Acta Scientific Dental Sciences (ISSN: 2581-4893)

Volume 5 Issue 9 September 2021

Review Article

OSMF: Hearing Forfeiture by Fibrosis

Sreepradha C[1]*, Vaishali MR[2], Maria Priscilla David[3] and Roopashri G[2]

[1]Assistant Professor, Department of Oral and Medicine and Radiology, GDCRI, Bellary, India

[2]Reader, Department of Oral and Medicine and Radiology, M R Ambedkar Dental College and Hospital, Bangalore, India

[3]Professor and HOD, Department of Oral and Medicine and Radiology, M R Ambedkar Dental College and Hospital, Bangalore, India

***Corresponding Author:** Sreepradha C, Assistant Professor, Department of Oral and Medicine and Radiology, GDCRI, Bellary, India.

Received: August 13, 2021

Published: August 28, 2021

Abstract

Oral squamous cell carcinoma has been ranked at the sixth position amongst the malignant neoplasms, with primarily the incidence of a precursor/pre-cancer such as oral leukoplakia, oral erythroplakia oral submucous fibrosis. The aetiology of OSMF is multifactorial and embraces local factors as well as systemic factors seen to affect retromolar region, buccal mucosa, followed by palatal fauces and uvula, soft palate, tongue later trailed by the labial mucosa. Clinical features include tongue stiffness, blanched mucosa, fibrosis, depigmented gingiva, rubbery soft palate with decreased mobility and blanched and atrophic tonsils, and shrunken bud like uvula with impairment of activities such as eating as well as other functional activities. Impaired hearing assessment is a part of the diagnostic protocol and is the most ignored part of diagnosis. Tympanometry and audiometry form a part of the hearing diagnostic tools and should be made a part of the same.

Keywords: Oral Squamous Cell Carcinoma; Oral Submucous Fibrosis; Tympanometry; Inflammation; Disease; Audiometry

Introduction

Oral squamous cell carcinoma has been ranked at the sixth position amongst the malignant neoplasms, accounting to about 300,000 cases worldwide. The five year survival rate for oral squamous cell carcinoma has endured at a circa of 50% over many decades [1].

OSCC is not an acute disease process and forms by a long cycle which encompasses genetic, epigenetic as well as metabolic modifications which would be due to a consequence of exposure to carcinogens, with primarily the incidence of a precursor/pre-cancer such as oral leukoplakia, oral erythroplakia oral submucous fibrosis. The most commonly testified etiological agents being tobacco, alcohol, betel quid comprising of areca nut [2].

Globally, about 2.5 million individuals have been accounted to have OSMF, with the tag of being the leading potentially malignant condition of South Asia with a high rate of prevalence in India [3-5]. It accounts for about 5% in women and 2% in men [6]. Majority of the patients diagnosed with OSF are between the age of 20 - 40years and shows a malignant transformation rate of about 7 to 13% [7].

Oral submucous fibrosis (OSMF) is a chronic inflammatory disease of the oral soft tissues with progressive juxta-epithelial fibrosis resulting in increasing difficulty in chewing, swallowing, speaking and mouth opening, often associated with burning sensation inside oral cavity that is aggravated on exposure to spicy food [8].

Aetiopathogenesis

The aetiology of OSMF is multifactorial and embraces local factors like areca nut, chilli as well as systemic factors like nutritional deficiency, genetic predisposition and autoimmunity, but areca nut chewing is the main causative agent [9].

The habit of chewing betel nut (*Areca catechu*) is well-thought-out to be the key etiological agent, with others factors being genetic predisposition, infections and viral agents, carcinogens, nutritional and immunologic factors.[8] The role of chilli in the pathogenesis of OSMF still is debatable and is projected to be an allergic response to capsaicin owing to allergen induced eosinophilia [5]. The combination of areca nut as well as tobacco has steered to a sharp upsurge in the occurrence of OSMF [4].

Areca nut comprises of alkaloids particularly arecoline and guavacoline, with a wide range of parasympatheticomimetic effects, which modulate matrix metalloproteinases, lysyl oxidases and collagenases, which in turn affect the collagen metabolism leading to fibrosis [10]. Synchronously, a drop in the water-retaining proteoglycan level occurs, which errands a surge in type I collagen production and flavonoids (Catechin and Tannin) in the betel nut stabilizes the collagen fibers and makes them resistant to degradation by collagenase [5].

Clinical features

The most commonly affected sites are the retromolar region as well as the buccal mucosa, followed by soft palate, palatal fauces, uvula, tongue and labial mucosa [5]. Most common age being affected is at about 40 years with a female preponderance with a age range of 12 - 62 years [10].

Inability/restricted mouth opening complemented with burning sensation are the chief compliant (s) of the patient. Scrutiny shows blanched oral mucosa with marble-like appearance, attributed to inflammation, trailed by hypovascularity and fibrosis that may be associated with small vesicles and mucosal erosions [8].

