

Localized Reactive Hyperplastic Lesions of the gingiva:A clinico-pathological study of 636 lesions from Iraq

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Abstract

This study was conducted to establish the prevalence of different reactive hyperplastic lesions of the gingiva in a university-based dental school biopsy service.

Biopsy records over a 12-years period (1982-1994) were retrieved from the files of the department of Oral Pathology, College of Dentistry, University of Baghdad. The lesions were reclassified into: Fibrous lesions (like Focal Fibrous Hyperplasia FFH and Ossifying Fibrous Epulis OFE); vascular lesions (like Pyogenic granuloma) and Giant cell lesions (like Peripheral giant cell granuloma PGCG). The available clinical data regarding age, gender and location were obtained for each case.

The majority of lesions were pyogenic granuloma which constituted 49.2% with female predilection. Peoples in the first and second decade of life were more prone to develop gingival lesions especially in upper anterior region.

This study indicates some differences in age and gender distribution as well as in location between the different lesions.

Keywords: pyogenic granuloma, reactive gingival lesions, PGCG, focal fibrous hyperplasia, peripheral ossifying fibroma.

Introduction

Soft tissue enlargement of the oral cavity often presents a diagnostic challenge because a diverse group of pathologic processes can produce such lesions. Within these lesions a group of reactive hyperplasias which develop in response to a chronic, recurring tissue injury that stimulates an exuberant or excessive tissue repair response ⁽¹⁾. The term "epulis" is used clinically to describe any localized overgrowth on the gingiva. Histological examination of epulides indicates the vast majority are:

Focal Fibrous Hyperplasia (FFH), Peripheral Ossifying Fibroma (POF), Pyogenic Granuloma (PG) or Granuloma Peripheral Giant Cell $(PGCG)^{(2,5)}$. Their histopathological features are quite distinct but considerable overlap still exists among these lesions. Eversole and Rovin (3)speculated that the different histological entities of reactive hyperplasia may be due to connective tissue response to varied intensities of gingival irritation. This response may be influenced by the serum levels of certain endocrine hormones. Many of these lesions can be identified as specific entities on the basis of their histopathological features and are divided into fibrous, vascular and giant cell type ⁽⁴⁾. The present study reviews the clinical and hitopathological features of four main types of localized reactive lesions of the gingival and establishes the relative prevalence of the different biopsied gingival lesions in relation to age, gender and site distribution.

Materials and method

A search through 4027 consecutive biopsies submitted to the Oral Pathology Department, College of Dentistry, Baghdad University, Iraq over a twelve years period (from1982 till the end of 1994) yielded a total of 636 lesions which could be categorized as localized reactive hyperplastic gingival lesions.

The microscopic sections were reviewed and histological diagnosis was made according to the criteria of Daley et al ⁽⁵⁾. The available clinical data on the location of the lesion, age, and gender of the affected patients at the time of surgical excision were recorded and tabulated. The lesions were classified into: *Fibrous lesions* which predominantly comprised of collagenous connective tissue, this include: Focal Fibrous Hyperplasia (FFH) and Ossifying Fibrous Epulis (OFE).

- Vascular lesions which predominantly comprised of vascular channels containing erythrocytes, this include: Pyogenic Granuloma (PG) and Granuloma Gravidarum (GG).
- Giant cell lesion which predominantly comprised of multinucleated numerous giant cells in a fibro vascular connective tissue stroma. this include: Peripheral Giant Cell Granuloma (PGCG).

For the purpose of recording the site distribution of these lesions, each jaw was divided into: an incisor-cuspid region, a premolar region, and a molar region. Deciduous molars were included in the premolar region.

The total number of localized reactive gingival lesions was determined both as an absolute number and as a percentage of the total number of the lesions.

