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ORIGINAL ARTICLE

Chronic periapical lesions: clinical and morphological characterization of 508 cases

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Abstract:

Objective: To evaluate the clinical and morphological findings of chronic periapical lesions (CPL) composed of periapical granuloma (PG), radicular cyst (RC), and residual radicular cyst (RRC). **Methods:** Sample was collected from an Oral Pathology Service over 10-year period. Histomorphological analysis was performed qualitatively. For estatistical analysis were used Fischer's exact and Q2 tests, considering value $p<0.05$. **Results:** Among the 4.808 cases retrieved from the service, 508 (10.6%) cases were CPL. PG was the most frequent lesion (n=264; 51.96%). RC and PG were more prevalent among young adults, while RRC affected older patients ($p<0.0001$). Morphologically, lower grade of intensity of the inflammatory infiltrate (Grade I) was founded in RRC, compared to PG and RC (Grade II/III) ($p<0.0001$). Atrophic epithelium was more present in RRC, whereas hyperplastic epithelium was more observed in RC ($p=0.012$). Russell bodies and foamy macrophages were more frequent in PG ($p<0.05$). Rushton bodies and negative images of cholesterol crystals were more observed in RC ($p<0.05$). **Conclusions:** CPL are a common condition frequently associated with persistent endodontic infection and represent a significant clinical challenge in dental practice. Therefore, clinical and morphological comprehensive characterization of CPL is important for their correct diagnosis and treatment.

Keywords: Odontogenic cyst; Radicular cyst; Periapical granuloma; Residual radicular cyst.

INTRODUCTION

Chronic periapical lesions (CPL) refer to chronic inflammatory reactions that surround the tooth apex and are caused by infections in the root canal, when the bacteria involved in the etiopathogenesis of these infections and their toxic products pass through the apical foramen and to induce inflammation in connective tissue[1,2]. If the stimulus persists, the lesion can progress to one of its chronic forms, periapical granuloma (PG) and radicular cyst (RC)[3].

Clinically, PG is characterized as an asymptomatic lesion. Sporadic episodes of exacerbation associated with secondary infection have been reported[4]. Radiographically, PG appears as a well-delimited radiolucent lesion with sclerotic margins[5]. The histopathological characteristics of PG include granulation tissue containing a predominantly mononuclear intense inflammatory infiltrate, permeated by vascularized dense fibrous connective tissue. Epithelial rests of Malassez are present in some cases[6,7].

Statement of Clinical Significance
This study provides a detailed clinicopathological characterization of chronic periapical lesions, offering valuable diagnostic, epidemiological, and prognostic insights that enhance understanding of their biological behavior and support more accurate clinical decision-making in endodontic and periapical surgical management.

RC are inflammatory odontogenic cysts associated with a non-vital tooth that are generally asymptomatic and are discovered during routine examination[2,8]. Histopathologically, RC are characterized by a cystic cavity lined with non-keratinized stratified squamous epithelium with varying degrees of hyperplasia and occasional arch-shaped projections[9]. The cystic capsule is composed of fibrous connective tissue, permeated by a chronic inflammatory infiltrate of variable intensity[10].

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Residual radicular cysts (RRC) are cysts that remain in the gnathic bones after extraction of the tooth associated with a RC, but when the residual tissue was not properly removed by curettage. The histopathological characteristics of these lesions are similar to those of RC. In most cases, RRC exhibit a non-keratinized, atrophic, stratified squamous epithelial lining and a cystic capsule containing a less intense chronic inflammatory infiltrate[11].

CPL are a common condition in Brazil[1], that cause bone resorption[12]. In light of this context, gaining a better understanding of the clinical and morphological aspects is importance to more effectively elucidate its pathogenesis. In the literature, it possible to find some studies that investigated clinical findings of PG, RC and periapical abscess[1], as well as morphological findings of RC[2,13,14]. However, studies that investigate both clinical and morphological characterization of PG, RC, and RRC still are scarce.

Within this context, the present study evaluated the main morphological findings of a series of cases of PG, RC and RRC by histopathological, comparing them with clinical characteristics, to contribute to a better understanding of the clinical and morphological profile of these lesions.

