ORAL SUB-MUCOUS FIBROSIS: A REVIEW

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ABSTRACT
Oral submucous fibrosis (OSMF) is a chronic disease affecting any part of the oral cavity. Epithelial atrophy, juxtaepithelial inflammation and fibrosis of the lamina propria are common findings. It is most common in the countries of south-east Asia and shows greater predisposition towards the Indian ethnic group. In this review we discuss various components of OSMF, including the classification, aetiology, clinical presentation, pathogenesis, and a brief overview of its management.

KEYWORDS: Oral Submucous Fibrosis (OSMF), medical management, surgical management

INTRODUCTION
Oral submucous fibrosis (OSMF) is a persistent, progressive, pre-cancerous condition of the oral mucosa, which is related with betel quid chewing habit extensively widespread in Southeast Asia and in China. OSMF was defined as an “insidious, chronic disease affecting any part of the oral cavity and sometimes the pharynx and esophagus, characterized by a mucosal rigidity of varying intensity due to the fibro-elastic changes of the juxta-epithelial layer, resulting in a progressive inability to open the mouth.”[1]

Numerous factors such as chili consumption, nutritional deficiency states, areca nut chewing, hereditary susceptibility, autoimmunity and collagen disorders have been suggested to be concerned in the pathogenesis of this condition. It is in general accepted at present that areca nut quid plays a principal role in the etiology of OSMF.[2] The copper content of areca nut is elevated, and the levels of soluble copper in saliva may go up in areca quid chewers. The enzyme lysyl oxidase is found to be unregulated in OSMF. This is a copper reliant enzyme, which plays a key role in surplus collagen synthesis and its cross-linkage leading to OSMF.[3]

OSMF is mainly seen in India, Bangladesh, Sri Lanka, Pakistan, Taiwan, and China, with a reported frequency ranging up to 0.4% in Indian rural population.[4] In recent years marked increase in the rate of OSMF is observed in many parts of India like Bihar, Maharashtra, Gujarat and Madhya Pradesh. The younger generations are suffering more due to the advent of more and more commercially freeze-dried areca-nut products in different multicolored eye-catching pouches like gutkha and pan masala.[5]

History
OSMF has been well established in Indian medical literature since the time of Sushruta—a renowned Indian physician who lived in the era 600 B.C and was termed as ‘Vidari’. It was first described in the modern literature by Schwartz in 1952 who coined the term atrophia idiopathica mucosa oris to describe an oral fibrosing disease, he discovered in 5 Indian women from Kenya.[5] Joshi subsequently coined the term oral submucous fibrosis (OSMF) for the condition in 1953.[6]

This condition has been referred to under a number of names, diffuse oral submucous fibrosis[7], idiopathic scleroderma of the mouth[8], idiopathic palatal fibrosis.[9]

Prevalence
Global estimates from 1996 indicate that about 2.5 million people have OSMF.[10] However, results from studies conducted in 2002[11] indicate that more than 5 million people in India have OSF (0.5 percent of the Indian population). In addition, it is estimated that up to 20 percent of the world’s population consumes betel nut in some form,[12] so the prevalence of OSMF probably is higher than that noted in the published literature.

The rate varies from 0.2-2.3% in males and 1.2-4.57% in females in Indian communities.[13] Oral submucous fibrosis is widely prevalent in all age groups and across all socioeconomic strata in India. The occurrence of this condition in children is extremely rare. Youngest case reported in the literature was a 4-year-old girl.[18] A case of OSMF in a 12 year old girl was reported in 1993 and the etiology was traced to be the habit of chewing roasted areca nuts.[19] Another case of a 11 year old girl...
Etiopathogenesis

Although various factors have been implicated in the development of oral submucous fibrosis, the exact role of any one of these in the development, severity and extent of the disease is not clear, as the disease may still occur if none of these is present.

