

Progression and Outcomes of Oral Submucous Fibrosis: Insights from a Case Series

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ABSTRACT

In India, tobacco consumption—particularly in the form of chewing—is deeply entrenched in daily life. It is not uncommon for at least one member in a household to engage in this habit, either occasionally or regularly. Despite widespread awareness of its harmful ingredients, tobacco continues to be used for various reasons, including recreation, stress relief, and taste preference. Among the many health consequences linked to the habitual chewing of tobacco and areca nut, Oral Submucous Fibrosis (OSMF) has emerged as a significant and increasingly encountered clinical condition. OSMF often presents not only in its classical form but also with varied and sometimes masked clinical appearances, making early diagnosis challenging.

KEYWORDS: Oral submucous fibrosis, fibrotic bands, mouth stiffness, dysphagia, reduced mouth opening, areca nut, tobacco.

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INTRODUCTION

Oral Submucous Fibrosis (OSMF) is a debilitating, progressive, and irreversible collagen metabolic disorder that arises primarily due to chronic chewing of areca nut and its commercial preparations. It is particularly prevalent in regions like India and Southeast Asia, where betel quid and tobacco consumption are culturally and socially ingrained. OSMF affects the oral mucosa and, in some cases, may extend to the pharynx and esophagus, resulting in mucosal stiffness and marked functional limitations. Notably, the condition carries a significant risk of malignant transformation, emphasizing the need for timely diagnosis and intervention.

The disease is characterized by progressive fibrosis of the oral and submucosal tissues. It typically begins with inflammatory

changes and gradually progresses to fibrosis, resulting in symptoms such as a burning sensation in the mouth, reduced tongue mobility, palpable fibrotic bands, blanching of the oral mucosa, and progressive reduction in mouth opening. Other symptoms include depapillation of the tongue, leathery texture of the mucosa, shrunken or atrophic uvula, and difficulty in swallowing (dysphagia).

The pathogenesis of OSMF involves a chronic hypersensitivity reaction, which initiates juxta-epithelial inflammation. This leads to increased fibroblast activity and reduced degradation of collagen fibers. The fibroblasts undergo phenotypic changes, producing thicker, more stable collagen bundles that resist enzymatic breakdown. As fibrosis replaces the loosely arranged connective tissue, the elasticity and mobility of oral structures diminish, resulting in the

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hallmark functional impairments of OSMF. Additionally, phagocytic activity in the tissue is minimized, further exacerbating the fibrotic process.

While the habitual use of areca nut remains the most critical etiological factor, other contributors include tobacco consumption, genetic predisposition, nutritional deficiencies (such as iron and vitamins), and certain dietary habits. The diagnosis is predominantly clinical, often confirmed by histopathological analysis of biopsy samples. Various alternative terms have been used to describe OSMF, including idiopathic scleroderma of the mouth, juxta-epithelial fibrosis, idiopathic palatal fibrosis, diffuse oral submucous fibrosis, and sclerosing stomatitis.

Given its irreversible nature and potential for malignant transformation, early detection and appropriate management of OSMF are essential to prevent disease progression and improve patient outcomes.

CASE REPORTS

Case 1

A 55-year-old male patient reported to the Department of Oral Medicine and Radiology with the chief complaint of a burning sensation in the mouth for the past 3 months. On enquiry, the patient revealed a history of gutkha chewing 7–8 times a day for the last 25 years, though he had quit the habit at present. He had no relevant medical history; however, he had visited a dental clinic 2 days prior with the same complaint. No abnormalities were detected in his general and extraoral examination. He had reduced mouth opening of approximately 23 mm and exhibited poor oral hygiene status. On intraoral examination, the patient presented with diffuse blanching extending from the retromolar pad area posteriorly to the buccal mucosa with respect to the premolars bilaterally, and from the maxillary vestibule superiorly to the mandibular vestibule inferiorly, accompanied by a loss of flexibility in the buccal mucosa.



Case 2

A 20-year-old male patient reported to the Department of Oral Medicine and Radiology with the chief complaint of restricted mouth opening for the past two years and pain in the left cheek region. On enquiry, the patient revealed a habit of gutkha chewing 6–7 times a day for the last 6 years, along with cigarette smoking 10–11 times a day over the same period. He gave no relevant medical or dental history. No abnormalities were detected in the general and extraoral examination.

On intraoral examination, a reduced mouth opening of approximately 19 mm and poor oral hygiene status were observed. On further examination, diffuse blanching was

noted along with restricted tongue movement. A burning sensation was present while eating, with a VAS score of 8 out of 10.

On palpation, fibrous vertical bands were palpable on the right and left buccal mucosa, extending from the retromolar pad posteriorly to the anterior buccal mucosa in the maxillary vestibule. The uvula was deviated to the right side, and white fibrous bands were present in the soft palate region, with loss of flexibility in the buccal mucosal muscles, suggestive of OSMF Grade 3. No lymph nodes were palpable.

