Gingival and alveolar ridge tumor-like overgrowth lesions

Salwan Y.H. Bede, B.D.S., F.I.B.M.S. (1)

ABSTRACT

Background: Tumor-like overgrowth lesions of the oral mucosa are pathological growths that project above the normal contour of the oral surface. A practical classification can be made according to the site of origin, the etiology and the histological appearance. The aim of this article is to evaluate and analyze patients with gingival and alveolar ridge tumor-like overgrowth lesions in terms of surgical treatment, diagnosis and outcome.

Materials and Methods: Patients complaining of these lesions were treated by surgical excision under local or general anesthesia; the excised lesions were submitted for histopathological examination, during the follow up period the patients were examined for complications and recurrence.

Results: Pyogenic granuloma was the most frequently encountered lesion, followed by peripheral giant cell granuloma, fibrous hyperplasia, peripheral ossifying fibroma and neurofibroma. Complications were minimal and recurrence occurred in one patient.

Conclusion: Gingival and alveolar ridge overgrowths are common being mostly reactive rather than neoplastic in nature, global recurrence rate was 2.1%.

Keywords: Gingival, alveolar ridge, overgrowth. (J Bagh Coll Dentistry 2013; 25(3):110-114).

INTRODUCTION

Tumor-like overgrowth lesions of the oral mucosa are pathological growths that project above the normal contour of the oral surface[1].

A practical classification can be made according to the site of origin of the lesion, on the basis of the etiology and the histological appearance[2, 3].

Histologically these lesions have been classified into; granulomatous, fibromatous and giant cell lesions[4], another histological classification includes; fibrous, vascular and giant cell lesions[1].Different mechanisms can lead to the development of these lesions, most commonly reactive hyperplasia and neoplasia, the majority of tumor-like lesions of the oral mucosa are considered to be reactive rather than neoplastic in nature [1, 5, 6].

Reactive tumor-like overgrowths are common, and they often arise in response to local irritations like defective restorations, dental plaque and calculus, trauma or inflammation, also some lesions are drug induced or associated with systemic diseases and conditions [2, 5].

Gingival overgrowth is also termed Epulis, a term described by Axhausen, which is defined as a lump arising from the gingiva; it is a clinical description where the histological diagnosis is not verified[3, 7].

Common gingival overgrowths include; pyogenic granuloma, peripheral ossifying fibroma, peripheral fibroma (fibrous hyperplasia) and peripheral giant cell granuloma[5, 8].

Treatment of these lesions consists of surgical excision, with curettage of the adjacent dental and osseous tissues and histological analysis of the excised tissues [2,3,5].

(1)Lecturer. Department of Oral and Maxillo-facial Surgery. College of Dentistry, University of Baghdad.

Some of these lesions, although benign, have a tendency to recur especially with incomplete removal of the lesion or of the etiological factors involved[5].

The aim of this article is to evaluate and analyze patients with gingival and alveolar ridge tumor-like overgrowth lesions in terms of surgical treatment, diagnosis and outcome.

MATERIALS AND METHODS

In the period extending from May 2009 to October 2012, 47 patients attended the department of Oral and Maxillofacial surgery in the College of Dentistry, University of Baghdad and the Oral and maxillofacial surgery unit in Al-Yarmook teaching hospital complaining of gingival and/or alveolar ridge lesions, the duration of the lesions ranged from 3 months to 4 years, a thorough history was taken from the patients, a careful clinical and radiographic examination was carried out using Periapical, Occlusal and Orthopantomogram views.

The surgical treatment was carried out under local anesthesia in 42 patients using Lidocain 2% with Adrenaline 1:100.000, while general anesthesia was needed in 5 patients.

The surgical treatment consisted of excision of the whole lesion to the bone, as an excisional biopsy, this was done using surgical blade, and in some cases electrocautery was used with curettage of the underlying bone and the roots of the adjacent teeth. The exposed bone was covered with Iodoform gauze pack which was secured to the area using a tie over black silk suture 3/0, or using periodontal pack. The pack was left in place for 7-10 days and was removed afterwards, in four patients flap advancement and direct suturing was performed. The patients were given Antibiotics (Amoxicillin 500 mg plus

Metronidazole 500 mg three times daily) and Analgesics (Paracetamol 1000 mg as required) and mouth rinses for 7 days postoperatively.

The excised specimens were submitted for histopathological examination. All patients were followed up for a minimum of 3 months, during this period the patients were examined for complications and recurrence.

