

EFFECT OF ORAL HABITS OF PATIENTS ON THE OVEREXPRESSION OF EGFR PROTEIN IN ORAL SQUAMOUS CELL CARCINOMAS (OSCCS)

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Over activation of a growth promoting proto-oncogene such as epidermal growth factor receptor (EGFR) plays a major role in the development of epithelial malignancies. It has been shown that alteration in the gene due to long term exposure to various carcinogenic agents is the reason for this. Betel chewing, smoking and consumption of alcohol are recognized risk factors for development of OSCCs. Both epidemiological and laboratory studies have revealed that the long-term exposure to such ingredients which carry different carcinogenic agents results in malignant transformation. The aim of the present study was to observe the relationship between the frequency of different habits in patients with OSCCs and immuno-expression of EGFR protein in their carcinomas.

The sample consisted of 60 primary OSCC patients (males). Information on oral habits was recorded. Patients were stratified according to the severity of smoking, frequency of betel chewing and amount of alcohol consumption (mild = 1, moderate = 2, heavy = 3, habit free = 0). Over-expression of EGFR was assessed by immunohistochemistry using monoclonal antibody EGFR 113 (dil,1:20, Novocastra). The percentage of positive cells, and the intensity (+, ++, +++) of expression were recorded. Analysis was done using chi square test.

In the sample 86.6 % of the patients were betel chewers. Out of sixty, 62 % were smokers and 60 % consumed alcohol. EGFR positivity was 56 %. Over 50 % of the cancer cells were EGFR positive in 10/34 positive tumours. No significant difference was found in the over-expression of EGFR in OSCCs, either between smokers and non-smokers or between betel chewers and non-betel chewers. Intensity of EGFR expression in OSCCs was not significant both between smokers and non-smokers or chewers and non-chewers. There was a significant difference in the percentage of EGFR positive cells between heavy consumers of alcohol and mild to moderate consumers of alcohol ($\chi^2 = 19.4$, $p = 0.004$). Intensity of EGFR protein expression also significantly differed between heavy consumers of alcohol and mild to moderate consumers of alcohol ($\chi^2 = 12.5$, $p = 0.05$).

No association was found between smoking, betel chewing and EGFR immunoreactivity in the present sample. However, exposure to alcohol showed a significant increase in the protein expression. Further studies are needed to confirm the over-expression of EGFR in oral squamous cell carcinomas induced by alcohol.