Results

During the twelve year interval, 636 localized reactive hyperplastic lesions of the gingiva were recorded from a total biopsy of 4027 cases. This constituted about 15.79% of the total biopsies assessed during that period. 173 cases (27.2%) were with fibrous lesions. 137 of them (21.5%) were FFH and 36 of them (5.6%) were OFE. Vascular lesions represented by pyogenic granuloma and granuloma gravidarum constitute 49.2% of the total lesions and giant cell lesions represented by PGCG constitutes 23.58% of the total lesions.

The age of patients at the time of surgical excision ranged from 3 years to 81 years. The distribution of different types of reactive gingival lesions in relation to age is shown in table (1).

The most common gingival lesion was pyogenic granuloma and the peak was in the first and second decade of life (15.25% and 13.99% respectively). Focal fibrous hyperplasia (FFH) and Ossifying Fibrous Epulis (OFE) were most prevalent in the second decade of life (6.13% and 1.88% of total gingival lesions respectively). Peripheral giant cell granuloma (PGCG), on the other hand, demonstrated high distribution for the first three decades of life. Table (2) showed the distribution of localized reactive gingival lesions in relation to

<u>MDJ</u>

gender. Female patients constitute about 66.35% of the total biopsied gingival lesions and male's patients constitute only 33.65% of cases. The female to male ratio was about 2:1, with pyogenic granuloma as the most common gingival lesion among females. 38 cases were pregnancy Epulis which constitutes about 18.53% of total PG in females. The most frequently affected age group was between 21 to 30 years of age in females (75 cases out of 205 female PG), whereas the most frequently affected age group in males was between 11 to 20 years of age (45 cases out of 103 male PG) as shown in table (3).

The site distribution of these gingival lesions was shown in table (4). The most prevalent site for pyogenic granuloma and FFH together with OFE was in the upper incisorcuspid area, whereas the lower premolar region considered as the most prevalent site for PGCG.

Recurrent cases was also recorded in this study and PG recorded only 20 cases (6.38% of total PG cases), whereas PGCG recorded only 3 cases (2% of total PGCG cases), OFE showed no recurrence.

The average size of these gingival lesions was about 1cm in diameter with maximum recorded size for PG of about 7cm. in 32 years female which extend through out lower buccal premolar and molar region.

Discussion

This study is the largest report on the incidence of four main histological types of reactive hyperplasia of the gingiva in Iraq. Pyogenic granuloma was the most common lesion occurring over a wide age range with a peak incidence in the second decade of life. Females outnumbered males in this category. Similar observations were

reported by Kfir et al (4) and Angelopoulous⁽⁶⁾. Poor oral hygiene may be a precipitating factor in many PG patients ⁽⁷⁾. The vascular nature and the rapid growth of PG which are reported in this study may be due to the effect of some factors like inducible nitric oxide synthase (iNOS)⁽⁸⁾ vascular endothelial growth factor (VEGF)^{(9),} Fibroblast growth factor (bFGF)⁽¹⁰⁾ or connective tissue growth $(ctGF)^{(11)}$. The factor present investigation revealed that pyogenic geanulomas were slightly more common on the maxillary gingiva than mandibular gingiva; anterior areas are more frequently affected than posterior areas. Also these lesions are much more common on facial aspect of the gingiva than the lingual aspect. Similar findings were recorded by Vilman et al (12)

PG of the gingiva develops in up to 5% of pregnancy ⁽¹³⁾. In the present study granuloma gravidarum or pregnancy epulis constitutes about 18.5% of total PG in females. The development of this particular kind of gingival lesion, typical in pregnancy, not different from that appearing in non-pregnant women, suggests the existence of a relationship between the gingival lesions and the hormonal condition observed in pregnancy.

Progesterone and estrogen hormones render the gingival tissue more susceptible to chronic irritation caused by plaque and calculus (4,6). Recent studies have revealed that sex hormones manifest a variety of biological and immunological effects. Estrogen enhances VEGF production in macrophages, an effect that is antagonized by androgens and which may be related to the development of PG during pregnancy ⁽¹⁴⁾. On the basis of clinical presentation and histological appearance, some authors believe that pregnancy epulis simply represents a pyogenic granuloma whereas others

believe that the lesion is unique because of the apparent influence of female sex hormones ⁽¹⁵⁾. Increased prevalence of pregnancy epulis toward the end of pregnancy and the tendency for the lesion to shrink after delivery indicate a definite role for hormones in the etiology of this lesion $^{(16,17)}$.

