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Histological changes to palatal and paratubal muscles in oral submucous fibrosis

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Abstract
Oral submucous fibrosis (OSMF) is prevalent in the Indian subcontinent. In a large number of patients conductive deafness due to eustachian tube dysfunction has been found. The present study was, therefore, planned to assess the histopathological changes in palatal and paratubal muscles in oral submucous fibrosis. Incisional biopsy from the soft palate was taken in clinically proven cases of OSMF. In most of the cases, signs of chronic inflammation and fibrosis were seen in the submucosa. Dysplasia was noticed in seven (13.2 per cent) patients. Degenerative changes in palatal/paratubal muscles were found in the form of loss of cross striations in seven (13.2 per cent), oedematous muscle fibres in five (9.4 per cent) and atrophy in five (9.4 per cent) cases. It was concluded that there was definite involvement of palatal and paratubal muscles in OSMF. This could explain the eustachian tube dysfunction in these patients.

Key words: Oral Submucous Fibrosis; Eustachian Tube; Palatal Muscles

Introduction

Oral submucous fibrosis (OSMF) has been defined as a slowly progressive disease in which fibrous bands form in the oral mucosa leading to severe restriction of movement of the jaws including the tongue.[1] The aetiology of the disease is still obscure. Chronic irritation is thought to be the underlying cause in the pathogenesis of OSMF. The common irritants are betelnut, tobacco, chillies, 'MISI', clay chewing and alcohol.[2–7] The common sites of involvement are the mucosa and submucosa of the soft palate, anterior faucial pillars, buccal mucosa, gums, tongue and lips. Other sites include oropharynx and oesophagus.[8,9]

Histologically, Pindborg and Sirsat[10] described four consecutive stages depending upon hyalinization, fibroblastic response and inflammation. Paymaster in 1956[11] first postulated the pre-cancerous nature of OSMF and described the development of slow-growing squamous cell carcinomata in one third of OSMF cases. Many other investigators have reported the association of OSMF with oral epithelial dysplasia and cancer.[12]

Adwani suggested the involvement of the muscles in the process of fibrosis in 1982.[13] Binnie and Cawson[14] stated that the characteristic feature of OSMF as seen under the light microscope was a homogenous, collaginous sub-epithelial zone in which there were degenerating muscle fibres. El-Labban and Canniff[15] in their study demonstrated the ultrastructural findings of muscle degeneration in OSMF by taking punch biopsies from buccal mucosa.

Rao[16] noticed pain in the ears and conductive deafness in 15 out of 46 cases (32.6 per cent) and suggested that these symptoms were due to pharyngeal fibrosis causing occlusion of the eustachian tubes. Bhonsle[17] reported that the density of the fibrous deposit varied from a slight whitish area on the soft palate to a dense fibrosis causing fixation and shortening or deviation of the uvula and the soft palate.

The main muscles attached to the eustachian tube and the soft palate are the tensor veli palatini and levator veli palatini. These two muscles and the other accessory muscles are referred to as palatal/paratubal muscles. The cartilaginous portion of the eustachian tube and its musculature is a dynamic organ and its ventilatory function and patency may be impaired by muscle dysfunction or a disease process that encroach upon its pharyngeal orifice and the lumen.

Thus the palatal/paratubal muscles may be involved in the process of fibrosis in patients with OSMF. Hence, the present work was planned to study the involvement of muscles in relation to the eustachian tube in patients with OSMF.

Materials and methods

The study was conducted in the department of ENT, MLN Medical College and SRN Hospital, Allahabad, India on 53 patients diagnosed with OSMF on clinical grounds. Biopsies were taken from the soft palate under local anaesthesia to confirm the diagnosis histologically and to study the changes in the palatal/

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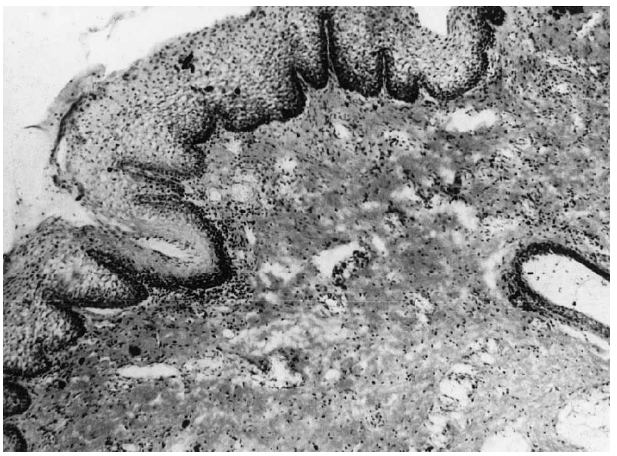

FIG. 1

Stratified squamous epithelium with dense fibrosis in the subepithelial zone (Van-Gieson's staining; ×75).

