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Fibro-Osseous Lesions- A Review

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ABSTRACT

Fibro-osseous lesions (FOLs) are a group of benign conditions involving the replacement of normal bone with fibrous tissue and mineralized components like bone or cementum-like material. These lesions primarily affect the jaws and craniofacial bones, with common types including fibrous dysplasia (FD), cemento-osseous dysplasia (COD), and fibro-osseous neoplasms. FOLs can be classified based on clinical behaviour, histology, and radiographic features. Fibrous dysplasia, which presents as monostotic or polyostotic, is characterized by a "ground-glass" appearance on radiographs. Cemento-osseous dysplasia is often asymptomatic and involves the gradual replacement of bone with fibrous and cementum-like material. Fibro-osseous neoplasms such as cementifying and ossifying fibromas are benign tumours that may cause bone expansion and deformities. Diagnosis of FOLs requires correlation of clinical, radiographic, and histopathological findings due to overlapping features. Treatment depends on symptoms and severity, ranging from observation to surgical intervention. Accurate diagnosis ensures appropriate management and optimal patient outcomes.

KEYWORDS

Fibro-osseous lesions, Fibrous dysplasia, Cemento-osseous dysplasia.

1. INTRODUCTION

Fibro osseous lesions (FOLs) are a poorly defined group of lesions that affect the jaws and craniofacial bones. They are spindle cell proliferations that contain woven bone.

These pathologic diseases include developmental, reactive, and dysplastic lesions, as well as neoplasms. All include the replacement of bone with a benign connective tissue matrix. This matrix has mineralization foci that vary in size and shape, resembling braided bone or circular acellular structures with high basophilic properties. Diagnosing FOLs requires both clinical and radiographic correlation, as incisional/excisional biopsy material alone is insufficient. The development pattern on conventional radiographs or CT scans of the head and face is the only distinguishing factor between these entities, which have overlapping histological properties. [1]

The 2017 WHO categorization of odontogenic and maxillofacial bone cancers includes cysts and FOLs as new lesion groups, in addition to the existing tumours. To avoid confusion, lesions can be defined based on their radiographic presentation, as histologic features overlap significantly. Thus FOLs fall into three categories: fibrous dysplasia (FD), cemento-ossifying fibroma (COF), and cemento-osseous dysplasia (COD). The classification now includes familial Gigantiform Cementoma (FGC), a rare autosomal illness with uncertain etiology and complex radiological features, as well as osteochondromatous lesions. [2]

The majority of these lesions have an unknown aetiology, but others are thought to be malignant and others to be connected to metabolic abnormalities, all of which provide significant diagnostic issues. While some FOL are thought to be real neoplasms with a high propensity for development if not totally removed, the unique anatomic position of these craniofacial lesions can lead to deadly complications such as encephalitis and meningitis. Histologically, these types of FOL are distinguished by the replacement of normal bone by a fibrous connective tissue matrix. The fibrous tissue has varied characteristics, such as varying levels of mineralization in the form of woven bone or cementum-like basophilic structures that are indistinguishable from cementicles. [4]

2. HISTORY

The evolution of fibro-osseous lesions (FOLs) demonstrates a growing awareness of these complicated bone disorders, beginning with early reports and progressing through contributions from significant scholars and recent diagnostic breakthroughs. Rudolf Virchow first identified FOLs in 1862 as "leontiasis ossea," a disorder characterized by skeletal abnormalities of the face that give it a lion-like look. This first description was ultimately characterized as fibrous dysplasia, and Boyko gave more insights in 1936, detailing craniofacial fibrous dysplasia and drawing attention to its clinical appearance.

