Case Report

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Reactive Fibrous Hyperplasia of Buccal Mucosa in a Nine Year Old Child

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ABSTRACT

Background: Oral cavity is an ideal niche for manifestation of various neoplastic or non neoplastic lesions. However of these, isolated non neoplastic soft tissue enlargements are usually inflammatory or reactive hyperplastic responses to local irritation or low grade injury. Diagnoses of such enlargements have always been a challenge to dental practitioners. Such lesions present as firm, nodular, sessile or pedunculated, non lobulated, smooth textured, with similar colour of mucosa, symptomatic or asymptomatic mass located on the buccal mucosa, tongue and lip mucosa along the line of occlusion. It is usually seen more commonly in fourth to sixth decade of life and occurrence in paediatric patients is extremely uncommon.

This report presents a very rare case of reactive fibrous hyperplasia of buccal mucosa in a nine year old male child with literature review related to etiology, clinical characteristics, differential diagnosis, complications and management.

Keywords: Fibrous, Hyperplasia, Inflammatory, Reactive, Buccal mucosa.

INTRODUCTION

Oral cavity is an ideal niche for manifestation of various neoplastic or non neoplastic lesions arising either from soft or hard tissue of oral cavity. One such manifestation of soft tissue lesions is the soft tissue tumour. Intraoral soft tissue tumour like lesion is said to be a pathologic growth that projects above the normal contour of the oral surface. The most common mechanism included reactive hyperplasia and neoplasia. Majority of localized overgrowths of the oral mucosa are considered to be reactive to traumatic irritants rather than neoplastic in nature.[1] The traumatic irritants include calculi, overhanging margins, restorations, foreign bodies, chronic biting, margins of caries and sharp spicules of bones and overextended boarders of appliances.[2] In certain instances however, unusual findings may leave dental practitioners with certain diagnostic uncertainty.[3] The localised lesions of the oral cavity have been broadly categorised as: Irritation fibroma, Peripheral ossifying fibroma, Squamous papilloma, Giant cell fibroma, Pyogenic granuloma and Peripheral giant cell granuloma.[4] The fibroma is the most common oral fibrous tumour like growth. Most, if not all fibromas represent reactive focal fibrous hyperplasia due to trauma or local irritation.[5] Fibrous hyperplasia (traumatic or irritation fibroma) is the healed end product of the inflammatory hyperplastic lesion. Clinically they appear either as pedunculated or sessile growth on any surface of the mucous membrane. The majority of such lesions are small and rarely larger than one centimetre. Recurrence of such lesion is mostly as a

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result of failure to eliminate the chronic irritation completely and do not have malignant potential.[6]

Most of the reported cases of reactive fibrous hyperplasia of oral mucosa were in adults, with an age range from 4th to 6th decade of life with very few being reported in paediatric age group.[7] It is extremely rare during the first decade of life.[7,8] The Irritation Fibroma has a 66% female predilection and can occur at any age, however it is usually biopsied in the fourth through sixth decades of life. Some authors suggest that there is no generalized sex predilection to females.[9] In every case, two contributory factors were proposed: The first one is relatively prominent soft tissue along the line of occlusion in oral cavity, that is frequently encountered with various chronic stimuli like calculi, overhanging margins of restorations, foreign bodies, chronic biting and sharp spicules of bones and overextended boarders of appliances placed inside mouths.[2] Secondly, continuous irritation to these tissues resulting in a chronic repair process that include granulation tissue and scar formation resulting in a fibrous submucosal mass.[9] The present paper reports here a case of reactive fibrous hyperplasia in a male child in his first decade of life that is very rare finding.

