REVIEW ARTICLE

Specific Clinical and Histopathological Classifications of Oral Submucous Fibrosis

Arti Singh Rajput[1], Satpal Yadav[2], Praveen Singh Rajput[3], Babita Singh Rajput[4]

ABSTRACT

Oral submucous fibrosis (OSMF) is a potentially malignant condition that predominantly occurs in Indians. This article provides an overview of specific Clinical and Histopathological classifications of Oral Submucous Fibrosis (OSMF). So that it can help to the clinicians and oral pathologists to the management and minimize the blind clinical trials and treatment modalities.

Keywords: Classification, OSMF, Precancer lesion

How to cite this article: Arti Singh Rajput, Satpal Yadav, Praveen Singh Rajput, Babita Singh Rajput. Specific clinical and histopathological classifications of oral submucous fibrosis. International Journal of Contemporary Medical Research. 2015;2(2): 182-185

[1]Dental S.R., Baba Saheb Ambedkar hospital, Delhi, [2]Senior Pediatric consultant, Jaipur Golden hospital and Gupta hospital, Delhi, [3,4]Medical officer, GRMC, Gwalior, M.P, India

Corresponding author: Dr. Arti Singh Rajput, Dental S.R., Baba Saheb Ambedkar hospital, Delhi, India

Source of Support: Nil

Conflict of Interest: None

INTRODUCTION

Oral submucous fibrosis is a world wide accepted chronic and potentially malignant condition of the oral cavity.[1] The key feature of the disease is submucosal fibrosis that affects the oral cavity and progressively involves the pharynx and the upper esophagus.[2]

Overall prevalence rate of OSMF in India to be about 0.2% to 0.5 % and prevalence by gender varying from 0.2-2.3% in males and 1.2-4.57% in females. The age range of Oral submucous fibrosis patients is 20 and 40 years .[3] Its etiopathological factors are ingestion of chillies, genetic susceptibility, nutritional deficiencies, altered salivary constituents, autoimmunity and collagen disorders.[1]

CLINICAL AND HISTOPATHOLOGICAL CLASSIFICATIONS OF ORAL SUBMUCO US FIBROSIS (OSMF)

Figure-1: Restricted mouth opening

Figure-2: Oral mucosa shows blanching and loss of elasticity

Figure-3: Histopathological view shows Marked fibrosis with hyaline changes extending from subepithelial to superficial muscle layers.

The severity of disease and the degree of mouth opening were co-related by Jain P and Suman V et al. (1988) as:

Normal mouth opening- 40-60 mm.
Grade I: 40-42mm.
Grade II: 31-39mm.
Grade III: 21-30mm.
Grade IV: 10mm-20mm.[1]

Sinor et al. (1990) reported mouth opening from 8 to 40 mm in OSMF cases as compared to 33 to 60 mm in controls.[4]

Khanna JN, Dave R (1995) classified oral submucous fibrosis into:

Grade I: Very early or incipient stage

Burning sensation; dryness of mouth; vesicles or ulcerations; irritation with spicy food; no change in mucosal colour; no fibrous bands palpable; mouth opening 36-40 mm; tongue protrusion normal.

Grade II: Early stage

Burning sensation and dryness of mouth, irritation with spicy food; oral mucosa blanched and loss of elasticity; no clear cut fibrotic band; slight restriction of mouth opening; tongue protrusion normal; mouth opening 26-35 mm.

Grade III: Moderately advanced stage

Burning sensation and dryness of mouth; irritation with spicy food; blanched; opaque; leather-like mucosa, vertical fibrotic bands on buccal mucosa making it stiff, considerable restriction of mouth opening, tongue protrusion not much affected, difficulty in eating and speaking, poor oral hygiene, mouth opening 15-25 mm.

Grade IV: Advanced stage

Burning sensation and dryness of mouth; irritation with spicy food; blanched; opaque; leather-like mucosa; thick fibrous bands on both buccal mucosa; retromolar area and at pterygomandibular raphe; very little mouth opening; restricted tongue protrusion; speech and eating very much impaired; very poor oral hygiene; mouth opening <15 mm.[5]

Khanna and Andrade (1995) grouped OSMF into 4groups based on clinical and histopathological features.

Group I: Very early changes

- Common symptom is burning sensation in the mouth.
- Acute ulceration and recurrent stomatitis.
- Not associated with mouth opening limitation.

