Fibro-Osseous Lesions of the Jaw: A Review Article

Dr. Anchal Rai¹*, Dr. Nitin Agarwal², Dr. Payal Tripathi³, Dr. Vasu S. Saxena⁴, Dr. Anas⁵, Dr. Braj Mohan Mishra⁶

¹PG Student, ²Prof and HOD, ³Professor, ⁴Associate Professor, ⁵⁶PG Student, Dept. of Omdr Career Postgraduate Institute of Dental Sciences and Hospital Lucknow, U.P, India

Abstract: Fibro-osseous lesions are group of lesions that are poorly defined and affect the jaws and craniofacial bones. It is characterized by the replacement of normal bone by fibrous connective tissue containing foci of mineralization that vary in amount and appearance. Fibro osseous lesion is benign and arising process. It includes diverse group of pathologic conditions that are developmental lesions, reactive or dysplastic diseases, and neoplasms. Therefore, classification and diagnosis of these lesions is controversial because there is significant overlap of clinical and histological features. Hereby we present the detailed review of different classification and description of fibro-osseous lesions.

Keywords: Benign, fibro osseous, hamartoma, osteoid, florid cemento-osseous dysplasia, ossifying fibroma, monostotic.

INTRODUCTION

Benign Fibro-osseous lesions is a group of lesions in which normal architecture of bone is replaced by fibrous connective tissue and later on, the lesion is infiltrated by osteoid and cementoid tissue. It is a benign and idiopathic process. Despite the advances in the understanding of these conditions, fibro-osseous lesions continue to present problems in classification, diagnosis, and management due to multiple histological and radiographic similarities [1]. The benign fibro-osseous lesions represent a clinically diverse group of disorders of bone that share similar histopathologic features. As a group, they are relatively common in the craniofacial complex, especially the jaws. Although the general concept of BFOL is relatively well known, specific diagnostic interpretation of individual cases is often bothersome. New concepts and controversies have arisen over the past 10 to 15 years regarding classification and diagnostic criteria [2]. The definitive diagnosis of FOL is only not possible alone by examination of incisional/excisional biopsy material and and it mainly depend on close clinical, radiological as well as molecular correlation [3]. Maxillofacial FOLs are of particular interest to the radiologist because they highlight the central role of the radiologist in the diagnostic process. This role arises because the pathology for all FOLs is identical, although they range widely in behavior, from dysplasia, hamartoma to benign neoplasia with occasional repetition. The Charles Waldron wrote “In absence of good clinical and radiologic information a pathologist can only state that a given biopsy is consistent with a FOL. With adequate clinical and radiologic information most lesions can be assigned with reasonable certainty into one of several categories”. Conversely in the absence of such information Eisenberg and Eisenbud stated that “pathologists today will often rightly decline to render a definitive diagnosis. Instead, the pathologist will resort to the diplomatic designation of benign fibro-osseous lesions. This is the only acceptable approach considering the potential for inappropriate treatment otherwise”. Therefore the identification of the majority of FOLs is made upon clinical and radiological features [4].

CLASSIFICATION

Various classifications of fibro-osseous lesions have been proposed by different authors as listed below [5].

1985 - Charles Waldron
1987 - Working classification by Mico M. Malek
1990 - Peiter J. Slootweg & Hellmuth Muller
1992 - World health organization (WHO) classification
1993 - Modified classification by Waldron
2001 - Brannon & Fowler classification

*Corresponding Author: Dr. Anchal Rai

2005 - WHO classification of FOL
2006 - Paul M. Speight & Roman Carlos classification
2008 - Eversole classification

Hereby, out of these we have followed the modified classification by Waldron which was given in the year 1993.

In 1993, Waldron had reviewed the subject of benign fibro-osseous lesions (BFOL) of jaws, and suggested a modification of his earlier classification to overcome the demerits of his own classification [5].

1. Fibrous dysplasia
2. Cemento-osseous dysplasia
   a. Periapical cemento-osseous dysplasia
   b. Focal cemento-osseous dysplasia
   c. Florid cemento-osseous dysplasia
3. Fibro-osseous neoplasm
   a. Cementifying /ossifying / cemento-ossifying fibroma