When the disease was first described in 1952, it was classified as an idiopathic disorder.[5]

Earlier workers correlated it with hypersensitivity to capsaicin (Capsicum annuum and Capsicum frutescens--an active ingredient in chilies -- secondary to chronic iron and/or vitamin B complex deficiencies; or exposure to cashew kernel oil.[18] Ramanathan summarized the evidence of OSMF being a mucosal change secondary to chronic iron and/or Vitamin B Complex deficiency. He suggested that the disease is an Asian analogue of sideropenic dysphagia.[19]

Currently, the habit of chewing areca nuts (the fruit of Areca catechu plant) is recognized as the most important etiologic agent in the pathogenesis of this condition. A number of epidemiological surveys, case-series reports, large sized cross sectional surveys, case-control studies, cohort and intervention studies provide over whelming evidence that areca nut is the main aetiological factor for OSMF.[17, 20-22] Four alkaloids have been conclusively identified in biochemical studies, arecoline, arecaidine, guvacine, guvacoline, of which arecoline is the main agent. The alkaloid component of the betel nut stimulates the inflammatory process.[20] An initial epithelial inflammation is followed by fibro-elastic changes in the lamina propria.[20, 23] Epithelial atrophy and collagen deposition result in the formation of dense fibrotic bands. Overactivity during chewing causes ischaemic changes. Subsequent fibrosis and scarring in the masticatory muscles contribute further to fibrotic band formation and trismus. These bands are visible in the palate, buccal and labial areas and, in later stages, in the pharyngeal and oesophageal areas. In vitro studies on human fibroblasts using areca extracts or chemically purified arecoline support the theory of fibroblastic proliferation and increased collagen formation that is also demonstrable histologically in human OSMF tissues.[24] The role of areca alkaloids, copper in fibroblast proliferation and increased collagen synthesis, stabilization of collagen structure by tannins and fibrogenic cytokines, genetic polymorphisms predisposing to OSMF, role of the collagen related genes Col1A2, COL3A1, Col6A1, COL6A3 and COL7A1 have been discussed by W.M. Tilakaratne et al.[25]

A possible autoimmune basis to the disease with demonstration of various auto-antibodies and an association with specific HLA antigens A10, DR3, DR7, and probably B7, along with haplophytic pairs A10/DR3, B8/DR3, and A10/88, has been found.[26] These pairs, together with the presence of autoantibodies and chronic inflammation of the oral mucosa, have been suggested as an autoimmune basis of oral submucous fibrosis.

Clinical features

The most frequently affected locations in oral submucous fibrosis are the buccal mucosa and the retromolar areas. It also commonly involves the soft palate, palatal fauces, uvula, tongue, and labial mucosa. It is generally believed that oral submucous fibrosis originates from the posterior part of the oral cavity and subsequently involves the anterior locations.[27] A study on the regional variations of this condition pointed out that such an observation would depend on whether the areca nut juice and the quid are swallowed or spat out.[28]

It manifests as a burning sensation in the mouth, intolerance to eating hot and spicy foods, Blanching and stiffness of the oral mucosa, trismus, vesiculation, excessive salivation, ulceration, pigmentation change, recurrent stomatitis, defective gustatory sensation, dryness of the mouth, gradual stiffening and reduced mobility of the soft palate and tongue leading to difficulty in swallowing and hyper nasality of voice, hoarseness of voice (with laryngeal involvement) and occasionally, mild hearing loss due to blockage of Eustachian tube.[29]

The precancerous nature of oral submucous fibrosis has been observed with development of slowly growing squamous cell carcinoma in one-third of oral submucous fibrosis patients.[30] In southern India, 40% of oral cancer patients had oral submucous fibrosis.[31] A 7.6% incidence of oral cancer in oral submucous fibrosis patients has been reported in a median 10-year follow-up period.[11] Pindborg et al. summarized the criteria in support of the precancerous nature of the disease as higher prevalence of leukoplakia among oral submucous fibrosis patients, high frequency of epithelial dysplasia, concurrent finding of oral submucous fibrosis in oral cancer patients, and histologic diagnosis of carcinoma without the clinical suspicion of it.[32]

