Importance of endodontic diagnosis regarding periapical cemento-osseous dysplasia: literature review

Adriana dos Santos **BELO**[1]
Rogério Vieira **SILVA**[2]
Renato Piai **PEREIRA**[3]
João Cláudio Borges **RODRIGUES**[2]

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ABSTRACT

Introduction: Periapical cemento-osseous dysplasia (PCOD) is a benign idiopathic lesion affecting more prevalently the region of mandibular incisors in women aged between 30 and 50 years old. PCOD has radiographic characteristics which may lead the general dentist to make mistakes including treatment planning, in case this lesion is diagnosed as one of the periapical diseases. **Objectives:** Therefore, the aim of this study was to describe this pathology by reviewing the literature. **Methods:** This literature review was conducted based on PubMed and SciELO, two of the largest databases in the world. Descriptors in English ("periapical cemento-osseous dysplasia") and Portuguese ("displasia cemento-óssea periapical") were used to search for the articles in order to evaluate the issue. **Results:** Twenty-four scientific articles were selected based on the following inclusion criteria: literature review or clinical case, written in English or Portuguese, published within the 1989-2016 period and describing aetiology, clinical and radiographic characteristics, diagnosis, treatment planning and prognosis regarding PCOD. **Conclusion:** It is important that the professional recognize the relevant aspects of PCOD, in order to elucidate the differential diagnosis and treatment and thus, prevent iatrogenic procedures such as unnecessary endodontic therapies.

Keywords: Diagnosis. Endodontics. Pathology. Oral.

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[1]Private practice (Salvador/BA, Brazil).
[2]Faculdade Independente do Nordeste, Curso de Odontologia (Vitória da Conquista/BA, Brazil).
[3]Universidade Estadual do Sudoeste da Bahia, Curso de Odontologia (Jequié/BA, Brazil).

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Contact address: Rogério Vieira Silva
Faculdade Independente do Nordeste (FAINOR)
Av. Luís Eduardo Magalhães, 1305, Candeias – Vitória da Conquista/BA
E-mail: roger.endo@hotmail.com

Introduction

Periapical cemento-osseous dysplasia (PCOD) is the most common form of cemento-osseous dysplasia presenting as an idiopathic lesion in which bone is replaced by non-neoplastic fibrous-osseous tissue.[2,16,18] Although its cause is unknown, some authors believe that it originates from the periodontal ligament,[19] whereas others state that it represents an uncommon reaction of the periapical bone.[4] The lesion typically affects the periapical area of lower incisors in black middle-aged women, but not exclusively.[17,19] Cases involving family as well as white women are described in the literature.[11,16]

This lesion is usually asymptomatic and is found in radiographic dental examination as a well-delimited radiolucent area close to the root apex, also appearing as a mixture of radiolucent/radiopaque area or radiopaque lesion depending on the phase in which it is diagnosed.[2,19] Despite being a well-recognised condition, patients are still submitted to endodontic therapy because of the equivocal diagnosis of endodontic origin lesions.

The diagnosis of PCOD is based on clinical and radiographic findings. Because the lesion is asymptomatic, it is usually found out incidentally when radiographic examination is performed for other purposes.[5,10,11,14,16,21] Pulp sensitivity tests are expected to prevent these mistakes as the teeth involved are healthy.[9,14] There is a general consensus that the PCOD requires no treatment. Therefore, the dental practitioner is supposed to perform a radiographic follow-up as the lesion rarely grows progressively.[4] Biopsy is not usually necessary in the cases of PCOD,[2] since its diagnosis is mainly achieved based on clinical and radiographic characteristics of the lesion.[16]

When clinical and radiographic findings are not associated with each other and there is little knowledge on this lesion, mistakes may occur due to equivocal diagnosis and lead to unnecessary endodontic treatment. It is of fundamental importance to carry out a clinical examination to determine the correct diagnosis of PCOD, with pulp sensitivity test being performed, rather than only radiographic evaluation, for establishing a treatment planning.[22]

The objective of the present article was to address relevant aspects on PCOD by reviewing the related literature, highlighting aetiology, clinical and radiographic characteristics, diagnosis and treatment planning. It is very important that the dental practitioner have knowledge on the relevant aspects of this pathology in order to achieve a differential diagnosis and determine adequate procedures, thus ensuring a high quality dental care.