Histology

- Fine fibrillar collagen network interspersed with marked edema.
- Blood vessels dilated and congested.
- Large aggregate of plump, young fibroblasts present with abundant cytoplasm.
- Inflammatory cells mainly consist of polymorphonuclear leukocytes with few eosinophils.
- Epithelium normal

Group II: Early cases

Buccal mucosa appears mottled and marble-like. Widespread sheets of fibrosis palpable. Patients with an interincisal distance of 26-35mm.

Histology

- Juxtaepithelial hyalinization present.
- Collagen present as thickened but separate bundles.
- Blood vessels dilated and congested.
- Young fibroblasts seen in moderate number.
- Inflammatory cells mainly consist of polymorpho nuclear leukocytes with few eosinophils and occasional plasma cells.
- Flattening or shortening of epithelial rete pegs evident with varying degree of keratinization.

Group III: Moderately advanced cases

- Trismus evident with an interincisal distance of 15-25mm.
- Buccal mucosa appears pale and firmly attached to underlying tissues.
- Atrophy of vermilion border.
- Vertical fibrous bands palpable at the soft palate, pterygomandibular raphe and anterior faucial pillars.

Histology

- Juxtaepithelial hyalinization present.
- Thickened collagen bundles faintly discernible, separate by very slight, residual edema.
- Blood vessels, mostly constricted.
- Mature fibroblasts with scanty cytoplasm and spindle shaped nuclei.
- Inflammatory exudate consists mainly of lymphocytes.
- Epithelium markedly atrophic with loss of rete pegs.
- Muscle fibers seen interspersed with thickened and dense collagen fibers.

Group IV A: Advanced cases

- Trismus is severe with interincisal distance of less than 15mm.
- The fauces are thickened, shortened and firm on palpation.
- Uvula is shrunken and appears as a small, fibrous bud.

- Tongue movements are limited.
- On palpation of lips, circular band felt around entire mouth

Group IV B: Advanced cases with premalignant and malignant changes.

- Hyperkeratosis, leukoplakia, or squamous cell carcinoma can be seen.

Histology

- Collagen hyalinized as smooth sheet.
- Extensive fibrosis obliterating the mucosal blood vessels and eliminating the melanocytes.
- Fibroblasts markedly absent within the hyalinized zones.
- Total loss of epithelial rete pegs.
- Mild to moderate atypia present.
- Extensive degeneration of muscle fibers evident.[6]

Pindborg and Sirsat (1996) described four consecutive stages:

Very early stage (Grade I):

- A finely fibrillar collagen, dispersed with marked edema.
- The fibroblastic response is strong.
- The blood vessels are sometimes normal, but more often they are dilated and congested.
- Inflammatory cells, mainly polymorpho nuclear leukocytes with an occasional eosinophils are present.

Early stage (Grade II):

- The juxta-epithelial area shows early hyalinization.
- Plump young fibroblasts are present in moderate numbers.
- The blood vessels are dilated and congested.
- The inflammatory cells are mostly mono-nuclear lymphocytes, eosinophils and an occasional plasma cell.

Moderately advanced stage (Grade III):

- The collagen is moderately hyalinized.
- The fibroblastic response is less marked, the cells present being mostly adult fibrocytes.
- Blood vessels are normal or constricted.
- The inflammatory exudates consist of lymphocytes and plasma cells, although an occasional eosinophil is seen.

Advanced stage (Grade IV):

- The collagen is completely hyalinized.
- The hyalinized areas are devoid of fibroblasts.
- Blood vessels are completely obliterated or narrowed.
- The inflammatory cells are lymphocytes and plasma cells.[7]

Haider et al. (2000) described the clinical and functional staging of OSMF.[8]

- Clinical stage
- Faucial bands only
- Faucial and buccal bands
- Faucial, buccal and labial bands
- Functional stage
- Mouth opening ≥20mm.
- Mouth opening 11-19mm
- Mouth opening ≤10mm.

Ranganathan K et al. (2001) clinically grouped OSMF into 4 groups based on mouth opening parameters:

1. Group I: Only clinical symptoms present with mucosal changes but no restriction of mouth opening (more than 35mm).
2. Group II: Restricted mouth opening (between 20 to 35mm).
3. Group III: Limited mouth opening (less than 20mm).
4. Group IV: Nil mouth opening with precancerous or cancerous changes in oral mucosa.[9,10]

Tilakaratne (2005) modified Pindborg's stages**:**

Early stage:

- Large number of lymphocytes in subepithelial, connective tissue zone along with myxoedematous changes.

