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Review Article

FIBRO OSSEOUS LESIONS IN MAXILLOFACIAL BONES

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ABSTRACT:
Fibro-osseous lesions of the maxillofacial bones should be classified based on their radiographic growth pattern. This method can simplify this category of lesions, which have considerable overlapping histologic features. These neoplasms can be grouped into three categories: (a) fibrous dysplasia; (b) ossifying fibroma; (c) and osseous dysplasia. Important lesions in the differential diagnosis are osteoblastoma and giant cell reparative granuloma.
Keywords: Fibrous dysplasia, Ossifying fibroma, Osseous dysplasia, Osteoblastoma, Giant cell reparative granuloma

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INTRODUCTION

Fibro-osseous lesions are a diverse group of processes that are characterized by replacement of normal bone by fibrous tissue containing a newly formed mineralized product. Commonly included among the fibro-osseous lesions of the jaws are fibrous dysplasia, cemento osseous dysplasia, and ossifying fibroma. Fibro-osseous-lesions of the maxillofacial bones are benign proliferations of spindle cells with varing amount of woven bone.[1]

CLASSIFICATION

Prior to reviewing the features of each entity subsumed under the heading of FOL of the craniofacial complex, we will proceed with a nosology of these lesions and as the discussion progresses, we will attempt to support the basis for this classification (Table 1). Whereas some investigators include giant cell lesions of bone with FOL, lesions of this nature will not be included here with the exception of the trabecular variant of Ossifying Fibroma which is essentially a FOL yet may contain foci of multinucleated giant cells.[2,3]

Table 1
Classification of benign fibro-osseous lesions of the craniofacial complex

I. Bone dysplasias
a. Fibrous dysplasia
i. Monostotic
ii. Polyostotic
iii. Polyostotic with endocrinopathy (McCune-Albright)
iv Osteofibrous dysplasia
b. Osteitis deformans
c. Pagetoid heritable bone dysplasias of childhood
d. Segmental odontomaxillary dysplasia
II. Cemento-osseous dysplasias
a. Focal cemento-osseous dysplasia
b. Florid cemento-osseous dysplasia
III. Inflammatory/reactive processes
a. Focal sclerosing osteomyelitis
b. Diffuse sclerosing osteomyelitis
c. Proliferative periostitis
IV. Metabolic Disease: hyperparathyroidism
V. Neoplastic lesions (Ossifying fibromas)
a. Ossifying fibroma NOS
b. Hyperparathyroidism jaw lesion syndrome
c. Juvenile ossifying fibroma
i. Trabecular type
ii. Psammomatoid type
c. Gigantiform cementomas

Table 2 lists the variations in histology among BFOL. These variant appearances may be unique to one disease yet in other instances, three or four entities may share the same histology even though they

represent separate and distinct clinicopathologic entities

Table 2: Microscopic similarities and dissimilarities among fibro-osseous lesions[4]

Fibrous element variations
Homogeneous plump monomorphic fibroblasts, hypercellularity, thin collagen fibers
Mature, hypocellular
Fasiculated, Storiform
Ossification (trabeculation) variations
Metaplastic woven bone
"Chinese/Hebrew" figure trabeculae
Lamellar bone trabeculae
Osteoblastic rimming
Mosaic resting/reversal lines
Trabecular paralleling
Cemental woven
Cemental microlamellar
Sharpey fiber fringe
Droplet (psammomatoid)
Curvilinear conglomerates ("Ginger root")

Essentially, the stromal element of BFOL may be quite homogeneous yet hypercellular with monomorphic appearing fibroblasts, whereas in others, the stroma is more mature or collagenous and then in yet others a storiform fibroblastic pattern prevails. The ossifications in BFOL can be quite heterogeneous even within a specific disease entity. Newly formed bone shows a woven pattern of collagen fiber orientation when viewed under polarized light. Mature bone exhibits a lamellar pattern as does dental cementum although the latter is microlamellar (Fig. 1). Many BFOLs are found to have both irregular trabeculae as well as spheroidal cementicle calcifications, so-called "cemento-ossifying" lesions. The ossification patterns seen in BFOL often represent the "age" of the lesion; formative processes in the early stages are more cellular and osteoblastic rimming of trabeculae is more prominent than older lesions of longer duration in which the stroma is more mature. As each entity is reviewed, the predominant histopathologic patterns will be specified (Genetic lesions are listed in Table 3)[4, 5]

