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Non-Endodontic Periapical Radiopaque Jaw Lesions: A Mini-Review

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ABSTRACT

Endodontic practice may be affected by periapical radiopaque/hyperdense lesions. Endodontists may have trouble diagnosing and treating radiopaque lesions. Inflammatory, non-inflammatory, neoplastic, dysplastic, metastatic or metabolic bone changes may occur. With the increased use of cone-beam CT, a detailed review of radiopaque lesions may help clinicians differentiate these lesions. Differentiating inflammatory, non-inflammatory and neoplastic disorders simplifies diagnosis and helps choose the best treatment. This literature review covers radiopaque/hyperdense lesion differential diagnoses, clinical, radiographic, histological and management issues.

Keywords: Periapical periodontitis; Differential diagnosis; Radiopaque; Endodontists

INTRODUCTION

Root canal infection treatment protocols require a precise diagnosis. Dental granuloma, radicular cyst and periapical abscess occur frequently and the radiographs show radiolucent inflammation and periapical bone resorption. Radiopaque lesions near the tooth apex may be endodontic or non-endodontic and require special attention. Low-grade chronic periapical inflammation may cause osteoblastic and osteoclastic activity. Imaging exams can reveal the margins of a lesion, its proximity to adjacent teeth and the internal composition, especially in calcified deposits. Periapical radiopaque lesions may be relevant to endodontic practice. Periapical radiographs and CBCT images help diagnose inflammatory, dysplastic and neoplastic periapical diseases. Many clinicians struggle to diagnose and treat radiopaque/hyperdense lesions since 1985, when Bender and Mori discussed the diagnosis of endodontic and periodontal periapical and periodontal radiopacities. This mini review aims to illustrate the radiopaque/hyperdense lesion clinical, radiographic, histological and management literature and to discuss differential diagnoses [1].

LITERATURE REVIEW

This study searched PubMed for studies, reviews and case reports on radiopaque periapical inflammatory, non-inflammatory, neoplastic and metabolic jaw lesions. The search was limited to English-language articles in indexed dental or medical journals on radiopaque lesion etiology, clinical aspects, radiographic characteristics, histological characteristics and management. This study included only relevant and pertinent articles from various dental specialties [2].

Reactional Osteogenesis (RO) constitutes an asymptomatic, localized radiopaque lesion in the maxillary sinus associated with secondary root canal infection in the molar apex. The sinus mucosa forms new bone on the maxillary sinus's superficial cortical due to the low-intensity and prolonged inflammation from the infected root canal. On conventional

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radiographs, RO appears as a poorly defined, dome-shaped, radiopaque thickening. CBCT images show a hyperdense, well-defined lesion with an irregular, round or ovoid shape.

Estrela et al. reported four cases of female patients aged 50-70 with this entity. Histologically, reactional osteogenesis bone tissue has distinct maturation stages, concentric lamellae and organized haversian systems. Inflammatory cells, mature tissue, and marrow may be present. If the apical infection persists after treating the root canal infection and bone reaction, surgery is recommended [3].

Condensing Osteitis (CO) (focal sclerosing osteomyelitis) is an asymptomatic, pathological, persistent osseous alteration caused by a chronic, low-grade pulpal inflammatory stimulus. CO appears as a diffuse radiopaque bone lesion in the alveolar process on periapical radiographs, usually at a mandibular premolar or molar apex or extraction site. Deep caries or extensive restorations may indicate pulp disease. CO may develop near radiolucent inflammatory periapical lesions (granuloma, cyst and abscess) or in enlarged periodontal ligament spaces. CBCT scans show CO as an ill-defined, non-expanding sclerotic lesion in close proximity to an infected tooth [4]. CO lesions are more common between the third and seventh decades of life, regardless of gender. Clinical examination and radiograph suffice to diagnose OC therefore a biopsy is not necessary. The microscopic image shows compact bone replacing the bone marrow and cancellous bone whereas bone marrow fibrosis and inflammation are rare. Root canal therapy is the treatment of choice.

