

Clinical and Histopathological Study of Fibro-osseous Lesions of the Jaws

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Seventy-two cases of benign fibro-osseous lesions of the jaws were reassessed, and a modified classification is proposed that may be helpful in their understanding and management. The anatomical distribution of these cases is described and compared to previously reported cases. The histogenesis of each particular lesion is also discussed. Correlative analysis of histopathologic and oral radiographic features is essential for definitive diagnosis.

Key Words: Fibro-osseous lesion, jaws, classification, histopathology, pathogenesis.

Benign fibro-osseous lesions of the jaws are described as the replacement of normal bone by tissue composed of collagen fibers and fibroblasts and containing varying amounts of a mineralized substance that may be bony or cementum-like in appearance (Waldron 1985).

Menzel (1872) has been credited with the first description of an osteofibroma thought to be an ossifying fibroma among the fibro-osseous lesions, whereas Montgomery (1972) first specified the term "ossifying fibroma". Subsequently, these lesions were classified somewhat arbitrarily, and a bewildering array of names such as ossifying fibroma, fibro-osteoma, osteofibroma or osteoma, have been applied to these lesions based on the relative amounts and character of bone and stroma observed in biopsy material (Harrison 1984). The term "fibrous dysplasia" of bone was first suggested by Lichtenstein (1938) and subsequently, this diagnosis became popular and almost all-inclusive for all fibro-osseous lesions of the jaws (Waldron 1970). However, Reed (1963) offered more rigid histologic criteria for the diagnosis of fibrous dysplasia, stating that this lesion represented an arrest of the maturation process at the woven-bone stage and that lamellar bone should be absent in an

otherwise uncomplicated case.

The fibrous connective tissue of the periodontal ligament contains mesenchymal blastic cells with the potential to form cementum, alveolar bone and fibrous tissue. Under pathologic conditions, such blastic cells are capable of producing tumors composed of either cementum, lamellar bone, fibrous tissue, or any combination of the three (Hamner *et al.* 1968). In particular, lesions composed only of cementum were designated by the broad diagnostic term "cementoma". From an etiologic standpoint, some of the cementum-forming lesions are possibly dysplastic, some may be reactive, and others are seemingly benign neoplasms (Waldron 1985).

Cooke (1957) subclassified fibro-osseous lesions as developmental, neoplastic, dysplastic and inflammatory, and Waldron and Giansanti (1973 a, b), Edwards and Corio (1984), and Waldron (1985) subclassified them as medullary or periodontal ligamentous in origin based on their presumed pathogenesis. However, until now there has been no completely satisfactory or universally accepted classification. Proper categorization of the fibro-osseous lesions of the jaws requires good correlative analysis of clinical, radiographic and microscopic features (Waldron 1985).

The aim of the present study is to classify these lesions based on their pathogenesis and analyze their clinical, radiographic and histologic characteristics.

MATERIALS AND METHODS

All benign fibro-osseous lesions of the jaws, ex-

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cluding peripheral lesions, accessioned at the Departments of Pathology of Yonsei Medical Center, Yong-Dong Severance Hospital (since April 1983) and Wonju Christian Hospital (over the ten-year period from 1977 to 1986) were reviewed. Clinical histories, radiographs, and the pathological material were studied. Individual sections were stained with hematoxylin and eosin. Special stains, including Masson's trichrome, silver impregnation for reticulin,

and periodic-acid-schiff (PAS) reactions, were also examined.

RESULTS

Modified Classification and Distribution

A modified classification of fibro-osseous lesions of the jaws was proposed according to the reports of Cooke (1957), Waldron and Giansanti (1973 a,b), Edwards and Corio (1984), and Waldron (1985). In this study, these lesions were classified on the basis of their origin (medullary or periodontal ligamentous), and each was further subdivided according to its neoplastic or non-neoplastic nature. Neoplastic lesions of periodontal ligament origin were divided into cementifying fibroma, cementoblastoma, gigantiform cementoma, cemento-ossifying fibroma and ossifying fibroma, except for periapical cemental dysplasia. Whereas neoplastic lesions of medullary bone origin were divided into osteoma and osteoblastoma, non-neoplastic lesions were divided into chronic sclerosing osteomyelitis and fibrous dysplasia (Table 1). Among the 72 cases of fibro-osseous lesions of the jaws, 51 (70.8%) were of medullary bone origin and 21 (29.2%) were of periodontal ligament origin. The most frequent lesion of medullary bone origin was fibrous dysplasia (31 cases, 43.1%), followed by 15 cases (20.8%) of osteoma, 4 cases (5.6%) of chronic sclerosing osteomyelitis, and 1 case (1.4%) of osteoblastoma. The most common type of lesion of periodontal ligament origin was ossifying fibroma (9 cases, 12.5%), followed by 4 cases of cemento-ossifying fibroma, 3 cases each (4.2%) of gigantiform

