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Nonendodontic periapical lesions: a retrospective study in Chile

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Abstract

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Aim To determine the frequency with which the histopathological diagnosis of periapical lesions contributes to a change in the clinical diagnosis.

Methodology Cases having a clinical diagnosis of disease resulting from dental pulp necrosis were selected from the database of the Oral Pathology Reference Institute between 1975 and 2005. Cases with different histopathological diagnoses were determined and information about age and gender of the patient, location of associated tooth, pulp status and the histopathological diagnosis were recorded. The percentage of nonendodontic periapical lesions was then determined.

Results In the 30-year period, 43 706 biopsy specimens were received. Overall 4006 (9.13%) had a clinical diagnosis of pulpal necrosis with associated pathosis in the periradicular area. Within this group, 26 cases (0.65%) had a histopathological diagnosis of nonendodontic pathology. Keratocystic odontogenic tumour was the most frequent nonendodontic lesion (11 cases) in the periradicular region followed by central giant cell granuloma (three cases), chronic sinusitis (three cases) and one case each of the following lesions: nasopalatine duct cyst, lateral periodontal cyst, calcifying cystic odontogenic tumour, ameloblastic fibroma, squamous odontogenic tumour, cemental dysplasia, haemangioma, foreign body cell granuloma and amalgam tattoo.

Conclusions The histopathological study of periapical pathosis can occasionally reveal nonendodontic lesions. Odontogenic tumours made up the largest group.

Keywords: keratocystic odontogenic tumour, periapical misdiagnosis, periapical lesions.

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Introduction

Most pathosis in the periapical region is a consequence of dental pulp necrosis. However, there are other pathoses that occur in this site with no relationship to the pulp condition (Dahl 1991, Kuc *et al.* 2000, Peters & Lau 2003). These nonendodontic lesions will not heal following root canal treatment.

The distinction between endodontic and nonendodontic lesions in a periapical area is important as a variety of pathologic alterations have been reported for the latter; such as keratocystic odontogenic tumours (KCOTs) (previously named odontogenic keratocyst) (Garlock *et al.* 1998, Ali & Baughman 2003), ameloblastoma (Cunha *et al.* 2005), central giant cell granuloma (CGCG) (or central giant cell lesion) (Dahlkemper *et al.* 2000, Lombardi *et al.* 2006) intraosseous haemangioma (Orsini *et al.* 2000) and malignancies such as carcinoma of the antrum (Copeland 1980) and central adenoid cystic carcinoma (Favia *et al.* 2000). These pathoses behave differently and require treatment linked to other specialists; such as maxillofacial surgeons, oncologists, etc.

The purposes of this retrospective study were to determine: (i) the frequency with which histopathological diagnosis of periapical lesions change the clinical diagnosis and (ii) the incidence of different lesions in specimens diagnosed at the Oral Pathology Reference

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Material and methods

All periapical biopsy specimens with a clinical diagnosis resulting from pulp necrosis between 1975 and 2005 were taken from the IREPO archives. The cases with a histopathological diagnosis of nonendodontic lesions were selected and their clinical and histopathological information was reviewed for an update of biopsy diagnoses according to the World Health Organization Classification of Tumours (WHO) 2005. Clinical data included age and gender of the patient, location and size of the lesion, pulp status, clinical and histopathological diagnosis.

Results

Of the 43 706 biopsies of the IREPO between 1975 and 2005, 4006 (9.13%) had a clinical diagnosis associated with a sequelae of pulpal necrosis in the periapical area. Of these, 26 cases (0.65%) had a histopathological diagnosis of nonendodontic pathosis (Table 1). Information on dental status obtained from the archives of these 26 cases was: pulp necrosis in five cases, root-filled tooth in five cases and undetermined status in 16 cases.

According to the World Health Organization Classification of Tumours (WHO) 2005 classification, 14 lesions were odontogenic tumours as follows: 11 KCOTs, one ameloblastic fibroma, one calcifying cystic odontogenic tumour (Gorlin cyst) and one squamous odontogenic tumour.

