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Editorial

Oral Submucous Fibrosis: The Aetiology and Pathogenesis

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Oral submucous fibrosis (OSF) is a condition seen predominantly in people of Asian origin from India, Bangladesh, Sri Lanka, Pakistan, Taiwan, parts of Southern China and Polynesia [1]. It is a chronic progressive disorder that is potentially malignant and its clinical presentation rests on the stage of the disease at detection and diagnosis. Most patients usually present with rigidity of lip, tongue and palate leading to varying degrees of limitation of opening of the mouth and tongue movement in addition to intolerance to spicy food [2]. The distinguishing feature of OSF is that it affects most parts of the oral cavity, pharynx and upper third of the oesophagus. Extensive research has provided over whelming evidence that areca nut is the main aetiological factor for OSF [3,4]. There is a definite dose-dependent relationship between areca nut and causation of the disease [1]. The severity and the time taken for the progression of the disease also seems to vary according to the preparation of areca nut consumed. Commercially prepared products such as pan masala, guthka and mawa have higher concentrates of areca nut per use and cause OSF more rapidly than by self-prepared conventional betel quid which contain less amounts of areca nut [5].

Fibrosis and hyalinization of sub-epithelial tissues account for the majority of clinical features observed in OSF [6]. Areca alkaloids causing fibroblast proliferation and increased collagen synthesis hydrolysis of arecoline in to arecaidine is obligatory before fibroblast stimulation can take place [7], suggesting that arecaidine is the active metabolite in fibroblast stimulation. The copper content of areca nut is high and the levels of soluble copper in saliva may rise in volunteers who chew areca quid [8]. The enzyme lysyl oxidase, which is a copper dependent enzyme [9], is found to be upregulated in OSF [10] and plays a key role in collagen synthesis and its cross linkage. It is known that OSF is associated with inflammatory changes in at least some stages of the disease and prostaglandin is one of the main inflammatory mediators and its production is controlled by various enzymes such as cyclooxygenase (COX) [1].

Polymorphisms of the genes coding for TNF-α has been described as an important risk factor for OSF. Autoimmunity as an etiological factor for OSF has been examined due to slight female predilection and occurrence in the middle age [11,12] and the presence of circulating immune complexes, their immunoglobulin contents and the recognition of various auto antibodies in patients era. The precancerous nature of OSF was first described by Paymaster in 1956 when it was observed that slow growing squamous cell carcinoma (SCC) was seen in one third of the patients with the disease [13]. This was further confirmed by various groups and Pindborg in early 1970s put forward five criteria to prove that the disease is precancerous [14]. These criteria include, high occurrence of OSF in oral cancer patients, higher incidence of SCC in patients with OSF, histological diagnosis of cancer without any clinical suspicion in OSF, high frequency of epithelial dysplasia and higher prevalence of leukoplakia among OSF cases [15].

In conclusion, literature indicates that the main aetiological factors for OSF are the constituents of arecanut, mainly arecoline. OSF is on the rise due to the increase in use of betel nut and other similar products and this condition is expected to be observed more frequently with people from countries where its use is common immigrating to western countries. The individual mechanisms playing a role at various stages of the disease need in-depth study in order to propose appropriate therapeutic interventions. However, the most important requirement is to limit or regulate the free access and production of hazardous products available in the market which lead to OSF.

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