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Comparative analysis of clinical and histopathological features for oral submucous fibrosis

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Abstract:
Oral submucous fibrosis is a chronic potentially malignant disorder and understanding the relationship between its clinical presentation and histopathological changes is essential for accurate disease assessment. Therefore, it is of interest to evaluate the clinico-histopathological correlation in 100 patients with clinically diagnosed oral submucous fibrosis through detailed clinical examination and histopathological analysis of incisional biopsy specimens. Most patients presented with Stage II or Stage III disease, with progressive reduction in mouth opening and increasing burning sensation, mucosal blanching and fibrous bands accompanied by greater epithelial atrophy, dense collagen deposition, hyalinization, reduced vascularity and muscle degeneration. Clinical staging showed a significant positive correlation with histopathological grading ($p < 0.001$), showing parallel progression of clinical and microscopic disease severity. Thus, data shows the importance of integrating clinical and histopathological evaluation for precise staging, individualized treatment planning and early identification of patients at increased risk of malignant transformation.

Keywords: Oral submucous fibrosis (OSMF), histopathology, clinical staging, areca nut, fibrosis, potentially malignant disorder

Background:
Oral Submucous Fibrosis (OSMF) is a chronic, progressive, debilitating and potentially malignant condition affecting oral cavity and associated structures. It is marked by juxta-epithelial inflammatory responses and subsequent fibrosclerosis of the lamina propria and deeper connective tissue leading to stiffness of the oral mucosa and limitation of mouth opening **[1]**. The importance of OSMF as an oral potentially malignant disorder has been increased after being recognized as one of the most important OSPMD due to its high transformation risk to OSCA **[2]**. The disease is mainly restricted to South and Southeast Asia, especially in India where chewing areca nuts in form of gutkha, pan masala and betel quid is a prevalent socio-cultural practice. OSMF has significant impact on affected individuals with issues of pain, functional impairment, nutritional compromise and increased cancer risk and is a significant public health concern in the world **[3]**. OSMF has been a growing problem in recent decades, especially among young people. The spread of commercially ready products of areca nut with good marketing promotion and attractive packaging has been cited for this trend **[4]**. The main etiological factor involved in the initiation and progression of the disease has been identified as Areca nut. Areca nut chewing is strongly implicated in the pathogenesis of OSMF, with progressive fibrosis characterized by extensive collagen deposition and hyalinization of the connective tissue, accompanied by inflammatory and muscular changes **[5]**. The pathogenesis of OSMF is multifactorial and complex with interplay of environmental, genetic, nutritional and immunological factors. The areca nut alkaloids, copper ions, flavonoids, reactive oxygen species and inflammatory mediators all play a role in causing the activation of fibroblasts and cross-linking of collagen **[6]**. These changes lead to overproduction of collagen fibers and eventual fibrosis of the subepithelial connective tissue. Continued inflammation and oxidative stress further worsens structural changes and loss of functions in the oral mucosa, leading to progressive restrictions in oral function **[7]**. In the clinic, OSMF is a progressive, increasingly severe disease. Symptoms often occur early, such as burning sensation, intolerance to spicy food, recurrent ulceration, xerostomia and blanching of the mucous membranes. With disease progression, fibrous bands can be felt in the buccal mucosa, soft palate, faucial pillars and lips which results in a gradual decrease in mouth opening and tongue mobility **[8]**. Nutritional intake is often affected, with dysphagia and difficulty talking resulting from advanced disease, as well as trismus. Clinical staging systems are in common use, but sometimes there is a discrepancy between clinical severity and histopathological staging which is the importance of comprehensive clinicopathological assessment **[9]**. Histopathologically, the changes typical of OSMF are those of disease progression. Early lesions include edema, chronic inflammatory cell infiltration and loosely arranged collagen fibers. During the progression of fibrosis, the bundles of collagen become more dense, hyalinized and cross-linked **[10]**. Other microscopic characteristics are epithelial atrophy, loss of rete ridges, decrease in vascularity, chronic inflammatory infiltrate and degeneration of underlying

