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Editorial

Oral Field Cancerization

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The oral cavity is one of the most vulnerable sites for the development of potential malignancies, owing to its direct contact with various carcinogens. Squamous cell carcinoma is one of the most common malignancies developed in the oral cavity with an average survival rate of about 5 years [1].

Field cancerization was first described by Slaughter., et al. in 1953. It implies that oral cancer does not arise as an isolated cellular phenomenon, but rather as an anaplastic tendency involving many cells at once that results into a multifocal development process of cancer at various rates within the entire field in response to a carcinogen, such as in particular tobacco [2]. The oral cavity is considered to be the most susceptible to this process, because of its exposure to a wide range of environmental carcinogens which affect the entire mucosa and result into the simultaneous occurrence of premalignant states [1,2]. Because of this various molecular analyses have been carried out to investigate the genetic mutations and clonality for the validation of this carcinogenesis model. Field cancerization involves the formation of multiple patches of premalignant disease with a higher-than-expected rate of multiple local secondary primary tumors. In oral cavity, tobacco and alcohol produces a synergistic effect as primary carcinogens in the development of squamous cell carcinomas. The environmental carcinogens have a potential to reach a large area and to damage a large proportion of cells contributing to premalignant states within the entire exposed surface [3].

The process of carcinogenesis initiates from multiple genetic and epigenetic alterations in the mucosa which can lead to the clonal expansion of premalignant daughter cells in a particular field. The genetically altered stem cells produce a clonal unit composed of daughter cells from which the patch extends into the adjoining areas in successive steps following further modifications. This activates continuous cellular transformations that eventually lead to the replacement of the normal epithelium by a proliferating field. Nevertheless, there is a population of cells with early genetic changes, which does not exhibit any histological changes, therefore describing the concept of field cancerization [1,3,4].

The presence of a field with genetically altered cells is a risk factor for cancer. The large amount of preneoplastic cells in the proliferating fields is probably increasing the risk of cancer dramatically. The likelihood of developing a second primary tumor in a patient who once had head and neck squamous cell carcinomas is 20%. Other researches in this field have a strong prospective of exhibiting new diagnostic markers for early detection, modalities to prevent progression, and lastly ways to combat development of second primary tumor [1].

Early identification and management of field change is a vital determinant for prevention of cancer mortality and morbidity. Various tumor markers have been identified to help determine the field effect, the entire process is still controversial and therefore further investigations are still under progress in order to provide a better understanding of carcinogenesis and to use the biomarkers foreseen in this concept for cancer prevention purposes.

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