Review Article

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# GIANT CELL LESIONS: A REVIEW OF THERE RADIOLOGY

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## ABSTRACT:

Diagnosis of many lesions of the oral cavity is challenging to most clinicians because of their uncommon prevalence. A number of cystic, metabolic, osteodystrophic, microbial, tumour and tumour like lesions of the oral cavity present with characteristic giant cell lesions; which makes their diagnosis and study simpler. We have attempted to classify the common giant cell lesions of the oral cavity, giving a brief account of their diagnostic features, along with their recent treatment modalities.

**Keywords:** diagnosis, giant cell, radiographs

## INTRODUCTION

Giant cells are large multinucleated cells of different lineage and the lesions containing giant cells fascinate clinicians, radiologists and pathologists. All these groups are concerned with the problem of differential diagnosis. Pathologists are intrigued by the uncertainties regarding the origin and nature of the giant cells and by their beauty as objects seen under the microscope.[1]

Diagnosis of many giant cell lesions of the oral cavity is challenging because of their uncommon prevalence. Therefore, this review was designed to get an account of their diagnostic features specially radiographically and current treatment modalities.

**Classification of Giant cell lesions of the oral cavity [2]**

1. Tumour and tumour like lesions : Central giant cell granuloma, Peripheral giant cell granuloma, Giant cell fibroma, Giant cell tumour, Osteosarcoma, Rhabdomyosarcoma, Hodgkin's lymphoma.

2. Cystic lesions : Traumatic bone cyst, Aneurysmal bone cyst

3.Metabolic lesions: Hyperparathyroidism

4. Osteodystrophic lesions : Noonan-like multiple giant cell lesion syndrome

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5. Microbial lesions: Tuberculosis, Leprosy,Actinomycosis, Sarcoidosis.

6. Miscellaneous lesions : Cherubism, Paget's disease, Fibrous dysplasia

## 1. TUMOR AND TUMOR LIKE LESIONS

### CENTRAL GIANT CELL GRANULOMA

It is a relatively uncommon pathological condition accounting for less than 7% of all benign lesions of the jaws.[3] Initially termed giant cell reparative granuloma, these lesions are no longer believed to represent a reparative process.[4]

**Radiographic features:** In the systematic review of the literature regarding the radiographic features of CGCG, of the 232 cases that fulfilled the inclusion/exclusion criteria, 46% had a unilocular appearance and 34% had well defined borders. In 33% of the cases, root resorption was noted. Bony expansion, cortical plate perforation, and paresthesia were noted in 51%, 38%, and 6% of the cases, respectively.[3]

**Treatment:**The treatment modalities considered are surgical curettage, especially for smaller lesions. Aggressive lesions are occasionally treated by en bloc surgical resection. Recent approaches for the treatment of large or multiple lesions have included weekly intralesional corticosteroid injection, daily subcutaneous or intranasal administration of calcitonin, and the use of interferon alpha-2a. Radiation treatment, however, is contraindicated because of the potential for oncogenic potentiation.[5]

### PERIPHERAL GIANT CELL GRANULOMA

The PGCG, also known as osteoclastoma, giant cell reparative granuloma, or giant cell hyperplasia, are reactive exophytic lesions found in the oral cavity.[2]

**Radiographic features:** PGCG is reported to cause cupping resorption of the underlying alveolar bone and the presence of recurrent lesion is associated with root resorption.[6] The radiographic features usually seen are superficial resorption or cupping of the alveolar bone, widened periodontal ligament space which is associated frequently with tooth mobility but may represent lesion extension around the root,[7] there may be little radiographic evidence of some lesions in teeth-bearing areas because lesions may be small and primarily in the soft tissues. Larger lesions exhibit a superficial erosion of the cortical bone surface and may demonstrate some widening of the adjacent periodontal space. Close examination of the area may reveal small spicules of bone extending vertically into the base of the lesion. In edentulous areas, the cortical bone exhibits concave area of a resorption beneath the lesion, often referred to as "saucerisation".[8]

**Treatment:**The treatment advocated has been surgical excision and elimination of possible irritant factors, with recurrence being infrequent (approximately 10%).[2]