Table 1. Classification and distribution of fibro-osseous lesions of the jaws*

Type of lesion	No. of cases(%)
I. Periodontal ligament origin	21 (29.2)
a. Neoplastic	21 (29.2)
Cementifying fibroma	3 (4.2)
Benign cementoblastoma	2 (2.8)
Gigantiform cementoma	3 (4.2)
Cemento-ossifying fibroma	4 (5.6)
Ossifying fibroma	9 (12.5)
b. Non-neoplastic	0 (0)
Periapical cemental dysplasia	0 (0)
II. Medullary bone origin	51 (70.8)
a. Neoplastic	16 (22.2)
Osteoma	15 (20.8)
Osteoblastoma	1 (1.4)
b. Non-neoplastic	35 (48.6)
Chronic sclerosing osteomyelitis	4 (5.6)
Fibrous dysplasia	31 (43.1)
Total	72 (100)

*Proposed modified classification

Table 2. Age distribution

Age range(yr)	Origin	Periodontal ligament					Medullary bone				Total (%)
	Type	CF	CB	GC	COF	OF	OS	OB	CSO	FD	
0-9		1		1			1				2 (2.8)
10-19			2		2	3	2	1	1	12	24 (33.3)
20-29						3	2		1	12	18 (25.0)
30-39		1		2	1		2		1	3	10 (13.9)
40-49		1				2	3			2	8 (11.1)
50-59							3		1		4 (5.6)
60-69					1	1	1			2	5 (6.9)
70-79							1				1 (1.4)

CF: Cementifying fibroma, CB: Cementoblastoma, GC: Gigantiform cementoma,

COF: Cemento-ossifying fibroma, OF: Ossifying fibroma, OS: Osteoma,

OB: Osteoblastoma, CSO: Chronic sclerosing osteomyelitis,

FD: Fibrous dysplasia

Table 3. Sex distribution

Sex	Origin	Periodontal ligament					Medullary bone				Total (%)
	Type	CF	CB	GC	COF	OF	OS	OB	CSO	FD	
Male		1	1	1	2	1	6	1	2	12	27(37.5)
Female		2	1	2	2	8	9	0	2	19	45(62.5)

Table 4. Location

Location	Origin	Periodontal ligament					Medullary bone				Total
	Type	CF	CB	GC	COF	OF	OS	OB	CSO	FD	
Ant Max						2	3			4	9
Post Max					1	2	1		1	20	28
Ant Man					1	1	1				3
Post Man		3	2		2	4	7	1	3	3	25
Multiple Qu				3						4	7

Ant: Anterior, Max: Maxilla, Post: Posterior, Man: Mandible, Qu: Quadrant

Table 5. Chief complaints

Findings	Origin	Periodontal ligament					Medullary bone				Total (%)
	Type	CF	CB	GC	COF	OF	OS	OB	CSO	FD	
Facial disfigurement				3						4	7 (9.7)
Swelling		1	2		2	8	11			19	43 (59.7)
Swelling with pain		2			2		3	1	2	4	14 (19.4)
Pain									2	2	4 (5.6)
Nasal obstruction						1				1	2 (2.8)
Routine check							1			1	2 (2.8)

cementoma and cementifying fibroma and 2 cases (2.8%) of cementoblastoma (Table 1).

Age and Sex Distribution

The age of onset was most frequently during the second and third decades of life, accounting for 58.3% of the sample. In particular, 24 of 31 cases of fibrous dysplasia occurred during these decades (Table 2).

There was a greater prevalence in females (45 cases) than in males (27 cases) (Table 3).

Location

Lesions originating from the periodontal ligament occurred most frequently in the posterior mandible, accounting for 11 of 21 cases. The majority of lesions originating from medullary bone occurred in the

posterior maxilla. Three cases of gigantiform cementoma and four cases of fibrous dysplasia showed multifocal involvement of the jaws (Table 4).

Chief Complaints and Duration

Swelling with facial disfigurement was the most common complaint, noted in 64 patients (Table 5). The duration of illness ranged from several weeks to 20 years (Table 6).

