, is expressed abundantly on the normal cell membrane, but its expression progressively reduces through the phases of epithelial dysplasia, carcinoma-in-situ and invasive cancer [15]. The loss of membrane expression of E-cad is said to enhance the invasiveness of tumour by inhibiting cellular adhesion, differentiation, and apoptotic ability [16]. Vimentin, β-catenin, and p53, CD44, podoplanin, MMP-9, and EGFR are the other tumor markers that have roles in cancer biology, their expression patterns and implications in oral squamous cell carcinoma(OSCC) and squamous dysplasia may not provide the same level of specificity and reliability as E-cadherin in assessing tumor invasiveness and Epithelial mesenchymal transition. Altered expression of E-cadherin has been described in preinvasive lesions, and loss of expression has been noted in malignancies such as the cervix, oesophagus, including the head and neck [3,6]. Hence, the aim of this study was to investigate the Immunohistochemical (IHC) expression pattern of E-cadherin in premalignant and malignant lesions of the oral cavity and to examine the relationship between E-cadherin IHC expression and different grades of epithelial dysplasia.

MATERIALS & METHODS

This cross-sectional study , was carried out in the Department of Pathology at Great Eastern Medical School &hospital, Srikakulam, Andhra Pradesh. The study duration was one year, from July 2023 to June 2024 after the approval from the Institutional Ethical Committee (247/IEC/GEMS&H/2024). Clinical details were procured from the patients, histopathology requisition forms and the hospital information management system (HIMS). All cases underwent standard processing and were stained with hematoxylin and eosin (H&E) for analysis.

***Inclusion criteria**:* All biopsy samples of oral mucosa showing dysplastic and malignant squamous lesions were selected. All cases of oral dysplastic and malignant squamous lesions received during the study period were noted, and systematic random sampling was performed to select 80 cases

***Exclusion criteria**:* Cases with a history of prior chemotherapy or radiotherapy and cases where the tissue sample was very scanty and IHC could not be performed were excluded from the study.

Statistical Analysis: The data were analyzed using descriptive statistics and expressed as percentages. The relationship between E-cadherin immunoexpression and microscopic features (WHO histological grading) was evaluated by calculating Pearson's correlation coefficient (r). The results were tabulated, and p-values were determined using SPSS version 20.0 (SPSS v20.0) software. A p-value of less than 0.05 was considered statistically significant.

Immunohistochemistry procedure: Antibody to E-cadherin (EP700Y) (Rabbit Monoclonal Primary Antibody, prediluted, Cell Marque) was used as the primary antibody, and sections of infiltrating duct carcinoma of the breast were used as positive controls. Tissue sections without incubation with primary antibodies were used as negative controls.

Slides were placed at 60°C in a hot air oven for 60 minutes and then immersed in xylene for two changes of 10 minutes each for deparaffinization. Slides were passed through decreasing grades of alcohol and running water for 5 minutes each. Antigen retrieval was performed using Tris EDTA-based antigen retrieval solution with a pH of 9. The microwave method of antigen retrieval was employed in our Immunohistochemistry setup, also known as the Heat Induced Epitope Retrieval (HIER) method. Slides were washed three times with wash buffer (pH 7.2–7.6) for 1 minute each, following which peroxidase-free blocking reagent was applied to the sections and allowed to act for 10 minutes. The slides were kept in a humid chamber. The primary antibody was added to the respective slides and incubated for 60 minutes in a humid chamber at room temperature. Slides were washed with wash buffer, and Hi-Definition Amplifier was applied to the tissue sections and incubated for 30 minutes. Slides were washed again with wash buffer, and Polymer Hi-Definition HRP Label was added to the sections and incubated for 30 minutes inside the humid chamber at room temperature. After washing with wash buffer, DAB chromogen was added to the sections and left for 10 minutes in the humid chamber. The slides were then washed twice with distilled water and counterstained with hematoxylin. The slides were washed under running tap water for 10 minutes, serially dehydrated in alcohol, and mounted in DPX. After drying, the test slides were examined along with the control sections (stained simultaneously) by light microscopy.

Evaluation of immunohistochemistry: In the assessment of the expression of E-cadherin, the proportion of positive tumor cells was visually estimated under microscope. The immunostaining was graded [10] for statistical purposes as follows:

Table 1: Grading of immunostaining	
Negative:	Staining of 0 to 10% of cells
1+ (loss):	Staining of 11-25% of cells
2+ (weak):	Staining of 26 to 50% of cells
3+ (strong):	Staining of 51% to 75% of cells
4+ (intense):	Staining of more than 75% of cells.

