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Fibro-osseous Lesions of the Jaws

CHARLES A. WALDRON, DDS, MSD*

In the first volume of the *Journal of Oral Surgery*, Dr G. Victor Boyko presented a case of osteofibroma of the mandible associated with leontiasis ossea of the skull (Boyko GV: *J Oral Surg* 1:100, 1943). The patient was a 32-year-old white woman who had complaints of right mandibular enlargement and a prominence of the right frontal and temporal areas of uncertain duration in August 1939. Mandibular radiographs showed an area of reduced radiodensity. Skull films showed a marked increase in density of the inferior part of the right temporal region and of the frontal bone, with evidence of both bone destruction and proliferation in the inferior frontal and orbital regions. A mandibular biopsy was reported as osteofibroma. The patient was kept under observation for 8 months after the biopsy, with little change in her condition. A course of deep x-ray therapy was then delivered to the mandibular lesion. She subsequently developed a pathologic fracture that was treated by maxillomandibular fixation. The fracture stabilized and at the last clinical examination, in June 1942, the patient had a union with good functional occlusion and was reported to be in good mental and physical condition.

Our concepts regarding fibro-osseous lesions have undergone considerable refinement during the 50 years since Boyko's article was published. In the 1940s and early 1950s these lesions were commonly designated as localized osteitis fibrosa, osteofibroma, or fibrous osteoma. These terms have seldom been used in the last 30 or 40 years. The skull lesions in Boyko's case were designated as leontiasis ossea. This term was suggested by Vichow in 1862 for bone disease involving the upper facial bones that resulted in a lion-like appearance. Whereas some have equated leontiasis ossea with the facial deformity seen in some cases of Paget's disease, others have used the term to describe facial deformity resulting from a variety of disease processes. In any event, leontiasis ossea is a vague clinical term and implies no specific type of pathology.¹

It is likely that Boyko's patient would be diagnosed today as having craniofacial fibrous dysplasia. It is of great interest that this patient received a course of radiation therapy for her mandibular lesion, which was

advocated by many authorities at that time for treatment of these lesions. This therapy, of course, antedated recognition of the problem of postradiation bone sarcoma, which was not recognized until the early 1950s.

Despite the advances in our understanding of these conditions, fibro-osseous lesions continue to present problems in classification, diagnosis, and management. Although there is no universally agreed on classification, the following is suggested as a useful, working classification for fibro-osseous jaw lesions. It must be emphasized that precise diagnosis requires good clinical, radiologic, and histologic correlation because the histologic findings alone may be similar for lesions with diverse behavioral characteristics and prognosis. In the absence of good clinical and radiologic information, the pathologist can only state that a given biopsy is consistent with a benign fibro-osseous lesion. With adequate clinical, and radiologic data, most fibro-osseous jaw lesions can be assigned with reasonable certainty into one of several categories (Table 1). However, it must be admitted that some cases still defy exact classification.

Fibrous Dysplasia

The etiology of fibrous dysplasia (FD) is unknown. It is widely considered to be a developmental (hamaromatous) lesion. Although it is usually classified as a nonneoplastic disorder, some examples show neoplas-

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Table 1. Classification of Fibro-osseous Lesions

I. Fibrous dysplasia
II. Reactive (dysplastic) lesions arising in the tooth-bearing area. These are presumably of periodontal ligament origin. It is convenient to divide them into three types based on their radiologic features although they seem to represent the same pathologic process. periapical cemento-osseous dysplasia focal cemento-osseous dysplasia florid cemento-osseous dysplasia
III. Fibro-osseous neoplasms These are widely designated as cementifying fibroma, ossifying fibroma, or cemento-ossifying fibroma.

tic-like clinical features. FD occurs both in polyostotic and monostotic forms. Polyostotic FD is uncommon and may involve only multiple bones, or it may be accompanied by skin pigmentation and a variety of endocrine disturbances (McCune-Albright syndrome).

Monostotic FD is more common than the polyostotic type. The jaws and skull are among the most commonly affected bones. Although mandibular lesions may be truly monostotic, maxillary lesions, which are more common than mandibular lesions, often involve adjacent bones such as the zygoma, sphenoid, and occipital, and they are not in a strict sense "monostotic." The designation of craniofacial FD is appropriate for these lesions.^{2,3}

Clinically, a painless expansion of the affected area is the most common indicator of FD. The disease is most often detected during the first two decades of life. Milder examples may not be diagnosed until later in life, but a careful history will often indicate that the lesion was first noted during the first or second decades. The most typical radiographic feature is that of a ground-glass opacification, although early lesions may be largely radiolucent. The lesion is not radiographically well defined and it blends imperceptibly into the surrounding bone. This is an important feature in differentiating FD from ossifying fibroma, which is radiographically well defined. Lateral skull views of maxillary lesions will frequently show increased density of the base of the skull involving the occiput, sella turcica, roof of the orbit, and frontal bones. This is said to be the most characteristic radiographic feature of FD of the skull. Maxillary lesions commonly involve the maxillary sinus so that Waters' views show a radiodense area that largely or totally obliterates the maxillary sinus. Teeth in the involved area are not displaced and remain firm. Root resorption is seldom if ever associated with FD (Fig 1).

