INTERNATIONAL JOURNAL OF MEDICAL AND APPLIED SCIENCES
Earthjournals Publisher
E-ISSN:2320-3137
www.earthjournals.org

CASE REPORT

FIBROUS DYSPLASIA- REVIEW OF LITERATURE

Basetty Neelakantam Rajarathnam, Maria Priscilla David, Narayan Naveen Kumar

1. Dr. Syamala Reddy Dental College, Hospital & Research Centre, 111/1, SGR Main Road, Munnekolala, Marathalli, Bengaluru-37, Karnataka, India
2. M R Ambedkar Dental College & Hospital, 1/36 Cline road, Cooke Town, Bangalore-05, Karnataka, India.
3. Vydehi Institute of Dental Sciences and Research Centre, #82, EPIP area, Whitefield, Bangalore-66

Corresponding Author: Dr Basetty Neelakantam Rajarathnam,M.D.S. Senior Lecturer,DEPARTMENT: Oral Medicine & Radiology, Dr. Syamala Reddy Dental College, Hospital & Research Centre, 111/1, SGR Main Road, Munnekolala, Marathalli, Bengaluru-37, Karnataka, India.

Abstract
Fibro-osseous lesions of the maxillofacial bones comprise a diverse group of pathologic conditions that include developmental lesions, reactive or dysplastic diseases, and neoplasms. Benign fibro-osseous lesions are disturbances in bone metabolism where normal bone is replaced by a connective tissue matrix that gradually develops cemento-osseous tissue. Frequently most of the fibro-osseous lesions are diagnosed incidentally due to its asymptomatic nature. Here we report a case of fibrous dysplasia and periapical cemental dysplasia which was diagnosed after performing a detailed examination.
Keywords: Fibro-osseous Lesion, Fibrous Dysplasia.

INTRODUCTION
Fibro-osseous lesions of the maxillofacial bones are benign proliferations of spindle cells with varying amounts of woven bone. Many specific entities have been proposed based on histologic and radiographic features. However, because there is considerable overlap of histologic features in these lesions, the confusing nomenclature can be simplified by defining lesions based on their radiographic presentation. These neoplasms can all fit into one of three categories: (a) fibrous dysplasia; (b) ossifying fibroma; (c) and osseous dysplasia. These entities all have over- lapping histologic features and are defined only by their growth pattern as apparent on plain radiographs or CT scans of the head and face.[1]
Here we report a case of fibro-osseous lesion which was diagnosed incidentally after thorough clinical evaluation and imaging and its literature review.

CASE 1: A 52 year old male patient named Muniraj Singh (Fig 1a) visited to the department of Oral Medicine & Radiology, M.R Ambedkar Dental College & Hospital, Bengaluru, with chief complaint of pain in upper right back tooth since 15 days, which is dull and intermittent in nature. It aggravates on eating hard food substances and relieved on taking antibiotics and

analgesics. No significant medical, dental and habit history. On general physical examination, patient was well oriented, cooperative and no other abnormalities found. Bilateral submandibular lymph nodes are palpable, single, soft to firm in consistency, freely movable and tender. On intraoral examination, Presence of deep dental caries in relation to 14,15 and the teeth were tender on percussion and also, Presence of expanded (fusiform) edentulous alveolar ridge buuco-lingually from 33 to 38, (Fig 1b) the surface of the expansion appear normal. On palpation presence uniform expansion of buccal and lingual cortex of the complete third quadrant. Considering the signs and symptoms, chronic periapical abscess in relation to 14, 15 was made and fibro-osseous lesion was provisionally considered in relation to third quadrant. Mandibular lateral occlusal radiograph (Fig 1c) taken in relation to third quadrant. It revealed fusiform expansion of buccal and lingual cortical bone also presence of finger print pattern of trabecular bone in the anterior region of edentulous ridge of 31, 32, 41, 42.Orthopantomogram (Fig 1d) revealed diffuse radiopacity extending antero-posteriorly of edentulous ridge from 41-38. Supero-inferiorly from alveolar crest till inferior border of mandible. Presence of a tiny radiopacity surrounded by radiolucent rim above the mandibular canal on the left side. Presence of ill defined radiolucency in the periapical region of 14, 15. On basis of clinical and radiological evaluation monostotic fibrous dysplasia of mandible was made.

