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Immunoexpression of P53 in Correlation with Histological Grading of Oral Squamous Cell Carcinoma

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Abstract

Oral squamous cell carcinoma is the 6th most common carcinoma according to Union for International Carcinoma Control.[1,2] In India its prevalence is high due to personal habit and environmental influence. It is divided into mild, moderate and poor according to severity. Overexpression of mutant P53 protein, located on chromosome 17 is considered as a poor prognostic marker for SCC.[3,4] We collected the samples of oral carcinoma that came to our department and carried out an analysis using p53 Immunohistochemical stain alongwith Hematoxyline and Eosin stain . A total of 52 cases were analyzed. Total positivity was 88% in the cases studied. Amongst these, 81% well differentiated carcinoma, 92% moderately differentiated carcinoma and all poorly differentiated carcinoma showed positive result. The intensity of p53 increased as the grading increased from well differentiated to poorly differentiated showing strong relationship between p53 overexpression and tumour aggressiveness.

Keywords: Squamous cell carcinoma, Immunohistochemical, Hematoxyline and Eosin

Introduction

Oral squamous cell carcinoma is a multifactorial and multistep process. It is a leading cause of mortality and morbidity with worldwide incidence of 550,000 cases and 300,000 deaths each year.[5] More than 90% of oral carcinoma are squamous cell carcinoma.[6] In India, its incidence is 30 per 100,000 population. Male to female ratio ranges from 2:1 to 4:1.[1,2] Most oral squamous cell carcinoma arise in the epithelial lining of buccal mucosa, lip, floor of mouth, tongue, gingiva, oropharynx. However in recent years, several alternation in the expression of tumour suppressor gene and oncogene in the development of head and neck squamous cell carcinoma have been described.[7] TP53 is one of those tumour suppressor gene located on chromosome 17p13, which in turn encodes several isoform of p53 protein. Mutation within the p53 causes overexpression and accumulation of mutated p53 protein. The mutant protein is not easily digestible, hence it accumulates inside the carcinoma cell leading to immunohistochemical overexpression, which is associated with different neoplastic and paraneoplastic lesion.

Material and Method

Clinical details of the patient including age, sex, sign and symptom, duration, any previous history of carcinoma and all relevant documents was retrieved. Informed consent was taken. Specimen was subsequently fixed in 10% formalin, processed in a tissue processor, paraffin embedded and cut at 4-5 u thickness at rotatory microtome and studied with H&E stain for histopathological assessment of tumour and

grading is done according to Border's Classification into Mild, Moderate and Poorly differentiated SCC.[8] Immunohistochemical stain was done using p53 antibody. For p53 protein expression, nuclear positivity was assessed quantitatively. The percentage of positively stained cell in the whole layer of epithelium was determined by scanning the entire section and was recorded by assigning cases to one of the following categories[9,10] :

(a) 0 = no epithelial cell staining

(b) 1+ = upto 25 % cells showing positive staining

(c) 2+= upto 50% cells showing positive staining

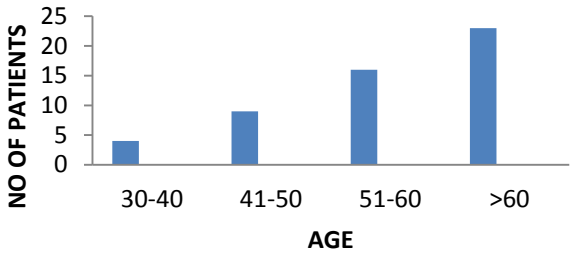
(d) 3+ = more than 50 % cells showing positive staining

Result and Observation

The data analysis is illustrated in the form of table, histogram, pie diagram, bar diagram.

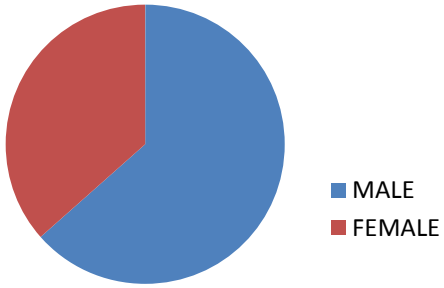
(A) Distribution of age in oral SCC

Amongst the 52 patients, 4 patients were in the age group 30-40 years, 9 patients were in the age group 41-50 years, 16 patients were in the age group 51-60 years and 23 patients were in the age group more than 60 years.



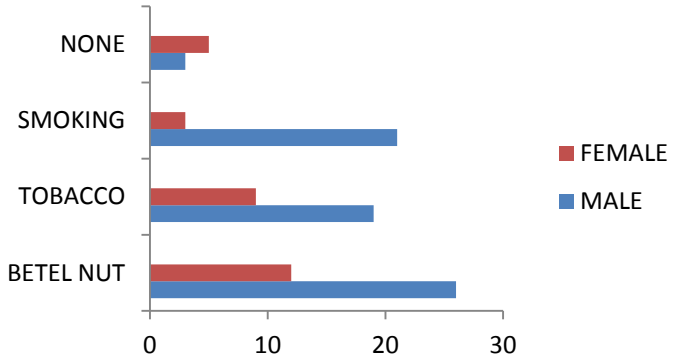
(B) Distribution of sex in oral SCC

Out of 52 patients, 33(63.46%) were male and 19(36.54%) were female.



(C) Distribution of patients in relation to their habit

Out of 33 male, 28(78.79%) took betel nut, 19(57.58%) took tobacco, 21(63.64%) were smoker and 3(9.1%) did not take anything. Similarly, out of 19 female, 12(63.16%) took betel nut, 9(47.37%) took tobacco, 4(21.05%) were smoker and 5(26.32%) did not take anything.