Histologically, classic FD shows irregularly shaped trabeculae of immature woven bone in a cellular fibroblastic stroma. The bone trabeculae tend to be del-

icate and are not connected to one another. They often assume curvilinear shapes resembling Chinese script writing. The bone trabeculae are not surrounded by osteoid rims or osteoblasts. Although there is wide agreement that FD of the long bones does not undergo lamellar maturation, jaw and skull lesions tend to be more ossified than their counterparts in the extra-gnathic skeleton. This is particularly true in specimens from older patients.

Serial biopsies in some cases have shown that histologically classic FD may undergo a progressive maturation to a lesion consisting of lamellar bone in a moderately cellular fibrous connective tissue stroma.

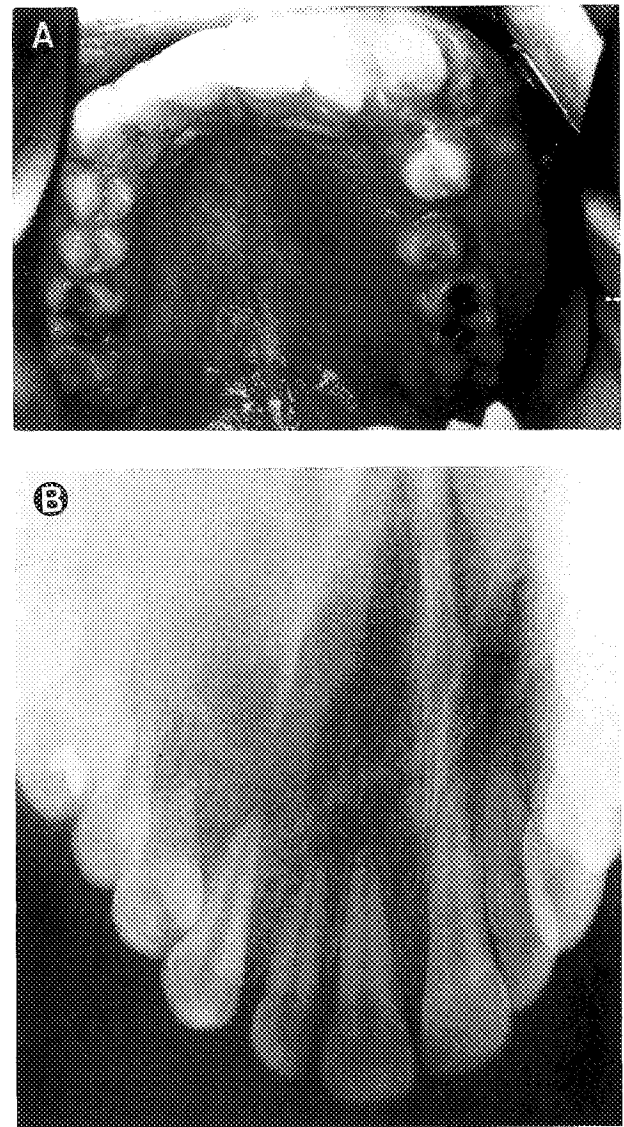


FIGURE 1. Craniofacial fibrous dysplasia. A, Intraoral view of lesion causing unilateral expansion of the maxilla in a white boy, age 11. B, Occlusal radiograph from a boy age 12 with fibrous dysplasia showing the ground-glass opacification and ill-defined margins. The clinical features in this patient were identical with those of the patient shown in A.

The bony trabeculae in these "mature" lesions tend to run parallel to one another⁴ (Fig 2).

Clinical management of FD of the jaws may be a major problem. Although small lesions, particularly in the mandible, may be amenable to complete resection, the diffuse nature and large size of many lesions, especially those of the maxillary complex, precludes their removal without extensive surgical procedures. In most cases, the disease tends to stabilize and essentially stops growing when skeletal maturation is reached. Some patients with minimal cosmetic or functional deformity may not require surgical treatment. However, cosmetic deformity, with associated psychological problems or functional deformity, may require surgical intervention in the younger patient. This usually entails surgical reduction of the lesion to an acceptable contour without attempting complete removal. Although this usually achieves a good cosmetic result, there may be a slow continued regrowth of the lesion. Reliable data as to the true incidence of continued growth after surgical

reduction of FD of the jaws are difficult to determine, but it is estimated that between 25% and 50% of patients will show some regrowth after a shave-down procedure.⁵ Regrowth after surgical reduction procedures appears to be more common in younger patients suggesting that surgical intervention should be delayed as long as possible.

