Original Article

Assessment of Immunohistochemical Expression to the TP63 Protein in Premalignant and Malignant Disorders of Oral Squamous Cell Carcinoma among Sample of Iraqi Patients

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Abstract

Background: The oral squamous cell carcinoma (OSCC) is the sixth most prevalent cancer recorded globally and is the leading cause of death. With an annual incidence of approximately 550,000 cases, early detection of genetic alterations in potentially malignant disorders (PMDs) can aid in the identification of lesions that may develop into cancer. TP63 is a protein known as tumor protein TP63. It is linked to the development of epithelial tumors and plays a crucial part in controlling the cell cycle. **Objective:** The aim of this study was to compare TP63 marker expression of the OSCC with oral premalignant tissues which is oral epithelial dysplasia (OED). **Materials and Methods:** TP63 was stained by immunohistochemical technique (IHC) on tissue sections from 35 (58.3%) of OED and 25 (41.7%) of patients with OSCC. For each lesion, the labeling index (LI), the staining intensity of the reaction, and the patterns of the TP63 reaction were evaluated. **Results:** Epithelial dysplasia with enhanced suprabasal expression and squamous cell carcinoma with diffused and peripheral patterns of response were both associated with overexpression of TP63. Labeling index (LI) of TP63 in patients with OSCC had the highest mean (61.04 ± 23.608) from epithelial dysplasia (54.77 ± 15.377) with significant differences ($P < 0.05$), and grades of OSCC showed statistically significant variations in the LI and intensity of reaction of TP63. **Conclusions:** As a result, TP63 can be a helpful signal of dysplastic alteration and a biomarker for the development of oral cancer.

Keywords: Dysplasia, IHC, malignant, oral squamous cell carcinoma, premalignant disorders, TP63.

Introduction

Oral squamous cell carcinoma (OSCC) is the most prevalent type of oral cancer. There has been a thorough analysis of the genetic and molecular changes, as well as the etiopathogenesis, of OSCC. Alcohol use and tobacco use are the two main risk factors for oral cancer. Tobacco smoking alone is a strong and independent risk factor for the cancers of the oral cavity and it has been estimated that the proportion of cancers of the oral cavity attributable to tobacco smoking is between 43% and 60%.[1,2] The quantity and length period of cigarette smoking are directly related to the risk of mouth cancer. Compared to cigarette smokers, those who smoke pipes and cigars are more likely to get oral cavity cancer. Due to their structural closeness, the *TP63* gene and p73 together make up the p53 gene family.[3,4] Two more p53 family members, which were TP63 and p73 discovered almost twenty years after the p53 gene was first reported based on some structural similarities. There is growing evidence that the active pathways in (SCCs) from many organs are similar.[4,5] Amplification of the chromosome segment between 3q26 and 3q28, which contains TP63, is one of the most frequent genomic aberrations shared by SCC of the lung, head and neck, esophagus, cervix, and bladder.[6] Nuclear TP63 expression is primarily located in

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the basal proliferative compartment of normal stratified squamous epithelium, with a decrease in expression in the more superficial layers.[7]

Overall, the TP63 isoforms TATP63 and NTP63 (and their target genes) show unique roles in preserving cellular metabolic homeostasis. Whereas NTP63 regulates glycolytic effector proteins and links glycolysis to oxidative phosphorylation, TATP63 governs glucose and lipid metabolism.[8] In addition to controlling intrinsic cellular features, NTP63 controls signaling pathways that affect the extracellular microenvironment. By doing so, NTP63 may alter the extracellular microenvironment in a way that promotes tumor migration and metastasis. A significant component of the extracellular matrix known as hyaluronic acid was expressed in HNSCC cell lines as a result of NTP63.[9]

To determine if TP63 immunohistochemistry expression correlates with study groups, we sought to examine the immunohistochemical staining pattern, intensity, and LI of the TP63 tumor protein in various histological grades of oral squamous cell carcinoma and dysplasia lesions. Hence, our TP63 expression analysis suggested that these gene products may be helpful for a more accurate diagnosis of OSCC and oral dysplasia.

