J. Oral Diagn 2024;09:e262

REVIEW ARTICLE

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Familial florid cemento-osseous dysplasia: an overview of a rare entity

Abstract:

Familial florid cemento-osseous dysplasia (FFCOD) is distinct from the sporadic variant and may often be confused with familial conditions presenting with lesions resembling cemento-ossifying fibromas. The current review aims to elucidate the FFCOD variant better and discuss distinguishing features with sporadic florid COD. A review of the literature on FFCOD cases using Google Scholar and PubMed was performed and summarised. A total of 11 articles with 36 patients were included in the current review. The clinical and radiologic presentations and the pertinent differences from the sporadic variant were discussed. The familial form shows advanced sclerosis and extensive distribution at a younger age, together with impacted teeth and bony expansion in the anterior mandible. Furthermore, distinguishing features from the most important differential diagnoses of other hereditary fibro-osseous conditions, including familial gigantiform cementoma (FGC), hyperparathyroidism jaw tumour syndrome (HP-JTS) and gnathodiaphyseal dysplasia (GDD) are discussed.

Keywords: benign fibro-osseous lesions, cemento-osseous dysplasia, familial florid cemento-osseous dysplasia.

INTRODUCTION

The latest 5th edition of the WHO Classification of Head and Neck Tumors includes (cemento)-ossifying fibroma, fibrous dysplasia, cemento-osseous dysplasia, familial gigantiform cementoma, and segmental odontomaxillary dysplasia under the category of benign fibro-osseous lesions (BFOLs) of the jawbones[1]. This category encompasses a group of developmental (fibrous dysplasia), neoplastic (ossifying fibromas) and dysplastic (cemento-osseous dysplasia and segmental odontomaxillary dysplasias) lesions[2]. These BFOLs have overlapping histopathologic features; therefore, clinical-radiologic correlation is paramount for definitive diagnosis[1,3,4].

Statement of Clinical Significance
The latest WHO classification included an additional variant of cemento-ossoeus dysplasia (COD), termed familial florid COD. This variant has a distinct presentation compared to sporadic florid COD and may often be confused with other familial conditions presenting with cemento-ossifying fibromas. The current review aims to elucidate this relatively new entity and discuss distinctive features.

Cemento-osseous dysplasia (COD) is the most common BFOL of the jawbones, showing a strong predilection for middle-aged females of African descent[1,5]. It is caused by the dysplastic replacement of normal bone by fibrous connective tissue intermixed with mineralised bony spicules. The latest edition of the WHO classification divided COD into variants based on their distribution, which includes focal, periapical and florid subtypes[1]. Focal COD remains confined to one location, often involving a posterior mandibular tooth, whereas florid COD refers to lesions that have multi-quadrant involvement. In contrast, periapical COD involves multiple teeth in the anterior mandibular region.

All variants of COD present with similar features and are often discovered incidentally on radiographic examination[3]. The lesions remain confined to the alveolar or tooth-bearing bone with a progression from radiolucent to mixed to ultimate radiopaque lesions with a radiolucent rim[1]. Due to the wide distribution of florid COD, large regions of the bone become sclerotic, with subsequent hypovascularity.

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Received: September 9, 2024. Accepted: October 8, 2024.
https://doi.org/10.5327/2525-5711.262

This disrupts normal haemodynamics of bone, resulting in secondary simple bone cyst (SBC) formation. Additionally, due to the sclerotic nature of the bone, secondary osteomyelitis can occur due to exposure of the sclerotic masses to the oral environment either via surgical manipulation (e.g. extraction) or physiological resorption of edentulous ridges. Florid COD may present with areas of limited bony expansion[1,4].

The latest WHO classification was the first to include an additional fourth COD variant, appropriately termed familial florid COD (FFCOD)[1]. This variant has a distinct presentation compared to sporadic florid COD and may often be confused with familial conditions presenting with lesions resembling cemento-ossifying fibromas. These include familial gigantiform cementoma (FGC), hyperparathyroidism jaw tumour syndrome (HPJTS), and gnathodiaphyseal dysplasia (GDD)[2].

The current review aims to better elucidate the relatively new familial florid variant of COD, and discuss distinguishing features from sporadic florid COD. A review of the literature on FFCOD cases was performed and summarised. Furthermore, the most important differential diagnoses of other hereditary fibro-osseous conditions are discussed in detail.