Results

In the current study, we have assessed 80 cases of premalignant and malignant squamous lesions of the mouth. Among them, 57 cases (71.25%) were malignant and of different grades, and 23 cases (28.75%) were premalignant.

In addition, out of 80 cases, 57 were oral squamous cell carcinoma (OSCC), with a male to female ratio of 3:1, comprising 44 males and 13 females.

Age, gender, and cancer information:

The age of the participants varied from 21 to 90 years. The majority of the participants belonged to the 41-50 years age group (13 patients) and 51-60 years age group (16 patients). Relatively fewer patients belonged to the younger age groups, namely 21-30 years (1 patient) and 31-40 years (12 patients). In the older age groups, there were 7 patients in the 61-70 years and 71-80 years age groups, and 1 patient above 80 years as shown in Table 2.

Table 2: Age distribution of premalignant and malignant cases			
	Dysplastic lesions	**Malignant lesions**	**Total**
Age group			
21- 30	1	1	2
31-40	2	12	14
41-50	8	13	21
51-60	5	16	21
61-70	6	7	13
71-80	1	7	8
81-90	0	1	1

In almost all age groups, the number of males was higher than that of females, indicating that these lesions are more likely to occur in mid to late life as shown in Table 3 &4.

Table 3: Gender& Age distribution of premalignant and malignant cases			
Age group	**Total**	**Male**	**Female**
21- 30	01	01	00
31-40	12	10	02
41-50	13	13	00
51-60	16	11	05
61-70	07	04	03
71-80	07	04	03
81-90	01	01	00

Table 4: Sex distribution of cases			
Cases	**Male**	**Female**	**Total**
Dysplastic lesions	17	6	23
Malignant lesions	44	13	57
Total	61	19	80

Among the malignant lesions, 37 cases (64.91%) had well-differentiated squamous cell carcinoma, and 12 cases (21.05%) had moderately differentiated squamous cell carcinoma. The lowest incidence of malignant lesions was weakly differentiated squamous cell carcinoma, which occurred in 8 cases (14.03%) as shown in Table 5.

Table 5: Distribution of malignant lesions		
Malignant lesions	**Number**	**Percentage**
Well differentiated SCC	37	64.91 %
Moderately differentiated SCC	12	21.05 %
Poorly differentiated SCC	8	14.03 %

Immunohistochemical studies for E-cadherin expression showed equal membrane staining of the epithelial layers in all normal tissues. There was also strong expression in well-differentiated squamous cell carcinoma, with 4+ staining in 13 out of 37 cases and 3+ staining in 14 out of 37. However, as there was a gradual loss of differentiation, the expression of E-cadherin fell drastically. Only two out of twelve cases of poorly differentiated carcinomas showed 3+ staining, while only one showed 4+ staining as shown in Table 6. There is a gradual loss of E-cadherin expression as there is a loss of differentiation Figure 1, 2 & 3.

Table 6: Pattern of E-Cadherin immunostaining in malignant lesions						
Grades of OSCC	**No.of cases**	**+4**	**+3**	**+2**	**+1**	**0**
Well differentiated	37	13	14	6	4	0
Moderatelydifferentiated	12	5	1	4	2	0
Poorly differentiated	8	0	1	2	3	2
Total	57	18	16	12	9	2

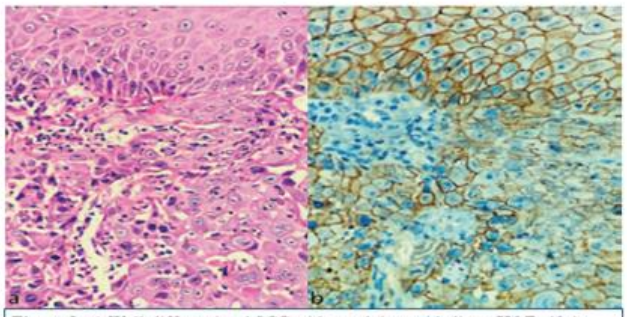

Figure 1: a) Well-differentiated SCC with overlying epithelium (H&E, 40x). b) Tumour showing reduced expression of E-cadherin, in the form of discontinuous membranous and few with absent staining, (2+, weak). Overlying epithelium shows normal expression (40x) (3+, strong)