Dr. Louis Lichtenstein was essential in establishing fibrous dysplasia as a distinct pathological entity in the 1930s, providing detailed descriptions of its clinical and histological aspects that served as the foundation for its inclusion in a larger group of fibro-osseous disorders. Later, Dr. James Waldron considerably enhanced our understanding of FOLs, particularly in the craniofacial region, helping to refine their classification and distinguish them from other bone illnesses. [3]

3. CLASSIFICATION

The authors offer numerous classification schemes for fibro-osseous lesions. They have been listed below. [4]

- Charles Waldron Classification of The Fibro-Osseous Lesions Of The Jaws (1985)
- Working Classification of Fibro-Osseous Lesions By Mico M. Malek (1987)
- Peiter J. Slootweg & Hellmuth Muller (1990)
- WHO Classification (1992)
- Waldron Modified Classification Of Fibro-Osseous Lesions Of Jaws (1993)
- Brannon & Fowler Classification (2001)
- WHO Classification Of Fibro-Osseous Lesions Of Jaws (2005)
- Paul M. Speight & Roman Carlos Classification (2006)
- Eversole Classification (2008)

In 1985, Charles A Waldron divided fibro osseous lesions into three categories based on clinical behaviour, histology, and radiographic findings. [4]

1. Fibrous Dysplasia
    a. Monostotic
    b. Polyostotic
2. Fibro-Osseous (Cemental) Lesions Presumably Arising In The Periodontal Ligament
    a. Periapical Cemental Dysplasia
    b. Localized Fibro-Osseous-Cemental Lesions (Probably Reactive In Nature)
    c. Florid Cement-Osseous Dysplasia (Gigantiform Cementoma)
    d. Ossifying & Cemenifying Fibroma
3. Fibro-Osseous Neoplasms of Uncertain Or Detectable Relationship To Those Arising In The Periodontal Ligament (Category II)
    a. Cemetoblastoma, Osteoblastoma & Osteoid Osteoma
    b. Juvenile Active Ossifying Fibroma & Other So Called Aggressive, Active Ossifying /Cementifying Fibromas.

In 1987, Mico M. Malek proposed a working classification of fibro-osseous lesions from the perspective of a diagnostic pathologist. Later, in 1990, Peiter J. Slootweg and Hellmuth Müller introduced a classification that primarily focused on histopathological features, emphasizing the need to include adjacent normal bone for accurate diagnosis. However, the discovery of cementum-like tissues in lesions at extra-gnathic sites suggested that these tissues might simply represent a normal variant of bone and that dental cementum is a specialized form of "bundle bone." [4]

Consequently, in the second edition of the WHO classification in 1992, three of the "cemental" lesions were reclassified under the "neoplasms and other tumors related to bone" category, leaving benign cementoblastoma as the only true neoplasm of dental cementum. This edition also introduced the term "cemento-osseous dysplasia," which encompassed conditions such as "florid cemento-osseous dysplasia," "periapical cemental dysplasia," and "other cemento-osseous dysplasias." [4]

Later, to address the shortcomings of his original classification, Waldron examined the subject of benign fibro-osseous lesions of the jaws (BFOL) in 1993 and proposed a change to his previous classification.

1. Fibrous Dysplasia
2. Cement-Osseous Dysplasia
    a. Periapical Cement-Osseous Dysplasia
    b. Focal Cement-Osseous Dysplasia
    c. Florid Cement-Osseous Dysplasia
3. Fibro-Osseous Neoplasm
    a. Cementifying Fibroma
    b. Ossifying Fibroma
    c. Cement-Ossifying Fibroma

Brannon and Fowler presented a categorization that differed from Waldron's and the WHO's by including more fibro-osseous lesions (FOL). Due to disagreements over cementum-like tissues, the 2005 WHO classification called cemento-osseous dysplasia (COD) osseous dysplasias, eliminating the term "cement". It emphasized diagnosing these lesions using clinical, histological, and radiological criteria. [4]

Speight and Carlos updated the classification in 2006, drawing on earlier system insights. Despite ongoing terminology challenges, this classification emphasized histopathological features to aid in diagnosis. Eversole et al. (2008) updated the categorization to include neoplasms, developmental dysplastic lesions, and inflammatory/reactive processes, emphasizing the importance of combining clinical, imaging, and microscopic examinations for a definite diagnosis. [4]

FIBROUS DYSPLASIA

Fibrous dysplasia (FD) is a benign, non-hereditary bone disorder characterized by the replacement of normal bone with fibro-osseous tissue, leading to bone deformity, pain, and structural weakness. It is caused by a post-zygotic activating mutation in the GNAS1 gene, which results in excessive cyclic AMP production and abnormal osteoblastic differentiation. This leads to the replacement of normal bone with fibrous tissue and immature woven bone. The extent of bone involvement depends on the timing of the mutation during embryogenesis [5].