Osteomyelitis begins in the medullary and cancellous bone and spreads to the haversian systems and periosteum. Due to the dense maxillary vascularity, osteomyelitis mostly affects the mandible. Osteomyelitis can sometimes be caused by an odontogenic infection. Chronic Osteomyelitis (COS) constitutes a bone infection with a duration of a month or longer. Chronic osteomyelitis can affect one part of the mandible or the entire hemi-mandible [5]. Its cause may be odontogenic or unknown. Chronic osteomyelitis is associated with several symptoms and clinical signs. Initially osteomyelitis limits the jaw opening while fever, leukocytosis and suppuration are not present. A mandibular enlargement is noticed without soft tissue involvement. Chronic osteomyelitis manifests 2 epidemiological peaks, first in adolescence and then after the 5th decade [6]. After some time, infectious foci, suppuration, intra- and extraoral fistulas and soft tissue involvement can be observed. As a result, sequestration and pathologic fractures may occur. Imaging is necessary to rule out malignant tumors in chronic osteomyelitis and a biopsy may also be taken under consideration. Imaging initially reveals a radiolucent/radiopaque area, with concurrent reaction from the periosteum and subperiosteal bone and sclerotic alterations [7]. Both the acute and chronic osteomyelitis treatment require antibiotics intake, removal of any responsible teeth, bone curettage and debridement and removal of any necrotic bone fragment. In case of chronic refractory osteomyelitis, decortication may be necessary to reach the infected medullary bone or even surgical resection [8].

Osteomyelitis with Proliferative Periostitis (OSPP) causes de novo periosteal bone formation due to inflammation. A gradually growing oedema in the inferior border and buccal cortex of the mandible near the molars can be observed. Radiographs of OSPP show radiopaque concentric parallel layers of new bone deposition in the periosteum (onion skin appearance) without cortical perforation. Chronic osteomyelitis tissue samples may show osteoblastic rimming and acute and chronic inflammatory cells. OSPP may be treated either endodontically or surgically. In case of excessive lamellar bone growth, surgical remodeling may be necessary [9].

Idiopathic Osteosclerosis (IO) is an asymptomatic, non-expansive, radiopaque lesion in posterior alveolar processes. IO most often affects the lower first molar's periapical region, followed by the mandibular first and second premolars. IO lesions may be superimposed over the root of a tooth with a less visible periodontal membrane, under the apex of a tooth separated by a visible membrane or separated from a tooth closer to the inferior border [10]. IO radiographs show a well-defined radiopaque/hyperdense bony lesion that may be round, elliptical or irregular with a ground-glass appearance. In the absence of other causes, radiopaque areas near the apices of asymptomatic, healthy teeth or small restorations may indicate IO. As long as IO and the adjacent tooth are stable, they do not require any treatment. IO has dense lamellar bone and small marrow channels without any inflammatory cell infiltration. The diagnosis doesn’t require a biopsy [11].

DISCUSSION

Cemento-Osseous Dysplasia (COD) is the most common fibro osseous lesion in the oral cavity. COD, a group of benign lesions with an unknown cause, replaces bone tissue with fibrous tissue containing newly formed mineralized structures. These lesions are radiolucent or radiopaque depending on disease maturity. COD has three radiographic subtypes: Focal, periapical and florid. All of them remain asymptomatic and are discovered accidentally in regular radiological checkups. Focal COD is a single, asymptomatic lesion in the posterior mandible or maxilla and may arise in close proximity to the root of the tooth or in an edentulous area [12]. Females are more commonly affected, aged 30-40 and the most common location is the posterior mandible. Black patients are also more commonly affected. Asymptomatic periapical COD occurs near one or more vital mandibular anterior teeth. Black middle-aged females are more commonly affected. Florid COD affects multiple upper and lower jaw quadrants. Black middle-aged females are more commonly affected. COD can appear as a radiolucent lesion or a mixed or radiopaque lesion near mandibular tooth apices or tooth extraction sites [13]. Radiolucent lesions resemble periapical endodontic infections, but pulp vitality tests are positive. The round mixed and radiopaque lesions have a well-defined radiolucent periphery around the radiopaque areas and do not appear to merge with the tooth's root. COD has diverse calcified structures