The malignant transformation rate for OSMF is 7 to 30 percent.[12,33]

The characteristic histologic features of OSMF consist of, atrophic epithelium often keratinized, generally without rete ridges, and in advanced cases it may be...
ribbon-like with juxtaepithelial hyalinization and collagen of varying density.[31]

**Staging**

Pindborg et al described 4 consecutive stages of oral submucous fibrosis based on histologic findings: very early stage, early stage, moderately advanced stage, and advanced stage.[34]

Khanna and Andrade in 1995 developed a classification system for the surgical management of trismus.[35]

- **Group I:** Very early stage without mouth opening limitations with an interincisal distance of greater than 35 mm.
- **Group II:** Early stage with an interincisal distance of 26-35 mm.
- **Group III:** Moderately advanced cases with an interincisal distance of 15-25 mm. Fibrotic bands are visible at the soft palate, and pterygomandibular raphe and anterior pillars of fauces.
- **Group IVA:** Advanced stage: Trismus is severe, with an interincisal distance of less than 15 mm and extensive fibrosis of the oral mucosa.
- **Group IVB:** Disease is most advanced, with premalignant and malignant changes throughout the mucosa.

Divya Mehrotra et al suggested a clinical grading of the disease and treatment methods as[36]

- **Grade I:** stomatitis and burning sensation in the buccal mucosa with no detection of fibres. Suggested treatment for this group is abstinence from habit and medicinal management.
- **Grade II:** symptoms of grade I, palpable fibrous bands, involvement of soft palate, and maximum mouth opening 26-35 mm. Suggested treatment: abstinence from habit and medicinal management.
- **Grade III:** symptoms of grade II, blanched oral mucosa, involvement of tongue, and maximal mouth opening 6-25 mm. Suggested treatment: abstinence from habit and surgical management.
- **Grade IV:** symptoms of grade III, fibrosis of lips, and mouth opening ≤5 mm. Suggested treatment: abstinence from habit and surgical management.

S. M. Haider et al gave the following staging system[36]

**Clinical and functional staging**

- **Clinical stage**
  1. Faucial bands only
  2. Faucial and buccal bands
  3. Faucial, buccal, and labial bands

- **Functional stage**
  A Mouth opening ≥ 20 mm
  B Mouth opening 11–19 mm
  C Mouth opening ≤10 mm

**Management**

In current years many researchers have elicited and worked upon, the existing etiology/pathophysiology concerned to OSMF. Until date, no efficient treatment is accessible for this progressively disabling condition with elevated malignant potential. Management of OSMF thus postulates major challenge for an oral physician/oral surgeon.

Various types of treatment modalities include conservative methods, medical management, and invasive methods like surgical elimination of the fibrotic bands and combined therapy.

**Conservative treatment**

Conservative treatment can be categorized into restriction of habits/behavioral therapy, nutritional or supportive therapy and oral physiotherapy.

**Restriction of habits/behavioral therapy**

The consumption of chilies, pan, betel nut, spices and commercially available, guthka, pan masala, is ever-increasing in India. Accordingly people should be encouraged to stop these habits so as to prevent OSMF. Affected patients should be explained about the disease and possible malignant potential of OSMF. Possible irritants should be removed. Those patients with this incurable, persistent fibro-elastic scarring disease necessitate being fully motivated. Motivation is essential at least in the early stages, as it could possibly slow the progress of the disease[38]

An intervention study has shown a decline in the risk for OSMF as a consequence of education against chewing habits. There was a drop in the incidence of OSMF from 21.3/100,000 persons among men in the control cohort to 8.3 in the intervention cohort, and from 45.7 to 29.0 among women in the control cohort. This reduction in the percentage highlighted the decreased risk with the decline in areca nut habit.[39] Avon et al.[40] also reported improvement in mucosal lesion as well as clinical symptoms subsequent to the cessation of the areca nut chewing habit.