Methodology

A literature review was performed by using two major databases worldwide, namely, PubMed and SciELO, with the objective of assessing their content in the current literature. Articles with the descriptors in English (i.e. "periapical cemento-osseous dysplasia") and in Portuguese (i.e. "displasia cemento-óssea periapical") were selected, resulting in 24 relevant scientific studies (literature reviews or case reports written in English or Portuguese between the years of 1989 and 2016). The articles had to use only human samples for the study of aetiology, clinical and radiographic characteristics, diagnosis, treatment planning and prognosis regarding periapical cemento-osseous dysplasia.

Results

Aetiology and localization

Osseous dysplasia is defined as a reactive non-neoplastic process developing in the periapical region, characterized by the replacement of normal bone tissue with fibrous tissue and metaplastic bone. This pathological alteration may assume several clinical forms and thus they are given different names. When this process occurs in the periapical region of anterior lower teeth, it is called periapical cemento-osseous dysplasia.[23]

Although its aetiology still remains unknown, some authors believe that this dysplasia represents an unusual reaction of the periapical bone to a local irritating factor, whereas others still suggest that dental trauma, hormonal disorders, systemic and genetic factors may be involved in the development of PCOD.[8]

Case reports show that when the lesion occurs in the periapical region of lower incisors, affecting the adjacent teeth, it is termed as periapical osseous dysplasia. Similar lesions commonly occurring in the posterior region of the mandible or maxilla are termed as focal osseous dysplasia, whereas involving two or more quadrants of the mandible or maxilla are termed as florid or generalized osseous dysplasia.[9]

Clinical, radiologic and histological characteristics

Each lesion is self-limiting,[15] typically with no expansion of cortical bone[13] and rarely exceeding 1.0 cm in diameter in those cases of individual lesions.[5,8] Radiographic and histological assessments show that the lesion is undifferentiated from the fibroma.[3] The majority of these lesions are smaller than 0.5 cm in size[19] and dental mobility rarely occurs.[13] This lesion is sometimes associated with mild chronic trauma[2,20]

The lesions are characteristically asymptomatic, but when they are located close to the mentonian foramen, painful symptoms or paresthesia may occur due to neural compression.[10]

A study described a clinical case in which a 51-year-old Caucasian woman had PCOD involving the lower incisors. These lesions are usually asymptomatic, but the patient presented with mild painful symptoms on palpation and percussion of the involved teeth, with clinical examination showing wear of the incisal borders of these teeth. According to the authors, although mild dental trauma has been suggested to be a possible causal factor for cemento-osseous dysplasia, it is also possible that the symptoms reported by the patient were not related to the presence of dysplasia as the primary cause, but instead to occlusal disorders, which were then treated with occlusal therapy.[16]

In another study conducted with 54 Japanese patients (91% female) presenting cemento-osseous dysplasia in the maxilla, it was found that there was no change in the lesion size (although the amount of lesions might increase), no displacement of teeth involved, and no patient with relevant familial history.[4]

A study by Thakkar, Horner and Sloan[15] reported a case of a family who had PCOD. Four of the seven children of a patient who had the lesion were examined, with three of them presenting similar lesions, predominantly involving the lower incisors. According to the authors, there seems to be a genetic basis for the lesion in this family, with the probable mode of inheritance being autosomal dominant. However, one cannot disregard environmental factors or other inheritance modes without gathering more data to demonstrate scientifically any genetic and familial relationship with cemento-osseous dysplasia.[15]

Still, according to the afore-mentioned study, the lack of early evidence for familial incidence of PCOD may be simple. The asymptomatic nature of the condition contributes to the fact that some cases are not diagnosed because the patients do not seek treatment, which may be the reason why cases of familial dysplasia have not been reported elsewhere. In fact, studies with methodology to demonstrate this genetic relationship are scanty in the literature.[15]

PCOD has a natural evolutive history that may last from months to years, with the lesion having several radiographic aspects depending on the phase in which it is.[10] A series of radiographic studies demonstrated that the first lesion, in many cases, present as a circumscribed radiolucid area[19] due to the formation of fibrous tissue.[10] The adjacent lesions merge over time, forming a linear pattern of radio-transparency covering the apices of several teeth.[18] This initial phase is called osteolytic[16] and exhibits increasing degrees of calcification over the years.[19]

A precise radiographic examination can reveal an intact lamina dura,[14,18] which suggests the conduction of a differential diagnosis for inflammatory periapical lesions.[16] However, Wilcox and Walton[21] disagree with this idea by stating that there is actually loss of lamina dura.