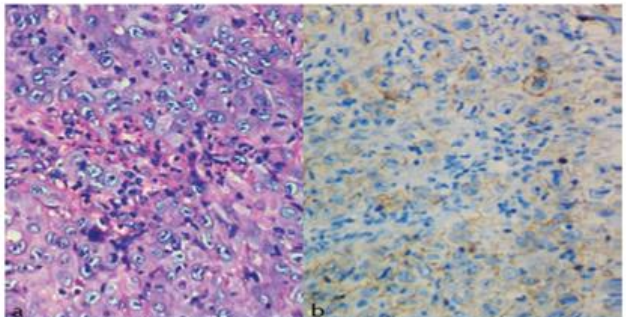

Figure 2: a) Moderately differentiated SCC showing tumour cells in sheets (H&E, 40x). b) E-cadherin expression showing tumour cells with decreased intensity of staining and loss of expression in few cells (40x). (1+, loss).

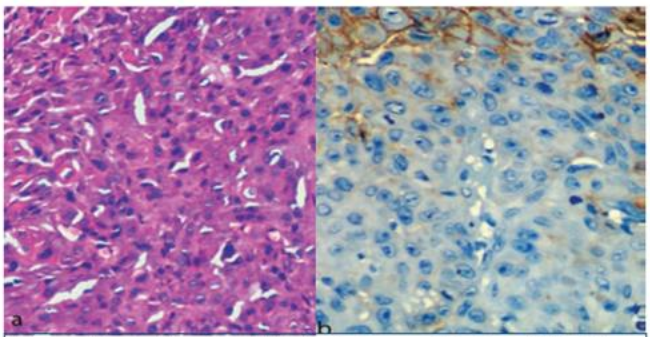

Figure 3: a)Poorly differentiated SCC showing tumour cells in sheets (H&E, 40x). b) E-cadherin expression showing predominantly loss of expression in tumour cells (40x). (0, negative).

E-cadherin expression in premalignant lesions varies according to the degree of dysplasia. +4 expression was seen in seven lesions with mild dysplasia. In mild dysplasia, three lesions had +3 expression and six had +4 expression. Two

lesions showed +2 expression and one lesion showed +3 expression, indicating that expression was lost in extreme dysplasia. Of the twenty-three lesions impacted by dysplasia, two had +2 expression, eight had +3 expression, and thirteen had +4 expression as shown in Table 7.

Table 7: Pattern of E- cadherin immunostaining in premalignant lesions

E-cadherin expression	Degree of Dysplasia			
	Mild Dysplasia	Moderate Dysplasia	Severe Dysplasia	Total
+4	07	06	00	13
+3	04	03	01	08
+2	00	00	02	02
+1	00	00	00	00
0	00	00	00	00
TOTAL	11	09	03	23

The findings demonstrated that premalignant lesions with a lower grade had increased E-cadherin expression ($p = 0.0163$) as shown in Figure 4,5&6 . This was also seen in malignant tumors, with a somewhat negative correlation ($p = 0.00037$). A moderately negative correlation ($p < 0.0001$) was found between the expression of E-cadherin in malignant and premalignant lesionsas shown in Table 8. E-cadherin expression in premalignant lesions with and without dysplasia showed a slight negative correlation, however it was not statistically significant .

Table 8: Correlation of E- Cadherin expression in premalignant and malignant lesions

Correlation of E- cadherin expression	R- value	p- value
With grades of premalignancy	-0.495	0.0163
With grades of malignancy	-0.46	0.00037
Premalignant vs malignant	-0.39	0.00031

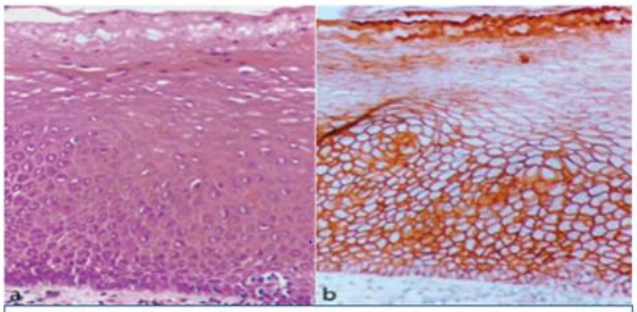

Figure 4: a) Higher magnification of mild dysplasia showing, crowding, anisonucleosis and mild nuclear pleomorphism in lower 1/3rd of the epithelium (H&E, 40x).
b) E-cadherin expression in mild dysplasia showing expression similar to normal mucosa with mild reduction of staining of cells in upper prickle layer (40x). (4+, intense).

