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Clinical Presentation and Management of Peripheral Giant Cell Granulomas in Children: 2 Cases Report

SUMMARY

Objective(s)*: Peripheral giant cell granuloma (PGCG) is a reactive, proliferative, exophytic lesion developing on the gingiva and alveolar ridge, originating from the periosteum or periodontal membrane. The lesion develops mostly in adults, commonly in the lower jaw, with slight female predilection although is uncommon in children.*

Cases Report*: Two boys, 11 and 8-years-old respectively, otherwise healthy, presented with gingival exophytic lesions in our clinic. In the first case the lesion was located in the right maxilla and appeared 4 months ago, whereas in the second case the fast growing lesion was located in the mandible and appeared 2 months ago. The lesions were red-blue enlargements, irregular and elliptical in shape respectively, soft to firm on palpation. Based on clinical examination, the initial diagnosis was assumed to be a type of reactive hyperplasia. OPG and CBCT showed no evidence of bone pathology. Blood, biochemical and hormonal investigations were within the normal values. Both lesions were surgically removed and histological examination established the diagnosis of PGCG. 4 consecutive follow ups have been done, with no evidence of recurrence.*

Conclusion*: This uncommon lesion in children should be included in the differential diagnosis of reactive hyperplasia. The treatment of PGCG comprises surgical resection, along with suppression of the underlying etiologic factors.*

Keywords: Peripheral Giant Cell Granuloma; Dental Treatment; Giant Cell Epulis

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Introduction

Peripheral giant cell granuloma (PGCG) is categorized as a reactive hyperplastic lesion. It is one of the most common giant cell lesions of the jaws[1]. It originates from connective tissue of the periodontal membrane and the periosteum due to a chronic trauma or irritation[2,3]. Chronic trauma is capable to induce inflammatory phenomena, which are characterized by the presence of inflammatory cells, formation of granulation tissue and tissue overgrowth. The lesion has an exophytic and proliferative appearance, due to the reparatory processes that take place, but it is not a neoplasm. Some of the possible causes may be ill-fitting restorations and dentures, plaque, calculus, food impaction, tooth extraction, tooth fracture, chronic trauma and orthodontic appliances[4,5]. However, there are cases where the responsible factor cannot be diagnosed.

Clinically, PGCG manifests as a firm, soft nodule, usually with ulcerated surface. The colour of the lesion ranges from red to purple or blue. The mean diameter of the lesion, typically located in the interdental papilla, the gingival level or the alveolar margin of the premolars or molars of the mandible, is 1-2cm[6]. The patient may complain of pain caused by repeated trauma, although lesion is usually painless. Radiographically, there are no changes in the underlining bone; however, lesions of great diameter are able to cause superficial erosions. Histologically, many multinucleated giant cells are present in a cellular and vascular stroma. The epithelium has a squamous structure and the connective tissue is characterized by inflammatory infiltration and small blood

vessels[7]. The treatment of choice consists of surgical excision with careful removal of the entire lesion to avoid a recurrence[8]. Most of the affected patients are the adults, in their fourth to sixth decade of life, whereas a few cases have been reported to occur in children, which involved more aggressive lesions, .

This article describes clinical presentation, diagnostic sequence and the management of 2 cases of the PGCG in children.

Patient Presentation

2 boys, 11 and 8-years-old respectively, with non-contributory medical history were referred to our Clinic. An informed consent was obtained from the patients' families for the case presentations. The chief complaint was the appearance of a fast growing exophytic lesion. In the first case, the lesion was located in the upper right maxillary region, which appeared 4 months ago. The intraoral examination identified a red-blue exophytic lesion from #14-17, measuring 3 x 1.5 cm in size, irregular in shape, soft consistency and slightly painful on palpation (Fig. 1). In the second case, the lesion was located in the mandible, which appeared 2 months ago. The clinical examination showed a painless red enlargement between teeth #31 and #32, which moved the teeth apart (Fig. 2). The lesion was elliptical in shape (maximum diameter of 1.5 cm), smooth and firm on palpation. The radiographic examination of both lesions with conventional panoramic radiograph (OPG) and cone beam computerized tomography (CBCT) showed no signs of the bone involvement (Figs. 3 and 4).

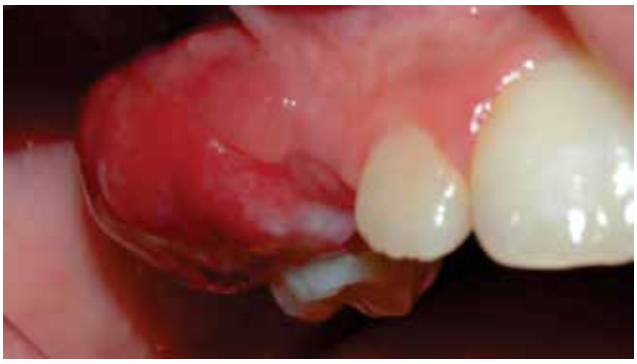

Figure 1. Patient 1. The initial clinical appearance of the lesion in the maxilla

