**ORAL PYOGENIC GRANULOMA. REVIEW OF 10 CASES**

**SUMMARY**

Pyogenic granuloma is a commonly occurring inflammatory hyperplasia of the skin and oral mucosa. It is not associated with pus as its name suggests and histologically it resembles an angiomatous lesion rather than a granulomatous lesion. The present study reports 10 cases (age range 10-46 years old; mean: 31.6 years; male:female ratio, 1:1) on patients that visited Dicle University, Dentistry Faculty, Department of Oral and Maxillofacial Surgery. We report the location, size, course and treatment of each lesion, comparing the results obtained to those reported in the literature. We discuss differential diagnosis with respect to other entities, peripheral ossifying fibroma, peripheral giant cell granuloma, hemangioma and fibroma, all of which show very similar histological appearance to pyogenic granuloma.

**INTRODUCTION**

Pyogenic granuloma (PG) or granuloma pyogenicum is a well-known oral lesion. It is a localized granulation tissue overgrowth in reaction to mild irritation (1). PG is a hyperactive benign inflammatory lesion that occurs mostly on the mucosa of females with high levels of steroid hormones. It is generally believed that female sex hormones play important roles in its pathogenesis (2). PG of the oral cavity is known to involve the gingiva most commonly. The majority remains small and lesions more than 1 cm in diameter are rare on the cheeks, tongue, and floor of the mouth possibly because masticatory trauma restricts their size through necrosis and ulceration (3). Radiographic findings are absent in pyogenic granuloma. PG is a benign lesion; therefore, surgical excision is the treatment of choice. To avoid the possibility of recurrence the lesion must be excised down to the underlying periosteum and predisposing irritant must be removed (2).

The present study reports 10 cases of PG in patients attending our clinic in the Southeastern Anatolia Region of Turkey, with specific consideration of epidemiological factors and differential diagnosis.

**MATERIAL AND METHOD**

Over the period of March 2012- April 2013, a total of 10 patients with PGCG (5 women and 5 men) attended our clinic (Dicle University Dentistry Faculty Department of Oral and Maxillofacial Surgery). Patient age ranged from 10-46 years. In each case we recorded age, sex, lesion location (upper or lower jaw, anterior or posterior position), lesion size, and time-of-onset. Possible correlation between lesion size and time-since-onset was investigated by Spearman rank correlation. Additionally, all patients underwent panoramic and periapical radiography.

**RESULTS**

The basic characteristics of this series are summarized in Table 1. Mean patient age was 31.6 years (range 10-46 years). Sex range (5 men- 5 women) and also location of the lesions (5 in upper jaw-5 in lower jaw) were equal. none of these proportions differs significantly from 1:1. Mean lesion diameter was 1,5 cm, with two granulomas larger than 2 cm. Time-since-onset ranged between 3-20 months.

**DISCUSSION**

All cases included in the present study showed the characteristic clinical appearance of PG i.e. soft, painless, and deep red to reddish‑purple in color. Intraorally, the gingiva is the most common site of involvement (about 60%-70%), followed by the lips (14%), tongue (9%), buccal mucosa (7%), and palate (2%) (4). The maxilla more than the mandible, the anterior region than the posterior with the buccal surfaces being affected more than the lingual surfaces (5,6). In our series all lesions showed gingival localization; the more cases in posterior part (6 cases) than in the anterior part (4 cases) and lower-to-upper jaw ratio was 1:1 (Figs 1,2). Neither these proportions differ significantly from the previous studies (though of course this may simply reflect the small size of the series). These lesions are more common with a higher incidence in females than males by 1.5:1 ratio (7,8). In the current study male-to-female ratio was 1:1.

The authors stated that oral pyogenic granuloma arises as a result of some minor trauma to the tissues that provide a pathway for invasion of microorganisms. Bhaskar et al. in their study observed that oral pyogenic granuloma comprized about 1.85% of all oral pathoses, other than caries and gingivitis treated at US Army Institute of Dental esearch (2). Some investigators consider pyogenic granuloma as a “reactive” or “reparative” tumor process. Regezi et al. suggest that pyogenic granuloma represents an exuberant connective tissue proliferation to a known stimulus or injury like calculus or foreign material within the gingival crevice (1). Several “etiologic factors” such as trauma, injury to a primary tooth, chronic irritation, hormones, drugs, gingival inflammation, preexisting vascular lesions, chronic irritation due to exfoliation of primary teeth, eruption of permanent teeth, defective fillings in the region of tumor, food impaction, total periodontitis, toothbrush trauma, etc. have been suggested as etiological factors where patients presented with these findings (9). We observed supra and subgingival calculus with resorption of the alveolar bone and thickening of the periodontal space in 5 patients (%50) reported here. In case 9, chronic infected roots was involved by the lesion and in case 7, the lesion was seen surrounding an unerupted first premolar teeth (Fig 2).

