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Management of central giant cell granuloma: discussion of two cases

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Background. The Central Giant Cell Granuloma (CGCG) is an uncommon benign lesion of the jaws. It occurs predominantly in children and young adults, and may cause local destruction of bone and displacement of teeth.

Case Report. Two case reports are presented illustrating the joint management of children with this condition by paediatric dentistry and maxillofacial surgery teams.

Conclusion. The role of the paediatric dental team is extensive in children with CGCG and coordination of care from both teams is essential to ensure that the highest quality of care is provided.

Introduction

A central giant cell granuloma (CGCG) is a benign lesion accounting for approximately 7% of all non-neoplastic tumours involving the jaws[1]. Jaffe first differentiated CGCG from neoplastic giant cell tumours of bone on clinical and histological grounds in 1953[2]. Those affected are predominately children and young adults, with nearly half of all cases manifesting before the age of 20 years[3]. Occurrence of the lesion has widely been reported as arising more commonly in females[3–13]; however, in the first decade of life, the presentation of the lesion is predominately male[4,8,12]. Single lesions occur more frequently in the mandible than the maxilla, with a prevalence of 60–85%[3–12,14,15]. Children with this lesion are likely to present to the paediatric dental department, as illustrated in the following two cases. The aim of this report is to describe the presentation, investigation, diagnosis and treatment of two such paediatric cases.

Case reports

Case report 1

A fit and well 7-year-old male presented with a firm, localized non-tender swelling of the mandible that had been present for 3 months. This had not improved with antibiotics prescribed by a general dental practitioner. Paraesthesia of the right lower lip had been present for 3 weeks. Intermittent bleeding from the lesion had resulted in absence from school, since the situation was perceived by the school authorities as a health and safety issue.

Clinically, the boy was pale with a palpable, non-tender node in the right submandibular region. Intraorally, an exophytic swelling was present, involving 84, 85 and 46. The lesion was ulcerated as a result of repeated occlusal trauma from opposing 55 (Fig. 1). Both 84 and 46 were mobile. Grossly carious deciduous molars were noted in all four quadrants.

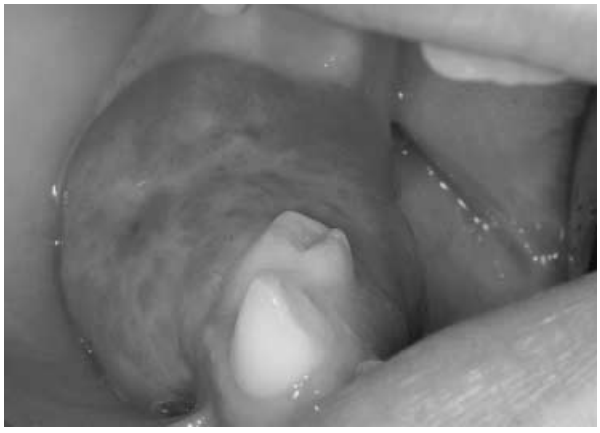

Fig. 1. Intraoral view of case 1 showing the extent and appearance of an exophytic ulcerated lesion.

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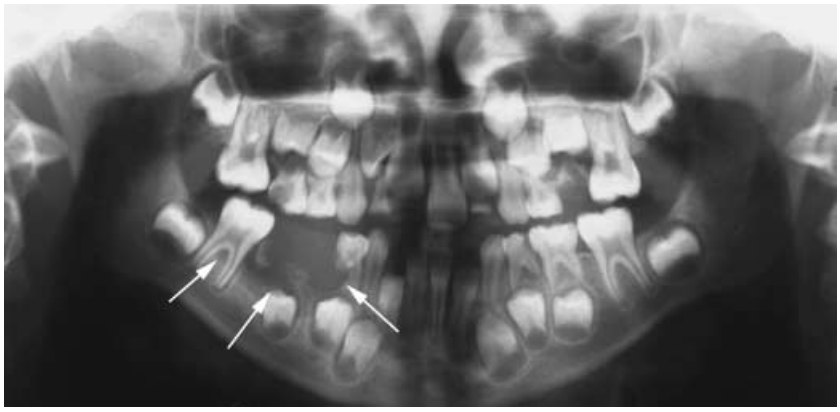

Fig. 2. Orthpantomograph of case 1 taken at presentation showing a diffuse radiolucent area in the right mandible, pathological root resorption of 84, and the involvement of 46, 85 and 45.