Radiographic Findings

Radiographic findings permitted separation of the lesions into those with well-defined and ill-defined borders. The well-defined group was subdivided into those with radiolucent, ground-glass, targetoid and radiopaque appearance, and the ill-defined group into those showing a radiolucent, ground-glass, mottl-

Table 6. Duration

Years	Origin	Periodontal ligament					Medullary bone				Total (%)
	Type	CF	CB	GC	COF	OF	OS	OB	CSO	FD	
0-1			1	3	2	1	6	1	2	9	
2-5		3	1		1	3	1		1	7	
6-10						2	4			6	
10-20						1	1			6	
Unknown					1	2	3		1	3	

Table 7. Radiographic findings

Findings	Origin	Periodontal ligament					Medullary bone				
	Type	CF	CB	GC	COF	OF	OS	OB	CSO	FD	
Well-defined	Radiolucent					2					
	Ground-glass					1	2				
	Targetoid	2	2		2	1		1			
	Radiopaque				1		8				
Ill-defined	Radiolucent										1
	Ground-glass										13
	Mottled								4		2
	Diffuse sclerosis			3							3
	Unchecked	1			1	5	5				12

ed and diffusely sclerotic appearance. Radiographic studies showed a well-defined appearance in all lesions originating from the periodontal ligament except gigantiform cementoma. Non-neoplastic lesions originating from medullary bone were ill-defined, whereas neoplastic lesions were well-delineated (Table 7).

Histopathologic Findings

Periodontal ligament origin: Assessment of histologic features was undertaken by H-E, Masson's trichrome, reticulin and PAS stains. With the use of special stains, the mineralized tissue and the stromal elements were evaluated on the basis of the following histological parameters.

Lamellar bone was designated as compact or trabecular, showing parallel lamellations in H-E, trichrome and reticulin stains. Immature woven bone was designated as an irregular osteoid periphery blending into the fibrous stroma in trichrome stains and irregular osseous trabeculae characterized by feathered margins in reticulin stains. Cementum was defined as oval or elliptically shaped laminated struc-

tures that were deeply stained by PAS; dystrophic calcification was characterized by an irregular basophilic calcified structure that did not seem bony or cementum-like in appearance. Variations in stromal elements were tabulated to record hyalinization, myxoid change and associated inflammatory reactions.

Immature woven bone was seen in one case of cemento-ossifying fibroma and 2 cases of ossifying fibroma, but not in cementum-forming lesions. Only one case of gigantiform cementoma showed myxoid change. Inflammatory reaction was usually associated with cementum-forming lesions (Table 8, Figs. 1,2,3 and 4).

Medullary bone origin: Histologic features were assessed for the presence of lamellae or immature woven bone, dystrophic calcification, osteoid formation and osteoblast rimming. Lamellar bone but not immature woven bone was observed in osteoma, osteoblastoma, and chronic sclerosing osteomyelitis. In fibrous dysplasia, lamellar bone formation was observed in 17 cases, and immature woven bone in all but 2 cases. Dystrophic calcification was seen in 19 of 31 cases and was more prevalent in the

Table 8. Histologic characteristics of periodontal ligament origin

	Cementifying fibroma	Cemento-blastoma	Gigantiform cementoma	Cemento-ossifying fibroma	Ossifying fibroma
Mineral tissue					
Lamellar bone	0	1/2	2/3	4/4	8/9
Immature woven bone	0	0	0	1/4	2/9
Cementum	3/3	2/2	3/3	4/4	0
Dystrophic calcification	1/3	1/2	1/3	3/4	7/9
Stroma					
Hyalinization	0	0	0	0	3/9
Myxoid change	0	0	1/3	0	0
Inflammation	2/3	1/2	3/3	2/4	0

