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RESEARCH ARTICLE

FLORID CEMENTO-OSSEOUS DYSPLASIA: A CASE REPORT WITH BRIEF SYNOPSIS

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ABSTRACT

Florid cemento-osseous dysplasia clearly looks to be a form of bone and/or cementoid tissues limited to jaw bones. It commonly affects middle-aged women in the fourth and fifth decade. It is usually asymptomatic and is incidentally diagnosed based upon clinical and radiographic examinations. Radio graphically the lesion shows bilaterally diffuse irregularly shaped radiopacities in the alveolar processes involving at least two quadrants. Management of an asymptomatic patient consists of regular follow up with reinforcement of good oral hygiene to prevent loss of teeth and management of the symptomatic patient is more difficult due to dysplastic bone with compromised blood supply, which is susceptible to infection. This article presents a rare case report of a 45-year-old south Indian female who was clinically and radiographically diagnosed as florid cemento-osseous dysplasia affecting the entire mandible.

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INTRODUCTION

Fibro-osseous lesions are group of jaw disorders in which a cellular bone or cementum like round structures are replaced with benign fibrous connective tissue matrix. The radiographical appearance plays an important role in the diagnosis of these lesions (Melrose et al., 1976). Cemento–osseous dysplasia's (COD), are well-known to arise from periodontal ligament tissues**.** Classification of cemento-osseous lesions of the jaws has long been argumentative dilemma for pathologists and clinicians. The World Health Organization (WHO) workshop group based upon the extent and radiographic appearances on head and neck tumors classified osseous dysplasia into four subtypes (Fig 1) (Pitak-Arnnop, 2009). Later in the year 1992, the World Health Organizationre-classified cementomatous lesions based on age, sex, location of the lesion, histopathologic, radiographic and clinical characteristics (Fig 2) (Goncalves, 2005).

Fcod Synopsis

The term florid cemento osseous dysplasia was first proposed by Melrose et al in 1976 to describe a wide-spread, extensive manifestations of the disease[1]. FCOD is a very uncommon benign, non-neoplastic and self-limiting jaw lesion with an unknown etiology that exhibits as an exuberant multi quadrant masses of bone and cementum.

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It has been deciphered as a dysplastic lesion or developmental anomaly arising in the tooth bearing areas. These lesions have a striking tendency towards being bilateral, often symmetrical and predominantly seen from the mandibular molar to premolar region (Goncalves, 2005) Waldron et al. have elucidated that reactive or dysplastic changes in the periodontal ligament might cause the disease. FCOD seems to have a female preponderance with middle aged black woman affected more frequently than the Caucasians and the Asian population (Mangala, 2006; Waldron, 1985).

Clinical Features

Lesions are often asymptomatic and found accidentally during radiographic examination, but some patients present clinically with pain, swelling, purulent discharge and sequestrum formation. Frequently, these lesions are strictly localised to the tooth-bearing areas and not associated with any other skeletal disease but sometimes gives rise to extreme deformities and functional disturbances caused by dysplastic hyper proliferation. Etiological factors such as denture wear, tooth extraction, compromises the condition making the vascular tissue of the lesion expose to the oral cavity (Pitak-Arnnop, 2009; Wood, ?).

Radiographic features

FCOD usually exhibits as a diffuse distribution of dense, lobulated multiple radiopaque masses interspersed with ill-defined radiolucent/radio-opaque sclerotic border confined to the alveolar process in either or both the jaws but with mandibular predilection (Pitak-Arnnop, 2009).