The size varies in diameter from a few millimeters to several centimeters. Rarely PG exceeds 2.5 cm in size and it usually reaches its full size within weeks or months, remaining indefinitely thereafter (5,6). 2 of our 10 lesions had a diameter greater 2,5 cm (cases 7 and 9; Table 1). This can be related to time of onset, since lesion diameter and onset time were significantly correlated. Note, however, that the correlation was not absolute: in one of in these 2 cases , onset time was only 2 months, but diameter was 2.5 cm. This lack of absolute concordance between lesion size and onset time is probably attributable to the diverse possible causes of PG, and the variation in the intensity with which the causal factors act.

In view of these clinical characteristics of PG, a number of other entities should be considered in differential diagnosis. Differential diagnosis included peripheral giant cell granuloma, peripheral ossifying fibroma, metastatic cancer, hemangioma, pregnancy tumor, conventional granulation tissue hyperplasia, Kaposi’s sarcoma, bacillary angiomatosis and non-Hodgkins lymphoma (10-13). Peripheral giant cell granuloma can be histologically identified due to the presence of multinucleated giant cells and lack of an infectious source (14). Ossifying fibroma or peripheral odontogenic fibroma occurs exclusively on the gingiva; however, it has a minimal vascular component unlike a pyogenic granuloma (6,15). Due to the proliferating blood vessels differential diagnosis of pyogenic granuloma from a hemangioma is made histologically in which hemangioma shows endothelial cell proliferation without acute inflammatory cell infiltrate, which is a common finding in pyogenic granuloma (12). Metastic tumors of the oral cavity are rare and attached gingiva is commonly affected, clinically they resemble reactive or hyperplastic lesions such as pyogenic granuloma, but microscopically they usually resemble the tumor of origin, which usually is distant from the metastatic lesion seen in the oral cavity (16.) The diagnosis of pregnancy tumor is based on the history and the apparent influence of the female sex hormones (17,18). Conventional hyperplastic gingival inflammation resembles pyogenic granuloma in histopathologic sections and it is impossible for the pathologist to reach a diagnosis and in such cases the surgeons description of the lesion is relied on. Pyogenic granuloma is distinguished from Kaposi’s sarcoma in Acquired immune deficiency syndrome due to the proliferation of dysplastic spindle cells, vascular clefts, extravasated erythrocytes and intracellular hyaline bodies none of which are seen in pyogenic granuloma. (19).

Treatment of pyogenic granuloma involves a complete surgical excision (16). After surgical excision of lesions, curettage of underlying tissue is recommended (20). Excision with 2 mm margins at its clinical periphery and to a depth to the periosteum or to the causative agent. Any foreign body, calculus, or defective restoration should be removed as part of the excision (21). Various other treatment modalities such as use of Nd: YAG laser, carbon dioxide laser, flash lamp pulse dye laser, cryosurgery, electrodessication, sodium tetradecyl sulfate sclerotherapy and use of intra lesional steroids have been used by various clinicians (22,23). Bhaskar and Jacoway has reported recurrence rate of 15.8% after conservative excision (2). Vilmann et al. observed that gingival cases show a much higher recurrence rate than lesions from other oral mucosal sites (24). Sapp et al. stated that oral pyogenic granulomas have a relatively high rate of recurrence after simple excision (25). Lawoyin et al. observed no recurrence in cases treated by surgical excision (26). Al‑Khateeb et al. observed a recurrence rate of 5.8% in his study (27). In the current study, all lesions were surgically excised and was sent for histopathologic examination. The scaling and root planning of the adjacent teeth was completed to remove all the local irritants, which could have been the primary etiologic factor. We followed up all cases for a period of 1 year and there has been no recurrence so far.

***Table 1:***Summarized characteristics of the 10 cases of pyogenic granuloma considered in the present study

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Case | Age  (years) | Sex | Jaw | Location | Size  (cm) | TSO  (months) |
| 1 | 42 | M | L | Posterior | 1 | 3 |
| 2 | 36 | F | L | Posterior | 1.5 | 4 |
| 3 | 44 | F | U | Anterior | 1.5 | 4 |
| 4 | 45 | F | L | Posterior | 1 | 3 |
| 5 | 14 | F | L | Posterior | 1.5 | 2 |
| 6 | 32 | F | U | Anterior | 1 | 2 |
| 7 | 10 | M | U | Posterior | 2.5 | 2 |
| 8 | 32 | F | L | Anterior | 1.5 | 6 |
| 9 | 46 | M | U | Posterior | 2.5 | 20 |
| 10 | 15 | F | U | Anterior | 1 | 1 |
|  | Mean  31.6 | F/M  5:5 | U/L  5:5 | P/A  6:4 | Mean  1.5 | Mean  4.7 |
| F: female, M: male, U: upper, L: lower, A: anterior, P: posterior, TSO: time-of-onset. | | | | | | |

Figure Legends:

Figure 1 : The lesions in the upper jaw

Figure 2 : The lesions in the lower jaw

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