CASE REPORT

A 9 year old male child reported to Department of Pedodontics and Preventive Dentistry with a chief complaint of a large intra-oral painless swelling on the right side cheek. Child's father gave history of appearance of a small nodular intraoral swelling around 10 months back that gradually increased to present size. Overlying cheek mucosa and skin was normal and swelling was nontender [Figure-1a]. Gross clinical intra-oral examination of the lesion revealed presence of an ovoid, smooth textured, sessile, non-ulcerated, non-tender, non-lobulated soft tissue mass of approximately 10×5×8 mm size, located between the maxillary and mandibular first molar regions on buccal mucosa. The lesion was freely mobile and firm in consistency. Colour of overlying mucosa of lesion was similar to adjacent buccal mucosa. Complete haematological investigations with bleeding time and clotting time were done that revealed values within normal range.

Differential Diagnosis: Depending on clinical features, various similar clinical conditions can be diagnosed differentially those are observed in oral cavity. These conditions include traumatic fibroma, pyogenic granuloma, fibrous hyperplasia, focal papilloma, lypoma, haemangioma, lymphangioma, inflammatory pseudotumour, infected benign minor salivary gland tumour, foreign body granuloma and traumatic neuroma. Final diagnosis is provided by histopathologic analysis, although clinical aspects are characteristic of the lesion.[10]

TREATMENT

The child was prescribed antibiotics, analgesic and antiseptic mouthwash a day prior to procedure to reduce post operative pain and complications while healing. The patient was co-operative and slightly anxious about the procedure. The technique of complete surgical removal of the lesions under local anaesthesia was selected among the possible therapeutic options, and surgery was performed at the outpatient department with no need of hospitalization. Profuse anaesthesia to the operative site was achieved by infiltration local anaesthesia (2% lignocaine, with 1:80,000 adrenaline) around the lesions, using short half inch needle mounted on an aspirating syringe. The base of the lesions was clamped with toothed tissue forcep and complete mass was excised in-toto with a no. 15 scalpel blade from base of the buccal mucosa. The free edges of the wound were sutured back with 4-0 chromic catgut [Figure 1b]. The parotid duct papilla was identified and saliva was expressed by digital manoeuvre, confirming that it is not severed. The excised mass was sent for histo-pathological examination to the Department of Oral Pathology [Figure 2a]. Patient was recalled after 1 week and 3 months for follow up of wound healing. Postoperative care with prescription of antibiotics, pain relievers and antiseptic mouth wash was continued for one week. The patients reported back to the department after 7 days for clinical follow-up and evaluation of the healing process. Manoeuvre to express saliva from Stensen's duct was repeated to make sure that it was not damaged [Figure 2b].

Macroscopic examination revealed a pink, ovoid, firm, smooth surfaced, sessile, non-ulcerated soft tissue mass of approximately 10×5×8 mm size [Figure 2a]. Histological examination of the tissue

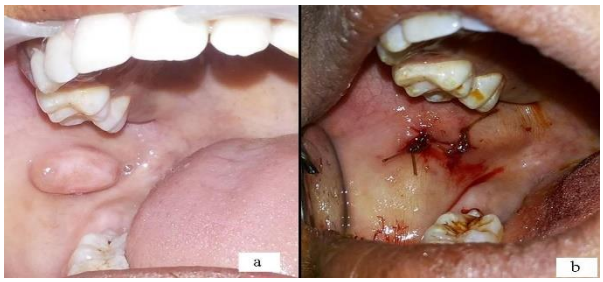

Fig 1a: Intra oral soft tissue mass on buccal mucosa.

Fig 1b: Complete excision of soft tissue mass with sutured wound.

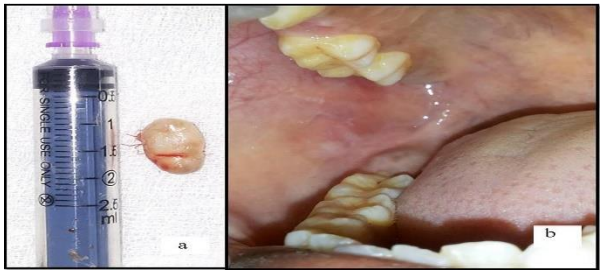

Fig 2a: Excised soft tissue mass from buccal mucosa.

Fig 2b – Healed wound after 1 week.

