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Tissue Reactive Hyperplastic Lesions of Oral Mucosa: A 10 Years Retrospective Study and Literature Review

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ABSTRACT

Objective: Tissue Reactive hyperplastic lesions (TRHL) are reactions to local injury of the oral mucous membrane. The cause varies from mechanical injury to hormonal imbalance. Since they closely resemble each other in clinical as well as histological picture thus confusing the examiner all the more.

Materials and Method: The four commonly found reactive lesions are Focal fibrous hyperplasia (FFH), Pyogenic granuloma (PG), Peripheral ossifying fibroma (POF), Peripheral giant cell granuloma (PGCG). The present study reviewed 238 cases, over a period of 10 years focussed on these four lesions. The clinical and histopathological records were assessed and documented. Any incomplete record was discarded. The records were categorised based on prevalence of jaw distribution, age, size and gender. Statistical analysis was done using chi square test and corelation was assessed using Pearsons's chi square ratio.

Results: The fibrous hyperplastic lesion was most prevalent lesion followed by Pyogenic granuloma, Peripheral ossifying fibroma and least was Peripheral giant cell granuloma. The most common site is buccal mucosa and gingiva. The age group which is most susceptible is from 3rd decade to 5th decade.

Conclusion: The lesions are difficult to identify and recurrence rate often leaves the patient and operator troubled. Hence thorough knowledge and understanding of cause and manifestation in particular is warranted for proper diagnosis and treatment.

Keywords: *Reactive hyperplastic lesions, Focal fibrous hyperplasia, Pyogenic granuloma, Peripheral ossifying fibroma, Peripheral giant cell granuloma*

1. INTRODUCTION

The pathologies observed in the oral tissues have been diverse and includes both soft tissue as well as hard tissue. This also includes reactive lesions caused from irritation, inflammation, developmental disorders and neoplastic conditions which include benign or malignant lesions.[1] These lesions are primarily comprised of inflammatory hyperplasia and the clinical features are quite overlapping and confusing making the distinguishing all the more difficult.[2] It was postulated that these reactive lesions cause an irritation to connective tissue leading to inflammatory hyperplasia.[3] The close imitation of these lesions to each other, pose difficulty in accurate diagnosis and treatment planning. Thus, an elaborate knowledge of clinico-histopathological features of each and every entity is required.

Tissue Reactive hyperplastic lesion (TRHL) is a term which includes entities such as traumatic fibroma, pyogenic granuloma (including pregnancy tumor), epulis fissuratum, peripheral giant cell granuloma and peripheral ossifying fibroma. The clinical features of these entities overlap resulting in confusion. They vary from sessile to pedunculated growths, smooth shiny surface or irregular surfaces with superimposed injury, colour may vary from pink to red with varied consistency.[4] Normally the duration of such lesion is long because of slow growth over a period of time; this results in ulceration on the surface due to trauma.[5] This change in clinical appearance resembles neoplastic proliferations, thus posing difficulty in identification. The connective tissue components involved in the lesions and their presence and alteration helps them differentiate histologically when combined with clinical picture.

The histological classification of TRHL has been described previously. Kfir et al[6] had proposed a histological classification of TRHLs: "Angiomatous hyperplasia (AH), Focal fibrous hyperplasia (FFH), Peripheral ossifying fibroma (POF), Peripheral giant cell granuloma (PGCG)." Neville had classified the reactive lesions in four categories which has been widely accepted. It includes:[7] 1. Focal fibrous hyperplasia (FFH) 2. Pyogenic granuloma (PG) 3. Peripheral ossifying fibroma (POF) 4. Peripheral giant cell granuloma (PGCG)

FFH reports to have a prevalence of 56-61% as reported in the literature earning the repute of most common reactive lesion followed by PG (19-27%). The next prevalent lesion was POF (10-18%) and PGCG (1.5 – 7%).[5,8] Since the lesions provide with similar yet unique clinical and histopathological features pertaining to each entity, early detection and identification will result in better diagnosis, adequate treatment and less complications. The present literature was assimilated to identify the distribution of these lesions and to review the identification characteristics.