MATERIAL AND METHODS

Ethical aspects

The study was approved by the Human Research Ethics Committee of UEPB, Campina Grande, Brazil, Approval Protocol Number: 4,197,082, in accordance with the Declaration of the Helsinki. As this was a retrospective study, the Consent to participate declaration was not required.

Study design

This was a cross-sectional, retrospective and observational study with semi-quantitative analysis of the morphological features of PG, RC, and RRC cases. The study population consisted of all cases diagnosed and stored at the Laboratory of Oral Histopathology, Department of Dentistry, UEPB (Campus I, Campina Grande) from 2011 to 2021.

Sample

Paraffin-embedded cases of PG, RC, and RRC diagnosed and stored at our service were obtained by convenience (non-probability) sampling. All cases were diagnosed clinically and morphologically as CPL. Including cases obtained from alveolar curettage after dental extraction.

Cases of PG, RC and RRC with sufficient amounts of biological material for morphological analysis were included in the sample. For RC and RRC, specimens exhibiting a pathological cavity lined completely or partially with non-keratinized, stratified squamous epithelium and sufficient amounts of a fibrous capsule for analysis were included. Cases of PG presenting remnants of odontogenic epithelium were excluded to avoid overlap with lesions that could represent early or developing RC. Cases of PG and RC associated with teeth previously submitted to endodontic treatment were excluded from the study to avoid potential confounding factors related to treatment-induced changes.

Clinical and morphological study

Patient sex and age and anatomical location of the lesions were obtained from the patient records.

Histomorphological features were analyzed in hematoxylin/eosin-stained 5-µm-thick sections under a light microscope (Leica DM 500, Leica Microsystems Vertrieb GmbH, Wetzlar, Germany) at 40x, 100x and 400x magnification. The following histopathological characteristics were analyzed by a previously trained examiner: intensity of the inflammatory infiltrate in connective tissue/capsule, pattern of the epithelial lining, multinucleated giant cells, foamy macrophages, Rushton and Russell bodies, negative images of cholesterol crystals, microorganism colonies, hyaline ring granuloma, odontogenic epithelial remnants, and foreign body granuloma.

The intensity of the inflammatory infiltrate was classified following the criteria of as grade I, II or III according to distribution and quantity[10]. For RC and RRC, the pattern of the epithelial lining was classified as atrophic (2 to 10 layers of cells) or hyperplastic (more than 10 layers of cells forming arch-shaped projections)[15]. The other morphological features (vacuolized cells, multinucleated giant cells, foamy macrophages, Rushton bodies, Russell bodies, negative images of cholesterol crystals, microorganism colonies, hyaline ring granuloma, remnants of odontogenic epithelium, foreing body granuloma and mucosal cells) were classified as present or absent.

Statistical analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS, version

21.0; IBM Corp., Armonk, NY, USA). Fisher's exact test, chi-squared test and Mann-Whitney test were used, adopting a p value <0.05.

RESULTS

Among the 4,808 cases diagnosed at the oral pathology service of UEPB from 2011 to 2021, 508 (10.56%) were CPL; of these, 264 (51.96%) were PG, 229 (45.07%) were RC, and 15 (2.95%) were RRC.

There was a predominance of women in all groups of lesions. Regarding anatomic location, the posterior mandible was the most affected site in PG (n=82; 31.1%) and RC (n=70; 30.6%), while the anterior maxilla was more frequently affected in RRC (n=7; 46.7%) (Table 1).

Table 1. Clinical and pathological aspects of the 508 chronic periapical lesions.