Two cysts were found, one lateral periodontal cyst and the other a nasopalatine duct cyst. The other nine lesions were: three chronic sinusitis, three CGCG, one haemangioma, one amalgam tattoo, one cemental dysplasia (periapical osseous dysplasia) and one case of foreign body granuloma. The largest lesions were one KLOT and one CGCG, both involving five teeth.

From the 26 histopathological diagnoses of non-endodontic lesions the most common were KCOTs (42.3%), observed in eight women and three men with an age range of 14–73 years. Of these 11 KCOTs six were located in the maxilla, five of them in the anterior portion of the maxilla and one in the posterior portion; the other five were in the molar region of the mandible.

Table 2 shows the distribution of nonendodontic periapical lesions in the present study and a Canadian study (Kuc *et al.* 2000). The incidence of these lesions, are expressed as a percentage of the total number of cases with pulpal necrosis with periradicular pathosis sent for a histopathological diagnosis. No malignant lesions were found.

Discussion

Most studies of periapical lesions show that between 75% and 95% are complications resulting from pulpal necrosis and that the majority would respond to conservative root canal treatment (Kuc *et al.* 2000, Peters & Lau 2003). For example, in 70% of cases (Murphy *et al.* 1991) healing of these periapical radiolucencies occurred at least 12 months after nonsurgical root canal treatment. Some studies have reported pathologic entities associated with root apexes examples being: KCOT (Nohl & Gulabivala 1996, Garlock

Table 1 Distribution of nonendodontic periapical lesions and clinical parameters

		Gender			Location	
Histologic diagnosis	Cases, *n* (%)	F	M	Age range (years)	Maxilla	Manbible
Keratocystic odontogenic tumour	11 (42.3)	8	3	14–73	6	5
Chronic sinusitis	3 (11.5)	2	1	12–53	3	
Central giant cell granulomas	3 (11.5)	1	2	40–76	2	1
Nasopalatine duct cyst	1 (3.8)	0	1	40	1	
Foreign body granuloma	1 (3.8)	1	0	74	1	
Calcifying cystic odontogenic tumour	1 (3.8)	1	0	39		1
Cemental dysplasia periapical	1 (3.8)	1	0	52	1	
Periodontal lateral cyst	1 (3.8)	0	1	31	1	
Haemangioma	1 (3.8)	0	1	21		1
Amalgam tattoo	1 (3.8)	1	0	50	1	
Ameloblastic fibroma	1 (3.8)	0	1	9		1
Squamous odontogenic tumour	1 (3.8)	0	1	29	1	
Total	26 (100)	15	11	9–76	16	10

Table 2 Comparison of two series of nonendodontic periapical lesions

Histologic diagnosis	Present study (2007) (Chile), *n* (%)	Kuc *et al.* (2000) (Canada), *n* (%)
Keratocystic odontogenic tumour	11 (0.27)	0 (0)
Chronic sinusitis	3 (0.07)	0 (0)
Central giant cell granulomas	3 (0.07)	2 (0.25)
Nasopalatine duct cyst	1 (0.02)	1 (0.2)
Calcifying cyst odontogenic tumour	1 (0.02)	0 (0)
Foreign body granuloma	1 (0.02)	0 (0)
Periodontal lateral cyst	1 (0.02)	1 (0.12)
Ameloblastic fibroma	1 (0.02)	0 (0)
Haemangioma	1 (0.02)	0 (0)
Amalgam tattoo	1 (0.02)	0 (0)
Cemental dysplasia periapical	1 (0.02)	0 (0)
Squamous odontogenic tumour	1 (0.02)	0 (0)
Fibro-osseous lesion	0 (0)	1 (0.12)
Calcifying epithelial odontogenic tumour	0 (0)	1 (0.12)
Myxoma odontogenic	0 (0)	1 (0.12)
Multiple myeloma	0 (0)	1 (0.12)
Total	26 (0.65)	8 (1)

et al. 1998, Ali & Baughman 2003), CGCG (Dahlkemper *et al.* 2000, Lombardi *et al.* 2006), ameloblastoma (Navarro *et al.* 2004, Cunha *et al.* 2005), paradental cyst (Silva *et al.* 2003), haemangioma (Orsini *et al.* 2000), glandular tissue (Childers *et al.* 1990), squamous cell carcinoma (Copeland 1980) and adenoid cyst carcinoma (Favia *et al.* 2000).