## GIANT CELL TUMOR

In I854 Sir James Paget credited Lebert as being the first to describe the giant cell tumor.[9] Giant cell tumor of bone is a rare, aggressive noncancerous (benign) tumor.[6] It is a primary locally aggressive bone neoplasm characterized by stromal mononucleated cells associated with uniformly distributed osteoclast-like giant multinucleated cells.[10]

**Radiographic features:** The roentgenographic appearance of the giant cell tumor is said to be characteristic. There is a bulky, roughly spherical or ovoid shadow, the margins of which are fairly definitely demarcated. The shadow is not smoothly circular in outline, but here and there presents small lobulations. There are incomplete trabeculae at the periphery of the tumor. There is no periosteal reaction, and the cortex and the periosteum beyond the tumor appear to be normal. The periosteum is never separated from the uninvolved bone, so that there is no so called reactive triangle. Kirklin and Moore have expressed the opinion that there is a tendency for the tumor to break down the trabeculae and to form a conglomerate mass. They described a second roentgenographic type in which the growth may simulate the appearance of a metastatic tumor.[9]

**Treatment:** The choice of treatment lies between curettage of the tumor bearing area, and roentgen irradiation, or a combination of the two. As early as 191O Bloodgood sponsored curettage followed by chemical cauterization of the cavity. Roentgen therapy is always to be recommended following this procedure. A few investigators recommend the use of Coley's toxins in the treatment of the condition.[9]

## OSTEOSARCOMA

Osteosarcoma or osteogenic sarcoma is defined as a primary intramedullary high grade malignant tumour in which the neoplastic cells produce osteoid.[11]

It is the third most common cancer in adolescence, occurring less frequently than only lymphomas and brain tumors. It is thought to arise from a primitive mesenchymal bone-forming cell and is characterized by the production of osteoid.[12]

**Radiographic features:**Radiographs of most fully developed osteosarcomas of the jaws showed a unicentric bone destructive lesion with indefinite margins. The roentgenographic appearance of a particular osteosarcoma usually will be sclerotic, lytic or mixed, in which areas of calcification and of lysis are intermingled, Some osteosarcomas presented a “sun-ray” effect, with ossific laminae radiating in a sunburst pattern about the periphery of the lesion or from the surface of the affected bone.. In some cases, an early osteosarcoma of the jaws may show a symmetrically widened periodontal membrane space about one or more teeth on a periapical dental radiograph, apparently before showing any other prominent radiographic evidence of its presence.[13]

**Treatment:** osteosarcomas of the jaw are reported to be treated by surgery, chemotherapy or a combination of the both.[14]

## 2. CYSTIC LESIONS

### TRAUMATIC BONE CYST

In 1929, Lucas and Blum for the first time described traumatic bone cyst as a separate disease entity. It was later defined by Rushton as a single cyst that has no epithelial lining, has an intact bony wall, is fluid filled, and has no evidence of acute or chronic inflammation.[15]

Traumatic bone cysts have been referred to in different ways: hemorrhagic bone cyst, simple bone cyst, hemorrhagic traumatic bone cyst, progressive bone cavity, unicameral bone cyst, and extravasation cyst.[2]

**Radiographic features:** The radiographic feature of TBC showed a radio transparent image with irregular or scalloped but well defined margins.[2], TBC appears as scalloped unilocular radiolucency.[15] TBCs also appear as a radiotransparent image with irregular or scalloped but well defined margins. The size is variable, and a fine sclerotic margin is sometimes seen. When the radiotransparency affects the interdental bone spaces, the lesion appears lobular or scalloped. Root reabsorption is rare, and can cause disappearance of the hard lamina in 16-62% of cases.[16]

**Treatment:**The treatment included careful curettage of the bone walls, with satisfactory results characterized by progressive bone regeneration and the absence of relapses. Nevertheless, Kuttenberg et al and Baqain et al reported two cases of relapse following surgical curettage. In both cases, re-treatment of the lesions sufficed to solve the problem. Other alternative treatments such as filling of the cavity with bovine lyophilized bone or the introduction of autologous blood with bone from the patient or hydroxyapatite, may be of interest in those cases where conventional management fails. These techniques may be particularly useful when osseointegrated implant rehabilitation of the affected zone is required. However, the introduction of radiopaque materials in the cavity may complicate the diagnosis of possible lesion relapse.[16]