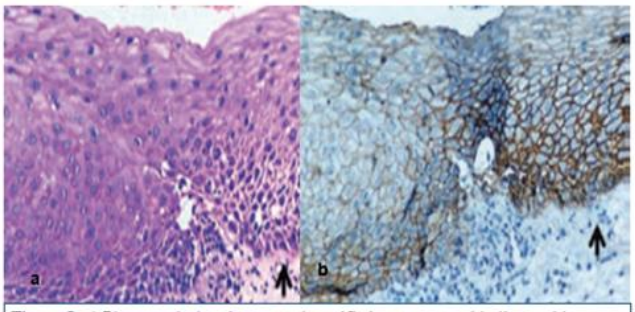

Figure 5: a) Photograph showing normal stratified squamous epithelium with adjacent areas showing moderate dysplasia (single arrow) (H&E, 40x)
b) Photograph showing E-cadherin expression in normal stratified squamous epithelium with adjacent areas showing moderate dysplasia (single arrow)

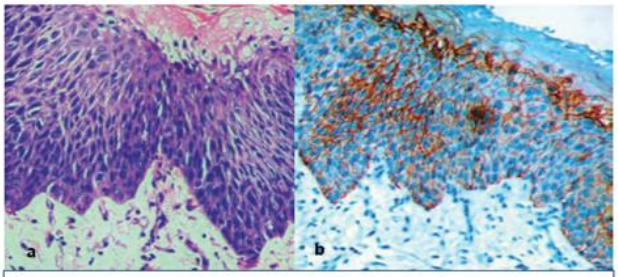

Figure 6: a) Photograph showing severe dysplasia. Cells in more than 2/3rd of the epithelium show loss of polarity, marked anisonucleosis, nuclear pleomorphism and hyperchromasia. The cells in superficial layer show maturation with keratinisation (H&E, 40x). b) E-cadherin expression is reduced in all the layers with only focal areas and few cells in upper prickle layer showing normal staining (40x). (3+, strong).

DISCUSSION

The present trend in cancer studies is changing towards understanding the molecular and genetic pathways behind cancer, rather from being constrained to morphological diagnosis. Immunohistochemistry (IHC) is an auxiliary test that has a direct link to the genetic evolution of cancer. Enhanced knowledge of the molecular causes of cancer can help in precise diagnosis and successful management techniques [10]. The process of malignancy in tumours is connected to the loss of epithelial differentiation and the gain of a mesenchymal phenotype. This is characterized by a decrease in epithelial gene expression and an increase in mesenchymal gene expression, which leads to increased cell motility, loss of cell adhesion, and loss of polarity. These molecular processes occur early in the pathogenesis of cancer, even in dysplasia. The

discovery of these transition molecules, like as E-cadherin, can be exploited as biomarkers for the detection of high-risk lesions [4].

In our study, E-cadherin was extensively and intensely expressed in oral premalignant lesions without dysplasia and with mild dysplasia, just like in normal mucosa. With the advancement of the grades of dysplasia, the intensity of E-cadherin expression dropped. Twenty percent of cases with moderate

dysplasia had weak E-cadherin expression, while fifty percent of those with severe dysplasia had weak or no E-cadherin expression. This expression pattern is consistent with recent investigations conducted by Sharma J et al. [17] and Thankam DR et al. [16]. Gupta A et al. found high levels of E-cadherin in 14.28% of instances of severe dysplasia, which contradicts our findings as shown in Table 9.

Table 9: Summary of comparison of E-cadherin expression in premalignant lesions with other studies {percentage (number of cases)} [17-19]

Study	Grading of E-cadherin expression	Present Study (n=23)	Basavraj. et.al(n=27)	Sharmaj. et.al(n=40)	Gupta.a. et.al (n=28)	Thankam. D.R.et.al (n=21)
Year of study		2025	2023	2022	2018	2021
Mild dysplasia	0-50%	-	-	100%(8)	-	-
	51-75% strong	17.39%(4)	28.57%(2)		14.28(1)	50%(1)
	76-100% Intense	30.43% (7)	71.43%(5)		85.71(6)	50%(1)
Moderate Dysplasia	0-25%		0%	-		
	26-50% weak		20%(1)	62.5%(3)		
	51-75% strong	13.04% (3)	40%(2)	37.5%(5)	57.14%(4)	100% (3)
	76-100% Intense	26.08% (6)	40%(2)		42.85%(3)	
Severe dysplasia	0-10% negative		0 %			
	11-25% loss	8.69%(2)	25%(1)	50%(2)		
	26-50% weak	4.34%(1)	25%(1)	50%(2)		
	51-75% strong		50%(2)		85.71(6)	100%(3)
	76-100% intense		0%		14.28(1)	

Sridevi U et al. found poor expression of E-cadherin in 75% of patients of moderate dysplasia (10). In Oral Submucous Fibrosis (OSMF), significant expression was identified in 60% of patients with moderate dysplasia, and in 33% of instances of leukoplakia with moderate dysplasia. However, E-cadherin expression in instances of dysplasia was not statistically significant.