Malignant change in fibrous dysplasia, usually an osteosarcoma, rarely has been reported. Most examples have been seen in patients who have received prior radiation therapy for FD, but there are some examples of spontaneous sarcomatous change.^{6,7} Radiation therapy for FD is definitely contraindicated as it carries the risk of postirradiation bone sarcoma. It is wise to keep any patient with FD under long-term follow-up and any patient showing clinical or radiologic evidence of change after a long period should be subjected to adequate biopsy to rule out sarcomatous change.

Reactive (Dysplastic) Fibro-osseous-cemental Lesions of the Tooth-Bearing Areas

These are the most common fibro-osseous lesions of the jaws. Their etiology is unknown, but they appear to originate from elements of the periodontal ligament. Based on the clinical and radiologic features, it is convenient to divide these lesions into three subtypes, although they appear to represent only variants of the same basic disease process.

PERIAPICAL CEMENTO-OSSEOUS DYSPLASIA

Periapical cemento-osseous dysplasia (PCOD) is a reasonably well-defined clinical-radiologic entity. The lesions predominantly involve the apical areas of vital mandibular incisor teeth. Multiple lesions are often present. There is a striking predilection for both female and black patients. Most patients are older than 30 years when the lesions are first noted and the condition is seldom seen in a patient who is younger than 20 years. PCOD is invariably an asymptomatic lesion that is discovered on a radiographic examination.

Radiographically the lesion appears as a well-circumscribed radiolucent, mixed radiolucent/radiopaque, or radiopaque lesion involving the apices of one or several teeth. Individual lesions are seldom more than 1.0 cm in diameter and most are less than 0.5 cm in size. In many cases, serial radiographic study has shown that the lesion first presents as a circumscribed radiolucent lesion, which over the course of several years shows increasing degrees of calcification. Individual lesions show little tendency to enlarge. Despite the fact that PCOD is a well-recognized condition, patients are still subjected to endodontic therapy for the mistaken diagnosis of periapical cyst or granuloma. Accurate pulp testing should avoid these errors.

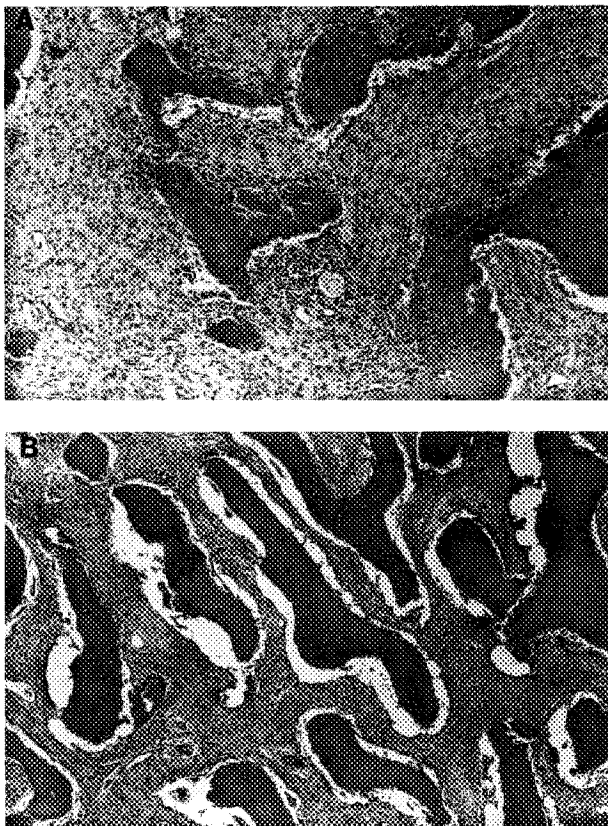


FIGURE 2. Photomicrographs of biopsies from patients with fibrous dysplasia. *A*, Biopsy from maxillary fibrous dysplasia in a white boy age 8. Irregular shaped trabeculae of woven bone are present in a cellular fibrous connective tissue stroma. (hematoxylin-eosin stain, original magnification $\times 60$). *B*, Photomicrograph from a reduction procedure for fibrous dysplasia in a 45-year-old black woman. This patient had a slowly progressing maxillary lesion for many years. Trabeculae of mature lamellar bone are present in a mature fibrous stroma (hematoxylin-eosin stain, original magnification $\times 60$).

There is general agreement that PCOD does not require treatment and in the typical case (ie, a black female with multiple lesions involving vital teeth) no further intervention is required. Isolated lesions in a less typical clinical-radiologic situation may dictate a biopsy to rule out a more significant pathological process. Because a biopsy is seldom performed on the lesion, there are no reports of the histologic features in any significant series of cases. The microscopic findings reported in limited numbers of cases are identical with those of focal cemento-osseous dysplasia (FCOD).