**Nutritional or supportive therapy**

Micronutrients and minerals such as vitamin A, B, C, D and E, iron, copper, calcium, zinc, magnesium, and selenium can efficiently diminish the oxidant levels. A low ingestion of fruits and vegetables is linked with an increased risk for pre-cancers and cancers.[41]

Ingestion of red tomatoes, green leafy vegetables and fresh fruits should be included in the regular diet since these deliver protection against the increased risk of cancer by rising levels of antioxidants.[37,38] Lycopene is a carotenoid present in tomatoes has been revealed to have a number of effective antioxidant and anticarcinogenic properties and has established intense benefits in precancerous lesions for instance leukoplakia. Lycopene has been shown to reduce hepatic fibrogenesis and may exert a similar inhibition on abnormal fibroblasts in OSMF.[42]
Intake of green tea should be incorporated in the diet chart. Polyphenols in green tea have considerable free radical scavenging activity and can protect cells from DNA damage caused by reactive oxygen species. It can also hinder tumor cell proliferation and induce apoptosis. Tea catechins have been shown to reduce angiogenesis and tumor cell invasiveness. Furthermore, green teas have been shown to activate detoxification enzyme, such as glutathione S-transferase which may help defend next to tumor development. Thus, many of the potential beneficial special effects of tea have been ascribed to the strong antioxidant activity of tea polyphenols.\(^{[43]}\)

Various studies have concerned the deficiency of iron both as a cause and consequence in etiopathogenesis of OSMF. Thus, routine assessment of hemoglobin levels followed by iron supplements should be incorporated in the treatment plan.\(^{[37,38]}\)

Immune milk is a variety of skimmed milk formed from cows immunized with several human intestinal bacteria. It has high-quality anti-inflammatory effect and contains a reasonable amount of vitamins such as A, B1, B2, B6, B12, C, pantothenic acid, nicotinic acid, folic acid, iron, copper and zinc. Chemically it is alike to commercial milk however it contains 20-30% elevated concentration of IgG Type 1 antibody. Tai et al. showed that oral supervision of milk from cows immunized with human intestinal bacteria lead to a substantial improvement in signs and symptoms of patients with OSMF. Presence of IgG antibody in immunized milk might restrain the inflammatory reaction and modulate cytokine assemblage in OSMF patients which is foremost to significant improvement.\(^{[44]}\)

**Oral physiotherapy**

Physiotherapy can transform tissue remodeling during promotion of physical movements within physiological limits producing noteworthy results. Physiotherapy in OSMF can be in the form of physical exercise regimen and splints or other devices. Muscle stretching exercises intended for the mouth may be supportive to avoid further limitation of mouth movements. This includes forceful mouth opening with the assist of sticks, hot water gargling and ballooning of mouth. This is considered to put pressure on fibrous bands. Forceful mouth opening have been tried with mouth gag and acrylic surgical screw.\(^{[1,38]}\)

Cox and Zoellner advocated 5 times every day for physiotherapy by inter-positioning tongue spatulas stuck between teeth and addition of a new spatula every 5-10 days for 4 months. It was observed that oral opening was enhanced in OSMF patients.\(^{[45]}\)

**Microwave diathermy**

Heat has been adequately used in the form of hot rinses, lukewarm water or selective deep heating therapies like shortwave and microwave diathermy. Heat therapy acts by fibrinolysis of bands. Short-wave produces sharp localized deep heat, avoids the unintentional heating of superficial facial tissues like skin and adipose tissue. Microwave diathermy selectively heats only juxtaepithelial connective tissue and limiting the area to be treated. Thus, it is easy to apply with minimum discomfort.\(^{[38,41]}\)

Gupta et al. advocated diathermy every day for 20 min at each site of lesion by means of 20-25 watts of energy to create comfortable warmth. Such 15 sittings were given to each patient and found helpful for the moderately advanced stage of OSMF.\(^{[46]}\)

**Medical treatment**

Treatment includes intralesional injection of steroids, placentrex and fibrinolytic agents. Medical treatment is symptomatic and intended at improving movements. The medical management has been summarized in the following table given by Auluck et al.\(^{[65]}\)