1.

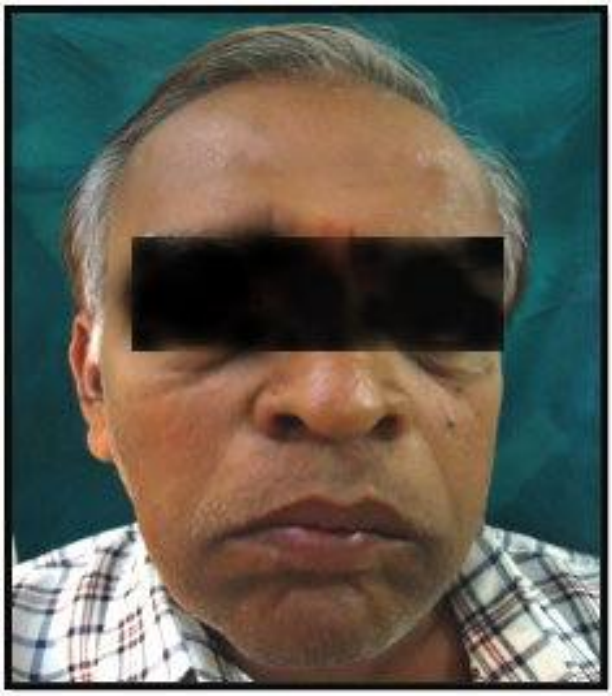

FIG 1A : FRONTAL VIEW OF PATIENT

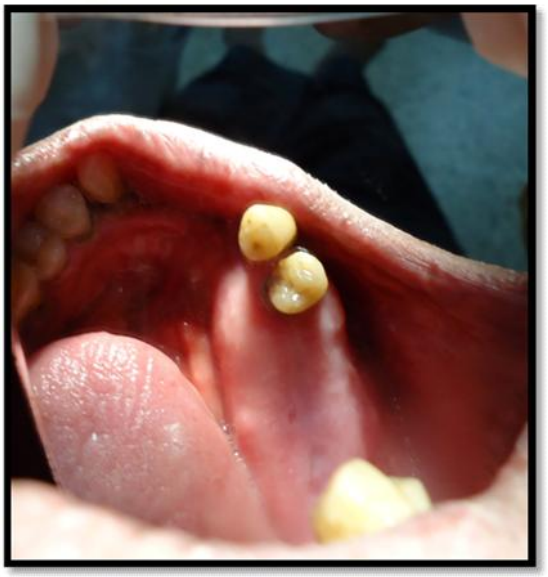

FIG 1B: INTRAORAL VIEW SHOWING FUSIFORM EXPANSION OF THIRD QUADRANT

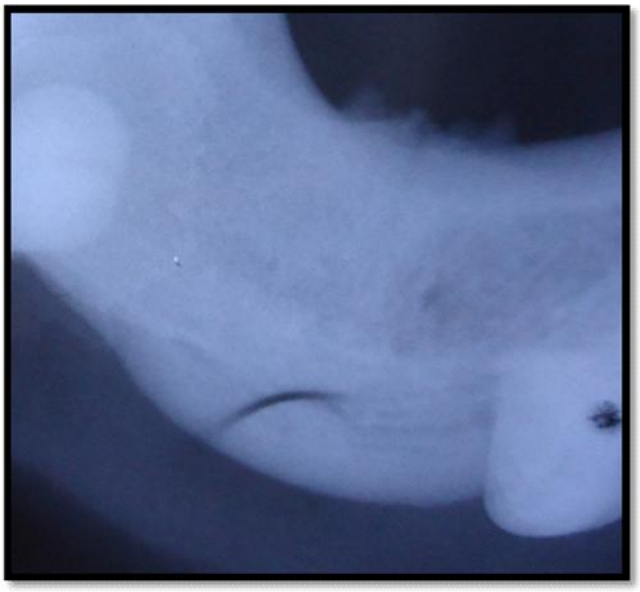

FIG 1C: OCCLUSAL RADIOGRAPH, ANTERIORLY SHOWING FINGERPRINT PATTERN.