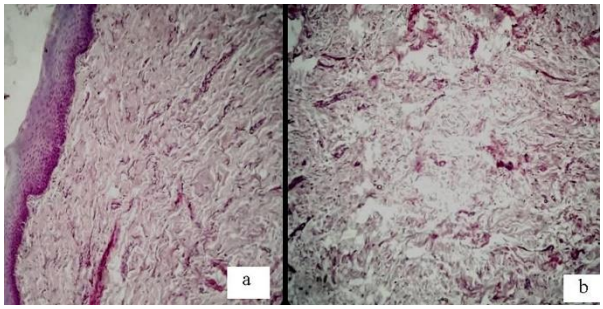

Fig 3a & 3b – Histo-pathological photomicrograph of reactive fibrous hyperplasia; Haematoxylin and Eosin stain (×10 view)

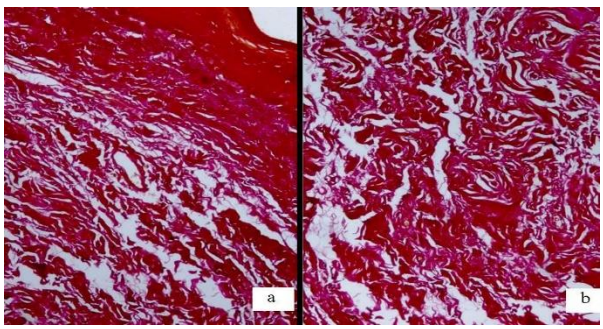

Fig 4a & 4b: Histo-pathological photomicrograph of reactive fibrous hyperplasia; Von Giesson stain (×10 view)

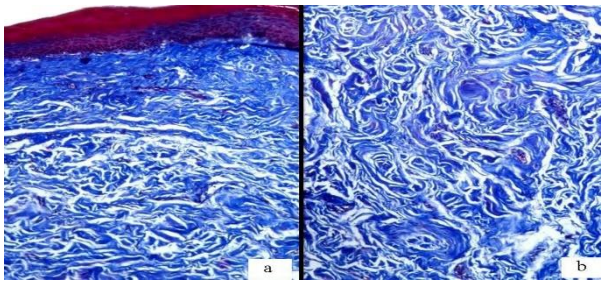

Fig 5a & 5b: Histo-pathological photomicrograph of reactive fibrous hyperplasia; Masson's Trichrome stain (×10 view)

section as stained by Haematoxylin and Eosin stain (x10 view) showed stratified squamous epithelium and underlying connective tissue stroma. The overlying epithelium was atrophic stretched parakeratinised and non-ulcerated. There was shortening of rete pegs and epithelium connective tissue interphase was smooth [Figure 3a]. The connective tissue stroma consisted of minimal oedema, dense collagen bundles with no cellular atypia, matured granulation tissue formation and moderately dense inflammatory (lymphoplasmocytic) infiltrate. Compressed blood vessels engorged with RBCs were also evident in connective tissue stroma [Figure 3b]. Tissue section as stained by Von Giesson (x10 view), [Figure 4a & 4b] and Masson's Trichrome (x10 view), [Figure 5a & 5b] stain showed parallely arranged, densely packed strands of collagen with interlacing arrangement of collagen bundles. Spindle shaped fibroblast with wavy nuclei and thick arrangement of mature collagen was also evident. The overall histopathologic features were confirmatory of reactive fibrous hyperplasia (mature type) of buccal mucosa.

On recall appointment and follow up after one month, healed buccal mucosa at the site of lesion was observed with no signs of recurrence. Digital manoeuvre of Stensen's duct was performed successfully with no signs of duct damage.