Materials and Methods

Of 60 archival FFPE sections, 25 were OSCC cases and 35 were epithelial dysplasia cases. After reviewing the Hematoxylin and Eosin (H and E) stained sections, these sections were chosen from the Ghazy Al-Hariri Hospital for surgical specialties and the Department of Histopathology of Teaching Laboratories, both of which are part of the Baghdad Medical City. Sections of the paraffin-embedded blocks from the chosen cases were cut at a thickness of 4 μm, and they were then stained on positive charge slides with the IHC marker TP63. Positive control material was chosen to be normal prostate tissue that expressed TP63 positive signals.

Immunohistochemical assay

The manufacturer's product (#E-IR-R213A from Elabscience Biotechnology) was immunohistochemically stained for the expression of TP63 in accordance with the manufacturer's instructions. In a nutshell, tissue sections that were cut at a thickness of four μm were mounted on glass slides that had been treated with poly-l-lysine. Epitope retrieval solution (Leica 10x, pH = 9) was heated once over a 20–30 min period to boiling temperature.[10] A two-step, broad-spectrum immunohistochemical detection method called the two-step Plus can directly boost an antibody's signal of binding to an antigen. This strategy successfully avoids the space-steric barrier caused by excess polymer molecules while preserving the antibody's ability to precisely bind to antigens. The primary antibody is a mouse-derived monoclonal antibody from Elabscience Biotechnology Inc. with the designation PA6082. The unique polymer auxiliary agent for macromolecular detection technique enables better primary antibody binding detection. To mitigate the effects of variable water alkalinity and acidity on the DAB chromogenic agent, this kit contains a diluent for the concentrated DAB solution.[10]

After mounting the slides, counterstaining with Harri's hematoxylin for two minutes, and performing positive and negative staining controls for each batch of the research sample, the slides were stained. Healthy human prostate tissues that had been proven to exhibit an immunological response to TP63 in prostatic epithelial basal cells served as positive controls. By omitting the primary antibody stage, a negative control will be provided.[10]

Evaluation of the IHC signals

The existence of TP63 signals, the proportion of scoring positive staining cells (LI; labeling index), and the pattern (localization of staining) of TP63 expression were all evaluated in the results. Positive control (prostrate epithelium) and negative control immunohistochemical staining were taken into consideration. To avoid interobserver bias, the grading was carried out independently by two observers. After mounting the slides, counterstaining with Harri's hematoxylin for two minutes, and performing positive and negative staining controls for each batch of the research sample, the slides were stained. Healthy human prostate tissues that had been proven to exhibit an immunological response to TP63 in prostatic epithelial basal cells served as positive controls. By omitting the primary antibody stage, a negative control could be carried out.[11]

Counting criteria

Staining sections under a HUMAN light microscope were examined and assessed. Positive cases were those in which DAB consistently appeared brown in the epithelial cells' nuclei under low power. Negative sections were those in which epithelial cell immunostaining was completely absent.[11] The positive of individual cells was assessed at a 10x magnification. Positive cells were those that had consistent brown nuclear staining, whereas negative cells only showed hematoxylin staining.[11]

At a 40x and 10x magnification, a qualitative (semi-quantitative) evaluation of the degree of TP63 positivity in the basal/parabasal, suprabasal, and superficial layers of different stages of oral epithelial dysplasia was carried out. The pattern of expression in OSCC was divided into three categories based on the staining intensity of tumor islands: diffuse, central, and peripheral. The staining intensity of the positive results was then assessed. According to a study by Castle *et al.*, the staining intensity in cases of staining heterogeneity was classified as (+) light staining,