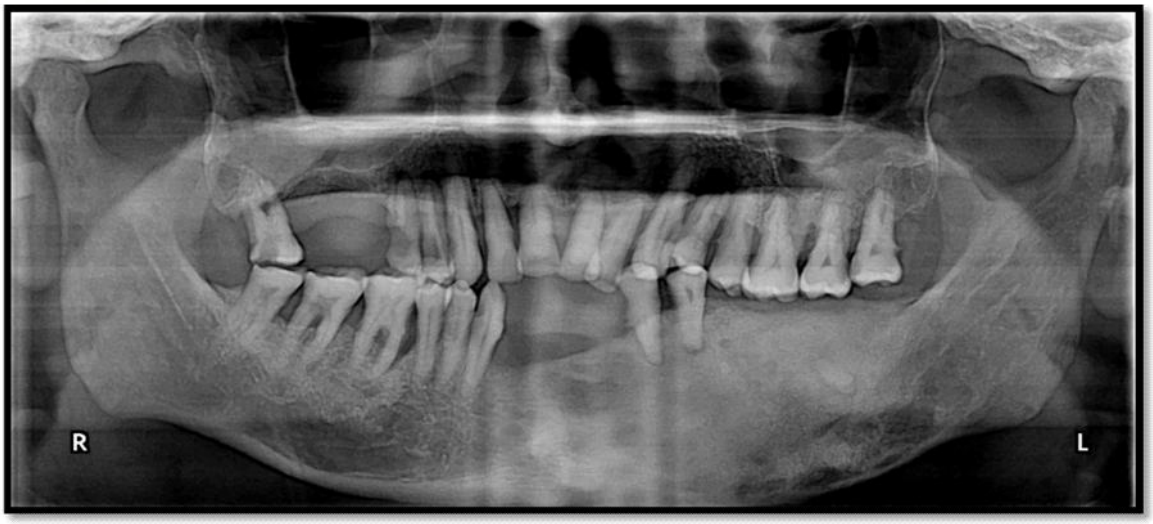

FIG 1D: ORTHOPANTOGRAM SHOWING THE DIFFUSE RADIOLUCENCY IN THIRD QUADRANT

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[a]
- 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
- d. Gigantiform cementomas

[a] Osteofibrous dysplasia is found in the fibula and tibia only

Table 2 Microscopic similarities and dissimilarities among fibro- osseous lesions

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’’)

DISCUSSION:

By definition, all Benign Fibro Osseous Lesions (BFOL) possess an osseous and a fibrous tissue component. Prior to reviewing the features of each entity subsumed under the heading of BFOL of the craniofacial complex, a nosology of these lesions is given in Table 1 & 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.[2]

Fibrous Dysplasia

The original term FD was introduced by Liechtenstein in 1938.[3]Fibrous dysplasia is a developmental or growth disorder in which normal bone is replaced by abnormal fibrous tissue that contains small, abnormally arranged bone trabeculae. It is considered by some authors to be a hamartomatous malformation that presumably results from an idiopathic arrest in maturation of bone at the woven bone stage.[4] Fibrous dysplasia is a benign dysplastic process of altered osteogenesis that may occur within a single bone (monostotic) or multiple bones (polyostotic). When polyostotic fibro-osseous lesions typical for fibrous dysplasia are associated with other anomalies and endocrinopathy, this variant form constitutes the McCune- Albright syndrome (MAS). The molecular underpinning of this related group of diseases is a mutation in the gene that encodes the G protein alpha-subunit (Gs-alpha) that couples cAMP to hormone receptors.[2]If the mutation occurs in one of the undifferentiated stem cells during early embryologic life, the osteoblasts, melanocytes, and endocrine cells that represent the progeny of that mutated cell all will carry that mutation and express the mutated gene. The clinical presentation of multiple bone lesions. cutaneous pigmentation, and endocrine disturbances would result. Skeletal progenitor cells at later stages of embryonic development are assumed to migrate and differentiate as part of

the process of normal skeletal formation. If the mutation occurs during this later period, the progeny of the mutated cell will disperse and participate in the formation of the skeleton resulting in multiple bone lesions of fibrous dysplasia. Finally, if the mutation occurs during postnatal life, the progeny of that mutated cell are essentiaily confined to one site, resulting in fibrous dysplasia affecting a single bone.[5]