### ANEURYSMAL BONE CYST

Aneurysmal bone cyst has been described as a pseudocyst due to the absence of an epithelial lining. It is rarely found in the jaws and comprises of 1% of all nonepithelial and non odontogenic cysts of the jaws. Despite its recognition in the jaws since 1958, it is still an uncommon finding in the facial bones. According to Jaffe, the word aneurysmal was used in relation to the blowout distension of part of the affected bone area.[17]

The WHO defines ABC as a benign intra-osseous lesion, characterized by

blood-filled spaces of varying size associated with a fibroblastic stroma containing multinucleated giant cells, osteoid and woven bone.[18]

**Radiographic features:** Radiographically, aneurysmal bone cyst may appear as a unilocular or multilocular radiolucency with expansion and thinning of the surrounding cortical bone. It has also been described as having a honeycomb or soap bubble appearance since it may be traversed by thin bony septa. But there is no pathognomonic radiographic appearance for ABC. [17] Sometimes we can also see a destruction of the bone cortex and periosteal reaction.[19]CT often shows an osteolytic and expandable multinuclear mass with surrounding bone shell and displaced surrounding normal structures. The classic blow-out appearance has been described. A clearly-defined body wall (bony septa) imparting a soap bubble or honeycomb appearance has been reported.[20]

**Treatment:**There is no uniform treatment and management of ABC due to its varied nature. The usual treatment of choice is curettage as it is a benign lesion. The failure to remove the lesion completely has been associated with a recurrence, although there has been a report of a case whereby the lesion regressed spontaneously. Some authors have also recommended supplementing curettage with cryotherapy. The defect can be filled up with bone chips prior to cryosurgery. Segmental resections are performed with immediate bone grafting if the lesions have been found to be extensive and cause functional and cosmetic deformities. Radiation is not recommended as sarcomatous change has been reported in these lesions after irradiation. A high recurrence rate of 53 - 66% has been reported for ABC in the jaws. Therefore, a close follow up of the cases is recommended.[17]

## 3. METABOLIC LESIONS

### HYPERPARATHYROIDISM

Hyperparathyroidism (HPT) is characterized by hypersecretion of parathyroid hormone.[21] HPT is divided into primary, secondary and tertiary categories. Classic skeletal lesions, which are bone resorption, bone cysts, brown tumours and generalized osteopenia, occur in less than 5% of cases. The ribs, clavicles, pelvic girdle, and the mandible are the most often involved bones.[2] Bone lesions develop in an estimated 10% to 15% of patients with hyperparathyroidism. Multiple lesions may be identified simultaneously.

**Radiographic features:** Radiographically, lesions are characterized as well defined radiolucent areas, unilocular or multilocular. The characteristic radiographic findings are the presence of widespread loss of the lamina dura, and changes in the pattern of the trabecular bone of the jaws.[21] Demineralization and thinning of cortical boundaries such as the inferior border and the mandibular canal , a generalized decrease in the density of mandible and maxilla with a generalized rarefaction

(numerous small, randomly oriented trabeculae) and the teeth were standing out. Presence of complete effacement of lamina dura in all the teeth present, which appeared to be "floating in air." It also revealed the presence of a gross destructive lesion having poor osseous density and multiple cyst-like cavities with poorly corticated and irregular margins.[22]

**Treatment:** Treatment for brown tumour in the jaws includes enucleation and curettage, radical resection and reconstruction, radiation therapy, and chemotherapy.[2]

Studies have reported that the treatment of HPT is the first step in the management of the brown tumor, as spontaneous regression of the lesion often occurs. However, several cases of brown tumor that did not disappear or even grew after normalization of HPT level have been reported. In these cases brown tumor resection should be the preferred treatment.[21]

## 4. OSTEODYSTROPHIC LESIONS

### NOONAN-LIKE MULTIPLE GIANT CELL LESION SYNDROME

Noonan's syndrome (NS) is one of the most common genetic conditions affecting the nervous system. It was first reported by Kobylinski in 1883 and described by Noonan and Ehmke in 1963. The estimated prevalence is reported to be 1:1000 to 1: 2500 live births. It is a relatively common autosomal dominant condition with normal karyotype (46XY).[23]

