CASE REPORT

Focal cemento-osseous dysplasia involving a mandibular lateral incisor

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Abstract


\textbf{Aim} To report a case of focal cemento-osseous dysplasia (FCOD) affecting a single tooth misdiagnosed as an inflammatory periapical lesion.

\textbf{Summary} The patient, a black 47-year-old woman complained of pain affecting the right side of the mandible. Routine X-ray examination discovered a periapical radiolucency on the mandibular left lateral incisor (tooth 32), which was otherwise normal and not carious. As the response of this tooth to a vitality test was doubtful, the lesion was diagnosed as a periapical granuloma or cyst secondary to pulp necrosis. Endodontic treatment and curettage of the periapical lesion were performed, and histological examination of the curettage material revealed a localized osseous dysplasia.

\textbf{Key learning points}
\begin{itemize}
  \item FCOD may rarely affect only one tooth, resembling a periapical granuloma or cyst.
  \item Careful diagnosis is of paramount importance in cases of questionable periapical lesions affecting normal-looking teeth, before beginning treatment.
  \item FCOD generally requires no treatment. Biopsy is warranted in case of doubt.
\end{itemize}

\textbf{Keywords}: cementoma, fibro-osseous lesions, incisor, periapical diseases.

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Introduction

Focal cemento-osseous dysplasia (FCOD) in the tooth-bearing areas of the jaws is an asymptomatic benign condition, belonging to the spectrum of fibro-osseous lesions. FCOD and periapical cemento-osseous dysplasia are different names for the same pathological process (Summerlin & Tomich 1994, Waldron 1995, Su \textit{et al.} 1997a,b).

The aetiology and pathogenesis of FCOD are unknown, and this lesion is considered to be a reactive or a dysplastic process in the periapical tissues. Usually, it affects two or more

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mandibular anterior teeth, and the radiographic appearance varies depending on the state of development. In rare cases, the lesion may affect only one tooth, and thus mimic an apical granuloma or a cyst (Forman 1975, Wilcox & Walton 1989, Smith et al. 1998, Drazic & Minic 1999).

A case of a solitary FCOD misdiagnosed as an inflammatory periapical lesion resulting in unnecessary treatment is reported.

Case report

The patient, a 47-year-old black woman, presented complaining of pain in the right mandible. Routine radiographic examination revealed a corono-radicular oblique fracture of tooth 45. It also showed a periapical radiolucent lesion approximately 9 mm in diameter with well-defined borders (Fig. 1), affecting the otherwise normal-looking left lateral mandibular incisor (tooth 32).

Upon clinical examination, tooth 32 showed a normal coronal morphology and colour, without signs of carious lesions. No mobility or tenderness to percussion were found. Cold sensitivity test was inconclusive. No other vitality tests were undertaken. The patient’s medical history was noncontributory.

A surgical excision of the periapical lesion was performed. On the basis of the intra-operative findings during the curettage, it was suspected that the lesion was not a periapical granuloma or cyst of endodontic origin. The curetted material was submitted for histopathological examination.

Figure 1 Periapical radiograph showing a well-circumscribed unilocular apical radiolucency associated with tooth 32.
Macrosopic examination of the surgical sample showed small fragments of gritty, haemorrhagic tissue. Histological examination revealed that they consisted of fibrous tissue, with varying cellular content, associated with irregular trabeculae of bone or deposits of cementum-like substance (Fig. 2). Peripheral osteoblasts were also present along some of the calcified masses. There were no inflammatory cells in the fibrous tissue. The bone fragments showed an irregular woven structure under polarized light. The final histological diagnosis was FCOD.

The patient was subsequently lost to follow-up.

