ISSN 0973-2063 (online) 0973-8894 (print)

Bioinformation 22(10): 7595-7598 (2026)

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BIOINFORMATION
Discovery at the interface of physical and biological sciences

BIOMEDICAL INFORMATICS

www.bioinformation.net

Volume 22(10)

Review

Received October 1, 2026; Revised October 31, 2026; Accepted October 31, 2026, Published October 31, 2026

DOI: 10.6026/973206300227595

SJIF 2026 (Scientific Journal Impact Factor for 2026) = 8.478
2022 Impact Factor (2023 Clarivate Inc. release) is 1.9

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Citation: Abdul, Bioinformation 22(10): 7595-7598 (2026)

Muscle degeneration and ultrastructural changes in oral submucous fibrosis: A review

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ISSN 0973-2063 (online) 0973-8894 (print)
Bioinformation 22(10): 7595-7598 (2026)
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Abstract:
Oral submucous fibrosis (OSMF) can involve skeletal muscle beneath fibrotic mucosa. Therefore, it is of interest to examine the muscle findings from microscopy, ultrasound and electromyography. Reported changes include fibre atrophy, myofibril disruption and altered muscle activity. Current studies are small and do not establish muscle degeneration as the cause of trismus. Prospective studies should relate muscle changes to oral aperture and treatment response.

Keywords: Oral submucous fibrosis (OSMF), skeletal muscle, ultrastructure, muscle degeneration, masseter, buccinators, trismus, narrative review

Background:
Oral submucous fibrosis is a fibrosing oral potentially malignant disorder in which repeated exposure to areca nut and related preparations produces progressive connective-tissue alterations. Its morbidity extends beyond a visible mucosal lesion: burning, impaired tongue mobility, limited cheek elasticity and trismus can interfere with nutrition, oral hygiene and dental treatment. Recent reviews have emphasized interactions among extracellular matrix deposition, altered tissue signalling and malignant potential **[1].** Pathobiological studies describe excessive collagen synthesis, reduced degradation, fibroblast activation and inflammation as contributors to the fibrotic phenotype **[2].** Therefore, it is of interest to explore the skeletal muscle findings across microscopic, imaging and physiological investigations without treating heterogeneous reports as a pooled estimate.

Approach to the narrative review:
This narrative review synthesizes selected clinical, pathological and experimental publications cited in the source manuscript. Studies addressing muscle ultrastructure, oral and masticatory muscle morphology, electromyographic findings and functional implications were given particular attention, together with background work on OSMF pathogenesis. The articles were examined for the muscular structure studied, the method of observation, the direction of reported changes and plausible explanations for disagreement. Because this is a qualitative narrative synthesis, there was no protocol registration, formal PRISMA selection flow, pooled effect estimate or standardized risk-of-bias scoring. Coverage should not be interpreted as exhaustive.

Clinical setting and functional phenotype:
The broad clinical spectrum of OSMF requires separating mucosal signs, oral aperture and muscle-specific impairment. Early oral burning and mucosal blanching may precede marked trismus, whereas more advanced tissue rigidity can coexist with reduced mandibular excursion, problems chewing and substantial oral disability. Clinical grading frameworks have been proposed to describe this progression, but graded oral aperture is an integrated measurement of mucosal, muscular, articular and behavioural influences **[3].** Clinical functional restriction should not be attributed entirely to fibrosis without considering coexisting temporomandibular conditions, pain, parafunction and disuse. Disease severity assessments may therefore benefit from documenting interincisal distance and jaw movement together with symptoms, habitual chewing exposure and relevant muscle findings. Reports comparing clinical grading systems suggest that apparent associations depend on how OSMF stage is operationalized **[4].**

Mechanisms potentially linking fibrosis to muscle injury:
Profibrotic signalling may change the immediate mechanical and biochemical environment of superficial skeletal muscles. Dense collagen bundles can constrain tissue excursion, impair local microvascular exchange and expose muscle fibres to chronic compression or reduced activity. Histological observations have been interpreted in light of these processes, but direct causal evidence demonstrating a unique pathway from submucosal fibrosis to myocyte damage remains incomplete. Histopathological descriptions of progressive OSMF support the plausibility of multifactorial tissue involvement **[5].** Areca-nut-related exposure may also sustain local inflammatory and oxidative stress pathways. These processes offer possible explanations for muscular changes but should not be confused with proven direct toxicity to human buccinator or masseter fibres. Contemporary mechanistic work encourages separating matrix deposition, neurovascular changes and muscle-cell responses in future studies **[6].**

Histopathological evidence of muscular involvement:
Early pathological observations drew attention to skeletal muscle lying beneath densely fibrotic oral mucosa. Biopsy series describe variation in fibre calibre, separation of muscle bundles by connective tissue and occasional degenerative features. Such changes may be focal; a single biopsy cannot establish whether the process is present throughout the musculature involved in mandibular excursion. Studies focusing on mouth-opening treatments also underscore the importance of persistent mechanical and tissue-level constraints **[7]**. The classic ultrastructural report by El-Labban and Canniff described damage extending to skeletal muscle in OSMF biopsies, supporting the proposition that the disorder is not confined strictly to collagen in the superficial lamina propria **[8].** Histological examination of palatal and related musculature later broadened the structures considered relevant to speech, swallowing and oral movement **[9].** These reports are valuable for documenting possible pathology but are limited by tissue selection, processing artefacts and lack of prospective linkage with objective mechanical function.