FD can be classified into monostotic and polyostotic forms. Monostotic FD is the most common type, affecting a single bone, typically the ribs, femur, tibia, or craniofacial bones. It is usually asymptomatic but may present with mild swelling, deformity, or pathological fractures. Craniofacial fibrous dysplasia, a subtype of monostotic FD, affects the skull and facial bones—most often the maxilla—and may cause facial asymmetry, proptosis, vision impairment, or hearing loss. Polyostotic FD, a more severe form, involves multiple bones and is associated with skeletal deformities and fractures. It can be seen in syndromic conditions such as McCune-Albright Syndrome, which presents with café-au-lait pigmentation and endocrine dysfunctions, and Mazabraud Syndrome, which is characterized by the presence of soft tissue myxomas[5].

Clinically, fibrous dysplasia usually manifests in childhood or adolescence, often before the age of 30. It presents as a slow-growing, painless swelling that may lead to facial asymmetry. While most cases are asymptomatic, some patients may experience mild pain or discomfort. Involvement of the jawbones can cause displacement of teeth, though they typically remain vital. Polyostotic fibrous dysplasia is sometimes associated with syndromic conditions such as McCune-Albright syndrome or Jaffe-Lichtenstein syndrome, which present with additional systemic manifestations. Due to its progressive nature, early diagnosis is crucial for monitoring and management. A thorough correlation between clinical, radiological, and histopathological findings is essential to distinguish fibrous dysplasia from other fibro-osseous lesions and ensure appropriate treatment planning [6].

Radiographically, fibrous dysplasia appears as a poorly defined, expansile bony mass with a characteristic "ground-glass" or "whorled" appearance. The lesion blends with the surrounding bone, causing cortical expansion without perforation. Depending on its stage, it may exhibit varying degrees of radiopacity and radiolucency. The maxilla is more commonly involved than the mandible, and lesions may be monostotic or polyostotic. Advanced imaging techniques such as CT scans further highlight the classic ground-glass pattern, aiding in diagnosis and differentiation from other bone pathologies [6].

HISTOPATHOLOGICAL FEATURES

Histopathologically, fibrous dysplasia is characterized by the replacement of normal bone with irregularly shaped, immature woven bone trabeculae embedded within a fibrocellular stroma. These trabeculae, which lack a lamellar organization, exhibit a distinctive "Chinese character" or "alphabet soup" appearance. A key diagnostic feature is the absence of osteoblastic rimming, which helps differentiate fibrous dysplasia from other fibro-osseous lesions such as ossifying fibroma and osteoblastoma. The fibrous stroma consists of moderately cellular, spindle-shaped fibroblasts with loosely arranged collagen fibers and lacks inflammatory infiltrates. Unlike cemento-osseous dysplasia or ossifying fibroma, fibrous dysplasia does not contain basophilic, cementum-like deposits. Additionally, the lesion is not encapsulated and merges gradually with the surrounding normal bone, in contrast to ossifying fibroma, which is well-demarcated and encapsulated. The vascularity in fibrous dysplasia is generally mild, without significant hemorrhagic changes or aneurysmal bone cyst-like features. In polyostotic cases, particularly those associated with McCune-Albright syndrome, scattered multinucleated osteoclast-like giant cells may be present. Due to overlapping histopathological

features with other fibro-osseous lesions, a definitive diagnosis of fibrous dysplasia requires correlation with clinical and radiological findings [6].