histopathologically. COD formation begins with a vascular fibrous stroma with osteoid and basophilic cementoid structures. Later in the disease, the stroma becomes more fibrotic and osteoid trabeculae, thicker curvilinear bony trabeculae and cementoid masses may appear [14]. COD can be diagnosed clinically and radiographically without biopsy or surgery. Radiographic follow-up is needed to confirm COD diagnosis and prevent odontogenic infections in teeth overlying the lesional tissue. COD's avascular and dysplastic bone enables the odontogenic infections to spread, causing osteomyelitis and bone sequestration.

Fibrous Dysplasia (FD) is a benign developmental disorder that causes abnormal osteogenesis and fibrous tissue with abnormal mineralization to replace normal bone. A mutation in the G protein alpha-subunit (Gs-alpha) gene disrupts guanosine triphosphatase and increases cAMP, which stimulates endocrine receptors. Increased intracellular cAMP in bone marrow osteoprogenitor cells may affect preosteoblast proliferation and differentiation. FD can be monostotic or polyostotic [15]. The rare polyostotic form may be linked to McCunee-Albright syndrome, which causes hormonal changes, precocious puberty, skin pigmentation and other endocrine abnormalities. The monostotic form of FD affects the craniofacial bones, including the maxilla, which is the most affected jaw site. These lesions are common in the first to third decades of life and have no gender or race preference. Asymptomatic bone enlargement can cause buccolingual and inferior cortical bone expansion, maxillary sinus obliteration, root and tooth displacement, root resorption, inferior mandibular canal dislocation and malocclusion in monostotic FD. Radiographically, FD could range from a radiolucent lesion to a completely radiopaque lesion, with the majority of cases displaying mixed and radiopaque images that blend into the normal bone margins, assuming a ground-glass appearance [16]. In addition, conventional radiographs may reveal a narrowing of the periodontal ligament space and a mixture of lamina dura and lesional bone in the lamina dura. Biopsy is required to diagnose FD; however, clinical and radiographic characteristics, as well as microscopic characteristics, are crucial for FD diagnosis. Due to the clinical and histological similarity of fibrodysplasia to other benign fibro-osseous lesions, GNAS mutational analysis may be an essential diagnostic adjunct.

Histopathological characteristics of FD include osteogenesis manifested by thin osteoid trabeculae anastomosing in a hypercellular fibroblastic stroma. In close proximity to the trabecula, a ring of osteoblasts could be seen. In advanced stages, the osteoid trabeculae thicken and assume characteristics of a "Chinese figure". In the majority of cases, conservative surgical treatment involving cosmetic recontouring surgery and follow-up are the primary modalities for managing FD cases; however, new growth may occasionally occur, albeit at a slow rate [17].

Neoplasms involving the jaw bone include both benign and malignant entities, which are further divided into primary and metastatic malignancies. The benign tumors include odontomas, osteomas, osteoblastomas, osteoid osteomas, central ossifying fibromas, cementoblastomas, Gorlin cysts, ameloblastic fibro-odontomas and adenomatoid odontogenic tumors. The malignant tumors include osteosarcomas, chondrosarcomas and metastatic tumors originating from lung, kidney, liver, prostate, breast, genital organs and colorectum [18].