muscle fibers. These histological changes have a direct impact on the functional deficits seen in the clinic and could explain the risk of malignant transformation **[11]**. There are a number of clinical and histopathological classification systems for staging OSMF that have been proposed. The clinical staging is primarily judged by mouth opening and functional capacity while the histopathological grading is judged by the degree of fibrosis, collagen deposition, epithelial changes, inflammatory response and vascular changes. There is a correlation between the severity of clinical symptoms and the degree of microscopic progression, which should be taken into consideration for prognosis, treatment planning and long-term follow-up of patients with affected tissues **[12]**. A major concern of the OSMF is its malignant transformation potential. Epithélial dedifferentiation, hypoxia, vascular changes and molecular disruption in the fibrotic microenvironment are conducive to carcinogenesis. Hypoxia-inducible factors, EMT pathways, cytokines, growth factors and a number of genetic regulators have been shown to play a role in disease progression and malignant transformation in recent studies **[13]**. Further molecular biology and computational studies have improved understanding of the pathogenesis of OSMF and led to the identification of targets for future therapy **[14]**. Therefore, it is of interest to analyze the clinical and histopathological features of OSMF among 100 patients and to see the correlation of clinical staging with histopathological grading of the disease.

Materials and Methodology:

Study design and setting:

A cross sectional, comparative observational study was conducted in the Department of Oral Medicine and Radiology in association with Oral Pathology Department of a tertiary care dental college during a 18 month period in the hospital setting.

Study population:

100 patients, clinically diagnosed with Oral Submucous Fibrosis (OSMF) were included in the study, during the study period.

Sample size:

The number of patients selected for the study was 100, which was influenced by the number of clinically diagnosed OSMF cases during the study period and was sufficient in order to have a good statistical power for comparison between clinical stages and histopathological grades.

Inclusion criteria:

[1] Patients older than 18 year.
[2] Clinically diagnosed cases of Oral Submucous Fibrosis.
[3] Patients with a history of chewing areca nut, gutkha, pan masala and betel quid.
[4] Patients who are willing to participate and sign informed consent.
[5] Patients who are willing to have a biopsy for histopathological assessment.

Exclusion criteria:

[1] Previous patients with OSMF.
[2] Patients with the diagnosis of malignancy put at presentation in the oral cavity.
[3] Patients who have systemic fibrotic disorders.
[4] Immunosuppressive therapy patients.
[5] Women who are pregnant or breastfeeding.
[6] Patients unwilling to participate in the study.

Ethical considerations:

The Institutional Ethics Committee gave ethical approval prior to the start of the study. All participants gave written informed consent, in which they were informed about the purpose and content of the study.

Data collection procedure:

Clinical evaluation:

A detailed case history was recorded for each participant including:

[1] Age
[2] Gender
[3] Occupation
[4] Duration, frequency of consuming areca nut/gutkha
[5] Type of chewing habit
[6] Duration of symptoms

A thorough oral exam was conducted with suitable lighting. The following clinical parameters were noted:

[1] **Burning Sensation** - Evaluated with a Visual Analogue Scale (VAS) from 0-10.
[2] **Mouth Opening** - As the distance between the incisors (the greatest distance) measured with a calibrated Vernier caliper and reported in mm.
[3] **Tongue Protrusion** - From the mesio-incisal edge of the maxillary central incisors to the tip of the protruded tongue.
[4] **Cheek Flexibility** - Based on accepted clinical procedures.
[5] **Mucosal Blanching** - Observed/not observed.
[6] **Fibrous Bands** - Palpable fibrous bands were recorded and their location was noted.
[7] **Clinical Staging** - The clinical staging was done as per Khanna and Andrade's clinical classification based on mouth opening and the severity of the signs of the disease.

Histopathological evaluation:

Biopsy procedure:

Representative fibrotic areas were biopsied by incisional biopsy, preferably from the buccal mucosa, after injection of local anaesthetic. Tissues were promptly fixed in 10% neutral buffered formalin.

Histological processing:

The specimens were processed in the usual way and embedded in paraffin wax. Specimens were cut into multiple 4-5µ slices and stained with Hematoxylin and Eosin (H&E) stain. Histopathological Assessment Slides were examined independently by two experienced oral pathologists. The following histopathological parameters were analyzed:

[1] Epithelial thickness
[2] Degree of epithelial atrophy
[3] Abnormal cells that could develop into cancer
[4] Inflammatory cell infiltration
[5] Collagen fiber arrangement

Collagen hyalinization:
[1] Vascularity
[2] Muscle degeneration
[3] Fibrosis severity

Histopathological grading:
A grading system, following the Pindborg and Sirsat histopathological criteria, was used:
[1] Grade I (Early Stage)
[2] Average Grade II (Early Stage)
[3] Grade III (Moderately Advanced Stage)
[4] Subtotalized MDMA use (IV–Advanced Stage).