Clinical aspects	Lesions						p-value
	PG		RC		RRC		
	(n)	(%)	(n)	(%)	(n)	(%)	
Sex							
Male	111	42	90	39,3	7	46.7	p=0.744*
Female	153	58	139	60.7	8	53.3	
Mean of age	37.43+15.85 (4-84)		36.92+16.15 (6-78)		57+15.77 (17-76)		p= 0.729[a] **p<0.0001**[b] **p<0.0001**[c]
Lesion location							
Maxilla	126	55	118	44.6	9	60	p=0.884*
Mandible	95	41.4	81	30.6	6	40	
Type of epithelium							
Atrophic	N/A		51	22.3	8	53.3	**p=0.012**[+]
Hyperplastic	N/A		178	77.7	7	46.7	
Pseudostratified ciliated epithelium	N/A		9	3.9	2	13.3	
Squamous odontogenic tumor-like proliferations	N/A		4	1.7	0	0	
Intensity of inflammatory infiltrate							
Grade I	18	22.3	51	22.3	10	66.7	**p<0.0001***
Grade II/III	246	93.1	177	77.2	5	33.3	
Morphological aspects							
Vacuolized cells	2	0.8	5	2.2	0	0	
Multinucleated giant cells	31	11.7	29	12.7	3	20	p=0.632*
Foamy macrophages	84	31.8	45	19.7	2	13.3	**p=0.005***
Rushton bodies	10	3.8	25	10.9	2	13.3	**p=0.007***
Russell bodies	160	60.6	84	36.7	1	6.7	**p<0.0001***
Negative images of cholesterol crystals	20	7.6	31	13.5	6	40	**p<0.0001***
Microorganism colonies	12	4.5	7	3.1	0	0	p=508*
Hyaline ring granuloma	9	3.4	5	2.2	0	0	p=570*
Remnants of odontogenic epithelium	N/A		30	13.1	2	13.3	p=508*
Foreing body granuloma	2	0.8	0	0	0	0	p=395*
Mucosal cells	N/A		4	1.7	1	6.7	p=511*

PG: periapical granuloma; RC: radicular cyst; RRC: residual radicular cyst; N/A: not applicable.
p<0.05 indicates a statistically significant difference and is highlighted in bold.
*Pearson χ^2; [+]Test exact fisher´s.
[a,b,c]Mann-whitney test; [a]RC x PG, [b]RC x RRC, [c]PG x RRC.

The mean patient age was 37.43±15.85 years (4-84) and 36.92±16.15 years (6-78) in cases of PG and RC, respectively, while patients with RRC were older [57±15.77 years (17-76)], with a significant difference between these groups (p<0.0001) (Table 1).

Analysis of the morphological findings showed a higher frequency of grade II/III inflammatory infiltrates in PG (n=246; 72.3%) and RC (n=112; 48.9%) and of grade I infiltrates in RRC (n=10; 66.7%), with a statistically significant difference between groups (p<0.0001) (Table 1). There was a significant difference in the type of epithelium between RC and RRC, with a predominance of hyperplastic epithelium in the former and atrophic epithelium in the latter (Table 1, Figure 1).

We also found a higher frequency of Russell bodies, negative images of cholesterol crystals, foamy macrophages, and Rushton bodies in PG and RC, with a significant difference compared to RRC (p<0.0001, p<0.0001, p=0.005 and p=0.007, respectively) (Table 1, Figure 2).