0/0: Involved case/Total case

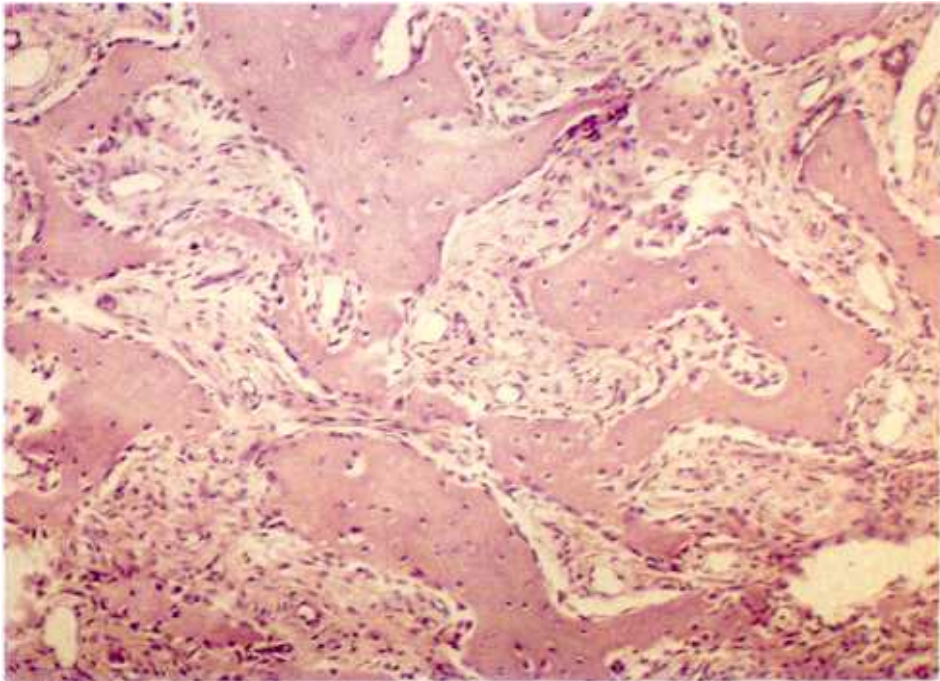


Fig. 1. Ossifying fibroma showing bony trabeculae with osteoblast rimming (H-E, $\times 100$).

craniofacial type than in the monostotic type. Osteoblast rimming was observed in all lesions originating from medullary bone except polyostotic fibrous dysplasia. Osteoblastoma and chronic sclerosing osteomyelitis revealed osteoid formation, but fibrous dysplasia and osteoma did not (Figs. 5 and 6).

DISCUSSION

Fibro-osseous lesions of the jaws comprise a controversial group of pathologic conditions that causes difficulty in classification, pathogenesis and treatment.

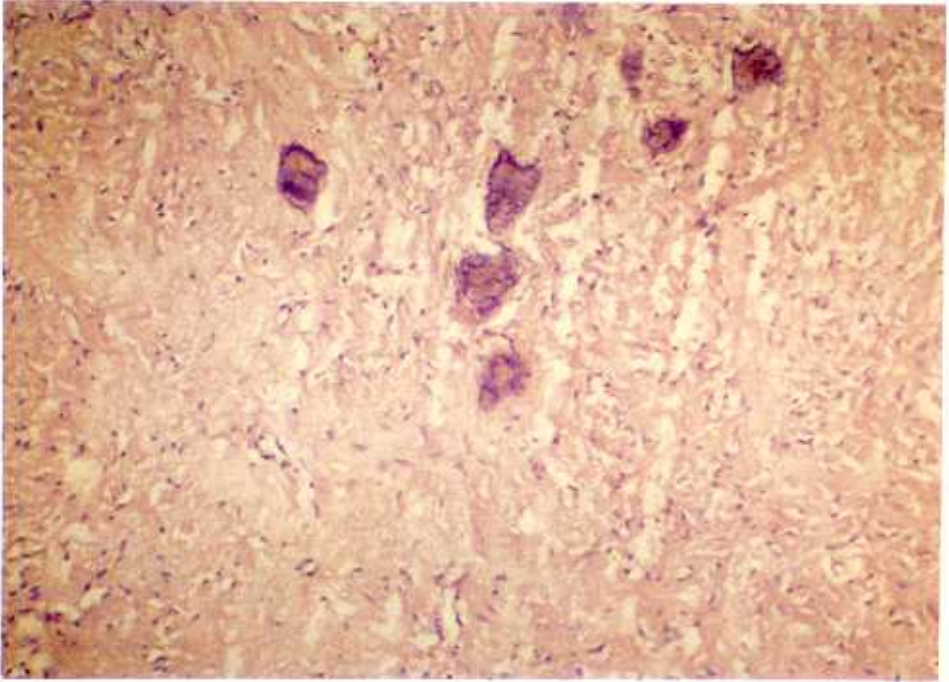


Fig. 2. Ossifying fibroma showing hyalinized stroma with dystrophic calcifications (H-E, $\times 100$).



Fig. 3. Giantiform cementoma showing acellular cemental mass (H-E, $\times 100$).