FOCAL CEMENTO-OSSEOUS DYSPLASIA

FCOD has received scant attention in the literature although it likely represents the most common fibro-osseous lesion seen in the oral pathology laboratory. Robinson properly noted in 1956 that some fibro-osseous lesions of the jaws did not meet the criteria for FD nor for ossifying fibroma. He suggested that these lesions be designated as an "osseous dysplasia reaction of bone to injury."⁸ Waldron also discussed these lesions in 1985 under the designation of "localized fibro-osseous cemental lesions—presumably reactive in nature."³ Tomich and Summerlin suggested in 1989 that FCOD is the most appropriate designation for these lesions.⁹ In a review of jaw fibro-osseous lesions at Indiana University, Tomich and Summerlin identified 175 cases of FCOD, as compared with 45 examples of ossifying fibroma. FCOD does not have the clinical and radiologic features of PCOD nor of florid cemento-osseous dysplasia (FLCOD), although the histologic features are essentially similar. In Tomich and Summerlin's review, 87% of cases were found in females and 79% were found in the posterior mandible, most often in edentulous areas. The majority of cases occur in the fourth and fifth decades of life.⁹

FCOD is almost invariably an asymptomatic lesion that is discovered on a radiographic examination. It presents as a fairly well demarcated radiolucent or mixed radiolucent/radiopaque area. However, some examples are largely sclerotic. Most lesions are less than 2.0 cm in size, but larger lesions may be seen. The majority of cases do not show bone expansion, but large lesions may cause slight jaw enlargement. On surgical exploration, the tissue occupying the defect is gritty, hemorrhagic, and is removed by curettage in small fragments, often with some difficulty. This is an important observation and contrasts with the relatively avascular and well-circumscribed nature of an ossifying fibroma, which tends to enucleate with relative ease. Microscopically FCOD shows areas of cellular fibrous tissue containing numerous small blood vessels. Irregular trabeculae of woven bone and/or cementum-like calcifications are intermingled among the fibroblastic

stroma. Scattered foci of multinucleated giant cells may be seen (Fig 3).

Simple bone cysts also may occur in FCOD lesions.¹⁰ These cysts may occasionally comprise a large component of the total lesion. The pathogenesis of these cysts developing in cemento-osseous dysplasia is not well understood. In some cases serial radiographic study has shown that the area of cemento-osseous dysplasia was present for some years before the cyst developed. In contrast to the rapid repair noted in the typical simple (traumatic) cyst in young patients, cysts associated with cemento-osseous dysplasia "heal" more slowly and the tissue filling the defect maintains an abnormal radiographic appearance.^{11,12}

In many cases of FCOD, serial radiographic study after biopsy has shown that the lesions have little or no tendency to enlarge even after only partial removal of the lesional tissue. The patients also tend to remain asymptomatic. Whether or not there should be further surgical intervention after partial removal for biopsy is debatable, but the long-term prognosis appears excellent. Separation of some larger examples of FCOD from ossifying fibroma may be arbitrary and which

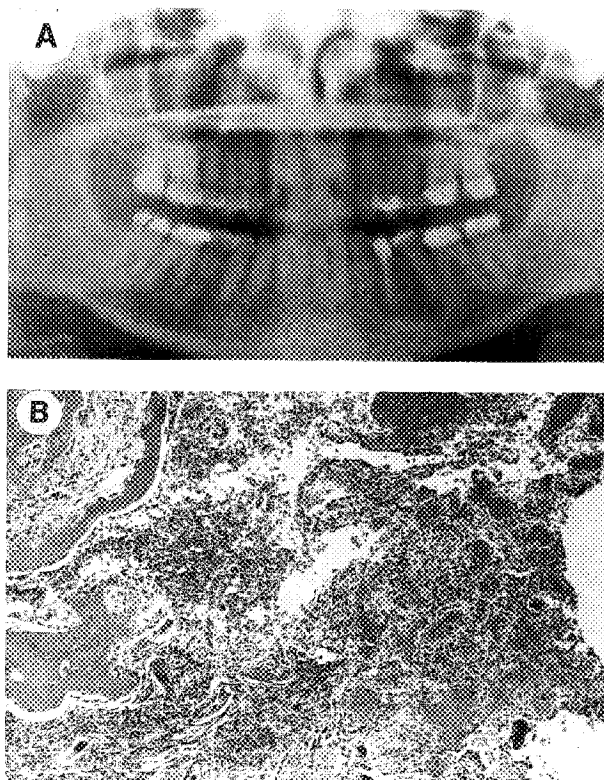


FIGURE 3. Focal cemento-osseous dysplasia in the mandibular left premolar region of a white woman age 44. *A*, Radiograph of the lesion partially removed at biopsy 5 years before showing no appreciable change. *B*, Photomicrograph from biopsy specimen showing hemorrhagic fibrous tissue containing trabeculae of woven bone and ovoid, acellular, cementum-like structures (hematoxylin-eosin stain, original magnification $\times 60$).

diagnosis to render in such cases may be a matter of personal preference.