(++) moderate staining, and (+++) intense staining. Each observation was carried out by two more observers to eliminate interobserver bias.[11,12] Quantitative labeling index evaluation (LI) positively and negatively stained cells were counted. Three non-overlapping fields were selected using an Olympus microscope, and photomicrographs were taken at magnifications of 40x and 10x. The results were expressed as a percentage of positive cells on average. Negative specimens comprised 10% or less positive cells, as defined below: approximately 10% of tumor nuclei are negative, 10%–25% are weakly positive, 26%–75% are moderately positive, and 76%–100% are strongly positive. Only nuclear reactivity was found beneficial.[12,13]

Statistical analysis

To ascertain whether there was a correlation of TP63 protein expression with study groups, data were evaluated using means, standard deviations, and tables of frequencies. Using SPSS statistical software, version 25, and a Chi-square test was run with a significance level of P = 0.05.[14]

Ethics statement

The protocol of the study was approved by the ethical committee of the Institutional Review Board (IRB) in the Al-Nahrain University College of Medicine (at November 11, 2021.) under code number 59.

Results

The chi-square test was used for statistical analysis, and a *P* value of 0.05 or less was considered significant. The study groups were split into two, one contained epithelial dysplasia (Group I) and the other with OSCC (Group II). TP63 expression was observed in all cases except six, four from Group I and two from Group II [Table 1]. TP63 expression was found in the suprabasal layer of dysplasia, as well as in the basal and superficial layers as indicated in Figure 1.

The mean LI of TP63 ranged from 54.77 ± 15.377 to 61.04 ± 23.608, with a minimum value of 26 seen in dysplasia and a maximum value of 90 seen in OSCC [Table 2].

Table 1: Qualitative assessment of TP63 expression

Study groups	No expression	Expression	Total	$\chi 2$ (*P*)
Dysplasia (Group I)	4 (6.7%)	31 (51.7%)	35 (58.3%)	0.02
OSCC (Group II)	2 (3.3%)	23 (38.3%)	25 (41.7%)	
Total	6 (10%)	54 (90%)	60 (100%)	