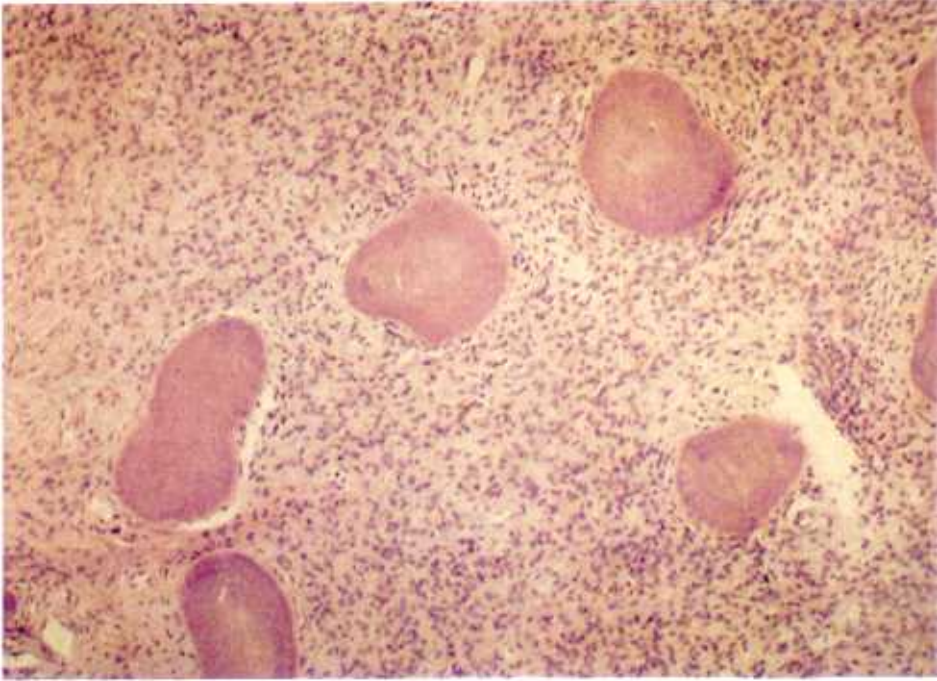


Fig. 4. Cementifying fibroma showing cementicles in the fibrous stroma (H-E, $\times 100$).

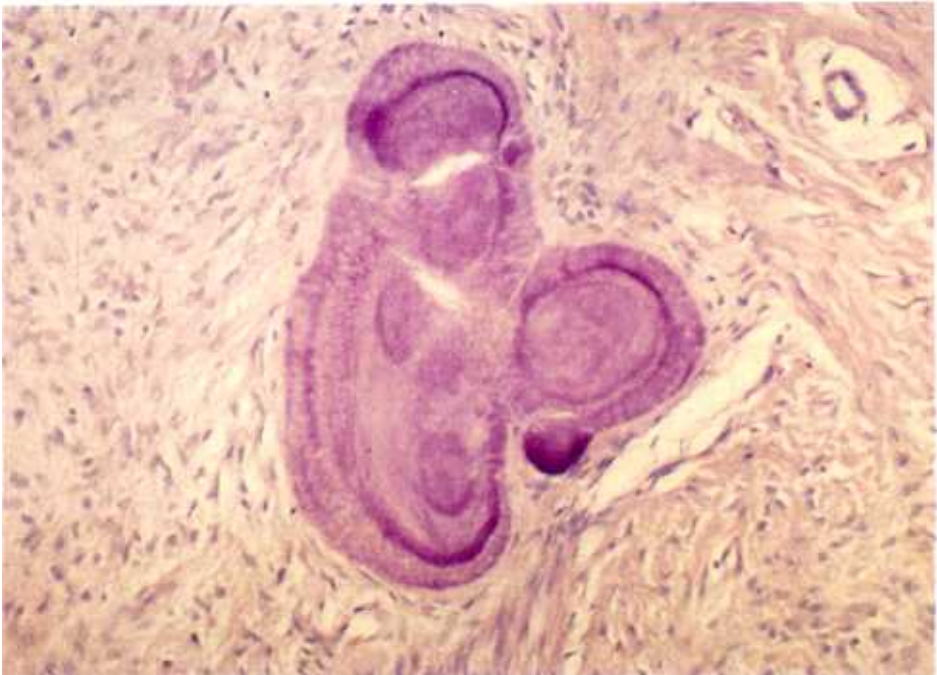


Fig. 5. Cementum showing concentric lamellation pattern (PAS, $\times 200$).