FLORID CEMENTO-OSSEOUS DYSPLASIA

FLCOD is a reasonably well-defined entity within the group of cemento-osseous dysplasias. It is seen almost exclusively in middle-aged to elderly black females.¹² In the past this condition has been designated as sclerosing osteitis,¹³ multiple enostoses,¹⁴ diffuse chronic osteomyelitis,¹⁵ and gigantiform cementoma.⁴ Laboratory and radiologic studies have shown that the disease is limited to the tooth-bearing areas of the jaws and the patients do not have evidence of disease in other parts of the skeleton. The etiology of FLCOD is unknown and there is no good explanation for its sex and racial predilection. In some instances the disease appears to have a familial distribution. There is a striking tendency toward bilateral, symmetrical involvement and it is not unusual to find extensive lesions in all four posterior quadrants of the jaws. Many patients are partially or totally edentulous when the condition is first detected. The process may be totally asymptomatic and, in such cases, it is detected when radiographs are taken for some other purpose. When the lesions are large, jaw expansion may be noted. Symptoms such as dull pain or drainage are almost always associated with exposure of the sclerotic calcified masses to the oral cavity as the result of progressive alveolar atrophy under a denture or after extraction of teeth in the involved area (Fig 4).

The most common radiographic presentation is densely sclerotic lobular masses often symmetrically located in various areas of the jaws. These are usually admixed with less well-defined areas of a mixed radiolucent/radiopaque pattern. If the patient has remaining lower anterior teeth, they will often show circumscribed periapical lesions typical of PCOD. Well-defined lucent areas may be present and these represent simple bone cysts, which are not uncommonly associated with FLCOD. Microscopically FLCOD shows an admixture of woven bone trabeculae and droplets of cementum-

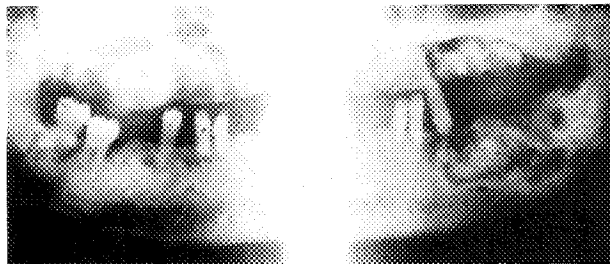


FIGURE 4. Florid cemento-osseous dysplasia in a black 58-year-old woman. All four quadrants are affected. The sclerotic masses in the left maxilla and mandible are exposed to the mouth and are sequestering.

like calcifications in a fibroblastic stroma. The cementum-like calcifications often fuse to form coalescing masses. The large globular calcifications are usually of a cementum-like morphology, although some pathologists consider them to be sclerotic bone in nature.

Management of FLCOD is often difficult and not very satisfactory. In the asymptomatic patient, it is probably wise to keep the patient under observation without surgical intervention because the radiologic features are diagnostic. Because the onset of symptoms is usually associated with exposure of the sclerotic masses to the oral cavity, biopsy or elective extraction of teeth in the involved area should be avoided. Management of the symptomatic patient is more difficult. At this stage there is an inflammatory component to the disease, and the process is basically a chronic sclerosing osteomyelitis involving dysplastic bone. Antibiotics should be administered, but may not be very effective. Sequestration of the cementum-like masses will occur slowly and this will be followed by healing. Saucerization or surgical excision of the sclerotic masses is often not successful and may make matters worse.

Fibro-osseous Neoplasms

Fibro-osseous neoplasms remain somewhat controversial, and differing concepts have been advanced regarding their nature and the proper terminology for them. The 1972 World Health Organization (WHO) classification separated the cementifying fibroma (CF), which was considered to represent an odontogenic tumor, from ossifying fibroma (OF), which was considered to be a tumor of osseous origin.¹⁶ This separation has been followed in many standard oral pathology texts.^{17,18} However, today there is general agreement that CF and OF represent only histologic variants of the same lesion and the 1992 WHO classification groups them under a single designation as cemento-ossifying fibroma.^{4,19} The tumor is defined as a demarcated and occasionally encapsulated lesion consisting of fibrous tissue containing variable amounts of mineralized material resembling bone and/or cementum. Although some of these tumors show only cementum-like calcifications and other may contain only bone, admixture of the two types of calcifications are commonly encountered. Whether or not these amorphous cementum-like calcifications truly represent cementum is uncertain because similar calcified structures may be encountered in fibro-osseous lesions of the skull that are anatomically far removed from the jaws.²⁰ Similar cementum-like bodies are also occasionally seen in extragnathic bone lesions, which makes their cemental nature very unlikely.

The common types of OF may occur in patients over a wide age range, but they are most often diagnosed during the third and fourth decades of life. The

lesions are largely restricted to the tooth-bearing areas of the jaws, although posterior mandibular lesions may extend upward into the ascending ramus for some distance. About 70% to 80% of these lesions occur in the mandible, most often in the premolar-molar region, and there is a definite female predilection. There is considerable evidence that these tumors originate from elements of the periodontal ligament.²¹ Large OFs are associated with a slowly progressing enlargement of the affected bone.