These mutations result in GTPase perturbations that lead to prolonged Gs- alpha activation and stimulation of endocrine receptors. The lesions of fibrous dysplasia show elevated intracellular cAMP in bone marrow osteoprogenitor cells and these molecular changes probably initiate cell proliferation with differentiation defects. Gs-alpha mutation is also seen in 40% of pituitary tumors causing acromegaly. In fibrous dysplasia, these genetic lesions are postzygotic, somatic and result in cellular mosaicism, a genetic process that accounts for the fact the not all bones in polyostotic disease are affected; the normal appearing bone in affected individuals is devoid of genotypic lesions. Albright's hereditary osteodystrophy (psuedohypoparathyroidsm) also involves a debilitating mutation in the Gs-alpha gene with a wide range of skeletal anomalies, yet fibro-osseous lesions are not extant.[2]

Monostotic fibrous dysplasia of the jaws, when the disease is limited to a single bone, it is termed monostotic fibrous dysplasia. This type accounts for about 80% to 85% of all cases, with the jaws being among the most commonly affected sites.[5] Most examples of monostotic fibrous dysplasia are diagnosed during the second decade of life. Males and females are affected with about equal frequency. A painless swelling of the affected area is the most common feature. Growth is generally slow, and the patient or parents are often unable to recall when the lesion was noted first. Occasionally, however, the growth may be fairly rapid. The maxilla is involved more often than the mandible.[5]In a systematic review of previous studies of FD McDonald and Jankowski determined that the most common presenting complaints was swelling in 94% of cases and pain in 15%.[6]

Monostotic fibrous dysplasia occurring in frontal, ethmoidal, temporal and calavarial bones are also reported.[2]Teeth involved in the lesion usually remain firm but may be displaced by the bony mass.[5]Monostotic fibrous dysplasia of the craniofacial complex is often confused with other BFOL, typically ossifying fibroma and diffuse sclerosing osteomyelitis of the mandible, diseases that manifest unique clinicoradiologic features. Radiographic features vary depending upon the stage of the disease and have been sub-classified into three different patterns: pagetoid type 56%, sclerotic type 23%, and the radiolucent type 21%. Both the patients had sclerotic lesions, the commonest form of involvement seen in facial bones and bones of the base of the skull. The lytic and pagetoid types usually involve the calvarial bones.[4]

Early onset lesions are radiolucent and later progressively calcify, culminating in a ''ground glass'' or mottled mixed radiolucent/radiopaque pattern. Critical to the diagnosis is the fact that fibrous dysplasia fails to manifest any discrete margins; rather, the lesional bone subtly blends into the surrounding normal appearing bone.[2] Involvement of the mandible often results not only in expansion of the lingual and buccal plates but also bulging of the lower border. Superior displacement of the inferior alveolar canal is not uncommon. Periapical radiographs of the involved dentition often demonstrate narrowing of the periodontal ligament space with an ill-defined lamina dura that blends with the abnormal bone pattern.When the maxilla is involved the lesional tissue displaces the sinus floor superiorly and commonly obliterates the maxillary sinus.