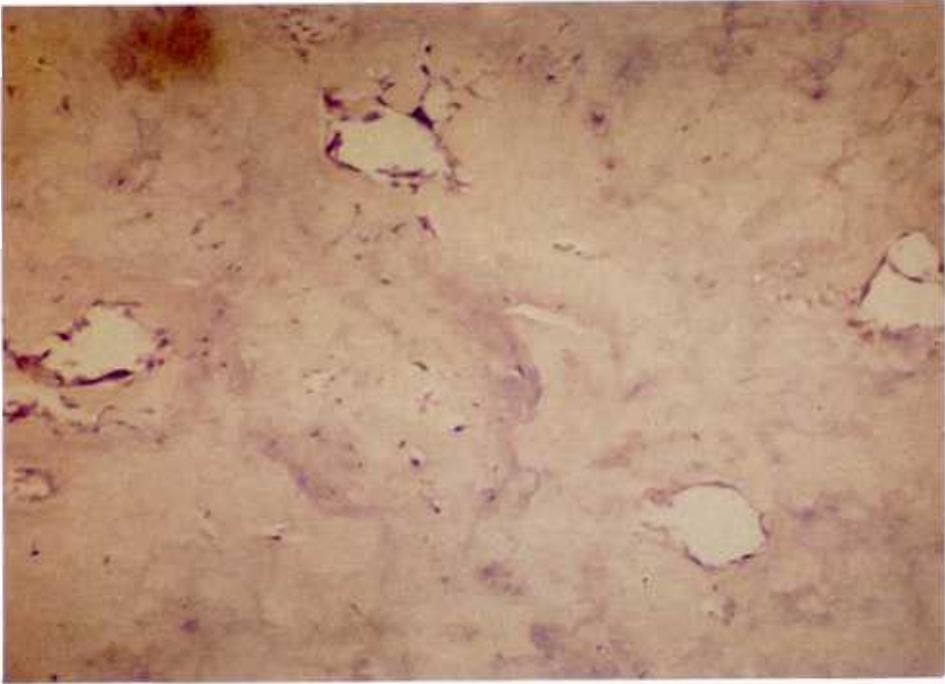


Fig. 6. Cementoblastoma showing a solid mass containing multiple basophilic reversal lines and cementoblasts (H-E, $\times 100$)

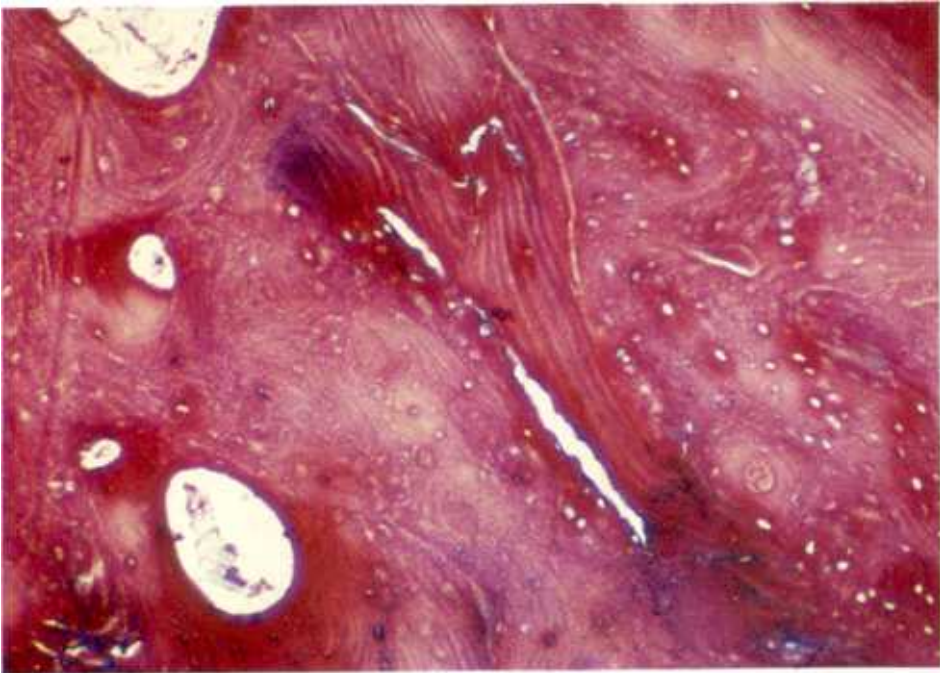


Fig. 7. Osteoma showing mature cortical bone (Masson's trichrome, $\times 200$).