RESULTS

The study group consisted of 47 patients, 26 females (55.3%) and 21 males (44.7%), the age of the patients ranged from 3-70 years with an average of 41.5 years. The duration of the lesions ranged from 3months to 4 years with an average of 12.25 months. The size of the lesions varied from about 1 cm to about 5 cm in their greatest dimension. In 7 patients, slight resorption of the underlying bone was evident radiographically. Ten patients were diabetics, two were hypertensive and two female patients were pregnant in the third trimester, the lesion was excised after parturition.

The gingiva and the alveolar ridge of the mandible were involved in 28 (59.6%) patients while the remaining 19 (40.4%) patients had maxillary gingival and/or alveolar ridge lesions.

Histopathological examination revealed inflammatory/reactive lesions in 46 (97.9%) of the cases and neoplastic lesion in only one (2.1%) case. In details the diagnoses were pyogenic granuloma (Fig.1) in 20 patients (42.5%), peripheral giant cell granuloma in 19 patients (40.4%), fibrous hyperplasia in 6 patients (12.7%), peripheral ossifying fibroma in one patient (2.1%) and neurofibroma (Fig.2) in one patient (2.1%).

In patients diagnosed with pyogenic granuloma the mandibular gingiva and alveolar ridge mucosa was affected in 9 patients and the maxilla was affected in 11 patients, and females were more affected (13 patients) than males (7 patients).

Peripheral giant cell granuloma affected the mandibular gingiva and alveolar ridge mucosa in 13 patients, whereas the maxilla was affected in 6 patients, and males were more affected (11 patients) than females (8 patients). Fibrous hyperplasia affected the mandible in 4 patients, and affected the maxilla in 2 patient, females were more affected (4 patients) while only 2 males were diagnosed with fibrous hyperplasia. (Table 1) summarizes the results.

No serious complications occurred after surgery other than the temporary inflammatory postoperative reaction, in 4 patients ulceration of the excision area occurred which was due to the early loss of the pack, the ulcers persisted for 2-3 weeks, and those patients were kept on mouth washes until the complete healing of the area.

In one patient diagnosed with peripheral giant cell granuloma recurrence occurred one year after the surgical excision, making the recurrence rate of peripheral giant cell granuloma 5.2% and a global recurrence rate in this study 2.1%. The lesion was re-excised (Fig.3 A, B and C), the laboratory investigations for the patient with recurrent peripheral giant cell granuloma revealed normal serum calcium and phosphorus and parathyroid hormone levels thus excluding hyperparathyroidism.

DISCUSSION

Gingival and alveolar ridge overgrowths are common [2,5,9]. Most of these lesions are inflammatory and reactive in nature rather than neoplastic, it is estimated that about 85% to more than 90% of these lesions are reactive while the remainder is neoplastic in origin[9, 10]. In this paper the reactive lesions mounted for more than 97% of the lesions, while the neoplastic ones were only in 2.1% of the cases

Pyogenic granuloma was the most common lesion encountered in this study; this finding was also seen in other studies [9,10] . In some studies [11, 12], pyogenic granuloma was found to be the second most common lesion in the oral cavity. The term lobular capillary hemangioma was first introduced in 1980 by Mills et al, as an underlying lesion of pyogenic granuloma and now this term is used synonymously with it [13,14]. In this study pyogenic granuloma affected females more than males and maxilla was affected more than the mandible. These findings are in agreement with the findings in another study[15] that analyzed the incidence of pyogenic granuloma in the oral cavity and found also that the most common site of occurrence is the gingiva. A recurrence rate ranging from 5.8%-10% was reported[8, 15], but in this study no recurrence was noticed after the surgical excision during the time of follow up.

Peripheral giant cell granuloma, also known as giant cell epulis, giant cell reparative granuloma, Osteoclastoma or giant cell hyperplasia[16], was the second most common reactive lesion; another study[11] found that this lesion was the most commonly encountered. Studies have demonstrated that females are affected more than males [16-19], but in this study slight male predominance was noticed. Also the mandible was affected more than the maxilla in this study, a finding that was also seen in other studies [18,19]. The recurrence rate of peripheral giant cell granuloma in this study was 5.2%, other

authors reported a recurrence rate that ranges between 1.4%-10% [8, 17].

