GIANT CELL LESIONS OF ORAL CAVITY

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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 clinical, histological and diagnostic features, along with their recent treatment modalities.

Introduction

Giant cells are large multinucleated cells of different lineage.[1] Foreign body giant cells, Langhan's giant cells, Touton giant cells, tumour giant cells, are various types of giant cells apart from the miscellaneous types of giant cells such as Aschoff cells of rheumatic nodule and Reed Sternberg cells of Hodgkin's lymphoma.

Classification

1. Microbial lesions: Tuberculosis, Leprosy, Actinomycosis, Sarcoidosis
2. Tumour and tumour like lesions : Central giant cell granuloma, Peripheral giant cell granuloma, Giant cell fibroma, Giant cell tumour, Osteosarcoma, Rhabdomyosarcoma, Hodgkin's lymphoma
3. Cystic lesions : Traumatic bone cyst, Aneurysmal bone cyst
4. Metabolic lesions : Hyperparathyroidism
5. Osteodystrophic lesions : Noonan-like multiple giant cell lesion syndrome
6. Miscellaneous lesions : Cherubism, Paget's disease, Fibrous dysplasia

ORAL TUBERCULOSIS

Tuberculosis lesions of the mouth may be either primary, or secondary to pulmonary tuberculosis,[2] with secondary lesions being more common. The typical oral lesions consist of a stellate ulcer, most commonly on the dorsum of the tongue.[3] They may also occur on the gingiva, floor of mouth, palate, lips and buccal mucosa.[4] The diagnosis of tuberculosis is confirmed by the presence of acid-fast bacilli in the specimen, or more likely by culture of tuberculosis bacilli.

ORAL LEPROSY

Leprosy lesions are common in the lepromatous form,[5] with the prevalence ranging from 19% to 60% of the patients. The presence of oral lesions is directly proportional to the duration of the disease, indicating that these are a late manifestation.[5]The oral lesions in leprosy develop insidiously and are generally asymptomatic and secondary to nasal changes.[6] The most frequently affected site is the hard palate.[6]

ORAL ACTINOMYCOSIS

Cervicofacial Actinomycosis is the most common form of this rare disease.[7] .The sites most commonly involved include the submandibular space [7], cheek, parotid gland, teeth, tongue, [8] nasal cavity, gingival and oral space. There is a slight male prevalence in young adults. FNAC is the method of choice in the diagnosis of cervical Actinomycosis.

ORAL SARCOIDOSIS

Head and neck lesions of sarcoidosis are manifested in 10 to 15% of patients. Oral involvement generally appears in patients with chronic multisystem sarcoidosis and seldom occurs in the acute stage. The oral lesions may be solitary, multiple or part of a generalized disease. In some cases, oral involvement is the first, or only, manifestation of the disease[9]and appears as a non tender well-circumscribed brownish red swelling. Oral glucocorticoids are the first-line treatment for sarcoidosis.

CENTRAL GIANT CELL GRANULOMA

The WHO has defined it as an intraosseous lesion consisting of cellular fibrous tissue that contains multiple foci of hemorrhage, aggregations of multinucleated giant

cells and occasionally trabeculae of woven bone. Some authors separate CGCG into two types, referring to its clinical and radiographic features as (a) Nonaggressive lesion which is usually slow growing and asymptomatic; [10] and (b) Aggressive lesion which is usually found in younger patients and is painful, grows rapidly, is larger overall, and often causes cortical perforation and root resorption and has a tendency to recur.[11] Traditionally management has been surgical, by means of excision and curettage.

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. PGCG's may present itself as polypoid or nodular lesions, predominantly bluish red with a smooth shiny or mamillated surface. They are variable in size, though reportedly rarely exceed 2 cm in diameter, and are generally soft or rubbery to touch. They are basically asymptomatic, unless they interfere with occlusion and affect largely the lower jaw in the premolar and molar regions.[12] Treatment is usually by surgical excision and elimination of possible irritant factors, with recurrence being infrequent (approximately 10%).[12]

GIANT CELL FIBROMA

GCF was named for its characteristically large, stellate-shaped, mononuclear and multinucleated giant cells[13]. There is no gender predilection for GCF, but it is a lesion of the young; found most commonly in the first three decades of life.[13] It presents clinically as an asymptomatic raised lesion, 1cm or smaller in diameter.[13] It may be pedunculated or sessile and is found most commonly on the gingiva. The treatment of choice is conservative surgical excision.[14]

GIANT CELL TUMOUR

Giant cell tumor of bone (GCT) is a rare, aggressive non-cancerous (benign) tumor. It generally occurs in adults between the ages of 20 and 40 years. Giant cell tumors occur in approximately one person per million per year.[15] They most frequently occur around the knee joint in the lower end of the thighbone (femur) or the upper end of the shinbone (tibia) and can occur in mandible and wrist and the hip bones. Treatment includes scooping out the tumor (curettage) [16]