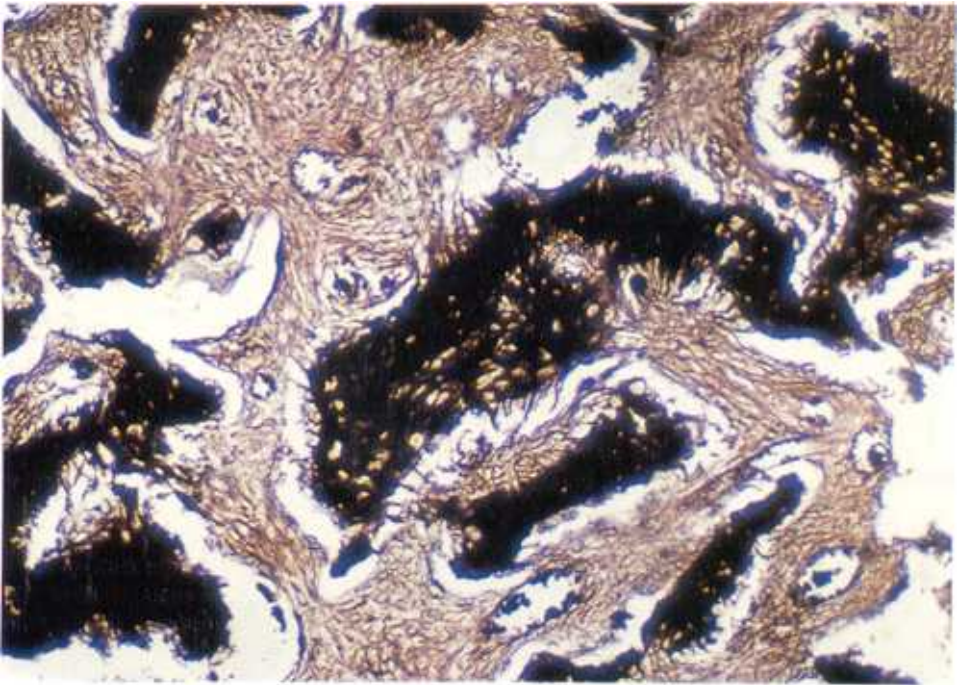


Fig. 8. Fibrous dysplasia showing immature bone with feathery margins (Reticulin, $\times 100$).

Table 9. Histologic characteristics of medullary bone origin

	Osteoma	Osteoblastoma	Chronic Sclerosing osteomyelitis	Fibrous dysplasia		
				Monostotic	Craniofacial	Polyostotic
Lamellar bone	15/15	1/1	4/4	11/21	5/9	1/1
Immature woven bone	0	0	0	20/21	8/9	1/1
Dystrophic calcification	1/15	0	1/4	10/21	8/9	1/1
Osteoid formation	0	1/1	4/4	0	0	0
Osteoblast rimming	2/15	1/1	4/4	6/21	3/9	0

Cooke (1957) classified these lesions as developmental, neoplastic, dystrophic and inflammatory; Hamner *et al.* (1968) divided lesions originating in the periodontal ligament into cementoid, osteoid, cemento-osteoid and fibroid lesions, and Waldron and Giansanti (1973 a, b), and Edwards and Corio (1984) classified them as being of periodontal ligamentous and medullary origin. However, the former authors did not describe lesions of medullary bone origin except for fibrous dysplasia, and the latter stated that chronic sclerosing osteomyelitis was of periodontal ligament origin.

Benign fibro-osseous lesions have been classified as being of periodontal ligament or of medullary bone

origin, and as being either neoplastic or non-neoplastic in character. This might be a clinically acceptable classification on the basis of the reports of Cooke (1957) and Waldron (1985). Consideration of the histogenesis of fibro-osseous lesions, particularly those occurring in the jaw, could theoretically permit distinction between those of periodontal ligament origin and those of medullary bone origin. In 1958, Fullmer and Lillie discovered a previously undescribed connective tissue fiber, the oxytalan fiber of the human periodontal ligament and gingiva, and Hamner *et al.* (1966) suggested that these fibers could serve as a marker for lesions arising from the periodontal ligament.

However, they did not prove that the presence of ox-talan fibers would permit one to distinguish conclusively between lesions of odontogenic and non-odontogenic origin. Although classifying cementum-forming lesions as being of periodontal ligamentous origin, all of the authors controverted the problem of ossifying fibroma composed of pure bone tissues. Since an ossifying fibroma of the jaws frequently develops in the vicinity of constricted teeth, it is reasonable to accept such a lesion as being of periodontal ligament origin. We suggest that ossifying fibromas in other areas originate from undifferentiated mesenchymal periosteal cells.