LITERATURE REVIEW

A review of the published literature using Google Scholar and Pubmed was performed. The search terms included "familial florid cemento-osseous dysplasia", "familial florid osseous dysplasia", "inherited osseous dysplasia", and "hereditary osseous dysplasia". For inclusion in the current review, cases had to present with a familial history of similar lesions and radiologic evidence of lesions resembling florid COD.

The search revealed a total number of 13 articles for further review. The references used in these articles were also accessed and evaluated. Two articles reported under 'familial florid cemento-osseous dysplasia' were excluded from the current review. The reported cases from Toffanin et al.[6] had a similar radiologic appearance to FFCOD; however, multiple family members reported multiple non-traumatic bone fractures, and one patient had raised alkaline phosphatase levels. Considering this history, the symptoms could not be distinguished from gnathodiaphyseal dysplasia. The cases reported by Kucukkurt et al.[7] were also excluded based on radiologic evaluation, with these patients presenting with multiple supernumerary teeth/odontomas and lesions outside of the alveolar bone resembling multiple osteomas. The combined features likely represent cases of Gardner syndrome. Therefore, a total of 11 articles consisting of 36 patients were included as part of the current study (Table 1)[8–18].

DISCUSSION

Clinical presentation

Sporadic florid COD shows a strong female predilection, with a female-to-male ratio of up to 100:1 and presents at a slightly later mean age than the other COD variants (49.3 years)[5,19]. There is a strong predilection to occur in black patients[5,19]. Although lesions most commonly occur in the mandible, concurrent involvement of the maxilla may be seen in 27% of cases[5]. The lesions are often discovered incidentally on radiographic examination[3]. In some cases, associated clinical signs and symptoms, related to secondary osteomyelitis, lead to the discovery of the lesions. Secondary infection/osteomyelitis is commonly seen in patients with florid COD, being described in 59.6% of cases. Associated swelling is less prevalent, but well-described in approximately 31.6% of cases[5,19].

Familial florid COD has a hereditary component; therefore, several family members may be concurrently affected by the disease[17]. It is, therefore, of utmost importance that should the familial variant be suspected, other family members should be encouraged to undergo investigative radiologic examinations. The predilection for females is not as strong, and male patients are affected in greater frequencies than the sporadic variant (2:1 female-to-male ratio)[17]. Due to the autosomal dominant inheritance pattern, FFCOD may also occur in white population groups[17]. It also presents at a significantly younger mean age (34.5 years) compared to the sporadic variant[17]. In FFCOD, the lesions progress rapidly to the sclerotic phase of the disease, resulting in multiple impactions of permanent teeth with over-retained primary teeth seen in roughly a third of cases. Furthermore, patients often present with extraoral swelling in the anterior mandibular area, resulting in chin prominence. The lesions have a similar distribution to the sporadic variant, with the maxilla having a more advanced presentation. In FFCOD, the lesions are generally more advanced in their distribution and sclerosis than in the sporadic variant. Due to the earlier onset of sclerosis, associated osteomyelitis is more common and may even occur in the maxilla. In contrast, the maxilla is relatively spared from osteomyelitis in the sporadic variant.

Table 1. Characteristics of familial florid cemento-osseous dysplasia cases reported in the literature.