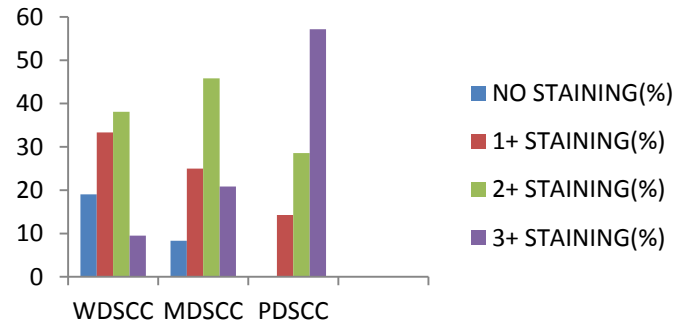
(D) Type of oral SCC

Type of SCC	No of Patients
Well differentiated	21
Moderately differentiated	24
Poorly differentiated	7

(E) Intensity of p53 in different grades of oral SCC

Out of 21 WDSCC, 4(19.05%) cases show no staining with p53, 7(33.33%) cases show 1+ staining pattern, 8(38.1%) cases show 2+ staining pattern and 2(9.5%) cases show 3+ staining pattern. Out of 24 MDSCC, 2(8.33%) cases did not stain with p53 and 6(25%) cases, 11(45.83%) cases and 5(20.83%) cases showed 1+, 2+ and

3+ staining pattern gradually. Out of all the positive staining PDSCC cases, 1(14.29%) cases, 2(28.57%) cases and 4(57.14%) cases showed 1+, 2+, 3+ staining pattern gradually.



Discussion

From our study it has been found that as the grading increases from well differentiated to poorly differentiated, p53 nuclear staining intensity also increases. This indicates that p53 is a poor prognostic marker and associated with poor survival rate.

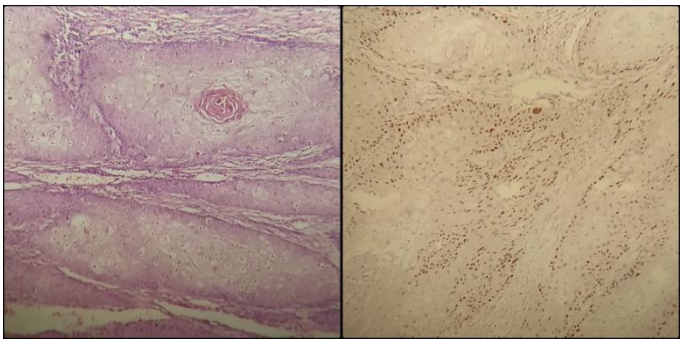


Fig 1: Well differentiated Squamous cell carcinoma: H&E stain (left), P53 IHC (right).

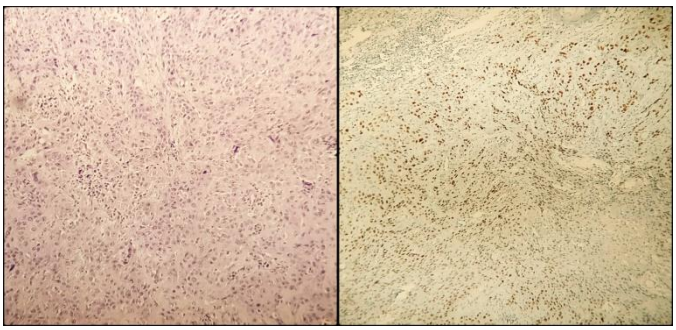


Fig 2: Moderately differentiated Squamous cell carcinoma: H&E stain (left), P53 IHC (right)

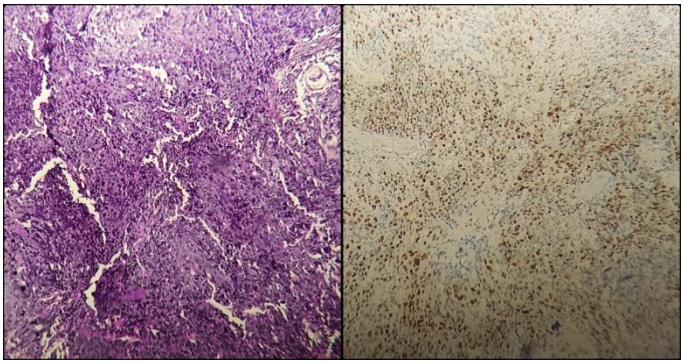


Fig 3: Poorly differentiated Squamous cell carcinoma: H&E stain (left), P53 IHC (right)

Previous literature also showed association of p53 expression with tumour grade and histological features. Dave et al. in a study of 40 cases of HNSCC found a significant association of p53 expression with tumour grade and other histological parameters like degree of kerartinization.[11]

Ayaz Manzoor Dar et al. also carried out a study and also found a significant relationship between increased p53 expression and poor histological grading.[7]

Shin et al. also showed that expression of p53 protein in primary head and neck squamous cell carcinoma was significantly predictive of shorter survival because of its association with earlier development of both tumour recurrence and second primary tumour.[12]

However the complete absence of p53 positivity in some squamous cell carcinoma was explained by Nylander et al. as the tumour either comprise of wild tumour protein or have change in the function in TP53 gene resulting in production of a truncated, non-functional and non-detectable protein.[13]

Conclusion

It was prominent from the study that there is an association of P53 immunoexpression with histological grading of tumour with increased immunoreactivity from mild to poorly differentiated SCC, Hence p53 is considered as a bad prognostic factor of oral squamous

cell carcinoma. So P53 can be used in future as targeted therapy to control oral SCC.

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