χ^2 = chi-square test (degree of freedom = 1); $P < 0.05$ significant

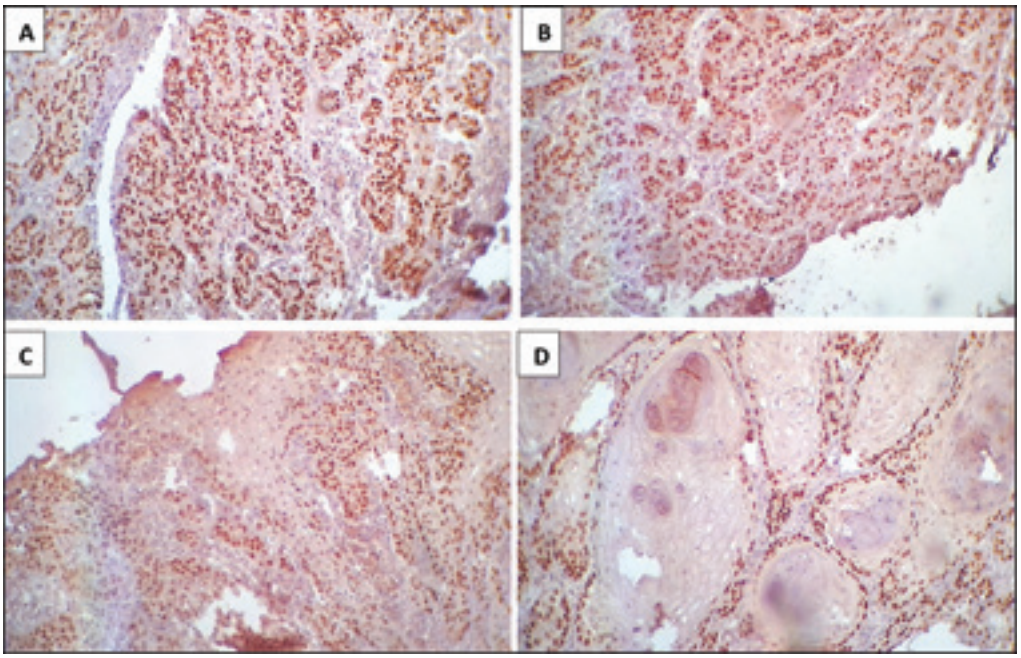


Figure 1: IHC Distribution of TP63 protein in different oral tissues. (A) TP63 expression in WDSCC diffuse pattern of reaction, high score, intense staining intensity (40x). (B) TP63 expression in MDSCC diffuse pattern of reaction high score, intense staining intensity (40x). (C) TP63 expression in dysplastic epithelium in basal and suprabasal layers, moderate intensity and score (40×). (D) TP63 expression in invasive OSCC, central and diffuse patterns or reaction around tumor mass, light intensity, and moderate score (40×)

Table 2: Mean L1 in different groups (positive cases)

Study Groups	Numbers	Mean ± Std. deviation	Minimum	Maximum
Dysplasia (Group I)	31 (51.7%)	54.77 ± 15.377	26	93
OSCC (Group II)	23 (38.3%)	61.04 ± 23.608	20	90
Total	54 (90%)	57.44 ± 19.364	20	93

Table 3: Clinico-pathological parameters and positive TP36 expression of study groups

Characteristics		N (%)	Positive TP63 expression	*P* Value
Study groups	Dysplasia (group I)	35 (58.3%)	31 (51.7%)	0.02 < 0.05
	OSCC (Group II)	25 (41.7%)	23 (38.3%)	
Age (years)**	(24 – 45)	22 (36.7%)	19 (31.7%)	0.2 > 0.05
	(46 – 65)	28 (46.7%)	27 (45%)	
	(≥ 66)	10 (16.7%)	8 (13.3%)	
Genders**	Males	40 (66.7%)	35 (58.3%)	0.2 > 0.05
	Females	20 (33.3%)	19 (31.7%)	
Tumor grades*	WDSCC	12 (20%)	12 (20%)	0.05 ≤ 0.05
	MDSCC	8 (13.3%)	8 (13.3%)	
	PDSCC	5(8.3%)	3 (5.0%)	
TNM stage*	Stage I	2 (3.3%)	2 (3.3%)	0.6 > 0.05
	Stage II	11 (18.3%)	9 (15%)	
	Stage III	10 (16.7%)	10 (16.7%)	
	Stage IV	2 (3.3%)	2 (3.3%)	
Metastasis*	Metastatic	7 (11.7%)	7 (11.7%)	0.6 > 0.05
	Non-metastatic	18 (30.0%)	16 (26.7%)	

* for (OSCC) only

** for (OSCC) and (Dysplasia)

Well-differentiated (WDSCC)

Moderately-differentiated (MDSCC)

Poorly-differentiated (PDSCC)

Table 4: Cross-tabulation for percentage and intensity staining of TP63-positive cells in study groups

Groups	Total	TP63 expression score N (%)			Staining intensity N (%)			Negative
		Weak	Moderate	Strong	(+)	(++)	(+++)	
Dysplasia	35(58.3%)	5(8.3%)	22(36.7%)	4 (6.7%)	5(8.3%)	18(30%)	8 (13.3%)	4 (6.7%)
OSCC	25(41.7%)	4(6.7%)	7 (11.7%)	12(20%)	6 (10%)	10(16.7%)	7 (11.7%)	2 (3.3%)
Total	60 (100%)	9 (15%)	29(48.3%)	16(26.7%)	11(18.3%)	28 (46.7%)	15 (25%)	6 (10%)
P Value			0.01			0.6		

The current study included 25 (41.7%) of OSCC patients, with 23 (38.3%) having positive TP63 expression. Forty males (66.7%) having 35 (58.3%) positive TP63 expression versus twenty females (33.3%) in a ratio of 19 (31.7%). The age range of OSCC patients ranged from 24 to 82 years, with a mean age of 49.0 ± 13.5 years. WDSCC 12 (20%) was found in nearly 95% of OSCC patients, with notable heterogeneity between grades. Clinical TNM stage II was the most common in 11 (18.3%) of OSCC patients, followed by stage III 10 (16.7%), and only seven cases (11.7%) get metastasize as indicated in Table 3.