Fibrous dysplasia.[5]In McDonald-Jankowski's study, the most common radiographic presentation of FD was a poorly defined, ovoid (fusiform) area of dysplastic bone exhibiting a ground glass appearance. Petrikowski and others suggested that upward displacement of the mandibular canal may be unique to FD and could be pathognomonic.[6]

Radionuclide scanning in fibrous dysplasia shows areas of intensely increased uptake that is due to diffuse microscopic ossification. Scintigraphy is helpful in determining the activity and potential multicentricity of the lesion and is specifically helpful in diagnosing when plain radiographs are equivocal.CT accurately establishes the diagnosis and extent of bone involvement. Involvement of optic canals, orbital fissures, frontonasal ducts, and ostiomeatal complex can be best evaluated by CT scanning. CT characteristics of fibrous dysplasia include expansion of the involved bone with heterogenous pattern of CT densities associated with scattered or confluent islands of bone formation. CT attenuation levels have been reported to range from 34 to 513 Hounsfield (HU) depending on the fibrous tissue and bone content. On magnetic resonance imaging (MRI), fibrous dysplasia exhibits homogenous and moderately low signal intensity on T1-weighted images. On T2-weighted images, the tissue usually exhibits very high signal intensity. After intravenous Gd-diethylenetriaminepentaacetate (Gd-DTPA) lesions display moderate to significant central contrast enhancement with some rim enhancement. The degree of contrast enhancement on T1-weighted images depends on the amount and degree of bone trabeculae and collagen present. Both CT and MRI are excellent imaging modalities in defining the constrictive effect of craniofacial fibrous dysplasia on the orbit, optic canals, and adjacent paranasal sinuses.[4]

The differential diagnosis with similar radiographic appearance such as ameloblastoma, ameloblastic fibroma, ameloblastic odontoma, ameloblastic fibro- odontoma, cental giant cell granuloma, odontogenic cyst, ossifying fibroma, osseous dysplasia, chronic sclerosing osteomyelitis and osteosarcoma should be considered.[7]

Histologically a fibro-osseous pattern is typically seen, yet akin to the imaging features, subtle changes are seen at various stages of the disease's natural history. In the early formative phase, pronounced osteogenesis is seen with thin osteoid anastomosing trabeculae that are rimmed with osteoblasts. The stromal fibroblastic element is proliferative and hypercellular although no pleomorphism can be seen. With ensuing weeks, the trabeculae thicken, yet the osseous collagen pattern remains woven and the trabeculae assume the classic ''Chinese figure'' characteristics. The fibrous element continues to be hypercellular. In later stages of the disease woven bone is replaced by lamellar bone trabeculae; extensive remodeling may result in a mosaic pattern of resting and reversal lines (Fig. 4).[2]

Another variant of fibrous dysplasia is osteofibrous dysplasia, a pediatric disease that is typically found in the lower extremity long bones and is a fibro-osseous process in which epithelial nests and islands are found dispersed throughout the lesion. Oftentimes the nests are quite small and are seen only with immunostaining for cytokeratins. These lesions show radiologic and histologic features typical for fibrous dysplasia and many researchers take the position that osteofibrous dysplasia is merely a variant of fibrous dysplasia. Others consider this variant to be an adamantinoma with a reactive fibro-osseous component. Genetic analyses have failed to identify any G(s) alpha mutations in osteofibrous dysplasia. Long term follow up has failed to demonstrate the emergence of classic adamantinoma from these lesions. Indeed, many cases

spontaneously resolve. Osteofibrous dysplasia has not been described in the craniofacial complex although one of the author has encountered a maxillary fibro-osseous lesion with multiple epithelial nests, probably odontogenic in origin, dispersed throughout the fibrous stroma.[2]

Fibrous dysplasia may also be associated with soft tissue myxomas, the Mazabraud syndrome.[2]

Clinical management of fibrous dysplasia of the jaws may present a major problem. Although smaller lesions, particularly in the mandible, may be surgically resected in their entirety without too much difficulty, the diffuse nature and large size of many lesions, particularly those of the maxilla, preclude removal without extensive surgery. In many cases, the disease tends to stabilize and essentially stops enlarging when skeletal maturation is reached. Some lesions. however, continue to grow. although generally slowly. in adult patients.

Some patients with minimal cosmetic or functional deformity may not require or desire surgical treatment. Cosmetic deformity with associated psychologic problems or functional deformity may dictate surgical intervention in the younger patient. Such a procedure usually entails surgical reduction of the lesion to an acceptable contour with out attempt store move the entire lesion. The cosmetic result is usually good, but regrowth of the lesion occurs over time.