OSTEOSARCOMA

Approximately 7% of all primary OS arise in the jaw bones.[17]The mandible is more commonly involved than the maxilla (1.5:1) [17] Consideration should be given to the possibility of oral metastases in patients with known primary malignant disease and biopsy is essential for establishing the diagnosis.[18]

RHABDOMYOSARCOMA

Rhabdomyosarcoma (RMS) is a mesenchymal malignant neoplasm that exhibits skeletal muscle cells with varying differentiation degrees. It occurs most often in the head and neck region, genitourinary tract, retroperitoneum and to a lesser extent the extremities.[19] In the head and neck, the most frequently affected sites are orbit, paranasal sinuses, soft tissues of the cheek and the neck. In the oral cavity the most common sites are tongue, palate and buccal mucosa.[19]The diagnosis of RMS is confirmed through biopsy of the primary tumor.

HODGKIN'S LYMPHOMA

Lymphomas are the second most frequent malignancies affecting the head and neck region after carcinomas.[20]Hodgkin's lymphoma is a disease affecting primarily lymph nodes with secondary extranodal spread. It accounts for 1 - 5% of head and neck tumors.[20] Immunophenotypically, Reed - Sternberg cells are positive for CD15/CD30 and negative for CD45/CD20 both in nodal and extra nodal disease.

TRAUMATIC BONE CYST

TBCs 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. The underlying etiopathogenesis is not clear. Simple curettage of the bone walls is performed, with healing after 6-12 months.[21]. Radiologically, TBCs appear as a radio transparent image with irregular or scalloped but well defined margins.[21] Histologically, the lesion appears as a cancellous bone cavity that may be vacant and without a lining, or presents as a thin connective tissue layer with a scant liquid content.[21]

ANEURYSMAL BONE CYST

ABC is characterised by the replacement of the bone by spongy fibro-osseous tissue, and is a locally destructive and multicystic lesion filled with blood.[22]Only 2% of ABCs are found in the head and neck, with 66% of these being located in the mandible.[23] Histologically, the ABC is characterised by large blood-filled spaces which do not have an endothelial lining. Instead, the cyst wall and septa are made up of fibroblasts, myofibroblasts, histiocytes, congested vessels, osteoblasts, osteoid, bone and degenerated calcifying fibromyxoid tissue. Surgery and curettage of the cavity is the main treatment of ABC.

HYPERPARATHYROIDISM

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.[24] The ribs, clavicles, pelvic girdle, and the mandible are the most often involved bones. Both primary and secondary HPT are prone to cause loss of the lamina dura. Bone alterations are the

main consequences of this endocrine condition, including the development of an osteolytic lesion called brown tumor.[25] It represents the terminal stage of the bone remodelling process during secondary HPT. The treatment for brown tumour in the jaws includes enucleation and curettage, radical resection and reconstruction, radiation therapy, and chemotherapy.

NOONAN-LIKE MULTIPLE GIANT CELL LESION SYNDROME

Noonan syndrome (NS) is a common autosomal dominant multiple congenital anomaly disorder, characterised by short stature, craniofacial dysmorphisms and congenital heart defects (CHD), in particular pulmonary valve stenosis (PVS). Treatment focuses on the problems that occur and is usually multidisciplinary as in most other syndromes[26]

CHERUBISM

Cherubism, or familial intraosseous fibrous expansion of the mandible, is a disease characterised by the presence of giant cells and fibrous tissue proliferation. Histopathology reveals numerous multinucleate giant cells which are tartarate resistant acid phosphatase positive (characteristic of osteoclast). These cells are scattered in between mononuclear spindle cells. Eosinophilic cuffing of vessels is specific for cherubism. [27]. Surgery should be reserved for the cases presenting with ugly features or with functional abnormality. [28]

PAGET'S DISEASE

It is a localized disorder of bone remodelling characterized by an increase in osteoclast-mediated bone resorption and a compensatory increase in new bone formation.[29] PDB is characterized by increased bone turnover, which can affect a single bone (monostotic) or multiple bones (polyostotic. According to the literature, skull involvement in Paget's disease occurs in 65–70% of advanced polyostotic cases.[30] An alkaline phosphatase level diagnostic of Paget's is above 1000 IU/ml.[31]

FIBROUS DYSPLASIA

It is the replacement of normal bone with fibrous tissue causing painless expansile lesions that impair cosmetic and structural function of bone. They constitute 7% of all nonmalignant bone tumors and may be either monostotic or polyostotic[32] .The monostotic form is more commonly found in the facial skeletal region. The normal bone is replaced by tissue that is more radiolucent, with a grayish "ground-glass" pattern that is similar to the density of cancellous bone but is homogeneous, with no visible trabecular pattern.

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