



CASE REPORT

Periapical radiolucency mimicking an odontogenic cyst

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Abstract

Nary Filho H, Matsumoto MA, Fraga SC, Gonçalves ES, Sérvulo F. Periapical radiolucency mimicking an odontogenic cyst. *International Endodontic Journal*, 37, 337–344, 2004.

Aim To present a clinical case of a giant cell lesion located in an unusual site, initially misdiagnosed and treated as an odontogenic cyst.

Summary Periapical radiolucencies often suggest the presence of odontogenic pathosis, usually inflammatory granulomas or cysts. The high frequency of such lesions tends to lead clinicians to arrive at a diagnosis without completing a comprehensive assessment of the patient or carrying out the full range of available diagnostic tests. A case report of a giant cell lesion, which was misdiagnosed and treated initially as an odontogenic lesion because of its unusual location, is presented.

Key learning points

- Clinical signs and radiographic appearance are usually sufficient to reach a diagnosis of periapical pathosis.
- When traditional treatment does not lead to success, a biopsy should be considered to ascertain the diagnosis and allow the correct treatment to be provided.
- Histological examination of soft tissue removed during endodontic surgery is essential.

Keywords: benign neoplasms, central giant cell lesion, periapical pathology.

Received 13 February 2003; accepted 29 December 2003

Introduction

Giant cell granulomas of the jaws are considered to be benign lesions of the maxillofacial region, which occur in two forms, i.e. central, when they arise inside the bone, or peripheral, when they occur in soft tissue (Waldron 1995). Initially called giant cell reparative granuloma (Jaffe 1953) because of its apparently less aggressive clinical behaviour compared to giant cell tumours affecting long bones, the current terminology 'giant cell lesion' makes no

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distinction. The term 'reparative' has been eliminated (Waldron & Shafer 1966) as it does not reflect the biological behaviour of the lesion.

Two distinct forms are currently recognized: slow-growing, nonaggressive and asymptomatic forms that do not perforate cortical bone; and painful rapidly growing forms that result in expansion and perforation of the cortical plate and even in tooth resorption. The latter has significant rates of recurrence after surgical excision, characterizing neoplastic behaviour (Waldron & Shafer 1966, Guralnick 1972, Pedersen 1973, Chuong *et al.* 1986, Whitaker & Waldron 1993). Despite these differences, the histological pattern of the two forms is similar. Studies have been performed in order to detect morphological and histochemical characteristics that would explain the differences (Whitaker & Waldron 1993, Whitaker *et al.* 1993).

The aetiology of these lesions remains unclear. Local trauma and haemorrhage are the most frequently reported factors, although they are not always present (Glickman 1988). The influence of sex hormones, such as oestrogen and progesterone, has been proposed as a result of the fact that more young women than men and elderly individuals are affected (Whitaker & Bouquot 1994). Genetic causes have also been investigated (Zhu & Qui 1994).

The radiographic features of these lesions are nonpathognomonic, a fact that makes their diagnosis difficult (Selden 2000). According to a radiographic analysis of 80 cases of central giant cell lesions of the maxilla (Kaffe *et al.* 1996), they demonstrated variable sizes and uni- or multilocular radiolucencies associated with tooth displacement or resorption. The mandible was affected more commonly than the maxilla, especially in the anterior segment (Whitaker & Waldron 1993). When localized close to the periapical region or laterally to the roots of the teeth, these lesions are easily confused with inflammatory odontogenic lesions (Glickman 1988, Dahlkemper *et al.* 2000).

The objective of this report is to present a clinical case of a central giant cell lesion located in the periapical region that was initially misdiagnosed as an inflammatory odontogenic cyst; it was identified by chance as a radiographic finding during orthodontic review, and was treated accordingly. The aetiology of the lesion and the outcome of treatment are discussed. In addition, the report emphasizes the need for histopathological examination of any tissue removed from the periapical region during endodontic surgery, especially when root canal treatment does not lead to a predictable response. In this way, further complications for the patient should be avoided and more appropriate treatment can be instituted to specific pathologies.