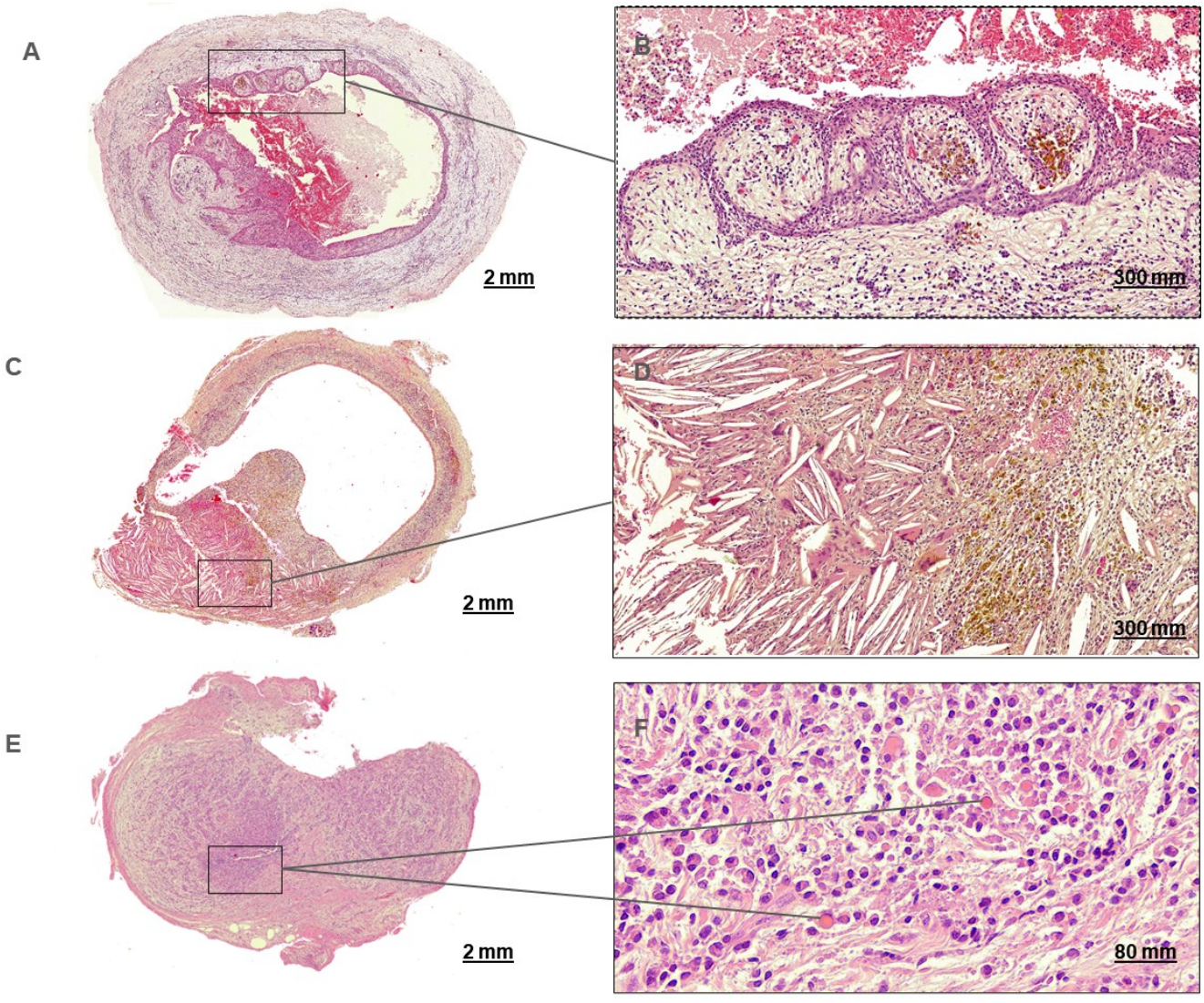

Figure 1 (A, B) Micrographs of radicular cyst showing hyperplastic stratified squamous epithelial lining. **(C)** Residual radicular cyst with atrophic epithelial lining. **(D)** At higher magnification, residual radicular cyst with details of negative images of cholesterol crystals associated with numerous multinucleated giant cells. Intense deposition of brownish pigmentation consistent with hemosiderin. **(E)** At lower magnification, periapical granuloma showing grade III inflammatory infiltrate, predominantly mononuclear, and **(F)** At higher magnification, periapical granuloma showing inflammatory infiltrate composed predominantly of lymphocytes and plasmocytes. Russell bodies are observed (arrows) (hematoxylin and eosin).

In addition, a statistically significant association was observed between the degree of the inflammatory infiltrate and the type of epithelium in RRC (p=0.007), with a predominance of a grade I inflammatory infiltrate in lesions with atrophic epithelium (Table 2).

In RC group, the degree of inflammatory infiltrate was significantly associated with the presence of Russell bodies (p=0.0001), with a predominance of grade II/III when these bodies were present. In PG group, there was an association of grade II/III inflammatory infiltration with the presence of foamy macrophages (p=0.001) (Table 3).

DISCUSSION

Our clinical-pathological study shows that CPL are common lesions in oral pathology services