<table>
<thead>
<tr>
<th>Treatment modality for OSF (Auluck et al., 2008).</th>
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<tbody>
<tr>
<td>Treatment</td>
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<tr>
<td>Micronutrients and minerals</td>
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<tr>
<td>Milk from immunized cows</td>
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<tr>
<td>Lycopene</td>
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<td>Pentoxyfilline</td>
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<td>Interferon gamma</td>
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<td>Steroids</td>
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<td>Placental extracts</td>
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<td>Turmeric</td>
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<td>Chymotrypsin, hyaluronidase and dexamethasone</td>
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Steroids
Steroids are well recognized to act as immunosuppressive agents causing inhibition of inflammation found in OSMF lesions, thus reducing this fibro-collagenous condition. In addition, steroids can slow down the proliferation of fibroblasts and thus reducing the number of collagen fibers. Steroids can also liberate cellular proteases in the connective tissue, which in turn can activate the collagenase and zymogen that consume insoluble collagen.\[^{[1,4,47]}\]

Submucosal intralesional injections weekly or topical application of steroids in patients with moderate OSMF may help to avoid additional damage. Steroid ointment applied topically may be helpful in ulcers and painful oral mucosa. A local injection of hydrocortisone 1.5 cc is found to be efficient.\[^{[44]}\]

Hyaluronidase
*In vitro* studies, hyaluronidase attacks quickly on collagen from OSMF patients than on normal collagen. Hyaluronidase degrades the hyaluronic acid matrix, lowers the thickness of intracellular cemental substances as well as activating definite plasmatic mechanisms. As a result, reprieve of trismus may be predictable through softening and diminishing of fibrous tissue.\[^{[9]}\]

The use of topical hyaluronidase has been revealed to improve symptoms more quickly than steroids only. Hyaluronidase can also be supplementary to intralesional steroid preparations. The mixture of steroids and hyaluronidase showed improved long-term results. Kakar *et al.* found that injection of 1500 IU of hyaluronidase and dexamethasone (4 mg) locally for 7 weeks gave superior results if it was followed by 3 weeks of hyaluronidase injections.\[^{[48]}\]

Collagenase
An endogenous collagenase action in normal oral mucosa exhibits 3 to 5 fold higher levels than that of OSMF tissues. Reduced content of functional collagenase observed in OSMF patients is one of the mechanisms accountable for collagen accumulation. Lin and Lin found that intra-lesional collagenase injections not only results in a noteworthy improvement of mouth opening, but also experience a striking decline in hypersensitivity to spices, cold, and heat which helps to re-establish eating function.\[^{[1,49]}\]

Placental extracts
The injection placentrex is an aqueous extort of human placenta containing nucleotides, enzymes, aminoacids, steroids and vitamins. It acts by “biogenic stimulation”. Its use is mainly due to the method of “tissue therapy” introduced by Filatov in 1933 and later in 1953. His theory states, “animal and vegetable tissues, when detached from the parent body and exposed to conditions unfavorable, but not mortal to their existence, undergo biological readjustment leading to the development of substances in condition of their survival to ensure their vitality. Such tissues or their extracts, implanted or injected into the body after conflict to pathogenic factors, stimulate the metabolic or regenerative processes, thereby favoring recovery.\[^{[38]}\]

The aqueous extract of placenta acts as follows
1. Hasten cellular metabolism
2. Aids in the assimilation of exudates
3. Stimulates regenerative development
4. Increases physiological purpose of organs
5. Produces noteworthy enhancement of wound healing
6. Has an anti-inflammatory consequence.\[^{[38]}\]
7. Sur and Bis was showed that it is a necessary biogenic stimulator. It stimulates pituitary adrenal cortex and regulates metabolism of tissue. It also increases vascularity of tissue.\[^{[50]}\]

Katharia *et al.* carried out a study on 22 OSMF patients and injection of 2 ml placental extract (Inj. placentrex) was given locally in the preset areas, once a week up to a total duration of one month. The results showed that the severity of the disease was reduced.\[^{[54]}\]