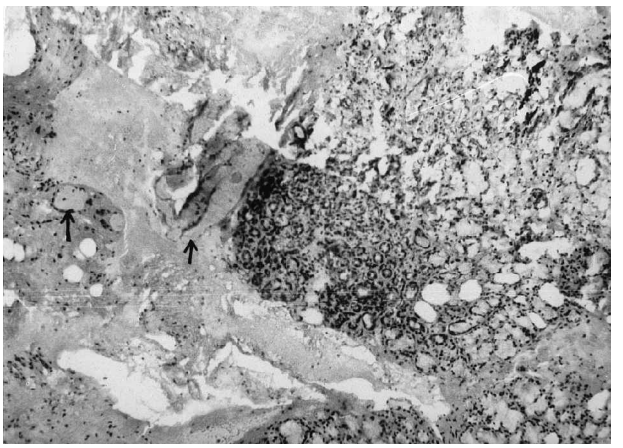

FIG. 3

Degenerated muscle fibres (arrow) with inflammatory infiltrate and fibrous tissue (H&E; ×750).

paratubal muscles. Elliptical incisions about 0.5 to 1 cm, were made and two specimens taken, one from the mucosa and submucosa and another from deeper tissue to include the muscle layer.

The biopsy specimens were preserved in Boin's solution which was formed by mixing a saturated aqueous solution of picric acid (75 ml), 40 per cent formaldehyde (25 ml) and glacial acetic acid (5 ml). Boin's solution was used as the fixative. It penetrated rapidly and evenly and prevented tissue shrinkage.

After routine processing, 3–5 μm sections were cut from paraffin wax embedded tissue and stained with haematoxylin and eosin and Van-Gieson's stain. Light microscopy was used to study the histological changes.

Results

The covering epithelium was atrophic in 20 out of 53 specimens. A subepithelial deposition of amorphous material that stained with specific collagen stain (Van Gieson's stain) was seen in each case (Figure 1). All specimens revealed fibrosis which was mild in 11 (20.7 per cent) cases, moderate in 37 (69.8 per cent) and dense in five (9.4 per cent) cases. Thickened epithelium with deep invaginations of epithelial pegs into lamina propria was seen in many of the sections. Chronic inflammatory cells were seen in all cases, mild in 10 (18.8 per cent) and moderate in the rest of the cases. Vascularity was mild in 13.2 per cent and moderate in 86.7 per cent of cases. Dysplastic changes were seen in seven cases only. Out of these, three revealed severe dysplasia. Areas of acanthosis and parakeratosis were also seen. Few sections showed minor salivary gland tissue surrounded by fibrotic tissue. In some sections the minor salivary gland tissue was found encroaching the epithelium because of dense fibrosis and many times no subepithelial zone was seen (Figure 2).

Degenerative changes in the muscle fibres were seen in 11 (20.8 per cent) cases (Figures 2 and 3). Most of these changes were in the form of loss of cross striations (Figure 4). Other degenerative changes were oedema in the muscle fibres surrounded by inflammatory cells in five (9.4 per cent) (Figure 5) and atrophy of muscle fibres in five (9.4 per cent) cases.

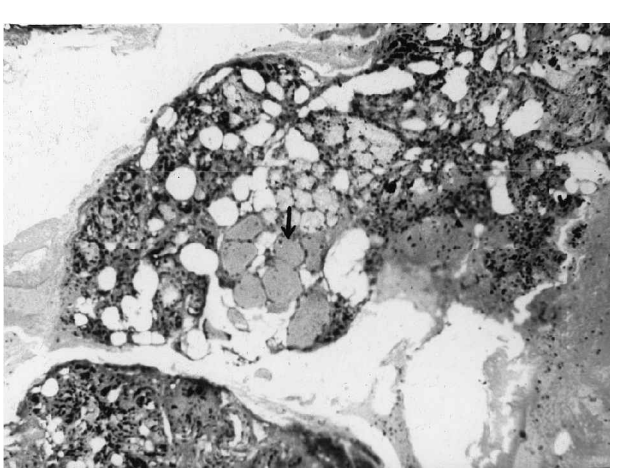

FIG. 2

Degenerated muscle fibres (arrow) surrounded by fatty tissue and minor salivary gland tissue with no evidence of subepithelial zone (H&E; ×75).

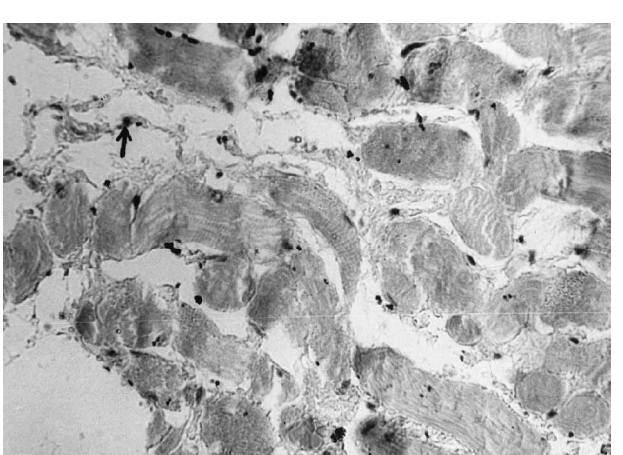

FIG. 4

Fragmented and degenerated muscle fibres with loss of cross striations and thin fibrous strands (arrow) in between the muscle fibres (H&E; ×750).