Fibrous dysplasia

Fibrous dysplasia is a benign pathologic condition of the bone in which the normal bone is replaced by the fibrous tissues connective tissue. Fibrous dysplasia is not of periodontal ligament origin. It is considered as hamartomatous fibroosseous lesion. Though the exact etiology is not known. Fibrous dysplasia was first described by Lichtenstein 1938 as a disorder characterized by progressive replacement of normal bone by fibrous tissue elements. Fibrous dysplasia affecting the jaws is an uncommon developmental anomaly. It may present as Monostotic (74%), Polystotic (13%) and Craniofacial (13%) varieties [6]. It is a rare condition occurs due to the post zygotic mutation in the GNAS1 gene [6]. FD is classified as monostotic and polyostotic. It is monostotic when it affects a single bone or, less commonly, polyostotic when it involves multiple bones. About 3% to 5% of polyostotic. FD is also associated with extraosseous manifestations. McCune-Albright syndrome (MAS) is an infrequent disorder characterized by the clinical trial of polyostotic FD, skin hyperpigmentation (câfè au lait spots), and multiple endocrinopathies, including gonadal hyperfunction leading to sexual precocity (especially in females) [7]. Mazabraud syndrome is another rare disease that is characterized primarily by polyostotic FD and intramuscular myxomas [7]. Fibrous dysplasia is primarily a disease of childhood, its progression is arrested when adult life is reached and skeletal growth ends. It is believed that in cases in which the disorder is first encountered later in life, the skeletal lesions originated during childhood [8]. The lesions of fibrous dysplasia are common in maxilla as compared to mandible, and the posterior aspects of the jaw are more often involved than the anterior [6]. FD usually arises within the first or second decades of life, exhibit clinically as a slow-growing, painless expansion of the involved bone. Facial asymmetry may be obvious. In rare cases, the expansion may be more rapid or begin to accelerate after a period of slow growth, resulting in marked facial deformity and possible nerve entrapment. Many of these cases often leads to the development of an aneurysmal bone cyst (ABC) or, less commonly, malignant transformation. On a plain or panoramic film, early lesions may appear well defined and radiolucent, whereas later lesions may appear largely sclerotic. However, classic FD has a ground-glass or orange-peel appearance, with poorly visible borders that appear to blend in with the surrounding, unaffected bone [7]. The basic lesion being a replacement of medullary bone by fibrous tissue, areas of decreased density may be seen. Since the involved bone is often expanded, and its cortex thinned, the radiographic picture takes on a cystic appearance. There may be ridges on the inner surface of the cortex of the bone giving the impact of a multilocular lesion. The presence of bony trabeculae in many of the apparently cystic areas gives rise to increased densities resembling ground glass or "columns of slowly ascending smoke" [8]. The histologic appearance of FD, as with other BFOLs, varies depending on the stage of the lesion. Early FD usually exhibits a moderately cellular, fibrous stroma containing disorganized arranged, uniform, benign appearing, spindle-shaped to ovoid fibroblasts. The trabeculae tend to be delicate and curvilinear and have been likened to Chinese script-writing [7]. The surgical management of a small, monostotic mandibular lesion is much less troublesome than treatment of larger, more diffuse lesions or of craniofacial FD [6]. It has been suggested that since the growth of FD often tends to stabilize and occasionally arrest when skeletal maturity is achieved, surgical intervention in children and adolescents with more extensive lesions should be deferred as long as possible [7].

Fig-1: Showing fibrous dysplasia on the right side of the maxilla

Fibrous dysplasia:

“orange peel” appearance of fine dense trabeculae seen on intra-oral radiography in late stage.
Cemento-osseous dysplasia

Cemento-osseous dysplasia is a benign condition of the jaws that may arise from the fibroblasts of the periodontal ligaments. The pathogenesis of the cemento-osseous dysplasias is uncertain, although they appear to represent some form of reactive or dysplastic process. Cemento-osseous dysplasias are divided into three groups: periapical, focal and florid cementosseous dysplasia [9].

Fig-2: Intraoral radiograph showing characteristic feature of fibrous dysplasia that is orange peel appearance

Periapical cemento-osseous dysplasia

Periapical cemento-osseous dysplasia (PCOD) is also known as cementoma but it is not an neoplasm and it is an asymptomatic benign lesion [9]. Periapical cemento-osseous dysplasia mainly occurs in melanoderm women, at mid-age (40- 50 age-ranges) and rarely below 20 years-old. The most common site of appearance is mandible periapical region [10]. PCOD occurs most commonly in the mandible anterior region of patients. It displays three different characteristics according to its period. In the first period, which is also called osteolytic period, circular and elliptical resorption areas are seen. In the second period, which may also be called as cementoblastic period or intermediate period? In the third period, lesions display massive calcifications [9].

A radiographic study revealed that the lesions size is about 1.8 cm, ranging from 0.2 to 11 cm. Additionally, although the lesions are close to the tooth apex, the periodontal ligament remains clearly visible in radiographs [10]. Due to the nature and evolution of this lesion, no treatment is necessary. Because the teeth remain vital, tooth extraction or endodontic treatment should not be performed. Dental prophylaxis and oral hygiene instruction reinforcement to prevent periodontal disease and caries lesions which can lead to tooth loss and regular follow up examination is required [10].