2. METHOD

The archived records from department of oral medicine and radiology and oral pathology from past 10 years were assessed. A detailed case history of description of the lesion and information availability about the age, gender of the patient, affected jaw and site was considered for be included in the study. The histological findings were also taken into account to establish the diagnosis. The records were correlated clinically and histopathologically. In case of missing of the either, the records were excluded. The complete records included were then categorised as per the occurrence of the lesion into jaw distribution, site distribution, age distribution, and gender distribution. Jaw distribution was into maxillary and mandibular, site distribution was done into labial mucosa, buccal mucosa, gingiva and palate, age distribution was classified into decades of 10-19 years, 20-29 years, 30-39 years, 40-49 years, 50-59 years and 60 years & above, gender distribution was done as male and female. The data was tabulated and descriptive statistics were applied using SPSS version 25 (IBM, Statistical package for Social Sciences). Results were expressed in terms of frequencies and percentages using Chi square test, Pearson coefficient, likelihood ratio and linear by linear association.

3. RESULTS

The lesions were classified into fibroma, pyogenic granuloma, peripheral ossifying fibroma and peripheral giant cell granuloma. Frequency and percentage distribution were expressed for each category; as per observed on the jaw: maxillary and mandibular, age: as observed in each decade starting from 10 years to 60 and above, site: labial and buccal mucosa, gingiva and palate and gender: male or female. Of the 238 lesions included fibroma was observed in highest percentage (55.5%), followed by pyogenic granuloma (33.6%), POF (8%) and PGCG (2.9%). The lesions were seen more in mandible than in maxillary. The 30-39 and 40-49 years of age were most prone to develop an hyperreactive lesion. PGCG has shown an inkling towards older age group. Fibroma were prevalent on buccal mucosa while PG was seen on gingiva and was mostly seen in females. Fibroma on the other hand was observed in males. POF was also prevalent in males and at buccal mucosa. (Table 1). A non-linear association was observed for all the lesions as seen in Pearson chi square ratio and likelihood ratio.

Table 1: Table showing frequency and percentage distribution of tissue hyperplastic lesion

	Count (% within)	Type				Pearson Chi-Square	Likelihood Ratio	Linear-by-Linear Association
		Fibroma	PG	POF	PGCG			
	Total	132(55.5%)	80 (33.6%)	19 (8.0%)	7 (2.9%)			
Jaw distribution	Maxillary	52 (47.7%)	38 (34.9%)	15 (13.8%)	4 (3.7%)	.011	.010	.005
	Mandibular	80 (62.0%)	42 (32.6%)	4 (3.1%)	3 (2.3%)			
Age distribution	10-19 years	10 (47.6%)	6 (28.6%)	4 (19.0%)	1 (4.8%)	.135	.170	.282
	20-29 years	22 (42.3%)	24 (46.2%)	5 (9.6%)	1 (1.9%)			
	30-39 years	44 (60.3%)	24 (32.9%)	4 (5.5%)	1 (1.4%)			
	40-49 years	32 (68.1%)	12 (25.5%)	2 (4.3%)	1 (2.1%)			
	50-59 years	17 (53.1%)	8 (25.0%)	4 (12.5%)	3 (9.4%)			
	60+ years	7 (53.8%)	6 (46.2%)	0 (0.0%)	0 (0.0%)			
Site distribution	Labial mucosa	33 (86.8%)	5 (13.2%)	0 (0.0%)	0 (0.0%)	.000	.000	.000
	Buccal mucosa	64 (71.1%)	10 (11.1%)	12 (13.3%)	4 (4.4%)			
	Gingiva	20 (29.9%)	40 (59.7%)	4 (6.0%)	3 (4.5%)			
	Palate	15 (34.9%)	25 (58.1%)	3 (7.0%)	0 (0.0%)			
Gender distribution	Male	72 (61.5%)	30 (25.6%)	12 (10.3%)	3 (2.6%)	.058	.056	.365
	Female	60 (49.6%)	50 (41.3%)	7 (5.8%)	4 (3.3%)			

Discussion

The various tissue hyper reactive lesions have been a puzzle to all the specialities alike; they are similar in appearance and often puzzling. The chairside diagnosis often become difficult as the histological examination becomes necessary in order to confirm their origin and features. The present study attempted to explain the differentiating features to make clinical diagnosis a bit easy. The samples obtained were over a period of 10 years to assess the prevalence in which the fibroma or fibrous hyperplasia was found to be maximum and PGCG was minimum in distribution. The following differentiating characteristics were found in the histological examination. (Table 2)