The prevalence of regrowth after surgical reduction is difficult to determine, but it has been estimated that between 25% and 50% of patients show some regrowth after surgicalshave-down of the lesion. The regrowth is more common in younger patients, and many surgeons believe that surgical intervention should be delayed for as long as possible.[5]Concentration of serum alkaline phosphatase (ALP) may be important marker for detection of the recurrence of the lesion. The patients who had FD, have higher ALP, this may be a reliable marker for estimating tumor progress and a sudden rise in ALP was correlated with the regrowth of FD by Park et al.[7]

Malignant change usually development of an osteosarcoma, has been rarely associated with fibrous dysplasia. Most examples have been found in patients who had received radiation therapy for fibrous dysplasia. But a few examples of spontaneous sarcomatous changes have been reported. Radiation therapy for fibrous dysplasia is contraindicated because it carries the risk for development of postirradiation bone sarcoma.[5]

The complications of the lesions involving sphenoid, orbital, frontal bones, are proptosis, visual disturbances, facial asymmetry and orbital dystopia. The fifth nerve impairment, hearing loss and seizure disorders have been reported as neurological complications.[7]

Polyostotic Fibrous Dysplasia is the term applied to involvement of multiple bones throughout theskeleton with ribs and long bones most commonly affected along with jaw andcraniofacial region.McCune-Albright syndrome (MAS) is defined as the association of PFD,precocious puberty, cafe-au-lait spots, and other endocrinopathies due to hyperactivity of various endocrine glands. Fuller Albright first described this syndrome in 1937. McCune-Albright syndrome has been shown to be due to a post zygotic activating mutation of the GS alpha gene in the affected tissues. The GS alpha subunit is the component of the G-protein complex, which couples hormone receptors to adenylate cyclase (the intracellular second messenger) in a submembrane site. It then mediates the cellular effects of hormone binding. [8]

There are two apparently separate types of PFD which are –

- Jaffe's type
- Albright's syndrome.[9]

Other endocrine disturbances like hyperthyroidism, cushing's syndrome, hyperparathyroidism, acromegaly, goiter and gynacomastia may also occur. In addition to these, the occasional occurrence of multiple intramuscular soft tissue myomas as extra-skeletal manifestations of PFD has also been noted.[3]

Happle suggested that MAS was due to somatic mosaicism, with the non- mosaic state being lethal (Happle's hypothesis). In somatic mosaicism, mutation occurs in somatic cells postzygotically rather than in germ cell. Depending on the time, number, and type of cells in which the mutation occurs there could be varying severity and extent of disease manifestation. GNAS1mutation similar to that in MAS have been identified in other types of fibrous dysplasia, except in Jaffe-Lichtenstein syndrome.

It was stated that the activating mutations in the gene encoding for α subunit of stimulatory G protein is involved in the pathogenesis of PFD, pituitary adenoma and MAS.[10]

PFD occurs in about 20-30 % cases of FD. The disease usually manifests early in life with an evident deformity of long bones, often unilateral in distribution. It has insidious onset. Recurrent bone pain is the most common presenting skeletal symptoms.[11]

Because of the severe bone changes, spontaneous fractures are a common complication of the disease. The structural integrity of the bone is weakened and the weight bearing areas become bowed. The curvature of the femoral neck and proximal shaft of the femur markedly increase causing a 'shepherd crook deformity', which is a characteristic sign of the disease. Overgrowth of adjacent soft tissues may be present. Two apparently separate types of PFD are described as -

Jaffe's type - FD involving a variable number of bones, accompanied by pigmented lesions of the skin or "cafe-au-lait" spots of thin light brown color. It is mild and non- progressive form. This type occurs in about 50% of the cases.