Case report

A 16-year-old female patient was referred to the Oral and Maxillofacial Surgery Service of the Dentistry Department, Sagrado Coração University, Bauru, São Paulo, Brazil, for evaluation and treatment of a lesion located in the mandible and detected upon routine orthodontic follow-up radiography. Sensitivity tests of the involved teeth performed by the clinician who referred the patient were negative for the mandibular left central incisor. Based on this result, root canal treatment was initiated, with slight pulp bleeding during canal access. As neither clinical nor radiographic findings indicated regression of the lesion 8 months after root canal treatment, the patient was referred.

The patient did not report any symptoms, and her medical history was not significant. An initial clinical examination revealed a hard swelling of the lingual cortex in the region of the incisors (Fig. 1) covered with normal mucosa. Radiography revealed an oval, regular and well-delineated radiolucent area located between teeth 31 and 32. Tooth 31 had been subjected to root canal treatment, with satisfactory technical results (Fig. 2) but inadequate periapical bone healing.

On an outpatient basis and under local anaesthesia, an excisional biopsy of the lesion was performed 1 week after her first visit that consisted of simple surgical curettage. In



Figure 1 Clinical aspect of the affected area. Normal mucosa covering expanded lingual cortex related to tooth 31.

view of the acceptable quality of the root filling, no further endodontic therapy was undertaken.

During the surgical procedure, the presence of a solid lesion was verified (Fig. 3), which indicated the need for more aggressive curettage with cortical scarification using a bur; this resulted in a bone defect that involved the vestibular wall. Similarly, the root was polished because of the involvement of the periodontal ligament, but without visual verification of resorbed areas within the root. Because of the defect, the area was filled with an autogenous bone graft obtained from the chin region with a trephine bur (Fig. 4a,b). The harvested bone block was trimmed to shape the cavity; and the wound was sutured. No membrane was employed.

The specimen was sent for pathological examination. Macroscopically, soft, dark-red tissue that crumbled upon manipulation was observed. Microscopically, fibrous connective tissue, rich in spindle-shaped and ovoid cells containing numerous blood vessels and multinucleated giant cells, was noted. Areas of osteogenesis and angiogenesis were also present, as well as occasional fragments of bone tissue located at the periphery of the lesion. The appearance was consistent with a central giant cell lesion (Fig. 5).

Follow-up of the patient during a preliminary period of 2 years following curettage of the lesion showed healing of the area, without any signs of recurrence and with re-establishment of the lamina dura and periodontal architecture (Fig. 6a–d).



Figure 2 Radiographic aspect of the lesion between teeth 31 and 32. A radiolucent area which did not respond to the endodontic treatment of tooth 31.



Figure 3 After the exposure of the area, a solid lesion was found, discounting a clinical diagnosis of odontogenic cyst.

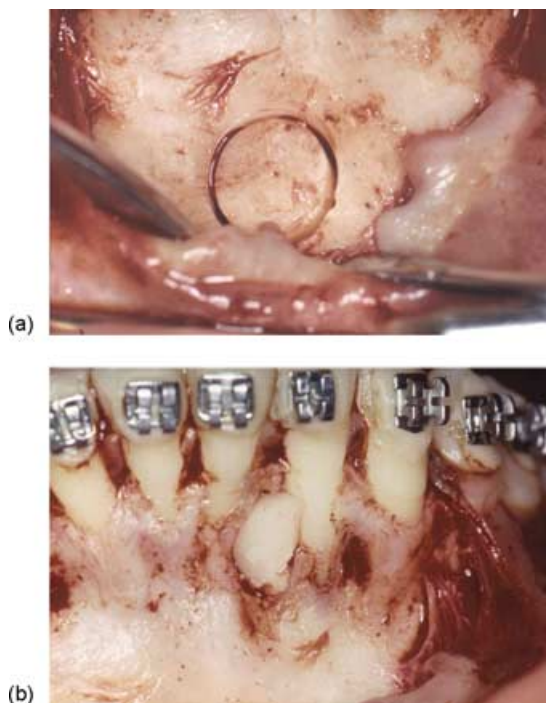


Figure 4 (a) After the curettage of the lesion, autogenous bone graft harvested from the chin was used to fill in the defect; (b) bone graft positioned.