**Radiographic features:** Panoramic radiographs revealed multiple, illdefined, radiolucent, multilocular, bilateral images extending over the body and angle of the mandible, however, the mandibular cortical bone was unaffected.[24]

**Treatment:** Treatment must be focused on the problems that occur and is usually multidisciplinary as in most other syndromes.[2]

## 6. MISCELLANEOUS LESIONS

### CHERUBISM

Cherubism was first described by Jones in 1933 as 'familial multilocular cystic disease of the jaws'. He later coined the descriptive term 'cherubism' when he likened the classical characteristics of full round cheeks and upward cast of the eyes to the angelic look of the cherubs immortalized by Renaissance art.[25]

Cherubism is a rare, autosomal dominant, self-limiting disorder that exhibits 100% male and 50% to 70% female penetrance.[26]

**Radiographic features:** radiographically, the lesions usually manifest as bilateral, multilocular radiolucencies with concomitant bony expansion. Numerous impacted or displaced teeth may be identified.[26] The lesions of cherubism can be classified according to their

extent: grade I, bilateral involvement of the ascending ramus of mandible; grade II, bilateral involvement of the ascending ramus of mandible and maxillary tuberosity; grade III, complete involvement of the maxilla and mandible compromising the coronoid processes and condyles. Radiographically identified multilocular radiolucent lesions mainly are affecting the body and ascending ramus of the mandible. The coronoid process might be involved and the condition extends to the maxilla, preferentially to the region of maxillary tuberosity. The radiolucencies are mainly bilateral and may affect the lower portion and fundus of the orbit which cortical bone appears to be thin but intact. The teeth are found to be displaced and impacted and root resorption is observed; the mandibular canal is often displaced. The facial sinuses frequently appear to be obliterated, resuming their pneumatic function after regression of disease activity. Some authors have reported involvement of the condyles.[27]

**Treatment:** In most cases, cherubism is a self-limiting disease that often undergoes spontaneous resolution after puberty. Thus, surgical intervention usually is unnecessary. However, patients may experience functional and psychological disturbances, in which case treatment may be needed. Studies suggest that intralesional calcitonin injection or systemic calcitonin administration may induce complete remission of lesions in affected children, with minimal adverse effects.[26]

## PAGET'S DISEASE

Sir James Paget, after whom this disease is known, first gave a clear account of its main features in 1876 at the age of 62, but it was Professor Czerny of Freiburg in 1873 who suggested the term osteitis deformans, meaning an enlargement and disfigurement of bones resulting from some inflammatory process.[28]

**Radiographic features:** In the early stages of the disease, PDB may manifest as an ill-defined, radiolucent lesion with poorly defined borders that blend into the surrounding bone. The lesion may exhibit ground-glass opacification. As the disease progresses, confluent radiolucentradiopaque areas often are seen. These affected areas often are described as having a "cotton-wool" appearance. Teeth in the affected regions usually demonstrate hypercementosis.[26]

The cotton wool patches, as these patches of sclerosed bone are so aptly described, had fluffy ill defined margins and were largest in the tuberosity region. In the mandible the fibrillar pattern was more marked throughout the body of the bone and was not necessarily related to the vicinity of the teeth. The cortical plate of either jaw was lost and the outline of the bone became ill-defined. Another picture was seen in the maxilla when there was a general, even slight increase in density, without any cotton wool patches, giving a ground glass appearance. The greatest

expansion of the maxilla occurred bucally over the antrum. Changes in the bone pattern were sometimes seen before there was any change in the outline. [28]