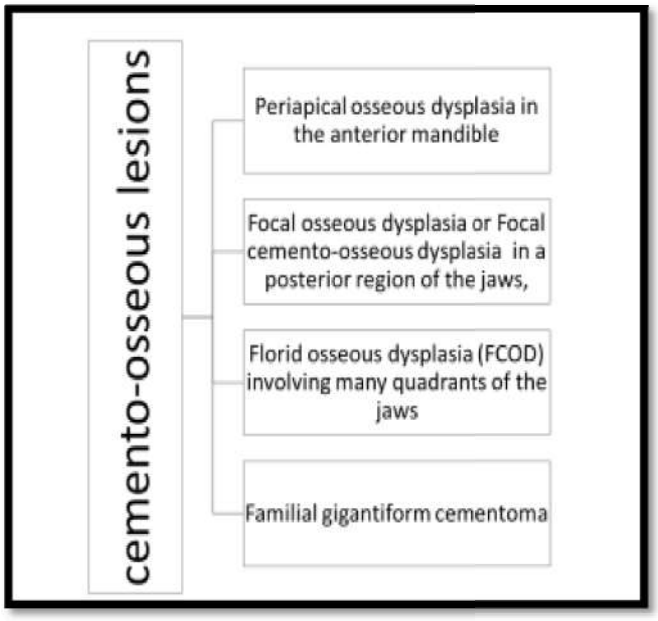

Fig. 1. Classification of cementoosseous lesions

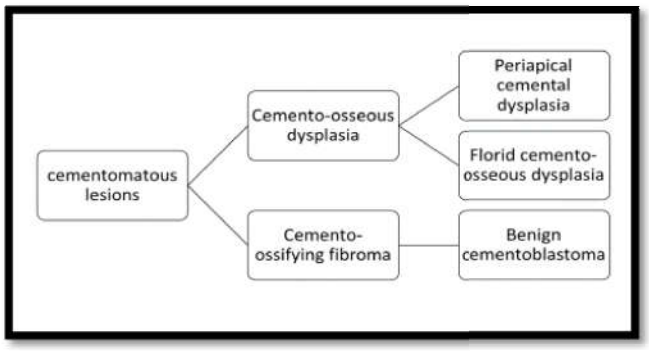

Fig. 2. Classification of cemento osseous lesions by WHO in (1992)

When the lesions are large, jaw expansion and facial deformity are likely to be present. The internal structure of the lesion can vary from an equal mixture of radiolucent and radiopaque lesions to almost completely radiopaque. Its mineralization increases with time and may vary in different stages depending on the degree of calcification. Immature FCOD manifests as a densely sclerotic lobular or irregular shaped radiopacities with a radiolucent halo which separates the lesion from the surrounding bone. Upon maturation, FCOD involves multiple sites throughout the jaw. Large FCOD lesions can displace the anatomical structures causing enlargement of alveolar bone by displacement of buccal and lingual cortical plates. The roots of associated teeth may have a considerable amount of hypercementosis, which may fuse with the abnormal surrounding cemental tissue of the lesion (Pitak-Arnnop, 2009; White, ?).

Histopathological features

These lesions comprised of fragments of cellular mesenchymal tissue of spindle shaped fibroblasts and collagen fibers with numerous small vessels. The magnitude of each mineralized material varies from lesion to lesion and from area to area in the individual site of involvement. As the lesion matures and becomes more sclerotic, the ratio of fibrous connective tissue to mineralized material decreases and bony trabeculae become thick, curved and matches the shape of ginger roots.

The individual trabeculae fuse and form lobular masses composed of sheets or fused globules of relatively acellular and disorganized cemento-osseous material (Pitak-Arnnop, 2009; Neville, ?).

Differential diagnosis

The term cementoosseous dysplasia is a histopathological term, yet the diagnosis can be solely made by clinical and radiographic findings. FCOD is a benign fibro-osseous lesion that must be differentiated from other benign fibro-osseous lesions (ref Table 2) such as chronic diffuse sclerosing osteomyelitis (CDSO), periapical cemental dysplasia, ossifying fibroma (OF), fibrous dysplasia (FD) and Paget's disease of bone (PD)on the basis of combined clinical, radiographic, and histological features (Pitak-Arnnop, 2009; Wood, ?; White, ?).