Radiographic investigation revealed a poorly circumscribed radiolucency involving the erupted 84, 85 and 46, and the unerupted 45 (Fig. 2). Widening of the periodontal ligament of the mesial root of 46 was evident, along with pathological root resorption of 84. A provisional diagnosis of CGCG was made and relevant haematological and biochemical investigations were undertaken to confirm the above and eliminate hyperparathyroidism. Low haemoglobin, and elevated creatinine and phosphate levels were noted (Table 1).

An incisional biopsy and extraction of all grossly carious deciduous teeth was performed under general anaesthesia. Histology confirmed the diagnosis of CGCG. The lesion was subsequently excised with the sacrifice of the unerupted 45 and erupted 46. No perforation of the cortical plate was noted at the time of excision. Both 26 and 36 were fissure sealed at surgery, and in view of the patient's age and dental development, compensating extraction of 16 was also undertaken. Currently, the patient remains well and is under regular joint review.

Case report 2

A fit and well 9-year-old male presented with a firm, localized non-tender facial swelling of the right maxilla with a duration of 2 weeks that had not responded to antibiotics. Clinically, the swelling extended intraorally from 11 to 54, with 11, 12 and 53 exhibiting increased mobility.

Radiographs confirmed a poorly circumscribed radiolucency with displacement of adjacent teeth and root resorption of 53.

Following a provisional diagnosis of CGCG, appropriate investigations were undertaken, including serum calcium and phosphate (Table 1), computed tomography scan (Fig. 3) and incisional biopsy, which confirmed the diagnosis.

The lesion was excised via an intraoral approach with the sacrifice of 11, 12, 13, 14 and 54 (Figs 4 & 5). A small antral communication was noted on the anterior wall at the time of excision. A subsequent procedure was carried out 6 weeks

Table 1. Relevant haematological and biochemical results.

Investigation	Case 1	Case 2	Reference range
Haemoglobin	10.8	Normal	11.5–15.5 g dL^{-1}
Creatinine	100	Normal	20–60 µmol L^{-1}
Phosphate	2.02	1·73	0.8–1.9 mmol L^{-1}
C-reactive protein	5.7	Normal	0–5 mg L^{-1}
Parathyroid hormone	13	16	8–63 ng L^{-1}

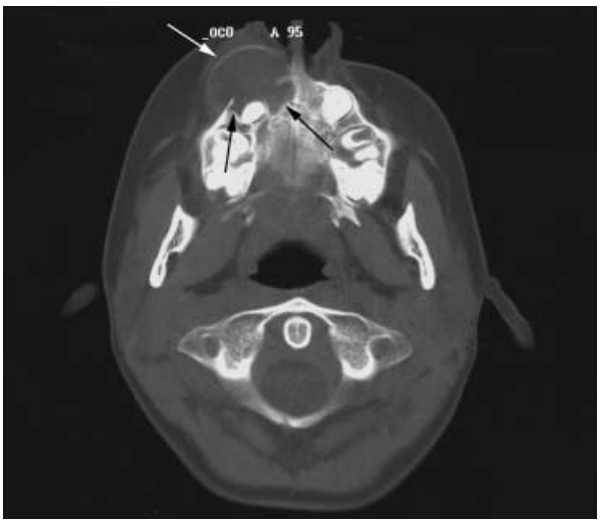

Fig. 3. Computed tomography scan of case 2 showing a cystic expansile lesion in the right maxilla that is expanding labially with well-defined margins.

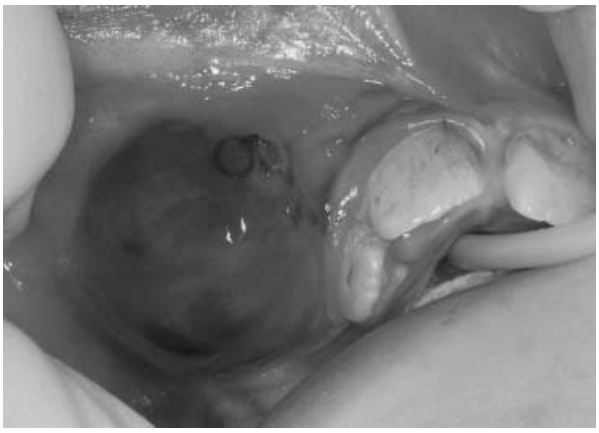

Fig. 4. Intraoral appearance of the lesion immediately before case 2 underwent curettage.

later using an extraoral approach. The extraoral incision employed was a modification of the Weber-Dieffenbach (Ferguson) incision routinely used for maxillectomy procedures[16]. The incision passes across the lower eyelid in the first skin crease below the eyelashes. Both skin and orbicularis oculi muscle are separated from the orbital septum, down to the infraorbital rim, where a periosteal incision is made. At the junction of the skin incision with the nose, the incision is carried down to bone and passes down the lateral wall of the nose, where it meets cheek skin. At the alar crease, the incision is turned laterally and passes around the base of the alar lobule. In a maxillectomy procedure, it would continue across the base of the nose at the junction with upper lip, and then the upper lip would be split by an incision taken down the edge of the philtrum. This latter part was unnecessary in providing adequate access to the tumour in the paranasal region, and the incision stopped inferior to the alar lobule.