Disease progression may show fibrosis, blanched oral mucosa, tongue stiffness, blanched, leathery floor of the mouth, fibrotic, depigmented gingiva, rubbery soft palate with diminished mobility and atrophic tonsils, and shrunken uvula bud with weakening of functional activities such as eating, whistling, blowing, sucking.[5] Other symptoms are increased salivation, change of gustatory sensation, hearing loss due to stenosis of the Eustachian tubes, dryness of the mouth, nasal tonality to the voice and dysphagia to solids. Pindborg has classified OSMF into 3 stages depending on the clinical features seen.

As time progresses increased fibrosis leads to loss of resilience, which in turn interferes with speech, tongue mobility as well as decreased ability to open the mouth. Till date, many studies of OSMF have demonstrated progressive degeneration of the underlying musculature, causing impairment of function. Palatal envelopment has been observed in more than 50% of patients with fibrosis apparent in the faucial pillars [11].

Association of the palatal and paratubal muscles (levator veli palatini, tensor veli palatini, tensor tympani and salpingopharyngeus) together, which regulate the patency and function of the pharyngeal orifice, results in impairment of eustachian tube function and patency, leading to pain in the ear along with loss of hearing [11-13].

Diagnosis and investigations

Clinical diagnosis of the disease process is sufficient; however an incisional/punch biopsy would provide us with a complete confirmatory diagnosis. The histopathology report would show increased fibrosis as well as reduced blood supply in the region.

Additional investigations consist of haematological, serological, immunological and biochemical factors which may reveal findings such as a raised ESR, slight eosinophilia, microcytosis and hyperchromic indicative of anaemia.

Advanced assessment by the use of tympanometry as well as audiometry for the impact on hearing loss is indicated for a better understanding. Biopsy specimens from soft palate reported degenerative changes in palatal and paratubal muscles in the form of atrophy, loss of cross striations and oedema of myoepithelium [14].

Tympanometry is a process to gauge the role of the middle ear. It provides a detailed illustration of the air pressure in the external ear canal to impedance of the ear drum and middle ear system. It is a hand held probe that is introduced into the ear which entails of three tubes with a loudspeaker, a microphone and a pump. A tone is conveyed through the loudspeaker as the pressure changes within the sealed canal, with the microphone measuring the amount of sound that is reflected back during the pressure sweep (sound wave coming from the eardrum), displayed in grid form – the tympanogram [14].

Diverse type of tympanogram are:

Audiometry

Pure-tone audiometry, a behavioural test measures hearing sensitivity. Audiometry is an electronic device which produces pure tones. Pure tone is delivered to the ear through headphone for air conduction and by bone vibrator for bone conduction. Hearing level in decibels above the normal threshold is plotted. The frequency, ranges between 250 to 800Hz. The pure tone average is the average of the hearing threshold levels at 500, 1,000, 2,000 Hz only [14].

Scale of hearing impairment (Modified from Goodman, 1965):

- Grade 1: 10 - 15 dB - Normal hearing
- Grade 2: 16 - 25 dB - Minimal hearing loss
- Grade 3: 26 - 40 dB - Mild hearing loss
- Grade 4: 41 - 55 dB - Moderate hearing loss
- Grade 5: 56 - 70 dB - Moderate to severe hearing loss
- Grade 6: 71 - 90 dB - Severe hearing loss
- Grade 7: > 90 dB - Profound deafness.

Qualitative Hearing was classified into normal hearing, conductive, sensorineural and mixed hearing loss [14].

Abnormal or impaired Eustachian tube functions (i.e. impaired opening or closing) may cause pathological changes in the middle ear. This in turn can lead to hearing disabilities [15]. Involvement of the palatal and paratubal muscles (levatorveli palatine, tensor veli palatini, tensor tympani and salpingopharyngeus), which regulate the patency and function of the pharyngeal orifice, results in impairment of eustachian tube function and patency [12], leading to pain in the ear along with loss of hearing.

Differential diagnosis

The differential diagnosis includes anaemia and scleroderma which can be distinguished by other cutaneous, systemic and characteristic radiographic and laboratory findings [5].

Management

Management of OSMF includes use of hyaluronidase and corticosteroids or a combination of both. Other treatment modalities include antioxidants, Immunomodulators, Physiotherapy, Interferon-γ, Hyper Baric Oxygen (HBO) therapy, Curcumin, Oxitard, Aloe vera, Surgery [5,8,16].

Therefore, the protocol for OSF management should include ENT consultation and hearing impairment evaluation. It is suggested that further studies be conducted with larger sample size and advanced technology for diagnosis and management of hearing impairment.

Conclusion

Even though it is easy to diagnose but the irreversible condition reflects the failure of the present treatment modalities. The lack of knowledge and the delay in seeking treatment leads to the progression of the disease. Hence, more focus should be emphasized in detecting newer treatment modalities which is the need of the hour and the future.

Therefore, as clinicians it would be required to examine all cases of OSF for hearing impairment so as to enable referral to the concerned specialist for further evaluation of hearing loss and increase the likelihood of the treatment in order to achieve favourable prognosis.

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