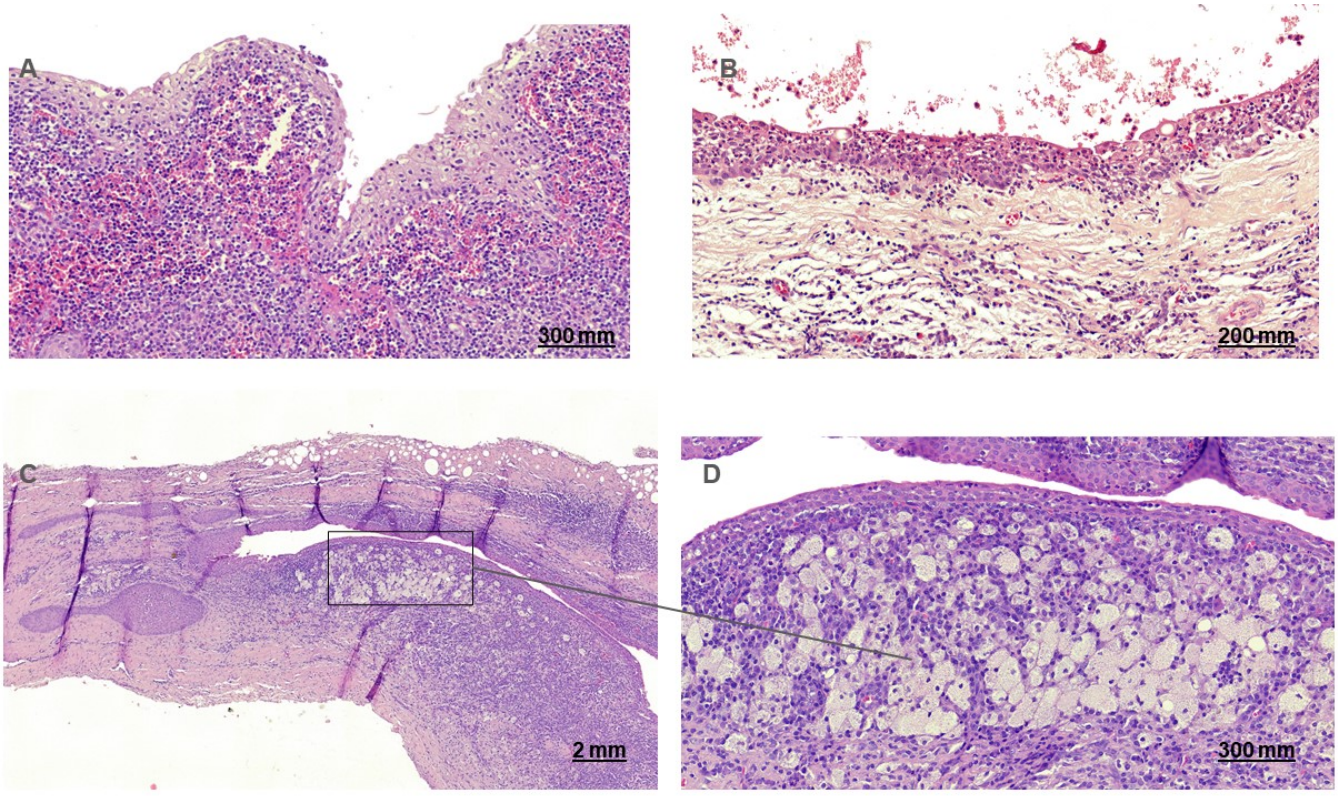

Figure 2. (A) Micrographs of radicular cyst showing hyperplastic stratified squamous epithelial lining and capsule with grade III inflammatory infiltrate. **(B)** Residual radicular cyst with atrophic epithelial lining and capsule with grade I inflammatory infiltrate. **(C)** Radicular cyst with hyperplastic stratified squamous epithelial lining, capsule with grade III inflammatory infiltrate and epithelial plaque resembling squamous odontogenic tumor. **(D)** At higher magnification, radicular cyst with details showing numerous foamy macrophages (hematoxylin and eosin).

Table 2. Association of morphological parameters: intensity of inflammatory infiltrate x type of epithelium.

Type of epithelium	Intensity of inflammatory infiltrate		
	I	II/III	p-value
RC			
Atrophic	16	35	p=0.128*
Hyperplastic	36	142	
RRC			
Atrophic	8	0	**p=0.007***
Hyperplastic	2	5	

RC: radicular cyst; RRC: residual radicular cyst.
p<0.05 indicates a statistically significant difference and is highlighted in bold.
*Test exact fisher´s.

Table 3. Association between lesion type, Intensity of the inflammatory infiltrate, and the presence of Russell bodies and foamy macrophages.