Habit cessation counselling was done. The patient was prescribed medications and recalled after 15 days.



Case 3

A 29-year-old male patient reported to the Department of Oral Medicine and Radiology with the chief complaint of decayed teeth for the past two to three months and expressed interest in getting them filled. On enquiry, the patient revealed a habit of gutkha chewing 5–6 times a day for the last 3 years, along with alcohol consumption 2–3 times a month over the same period. He gave no relevant medical or dental history. No abnormalities were detected in the general and extraoral examination.

On intraoral examination, a reduced mouth opening of around 28 mm, poor oral hygiene status, decayed tooth 46, generalized gingival inflammation, and generalized bleeding on probing were observed. Further examination revealed diffuse blanching extending from the corner of the mouth anteriorly to the maxillary tuberosity region posteriorly, involving the retromolar pad area. It extended superiorly from

the buccal mucosa (15,16,26) to the mandibular vestibule inferiorly, along with restricted tongue movement.

A burning sensation was present while eating, with a VAS score of 10 out of 10. On palpation, fibrous vertical bands were palpable with an inflamed and deviated uvula to the left side, suggestive of OSMF Grade 3.

Further examination revealed ulcerations on the upper and lower lip region (13, 24, 35, 43) measuring 0.5 mm in diameter; on the left buccal mucosa, an ulcer of 0.8 mm anteroposteriorly and 0.4 mm superoinferiorly (26,27); and on the right buccal mucosa (17), an ulcer measuring 0.7 mm × 0.3 mm. All ulcers were flat, tender, non-scrapable, and suggestive of recurrent major aphthous ulcers.

Habit cessation counselling was done. The patient was prescribed medications and recalled after one week. The patient was referred to the Department of Periodontics for oral prophylaxis.



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Case 4

A 22-year-old male patient reported to the Department of Oral Medicine and Radiology with the chief complaint of dirty deposits on his teeth for the past two to three months and wanted to get them cleaned. The patient also complained of a burning sensation on the buccal mucosa. On enquiry, he revealed a habit of gutkha chewing 4–5 times a day for the past 2–3 months. The patient gave a history of sickle cell anemia. No abnormalities were detected in the general and extraoral examination.

On intraoral examination, a reduced mouth opening of approximately 30 mm, poor oral hygiene status, and

generalized gingival inflammation were observed. On further examination, blanching was observed on the right buccal mucosa, along with restricted mouth opening. A burning sensation was present with a VAS score of 3 out of 10.

On palpation, vertical fibrous bands were present on the right buccal mucosa with loss of flexibility, suggestive of OSMF Grade 1.

Habit cessation counselling was done. The patient was prescribed medications and recalled after 15 days. He was referred to the Department of Periodontics for oral prophylaxis



Case 5

A 22-year-old male patient reported to the Department of Oral Medicine and Radiology with the chief complaint of pain in the upper back left tooth region for the past 2–3 days. On enquiry, the patient revealed a habit of gutkha chewing 2–3 times a day for the past 1 year but had quit the habit 3 months ago. He gave no relevant medical or dental history. No abnormalities were detected in the general and extraoral examination.

On intraoral examination, a reduced mouth opening of 21 mm, poor oral hygiene status, difficulty in whistling, and loss of flexibility in the buccal musculature were observed.

Further examination revealed diffuse blanching in the left and right buccal mucosa, extending from the retromolar pad area posteriorly to the mandibular labial vestibule anteriorly, and from the maxillary vestibule superiorly (15,16,17,25,26,27) to the mandibular vestibule inferiorly (36,37,38,46,47).

A burning sensation was present when eating spicy food, with a VAS score of 4 out of 10. On palpation, fibrous vertical bands were palpable in the buccal mucosa, and the uvula appeared shrunken, suggestive of OSMF Grade 3.

Habit cessation counselling was done. The patient was prescribed medication and recalled after 15 days. He was referred to the Department of Oral Surgery for further treatment.



CASE DISCUSSION

Oral Submucous Fibrosis (OSMF) is a chronic, progressive, and potentially malignant condition that primarily affects the oral cavity. It is characterized by fibrosis (scarring) of the submucosal tissues, leading to progressive reduction in mouth opening (trismus), difficulty swallowing (dysphagia), and other oral dysfunctions. OSMF is closely associated with the habitual chewing of areca nut (betel nut) and tobacco, common in Southeast Asia and India, though it has been reported globally.

PATHOPHYSIOLOGY

OSMF involves a pathological increase in collagen production in the submucosal layer of the oral tissues. This abnormal collagen accumulation results in fibrosis, leading to restricted mouth opening, mucosal atrophy, and stiffness of the affected areas.[1] The condition is thought to be triggered by an inflammatory response initiated by the alkaloids in areca nut and tobacco, leading to fibroblast proliferation and excessive collagen production.[2] Other theories suggest that immune responses, particularly involving cytokines such as TGF- β (Transforming Growth Factor-beta), may play a role in the fibrosis process. A recent study has identified the role of oxidative stress and DNA damage as important contributors to OSMF's pathogenesis.[3] The prolonged exposure to areca nut and tobacco may lead to the activation of pro-inflammatory pathways, leading to tissue damage and abnormal wound healing.