**Treatment:**

Slowly evolving PDB or in asymptomatic patients, treatment often is unnecessary or is delayed until the level of alkaline phosphatase is more than 25% to 50% above normal. Parathyroid hormone antagonists, including calcitonin and bisphosphonates, usually are used to reduce the rate of bone turnover. Surgery is not usually indicated.while there is no specific treatment of Paget's disease, very careful case management is needed to avoid complications when the jaws are involved. The teeth and gingivae must be carefully attended to, in order to avoid inflammatory episodes that may lead to osteomyelitis. If a tooth has to be extracted, it must be done surgically and the socket sutured afterwards not only to control haemorrhage but infection, and to avoid an oral antral fistula in the upper jaw. Dentures must be well designed and if necessary frequently remade to avoid any pressure necrosis of the bone. Should a reduction of the alveolus be deemed advisable for prosthetic reasons, care must be taken to see that no dense superficial sclerotic nodules of bone are left behind or they may sequestrate. Since the marrow is so vascular, haemorrhage is considerable. In the late stages of the disease analgesics will be needed to relieve the continuous dull ache, and in very severe cases an alcohol injection to the trigeminal ganglion may be necessary, such as is done for other major trigeminal neuralgias. A patient with Paget's disease of the jaws should be seen every year if the complications associated with the teeth both natural and artificial are to be avoided.[28]

## FIBROUS DYSPLASIA

It is a rare localised disease often associated with bony deformities caused by the abnormal proliferation of fibrous tissue interspersed with normal or immature bone because of poorly differentiated, mutated osteoblasts.[29] It frequently affects the jawbones. They constitute 7% of all nonmalignant bone tumors.[2] The lesion is classified into two forms: monostotic (75–80%) and polyostotic types (20–25%).[30]

**Radiographic features:** fibrous dysplasia is radiolucent, with a grayish "ground-glass" pattern that is similar to the density of cancellous bone but is homogeneous, with no visible trabecular pattern.[6] CT and MRI scans also may be useful for establishing the full extent of the lesion and for assessing the degree of bony expansion. Bone scintigraphy is a sensitive imaging modality that is useful for detecting early fibrous dysplasia, as well as for determining the extent of polyostotic disease.[26]

**Treatment:** Surgical management of a small, monostotic mandibular lesion is much less problematic than treatment of larger, more diffuse lesions or of

craniofacial fibrous dysplasia. It has been suggested that since the growth of FD often tends to stabilize and occasionally stops when skeletal maturity is attained, surgical intervention in children and adolescents with more extensive lesions should be delayed as long as possible. However, in some cases, fibrous dysplasia may persist into late adult life. Conversely, spontaneous regression of fibrous dysplasia also has been reported. If treatment is necessary, especially in young patients with significant cosmetic or functional deformity, therapy should be limited to a contouring procedure, without complete resection, to minimize morbidity. However, it has been estimated that 25% to 50% of patients will exhibit subsequent regrowth of the lesion and, thus, may require multiple shave-down procedures. Complete surgical resection is recommended for patients with rapidly expanding fibrous dysplasia, lesions that encroach on the orbit, and lesions secondarily involved with an aneurismal bone cyst or a malignancy. Radiation therapy is contraindicated owing to the increased risk of malignant transformation. Long-term clinical and radiographic follow-up is recommended for any patient with fibrous dysplasia. [2]