Variable		Intensity of inflammatory infiltrate		p-value
		I	II/III	
Russell bodies				
PG	P	9	151	p=0.454*
	A	9	95	
RC	P	9	75	**p=0.001***
	A	43	102	
RRC	P	0	1	p=0.333*
	A	10	4	
Foamy macrophages				
PG	P	0	84	**p=0.001***
	A	18	162	
RC	P	8	37	p=0.433*
	A	44	140	
RRC	P	1	1	p=1.000*
	A	9	4	

PG: periapical granuloma; RC: radicular cyst; RRC: residual radicular cyst; P: present; A: absent.
$p<0.05$ indicates a statistically significant difference and is highlighted in bold.
*Test exact fisher´s.

considering that, at our service, 10.6% of the 4,808 cases were CPL. An important aspect is that our study involved the largest sample of CPL among the studies found in the literature[1,11-14] and these cases were characterized clinically and histopathologically. Furthermore, Chen et al.[13] and Santos et al.[14] evaluated clinical and morphological features only in RC, while our study assessed cases of PG, RC, and RRC, thus highlighting the importance of the data found here for correct characterization of the clinical and histopathological features of CPL.

Among all CPL evaluated, PG were the most frequent, accounting for approximately 52% of cases. In contrast, Omoregie et al.[16], Couto et al.[1] and Alotaibi et al.[17] identified RC as the most common lesions. This high frequency of CPL may be related to the high caries rates in Brazil, which cause high tooth loss and mainly affect the first permanent lower molar in adolescents and younger individuals[18]. Interestingly, in our study, similar to Couto et al.,[1] RC and PG were more prevalent among young adults, while RRC affected older patients. This finding suggests a distinct clinical behavior of RC and RRC, which requires further research.

Although CPL are common in clinical practice, biopsies of these lesions are rarely sent for histopathological diagnosis[1]. According to Omoregie et al.[16], many dentists continue to question the real need for performing biopsies of periradicular lesions. It is believed that routine biopsy of CPL is necessary if the clinical diagnosis of these lesions is uncertain and in the case of an inadequate response to endodontic treatment. However, it is important to note that CPL can be large, causing bone resorption that may compromise adjacent teeth[12], Hence, awareness of dentists is the key pillar for the correct referral and diagnosis of CPL.

Histopathologically, RC and RRC appear as a pathological cavity lined with epithelial tissue[19] where epithelial proliferation is stimulated by interleukins and growth factors such as IL-1, IL-6, keratinocyte growth factor (KGF) and transforming growth factor (TGF)[20]. This epithelial proliferation is associated with vascular fibrous connective tissue exhibiting different degrees of an inflammatory infiltrate[20]. In our study, RC mostly exhibited hyperplastic epithelium and a grade III inflammatory infiltrate, which may be related to the intense metabolic activity of these lesions[21]. Within this context, the balance between proinflammatory and anti-inflammatory cytokines is responsible for controlling host responses to antigen stimulation in chronic inflammatory processes[22]. This fact may also explain the findings regarding RRC, which exhibited atrophic epithelium and a grade I inflammatory infiltrate, indicating that the main antigenic stimuli are no longer present and

reinforcing our suggestion for evaluating these lesions as distinct entities.

In our study, the epithelial lining of RC and RRC was a non-keratinized stratified squamous epithelium, sometimes exhibiting mucous and ciliated cells, similar to the findings reported in the literature[9,14,23]. It is believed that these mucous cells are of metaplastic origin but the exact cause of their development is unknown[23]. Ciliated cells are described in the literature as the probable cause of epithelial metaplasia triggered by the presence of remnant respiratory tissues during embryogenesis or inflammatory stimuli in the epithelium of these lesions[14,23]. Another finding was the presence of squamous inclusions in the cystic capsule of RC, which is an uncommon morphological finding[14,23,24]. The pathogenesis of these inclusions is still poorly understood; however, it is believed that they originate from the epithelial rests of Malassez[24]. Although these inclusions do not alter the biological behavior of the lesion, they resemble the histological characteristics of odontogenic neoplasms such as squamous odontogenic tumor[24]. Therefore, an incorrect interpretation of these histopathological findings in RC can lead to diagnostic errors and consequent inadequate treatments.