Albright's syndrome -FD even more severe, involving nearly all bones in the skeleton accompanied by pigmented lesions of the skin in addition to endocrine disturbances of varying types. Female patients exhibit precocious puberty, sometimes beginning the age of 2 or 3 years. Vaginal bleeding is a common manifestation.[9]

Cutaneous pigmentation in PFD is ipsilateral to the side of bony lesions, a feature that differentiates pigmentation of this disease from that in neurofibromatosis. The pigmented macules or cafe-au-lait spots are related to increased amounts of melanin in the basal cells of the epidermis. They are arranged in a linear or segmental pattern near the midline of the body, usually overlying the lower lumber spine, sacrum, buttocks, neck and shoulders. Similar lesions may occur on lips and oral mucosa also.

Some severely affected patients may present with associated hepatic, cardiac and gastrointestinal dysfunction (i.e. elevated hepatic transaminases, gastrointestinal polyposis and cardiomyopathy.[5]

Mazabraud's syndrome is a rare disease caused due to association of FD and intramuscular myxoma that occur in the same anatomical region. Patients with soft tissue myxomas should be thoroughly examined for FD as greater risk of sarcomatous transformation in FD with Mazabraud's syndrome has been reported. Malignant transformation may include

OsteosarcoMcCune Albright Syndrome (most common), ChondrosarcoMcCune Albright Syndrome, FibrosarcoMcCune Albright Syndrome and LiposarcoMcCune Albright Syndrome. These malignancies occur most commonly in the setting of therapeutic irradiation exposure. Females may have a greater risk for breast cancer, probably due to their prolonged exposure to elevated estrogen levels. The underlying GS alpha gene mutation may also play a role in this.[9]

The oral manifestations of PFD are related to severe disturbance of bony tissue. One third of the polyostotic patients have lesions in the mandible. There may be expansion and deformity of the jaws and the eruption pattern of the teeth is disturbed because of loss of support of the developing teeth. The endocrine disturbance also, may alter the time of eruption of the teeth.[9]

The lesions include a radiolucent lesion in the diaphysis or metaphysis with endosteal scalloping. It may be present with or without cortical expansion. The radiolucent lesion has a thick sclerotic border and is called 'rind sign'.[9] In general, the medullary portion of bone is mostly involved and present irregular trabeculations that give multilocular cystic appearance.

Among the skull and facial bones, the frontal bone is most frequently involved and then the sphenoid bone, with obliteration of frontal and sphenoid sinuses. Most commonly, maxillary and mandibular involvement has a mixed, radiolucent and radiopaque pattern with displacement of teeth and distortion of nasal cavities.[12]

Histological Features are similar to that of MFD. The lesion is composed of fibrillar connective tissue within which numerous trabeculae, woven immature bone, irregular in shape, can be seen.[5] The osteocytes are quite large and collagen fibers of these trabeculae can often be observed extending into fibrous tissue. Bone formation by stellate osteoblasts can be seen but rows of cuboidal osteoblasts remaining on the surface of the trabeculae (osteoblastic rimming) are absent. These trabeculae typically have wide osteoid seams. Osteoclastic activity may also be seen in some areas.[9]

Depends on biologic behaviour of the lesion in each patient. Growth of FD is cyclic with greatest change occurring during childhood, onset of puberty or pregnancy. Surgical management include either contour excision or en bloc resection with or without bone grafting. Bisphosphonates are used in cases of symptomatic PFD to decrease bone pain.[13]

Cherubism, familial fibrous dysplasia, is a benign dysplastic bone disease that is limited to the maxilla and mandible and is not included in the classification of fibro- osseous lesions. A brief discussion of this entity and its syndromic associations is discussed here, only because it was at one time considered to be a specific variant of fibrous dysplasia (which it is not). It has its onset in childhood and is inherited as an autosomal dominant trait.[14]The gene for cherubism maps to chromosome 4p16, the site for adaptor protein 3BP2 which positively regulates the high affinity IgE receptor (FcepsilonRI)-mediated activation of degranulation in mast cells.[2]Over-expression of 3BP2 mutant protein inhibits antigen-induced mast cell activation as well as numerous other internal signaling pathway proteins.