Discussion

Clinical and radiographic evaluations of intraosseous lesions, especially when located adjacent to roots, are subject to diagnostic confusion. The high incidence of odontogenic lesions often leads clinicians to ignore other possibilities, such as central giant cell lesions, within the differential diagnosis (Kaffe *et al.* 1996). After all, common lesions occur commonly and the occurrence of other pathosis in the region of the incisors is infrequent. In addition, there may be difficulties in pulp vitality assessment procedures, leading to imprecise evaluation. Considered to be a conservative treatment, root canal treatment is still the procedure of choice before surgery, and may often be conducted in rare cases of non-dental pathology.

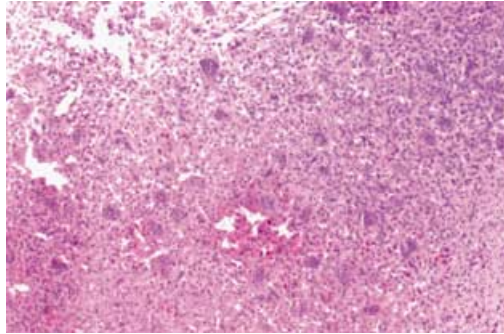


Figure 5 Central giant cell lesion showing numerous multinucleated giant cells in a loose fibrous connective tissue.

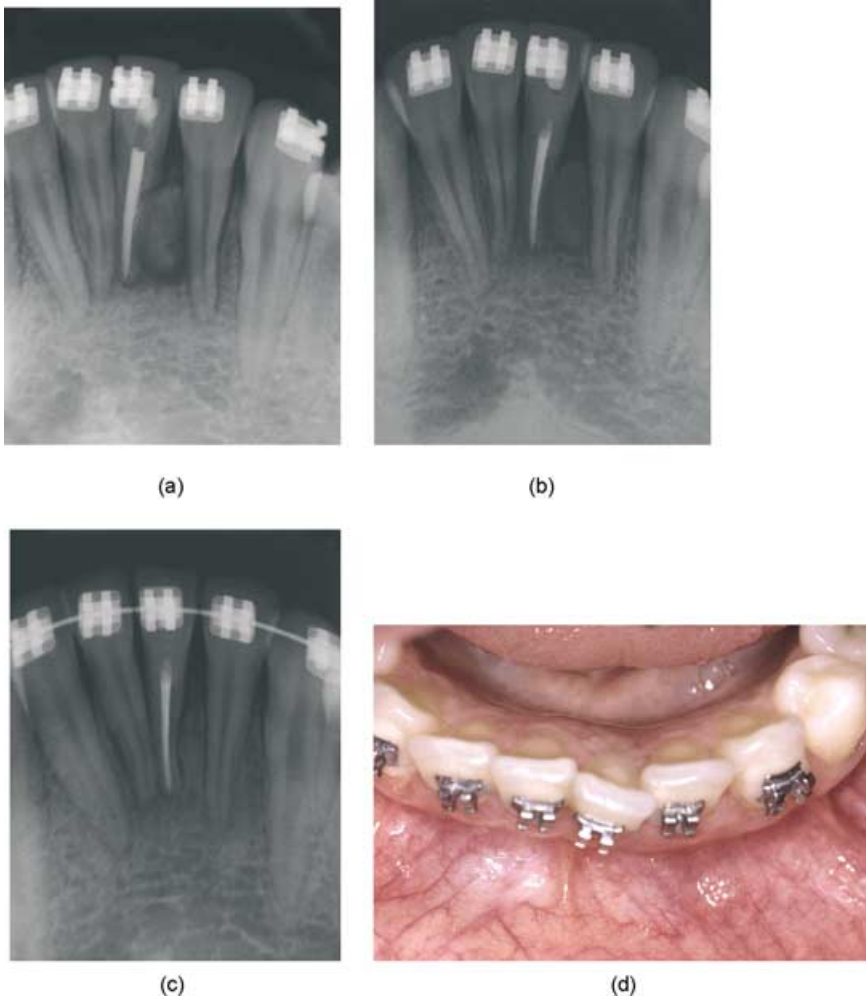


Figure 6 (a) Radiograph 1 month after curettage and bone graft; (b) radiograph after 4 months; note the incorporation of the bone graft; (c) radiograph after 2 years, with complete re-establishment of the lamina dura; (d) clinical aspect after 2 years.