DISCUSSION

Intraoral localized overgrowths of oral mucosa most of the time are focal reactive fibrous hyperplasia due to chronic low grade local irritation. However an interesting point to note is that, such lesions are neoplasia of connective tissue origin and microscopically similar to inflammatory

hyperplasia. Hyperplasia is a self limiting process unlike neoplasia and hyperplastic cells sometimes show regression after removal of the stimulus. Neoplastic tissues sometimes resemble that of hyperplastic tissue that do not regress. Hence it is said that neoplasms can occur from the chronic irritation.[2,6] Reactive fibrous hyperplasias of oral mucosa are asymptomatic lesions found more frequently on the buccal mucosa, between the fourth to sixth decade of life. They may present a smooth or nodular appearance, firm to hard consistency and sessile or pedunculated base. Colour may be similar to mucosa or vary depending on extent of inflammation, measuring up to 2 cm in diameter and may display slow growth due to low mitotic index.[6] Such lesions are often encapsulated, usually well delimited and do not produce metastasis. Microscopically, reactive fibrous hyperplasia appear as a nodular mass of fibrous connective tissue with mature or immature collagen fibres mixed with fibroblasts and varying degree of inflammatory cell infiltrate of different cell lineage. Epithelium shows presence of stratified squamous parakeratinised cells well separated from connective tissue stroma.[11]

Reactive fibrous hyperplasia is considered to be a lesion of connective tissue origin, characterized by proliferation of fibroblasts and deposition of collagen fibres in short and cluttered beams, considered by some authors as a true neoplasm.[9] The case presented here revealed an ovoid smooth surfaced, sessile non tender, non lobulated and firm mass, suggesting clinical diagnosis of reactive fibrous hyperplasia of buccal mucosa due to traumatic origin that was confirmed histologically.

Two etiologic factors and pathogenic links between soft tissue trauma and reactive fibrous hyperplastic growth had been discussed in the literature. The first possibility is the direct trauma to connective tissue that causes rupture of vasculature and hematoma formation having a relatively short natural history of presentation termed as “traumatic hematoma” and commonly occurs in late adulthood.[7] The second possibility suggests reactive fibrous hyperplasia formation is due to deposition and differentiation of undifferentiated mesenchymal cells and inflammatory infiltrate to fibroblastic cells with intense collagen deposition, secondary to trauma and hematoma formation. Such lesions are termed “post-traumatic reactive fibrous hyperplasia”.[8] The average time between soft tissue trauma and reactive fibrous hyperplasia formation is almost 6 months to 1 year and occurs more commonly in the fourth to sixth decade. However large lesions of such type are very rare to occur in first decade of life.[8] Clinically such lesions appear as broad based, light in colour than the surrounding normal tissue, with the surface often appearing white due to hyperkeratosis or ulcerated surface due to secondary trauma. The growth potential of reactive fibrous hyperplasia does not exceed 10-20 mm in diameter.[9]

The general literatures have cited the reasons for a few of the oral lesions like irritation fibroma arising as a result of oral habits such as lip biting / sucking. Atypical lesions can occur in other regions of oral cavity like tongue due to improperly fitting orthodontic appliances.[12] Reactive fibrous hyperplasia has been also associated with oral practices like tongue piercing.[13] Rare association of reactive fibrous hyperplasia with a natal tooth in a four year and six month old child have also been reported showing association of local irritants as a major causes of these lesions.[14]

Several modalities of treatment for intraoral lesions are available, including marsupialisation, CO2 and Er,Cr:YSGG laser ablation,[15,16] cryotherapy and surgical enucleation.[11] The surgical enucleation is the most widely used technique for definitive removal of the lesion, with a wide safety margin to prevent recurrence.[17] This treatment is simple, inexpensive and can be performed under local anaesthesia with excellent prognosis and no recurrence. In Paediatric Dentistry, the procedure can be done using behaviour management techniques, which are relatively well tolerated by children.

CONCLUSION

It can be concluded from above discussion that the chronic low grade irritation injuries of soft tissues of oral cavity give rise to lesions that pose challenge to dental practitioners for correct diagnosis and decide proper treatment plan. Proper evaluation of such lesions or enlargements is important before coming to final diagnosis and

treatment modality. While excising such a reactive fibrous hyperplastic lesions and closing the wound care should be taken to avoid injury to Stensen's duct and parotid papilla.

CONFLICTS OF INTEREST

There are no conflicts of interest declared.

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