Discussion

According to the WHO classification (Kramer et al. 1992), four major types of benign fibro-osseous lesions were to be distinguished: cemento-ossifying fibroma, a lesion considered to be an osteogenic neoplasm (resulting from assimilation of the formerly named cementifying fibroma and ossifying fibroma), and three variants of non-neoplastic cemento-osseous dysplasias. The latter are:

1. periapical cemental dysplasia (formerly periapical fibrous dysplasia), affecting the mandibular incisor periapical region;
2. florid cemento-osseous dysplasia (formerly gigantiform cementoma or familial multiple cementoma), affecting several parts of the jaws as large masses of mineralized almost acellular tissues, especially in black persons;
3. other cemento-osseous dysplasias (lesions sharing some of the features of periapical cemental dysplasias and/or florid cemento-osseous dysplasias, but not their clinico-pathological pattern of presentation).

These lesions were and still are quite distinct from the benign cementoblastoma. Summerlin & Tomich (1994) and more recently, Su et al. (1997a,b) modified Kramer’s classification and proposed the overarching term of FCOD for the lesions affecting the mandibular incisor area formerly designated as periapical cemental dysplasia, and for similar
lesions situated elsewhere in the jaws. In this paper, this classification was followed and the term FCOD used.

FCOD has been described as having three developmental stages, each with specific radiographic features (Thoma 1954, Tanaka et al. 1987). In the early or osteolytic stage, resorption of alveolar bone is accompanied by proliferation of well-vascularized fibrous connective tissue. At this stage, radiographs show a well-defined radiolucent area with loss of periodontal ligament and lamina dura. In the intermediate or cementoblastic stage, small opacities appear within the radiolucent area, which consequently displays a mixture of radiolucent and radio-opaque architecture. This is because of the deposition of cementum-like droplets in the fibrous tissue. At this stage, the lesion could be histopathologically misdiagnosed as cemento-ossifying fibroma. The last mature, osteosclerotic and ‘inactive’ stage is characterized by a definite radio-opacity, present in the major part of the lesion. The histopathological pattern has been described as ‘ginger root-like’ owing to a great number of poorly cellular, thick, curvilinear, anastomosing bony trabeculae (Su et al. 1997a). Only at this stage a well-defined border may be seen as a thin radiolucent line.

According to Neville & Albenesius (1986) and Su et al. (1997b), FCOD has an age-peak incidence between the fourth and the fifth decades. This conclusion is in agreement with Chaudry et al. (1958) and Zegarelli et al. (1964), who reported that the peak incidence is between 30 and 45 years of age. The latter authors grouped all the fibro-osseous lesions instead of distinguishing between cemento-ossifying fibroma and FCOD; therefore, their findings are probably less reliable.

FCOD seems to have a striking predilection for black women: 70% of reported cases according to Zegarelli et al. (1964). Neville & Albenesius (1986) reported that 6% of subjects in a black population exhibited signs of FCOD.

The aetiology of FCOD is unknown. The hypothesis of a periodontal ligament origin of this lesion seems to be the most widely accepted. Whether trauma, caries, periodontal disease, infection or systemic diseases could be triggering factors is still to be elucidated. In this respect, Zegarelli et al. (1964) suggested a hormonal imbalance as a likely causative or contributory factor. A few cases of autosomal pattern of inheritance of familial periapical cemental dysplasia have been reported (Young et al. 1989, Thakkar et al. 1993).

The diagnosis of typical FCOD is usually straightforward and is based on clinical and radiological features. The differential diagnosis should consider the stage of development of the lesion and include periapical granuloma or cyst and chronic osteomyelitis in the osteolytic stage, whereas in the mixed and radio-opaque stages, chronic sclerosing osteomyelitis, ossifying/cementifying fibroma, odontoma and osteoblastoma should be considered (Forman 1975, Neville & Albenesius 1986, Natkin 1994).

No treatment is necessary for FCOD and follow-up is required to confirm the diagnosis. Some authors have suggested that transformation into florid cemento-osseous dysplasia was possible and should be considered at recall visits (Waldron 1993, Summerlin & Tomich 1994, Manganaro & Millet 1986).

Conclusions

Solitary FCOD may present a difficult diagnosis for the dental practitioner. This case highlights the necessity to make a careful differential diagnosis in doubtful cases. A detailed and careful clinical examination, together with judicious use of vitality tests and radiographical examination, is required. In this patient, the radiographical finding and the inconclusive vitality test led to a misdiagnosis and unnecessary treatment.

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References