Intermediate stage:

- Granulation changes close to the muscle layer and hyalinization appears in subepithelial zone where blood vessels are compressed by fibrous bundles. Reduced inflammatory cells in subepithelial layer.

Advanced stage:

- Inflammatory cell infiltrate is hardly seen. Number of blood vessels dramatically small in subepithelial zone. Marked fibrosis with hyaline changes extending from subepithelial to superficial muscle

layers. Atrophic, degenerative changes start in muscle fibers.

Utsunomiya H et al. (2005) histopathologically divided OSMF into 3 stages:

Early stage: Juxta-epithelial area of hyalinization. Dilated and congested blood vessels with large number of lymphocytes, eosinophils and occasional plasma cells in sub-epithelial zone along with myxo-edematous changes.

Intermediate stage: Hyalinization of sub-epithelial zone with compression of blood vessels, reduced inflammatory cell infiltrate, and granulation tissue changes close to muscle bundles.

Advanced stage: Number of blood vessels reduced, obliterated, or narrowed in sub-epithelial zone with no inflammatory cell infiltrate. Marked fibrosis and hyalinization extending from subepithelial to superficial muscle layers with atrophic degenerative changes of muscle fibers.[11]

OSMF cases were clinically categorized by Kiran Kumar et al. (2007) into three clinical stages according to ability to open the mouth.

Stage I: Mouth opening >45 mm.

Stage II: Restricted mouth opening 20-44 mm.

Stage III: Mouth opening < 20 mm.[8]

The histopathological grading followed by Kiran Kumar et al.

Grade I: Loose, thin and thick fibres.

Grade II: Loose or thick fibres with partial hyalinization.

Grade III:Complete hyalinization.[8]

CONCLUSION

Oral submucous fibrosis (OSMF) is now accepted globally as an Indian disease, having highest malignant potential (7.6%) than any other oral premalignant lesions. An attempt is provide and update the knowledge of classification system of OSMF. So that these classification can help to the clinicians and oral pathologists to the management and minimize the blind clinical trials and treatment modalities.

REFERENCES

1. Hasana Shamimul, Sherwania Osama, Ahmeda Sameer, Khana Mohd Abbas. Oral submucous fibrosis turning into malignancy: A case report and review of literature. J Orofac Sci. 2011;3:30-6.
2. Angadi Punnya V, Rao Sanjay S. Areca nut in pathogenesis of oral submucous fibrosis: revisited. Oral Maxillofac Surg 2011;15:1–9.
3. Yadav Sunil. Etiopathogenesis and management of oral submucous fibrosis**.** The Internet Journal of Bioengineering ISSN 1937; 8: 246.
4. Tepan MG et al. Use of tongue flap in submucous palatal fibrosis. The journal of laryngology and otology 1986; 100: 155-460.
5. Patidar Kalpana A, Parwani Rajkumar N., Wanjari Sangeeta Panjab. Correla -tion of salivary and serum IgG, IgA levels with total protein in oral submuc- ous fibrosis. Journal of Oral Science 2011; 53: 97-102,
6. JK Savita, HC Girish, Murgod Sanjay, Kumar Harish. Oral submucous fibrosis: A review [part 2].Journal of Health Sciences and Research 2011; 2:1-6.
7. Pandya Shruti, Chaudhary Ajay Kumar, Singh Mamta, Singh Mangal, Mehrotra Ravi. Correlation of histopathological diagnosis with habits and clinical findings in oral submucous fibrosis. Head and Neck Oncology 2009;1: 1758-3284.
8. Kiran Kumar K, Saraswathi TR, Ranganathan K, Uma Devi M, Elizabeth J Oral submucous fibrosis: A Clinico-histopathologic study in Chennai. Indian J of Dent Res 2007;18: 53-9.
9. Goel Saurabh, Ahmed Junaid, Singh Mohit Pal, Nahar Prashant. Oral submuc ous fibrosis: A clinico-histopathological comparative study in population of Southern Rajasthan. J Carcinogene Mu-tagene 2010;1:2-4.
10. Ranganathan K, Mishra Gauri. An overview of classification schemes for oral submucous fibrosis. Journal of Oral and maxillofacial pathology 2006; 10(2): 55-8.
11. George Antony, BS Sreenivasan, S Sunil et al. Potentially Malignant Disor ders of Oral Cavity. Oral and Maxillo- facial Pathology Journal 2011;2: 0976-1225.