Management

FCOD requires no treatment unless it is mortifying for the patient concerned. Nevertheless, asymptomatic patients are managed by regular recall examinations with prophylaxis and reinforcement of good home hygiene care to control periodontal disease. Management of the symptomatic patients are more challenging because chronic inflammation and infection develop within densely mineralized tissues and antibiotics are in appropriate due to their poor tissue diffusion. Indeed biopsy increases the risk of infection or may cause jaw fractures and it is not recommended to surgically remove these lesions as this often requires extensive surgery (Goncalves, 2005; Beylouni, 1998; Summerlin, 1994; Damm, 2001)

Case Report

A 45-year-old female patientreported to the department of periodontics, S.R.M. Dental College, with a chief complaint of shaking of her teeth in both the arches for past 1 month. Patient's medical history revealed no history of major illness or any systemic diseases. Extra oral examination reveals no signs of lymphnode enlargement or tenderness. On gingival examination it depicts reddish pink shiny smooth surface with melanin pigmentation and blunting of interdental papilla with soft and edematous in consistency and gingival swelling with presence of purulent exudates (Fig 3). Periodontal examination reveals partially edentulous arches with generalized probing pocket depth ranging from 5- 8 mm with generalised tooth mobility, and gingival recession along with furcation involvement in posterior teeth (Table 1).

Fig 3. Clinical picture showing severe gingival inflammation and exudate with severe periodontal destruction

Fig. 4. Ortho pantomograph reveals dense, multilocular radiopacities surrounded radiolucency in the right and left posterior areas of mandible

Table 1. Showing the hard tissue examination features

MOBILITY	RECESSION	FURCATION
Grade I mobility – 11,12,13,15,16,17,34,35,24,25 Grade III mobility – 27,28,47,48,41,42,43,31,32,37,38	Class II – 31,32,41,42,43,34,35,24,25,15,47,48,37,38,17, 18,27	Grade III – 47,48,37,38,17,27

Table 2. Shows the difference between other fibro osseous lesions and FCOD based on demographic data, prevalence, clinical and radiographic features [15-17]

	FCOD	PAGETS DISEASE	COF	CSO
Age of incidence	4th and 5th decades	5th and 6th decades	3rd and 4th decades	5th and 6th decades
Gender	Female predominance	male predominance	Female predominance	Female predominance
Race	64% Blacks	3% whites	53% Whites	70.0% blacks
Symptoms	38%	90%	51%	57.6% - abscess formation 79.8 % - swelling 20.7 % - Limited mouth opening
Radiographic borders	Well defined (53%) Ill-defined (47%)	well-circumscribed radiolucency, loss of lamina dura, pulpal radio-opacity	Mostly well defined (85%) Ill-defined (15%)	Sequester formation - 14.8 %, ill-defined osteolytic areas
Radiographic density	Lucency (31%) Irregular opacity (24%)	17% "cotton wool"-like radiopacities	Lucency (53%) Opacity (7%)	bone condensation (91.1%), periosteal thickening and bony irregularities (8.9%)
Periapex involvement	70%	----	7%	-----
Edentulous area	21%	----	8%	-------
Jaw segments	Predominantly in mandible Increased incidence in anterior and premolar regions of mandible	Maxillary involvement is about six times more common than mandible	Predominantly in mandible	85%body of the mandible from the alveolus to the inferior border and may extend into the ramus commonly in the mandible