Chymotrypsin
Chymotrypsin is an endopeptidase enzyme that can execute proteolysis. Chymotrypsin preferentially cleaves peptide amide bonds where the carboxyl side of the amide bond is a tyrosine, tryptophan, or phenylalanine of collagen fibers in OSMF. Chymotrypsin, thus acts as a proteolytic agent in the treatment of OSMF. Gupta and Sharma injected Chymotrypsin (5000 IU), dexamethasone (4 mg) and hyaluronidase (1500 IU) twice weekly for 10 weeks sub-mucosally in OSMF patients and observed good results.\[^{[57,52]}\]

Interferon (IFN)-gamma
IFN-gamma plays a significant role in the treatment of OSMF for the reason that it has immuno-regulatory effect. Haque *et al.* studied that IFN-gamma is a known anti-fibrotic cytokine, effect of which was considered on collagen synthesis by arecoline stimulated OSMF fibroblast. Results of this study showed that there was inhibition of collagen synthesis in existence of IFN-gamma. This clinical trial of IFN-gamma intra-lesional injections gave major progress in mouth opening.\[^{[53]}\]

Aloe vera
*A. vera*, popularly known as “babosa,” is a plant usually found the Northeast of Brazil. Its foliage, extract and resin present antimicrobial, anti-inflammatory and healing properties and are indicated to hepatic and stomach diseases.\[^{[54]}\]

Sudarshan *et al.* has carried out a preliminary study to contrast the efficacy of *A. vera* with antioxidants in the treatment for OSMF. Results of this study showed that *A. vera* response is enhanced in all the parameters evaluated and responded in all the clinic-histopathological stages chiefly in patients with mild-stage clinically and early-stage histopathologically. *A. vera* as well showed decline
in burning sensation, improvement in mouth opening and cheek flexibility. It was concluded that A. vera group reduces the burning sensation and recovers mouth opening thus enhances the patients’ fulfillment.\(^{55}\)

**Turmeric**

Curcumin (diferuloylmethane) found in turmeric, a natural yellow pigment exhibits anti-oxidant, anti-inflammatory and anti-cancer properties. Turmeric oil and turmeric oleoresin together offers defense against DNA damage. As such, it may fulfill two roles in the putative treatment of OSMF, both as an anti-inflammatory agent and as a chemopreventive agent. It also provides a base for a simple, safe, acceptable and cost effective interference for earlier stages of OSMF.\(^{37}\) Rai et al. conducted a study using curcumin in the treatment of oral precancers including 25 patients with OSMF. This study reported that OSMF was “cured by curcumin” due to increasing of local and systemic antioxidative status.\(^{56}\)

**Pentoxifylline therapy**

Pentoxifylline is a methylxanthine derivative that produces dose-related hemorrhheologic effects. It can act in these possible ways.

1. Microcirculation is progressed and platelet aggregation as well as granulocyte adhesion is reduced
2. Leukocyte deformability is amplified and as well as slows down neutrophil adhesion and activation
3. Has antithrombin, antiplasmin, and fibrinolytic activity
4. It can cause degranulation of neutrophils, increases natural killer cell activity and inhibits T-cell and B-cell activation
5. It can maintain cellular integrity and homeostasis following acute injury
6. Pentoxifylline reduces burn scar contractures
7. This drug can also reduce the symptoms in patients with OSMF, in addition to its function in improving the vascularity\(^{57}\)

Rajendren et al. used pentoxifylline as an accessory drug in OSMF treatment and after 7 months trial and 6-12 months follow-up, the patients showed progress in signs and symptoms as compared to controls.\(^{57}\)

**Surgery**

It is the technique of choice in patients with limited mouth opening and/or biopsy showing dysplastic or neoplastic changes.\(^{57}\)

The following surgical modalities have been used.