The expression of E-cadherin in Squamous Cell Carcinomas (SCC) decreased as the grade of malignancy increased. Well-differentiated SCC displayed robust expression in 41% of patients, similar to severe dysplasia. Moderately differentiated SCC displayed negative expression in 50% of patients. IHC staining was shown to be lower in focal areas with high-grade cytologic atypia in well- and moderately differentiated SCC than in other areas with primarily low-grade cytologic atypia.

Moreover, in well and moderately differentiated carcinomas, the intensity of E-cadherin staining in tumor cells was variable within the tumor. There was variation between cells at the invasive front and other cells. Moreover, in well and moderately differentiated carcinomas, the intensity of E-cadherin staining in tumor cells was variable within the tumor. There was variation between cells at the invasive front and other cells.

In poorly differentiated SCC, there was negative E-cadherin in all cases, with few cells (less than 10%) showing complete membranous staining where differentiation was maintained. This is consistent with Sharma J et al., Gupta A et al., and Khant HR [17,19,20]. However, Gupta A et al. observed mostly negative E-cadherin in moderately differentiated SCC, which is consistent with our study [19]. In poorly differentiated SCC, Sharma J et al., Gupta A et al. observed mostly negative expression and loss of E-cadherin, as in our study, while Khan HR et al. observed mostly strong expression [17,19,20]asshown in Table 10.

Table 10: Summary of comparison of E-cadherin expression in malignant lesions with other studies {percentage (number of cases)} [17,18,20].

Study	Grading of e cadherin expression	Present Study n=57	Basavraj. et.al n=27	Sharmaj. et.al n=20	Gupta.a. et.al n=21	Khan H.R.et.al n=37
Year of study		2025	2023	2022	2018	2021
Well differentiated	0-10% negative		17.65%(3)		-	-
	11-25% loss	7.01%(4)	29.41%(5)			

	26-50% weak	10.52% (6)	11.76%(2)	25%(2)	28.57(2)	16.66%(2)
	51-75 strong	24.56% (14)	41.18%(7)	75%(6)	57.14(4)	83.33%(10)
	76-100 Intense	22.80% (13)			14.28%(1)	
Moderately Differentiated	0-10% negative		50%(3)		57.14%(4)	
	11-25% loss	3.50% (2)	16.66%(1)	37.50%(3)	14.28%(1)	
	26-50% weak	7.01% (4)	16.66%(1)	62.50%(5)		53.84%(7)
	51-75 strong	1.75% (1)	16.66%(1)		28.57%(2)	46.15%(6)
	76-100 Intense	8.77% (5)				
Poorly differentiated	0-10% negative	3.50% (2)	100%(4)		100%(7)	
	11-25% loss	5.26% (3)		100%(4)		8.33%(1)
	26-50% weak	3.50% (2)				41.66(5)
	51-75% strong	1.75% (1)				50%(6)
	76-100%intense					

In our research, the pattern of E-cadherin expression demonstrated a statistically significant difference between the grades of dysplasia, grades of SCC, and between dysplasia and SCC. This is in agreement with Gupta A et al., Santos-García A et al., Yuwanati MB et al., and Kaur J et al., who demonstrated a statistical significance between E-cadherin expression and grades of dysplasia and SCC [17,21-23]. Zeidler SVV et al., Khan HR et al., Santos-García A et al., and Akhtar K et al. demonstrated a statistical significance between E-cadherin expression and grades of SCC [4,20,21,24].

CONCLUSION

We also observed that the greater the grade of dysplasia and malignancy, the lower the expression of E-cadherin. But there were some cases of moderate and severe dysplasia that had similar levels of E-cadherin expression as in mild dysplasia. Thus, E-cadherin alone cannot be used for the grading of dysplasia. E-cadherin alone cannot also differentiate between invasive lesions and severe dysplasia that is not invasive, since there were some cases of severe dysplasia and well-differentiated SCC that had similar levels of expression.

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