One of the interesting finding in the present study was the high number of PG affecting males between 11 and 20 years of age. The development of these lesions in this particular age group in males may be due to the effect of male sex hormones which reaches the highest levels during puberty with remarkable neglect of oral hygiene in this age group rendering gingival tissue more prone to develop exuberant tissue growth in response to local irritant like calculus and dental plaque.

PGCG is an exophytic lesion that is seen exclusively in the gingiva ⁽¹⁶⁾ and is clinically similar to PG, but with the appearance of multinucleated giant cells and lack of infectious source ⁽⁷⁾. PGCG shows a wide age distribution with peak incidence in patients between 41 and 50 years of age. Similar findings were reported by Bataineh and Al-Dwair (18) who conducted their study on Jordanian sample. PGCG appears to be more common in mandible than maxilla, particularly in lower premolar region; this finding is in agreement with that reported in previous studies (19, 20). Females are more frequently affected by PGCG than males with female to male ratio of about 2:1. This finding is not in agreement with the findings of Kfir et al ⁽⁴⁾ who showed no sex predilection for this lesion.

Most if no all irritation fibromas represent reactive focal fibrous hyperplasia due to trauma or local irritation. Although the tem FFH more accurately describes the clinical appearance and pathogenesis of this entity, it is not commonly used. It may

occur at any oral site, but it is seen mostly on buccal mucosa ⁽⁷⁾. In this study, FFH of the gingiva occur in the same age groups, site and female predilection when compared with PG lesions. This finding could support the notion that inflammatory or reactive hyperplasia of gingiva may be the same lesion at different stages of histological maturation ^(4, 5). FFH could represent a fibrous maturation of PG especially in those lesions with long duration.

Peripheral fibromas ossifving which is also known as Ossifying fibrous epulis (OFE) or peripheral fibroma with calcification occurred more frequently in females than males by a ratio of 2.25:1. There were bimodal peak incidences at the first and second decade of life. This finding is in partial agreement with other investigators ^(4, 19) who found a peak incidence of OFE between the second and third decade of life with average age of 28 years. Eversole and Rovin⁽³⁾ suggested that the loss of periodontium that accompany tooth loss in old age may explain the greater occurrence of OFE in the younger age group. The limitation of PGCG and OFE to the gingival supports a possible histogenic superficial derivation from the periodontal ligament which contains cells capable of producing bone

Conclusion

The author is with the opinion that FFH, PG, PGCG and OFE are gingival response to chronic, low grade irritation by plaque caused and calculus. or any other irritant. However, the histological appearance of each entity may be influenced by the intensity of the irritation, duration of the lesion and possibly hormonal effects like estrogen and progesterone. This may reflect some differences in age, gender as well as site distribution between different lesions.

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| Age / years | FFH | OFE | PG | PGCG |
|---------------|-------------|------------|-------------|--------------|
| | No. (%) | No. (%) | No. (%) | No. (%) |
| 0-10 | 4 (0.6%) | 1 (0.15%) | 24 (3.77%) | 18 (2.83%) |
| 11-20 | 23 (3.61%) | 11 (1.72%) | 97 (15.25%) | 25 (3.93%) |
| 21-30 | 39 (6.13%) | 12 (1.88%) | 89 (13.99%) | 25 (3.93%) |
| 31-40 | 20 (3.14%) | 6 (0.94%) | 43 (6.76%) | 20 (3.14%) |
| 41-50 | 20 (3.14%) | 5 (0.78%) | 33 (5.19%) | 33 (5.19%) |
| 51-60 | 26 (4.08%) | 0 | 15 (2.36%) | 17 (2.67%) |
| 61-70 | 4 (0.6%) | 0 | 6 (0.94%) | 10 (1.57%) |
| Older than 70 | 1 (0.15%) | 1 (0.15%) | 6 (0.94%) | 2 (0.3%) |
| Total | 137 (21.5%) | 36 (5.66%) | 313 (49.2%) | 150 (23.58%) |