An upper prosthesis was constructed for restoration of aesthetics and function until such time as a bone graft and implants are appropriate (Fig. 6). Currently, the patient remains well and continues to be under joint review.

Discussion

The clinical behaviour of CGCG is variable, ranging from slow-growing and asymptomatic to rapidly expanding lesions with pain, swelling, cortical bone perforation, root resorption and displacement of teeth[14]. Paraesthesia of the lip is an occasional finding[3–5,8,12].

The radiographic appearance may be either uni- or multilocular, with a tendency for the latter when the lesion is larger. The margins may be either well defined or irregular in appearance[3–5,7–10].

Histologically, CGCG consists of an intraosseous lesion with a cellular fibrous stroma containing aggregations of multinucleate giant cells, extravasated erythrocytes, and often, large sinusoidal spaces[1,6,8,11–14,17]. Importantly, it cannot

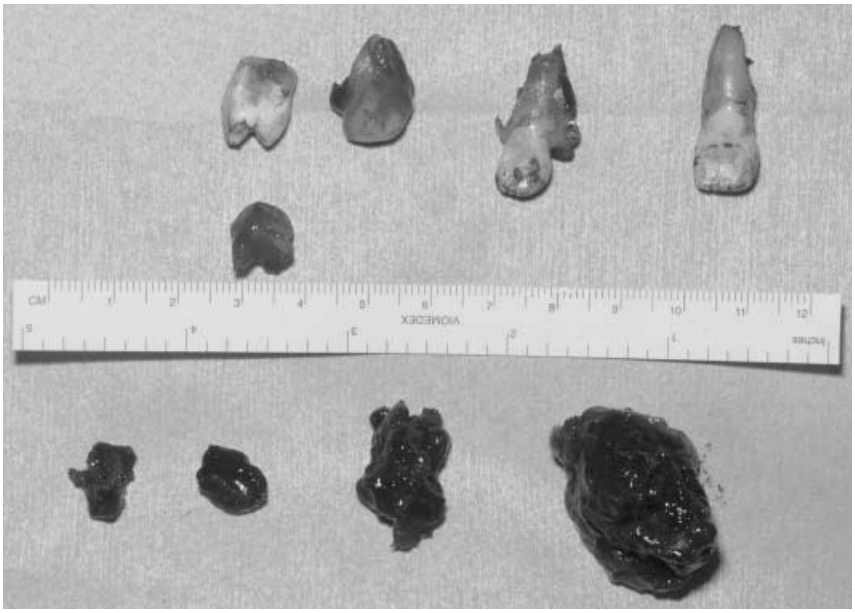

Fig. 5. Excised specimen from case 2, including unerupted 14 and 13, and erupted 54, 12 and 11.

Benign	Malignant
Aneurysmal bone cyst	Fibrosarcoma
Brown tumour of hyperparathyroidism	Lymphoma
Cementifying fibroma	Malignant fibrous histiocytoma
Cherubism	Malignant giant cell tumour of bone
Fibrous dysplasia	Osteogenic sarcoma
Ossifying fibroma	Giant cell tumour of bone

Table 2. Differential diagnosis of multinucleate giant cell lesion (in alphabetical order)