Because of the lack of healing after 8 months, surgical curettage was performed. Based on the characteristics of the lesion, some diagnostic alternatives, such as a lateral periodontal cyst and even a central giant cell lesion, were proposed. In such cases, microscopic analysis of the specimen obtained is extremely important. Comparing the clinical and histopathological diagnoses of 805 periapical lesions, Kuc *et al.* (2000) reported that 4.1% of cases referred as endodontic lesions did not match the clinical and radiographic findings. However, this percentage could be even higher in other situations, as microscopic analysis of the fragment removed during periradicular surgery is not always carried out, usually because clinicians do not routinely send specimens for histological examination.

Conservative surgical treatment was proposed in the present case based on the location, extension and apparently nonaggressive behaviour of the lesion, and when taking into account its radiographic appearance and clinical course. Curettage was performed and the tooth involved was retained; the immediate filling of the surgical area with autogenous bone graft obtained from the chin allowed for more rapid and predictable healing because of the osteogenic potential it provided in the large defect that was present. Few reports using autogenous bone grafts are found in literature, but those that are relevant show satisfactory results, even in areas of previous recurrent lesions (Becelli *et al.* 1998, Markt 2001). The chin represents an excellent graft donor site because of the quality of bone, the low morbidity associated with the procedure and the possibility of carrying out the procedure under local anaesthesia (Misch *et al.* 1992). In the present case, surgical access was possible during the removal of the lesion, thus avoiding surgical intervention in another region.

The root of the involved tooth was curetted because of the lack of alveolar bone and the involvement of the periodontal ligament. More radical surgical procedures, such as block resections, are indicated in cases where the aggressive biological behaviour of the lesion causes rapid bone destruction and pain (Bataineh *et al.* 2002). Treatments other than surgery, such as intralesional injection of corticosteroids (Rajeevan & Soumithran 1998, Carlos & Sedano 2002) and calcitonin (Lange *et al.* 1999, Pogrel *et al.* 1999) have been suggested. Such methods have potential for inhibition of osteoclastic cells in order to reduce the size of the lesions and even to cure them with satisfactory results (Lange *et al.* 1999, Pogrel *et al.* 1999, Rajeevan & Soumithran 1999, Carlos & Sedano 2002).

No history of associated trauma or any other relevant systemic alteration was detected during the preoperative history. The red-coloured blood observed by the clinician during the opening of tooth 31 should have been a clinical signal that the pulp tissue was not involved in the aetiopathogenesis of the lesion, and indicating that the orthodontic treatment may have had a strong association. A similar situation was reported by Pedersen (1973); however, in both cases, the use of orthodontic treatment in the occurrence of the lesion cannot be confirmed.

No clinical or radiographic pathosis was observed in the affected area 2 years following surgery. However, the possibility of later recurrence of the lesion requires follow-up for a prolonged period of time, as the histological pattern of the lesion does not match its biological behaviour.

Conclusions

Clinical signs and radiographic appearance are usually sufficient to arrive at the diagnosis of periapical pathosis. However, lesions other than those of dental origin can affect the jaws. When traditional treatment does not lead to success, a biopsy should be carried out to determine the diagnosis and make appropriate treatment possible.