Outcome measures:
Primary outcome:
There is a fair degree of correlation between clinical stage and histopathological grade of OSMF.

Secondary outcomes:
[1] The data for demographic variables are distributed.
[2] Association of disease severity with the chewing habits of the individuals.
[3] The comparison of clinical manifestations according to histopathological grades.
[4] Assessment of epithelial and connective tissue changes at the various stages.

Statistical analysis:
The data were tabulated in the Microsoft Excel and analysed by IBM SPSS Statistics Version 29.0. Data were presented as mean ± SD for continuous variables and frequencies (percentages) for categorical variables. The following statistical tests were used: Chi-square test for categorical variables, Fisher's exact test (where applicable), Independent t-test and ANOVA for comparison of means and Spearman correlation analysis was used to evaluate the association between clinical stage and histopathological grade. A p-value < 0.05 was deemed significant.

Result:

A total of 100 patients clinically diagnosed with Oral Submucous Fibrosis (OSMF) were included in the study. Clinical findings were compared with histopathological grading to determine the relationship between disease severity and microscopic tissue alterations. The majority of patients were males with a history of areca nut or gutkha chewing. Progressive worsening of clinical symptoms was observed with increasing histopathological grades. Among the 100 patients, Stage II OSMF constituted the largest proportion (33%), followed by Stage III (30%), Stage I (22%) and Stage IV (15%). Histopathologically, Grade II fibrosis was the most common finding (33%), followed by Grade III (32%), Grade I (20%) and Grade IV (15%). A statistically significant association was observed between clinical stage and histopathological grade ($p<0.001$). Patients with advanced clinical stages predominantly demonstrated advanced histopathological grades, indicating a strong correlation between clinical severity and microscopic progression of fibrosis **(Table 1)**. A significant deterioration in clinical parameters was observed with increasing histopathological grade. Mean burning sensation scores increased from 3.2 in Grade I to 8.2 in Grade IV ($p<0.001$). Mean mouth opening progressively decreased from 39.4 mm in Grade I to 18.9 mm in Grade IV ($p<0.001$). Similarly, tongue protrusion showed significant restriction in advanced grades. Clinical signs such as mucosal blanching and palpable fibrous bands became increasingly prevalent, reaching 100% among Grade IV patients. These findings show that worsening histopathological fibrosis is accompanied by greater functional impairment and symptom severity **(Table 2).** Histopathological alterations showed progressive worsening with increasing grade. Epithelial atrophy increased from 20% in Grade I to 100% in Grade IV cases. Dense collagen deposition and collagen hyalinization showed a similar trend, reflecting progressive fibrosis. Reduced vascularity and muscle degeneration became increasingly prominent in advanced grades. Dysplastic changes were absent in Grade I but observed in 26.7% of Grade IV cases, indicating an elevated risk of malignant transformation in advanced disease. All associations were statistically significant **(Table 3).**

Table 1: Distribution of patients according to clinical stage and histopathological grade

Clinical Stage	Grade I	Grade II	Grade III	Grade IV	Total
Stage I	12	8	2	0	22
Stage II	6	18	8	1	33
Stage III	2	6	17	5	30
Stage IV	0	1	5	9	15
Total	20	33	32	15	100

Chi-square = 52.84, p < 0.001

Table 2: Comparison of clinical parameters across histopathological grades

Parameter	Grade I (n=20)	Grade II (n=33)	Grade III (n=32)	Grade IV (n=15)	p value
Burning Sensation (VAS)	3.2 ± 1.1	4.8 ± 1.4	6.7 ± 1.5	8.2 ± 1.2	<0.001
Mouth Opening (mm)	39.4 ± 3.2	33.8 ± 3.8	27.1 ± 4.2	18.9 ± 3.5	<0.001
Tongue Protrusion (mm)	37.6 ± 4.1	33.2 ± 3.7	28.7 ± 4.4	22.8 ± 3.8	<0.001
Blanching Present (%)	40.0%	69.7%	90.6%	100%	<0.001
Fibrous Bands Present (%)	45.0%	72.7%	93.8%	100%	<0.001

Table 3: Histopathological features across different grades of OSMF

Histopathological Feature	Grade I (%)	Grade II (%)	Grade III (%)	Grade IV (%)	p value
Epithelial Atrophy	20.0	48.5	78.1	100.0	<0.001