Fibrous hyperplasia also called fibroma, irritation fibroma, traumatic fibroma or fibrous nodule, is reported to be the most common reactive lesion in the oral cavity [8,12,20], while some authors reported to be the second most common lesions in the oral cavity [9]. in this study, this lesion followed pyogenic granuloma and peripheral giant cell granuloma in prevalence, possibly because this study was limited to gingival and alveolar ridge mucosal lesions, another possible reason is that these lesions occur most commonly along the bite line in the buccal mucosa, and it is likely that they represent a fibrous maturation of a preexisting pyogenic granuloma [8].

The least common reactive lesion in this study was peripheral ossifying fibroma, it was first identified as a lesion by Shephard in 1844 [21] and its current name was given by Eversole and Rovin[22]. It is estimated that it accounts for about 15% of the solitary gingival growths[10]; it is also termed peripheral cemento-ossifying fibroma, peripheral odontogenic fibroma with cementogenesis, peripheral fibroma with osteogenesis, and peripheral fibroma with calcifications[23]. A recurrence rate ranging from 8-20% has been reported with an average time interval of 12 months [21, 22, 24, 25]. Comparison with this study was not possible since only one case was diagnosed as peripheral ossifying fibroma and no recurrence was noticed.

While 97.9% of the lesions were reactive and inflammatory in origin, only one patient (2.1%) in this study was diagnosed with neurofibroma which is neoplastic in origin, it is an uncommon tumor of the oral cavity and seen either as a solitary lesion or as a part of neurofibromatosis[26], it is the most common type of peripheral nerve neoplasms, and the most commonly reported intraoral site is the tongue and the buccal mucosa[8].

Treatment of all these solitary lesions entails surgical excision of the lesion down to the bone with removal of all the causes of local irritation [8, 9,19,27]. Other protocols like the use of electrocautery, Nd:YAG laser, flash lamp pulsed dye laser, cryosurgery, intralesional injection of ethanol or corticosteroids or sodium tetradecyl sulfate sclerotherapy have been proposed, but the literature shows no difference using these modalities [19, 27]. In the current study the traditional method of excision was used as it is readily available and requires no special equipment.

A limitation of this study is the small sample size; this can be attributed to lack of compliance and the loss of patients to follow up, which were excluded. Another reason is that this study is limited to lesions of the gingiva and alveolar ridge mucosa.

REFERENCES

1. Pour MAH, Rad M, Mojtahedi A. A survey of soft tissue tumor like lesions of the oral cavity: A clinicopathological study. Iranian J Pathol 2008; 3(2): 81-7.
2. Ballini A, Scattarella A, Crincoli V, Carlaio RG, Papa F, Perillo L, Romanazzo T, Bux MV, Nardi GM, Dituri A, Cantore S, Pettini F, Grassi FR. Surgical treatment of gingival overgrowth with 10 years follow up. Head Face Medi 2010; 6:19.
3. Seward GR, Harris M, McGowan DA. Killey and Kay's outline of oral surgery, part 1. 2nd ed. Bristol: Wright; 1987. pp: 297-313.
4. Anneroth G, Sigurdson A. Hyperplastic lesions of the gingiva and alveolar mucosa. Acta Odontol Scand 1983; 41(2): 75-86.
5. Rossman JA. Reactive lesions of the gingiva: Diagnosis and treatment options. The Open Pathology J 2011; 5: 23-32.
6. Carvalho Mde V, Iglesias DP, do Nascimento GJ, Sobral AP. Epidemiological study of 534 biopsies of oral mucosal lesions in elderly Brazilian patients. Gerodontology 2011; 28(2): 111-5.
7. Axhausen, G. Allgemeine Chirurgie in der Zahn-Mund- und Kieferheilkunde Hanser, München; 1947.
8. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and Maxillofacial pathology. 2nd ed. Philadelphia: WB Saunders; 2002. pp. 437-495.
9. Stablein MJ, Silverqlade LB. Comparative analysis of biopsy specimens from gingiva and alveolar mucosa. J Periodontol 1985; 56(11): 671-6.
10. Chaturvedia R, Guptab M, Jainc A. Peripheral ossifying fibroma: A case report. Ind J Dent 2012; 3(2): 89-93.
11. Mighetl AJ, Robinson PA, Hume WJ. Peripheral giant cell granuloma: a clinical study of 77 cases from 62 patients, and literature review. Oral diseases 1995; 1(1): 12-9.
12. Awange DO, Wakoli KA, Onyango JF, Chindia ML, Dimba EO, Guthua SW. Reactive localised inflammatory hyperplasia of the oral mucosa. East Afr Med J 2009; 86(2): 79-82.
13. Mills SE, Cooper PH, Fechner RE. Lobular capillary hemangiom: the underlying lesion of pyogenic granuloma: A study of 73 cases from the oral and nasal mucous membranes. Am J Surg Pathol 1980; 4: 470-9.
14. Toida M, Hasegawa T, Watanabe F, Kato K, Makita H, Fujitsuka H, et al. Lobular capillary hemangioma of the oral mucosa: Clinicopathological study of 43 cases with a special reference to immunohistochemical characterization of the vascular elements. Pathol Int 2003; 53: 1-7.
15. Al-Khateeb T, Ababneh K. Oral pyogenic granuloma in Jordanians: a retrospective analysis of 108 cases. J Oral Maxillofac Surg 2003; 61(11): 1285-8.
16. Etoz OA, Demirbas AE, Bulbul M, Akay E. The Peripheral Giant Cell Granuloma in Edentulous Patients: Report of Three Unique Cases. Eur J Dent 2010; 4(3): 329–33.