Author	Age	Sex	Population	Pain/ symptomatic	Impacted teeth	Over-retained primary teeth	Maxilla affected	Expansive lesions	Anterior mandibular expansion
Sedano et al.[8]	40	F	White	N	N	N	Y	Y	N
	NR	F	White	N	N	N	Y	Y	N
	NR	F	White	N	NR	NR	NR	NR	NR
	NR	M	White	N	NR	NR	NR	NR	NR
	NR	M	White	N	N	N	NR	N	N
	NR	F	White	N	NR	NR	NR	NR	NR
	NR	F	White	N	NR	NR	NR	NR	NR
	NR	M	White	N	NR	NR	NR	NR	NR
	NR	M	White	N	NR	NR	NR	NR	NR
Musella et al.[9]	33	F	White	N	N	N	Y	N	N
	71	F	White	Y	N	N	Y	Y	N
Thakkar et al.[10]	61	F	Black	N	N	N	Y	N	N
	34	M	Black	N	N	N	N	N	N
	30	F	Black	N	N	N	N	N	N
	26	F	Black	N	N	N	N	N	N
Coleman et al.[11]	32	F	Black	Y	Y	Y	Y	Y	Y
	13	M	Black	N	N	N	NR	N	Y
	9	F	Black	N	Y	Y	Y	Y	Y
Hatori et al.[12]	29	F	Asian	Y	N	N	Y	Y	N
	62	M	Asian	Y	N	N	Y	Y	N
Srivastava et al.[13]	NR	F	Asian	Y	N	N	Y	Y	N
	18	M	Asian	N	Y	Y	Y	Y	N
Sim et al.[14]	49	F	Asian	Y	N	N	Y	Y	N
	21	F	Asian	N	N	N	Y	Y	N
	21	F	Asian	N	N	N	Y	Y	N
Thorawat et al.[15]	24	F	Black	Y	Y	Y	Y	Y	N
	45	F	Black	N	Y	Y	Y	Y	N
Lv et al.[16]	49	M	Asian	Y	Y	N	Y	Y	N
	NR	M	Asian	N	N	N	Y	Y	N
	NR	M	Asian	N	NR	NR	NR	NR	NR
	NR	F	Asian	Y	NR	NR	NR	NR	NR
Nel et al.[17]	58	F	Black	Y	Y	N	Y	Y	Y
	18	F	Black	N	Y	Y	Y	Y	Y
	21	M	Black	Y	Y	Y	Y	Y	Y
Smit et al.[18]	34	F	Black	Y	N	N	Y	Y	Y
	31	F	Black	Y	Y	Y	Y	Y	Y
Total	Mean age = 34.54	24F:12M (2:1)	11Asian 14 Black 11 White	13/36 (36%)	10/28 (36%)	8/28 (29%)	23/26 (88%)	21/28 (75%)	8/28 (29%)

M: male; F: female; Y: yes; N: No; NR: not reported.

Radiologic presentation

Sporadic florid COD presents with lesions limited to the alveolar bone, located above the inferior alveolar nerve canal in the mandible. The lesions present initially as radiolucent with progression to mixed density and ultimately sclerotic masses surrounded by a thin radiolucent rim[19]. Lesions in close proximity may coalesce to form larger sclerotic masses (Figure 1). There may be an associated but separate well-defined radiolucency, in cases with SBC formation (Figure 2). Radiologic widening

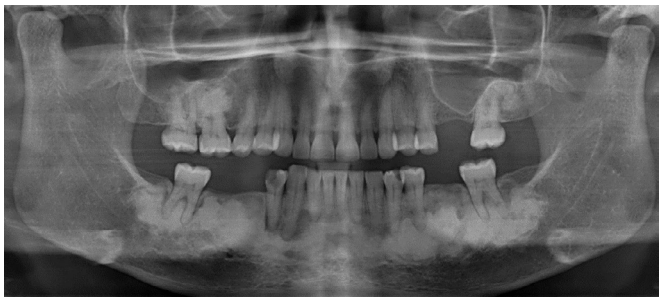

Figure 1. Florid cemento-osseous dysplasia showing coalescing sclerotic lesions with a radiolucent rim affecting all four quadrants.

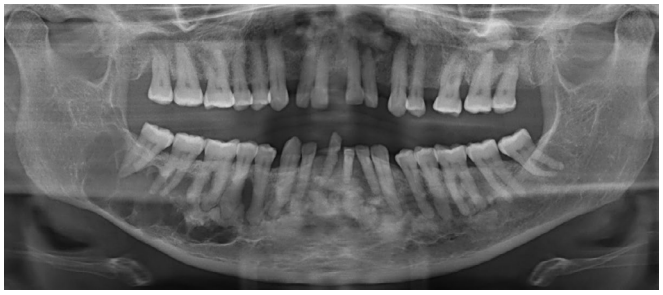

Figure 2. Florid cemento-osseous dysplasia with simple bone cyst formation in the right mandible.