Disclaimer

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References

- Bataineh AB, Al-Khateeb T, Rawashdeh MA (2002) The surgical treatment of central giant cell granuloma of the mandible. *Journal of Oral and Maxillofacial Surgery* **60**, 756–61.
- Becelli R, Cerulli G, Gasparini G (1998) Surgical and implantation reconstruction in a patient with giant-cell central reparative granuloma. *Journal of Craniofacial Surgery* **9**, 45–7.
- Carlos R, Sedano HO (2002) Intralesional corticosteroids as an alternative treatment for central giant cell granuloma. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics* **93**, 161–6.
- Chuong R, Kaban LB, Kozakewich H, Perez-Atayde A (1986) Central giant cell lesions of the jaws: a clinicopathologic study. *Journal of Oral and Maxillofacial Surgery* **44**, 708–13.
- Dahlkemper P, Wolcott JF, Pringle GA, Hicks ML (2000) Periapical central giant cell granuloma: a potential endodontic misdiagnosis. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics* **90**, 739–45.
- Glickman GN (1988) Central giant cell granuloma associated with a non-vital tooth: a case report. *International Endodontic Journal* **21**, 224–30.
- Guralnick WC (1972) Central giant-cell granuloma. *British Journal of Oral and Maxillofacial Surgery* **9**, 200–7.
- Jaffe HL (1953) Giant-cell reparative granuloma, traumatic bone cyst, and fibrous (fibro-osseous) dysplasia of the jawbones. *Oral Surgery* **6**, 159–75.
- Kaffe I, Ardekian L, Taicher S, Littner MM, Buchner A, Hashomer T (1996) Radiologic features of central giant cell granuloma of the jaws. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics* **81**, 720–6.
- Kuc I, Peters E, Pan J (2000) Comparison of clinical and histologic diagnoses in periapical lesions. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics* **89**, 333–7.
- Lange J, Rosenberg AJWP, van den Akker HP, Koole R, Wirts JJ, van den Berg H (1999) Treatment of central giant cell granuloma of the jaw with calcitonin. *International Journal of Oral and Maxillofacial Surgery* **28**, 372–6.
- Markt JC (2001) An endosseous, implant-retained obturator for the rehabilitation of a recurrent central giant cell granuloma: a clinical report. *Journal of Prosthetic Dentistry* **85**, 116–20.
- Misch CM, Misch CE, Resnik RR, Ismail YH (1992) Reconstruction of maxillary alveolar defects with mandibular symphysis grafts for dental implants: a preliminary procedural report. *International Journal of Oral and Maxillofacial Implants* **7**, 360–6.
- Pedersen GW (1973) Central giant-cell lesion of the maxilla: enucleation and immediate reconstruction. *Oral Surgery, Oral Medicine, Oral Pathology* **36**, 790–9.
- Pogrel MA, Regezi JA, Harris ST, Goldring SR (1999) Calcitonin treatment for central giant cell granulomas of the mandible: report of two cases. *Journal of Oral and Maxillofacial Surgery* **57**, 848–53.
- Rajeevan NS, Soumithran CS (1998) Intralesional corticosteroid injection for central giant cell granuloma. A case report. *International Journal of Oral and Maxillofacial Surgery* **27**, 303–4.
- Selden HS (2000) Central giant cell granuloma: a troublesome lesion. *Journal of Endodontics* **26**, 371–3.
- Waldron CA (1995) Bone pathology. In: Neville BW, Damm DD, Allen CM, Bouquot JE, eds. *Oral and Maxillofacial Pathology*, 1st edn. Philadelphia, USA: W.B. Saunders Co., pp. 443–92.
- Waldron CA, Shafer WG (1966) The central giant cell reparative granuloma of the jaws: an analysis of 38 cases. *American Journal of Clinical Pathology* **45**, 437–47.
- Whitaker SB, Bouquot JE (1994) Estrogen and progesterone receptor status of central giant cell lesions of the jaws. *Oral Surgery, Oral Medicine, Oral Pathology* **77**, 641–4.

- Whitaker SB, Waldron CA (1993) Central giant cells lesions of the jaws. A clinical, radiologic, and histopathologic study. *Oral Surgery, Oral Medicine, Oral Pathology* **75**, 199–208.
- Whitaker SB, Vigneswaran N, Budnick SD, Waldron CA (1993) Giant cell lesions of the jaws: evaluation of nucleolar organizer regions in lesions of varying behavior. *Journal of Oral Pathology and Medicine* **22**, 402–5.
- Zhu Q, Qui J (1994) Cytogenetic analysis on giant cell tumor of bone. *Chinese Journal of Pathology* **23**, 162–5.

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