Radiographically, the tumor is a well-defined unilocular lesion. Depending on the amount of mineralized material produced in the tumor, it may appear radiolucent or show varying degrees of radiopacity. Root resorption and/or root divergence of associated teeth may be noted in some cases. On surgical exploration, the tumor is found to be relatively hypovascular and well-demarcated from the surrounding tissue, permitting relatively easy separation from the surrounding bone (Fig 5). Some lesions will have a definite capsule. This demarcation from the surrounding tissue is an important feature in distinguishing OF from FD.

Histologically, OF shows a range of histologic patterns. The soft tissue component consists of fibrous connective tissue with varying degrees of cellularity. The calcified structures consist of rounded or lobulated basophilic masses (cementum-like), trabeculae of osteoid or bone, or combinations of the two. Differentiation between FD and OF on histologic appearance alone is often impossible, and the distinction between these conditions requires clinical and radiographic information, which should be made available to the pathologist.

The growth rate of OF varies considerably, but most tumors show a slowly progressive enlargement. Occasional examples may grow to massive size causing considerable cosmetic and functional deformity. The histologic features of a given case are not particularly



FIGURE 5. Radiograph of an ossifying fibroma of the mandible in a 38-year-old white man.

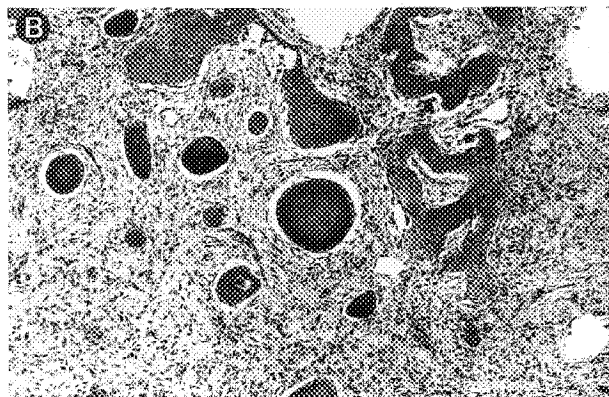
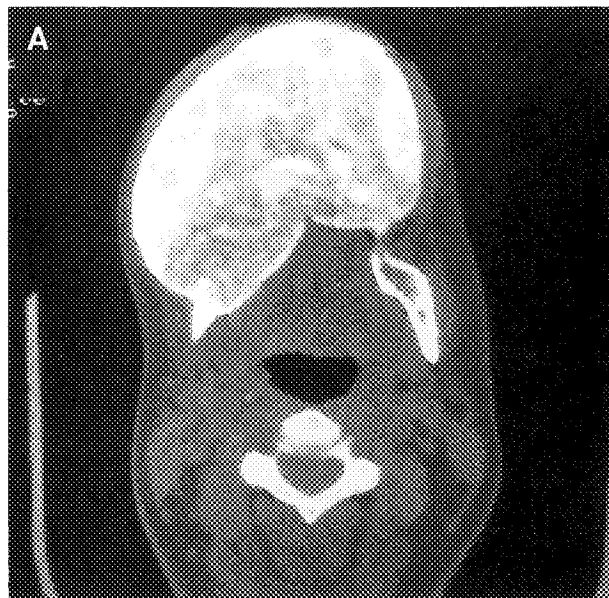


FIGURE 6. Massive ossifying fibroma of the anterior mandible in a black woman, age 39. *A*, Computed tomographic scan of lesion. It has had quadrupled in size over a 5-year period. *B*, Photomicrograph from the resection specimen. Trabeculae of woven bone and acellular cementum-like material are present in a mature fibrous stroma (hematoxylin-eosin stain, original magnification $\times 60$).

helpful in predicting the growth rate. An OF can usually be excised in one piece or removed by curettage in several large fragments. Massive tumors may require local resection and subsequent bone grafting. The prognosis is excellent and recurrence after removal seldom is encountered. Malignant transformation of an OF has not been documented (Fig 6).

Another and relatively rare type of fibro-osseous tumor has also been included under the "umbrella" of OF. These lesions are often designated as "juvenile," "active," or "aggressive" OFs, but there does not appear to be a generally agreed on criteria for separating these lesions from the more common types of OF. The juvenile OF (JOF) is most often seen in patients who are between 5 and 15 years of age (60% to 80% of cases),⁴ although examples have been diagnosed in

older patients. The majority of cases involve the maxilla, paranasal sinuses, and orbital and fronto-ethmoid bones, but mandibular lesions do occur. Johnson believes that the majority of these tumors arise in the paranasal sinuses and, with persistent growth, they involve the orbital, nasal, and cranial cavities, and the maxilla. He also believes that the mandibular examples arise from the myxoid dental papilla of developing teeth.²²

Clinically, the JOF is often characterized by a progressive and sometimes rapid expansion of the affected area. When the orbital bones and paranasal sinuses are involved, the patients may develop proptosis, exophthalmos, and bulbar displacement. The radiologic features are variable and, depending on the tumor's location and the amount of calcified tissue produced by the tumor, the lesion will show varying degrees of radiolucency and radiopacity. It may be fairly well demarcated or show invasion and erosion of the surrounding bone (Fig 7).