Table 2: Table summarizing clinical and histological features of tissue reactive hyperplastic lesion

REACTIVE LESIONS	FFH	PG	POF	PGCG
AGE (Decades)	4^{th} to 6^{th}	1^{st} to 2^{nd}	2^{nd} to 3^{rd}	3^{rd} to 6^{th}
SEX	Females	Females	Females	Females
SITE	Buccal Mucosa Followed by gingiva	Anterior facial maxillary gingiva	Anterior maxilla (interdental papillae)	Mandibular gingiva
SIZE	Milimeter to Centimeter	Milimeter to Centimeter	Less than 2 Centimeter	Less than 2 Centimeter
COLOUR	Pink	Bright red	Red pink	Blue purple
SURFACE	Smooth	Smooth Lobulated	Smooth Nodular	Smooth Nodular
ULCERATION	In case of Trauma	In case of Trauma	In case of Trauma	May not Present
BASE	Sessile	Pedunculated	Pedunculated/ Sessile	Pedunculated/Sessile
BLEEDING	Less	High	Less	High
RECURENCE	Rare	Frequent	Less Chance (16%)	Less Chance (10%)
HISTOLOGICAL	Fibrous connective tissue	Vascular fibroepithelial tissue	Calcification with cellular stroma	Multinucleated giant cell in stroma

Focal fibrous hyperplasia: Fibroma refers to a benign neoplastic lesion originated from fibrous tissue thus term focal fibrous hyperplasia was adapted[9]; as it gives more clarity about the lesion being a reactive response.[2,6] It usually results from chronic repair of granulomatous tissue and scar formation causing fibrous mass in submucosa.[10] It usually appears as raised sessile or pedunculated mass with a smooth surface, firm in consistency, asymptomatic with varying sizes. The colour of the lesion is usually pink; however, changes in consistency and surface may be seen in case of inflammation or trauma. Buccal mucosa was seen to be most commonly affected area in the present study and in the age range of third to fourth decade which has been backed up in previous study.[11]

The histological examination of the lesion revealed nodular or encapsulated, fibrous connective tissue with vascularity, immature and mature collagen fibres are present interspersed with fibroblasts and varying percentage of cell inflammatory infiltrate. Stratified squamous epithelium is present on the surface, with parakeratinized cells; epithelial atrophy may be seen in case of trauma.[2,12,13] Treatment of the lesion consists of excision and removal of source. Recurrence rates are slow if properly excised.[12]

Pyogenic Granuloma: Hullihen first reported pyogenic granuloma in 1844. Poncet and Dor reported the same and named it as "botryomycosis hominis" which was later changed to pyogenic granuloma or granuloma pyogenicum by Hartzell in 1904.[14] The lesion does not suppurate and not show histological features of granuloma thus it is a misnomer.[7] Clinical features of PG include smooth or lobulated lesion, sessile or pedunculated and colour showing a dominant reddish hue on the gingiva with sizes varying from few millimetres to few centimetres. The lesion is asymptomatic; surface may become ulcerated if affected by secondary trauma. This happens in cases where the lesion grows so as to affect routine functions such as mastication or hygiene.[15] Female predilection has been implied and is attributed to the hormones. The rise in levels of progesterone and oestrogen is the likely cause of proliferation of blood vessels resulting in overgrowth. However, presence of local irritants is observed in cases and the overgrowth is exaggerated in presence of hormones.[15] Daley et al[9] proposed that increase in levels of progesterone and oestrogen results in more susceptibility of inflamed gingival tissue to proliferate in presence of local irritants. The present study has shown male are being affected too in considerable numbers.