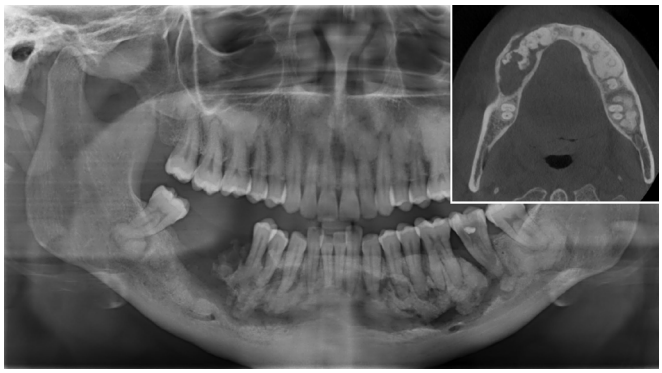

Figure 3. Florid cemento-osseous dysplasia with secondary osteomyelitis affecting most of the mandible following extraction of the right first mandibular molar 2 years ago. The expansion or widening of the radiolucent rim is seen as a sign of osteomyelitis. On CBCT imaging (insert), there is a slight bony expansion involving the left mandible, with a more pronounced expansion seen on the right side due to secondary infection.

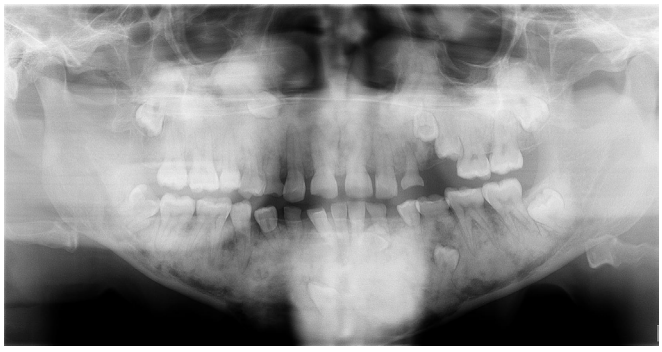

Figure 4. Presentation of familial florid cemento-osseous dysplasia with more advanced sclerosis seen in the mandible and maxilla confined to alveolar bone. The expansive lesions can be appreciated by displacement of the maxillary sinus and inferior alveolar nerve as well as prominence of the mental region. There are multiple teeth impactions and over-retained primary teeth.

or accentuation of the surrounding radiolucent rim may occur with secondary osteomyelitis (Figure 3). There may be slight expansion of the affected region which may become more pronounced when SBC or osteomyelitis accompanies these lesions.

Familial florid COD has differing radiologic presentations from the sporadic variant. Firstly, the expansion in the anterior mandibular region is more pronounced (Figure 4). The lesions are more extensive, with advanced involvement of the maxilla. The lesions often undergo sclerosis at a much earlier age. These unique features explain why FFCOD is often seen with associated impacted teeth and over-retained primary teeth, findings not seen in the sporadic variant (except for impacted third molars)[7]. Due to the earlier sclerosis, higher rates of osteomyelitis occur[17]. FFCOD cases often have radiologic signs of expansion, resulting in displacement of the inferior alveolar nerve canal or maxillary sinuses.

Histopathologic findings

BFOLs of the jaws share similar, overlapping histopathologic features. Regardless of the variant, COD is generally submitted as gritty tan-brown fragments. This contrasts with COF, in which the peripheral circumscription allows surgical enucleation or 'shelling-out' of the neoplasm. The well-circumscribed mass appears yellow-to-tan mass with a gritty texture on sectioning[1,3].

The histopathologic features of COD, regardless of the variant, are similar. COD consists of a highly vascular collagenised stroma with a plump spindle cell component that varies in cellularity. This vascular stroma, paired with the fragmented nature of the submitted specimens, aids in distinguishing COD from COF. As the lesion evolves, variably sized woven and cementum-like bone fragments appear. Osteoblastic rimming is usually limited. In the mature, late stage of the disease, COD comprises of dense sclerotic bone with minimal intervening marrow spaces[1,3,20]. The familial variant of florid COD presents with similar histological findings to COD and distinction is made on clinical and radiologic grounds.

Differential diagnosis

The expansive areas in cases of FFCOD may be mistaken for cemento-ossifying fibromas (COFs), should the background disease not be appreciated[3]. This makes distinguishing between FFCOD and genetic conditions with multiple associated COF-like lesions significant. These include HPT-JTS, FGC and GDD[21]. These conditions are primarily inherited in an autosomal dominant fashion, and are distinguished via their unique clinical features.