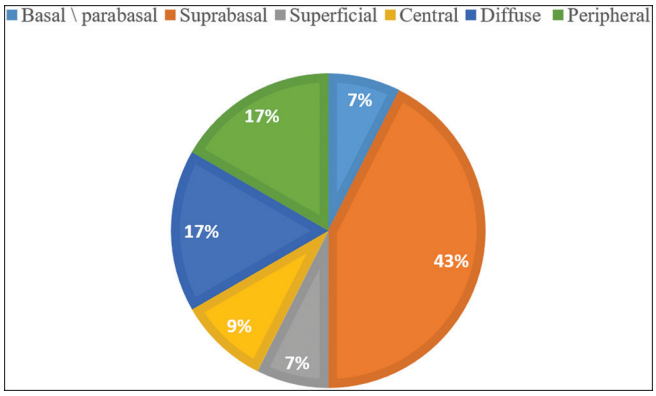
In most cases of dysplasia, TP63 positivity was in the moderate score 22(36.7%) and moderate intensity 18 (30%), but in OSCC, most positive TP63 cells were in the strong score 12 (20%) and moderate intensity of staining 10(16.7%) [Figure 1]. TP63 expression score was shown to be statistically significant between dysplasia and OSCC at $P < 0.05$ [Table 4].

According to Table 5, TP63 positive signals were in the mild dysplasia as (6%), moderate dysplasia (12%), and severe dysplasia (13%), while in OSCC grades, the WDSCC as (12%), MDSCC (8%), and PDSCC (3%). The majority of severe dysplasia sections (16.7%) had moderate positive TP63 expression and moderate intensity (11.7%). Interestingly, the majority of WDSCC sections had (10%) of high score positive TP63 expression and (13.3%) with moderate intensity of reactivity as seen in Figure 1.

Graph 1 shows that the majority of TP63 signals were classified as suprabasal in oral epithelial dysplasia among

Table 5: Cross-tabulation for percentage and intensity staining of TP63-positive cells in grades of study groups

Study groups		TP63 expression score N (%)			TP63 staining intensity N (%)			
		Weak	Moderate	High	(+)	(++)	(+++)	Negative
Dysplasia	Mild	1 (1.7%)	4 (6.7%)	1 (1.7%)	3 (5.0%)	1 (1.7%)	2 (3.3%)	1 (1.7%)
	Moderate	2 (3.3%)	8 (13.3%)	2 (3.3%)	1 (1.7%)	10(16.7%)	1 (1.7%)	1 (1.7%)
	Severe	2 (3.3%)	10 (16.7%)	1 (1.7%)	1 (1.7%)	7 (11.7%)	5(8.3%)	2 (3.3%)
P Value		0.2			0.2			
OSCC	WDSCC	4 (6.7%)	2 (3.3%)	6 (10%)	1 (1.7%)	8 (13.3%)	3 (5.0%)	0 (0.0%)
	MDSCC	0 (0.0%)	5(8.3%)	3 (5.0%)	5(8.3%)	1 (1.7%)	2 (3.3%)	0 (0.0%)
	PDSCC	0 (0.0%)	0 (0.0%)	3 (5.0%)	0 (0.0%)	1 (1.7%)	2 (3.3%)	2 (3.3%)
P Value			0.002				0.01	



Graph 1: Distribution of sample according to the patterns of TP63 expression between study groups

various stages of the basal/parabasal, suprabasal, and superficial layers. The TP63 pattern of expression in OSCC was classified as peripheral, central, or diffuse and the majority of cases were classified as peripheral and diffuse, with high significant differences ($P < 0.0001$) as shown in Figure 1.