Orthopantomograph revealed generalized horizontal bone loss evident till the apical thirds of tooth roots in relation to 48 to 41, 31 to 38 and 15 to 11, 21 to 25 with severely resorbed maxillary right and left posterior segments. The apical region of 47 reveals small, oval ill-defined multiple radiopacities surrounded within a well-defined radiolucent halo measuring a size of 5 x 3 mm, which separates the lesion from the surrounding bone and displaces the inferior alveolar canal. Lesion in relation to 48 reveals a well-defined radiolucency in the apical aspect and merging with adjacent lesion in 47 measuring of 2 x 3 mm size. The 35 region shows multiplelarge, irregular, dense radiopacities seen in the apical aspect extending towards the edentulous area of 36, displacing the inferior alveolar canal, measuring size of 8 x 4 mm. 38 area reveals a well-defined radiopacity surrounded within a well-defined radiolucent halo in the apical aspect measuring size of 3 x 5 mm. lesion in relation to the edentulous aspect of 46 reveals small, oval ill-defined radiopacity surrounded within a well-defined radiolucent halo measuring a size of 3 x 4 mm (Fig 4) showing various stages of disease severity. Thereby correlating the radiographic and clinical findings the present case is provisionally diagnosed as Florid COD. Because of its poor vasculature and density of bone the periapical infection may complicate into osteomyelitis which impedes further periodontal management. Therefore, the patient was motivated about the oral hygiene and referred to Oral & maxillofacial surgery for extraction of all teeth and advised for full mouth rehabilitation. The differential diagnosis is further reviewed in the discussion.

DISCUSSION

Osseous dysplasi as in the jaws are the most prevalent of the fibro-osseous lesions. These lesions are customarily referred to as cementoosseous dysplasias. Three different types of distribution patterns have been described; periapical cemento osseous dysplasia (PCOD), focal cemento osseous dysplasia and florid cemento osseous dysplasia (FCOD) (Waldron, 1985). The PCOD described when the lesions occur adjacent to the periapices of teeth, or in the vicinity of teeth in edentate cases. It affects multiple teeth of the maxilla and mandible, with median age of 39 years and female predominance more than 90%.Most commonly seen in blacks, Asians are also affected but to a much lesser extent. As the lesions become more extensive, expansion of the buccal and lingual cortices may occur. Radiographically circumscribed radiolucencies at apices of the tooth are seen (Waldron, 1970). Whereas, FCOD is more extensive and wider in distribution. If the periapical pattern present in 3 or 4 quadrants the term florid is often applied. FCOD follows a similar pattern of racial distribution as PCOD. A hereditary pattern has also been described and this may, in fact, be a different type of FCOD with a unique autosomal dominant inheritance pattern. The lesions are asymptomatic and teeth remain vital despite the changes in the supporting bone. Early patterns of FCOD have radiolucent patterns that are not well demarcated in the alveolar bone of tooth bearing areas. With time, the outlines increase and the mineralizations gradually opacify, with varying patterns of mixed radiolucency-radiopacity. As these lesions enlarge, they extend to the basal bone of the maxilla and mandible. The shapes of the mineralizing patterns vary from diffuse and lobular outlines that either gradually enlarge or coalesce to form larger mineralized patterns. The focal pattern is used to describe situations when the lesion is only identified in one area of a single tooth or even multiple adjacent teeth. The features are similar to POD.

These lesions have more localized pattern. Large early patterns of mixed radiolucent-radiopaque density that extend to the basal bone. The denser mineralized patterns are to be expected in older female patients (Waldron, 1970; Waldron, 1973; Doi, 2013). FCOD is a benign fibro-osseous lesion that must be differentiated from other benign fibro-osseous lesions such as periapical cemental dysplasia, ossifying fibroma (OF), chronic sclerosing osteomyelitis (CSO) and Paget's disease of bone (PD).

Conclusion

In conclusion, to the author's knowledge, FCOD is unusual in south Indian population. The present case reportdiagnosed with FCOD on the basis of clinical and radiographic findings in a tamilnadu female patient. Many lesions that occur in the jaw have a similar radiographical appearance, and it is often tough to distinguish among them. Furthermore, to get the accurate diagnosis, we must consider all the available diagnostic information, such as radiographic and clinical findings which would contribute to the diagnosis. In the present case report, CT and histological analysis would have added more value to the diagnosis, though clinical and radiographic investigations would be sufficient enough in arriving at FCOD diagnosis (Waldron, 1970; Waldron, 1973; Doi, 2013)

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