The histologic features ascribed to the JOF by various investigators are also variable and a range of features may be present. The tumor shows a cell-rich stroma of polyhedral and spindle-shaped cells that produce little collagen.

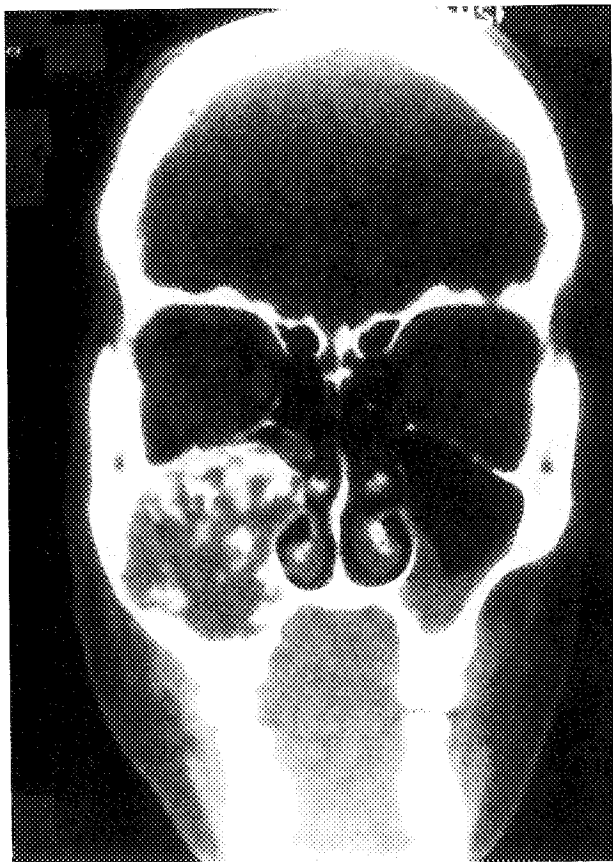


FIGURE 7. Computed tomographic scan of a large ossifying fibroma of the maxilla and maxillary sinus in a 10-year-old white girl.

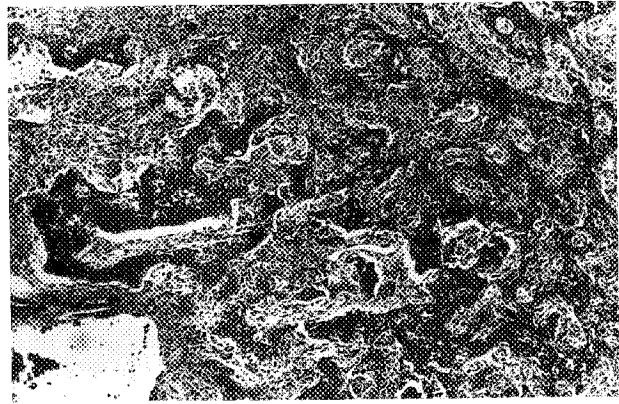


FIGURE 8. A biopsy specimen from patient in Figure 7 showing thin strands of ossoid in a cellular fibrous stroma (hematoxylin-eosin stain, original magnification $\times 60$).

According to Johnson, the characteristic mineralized component consists of numerous, small spherical ossicles of uniform size that are surrounded by osteoid rims.²² These structures have been designated by others as psammoma-like. Makek believes that lesions with this histologic pattern should be designated as psammous desmo-osteoblastoma.²³ Cystic structures apparently resulting from degeneration of myxoid stroma are often encountered in older lesions and may indicate regression of the tumor.

Other microscopic features attributed to the JOF include a cellular stroma with a low collagen fiber content. Small strands of immature cellular osteoid form within the lesion and these may bear a resemblance to the osteoid formed in an osteosarcoma.^{4,24} Foci of multinucleated giant cells also may be present. Maturation of the lesion results in formation of more typical trabeculae of woven bone. Lesions of this histologic type are designated as trabecular desmo-osteoblastoma by Makek (Fig 8).²³ Whether the psammoma-like histologic features and the trabecular pattern represent two separate entities or are only variants of a single lesion is not yet settled, but both have been designated as juvenile (active, aggressive) ossifying fibroma.