Ultrastructural changes in muscle fibres:
Transmission electron microscopy has revealed disruption of myofibrillar organization and variable organelle abnormalities in

affected tissue. A reported combination of fragmented contractile elements, atypical mitochondrial profiles and increased perimuscular fibrosis is biologically compatible with chronic injury or altered loading. Sumathi and colleagues explored the status of muscle in OSMF using histology and electron microscopy, highlighting the value of examining architecture below the resolution of routine light microscopy **[10].** Interpretation requires caution because fixation, sampling site and fibre orientation can resemble pathology. Ultrastructural appearance alone cannot show whether the change preceded restricted mouth opening or developed from chronic disuse. Quantitative stereology, fibre-type markers and standardized control sampling would help distinguish primary muscle degeneration from an adaptive reaction to longstanding fibrosis.

Ultrasonography and muscle morphology:

Ultrasonography offers a non-invasive view of muscle thickness and morphology, but its results depend on probe placement, occlusal clenching state, patient size and operator technique. Devathambi and Aswath evaluated masseter morphology in OSMF, providing an imaging counterpart to pathological reports **[11].** Other ultrasound work observed apparent increases in thickness in some patients, which need not contradict reports of fibre degeneration because bulk thickness and intrinsic tissue quality measure different dimensions **[12].** Morphometric and histopathological observations further suggest that stage and oral aperture do not relate uniformly to every muscle endpoint **[13].** Comparisons including areca-nut chewers or bruxism groups indicate that habitual muscle loading is an important potential confounder when interpreting a thicker masseter in OSMF **[14].** Prospective studies should distinguish resting thickness from contracted thickness and pair imaging with quantitative mouth-opening and chewing-function assessments.

Electromyography and treatment-related muscle activity:

Surface electromyography measures electrical activity during selected motor tasks rather than directly quantifying fibre viability or microscopic degeneration. In OSMF, altered activation can reflect pain inhibition, restricted movement, fatigue, adaptation to chewing habits or altered muscle recruitment. A clinical study of muscle activity around OSMF management illustrates the potential to document function longitudinally, but intervention-associated change may not be attributable to reversal of skeletal muscle pathology **[15].** More recent electromyographic observations similarly suggest that rehabilitation may be accompanied by changes in masticatory muscle function **[16].** Standardization of electrode placement, clench force, jaw position and recording conditions is essential for comparison. Electrical activity can complement but cannot replace histological or imaging assessment when the research question concerns structural degeneration.

Clinical implications and research priorities:

Muscle pathology should be regarded as one component of OSMF-related functional disability rather than a stand-alone diagnostic criterion. Clinical care must continue to prioritize cessation of areca-nut habits, symptom management, rehabilitation of mouth opening and appropriate surveillance for malignant transformation. Descriptive imaging or EMG tests are not yet sufficiently validated to guide individual treatment selection. Historical observations of deep-tissue involvement also argue against treating trismus as an exclusively superficial mechanical problem **[17].** A stronger evidence base would follow patients prospectively, document quantitative exposure histories, use consistent OSMF staging and collect synchronized measurements of oral aperture, pain, imaging, EMG and patient-reported function. When tissue sampling is clinically indicated, standardized immunohistochemistry and quantitative microscopy could distinguish inflammation, fibrosis, atrophy, mitochondrial change and fibre-type shifts. Investigators should examine anatomical subregions separately and report interobserver reproducibility. Evidence supporting reversal of muscle injury through a specific therapy requires controlled longitudinal comparisons rather than cross-sectional associations. Gupta and Jawanda **[18]** described muscular hyalinization and degeneration among the histopathological features of OSMF. Sayed Abdul *et al.* **[19]** examined buccinator biopsies from seven patients with OSMF and three healthy controls by light and transmission electron microscopy; they reported fibre atrophy and splitting, fibrosis, myofibrillar disorganization, mitochondrial abnormalities and autophagic vacuoles. These findings support deep-tissue involvement but, because of the small descriptive sample, do not establish whether muscle injury independently causes trismus.

Limitations of the evidence:

Existing studies vary in their populations, diagnostic criteria, stage distributions, muscle groups examined and measurement platforms. Histological samples represent limited regions and are not always matched to the muscles measured by ultrasound or electromyography. Most reports are observational; consequently, neither temporal ordering nor the independent contribution of muscle damage to trismus can be established. The present narrative synthesis does not provide pooled prevalence estimates or claims of a systematic search.

Conclusion:

Muscular changes in OSMF extend the pathological picture beyond submucosal collagen accumulation. Microscopy, ultrasound and electromyography provide complementary but non-interchangeable observations. Consistent longitudinal assessment is needed to clarify whether and how muscle degeneration contributes to trismus and responds to therapy.

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