Dense Collagen Deposition	30.0	66.7	90.6	100.0	<0.001
Collagen Hyalinization	15.0	45.5	84.4	100.0	<0.001
Reduced Vascularity	10.0	39.4	75.0	100.0	<0.001
Muscle Degeneration	5.0	24.2	59.4	93.3	<0.001
Dysplastic Changes	0	3.0	9.4	26.7	0.002

Discussion:

In the present study, the correlation of clinical manifestation and histopathological changes was evaluated in 100 patients with Oral Submucous Fibrosis (OSMF) diagnosis. The results revealed a strong relationship between a progressive microscopic change and increasing clinical severity. Advanced clinical stages showed higher restriction of mouth opening, intense burning sensation, extensive blanching of mucous membranes and presence of fibrous bands, associated with higher histopathological grades (epithelial atrophy, collagen hyalinization, less vascularization, muscle degeneration and dysplastic changes). The use of areca nuts and commercially available smokeless chewing products has led to OSMF being one of the most common oral potentially malignant disorders in South Asia. Recent studies show that there is an increasing prevalence among young people, highlighting the public health problem of the disease. Alka *et al.* found that progressive fibrosis, hypoxia and changes in angiogenesis play an important role in the pathogenesis of OSMF and is a mechanism responsible for the progression of disease and malignant transformation potential **[14]**. This is supported by the present study which showed that clinical stages were correlated with the severity of the histopathological changes, with functional impairment being more pronounced in advanced clinical stages. A good correlation was found between clinical staging and histopathological grading. Most patients with stage III and IV disease had Grade III and Grade IV changes in the histopathology. However, Goel *et al.*, in a clinico-histopathological study of 100 OSMF cases, reported no significant correlation between clinical staging and histopathological grading. The authors suggested that differences in the severity and extent of fibrosis across different oral mucosal regions and involved muscles may contribute to this discrepancy **[15]**. This association is another reminder of the significance of combining clinical and microscopic evaluation to make a correct diagnosis and staging of the disease. The progressive decrease in mouth opening with increasing grade of the histopathological changes was one of the most remarkable observations of this present study. This decrease is due to a rise in collagen deposition, fibrosis and muscle involvement **[16]**. The marked limitation in the ability to use the oral cavity is probably due to the presence of collagen bundles and hyalinization seen in advanced lesions with histopathological examination. Excessive accumulation of ECM has been described in studies of tissue remodeling and fibrosis in OSMF, which result in progressive trismus and oral rigidity **[17]**. Burning sensation was found to be more prevalent as the disease severity increased. This observation may be explained by the chronic inflammatory changes of the oral mucosa and epithelial atrophy. The thinner the epithelium, the more likely it is to become irritated by spicy foods and other irritating stimuli. Jaffri *et al.* reported that patients with OSMF experienced greater functional impairment, particularly difficulty with chewing and swallowing and greater psychological distress compared with patients with oral lichen planus, highlighting the substantial functional and psychosocial burden associated with OSMF **[18]**. The high rate of epithelial atrophy seen in the advanced grades of the present study substantiates this explanation. The most common clinical findings were blanching of the mucosa and palpable fibrous bands, which increased significantly as disease progresses. Blanching is caused by diminished vascularity and progressive fibrosis of the connective tissue. In the present study, a similar decrease in vascularity was noted in advanced grades by histological examination. The development of progressive collagen deposition and vascular compromise is also reported in the recent reviews on potentially malignant oral lesions, which explains the clinical manifestation of OSMF **[19]**. Further important findings were the gradual progression of epithelial atrophy, as the grade of the histopathological increased. Significant epithelial thinning was seen in all Grade IV lesions. This observation is of clinical significance because the epithelial atrophy allows carcinogens from areca nut and tobacco products to penetrate to deeper tissues. Naik *et al.* identified several clinical and histopathological features associated with malignant transformation in OSMF and reported epithelial dysplasia in 27% of cases, emphasizing the importance of identifying histological abnormalities even in clinically less advanced disease **[20]**. Advanced disease was characterized by collagen hyalinization and dense collagen deposition. The amount of fibrosis found in the present study is in line with the current knowledge of the pathogenesis of OSMF. Molecular studies in recent years have shown that progressive fibrosis of connective tissue is due to fibroblast activation, abnormal collagen metabolism and dysregulated extracellular matrix remodeling. In OSMF tissues, Zhang *et al.* reported that PDGF-BB and autophagic pathways are involved in the activation and phenotypic transformation of fibroblasts, which plays a role in the process of fibrosis **[17]**. The mechanisms involved are probably responsible for the widespread collagen deposition found in the advanced lesions in the present study. As the disease progressed, the degeneration of the muscles became more prominent. The muscle fiber degeneration was especially prominent in the Grade III and Grade IV lesions. Severe trismus and restriction of tongue movement may result from progressive fibrosis which progresses into surrounding muscle. These observations also suggest that the involvement of OSMF is not restricted to superficial connective tissues but extends to deeper tissues involved in oral function **[21]**. The finding of epithelial dysplasia in advanced lesions is of special interest. Dysplastic changes were found mainly in Grade IV, suggesting that those grades are at a higher risk for malignant transformation. Ellis *et al.* showed that the molecular and structural changes of the epithelium can be used to identify the progression to malignancy