Figure 2. Patient 2. A PGCG presented as a painless red enlargement between teeth #31 and 32, moving the teeth apart

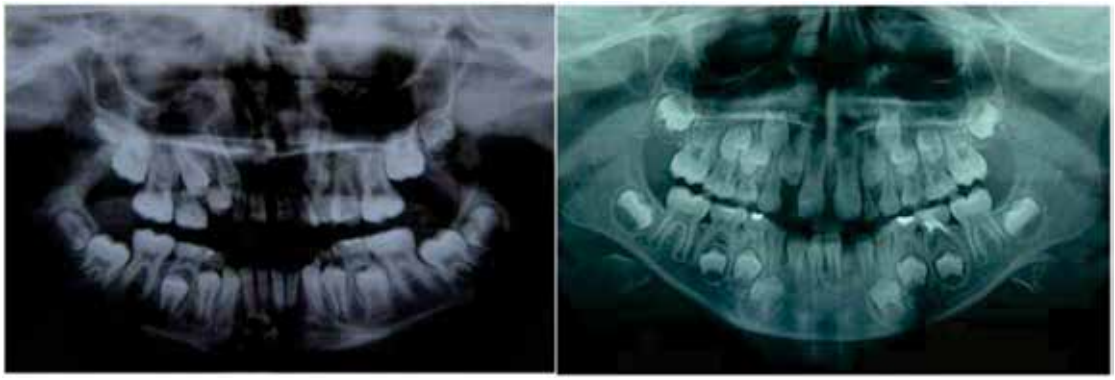

Figure 3. Panoramic radiographs of both patients, showing no evidence of bone involvement (patient 1 - left; patient 2 - right)

The selected treatment for both cases was the total surgical excision of the lesions, and in case 1, the deciduous tooth #55 was removed. Briefly, the lesions were surgically removed under local anaesthesia using initially steel scalpel for the resection, to preserve intact borders of the specimens for histological examination. Afterwards, electro-surgery device was utilized to minimize bleeding, and extensive curettage was performed with the exposure of the bone walls. The surgical area was covered by periodontal/surgical dressing in order to protect the surgical wound, which was removed after 2 days. Chlorhexidine gel was prescribed and the immediate postoperative period was uneventful.

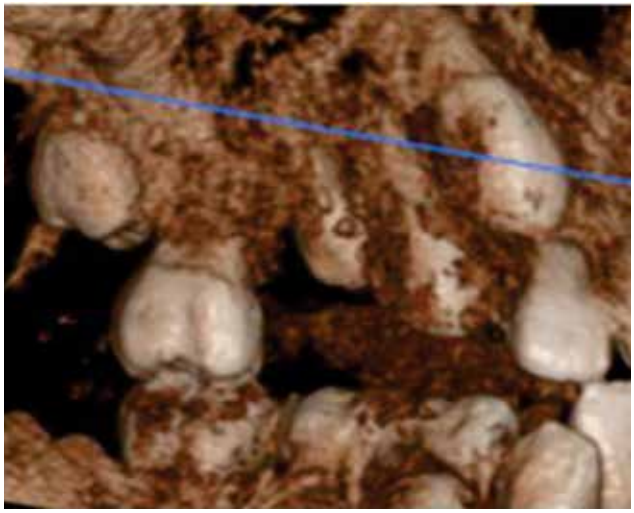

Figure 4. 3-dimensional CBCT of the area (patient 1) showing no signs of bone involvement after the removal of the deciduous tooth (#55)

Histological examination of the specimens was performed to establish the precise diagnosis. The additional blood, biochemical and hormone analysis, conducted in order to exclude the possibility of hyperparathyroidism, were within the normal values. The microscopic examination of the lesions revealed hyperplastic granulation tissue, capillaries and proliferation of multinucleated giant cells within haemorrhagic background. Adjacent acute and chronic inflammatory cells were also present (Fig. 5). The final diagnosis, based on the histological findings in both cases, was PGCG.

4 consecutive follow-ups have been done in a period of 2 years. The healing progress in both cases was satisfactory, and no evidence of recurrence was observed. At the final follow-up, there was no sign of pathology or recurrence and in the case 1 the permanent successor (#15) erupted normally (Fig. 6). Concerning the case 2, the patient was referred for orthodontic consultation.

