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Mini Review

The Controversy of Chronic Trauma and Oral Cancer

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Abstract

The presence of chronic trauma, for example an ill-fitted denture, as a risk factor for oral squamous cell carcinomas (OSCC) has been a controversy for a long time. Molecular and genetic studies shaded some light on the basis of oral carcinogenesis. At the same time, other factors such as inflammation has been proved to have mutagenic effect influencing the development of malignant tumors. Therefore, mechanical force of chronic trauma on the oral mucosa alone may not be carcinogenic. However, at some point, it may contribute in this phenomenon.

Keywords: *Oral Squamous Cell Carcinomas (OSCC); Chronic Trauma; Oral Cancer*

Introduction

It is well established that the major risk factors of head and neck squamous cell carcinomas (SCC) involve smoking and alcohol consumption and infection with HPV in non-smokers [1]. However, the presence of chronic trauma as a risk factor has been a controversy. Some studies showed no excess risk of cancers of the gum and buccal sulcus, but chronic physical irritation of the tongue's squamous epithelium by an ill-fitted denture is a risk factor for the development of oral cancer [2,3]. Others did not show association between disease status and use of dentures [4] or presence of broken teeth [2].

In oral squamous cell carcinomas (OSCC) the oral basal cells may acquire genetic alterations, including a mutation in TP53, resulting in a patch containing p53-mutated clonal units representing the first oncogenic changes in the mucosa that associated to additional genetic modifications originate the oral cancer [1,5]. For example, the activation of the oncogene Kras may result in benign lesion such as hyperplasia and/or papilloma. However, when Kras activation is associated with p53 mutation can result in OSCC [1]. Therefore, the hallmarks of cancer namely sustaining proliferative signaling, evading growth suppressors, resisting cell death, enabling replicative immortality, inducing angiogenesis, and activating invasion and metastasis [6] are the result of different type and number of oncogenic mutations [1].

In addition, it is also known that oral dysplasia presents loss of heterozygosity (LOH) at chromosomes 3p, 9p and 17p as a reflect of early changes in carcinogenesis, while carcinomas show alterations at chromosome arms 11q, 4q and 8p, a relatively late phase of carcinogenesis [5,7].

In this way, Bernardes *et al.* (2019) investigated the LOH in inflammatory fibrous hyperplasias a frequent lesion associated with the use of ill-fitting dentures and observed that LOH was present in 13% of the samples similarly to other reactive and inflammatory lesions. Therefore, the hypothesis that trauma associated with a dental prosthesis does not seem to have an important role in oral carcinogenesis [8].

However, inflammation is an enabling characteristic for its contributions to the acquisition of core hallmark capabilities through release of chemicals causing actively mutagenic effect for nearby cancer cells accelerating their genetic evolution for carcinogenesis [6].

It has been reported that the chronic physical irritation of oral mucosa by dentures contributes to the topical carcinogenic effect of tobacco in the mouth induced by absorption of dissolved tobacco-derived carcinogens [9]. Moreover, poor oral hygiene and presence of OSCC is associated with the presence of *Candida albicans*, a dimorphic fungus capable to produce nitrosamines, carcinogens that could act alone or in combination with other chemical compounds [2,10], activating specific proto-oncogenes and triggering the development of a cancer. Others have reported that *Candida* species could promote carcinogenesis by producing acetaldehyde from ethanol or even favor metastasis progression through inflammation [10]. However, there is no conclusive knowledge concerning the pathogenic role of this opportunistic infection on cancer development [10].

Conclusion

Taking on account the genetic/molecular mechanisms involved in the process of carcinogenesis it can be concluded that mechanical force of chronic trauma on the oral mucosa solely may not be carcinogenic. However, it may contribute with the process inducing establishment of inflammation, absorption of tobacco-derived carcinogens, and presence of *Candida albicans*, for example, that result in the activation of oncogenic pathways. Meanwhile, more studies are necessary to conclusively elucidate this controversy, and careful clinical examination and excellent dental practice are the best option for the patient for prevention and early diagnosis of oral cancer.

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