in the oral PMDs **[16]**. Finally, the presence of dysplastic changes in OSMF patients with advanced disease highlights the importance of frequent surveillance, biopsy evaluation and long-term follow-up. The overall results of the present study support that the severity of the disease is closely related to the severity of the disease at the histological level in OSMF. A strong correlation between clinical staging and microscopic grading is found, thus adopting a comprehensive clinicopathological assessment for the diagnosis, prognosis and treatment planning. It is important to identify and remove harmful habits early in the process in order to avoid the progression of disease and the risk of the development of malignancy.

Conclusion:

Clinical staging showed a significant correlation with histopathological grading, with advancing disease associated with greater functional impairment, epithelial atrophy, collagen hyalinization, vascular compromise and muscle degeneration. Histopathological examination remains essential for accurately assessing disease severity and identifying patients at increased risk of malignant transformation. These findings highlight the importance of early diagnosis, cessation of areca nut chewing and regular follow-up to improve outcomes and reduce the risk of oral cancer.

References:

[1] Tang J *et al. Int J Oral Sci.* 2025 **17**:8. [PMID: 39890798]
[2] Qin X *et al. Int J Mol Sci.* 2023 **24**:4992. [PMID: 36902423]
[3] Shih YH *et al. Int J Mol Sci.* 2019 **20**:2940. [PMID: 31208114]
[4] Kazemi K *et al. Dent J (Basel).* 2025 **13**:364. [PMID: 40863067]
[5] Adhane YB *et al. IP J Diagn Pathol Oncol.* 2021 **6**:211. [DOI: 10.18231/j.jdpo.2021.045]
[6] Mokal CN *et al. Front Cell Dev Biol.* 2026 **13**:1754209. [PMID: 41660015]
[7] Pandiar D *et al. Cureus.* 2023 **15**:e49107. [PMID: 38125265]
[8] Chhabra AK *et al. Cureus.* 2023 **15**:e47259. [PMID: 38022118]
[9] Mohammed F *et al. J Oral Pathol Med.* 2015 **44**:208. [PMID: 25047540]
[10] Ganganna K *et al. J Oral Maxillofac Pathol.* 2012 **16**:162. [PMID: 22923884]
[11] Rao NR *et al. J Otolaryngol Head Neck Surg.* 2020 **49**:3. [PMID: 31915073]
[12] More CB & Rao NR. *J Oral Biol Craniofac Res.* 2019 **9**:311. [PMID: 31334003]
[13] Gao G *et al. Front Pharmacol.* 2025 **16**:1540559. [PMID: 39959427]
[14] Alka H *et al. BMC Oral Health.* 2026 **26**:1233. [PMID: 42026549]
[15] Goel S *et al. J Carcinogene Mutagene.* 2010 **1**:1000108. [DOI: 10.4172/2157-2518.1000108]
[16] Ellis BG *et al. PLoS One.* 2022 **17**:e0266043. [PMID: 35333891]
[17] Zhang H *et al. Sci Rep.* 2025 **15**:15449. [PMID: 40316684]
[18] Jaffri SB *et al. Biol Clin Sci Res J.* 2024 **2024**:1339. [DOI: 10.54112/bcsrj.v2024i1.1339]
[19] https://link.springer.com/chapter/10.1007/978-3-031-84539-0_1
[20] Naik V *et al. BMC Oral Health.* 2025 **25**:1053. [PMID: 40604858]
[21] Pandya S *et al. Head Neck Oncol.* 2009 **1**:10. [PMID: 19409103]

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