TREATMENT

- Asymptomatic cases: Regular follow-up without intervention.
- Symptomatic cases:
    - Medical management: Bisphosphonates to reduce pain and bone turnover.
    - Surgical treatment:
        - Conservative recontouring for aesthetic concerns.
        - Bone grafting or osteotomy for severe deformities.
        - Radical resection is rarely needed.
- Radiation therapy is contraindicated due to the risk of malignant transformation to osteosarcoma.

CEMENTO- OSSEOUS DYSPLASIA

Cemento-Osseous Dysplasia (COD) is a benign, non-neoplastic fibro-osseous lesion that affects the jawbones, primarily in the tooth-bearing regions of the mandible. It is characterized by the progressive replacement of normal bone with fibrous tissue and cementum-like or osseous material. Although the exact etiology remains unclear, COD is believed to arise from dysregulated bone and cementum remodeling, possibly influenced by genetic predisposition, hormonal factors, or local inflammatory stimuli. It is more frequently observed in middle-aged women, particularly those of African, Asian, or Hispanic descent, suggesting a potential genetic or hormonal component. The lesion originates from the periodontal ligament and follows a self-limiting maturation process, transitioning from an initial radiolucent phase to a mixed and eventually radiopaque stage [7].

Clinically, COD is often asymptomatic and is usually detected incidentally during routine radiographic examinations. In some cases, patients may present with mild discomfort or swelling, though pain is typically absent unless secondary infection occurs. The condition does not cause tooth mobility or root resorption, and the affected teeth remain vital. COD can be classified into three subtypes: Periapical Cement-Osseous Dysplasia (PCOD), which occurs around the apices of vital anterior mandibular teeth; Focal Cement-Osseous Dysplasia (FCOD), which presents as a solitary lesion in the posterior mandible; and Florid Cement-Osseous Dysplasia (FCOD), which affects multiple quadrants of the jaw and may be associated with simple bone cysts. Florid COD, in rare cases, can lead to secondary infection due to exposure of sclerotic bone [7].

Radiographically, COD progresses through distinct stages. The early osteolytic stage appears as a well-defined radiolucent lesion, followed by a mixed stage, where radiolucent and radiopaque areas coexist. In the mature stage, the lesion becomes predominantly radiopaque with a dense, sclerotic appearance. Typically, COD does not exhibit cortical expansion or bone destruction, distinguishing it from aggressive neoplastic lesions. Advanced imaging techniques such as CT scans can help in confirming well-demarcated sclerotic masses without significant bone resorption [8].

HISTOPATHOLOGICAL FEATURES

Histopathologically, cemento-osseous dysplasia (COD) is characterized by a fibro-osseous matrix composed of a mixture of fibrous connective tissue, bone, and cementum-like deposits. In the early stages, the lesion consists predominantly of highly cellular fibroblastic tissue with minimal calcification. As the lesion matures, irregularly shaped bone trabeculae and spherical or lobulated basophilic cementum-like calcifications gradually increase in number. The calcified structures may appear fused or interspersed within the fibrous stroma. Unlike fibrous dysplasia, COD exhibits prominent cementum-like deposits that are often acellular and eosinophilic. Additionally, osteoblastic rimming may be observed in some areas, particularly around newly formed bone. The lesion is not encapsulated but remains well-demarcated from the surrounding bone. There is no evidence of inflammatory infiltration unless secondary infection occurs. As COD progresses, the lesion becomes more mineralized, transitioning from a predominantly fibrous stage to a mixed and eventually fully calcified, radiopaque stage. These histopathological features, along with clinical and radiographic correlation, are essential for an accurate diagnosis [8].

TREATMENT

- Asymptomatic cases: No treatment required; periodic radiographic follow-up.
- Symptomatic cases (infection, expansion, or exposure):
  - Antibiotics if infection occurs.
  - Avoid unnecessary extractions or invasive procedures to prevent secondary osteomyelitis.
  - Surgical debridement in cases of infected necrotic bone [8].