Odontoma is the most prevalent odontogenic tumor-like malformation (hamartoma) of unknown etiology that affects the tooth-bearing regions of the jaws. These lesions display complete differentiation of both epithelial and mesenchymal odontogenic cells, as well as enamel, dentin and sometimes cementum. Odontomas do not exhibit any gender preference and are most commonly observed in children and adolescents. Compound and complex odontomas are two distinct types of odontomas that can be distinguished by their level of histologic development and consequently, their level of mineralized content development. Compound odontoma is characterized by the presence of numerous tooth-like structures in the tooth-bearing regions of the jaws, particularly in the anterior maxilla. Complex odontoma is characterized by the presence of an amorphous calcification with dysplastic dentin covered by enamel in tooth-bearing regions, most commonly the posterior mandible [19]. Clinically, these two types of odontomas are slow-growing conditions with infrequent manifestations of clinical symptoms. Odontoma of the jaw is typically a radiographic incidental finding, with some cases identified during the investigation into the causes of delayed tooth eruption. There have been reports of bone expansion due to large odontomas, as well as pain and swelling from infected odontomas. Compound odontomas appear on radiographs as multiple radiopaque tooth-like structures surrounded by a thin radiolucent line indicating a distinct separation from the adjacent bone. Typically, the diameter of these lesions ranges between 10 mm and 20 mm. Complex odontomas have a distinctive radiographic pattern consisting of an amorphous radiopaque core surrounded by a radiolucent zone. On a histological level, odontomas consist of a capsule of connective tissue containing strands or islands of odontogenic epithelium, a tubular dentin formation and an enamel matrix. Odontomas are typically treated with local surgical excision and recurrences are uncommon [20].

Osteoma is a benign tumor composed of mature bone structures with cancellous or compact bone characteristics. Osteomas can develop anywhere on the skeleton, including the epidermis, dermis and muscles. However, these tumors are more commonly found in the mandible and maxilla, which are craniofacial bones. Apparently, three types of osteoma have been identified: Central osteoma, originating from the endosteum; peripheral osteoma, originating from the periosteum and osteoma originating from extraskeletal soft tissue. The majority of cases affecting the jawbones are of the peripheral type. Affecting the posterior mandible and condyle predominately. Despite the rarity of the central osteoma, its characteristics are well-described. It may or may not be asymptomatic and appears to be a persistent, slow-growing, expanding lesion more commonly found in the mandible,

primarily in the premolar region. In some cases, central osteomas are not associated with a tooth, but there is evidence linking them to root displacement or root resorption. Radiographically, a central osteoma appears as a round radiopaque mass with well-defined borders and no radiolucent halo between the lesion and the normal bone. The presence of mature bone with compact lamella or/and trabecular bone with sparse channels of fibrovascular tissue is shared by all three types of osteomas. Due to the similarity of central osteoma to other bone lesions, the final diagnosis should be based on the association of the histological findings, radiographic findings and the presence of evident growth, expansion and displacement of the teeth. In suspected cases, a biopsy is required and surgical excision is advised once the diagnosis has been confirmed. Recurrences following surgical treatment are uncommon.

Osteoblastoma is a benign, slow-growing bone tumor that represents about 1% of all primary bone tumors. The majority of these tumors are diagnosed in the second and third decades of life and are typically found in the axial skeleton; the mandible is the second most prevalent location. Although osteoblastomas are most common in the mandible, they can also affect the maxilla. Osteoblastomas are typically located centrally in the bone; however, periosteal lesions have also been reported. Osteoblastomas appear to have a preference for males; however, this preference may vary between studies. These tumors can be asymptomatic or they can cause severe pain, swelling and tenderness. Radiographic manifestations of osteoblastoma include a solitary round or oval radiolucent lesion with radiopaque structures and a well-defined sclerotic margin. Mature lesions are characterized by the presence of radiolucent rims and dense radiopaque structures in their centers. Some of these tumors may be located near the apex of a tooth's root, but they are unrelated to pulp necrosis. Osteoblastomas are hypothesized to have a high growth potential and to be radiographically larger than 10.5 mm in diameter. Osteoblastoma is characterized histopathologically by the presence of osteoid and bone trabeculae that permeate a well-vascularized connective tissue stroma. Bone trabeculae and osteoid tissue are surrounded by osteoblasts with typical morphological characteristics. Within the osteoid tissue, various degrees of mineralization with obvious basophilic reversal lines can be observed. In suspected cases, especially enlarged and painful lesions, incisional biopsies are required for diagnosis. Following histopathological verification, surgical treatment is advised. The surgical treatment of osteoblastomas can vary significantly based on their clinical behavior and location, ranging from the preferred en bloc resection to aggressive curettage in cases of tumors in tooth-bearing regions. Some reported cases of aggressive osteoblastomas had the potential for local invasion and a propensity to recur.