Table 3: Genomic alterations in fibro-osseous lesions

Disease	**Genomic lesions**
Adult osteitis deformans	Sequestosome 1 gene (SQSTM1), a scaffold protein in the NFkappaB pathway; Inactivating mutations in TNFRSF11B, which encodes osteoprotegerin (a decoy receptor for RANK ligand)
Childhood Paget disease (idiopathic hyper-Phosphatasemia	Insertion mutations in TNFRSF11A, a receptor activator of nuclear factor (NF)kappaB (RANK)-a critical regulator of osteoclast function
Hereditary inclusion body myopathy, Paget's disease and fronto-temporal dementia	Mutation in Valosin-containing protein (VCP), targeting the inhibitor of NFkappaB for degradation by the proteasome
Familial expansile osteolysis/expansile skeletal Hyperphosphatasia	Tandem duplications in TNFRSF11A
Fibrous dysplasia, McCune Albright syndrome	Mutations of the Gsalpha gene (GNAS), the alpha-subunit of the stimulatory G protein
Hyperparathyroidism associated ossifying fibroma	Mutations in tumor suppressor gene HRPT2
Psammomatoid Ossifying Fibroma	Chromosomal breakpoints t(X;2)(q26;q33); interstitial insertion of bands 2q24.2q33 into Xq26

HISTOPATHOLOGY

All fibro-osseous lesions of the jaw and face are variations of the same histological pattern. This pattern consists of a bland spindle cell population mixed with varying amounts of woven bone and occurs in fibrous dysplasia, ossifying fibroma, and osseous dysplasia. The most typical pattern is that seen with classic fibrous dysplasia. Spindle cells are intermixed throughout with woven bone. The woven bone is dispersed in the fibrous background in a pattern classically described as "Chinese Letters".Almost always, there are no phenotypic osteoblasts seen synthesizing this bone. The amount of woven bone production in these lesions varies. In some cases, the amount of woven bone is minimal and the lesion consists predominantly of very cellular spindle cells). Other times, woven bone can be quite abundant and occurs as large islands of bone. Sometimes woven bone shows early transformation into lamellar bone. Another pattern of bone formation is the formation of small osteoid globules. These globules are often called "cementicles".[6]

Lesions with abundant ossification in this matter have also been given the subtype as "cementoma". This pattern of ossification should not warrant a separate diagnostic category, and the term cementoma should not be used. Cementum has the same chemical make-up as bone in its relationship of type 1 collagen to

calcium hydroxyapatite crystals. When this tissue is not associated with the tooth root, as is normal cementum, it loses its identity as a specific tissue. Also, this globular pattern of bone formation is seen in lesions of fibrous dysplasia in the post cranial skeleton. It is also seen associated with other bone forming neoplasms such as osteoblastoma and osteosarcoma. Sometimes this pattern of mineralization occurs in the lining of unicameral bone cysts. For these reasons, we do not feel that this pattern of bone formation deserves a separate diagnostic category in the facial bones. Any of these three fibro-osseous lesions—fibrous dysplasia, ossifying fibroma, and osseous dysplasia—may have secondary aneurysmal bone cyst formation. This process can cause massive expansion of the lesions.[6,7]

FIBROUS DYSPLASIA

First described by Von Recklinghausen in 1891, fibrous dysplasia is a developmental defect of osseous tissue such that bone is produced with an abnormally thin cortex and marrow is replaced with fibrous tissue that demonstrates characteristic ground-glass appearance on x-ray examination.[9] The underlying defect in fibrous dysplasia is a mutation of the *GNAS1* gene, which leads to constitutive activation of gene products that preclude the maturation of osteoprogenitor cells and lead to development of abnormal bone matrix, trabeculae, and collagen, produced by undifferentiated mesenchymal cells. There exists a mainly self-limiting form of fibrous dysplasia classified as monostotic, which is characterized by dysplastic bone in a single location that remains relatively stable throughout life and a polyostotic form, which can exhibit aggressive growth placing adjacent structures at risk for compressive sequelae.[10],[11,12]

OSSIFYING FIBROMA

Ossifying fibromas are benign asymptomatic neoplasms of the maxillae that generally have slow growth and present proliferation of fibrous cell tissue, with a varying quantity of bone products that include bone, cement or a combination of these. They are often considered to be fibro-osseous lesions.[13]

Ossifying fibromas occur most often in the posterior region of the mandible and may also occur in the maxilla, commonly in the region of the canine fossa and in the area of the zygomatic arch. They are more common in females and present greatest incidence in the third and fourth decades of life. Facial asymmetry and tooth displacement may occasionally occur.