In the second phase, cementum droplets are deposited onto the lesion, resulting in a mixed appearance with radiolucid and radiopaque areas. In the third phase, calcification occurs and a defined radiopacity may appear with a thin radiolucid line.[10,15,21]

Histologically, the lesion possesses connective tissue undergoing various stages of maturation. In the initial phase, it presents a moderately collagenized mass with some circumscribed islets of mineralized cellular cement. In the most advanced phases, the islets increase in number and become grouped together. Normal bone tissue is also produced. The progression of the lesion into a more advanced stage reveals predominance of mineralized material.[10]

Evaluation of the histological appearance shows presence of a varied mixture of fibrous tissue and bone and/or cementum, thus being impossible to make a differential diagnosis for other osseous-fibrous lesions in the upper and lower maxilla. This can explain the fact that the lesion is mainly identified based on clinical examination, especially radiographic resources, rather than on its histological appearance.[10]

Diagnosis and treatment planning

Researchers have reported a clinical case in which twin sisters aged 38 years old presented PCOD. The involved teeth had neither caries nor restorations, but radiographic examination showed circumscribed radiolucid and radiopaque areas close to the apices of the lower incisors. Pulp sensitivity tests for cold, heat and electrical stimuli were positive for both patients. No treatment was planned after determining the diagnosis, except a periodical follow-up for observation. This case may represent the first genetic manifestation of the lesion found in twins, but further investigations and adequate methodologies are needed to determine any relationship.[5]

The majority of the studies on periapical lesions show that from 75 to 95% of them are complications resulting from pulp necrosis.[7] According to Perez, Ramos-Perez and Fregnani,[9] although the majority of periapical lesions are associated with teeth with pulp necrosis, from 0.6 to 4% of them are not of endodontic origin. Therefore, non-endodontic peri-radicular lesions represent a challenge for the dental practitioner as they can potentially be poorly diagnosed, leading to an inadequate treatment as a result. Therefore, a comprehensive clinical examination should be performed to disregard other types of lesions other than those of endodontic origin, thus avoiding diagnostic errors.[9]

The diagnostic process comprises a series of analyses, including epidemiological data, detailed anamnesis and judicious clinical-radiographic examination of the patient. Data on age, gender, skin colour, medical history, current disease status and occasional signals and symptoms should be carefully recorded. It is crucial to associate all the data gathered because there are lesions presenting well-defined clinical-radiographic profiles, as is the case of PCOD, thus assisting in the determination of a definitive diagnosis.[9]

The association of clinical findings with radiographic ones prevents errors, since osseous-cement dysplasia cannot be radiographically differentiated from a lesion resulting from pulp necrosis (e.g. periapical granuloma or periapical cyst) in the initial phase (osteolytic), leading to a poor diagnosis and consequently to unnecessary endodontic treatment.[18]

Cemento-osseous dysplasia can radiographically simulate endodontic pathologies. However, periapical pathologies of endodontic origin tend to be centrally located on the tooth's apex, regardless of radiographic angulation, in addition to the presence of aetiologies not occurring in periapical pathologies of non-endodontic origin, as is the case of PCOD.[21] In a prevalence study conducted by Pereira, Ribeiro and Bittencourt[8], the dental records (i.e. complete periapical radiographic documentation) of patients attending the Dentistry Department of the Bahia School of Medicine and Public Health in an 8-year period were analyzed. Of the 893 dental records selected according to the inclusion criteria, nine cases of patients with PCOD were found, representing a prevalence of 1%. It is worth emphasizing that all cases were of female patients older than 30 years old, with six being black.

According to Kawai et al,[4] a differential diagnosis should be established between the initial phases of PCOD and of ossifying fibroma due to the similarity in their radiographic images, which includes poorly-calcified periapical osteopetrosis and lesions of endodontic origin (e.g. granulomas, abscesses and cysts).

According to Pereira, Ribeiro and Bittencourt,[8] in the first phase of the lesion, the differential diagnosis includes not only periapical pathologies, but also occlusal trauma, florid cemento-osseous dysplasia and focal cemento-osseous dysplasia due to loss of bone structure and replacement with fibrosis. In the third phase, also known as maturation phase, the differential diagnosis should include odontoma, osteoblastoma and focal sclerosing osteomyelitis.