| Table (1):] | Distribution | of gingival | lesions by age |
|--------------|--------------|--------------------|----------------|
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Table (2): Distribution of gingival lesions by gender

| Gender | FFH | OFE | PG | PGCG | Total |
|---------|-------------|------------|----------------------------------------------------------------------|-----------------|--------------|
| | No. (%) | No. (%) | No. (%) | No. (%) | No. (%) |
| Males | 41 (6.4%) | 11 (1.72%) | 108 (16.98%) | 54 (8.48%) | 214 (33.65%) |
| Females | 96 (15.1%) | 25 (3.93%) | 205 (32.22%) Non Pregnant 167 (81.46%) Pregnant 38 (18.54%) | 96 (15.1%) | 422 (66.35%) |
| Total | 137 (21.5%) | 36 (5.65%) | 313 (49.2%) | 150 (23.58%) | 636 (100%) |

Table (3): Distribution of PG in relation to age group

| Ago / yoors | Male | Female | Total of PG | |
|---------------|--------------|--------------|-------------|--|
| Age / years | No. (%) | No. (%) | No. (%) | |
| 0-10 | 14 (4.47%) | 10 (3.19%) | 24 (7.66%) | |
| 11-20 | 45 (14.37%) | 52 (16.61%) | 97 (30.99%) | |
| 21-30 | 14 (4.47%) | 75 (23.96%) | 89 (28.43%) | |
| 31-40 | 7 (2.23%) | 36 (11.5%) | 43 (13.73%) | |
| 41-50 | 12 (3.83%) | 21 (6.71%) | 33 (10.54%) | |
| 51-60 | 9 (2.87%) | 6 (1.92%) | 15 (4.79%) | |
| 61-70 | 4 (1.27%0 | 2 (0.63%) | 6 (1.9%) | |
| Older than 70 | 3 (0.95%) | 3 (0.95%) | 6 (1.9%) | |
| Total | 108 (16-98%) | 205 (32.22%) | 313 (49.2%0 | |

| Table (4): Site distribution of gingival lesions |
|--------------------------------------------------|
|--------------------------------------------------|

| | Maxilla | | | Mandible | | | |
|-------|------------------------------|--------------------|-----------------|------------------------------|--------------------|-----------------|------------------|
| Site | Incisor- Cuspid Region | Premolar Region | Molar Region | Incisor- Cuspid Region | Premolar Region | Molar Region | Total No. (%) |
| FFH | 42 (6.6%) | 18 (2.83%) | 17 | 26 | 9 (1.41%) | 25 | 137 |
| 1111 | 42 (0.070) | 10 (2.0370) | (2.67%) | (4.08%) |)(1.41/0) | (3.93%) | (21.5%) |
| OFE | 10 | 5 (0.78%) | 3 (0.47%) | 8 (1.25%) | 8 (1.25%) | 2 (0.31%) | 36 |
| OPE | (1.57%) | 5 (0.7670) | 5 (0.4770) | 0 (1.2570) | 0(1.2570) | 2 (0.5170) | (5.66%) |
| PG | 95 | 36 (5.66%) | 38 | 66 | 31 | 47 (7.7%) | 313 |
| | (14.93%) | | (5.97%) | (10.37%) | (4.87%) | | (49.2%) |
| PGCG | 31 | 20 (3.14%) | 13 | 24 | 40 | 22 | 150 |
| rucu | (4.87%) | 20 (3.1470) | (2.04%) | (3.77%) | (6.29%) | (3.45%) | (23.58%) |
| Total | 178 | 79 | 71 | 124 | 88 | 96 | 636 |
| Total | (27.98%) | (12.42%) | (11.16%) | (19.49%) | (13.83%) | (15.09%) | (100%) |