The clinical management and prognosis of the JOF is also somewhat uncertain. Some lesions may be present for long periods with minimal symptoms. Others, particularly in very young children, may show rapid growth and aggressive local behavior. Conservative local excision or thorough curettage of the lesion appears to be the preferable treatment. Recurrence rates of 30% to 58% have been reported for JOF, which is in sharp contrast with the negligible recurrence rates associated with the more common types of OF. Recurrence of JOF also may be managed by local surgical excision.^{22,24} Sarcomatous transformation of JOF has not been shown and radical surgery for this tumor does not appear to be indicated.

A number of other disease processes involving the jaws may be clinically, radiologically and/or histologically confused with the "conventional" fibro-osseous lesions of the jaws. These include some types of chronic osteomyelitis and periostitis, Paget's disease, hyperparathyroidism, osteoblastoma, and low-grade osteosarcoma. Separation of these lesions from the "conventional" fibro-osseous lesions can usually be accomplished by thorough evaluation of the clinical, radiologic, and histologic features, although in some cases this may be very difficult.

References

1. Drury BJ: Paget's disease of the skull and facial bones. *J Bone Joint Surg [Am]* 44:174, 1962
2. Harris WH, Dudley HR Jr, Barry RJ: The natural history of fibrous dysplasia. *J Bone Joint Surg* 44A:207, 1962
3. Waldron CA: Fibro-osseous lesions of the jaws. *J Oral Maxillofac Surg* 43:249, 1985
4. Kramer IRH, Pindborg JJ, Shear M: *Histologic Typing of Odontogenic Tumors* (ed 2). Berlin, Germany, Springer-Verlag, 1992
5. Koopmans K: Fibreize dysplasie en fibro-osseuze-cementeize dysplasie van den kaken. Thesis. University Groningen, The Netherlands, 1990
6. Slow IN, Stern D, Friedman EW: Osteogenic sarcoma arising in a pre-existing fibrous dysplasia. *J Oral Surg* 29:126, 1971
7. Huvos AG: *Bone Tumors: Diagnosis, Treatment and Prognosis* (ed 2). Philadelphia, PA, Saunders, 1991, p 41
8. Robinson HBG: Osseous dysplasia: Reaction of bone to injury. *J Oral Surg* 14:3, 1956
9. Tomich C, Summerlin DJ: Focal cemento-osseous dysplasia. Presented at American Academy of Oral Pathology Meeting, Savannah, GA, April 25, 1989 (essay #11)
10. Higuchi Y, Nakamura N, Tashiro H: Clinico-pathologic study of cemento-osseous dysplasia producing cysts of the mandible. *Oral Surg Oral Med Oral Pathol* 65:339, 1988
11. Saito Y, Hoshina Y, Nagamine T, et al: Simple bone cyst: A clinical and histopathologic study of fifteen cases. *Oral Surg Oral Med Oral Pathol* 74:487, 1992
12. Melrose RJ, Abrams AA, Mills BG: Florid Osseous dysplasia. *Oral Surg Oral Med Oral Pathol* 41:62, 1976
13. Leband PF, Leacock AG: Sclerosing osteitis of the jaws. *J Oral Surg* 25:23, 1967
14. Bhaskar SN, Cutright DE: Multiple enostoses: Report of 16 cases. *J Oral Surg* 26:321, 1968
15. Shafer WG: Chronic sclerosing osteomyelitis. *Oral Surg Oral Med Oral Pathol* 15:138, 1957
16. Pindborg JJ, Kramer IRH: *Histologic Typing of Odontogenic Tumors, Jaw Cysts and Allied Lesions*. Geneva, Switzerland, World Health Organization, 1971
17. Shafer WG, Hine MK, Levy BM: *A Textbook of Oral Pathology* (ed 4). Philadelphia, PA, Saunders, 1983, p 142, 298
18. Regezi JA, Sciubba JJ: *Oral Pathology: Clinical-Pathologic Correlations*. Philadelphia, PA, Saunders, 1989, pp 359, 369
19. Eversole LR, Leider AS, Nelson K: Ossifying fibroma: A clinicopathologic study of 64 cases. *Oral Surg Oral Med Oral Pathol* 60:505, 1985
20. Margo C, Ragsdale B, Perman K et al: Psammomatoid (juvenile) ossifying fibroma of the orbit. *Ophthalmol* 92:150, 1985
21. Hamner JE, Scofield HH, Cornyn J: Benign fibro-osseous lesions of periodontal ligament origin. *Cancer* 22:861, 1968
22. Johnson LC, Yousefi M, Vink T, et al: Juvenile active ossifying fibroma: Its nature, dynamics and origin. *Acta Otolaryngol Suppl (Stockh)* 488, 1991
23. Makek M: So-called "fibro-osseous lesions" of tumorous origin: Biology confronts terminology. *J Craniomaxillofac Surg* 13:154, 1987
24. Fu Y-S, Perzin KH: Non-epithelial tumors of the nasal cavity, paranasal sinuses and nasopharynx: A clinicopathologic study. *Cancer* 33:1289, 1974