FIBRO-OSSEOUS NEOPLASM

Fibro-osseous neoplasms are benign bone tumors characterized by the replacement of normal bone with fibrous tissue and varying amounts of mineralized material, such as bone or cementum-like structures. These lesions differ from reactive fibro-osseous conditions, like Cement-Osseous Dysplasia, due to their progressive growth and potential for bone expansion. Fibro-osseous neoplasms are believed to result from a combination of genetic, environmental, and local factors, with post-zygotic mutations, such as those in the GNAS1 gene, leading to abnormal osteoblast and fibroblast differentiation. While some cases may have a hereditary component, most are considered sporadic, triggered by trauma, inflammation, or abnormal mesenchymal activity. Hormonal factors may also influence their development, particularly in syndromic cases like McCune-Albright Syndrome. Despite their benign nature, these neoplasms lack malignant potential but can cause significant structural changes [9].

There are three main types of fibro-osseous neoplasms: Cementifying Fibroma (CF), Ossifying Fibroma (OF), and Cemento-Ossifying Fibroma (COF). CF is a slow-growing tumor primarily composed of fibrous tissue and cementum-like calcifications, typically affecting the mandible and displacing adjacent teeth. OF is characterized by fibrous stroma and bone formation, often found in the posterior mandible and associated with facial asymmetry due to its higher growth potential. COF is a hybrid lesion containing both cementum-

like and bone-like components, often affecting the mandible and exhibiting a well-defined growth pattern. Each of these types presents with distinctive features and growth behavior, influencing clinical management and prognosis [9,10].

Clinically, fibro-osseous neoplasms often present as painless swelling or mass formation, which may lead to facial deformities if located in the craniofacial region. These lesions can cause functional impairment, such as difficulty with chewing or swallowing, depending on their size and location. In advanced cases, pathological fractures may occur due to weakened bone structure. Radiographically, these lesions initially appear as well-defined radiolucent areas with a characteristic "ground-glass" or "whorled" appearance, which later become more radiopaque with varying degrees of mineralization. As the lesions mature, they exhibit a mixed radiolucent-radiopaque pattern, eventually becoming predominantly radiopaque in their mature form. Advanced imaging techniques like CT or MRI provide more detailed insights into the lesion's extent, aiding in surgical planning and intervention [11,12].

HISTOPATHOLOGICAL FEATURES

Histopathologically, fibro-osseous neoplasms are characterized by a mixture of fibrous tissue and mineralized components such as bone, cementum, or a combination of both. The fibrous tissue is typically well-organized, often appearing dense and cellular with varying amounts of collagen fibers. The mineralized material can present as immature woven bone, cementum-like calcifications, or osseous material arranged in trabecular or nodular patterns. In Cementifying Fibroma, the cementum-like material is typically surrounded by fibrous stroma, while in Ossifying Fibroma, mature bone formation is more pronounced. The lesion may show varying degrees of mineralization, and the boundaries between the fibrous tissue and mineralized areas are usually well-defined. There may also be areas of fibrosis with osteoblastic rimming, which are indicative of active bone formation. The histological features, such as the arrangement and type of mineralized tissue, help in distinguishing fibro-osseous neoplasms from other fibro-osseous conditions and play a crucial role in determining the diagnosis [13,14].

TREATMENT

- Surgical excision with enucleation or curettage.
- Larger lesions may require segmental resection with bone grafting.
- Prognosis is good, with a low recurrence rate if completely removed.
- Regular radiographic follow-up is necessary to monitor for recurrence [15,16].

CONCLUSION

Fibro-osseous lesions (FOLs) of the maxillofacial bones encompass a diverse group of neoplastic and non-neoplastic conditions with overlapping histopathological, clinical, and radiological features. Accurate diagnosis requires a multidisciplinary approach, integrating clinical, radiographic, and histopathologic findings. Advanced imaging and collaboration with oral and maxillofacial radiologists can aid in distinguishing

between conditions like fibrous dysplasia, ossifying fibroma, osseous dysplasia, and others. Understanding the molecular biology and radiological patterns of these lesions is crucial for proper classification and management. Conservative treatment is recommended for many FOLs, except for lesions like osteoblastoma, which necessitate aggressive intervention. A thorough and precise diagnosis ensures appropriate therapeutic action and optimal patient outcomes. [17,18]

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