Fig-3: Showing periapical cementoosseous dysplasia on the lower anteriors

Focal cementosseous dysplasia

Focal cemento-osseous dysplasia (FCOD) is a benign lesion that occurs between the periapical and florid cemento-osseous dysplasia [9]. About 80 percent of cases seen in females, between 3rd and 5th decade, and it are more common in white than in black patients. FCOD may occur in any area of the jaws, although the posterior mandible is the predominant site [9]. FCOD has three developmental stages where there is resorption of alveolar bone and radiograph shows well defined radiolucent area with loss of pdl space and lamina dura, next stage is the cementoblastic stage in this there is the mixture of radiolucent and radiopaque elements, there is the deposition of cementum like droplets in fibrous tissue and the last stage is osteosclerotic or inactive stage in this radiopacity is seen in the lesion and ginger root like pattern is seen histopathologically [11]. The differential diagnosis could be periapical granuloma, chronic osteomyelitis ossifying fibroma, osteoblastoma [11]. Once a diagnosis has been established, no treatment is necessary [9].

Florid cementosseous dysplasia

Florid cemento-osseous dysplasia (FLCOD) is a condition that usually evident as multiple radiopaque cementum-like masses distributed throughout the jaws [9]. Florid cemento-osseous dysplasia is more commonly seen in middle-aged black women although it also may occur in Caucasians and Asians. The reason for this racial and gender predilection is unknown [12]. The condition usually involves multiple quadrants. The process may be totally asymptomatic [9]. Radiographically, the lesions appear as multiple sclerotic masses, located in two or more quadrants, usually in the tooth-bearing regions. They are often confined within the alveolar bone [12]. The diagnosis of florid cemento-osseous dysplasia is usually made on the basis of radiographic appearance. No treatment is indicated. The most common complication is the development of osteomyelitis in patients who wear full or partial dentures [9].

Fig-4: Intraoral radiograph showing periapical radiolucency with radiopacity on lower anteriors
Ossifying fibroma

Ossifying fibroma (OF) is a fibro-osseous lesion that arises from the periodontal membrane. Montgomery first used the term OF in the year 1927, by which the lesion is currently known [13]. Ossifying fibroma mainly develops in the maxilla and mandible but rarely in long bones, if it occurs in long bones does it affect the tibia and the fibula [16]. Ossifying fibromas also occurs in both central and peripheral locations of the jaw bones [15]. Ossifying fibroma is a benign neoplasm primarily found in the mandible, composed of both osseous and fibrous components. Phenotypical OF is characterized by slow, progressive enlargement of the affected jaw and is diagnosed between the 2nd and 4th decades, with women affected more commonly than men [14]. From the radiological outlook, more than 50% of the lesions exhibit an expansion of the jaws and 53% shows well-defined unilocular radiolucencies and 40% are mixed radiolucent-radiopaque lesions. The lesions unusually can be radiopaque [13]. Complete surgical removal is the recommended treatment. The juvenile ossifying fibroma (JOF) has been distinguished from OF because of differences in the clinical and histopathologic presentations. JOF occurs in children and adolescents, is characterized histologically by osteid trabeculae and shows a local aggressive growth [14]. Hyperparathyroidism-jaw tumour syndrome HPT-JT may also develop jaw tumours, mainly OF, which are distinct from the brown tumours associated with severe hyperparathyroidism [14].
Juvenile ossifying fibroma

Juvenile aggressive ossifying fibroma is an uncommon and contentious, benign but aggressive osteogenic neoplasm of the jaws commonly occurring in children and young adults [18]. JOF affects both males and females equally without any significant gender preference. Maxilla is more commonly affected than the mandible [18]. The term juvenile ossifying fibroma (JOF) has 2 specific histopathologic variants of ossifying fibroma of the craniofacial skeleton. These are attributed as psammomatoid juvenile ossifying fibroma (PsJOF) and trabecular juvenile ossifying fibroma (TrJOF) [17]. Psammomatoid type is more commonly seen and it affects patients from wider range (3 months–72 years). It occurs basically in the sino-nasal and orbital bones. In contrast, trabecular variant affects patients with a mean age range of 2–12 years and mostly affects the jaws [18]. The term juvenile (aggressive) ossifying fibroma was used in the 2nd edition of the World Health Organization classification of odontogenic tumors to depict a lesion affecting the jaws of children under the age of 15 years [17]. In the early stage of development JAOF appears as a unilobulated/multilobulated radiolucent lesion followed by radiopaque appearance and is surrounded by a uniform radiolucent trimming at later stages [18]. The clinical management of JAOF remains uncertain. Small lesions can be treated conservatively by curettage or enucleation. An open surgical approach, such as transfacial, is ideal for resecting large and irregular shaped tumors that infiltrate sinuses and fronto nasal [18].

CONCLUSION

Benign fibro-osseous lesions of the jaws represent a various group of conditions in which the diagnosis of the lesions are very contentious because of the similarities in the clinical and histopathological features. A consultation with an oral and maxillofacial radiologist can be very advantageous. When a biopsy is indicated, it should include the interface between lesional and normal adjacent tissue, and the pertinent radiographic information should be provided to the pathologist. Appropriate final diagnosis will help in proper therapeutic action. Correlation between the biologic behaviour of the lesion and clinical, radiologic and histopathologic data is essential in reaching an accurate diagnosis.

REFERENCES