It affects males and females equally and the full phenotype affects both the maxilla and mandible with varying degrees of expressivity in that some cases exhibit minimal maxillary involvement. In the classic presentation, the child presents with ''cherubic facies'' characterized by upward gaze due to osseous expansion of the maxilla and orbital floor and bilateral osseous expansion of the posterior mandibular body and ramus. Teeth remain unerupted and imaging studies reveal bilateral multilocular radiolucencies with expanded cortices. Espansile lesions of the temporal

bone have also been reported in Cherubism[2]. Microscopically cherubism is a fibrous proliferation without an osteogenic component. The collagenous network is immature with delicate areolar fibrils. Small vessels within the fibrous component exhibit a characteristic collagenous cuffing (i.e.: perivascular hyalinizing fibrosis) and randomly dispersed throughout are multinucleated giant cells with diffusely dispersed nuclei. Aggressive forms of the disease with more marked expansion occur and result in more severe functional and cosmetic deformity.[15]

Cherubism has occurred in association with three syndromes: Noonan syndrome, Ramon syndrome and type I Neurofibromatosis. The Noonan syndrome is characterized by short stature, cherubic facies, congenital heart defects, chest deformity and mild mental retardation. It may be sporadic or inherited as an autosomal dominant trait and occurs between one in 1000-2500. The Ramon syndrome consists of mental deficiency, epilepsy, cherubism, gingival fibromatosis, hypertrichosis, stunted growth and juvenile rheumatoid arthritis. The rare cases with NF1 association have shown multiple mutations (NF1, SH3BP2, PTPN11).[2]

CONCLUSION

Fibro-Osseous Lesions represent a spectrum of diseases which require a careful examination and detailed case history taking for arriving at a suitable diagnosis. An attentive dentist must be aware of various overlapping features of fibro-osseous lesions, which is important for diagnosing these incidental lesions.

REFERENCES

1. Mc Carthy EF. Fibro-Osseous Lesions of the Maxillofacial Bones. Head and Neck Pathol 2013;7:5–10
2. Eversole R, Su L, ElMofty S. Benign Fibro-Osseous Lesions of the Craniofacial Complex - A Review. Head and Neck Pathol 2008;2:177–202
3. Regezi JA, Sciubba J. Oral pathology: Clinical-pathological correlations 2nd ed. Philadelphia: WB Saunders;1993
4. Yadavalli G. Fibro-osseous Lesions of the Jaw: A Report of Two Cases. J Clin Imaging Sci. 2011;1:10
5. Neville BW, Damm DD, Allen CM, Bouquot JE (Eds). Oral and maxillofacial pathology. Philadelphia: WB Saunders;1995
6. MacDonald-Jankowski D. Fibrous dysplasia: a systematic review. Dentomaxillofacial Radiology 2009;38:196–215
7. Canıtezer G, Gunduz K, Ozden B and Kose HI. Monostotic Fibrous Dysplasia: A Case Report. Dentistry 2012;3:2
8. Robert E. Marx, Diane. Stern. Oral and Maxillofacial Pathology.
9. Shafer Hine. Levy. Shafer's Textbook of Oral Pathology 7th edition
10. Cohen M M, Howell R E. Etiology of fibrous dysplasia and McCune-Albright syndrome Int J of Oral and Maxfac surgery 1999; 28(5): 366-71
11. Lichtenstein L, Jaffe HL.Fibrous Dysplasia of Bone. Arch. Surg. 1938; 36: 777-816
12. Brannon RB, Fowler CB. Benign Fibrous Lesions: A Review of Current Concepts.Advances in Anatomic Pathology. 2001; 8(3): 126-143.
13. Papadaki ME, Troulis MJ, Kaban LB, Advances in diagnosis and Management of Fibro-osseous lesions Oral Maxillofacial Surg clin N Am 2005; 17:415-434
14. Meng XM, Yu SF, Yu GY. Clinicopathologic study of 24 cases of cherubism. Int J Oral Maxillofac Surg. 2005;34:350–6.
15. Wang CN, Song YL, Peng B, Lu DH, Fan MW, Li J, Ye XQ, Fan HL, Bian Z. The aggressive form of cherubism: report of two cases in unrelated families. Br J Oral Maxillofac Surg. 2006;44:322–4.