CPL are characterized by the presence of an inflammatory infiltrate composed mainly of mononuclear cells[8], such as lymphocytes, plasma cells and macrophages. Weber et al.[25] comment that macrophages are polarized to two different phenotypes: M1 and M2. M1 macrophages are effector cells that participate in proinflammatory processes, while M2 macrophages have the capacity to inhibit cytotoxic and inflammatory functions. França et al.[9] suggested that the polarization of these cells to interfere with epithelial proliferation in inflammatory cysts. In our study, the observation of a significant association between atrophic epithelium and a grade I inflammatory infiltrate in cases of RRC may suggest a possible relationship to polarization to M2 macrophages.

Macrophages that phagocytose cholesterol crystals are called foamy macrophages[26,27]. After phagocytosis of these cholesterol crystals, these macrophages can induce the release of IL-1 which, in turn, has a well-established role in bone resorption and in the persistence of the lesion[15,26]. The observation in our study of a significant difference in the presence of foamy macrophages with major amount on the PG, suggests that these cells play an important role in the persistence of these periapical lesion.

Several origins of cholesterol crystals have been proposed, including their origin from the granulation tissue of the cystic wall, from phagocytosis of the membrane of cells that have undergone cell death, or from lipid crystallization[14]. Histologically, these cholesterol crystals can appear as cytoplasmic inclusions in foamy macrophages or as negative images since they are lost during the histological processing of tissues[15,26,27]. The presence of negative images of cholesterol crystals and foamy macrophages may indicate the inflammatory nature of the lesions, as observed in our study in which these morphological findings were observed in cases of PG, RC and RRC.

Another morphological finding observed in our study were Rushton bodies, which were more frequent in RC. Rushton bodies exhibit morphological variety and may appear as linear, spherical, or concentric eosinophilic structures, located within the epithelial lining of odontogenic cysts, particularly inflammatory cysts[13,14], and, less common, in the cystic capsule[28]. Regarding their incidence, Babburi et al.[28] report that Rushton bodies occur in approximately 10% of cases of RC. This percentage is similar to that found in our study in which these structures were observed in 7% of CPL, with a significant difference in cases of RC. The origin of Rushton bodies is widely discussed in the literature and there are several theories, including their origin from hyaline degeneration of newly formed capillaries or the hypothesis that they represent secretory products of the odontogenic cystic epithelium[28]. The latter is the most plausible theory considering that Rushton bodies are found almost exclusively in odontogenic cysts.

Russell bodies were also a common morphological finding, observed in 48% of CPL cases in our study. In contrast, Santos et al.[14] detected the presence of Russell bodies in only 9.5% of cases of RC. Morphologically, Russell bodies appear as intra- or extracellular small, spherical, eosinophilic structures. In CPL, it is believed that Russell bodies are induced by the infectious process of the pulp tissue that hyperactivates plasma cells in the periapical region, with subsequent overproduction of immunoglobulins in cisterns of the endoplasmic reticulum[29]. The findings of our study corroborate this view, in which Russell bodies were much more frequent in cases of PG and RC, which are more active inflammatory lesions than RRC.

In summary, our study represents so far, the largest sample of CPL cases described in the literature, a fact that permits a comprehensive analysis of clinical data and of all morphological findings related to each lesion and, consequently, the establishment of a diagnostic profile of CPL.

CONCLUSION

CPL are a common condition frequently associated with endodontic infection and represent a significant clinical challenge in dental practice. The present results led us to conclude that the incidence of CPL is high and that a good understanding of their epidemiology and clinical and morphological characteristics is extremely valuable for the correct diagnosis of these lesions.

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AUTHORS' CONTRIBUTIONS

TBR: Conceptualization, Data curation, Writing – original draft. RKLM: Data curation, Investigation. NYAH: Data curation, Investigation, Methodology. JLSC: Formal analysis, Methodology, Validation, Visualization. MAGN: Validation, Visualization. CFWN: Formal analysis, Software, Validation, Visualization. PMA: Project administration, Supervision, Validation, Writing – review & editing.

CONFLICT OF INTEREST STATEMENT

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Competing interests: The authors have no relevant financial or non-financial interests to disclose.
Ethics approval: The study was approved by the Human Research Ethics Committee of UEPB, Campina Grande, Brazil, Approval Protocol Number: 4,197,082.

DATA AVAILABILITY STATEMENT

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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