ETIOLOGY AND RISK FACTORS

The primary etiological factor for OSMF is the consumption of areca nut (betel nut) and its combination with tobacco.[4] Areca nut contains alkaloids like arecoline, which is thought to cause fibrosis by stimulating fibroblasts.[5] The prevalence of OSMF is notably higher in Southeast Asia and the Indian subcontinent due to the widespread practice of betel quid chewing, which also involves tobacco.

Other risk factors include genetic predisposition, dietary deficiencies (e.g., vitamin A, iron, and B-complex vitamins), and possible viral infections, especially Epstein-Barr virus (EBV). The role of genetic susceptibility, especially polymorphisms in the genes encoding matrix metalloproteinases (MMPs), has been suggested as influencing an individual's risk of developing OSMF.[6]

CLINICAL FEATURES

OSMF presents with a range of symptoms, and the clinical features can vary depending on the stage of the disease:

1. **Trismus (Restricted mouth opening):** This is the hallmark feature, and it can progress to a point where mouth opening becomes severely limited.[7]
2. **Mucosal Atrophy:** Thinning of the mucosal tissues occurs, often with a pale or whitish appearance due to the fibrosis beneath.
3. **Burning Sensation:** Many patients experience a burning sensation, especially when consuming spicy or acidic foods.
4. **Dry Mouth (Xerostomia):** Reduced salivation is common, leading to dry mouth and increased susceptibility to dental decay.
5. **Blisters or Ulcers:** In advanced stages, areas of the mucosa may become ulcerated or develop fibrotic bands, which significantly restrict the ability to eat and speak.[8]
6. **Difficulty Swallowing (Dysphagia):** As fibrosis progresses, swallowing may become painful and difficult, especially for solid foods.
7. **Hyperpigmentation and Leucoplakia:** The condition can sometimes be associated with patches of white or pigmented lesions on the mucosa, and in some cases, these can become malignant.

In advanced stages, the condition increases the risk of oral squamous cell carcinoma (OSCC).[9]

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DIAGNOSIS

The diagnosis of OSMF is primarily clinical, based on history (habitual betel nut or tobacco use) and characteristic signs like restricted mouth opening and mucosal atrophy.[10] However, a biopsy is often necessary to confirm the diagnosis, as it provides histopathological evidence of fibrosis. Histological examination typically reveals:

- Thickened, hyalinized collagen fibers.
- A reduction in elastic fibers.
- Atrophic epithelium.
- Inflammatory infiltrate, often with an abundance of lymphocytes and plasma cells.[11]

Immunohistochemical studies may reveal markers like fibronectin and collagen as key components in the fibrotic process. Advanced diagnostic techniques, including imaging such as MRI or ultrasonography, may be used to assess the extent of fibrosis.[12]

TREATMENT

Currently, there is no definitive cure for OSMF, and the management is focused on symptomatic relief and slowing disease progression. Treatment modalities include:

1. Steroid Therapy: Systemic or intralesional corticosteroids (e.g., triamcinolone) are used to reduce inflammation and fibrosis.[13]
2. Antioxidants: Vitamins such as vitamin C and E, as well as other antioxidant therapies, are used to combat oxidative stress and promote mucosal healing.
3. Surgical Release of Fibrotic Bands: In advanced cases, surgical intervention may be required to release the fibrotic bands and restore mouth opening.
4. Lifestyle Modifications: Ceasing the consumption of areca nut and tobacco is critical in halting disease progression.
5. Physical Therapy: Jaw exercises and stretching techniques can improve mouth opening in early stages of OSMF.
6. Stem Cell Therapy: There is ongoing research into the use of stem cells in the treatment of OSMF, with the aim of regenerating damaged tissues and reversing fibrosis.[14]

PROGNOSIS

OSMF has a poor prognosis if left untreated. The condition has a potential for malignant transformation into oral squamous cell carcinoma (OSCC).[15] Studies show that around 7-10% of OSMF patients may eventually develop OSCC.[16] Malignant transformation is influenced by factors such as the duration of the disease, extent of fibrosis, and the intensity of the betel nut and tobacco consumption.[17] Early detection and intervention are critical for preventing malignant transformation and improving the quality of life.

CONCLUSION

OSMF is a significant health concern, particularly in areas with high prevalence of areca nut and tobacco use. Early diagnosis and intervention can reduce the risk of complications, including malignant transformation. Public

health initiatives aimed at reducing the consumption of these substances are essential to prevent the incidence of this debilitating condition.

DECLARATION OF PATIENTS CONSENT

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal.

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CONFLICTS OF INTEREST

There are no conflicts of interest.

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