In a Finnish study performed by Visnapuu et al,[17] 55 Caucasian patients carrying type-1 neurofibromatosis were examined for presence of PCOD. The lesion was detected in 34.8% of the adult women, but was absent in men and children. In fact, it was observed that PCOD is a new type of osseous lesion associated with neurofibromatosis. As some of the patients included in their study were submitted to endodontic treatment, one cannot exclude the possibility that some of the procedures might have been unnecessary as a result of inadequate examination.

Nigri et al[6] presented an unusual clinical case of PCOD. A 25-year-old Caucasian man presented the lesion, which was radiographically diagnosed as a radiolucid image involving the apices of the left lower incisors and canines, all positively responding to pulp

sensitivity tests. In the follow-up visit, nine years after the first visit, the cone beam computed tomograph showed increased lesion size and compromised cortical bone. The patient also reported discomfort in the region of the affected teeth.[6]

The radiographic follow-up of the lesions should be considered as the presence of neoplastic process (ossifying fibroma) or simple bone cyst should not be disregarded based only on radiographic evaluation. It is important to observe that PCOD has a limited potential for growth, differently from neoplastic lesions.[4]

Patients with periapical radiolucencies who were endodontically treated should be followed up. Biopsy is indicated for cases of periapical radiolucency persisting after endodontic therapy and cases of incomplete clinical history, since non-endodontic lesions with different clinical prognoses can be revealed, with keratocystic tumour being the most common lesion.[7]

Smith, Patel and Hoskinson[14] presented a clinical case in which the patient had been submitted to inadequate endodontic treatment of four lower incisors, but one of the teeth had been fractured during instrumentation. Nevertheless, it was a case of cemento-osseous dysplasia without the need of intervention. This case highlights the necessity of performing an adequate examination for the correct treatment and the importance of not starting the endodontic therapy without a clear diagnosis.[14]

In the study conducted by Ortega et al,[7] a total of 4,006 biopsies performed at the School of Dentistry of the University of Chile were assessed, all from patients clinically diagnosed with disease resulting from pulp necrosis. Of these cases, 26 (0.65%) had histopathological diagnosis of non-endodontic pathology in which keratocystic odontogenic tumour was the most frequent non-endodontic lesion (42.3%). There was also one case (3.8%) of PCOD in a 52-year-old woman. In these cases, the histopathological diagnosis contributed to changing the clinical diagnosis.

However, it is important to emphasise that biopsy is usually not needed in those cases of PCOD,[2] since its diagnosis is primarily based on clinical and radiographic characteristics of the lesions.[16]

Another important factor is that the surgery needed to remove pathological tissues is very aggressive due to the difficulty in finding a cleavage plane between the lesion and healthy surrounding tissue. It is also impossible to remove peri-radicular portions of dysplastic bone completely while preserving the vitality of the teeth involved.[10]

Intervention in a black woman with multiple lesions in sound teeth is not necessary, but biopsy in cases of isolated lesions involving less common clinical and radiologic situations can be indicated in order to disregard a more significant pathological process.[8,18]

In general, these lesions do not cause problems when they are in the phase of radio-transparency, but once a significant sclerosis is present, the cemento-osseous dysplastic lesions tend to become hypovascular and prone to necrosis with minimum provocation.[18]

Pippi, Della Rocca and Sfasciotti[10] reported the cases of seven women aged between 35 and 63 years old who had PCOD and were being followed up at the Oral Surgery Department of the University of Roma. In five of them, the treatment was just to follow up those lesions remaining unchanged in size and the teeth positive for pulp sensitivity. Two patients were surgically treated, one of them due to the fact that she was very distressed with the presence of the lesion. In such a case, six months after the surgery, there was loss of pulp sensitivity in two of the four teeth involved and consequently they were endodontically treated. In the other case, the surgical treatment was performed due to a notable decrease in the pulp sensitivity of the teeth involved. The endodontic treatment of these teeth was performed before surgery, which included apicoectomy to allow complete removal of pathological tissue from the inter-radicular region.[10]

According to Kawai et al,[4] the lesions can become secondarily infected probably because of their location next to the periapical areas of teeth frequently exposed to pulp and periodontal infections. Additionally, the calcified masses inside the lesions are prone to necrosis and osteomyelitis can be a secondary lesion.