Two things help distinguish this lesion from classic fibrous dysplasia. First, lesions have been shown not to harbor the mutation in GNAS 1α . Also, patients with this fibro-osseous lesion generally do not have accompanying postcranial lesions.

Because of the expansile destructive nature of ossifying fibroma surgical excision is usually required. The recurrence rate is more common in younger patients.[14]

FLORID OSSEOUS DYSPLASIA

Florid osseous dysplasia also known as Cemento-osseous dysplasia [COD] is a benign condition of the jaws that may arise from the fibroblasts of the periodontal ligaments. It is most common in African-American females. The three types are periapical cemental dysplasia (common in those of African descent), focal cemento-osseous dysplasia (Caucasians), and florid cemento-osseous dysplasia (African descent). Periapical occurs most commonly in the mandibular anterior teeth while focal appears predominantly in the mandibular posterior teeth and florid in both maxilla and mandible in multiple quadrants.

On radiographic evaluation, during the early stage, a COD lesion may be detected as a round or oval apical radiolucency with a well-defined radiopaque border. In the second mixed stage, a radiolucent lesion may include radiopacities. In the mature stage, the internal mixed area becomes completely radiopaque with a thin radiolucent periphery.

Commonly, no treatment is required and only regular follow-up examinations are recommended.[15]

DIFFERENTIAL DIAGNOSIS

Fibro-osseous lesions of the jaw and face must be differentiated from other bone lesions which may mimic them histologically and radiographically. The most important lesions in the differential diagnosis are osteoblastoma and giant cell reparative granuloma. Osteoblastoma is a benign radiolytic bone-forming neoplasm which is most common in the postcranial skeleton, particular the posterior elements of the spine. It is a slow, but relentlessly growing, neoplasm which may destroy the structural architecture of the bone. Osteoblastomas also occur in the maxillofacial region. Radiographically, they are lytic lesions with focal radiodensity. In this area, they exhibit the same behavior of relentless growth that they do in the postcranial skeleton. Sometimes they grow very large and are regarded as "aggressive" osteoblastomas.[16] Histologically, broad seams of interlacing osteoid are present with varying degrees of mineralization. The central feature to differentiate this pattern from fibro-osseous lesions is that the stroma does not consist of cellular spindle cells but rather a loose vascular stroma with numerous prominent epithelioid-type osteoblasts . This stromal component is the most important feature to differentiate osteoblastoma from a fibro-osseous process. Osteoblastomas must be curetted to stop their relentless growth.

The second lesion which is often confused with a fibro-osseous process is giant cell reparative granuloma. In the craniofacial bones, this lesion is a well-defined lytic process. This lesion consists of

multinucleated osteoclast-like giant cells, fibrous tissue and reactive bone in a zonal pattern. Often the giant cells are quite prominent and are associated with extravasated red blood cells. Giant cell reparative granuloma is a reactive process that is the result of the early repair of a large resorptive defect. A florid form of multiple reparative granulomas, known as cherubism is an autosomal dominant inherited syndrome cause by mutation in the SH3BP2 gene on chromosome 4p16-3.[17]

The natural history of both a single giant cell reparative granuloma and cherubism is to undergo spontaneous healing over time. As this healing process continues, the giant cells disappear leaving only the fibrous stroma and reactive bone in a zonal pattern. This zonal pattern is the most distinctive feature of giant cell reparative granuloma in both the early and in the healing phases and should distinguish giant cell reparative granuloma from a fibro-osseous lesion.

Another lesion which has been confused with fibro-osseous lesions is the so-called "sclerosing osteomyelitis". This should not be regarded as a specific entity in the jaw. It is the same process of chronic osteomyelitis in any bone which is characterized radiologically by broad zones of sclerosis. Histologically, there is abundant reactive bone and the intervening space is filled with fibroinflammatory tissue. This fibroinflammatory tissue enables this lesion to be recognized as chronic infection. However, the diagnosis of osteomyelitis can only be rendered provided there has been an intraoperative culture that is positive for organisms. We do not make the diagnosis of osteomyelitis without a positive culture.

Finally, osteosarcomas may occur in the jaw. However, most osteosarcomas in the face and jaw are chondroblastic osteosarcomas and are rarely confused with a fibro-osseous process. On occasion, a conventional osteoblastic osteosarcoma may occur in the jaw. These are easily distinguished from a fibro-osseous lesion in that the stroma shows distinctly pleomorphic cells with abundant atypical mitotic figures.[18]

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