- Simple excision of the fibrous bands can be done. However, this excision can result in contracture of the tissue and exacerbation of the condition
- Split-thickness skin grafting following bilateral temporalis myotomy or coronoidectomy. Trismus associated with OSMF possibly due to changes in the temporalis tendon secondary to OSMF; therefore, skin grafts may relieve symptoms
- Nasolabial flaps and lingual pedicle flaps can be performed. Surgery to create flaps is executed only in patients with OSMF in whom the tongue is not involved\(^{58}\)
- Buccal fat pad is used as a grafting source. The buccal fat pad is a flexible and lobulated mass, simply accessible, and mobilized. Mehrotra et al. compared the surgical treatment modalities in 100 patients using buccal fat pad, tongue flap, nasolabial flap, and split thickness graft and concluded that buccal fat pad graft was superior to all the other surgical procedures, and that can be done even under local anesthesia as a day care procedure.\(^{59}\)

Lasers offer oral surgeons with a new modality for treating OSMF. The erbium chromium ytrrium scandium gallium garnet (Er:Cr:YSGG) laser has a wavelength of 2780 nm, well absorbed by water and is used on oral soft tissue without creating thermal damage. The overall advantage of laser surgery include a somewhat bloodless operative field and thus outstanding visibility, reduced need for local anesthesia, the less probability of bacterial infection, reduced mechanical tissue trauma, fewer sutures, quicker healing, reduced post-operative edema, scarring and tissue shrinkage. Chaudhary et al., highlights the attempt in treating a moderate case of bilateral OSMF with Er:Cr:YSGG laser showed a better end result during follow-up.\(^{60,61}\)

Advantages of using lasers in treating OSMF were the charred tissue obtained after excision of bands provided a protective environment for the incised raw areas until the initial healing took place, resulting in less scar-tissue formation. Shah et al. reported that the ultimate outcome of using lasers in OSMF was excellent. The average increase of mouth opening was 15.0 mm in moderately advanced cases and 17.0 mm in early cases over a follow-up period of 3 months. Less scar formation led to more flexible and elastic mucosa. Due to this there was no recurrence or tissue shrinkage after surgery, maintaining good mouth opening.\(^{61}\)

**Combined therapy**

With the combination of peripheral vasodilators (nylidrin hydrochloride), vitamin D, E and B complex, placental extract, local and systemic corticosteroids and physiotherapy claim a high success rate in OSMF management. The grouping of steroids and topical hyaluronidase shows enhanced long-term results than used alone.\(^{37,62}\) Kumar et al. reported that combined therapy employing nutritional and iron supplements with intra-lesional injection therapy using hyaluronidase, dexamethasone and placenrix in addition to local anesthetic topical gel and topical application of triamcinolone acetonide 0.1% caused a marked improvement in patients signs and symptoms. Evidenced by improvement in color of the oral mucosa, decrease in blanching and decreased severity of burning sensation,
increased mouth opening and tongue protrusion. However, in most of the cases, depending on the stage of disease and extent of oral involvement, therapy consisting of a combination of drugs is preferred in cases with minimal impairment of mouth opening and surgery might be useful in patients with marked limitation of mouth opening.

**Surgical Care**

Surgical treatment is indicated in patients with severe trismus and/or biopsy results revealing dysplastic or neoplastic changes. Surgical modalities that have been used include the following.

Simple excision of the fibrous bands, excision of bands with myotomy with or without coronoidectomy, coverage of the raw area with skin grafts, fresh amnion, collagen membrane, buccal pad of fat, local flaps or vascularised free flaps, followed by active post-operative jaw physiotherapy with anti-oxidants and proper nutrition and regular follow-ups to ensure maintenance of oral opening and early detection of malignant changes if any.

Use of lasers for band excision also has been documented.

Coverage of the area with fibrin glue or Absorbable Atelocollagen also is being tried at various institutes.

**CONCLUSION**

Conclusion OSMF is a debilitating but preventable disease and considering the malignant potential of the diseases, early diagnosis and proper management is essential in reducing the mortality of oral cancer. Dentists can play an important role in both the education of patients about the perils of chewing betel quid and in the early diagnosis of such high-risk premalignant lesions and cancer.

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