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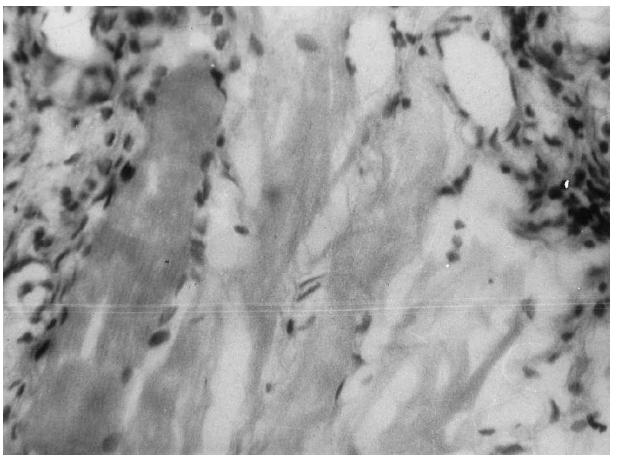

FIG. 5

Oedematous muscle fibres surrounded by inflammatory cells (H&E; ×750).

Discussion

Many studies have been conducted on OSMF in India and abroad during the past few years. The disease is fairly prevalent throughout the Indian subcontinent in all segments of population irrespective of caste, creed, age or socioeconomic status. The disease starts with vesicle formation and juxta-epithelial inflammatory reaction followed by fibro-elastotic changes of the lamina propria and epithelial atrophy leading to stiffness of the oral mucosa causing trismus and difficulty in eating.[3,18–20]

There are several studies available regarding histopathological features in OSMF that mainly deal with the changes that occur in the connective tissues. Su reported acanthosis and parakeratosis.[2] Sirsat and Khanolkar noticed a marked increase of dense collagen in the submucosa and notable hyaline degeneration in the connective tissue.[21] A marked thickening and acanthosis of the epithelium was also reported.[3] Sharan observed hypertrophy of epithelium with 'occasional areas of atrophy' and considerable liquefactive degeneration in the basal cell layer.[22] In contrast to these findings, Wahi *et al*.[23] noticed hyperplasia or atypical epithelial hyperplasia in 102 out of 104 cases with only one case showing atrophic epithelium. They also noticed keratinizing metaplasia in all the cases, an increased mitotic activity in the advanced lesions and squamous cell carcinoma in four cases. Mani and Singh[24] noticed a tendency towards epithelial atrophy associated with hyperorthokeratosis and pyknotic changes in the nuclei of the basal cell layer. Hyperplasia of the epithelium associated with hyperparakeratosis was also noticed. In the present study we observed atrophic epithelium in 20 out of 53 patients while in many other sections areas of acanthosis and parakeratosis were also seen.

However, only few authors have reported muscle degeneration in their study, they took punch biopsies from the buccal mucosa and reported changes in the buccal muscle layer.[14,15] In their electron microscopic study, El-Labban and Canniff[15] compared the ultrastructural changes in muscle fibres in patients with relatively normal and severely restricted mouth opening in OSMF patients. They found that the tissues from patients with restricted mouth opening showed severe degenerative changes in a high proportion of muscle fibres. These fibres contained large pools of homogenous material. Muscle cells or fibres exhibiting complete loss of their plasma membrane were also found. These muscle fibres were often surrounded by oedematous fluid.

In our study, biopsy was taken from the soft palate to include the palatal/paratubal muscles. We found muscle fibres in various stages of the degenerative process. Of these, the most striking were atrophy of the muscle fibres (9.4 per cent), loss of cross striations (13.2 per cent) (Figure 4) and oedematous infiltration of the muscle fibres in 9.4 per cent cases (Figure 5). To the best of our knowledge this is the only study which has focused its attention on the involvement of palatal and paratubal muscles in the fibrosis process in patients with OSMF.

In an interesting study, Tomoda *et al*.[25] concluded that with advancing age the muscle fibres of the human eustachian tube were prone to atrophy. Their observations suggested that the tensor palati produces rapid opening of the eustachian tube while the levator palati creates tension and dilates the pharyngeal orifice of the tube and both these functions deteriorate as muscles atrophy with age. Also the cartilaginous portion of the eustachian tube exhibits a valve-like action important in ventilating the middle ear. Dysfunction of the cartilaginous part due to any pathology in the nasopharynx is a causal factor in middle-ear pathology.

Oral submucous fibrosis is predominantly a disease of the oral cavity and oropharynx. However, fibrosis may extend into the nasopharynx and involve the pharyngeal orifice and the muscles attached to the cartilaginous portion of the eustachian tube. Since the palatal/paratubal muscles are attached to the soft palate, their involvement in the process of fibrosis causes malfunctioning of the eustachian tube.

The present study proves conclusively that there is definite involvement of palatal and paratubal muscles in 20.8 per cent cases of OSMF causing eustachian tube dysfunction in these cases.

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