The present retrospective study found that KCOTs have a larger potential for endodontic misdiagnosis. This finding is supported by Stajcic & Paljm (1987) who by histological means, studied 565 specimens of the periapical area around teeth with necrotic pulps that were either root filled or extracted. They found that odontogenic keratocysts represented 0.7% of the clinically and radiographically diagnosed radicular cysts.

Epidemiological reports, before the World Health Organization Classification of Tumours (WHO) 2005 classification, described keratocysts as the second most frequent developmental odontogenic cyst after the dentigerous cyst (Mosqueda *et al.* 2002).

It has been reported that 20% of maxillary KCOT's were in a periapical region. However, if both jaws are included the incidence in a periapical site decreases to 9% (Garlock *et al.* 1998). In the present study, no substantial differences were found between jaws, six cases were seen in the maxilla and five cases in the mandible. The KCOT is located in the posterior mandible in approximately 60–80% of cases (Neville *et al.* 2002). When this tumour is located in the maxilla, the canine region is the most frequently affected, 41% (Ali & Baughman 2003). This low frequency in the maxilla may have contributed to clinical misdiagnoses. However, in this study the large size of the KCOT was the main clinical feature signalling a different lesion.

Keratocystic odontogenic tumour is important because of its frequent recurrence and aggressive behaviour (Garlock *et al.* 1998, Peters & Lau 2003). Moreover, it could be associated with nevoid basal cell carcinoma syndrome or Gorlin syndrome (Neville *et al.* 2002). In the present study one recurring case was found after 8 years, no cases were associated with the syndrome.

The misdiagnosis of CGCG as an odontogenic cyst has been reported in previous studies (Dahlkemper *et al.* 2000, Lombardi *et al.* 2006). Dahlkemper *et al.* (2000) studied data of biopsy specimens diagnosed as CGCG, and found 20% associated with pulp necrosis; of these cases, 88% received root canal treatment prior to the final diagnosis. In the Lombardi *et al.* (2006) study two of four cases had received root canal treatment.

In the three cases of sinusitis found in this study, the possibility of an oral/dental infection could not be excluded. Although maxillary sinusitis is primarily considered to be of rhinogenous origin, dental infection can be a pre-disposing factor (Garming *et al.* 2004).

The 0.65% of nonendodontic periapical lesions found in this study is close to the 1% found in the study of Kuc *et al.* (2000) in which the specimens were also from an Oral Pathology Laboratory at Alberta University, Canada.

In at least two-thirds of the cases the teeth near the lesion had necrotic pulp or had received root canal treatment, thus there is a substantial coincidence of pulp and periapical pathosis with nonendodontic pathosis.

The histopathological diagnosis of periradicular lesions has been a subject of extensive debate (Walton 1998, 1999, 2000, Baughman 1999, Ellis 1999, Newton 1999, Ramer 1999, Summerlin 1999, Talacko *et al.* 2000). Although in this work the percentage of nonendodontic periapical lesions was low and only nonmalignancies were found, the consequences of these pathoses for the patient can be severe and therefore histopathological examination of all surgical specimens should be performed. Periapical radiolucencies that do not heal after root canal treatment or with an incomplete clinical history or with unusual radiographic image should receive surgical treatment and biopsy.

Conclusion

These results emphasize the importance of following up patients with periapical radiolucencies who have received root canal treatment and the need for histopathological study of every periapical lesions that is refractory to root canal treatment.

The histopathological study of periapical lesions can occasionally reveal nonendodontic lesions with different clinical prognosis. The odontogenic tumours make up the largest group, with the keratocystic odontogenic tumour being the most frequent nonendodontic lesion.

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