The most indicated procedure in cases of asymptomatic patients consists of regular follow-up examinations,[4,6,7,16,17] with emphasis on prophylaxis and good oral hygiene.[18]

Detailed anamnesis, quality radiographs and precise dental pulp testing are indispensable for correct diagnosis and important for following up the evolution of the clinical case. If there is a diagnostic error leading to endodontic

treatment, then this would possibly lead to re-treatment, apical surgery or even extraction because of the lack of expected regression of the lesion as viewed radiographically. The importance of following up lesions of PCOD is reported in the case of a healthy 26-year-old Caucasian woman, who was followed up eight times over a period of 12 years. During this time, the lesion changed in appearance, indicating several phases of PCOD.[12]

Cases of symptomatic patient, an unusual condition in the cases of PCOD, are more complex. In this phase, there is an inflammatory component and the process is basically a chronic osteomyelitis involving dysplastic bone and cementum. In these cases, the use of antibiotics is usually ineffective as the sclerotic cemental masses are slowly sequestrated before cure.[18]

A case report described an idiosyncratic emergence of PCOD in a 19-year-old woman whose main complaint was a painful swelling in the palatal region corresponding to the right premolars for 6 months and with 6-month evolution. Intraoral examination revealed a diffuse oval oedema of approximately 2 cm. No increase in cortical bone was detected, and the mucosa on the oedema was normal with no secondary alteration. The oedema was non-floating and had firm consistency. Mobility in teeth #14 and #15 was observed, but pulp sensitivity test revealed their vitality. Taking into consideration all the examinations performed, a temporary diagnosis of benign tumour was made. Analyses of the radiographic images reinforced the suspicion of benign tumour, and thus an excisional biopsy of tooth #14 was indicated as therapeutic procedure and for histopathological confirmation of the lesion. The definitive diagnosis was cemento-osseous dysplasia. In general, although the cases of PCOD do not require any treatment, it is essential to follow up the lesion due to the possibility of secondary osteomyelitis.[24]

Discussion

PCOD is a reasonably well-defined clinical and radiographic entity. There is a consensus in the literature that this lesion involves predominantly apical areas of sound mandibular incisors, but cases involving the premolar region have also been described elsewhere. Although isolated lesions may occur, the multiple ones are more frequent.[1,18]

The literature is unanimous in stating that the lesion affects mainly women, especially the black ones.[6,10] The occurrence in women ranges from 10:1 to 14:1[18]. Despite its predominance among black women, the diagnosis should not be based only on these characteristics, since the lesion also affects white women.[11,16] In order to explain the predominance among post-middle aged women, there is the hypothesis that these lesions represent a dysplastic process related to a hormonal imbalance that influences bone remodelling.[4]

There is also a consensus in the literature that the majority of the patients are older than 30 years old when the lesions are observed for the first time. The condition is rarely seen in patients younger than 20 years old.[8,18]

Three developmental phases of the lesion are described, each one with specific radiographic characteristics. The first phase (osteolytic) involves the proliferation of cementoblasts accompanied by bone resorption and loss of periodontal ligament.[21] Nevertheless, in this osteolytic phase the bone resorption is not accompanied by loss of periodontal ligament, which remains intact.[18] It is unusual that the lesion increases sufficiently in size to produce a detectable expansion of the cortical plate.[3] It is convenient to emphasise that radiographic images of the same lesion can show different aspects, but in different phases, as described earlier.[4]

It is very important to perform pulp sensitivity tests to determine a correct diagnosis. Lesions of non-endodontic origin usually do not affect blood supply or pulp innervation of adjacent teeth, which remain with normal pulp vitality.[17] Therefore, the presence of pulp vitality excludes the possibility of a chronic periapical lesion.[9] These are important aspects which should be disclosed to dentists.

There is a general consensus that PCOD does not require any treatment. The equivocal indication for endodontic treatment,[22] which compromises and impairs the condition, is an important fact that should be known by surgeon-dentists. The progressive growth of the lesion rarely occurs.[18] Once the radiopaque phase is reached, the lesion becomes stable and causes no complications.[8,16]

Conclusion

It is important that the dental practitioner recognizes the relevant aspects of PCOD, such as aetiology and clinical and radiographic characteristics, in order to perform the differential diagnosis and treatment planning, thus avoiding iatrogenic procedures and unnecessary endodontic therapies.

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