Discussion

Oral cancer is the sixth most common type of cancer worldwide and oral squamous cell carcinoma (OSCC) accounts for more than 90% of all head and neck cancers which are headed by clinically evident premalignant lesions. The most common premalignant lesion is leukoplakia, which can have variable degrees of epithelial dysplasia ranging from mild to severe. The severity of dysplasia increases the likelihood of acquiring invasive cancer. The prevalence of carcinomatous changes in oral leukoplakia ranges from 6.6% to 36%.[3]

The TP63 protein has been proven to perform two functions. Firstly; the production of TP63 protein in the proliferative layer of cells near the basement membrane of the normal oral mucosa, where it likely prevents basal cells from growing and so helps to maintain their basal cell state. However, when the oral dysplastic mucosa undergoes dysplastic change (i.e., transition from normal to epithelial dysplasia), dysplastic keratinocytes above the basal layer can shift to an embryogenesis-like state and continue to express TP63 protein, resulting in an anti-differentiation effect as well as a proliferative capacity of dysplastic cells in the oral dysplastic mucosa. Secondly; TP63 can contribute to the development of epithelial dysplasia by changing stem cell activity in the basal layer, resulting in an increase in the number of stem cells. This can lead to the altered distribution of oral epithelial dysplasia in the basal and suprabasal layers.[15]

The TP63 is a p53 homolog with a distinct involvement in epithelial physiology. TATP63, like p53, has tumor-suppressive properties,[16] but NTP63 isoform overexpression is common and serves as a differentiating hallmark of carcinogenesis. Several studies have been undertaken to study the role of TP63 in oral dysplasia and cancer, with mixed results. In normal and non-dysplastic mucosa, nuclear expression of TP63 in basal and parabasal keratinocytes of the epithelium is widely recognized, with NTP63 being the major functional isoform. The percentage of TP63-positive cells increases with dysplasia severity and expands into higher epithelial layers. ADditionally, TP63 overexpression, particularly NTP63, is prevalent in HNSCC tumors and cell lines generated from HNSCC. ADditionally, oral dysplasia associated with greater NTP63 expression and inflammatory cell infiltration is linked to an increased risk of cancer development and a worse prognosis.[17]

The TP63 monoclonal antibody for OSCC and dysplasia was used in this investigation to elucidate the involvement of this essential cell cycle regulator in this neoplasia. In our investigation, 31 (51.7%) dysplasia cases demonstrated TP63 positive expression, with mild dysplasia (10.0%), moderate dysplasia (20.0%), and severe dysplasia (21.7%), and moderate intensity of TP63 staining 10 (16.7%). This is consistent with the findings of a research by de Oliveira *et al.*[18] All positive cases of mild dysplasia had moderate expression.

The TP63 expression increased in tandem with dysplasia severity, according to Bortoluzzi *et al.*,[12] discovered, TP63 expression increased in line with the severity of dysplasia. Twenty-five (41.7%) cases of OSCC showed positive TP63

expression, with the majority of them having positive TP63 expression in 23 (38.3%) cases. In this study, 12 (20%) of WDSCC cases showed TP63 expression ranging from weak to strong. The majority of PDSCC cases had moderate to strong cyclin TP63 expression. This was a statistically significant result ($P < 0.05$).

Several local studies conducted recently in Iraq have investigated oral and other Squamous Cell Carcinoma using immunohistochemical markers for the prediction and detection of such an important type of cancer. These studies may reflect the wide prevalence of this cancer in Iraq.[19-21]

Conclusion

This study suggests that TP63 is implicated in carcinogenesis and is linked to changes ranging from early dysplasia to poorly differentiated carcinoma and that these changes are linked to enhanced expression patterns in dysplasia and OSCC. High TP63 expression in mild and severe dysplasia indicates that dysplasia severity increases with increased TP63 expression and may indicate a high probability of malignant transformation. TP63 expression was shown to be considerably higher in both well- and poorly differentiated SCC. These gene products' IHC demonstration may be a useful tool in detecting dysplasia and OSCC prognosis. Such studies with a bigger sample size in diverse parts of the oral cavity and evaluating all phases of SCC will shed additional light on the pathogenesis of oral cancer and the involvement of TP63.

Conflict of interest

There are no conflicts of interest.

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Nil.

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