17. Katsikeris N, Kakarantza-Angelopoulou E, Angelopoulos AP. Peripheral giant cell granuloma. Clinicopathologic study of 224 new cases and review of 956 reported cases. Inter J Oral Maxillofacial Surg 1988; 17(2): 94–9.
18. Motamedi MHK, Eshghyar N, Jafari SM, Lassemi E, Navi F, Abbas FM, Khalifeh S, Eshkevari PS. Peripheral and central giant cell granulomas of the jaws: A demographic study. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology 2007; 103(6): 39–43.
19. Adlakha VK, Chandna P, Rehani U, Rana V, Malik P. Peripheral giant cell granuloma. J Indian Soc Pedod Prev Dent 2010; 28(4): 293-6.
20. Kfir Y, Buchner A, Hansen LS. Reactive lesions of the gingiva. A clinicopathological study of 741 cases. J Periodontol. 1980; 51(11): 655-61.
21. Bhaskar SN, Jacoway JR. Peripheral fibroma and peripheral fibroma with calcification: report of 376 cases. J Am Dent Assoc 1966; 73:1312-20.
22. Eversole LR, Rovin S. Reactive lesions of gingiva. J Oral Pathol 1972; 1: 30-8.
23. Shetty P, Adyanthaya S. Peripheral Ossifying Fibroma- A Clinical and Histological Evaluation of 51 Cases. People's J Sci Res; 2012: 5(1): 9-14.
24. Cuisia ZE, Brannon RB. Peripheral ossifying fibroma-a clinical evaluation of 134 pediatric cases. Pediatr Dent 2001; 23: 245-8.
25. Das UM, Azher U. Peripheral ossifying fibroma. J Indian Soc Pedod Prev Dent 2009; 27(1): 49-51.
26. Richards D. Neurofibroma of the oral cavity. Br J Oral Surg 1983; 21(1): 36-43.
27. Jafarzadeh H, Sanatkhani M, Mohtasham N. Oral pyogenic granuloma: a review. J Oral Sci 2006; 48: 167-75.

Table 1: Distribution of patients according to gender and site of lesions

Diagnosis	Patients (%)		Site/Case (%)	
	Males	Females	Mandibular gingiva and alveolar ridge mucosa	Maxillary gingiva and alveolar ridge mucosa
Pyogenic granuloma	7 (14.9%)	13 (27.7%)	9 (19.2%)	11 (23.4%)
Peripheral giant cell granuloma	11 (23.4%)	8 (17%)	13 (27.7%)	6 (12.8%)
Fibrous hyperplasia	2 (4.2%)	4 (8.5%)	4 (8.5%)	2 (4.2%)
Peripheral ossifying fibroma	1 (2.1%)	0 (0%)	1 (2.1%)	0 (0%)
Neurofibroma	0 (0%)	1 (2.1%)	1 (2.1%)	0 (0%)
Total	**21 (44.7%)**	**26 (55.3%)**	**28 (59.6%)**	**19 (40.4%)**
	47 (100%)		**47 (100%)**	

Fig.1. Pyogenic granuloma

Fig.2. Neurofibroma.

Fig.3. Giant cell granuloma. A: at the time of presentation. B: 4 weeks after surgical excision. C: recurrence one year after excision.