Since first described by Norberg in 1930, gigantiform cementoma has shown an autosomal dominant pattern of heredity, and the condition has often been referred to as familial multiple cementoma. Though it is thought to be a neoplastic lesion, there is controversy concerning its pathogenesis. Some authors describe gigantiform cementoma as a reactive process that includes sclerotic cemental masses of the jaws (Waldron *et al.* 1975), florid-osseous dysplasia (Melrose *et al.* 1976) or florid cemento-osseous dysplasia (Waldron 1985); whereas others consider it to be dysplastic, developmental or neoplastic in character. Three examples in this study exhibited a familial tendency and were considered as neoplastic rather than reactive lesions in accordance with the views of Remagen and Uehlinger (1986) and Winer *et al.* (1972), because there had been relapses with continuous growth, a pattern of behavior different from the sclerotic cemental masses of the jaws and florid osseous dysplasia as described by Waldron *et al.* (1975) or Melrose *et al.* (1976).

In the fibro-osseous lesion, histopathologically, characterization of hard tissue is important to the diagnosis, and many attempts have been made to distinguish lamellar bone from woven bone and bone tissue from cementum. The use of polarized light to observe the direction or width of the collagen fiber in calcified structures has been widely used (Giansanti 1970 a,b; Hamner *et al.* 1968), as have histochemical methods (Eversole *et al.* 1972). In this study, we attempted to distinguish between the elements of hard tissue with the aid of Masson's trichrome, reticulin and PAS stains.

The lamellated structure of mature bone was more readily seen in trichrome and reticulin stains than in H-E stain. Whereas woven bone appeared as a feathered margin around bone trabeculae in reticulin stains, it seemed that the margin of bone trabeculae fused with the surrounding stromal tissues in

trichrome stains, thus suggesting that one could distinguish mature from woven bone. Woven bone was never observed in cementum-forming lesions but helpful in distinguishing between the two lesions, woven bone was present in fibrous dysplasia more frequently than in any other lesion. The lamellated structure seen in H-E, trichrome and reticulin stains represents a morphologic characteristic of woven bone. It distinguishes cementum from bone trabeculae because cementum is observed morphologically as an oval or elliptical calcified structure, but it is sometimes difficult to distinguish cementum-forming lesions because rounded dystrophic calcifications such as those seen in cementum can be formed in ossifying fibroma or fibrous dysplasia. In this study, we distinguished cementum from other calcified structures with relative ease because cementum was deeply stained by the PAS stain. Probably that was due to the presence of large amounts of mucopolysaccharides in the cementum. An inflammatory reaction was seen in 8 of 12 cases of cementum-forming lesions. The reason for the associated inflammatory reaction in a cementum-forming lesion is not clear but it is assumed to be related to the minute vascular distribution in the stroma. Dystrophic calcification similar to that seen in cementum was observed in the majority of fibro-osseous lesions but was more remarkable in ossifying fibroma and fibrous dysplasia. In particular, dystrophic calcification was observed in 3 cases of ossifying fibroma which showed stromal hyalinization, a finding that is at variance with those of Eversole *et al.* (1985), who claimed that dystrophic calcification appeared in the fibrous cellular stroma.

Among fibro-osseous lesions, the causes of fibrous dysplasia of the jaws are not well-known, but the possibilities include hamartoma, developmental arrest, endocrine disturbances, localized infection, reparative reaction against trauma, and hereditary factors. Harris *et al.* (1968) and Reed (1963) considered fibrous dysplasia to be the result of developmental arrest. But among the lesions found in craniofacial bone, osseous components are noted more frequently than in any other type of lesion. Some authors report that lamellar bone or osteoblast rimming is frequently observed in fibrous dysplasia of the jaws (Eversole *et al.* 1972, Harris *et al.* 1962, Waldron 1970). In this study, we observed osteoblast rimming in 9 cases and lamellar bone in 17 of 31 cases of fibrous dysplasia. These results are similar to those of Waldron (1970) and Harris *et al.* (1972).

In the case of fibrous dysplasia in craniofacial bone,

the frequency with which one encounters lamellar bone and osteoblast rimming is higher than in other areas, and it is thus difficult to differentiate between fibrous dysplasia and ossifying fibroma on the basis of histology alone. Waldron and Giansanti (1973 a) regarded it as appropriate to make a diagnosis of fibrous dysplasia when it corresponds with the clinical and radiographic findings of that condition, even if there is some lamellar bone or osteoblast rimming. In this study, osteoid formation was not observed in fibrous dysplasia, apart from bone-forming tumors.

In conclusion of this study, we suggest that classification based upon origin and pathogenesis, as illustrated in this study, is helpful in the understanding of fibro-osseous jaw lesions, as well as in the diagnosis and choice of treatment, and that analysis of histopathologic characteristics must be made in reference to clinical and radiologic findings.

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