

Comparative evaluation of complement factor I in dysplastic and normal oral mucosa using immunohistochemistry

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Abstract

Background and Aim: The importance of oral dysplasia is in this regard that a percentage of these lesions can progress to cancer. According to the research on cutaneous squamous cell carcinoma, complement factor I may play a role in progression of squamous cell carcinoma. The aim of present study was to evaluate immunohistochemical expression of complement factor I between normal and dysplastic oral mucosa.

Materials and Methods: In this case-control study, studied group was consisted of paraffin-embedded tissue blocks of 30 dysplastic oral mucosa samples and 30 normal oral mucosa samples (as control group). 4 μ sections were prepared from tissue blocks and stained with complement factor I antibody at pathology laboratory using immunohistochemistry technique. In this study, percentage of stained cells and staining intensity of them was considered and data analyzed by Mann-U-Whitney test. Significance level was set as P-value of lower than 0.05.

Result: 25 cases of normal mucosal samples and 20 cases of dysplastic mucosal samples didn't stain with this antibody. Mean percentage of stained cells in normal mucosa was 1.5 ± 4.1 and in dysplastic mucosa was 4.16 ± 6.7 . ($p < 0.1$) There was no statistically significant difference between normal and dysplastic oral mucosa with regard to mean percentage of stained cells. Semi-quantitative classification of percentage of stained cells also didn't show any statistically significant difference between normal and dysplastic oral mucosa. Also, with regard to staining intensity, no statistically significant difference was observed between normal and dysplastic oral mucosa.

Conclusion: According to immunohistochemical expression of complement factor I didn't have significant difference between normal and dysplastic oral mucosa, it seems that this factor don't play a role in initiation of carcinogenesis.

Keywords: dysplasia, oral mucosa, complement factor I