HPT-JTS is a syndrome resulting from a mutation of the *CDC73 (HRPT2)* tumour suppressor gene located on chromosome 1[22]. In this condition, patients have associated hyperparathyroidism with multiple parathyroid adenomas. The intrabony jaw lesions are indistinguishable from COFs; however, their multifocal distribution differs from sporadic COFs[21].

As the word 'gigantiform' implies, the intrabony lesions in FGC reach considerable sizes at a young age. Contrary to the name, this condition may be inherited or occur sporadically[23]. They often have multi-quadrant involvement of large, expansive lesions resembling COFs. The lesions usually present between 11-13 years of age, with a period of rapid growth noted between 14-16 years[24]. Only the jaw bones are affected in these patients, with no other extragnathic manifestations reported[25]. Unfortunately, a degree of ambiguity exists, as some authors report a history of long bone fractures without bowing[23]. Mutations in the *ANO5* gene, also termed *GDD1*, have been reported in cases of FGC, but not florid COD patients[23]. However, with the combination of various bone fractures, the question arises whether the patients in this study were better suited under a diagnosis of GDD. An allelic *ANO5* gene mutation (p.C356W) has also been identified in FFCOD patients, with no long bone fracture or other skeletal abnormalities noted in these patients[16]. The term expansive COD has been recommended to replace FGC as an entity, as not all cases have a hereditary component. However, many of these cases were reported in association with florid COD, and therefore, the possibility of FFCOD should also be considered[26].

GDD is a condition whereby extragnathic symptoms of brittle bones with associated long bone fractures have been reported in combination with COF-like lesions of the jaws. Mutations in the *ANO5* gene, or *GDD1* gene, have also been implemented in the pathogenesis of this condition[23,27]. Due to the overlapping clinical features and genetic associations between GDD and FGC, many reports have been erroneously attributed to one entity that may be better suited under the other. The distinction between entities likely revolves around extragnathic presentations. However, some authors suggest that there may be various presentations of the same entity[23].

Due to the considerable overlap and resultant confusion between FFCOD, FGC and GDD in the literature, El-Mofty[21] suggested overriding principles to aid in disease distinction until further comparative studies have been conducted. This includes the fact that FFCOD presents with an inherited and more advanced form of sporadic florid COD. FGC is a condition limited to the jawbones with extensive expanding COF-like lesions. In contrast, GDD has similar jaw lesions with extragnathic presentations of multiple long bone fractures.

Treatment

Treatment of FFCOD does not differ from the sporadic variants and is based primarily on the prevention of osteomyelitis through preservation of teeth and patient education. Due to the advanced sclerosis seen at a younger age in FFCOD, osteomyelitis results in significant morbidity[17]. In cases where surgical intervention is inevitable, such as teeth extraction, hyperbaric oxygen treatment[6,9] and prophylactic antibiotic treatment[9,12] have been used with varying degrees of success. Removing the sclerotic bony masses to reach normal bone has also been reported[17]. However, the wide distribution of lesions in the condition may result in extensive loss of bone volume.

CONCLUSION

There are differences in the presentation of FFCOD that help distinguish it from the sporadic variant of florid COD. Detailed radiologic examinations will highlight more advanced sclerosis and extensive distribution at a younger age. Multiple impactions with over-retained primary teeth are seen in the familial variant, which is not noted in sporadic cases. Although minimal bony expansion may accompany the sporadic variant of florid COD, FFCOD lesions show more advanced bony expansion, typically resulting in a prominent mental region.

AUTHORS' CONTRIBUTIONS

CS: Conceptualization, Investigation, Methodology, Writing – original draft. LR: Investigation, Methodology, Writing – review & editing. NRG: Investigation, Methodology, Writing – review & editing. FPF: Investigation, Project administration, Supervision, Writing – review & editing.

CONFLICT OF INTEREST STATEMENT

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Competing interests: The authors have no relevant financial or non-financial interests to disclose.

Ethics approval: All procedures followed the ethical standards of the Helsinki Declaration of 1975, as revised in 2008.

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