## REFERENCES:


1. Kramer IRH. Changing views on oral diseases.Proc.roy.Soc.Med 1974;67:271-276.
2. Varghese I, Prakash A. Giant cell lesions of the oral cavity. OMPJ 2011 Jun;2(1):107-110.
3. Stavropoulos F, Katz J. Central giant cell granulomas:A systematic review of the radiographic characteristics with the addition of 20 new cases. Dentomaxillofacial Radiology 2002; 31: 213-217.
4. Edwards PC, Fox J, Fantasia JE, Goldberg J, Kelsch RD.Bilateral central giant cell granulomas of the mandible in an 8 year old girl with Noonan syndrome(Noonan-like/multiple giant cell lesion syndrome). Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005 Mar; 99(3): 334-340.
5. Roberson JB, Crocker DJ, Schiller T. The diagnosis and treatment of central giant cell granuloma. J Am Dent Assoc 2011 June 2; 128: 81-84.
6. Madhusudan AS, Verma M, Nayak S, Dakwala P. Giant cell epulis: Report of 2 cases. http://www.idakerala.com.
7. Flaitz CM. Peripheral giant cell granuloma: A potentially aggressive lesion in children. American Academy of Pediatric Dentistry 2000; 22(3):232-233.
8. Subarnbhesaj A. Peripheral giant cell granuloma: A case report and review literature.KDJ 2005 Dec;8(2):125-130.
9. Major SG. Giant cell tumors of the jaws. Annals of surgery 1936 Dec; 104(6):1068-1079.
10. Junior RZB, Camargo OP, Ida CM, Baptista AM, Ribeiro MB, Bruno JM, Oliveira CRJCM. Primary malignancy in giant cell tumor: A case report. Sao Paulo Med J 2009; 127(5): 310-313.
11. Ajura AJ, Lau SH. A retrospective clinicopathological study of 59 osteogenic sarcoma of jaw bone archived in a stomatology unit. Malaysian J Pathol 2010;32(1):27-34.
12. Rajendran R, Sivapathasundharam B. Shafer's textbook of oral pathology. 6th ed. Noida(India): Elsevier; 2009.p.314-315,133,127,169,182,530, 711,706
13. Garrington GE, Scofield HH, Cornyn J, Hooker SP. Osteosarcoma of the jaws:Analysis of 56 cases. Cancer 1967 March; 20(3):377-391.
14. Picci P. Osteosarcoma(osteogenic sarcoma).Orphanet Journal of Rare Diseases 2007;2.
15. Kuhmichel A, Bouloux GF. Multifocal traumatic bone cysts:case report and current thoughts on etiology. J Oral Maxillofac Surg 2010; 68: 208-212.
16. Ballester IC, Figueiredo R, Aytes LB, Escoda CG. Traumatic bone cyst:A retrospective study of 21 cases. Med Oral Patol Oral Cir Bucal 2009 May 1; 14(5): e239-243.
17. Hussaini NZ, Zain R, Rahman ZA, Jalaludin MA. Aneurysmal bone cyst:A case report and review of the literature. Annals Dent Univ Malaya 1998; 5: 53-56.
18. Kaffe I, Naor H, Calderon S, Buchner A. Radiological and clinical features of aneurismal bone cyst of the jaws. Dentomaxillofacial Radiology 1999; 28:167-172.

19. Calleja JMLA, Carretero JLC, Martin JG, Burgueno M. Aneurysmal bone cyst of the mandible:Case presentation and review of the literature. Med Oral Patol Oral Cir Bucal 2007; 12: E401-403.
20. Tang IP, Shashinder S, Loganathan A, Anura MM, Zakarya S, Mun KS. Aneurysmal bone cyst of the maxilla. Singapore Med J 2009; 50(9): e326-328.
21. Fabue LC, Soriano YJ, Perez MGS. Dental management of patients with endocrine disorders. J Clin Exp Dent 2010; 2(4): e196-203.
22. Dinkar AD, Sahai S, Sharma M. Primary hyperparathyroidism presenting as an exophytic mandibular mass. Dentomaxillofacial Radiology 2007;36:360-363.
23. Yazdizadeh M, Tapia JL, Baharvand M, Radfar L. A case of neurofibromatosis-Noonan syndrome with a central giant cell granuloma. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004; 98: 316-320.
24. Cancino CMH, Gaiao L, Filho MSA, Oliveira FAM. Giant cell lesions with a Noonan-like phenotype:A case report. The Journal of Contemporary Dental Practice 2007 May 1; 8(4):1-6.
25. Meng XM, Yu SF, Yu GY. Clinicopathologic study of 24 cases of cherubism. Int.J.Oral Maxillofac.Surg 2005; 34: 350-356.
26. Alawi F. Benign fibro-osseous diseases of the maxillofacial bones. Am J Clin Pathol 2002; 118(1): S50-S70.
27. Lima G de M, Almeida JD, Cabral LA. Cherubism:Clinicoradiographic Features and Treatment. J Oral Maxillofac Res 2010 Apr-Jun; 1(2):e2:1-8.
28. Cooke BED. Paget's disease of the jaws:Fifteen cases. Ann R Coll Surg Engl 1956 Oct; 19(4): 223-240.
29. Kruse A, Pieles U, Reiner MO, Zunker C, Bredell MG, Gratz KW. Craniomaxillofacial fibrous dysplasia: A 10-year database 1996-2006. British Journal of Oral and Maxillofacial Surgery 2009; 47: 302-305.
30. Yasuoka T, Takagi N, Hatakeyama D, Yokoyama K. Fibrous dysplasia in the maxilla:Possible mechanism of bone remodeling by calcitonin treatment. Oral Oncology 2003; 39: 301-305.