Histologically the lesion consists of high vascular proliferation, engorged red blood cells, fibroepithelial stroma with

inflammatory cells such as neutrophils and macrophages. Surface is composed of parakeratinized stratified squamous epithelium. The histological variant may be of lobular capillary hemangioma type which shows small luminal blood vessels in large number. The other is non-lobular capillary hemangioma type (non-LCH) which shows in the centre, perivascular mesenchymal cells non-reactive for α smooth muscle actin and muscle specific actin.[15] The management include complete removal of lesion. Surgical excision along with thorough debridement of underlying epithelium and oral prophylaxis is recommended. In case of pregnant women, it is advised to wait till the full term as it may regress on its own but if not, surgical excision should be planned.[15]

Peripheral ossifying fibroma (POF): Shepherd first reported an entity in 1844 and termed as "alveolar exostosis". Lee proposed "calcifying fibroblastic granuloma" in 1968, however, Eversole and Rovin in 1972 coined the term Peripheral ossifying fibroma.[3,9] The origin of POF is supposed to be from periodontal ligament cells, surrounding periosteum or the interdental papilla; however, the origin is not fully established at present.[11] Clinically the lesion presents itself as painless, nodular mass, sessile or pedunculated, smooth, colour matching the surrounding mucosa, usually presents in papillary region; most common site in present study was buccal mucosa. Consistency of lesion vary from firm, fibrotic to hard depending on calcification.[11] Miller et al specified that immature POF bleeds easily and are soft thus appears like pyogenic granuloma, as the lesion becomes mature, they become firm and fibrotic.[16] The age predilection for the lesion is second to third decade with female predilection which can be due to hormonal influences[17] however our findings were inconclusive in this respect. The underlying bone is not affected but surface erosion can be observed at times. The calcifications in the lesion appears on the radiograph.[17]

Histologically, the surface contains stratified squamous epithelium. The connective tissue shows presence of fibroblasts, cellular stroma, fibrocytes and multinucleated giant cells. Areas of calcification are interspersed with cellular stroma throughout is observed. The calcification shows characteristics of bone, cementum and/or dystrophic calcification.[17] Orikin and Amaidas proposed that chronic irritation may lead to cells of periodontal ligament or periosteum to cause metaplasia, thus causing calcification.[18] Treatment is complete excision along with underlying epithelium, removal of source of irritation. Recurrence rate is high hence complete removal is advised. If the lesion involves surrounding teeth causing displacement or resorption, extraction is indicated.

Peripheral giant cell granuloma: It is a benign lesion, manifesting itself as soft tissue extra osseous nodule. The etiology of the lesion is debated however chronic irritations, extractions, injury to periodontal membrane or periosteum are listed.[11] Regarding the multinucleated nature of the lesion numerous hypotheses have been put forward. Chadwick et al suggested that the giant cells are osteoclasts left from physiological reaction to injury to surrounding structures.[19] Another hypothesis says that giant cells are reactionary component in response to the stimulus and the source of the cells are bone marrow monoclear cells which reach at the site via bloodstream.[20] The clinical features of PGCG includes asymptomatic growth from the deeper tissues, surface may be smooth or ulcerated, sessile or pedunculated. The colour ranges from red to reddish blue owing to high vascularity of the lesion, hence they bleed easily. It has potential for growth hence the size of the lesion varies. The site is usually from an interdental papilla or edentulous ridges; lesion tries to penetrate resulting in displacement of teeth. Mandible is more commonly affected than maxilla with peak incidence at 30-40 years.[11,21] The data obtained were inconclusive of exact statement.

The histological picture of the lesion consists of abundant osteoclast like multinucleated giant cells in a connective tissue stroma. There are numerous plump, spindle shaped fibroblasts with large number of blood vessels and extravasated RBCs. The nuclei of giant cells may be large vesicular or pyknotic. Stratified squamous epithelium is present which can be parakeratinized. There may be presence of mineralized tissue in the form of osteoid.[11,21]Treatment of the lesion is complete excision along with removal of irritation. The recurrence rate is high hence proper debridement is advised. Suture may be given if the area of resection is too big.

The present study tried to explain the differentiating features and explore the incidence of the lesions; however, prevalence was not assessed. The records were assessed based on the four most common hyperplastic lesions; other reactive lesions were not considered. The present literature made an attempt to explain the hyperplastic lesions and their characteristic.

4. CONCLUSION

Oral mucosa puts up with a vast array of reactions including mechanical or irritation injury, hormonal manifestations and iatrogenic trauma. The tissue reactive hyperplastic lesions are results of such trauma. The untreated and unidentified lesions may proliferate, posing dento-alveolar complications. The recurrence of these lesions is another concern. This review tries to solve the enigma of tissue reactive hyperplastic lesions.

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