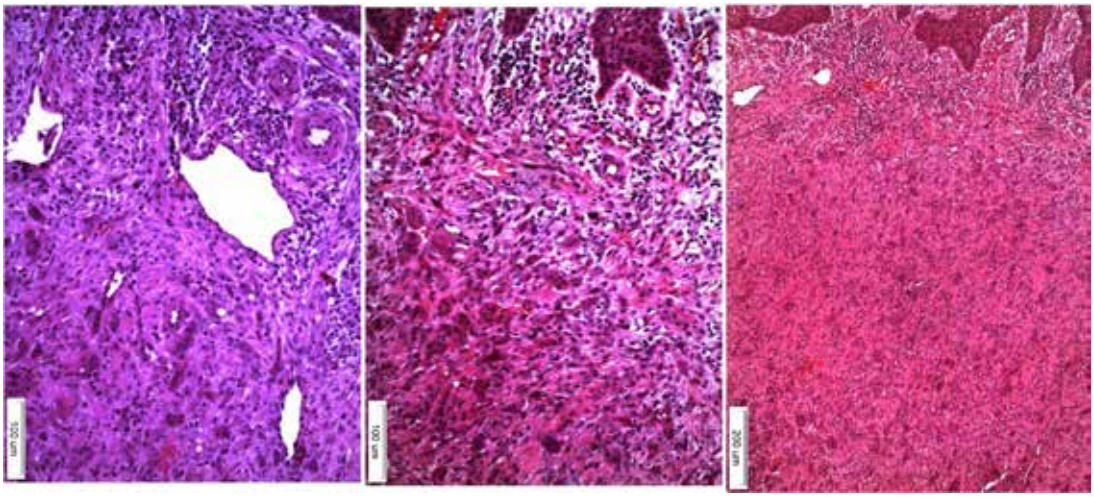

Figure 5. Histological appearance of the PGCG lesion (in various magnifications) showing features of hyperplastic granulation tissue, the presence of acute and chronic inflammatory cells, capillaries, and proliferation of multinucleated giant cells within haemorrhagic background (H&E stain x20)

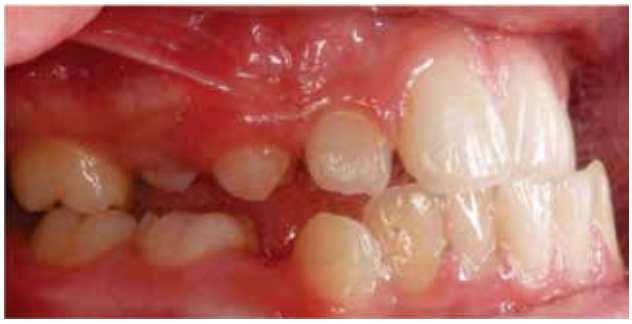

Figure 6. Clinical appearance of patient 1 in the final follow-up, showing no signs of pathology

Discussion

PGCG is a benign exophytic lesion, but not a true neoplasm. The etiologic factors are not yet clear. However, the main pathogenic mechanism of the PGCG is the prolonged repairing process due to chronic inflammation or trauma. According to the literature, the PGCG mostly appear in the fourth decade of life. The PGCG is very rare in children, as it has been suggested and confirmed by various studies[3,9,10]. In the paediatric reports, the frequency of PGCGs, ranged from 1% to 17% of all oral lesions[11], whereas the Greek report accounted 7% of the PGCGs

in their study population[12]. There is no consensus in the literature about the sex prevalence. There are studies in which the females are affected more[13], while in others the males are affected slightly more frequently[14], or the ratios are equal[10]. The most common location of the PGCG is the mandible, in the areas posterior teeth to canines[15].

The consistency of the lesion tends to be soft due to maturation process that changes the soft lesions to firm[16]. It is characterized as aggressive lesion, with the high rate of relapse and bone absorption of the interproximal crest area[3,17]. Children with the hypophosphatemic rickets (subclinical hyperparathyroidism) are high risk patients to develop the PGCG, since this condition can be influenced by sex hormones. The giant cells are potential target for the oestrogen action. The lesions that are developed imitate peripheral lesions and are called brown tumours. Histologically, the brown tumours cannot be distinguished from the giant cell lesions[10]. Consequently, when a lesion cannot be distinguished as arising in a peripheral or central location, the patient should be further evaluated to rule out the hyperparathyroidism[18]. In our cases, the hormone levels and the other haematological findings of the patients were within the normal range, so the possibility of a brown tumour was excluded.

The clinical differential diagnosis of the PGCG includes pyogenic granuloma, fibrous epulis, peripheral ossifying fibroma, inflammatory fibrous hyperplasia, peripheral odontogenic fibroma, cavernous haemangioma and papilloma[1]. The histological examination of the resected tissue is necessary to confirm the definitive diagnosis. Finally, the differential diagnosis from the central giant cell granuloma is very important and it's set by radiographic findings. The central giant cell granuloma is located inside the bone and is more aggressive condition with a different treatment[1]. In our cases, the differential diagnosis from the central giant cell granuloma was given after the radiographic examination, since the bone involvement wasn't evident.

The treatment of the PGCG consists of surgical excision with elimination of the lesion from the entire base, its aggressive curettage, followed by the removal of possible etiologic factors. However, recurrence rate ranges from 1.4% to 22%[18]. In our first case, the lesion was surgically removed along with the primary tooth.

The presented cases showed a proper management of PGCGs in children. Early diagnosis and the differential diagnosis from other conditions using clinical, radiographic, histological and biochemical findings were essential for the treatment plan. Finally, there were no signs of pathology, nor alveolar complications, especially in case 1 where the eruption of the permanent tooth was successful.

Conclusion

PGCG usually is not an aggressive lesion. However, the early detection and accurate diagnosis, based on clinical and radiographic findings as well as the histological examination, are essential in order to reduce dento-alveolar complications, such as bone loss or displacement of the dental germ.

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