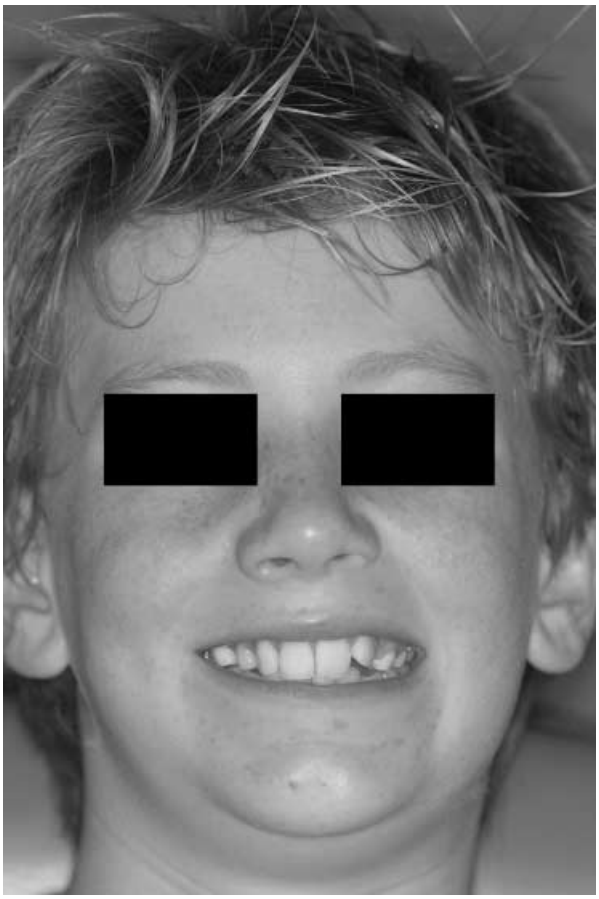

Fig. 6. Facial view of case 2 taken 6 months after an extraoral approach involving surgical curettage of central giant cell granuloma on the right maxilla, and replacement of 11, 12, and 53 with a removable prosthesis.

be distinguished on histological grounds alone from other multinucleate giant cell lesions of the jaws, including brown tumour of hyperparathyroidism, aneurysmal bone cyst and cherubism[4,14]. Table 2 illustrates an extensive list of both benign and malignant jaw lesions that may contain multinucleate giant cells.

Appropriate blood investigations are necessary, including serum chemistry, to exclude brown tumour of hyperparathyroidism. Additional blood tests could include full blood count, erythrocyte sedimentation rate, liver functions tests, B-2 microglobulin, serum calcium, urea and electrolytes, alkaline phosphatase, lactate dehydrogenase, and glucose to assist in exclusion of other conditions.

Provisional diagnoses of CGCG were made for both cases based on the history and clinical and radiographic findings. Key findings common to both included age and gender – both were male and in the first decade of life – together with clinical presentation of expanding exophytic lesions where radiographs revealed local destruction of bone and pathological root resorption of teeth.

Establishment of a definitive diagnosis, however, relies on a combination of the clinical and radiological findings with biochemistry and histopathology, the latter definitively excluding malignancy. Conventional management is by curettage or resection, which may be associated with loss of teeth, or in the younger patient, developing tooth germs. Non-surgical treatment includes systemic calcitonin therapy[18–20] and intralesional injections with corticosteroids[21–25]. In general, these studies have been on small numbers of patients with a limited period of follow up. Case 2 presented a problem with inadequate surgical clearance because of the complicated local anatomy of the maxilla, resulting in a second extraoral procedure being required; calcitonin therapy has been discussed as a possible future treatment if a further recurrence arises.

The reported recurrence rate following treatment varies among studies, ranging from 11% to 23%[6,8,9,11–13,15,17]. In a series reported in children, however, there was a recurrence of 41%[14]. Furthermore, the maxilla was the site for recurrence in five out of these seven cases. This may be explained by the fact that, in the mandible, lesions present in a relatively confined

anatomical area, whereas, in the maxilla, the situation is complicated by the more diverse anatomy, including the maxillary sinus, orbit and nasal cavities, resulting in increased perioperative morbidity and practical difficulty when undertaking radical curettage. This predilection for recurrence in the maxilla is supported by de Lange and van den Akker[4], and Whitaker and Waldron[8]. Cortical plate perforation has been shown to be a significant factor in risk of recurrence[6] and was found to be present in one of the two cases reported.

What this paper adds

- This paper adds to the currently available literature by focusing on the combined management of children with CGCG by both the maxillofacial and paediatric dentistry teams.

Why this paper is important to paediatric dentists

- This paper highlights the extensive participation of the paediatric dental team in initial diagnosis, subsequent treatment of co-existing dental disease as well as the implementation of a preventive programme and provision of prosthetic replacement of teeth if needed when managing a child with a CGCG.

Conclusion

The role of the paediatric team is extensive, including initial diagnosis and subsequent treatment of coexisting dental disease, as well as implementation of a preventive programme and provision of prosthetic replacement of teeth where there is loss of function or aesthetics after surgery. Coordination of care from both teams is essential to ensure that the highest quality of care is provided, efficient use of hospital attendances is attained, and there is minimal disruption to schooling and parental time off work, thereby enabling a swift and smooth return to normal family life.

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