Osteoid Osteoma (OO) is a rare benign bone tumor that occurs infrequently in the jaws. It occurs most frequently in the long bones, particularly the proximal femur and tibia and is typically diagnosed in the second and third decades of life, with a 3:1 male-to-female predilection. In the mandible, there is a slight preference for the occurrence of OO, with only two cases occurring in tooth-bearing regions. Mohammed et al. reported an unusual case of OO associated with the apex of the second mandibular premolar and the first mandibular molar. According to their report, the associated teeth responded positively to the vitality tests. Lesions of the craniofacial region, which typically range in size from 10 mm to 20 mm, may cause sensitivity and edema. OO has a diminished growth potential compared to osteoblastoma. These lesions are typically accompanied by severe pain that worsens at night, at which point nonsteroidal anti-inflammatory drugs may provide relief. There is a hypothesis that the intensity of this pain is due to the rich vascularity of OO, which leads to innervation of the free nerve endings into the lesion and the production of prostaglandins; as a result, nonsteroidal anti-inflammatory drugs that affect prostaglandins may alleviate OO-associated pain. Radiographically, OO is characterized as a round lytic lesion with a central nidus less than 10 mm in size, surrounded by a sclerotic bony margin. Although OO may resemble cemento-osseous dysplasia, its diagnosis requires a bone biopsy when bone enlargement or pain are present. OO has similar histological characteristics to osteoblastoma, including the presence of osteoid, fused trabeculae within a vascularized stromal tissue and a prominent osteoblast rim. The treatment of choice is surgical excision and its removal may include the affected teeth. In general, this condition has a favorable prognosis and recurrence after surgical treatment is uncommon.

Central Ossifying Fibroma (COF) is a benign fibro-osseous lesion with neoplastic behavior; it consists of fibrocellular tissue and mineralized material. COF arises in tooth-bearing areas, typically the mandible, with the majority of cases characterized by swelling and cortical expansion. Typically, Ossifying Fibroma (OF) is not associated with pain and some cases are discovered during routine radiographs. Conventional OF typically manifests between the second and fourth decades of life, with a female predilection. Juvenile Trabecular Ossifying Fibroma (JTOF) and juvenile Psammomatoid Ossifying Fibroma (JPOF) are the two variants of COF that develop predominantly in children and have a distinct histological pattern. Both variants tend to manifest in the craniofacial bones, particularly the paranasal sinuses and gnathic bones. Sinonasal presentation of juvenile OF is characterized by an aggressive clinical behavior, rapid growth, significant bone expansion and a high recurrence risk. Maxillary sinus lesions have a tendency to extend into the paranasal sinuses and orbit, demonstrating the aggressive clinical pattern described for juvenile OF. The radiographic appearance of OF depends on the stage of lesion maturity. Initially, OF may be represented radiographically as a round, unilocular radiolucency with a well-defined margin. Mature lesions may exhibit a distinct radiolucent-radiopaque mixture or they may be entirely represented by a well-defined radiopaque mass. OF may occasionally exhibit cortical bone displacement, tooth displacement and root resorption. Ramos-Perez et al. presented a case of OF that resembled chronic apical periodontitis closely. In their case report, conventional radiographs revealed a well-defined unilocular

radiolucent lesion in the apical region of an endodontically treated right mandibular canine. After adequate root canal treatment, OF has been suggested as a differential diagnosis for unusual or persistent apical radiolucencies. The authors also reported that this similarity between OF and apical periodontitis is extremely uncommon. For OF diagnosis, clinical and radiographic characteristics are essential. To ensure definitive identification, correlation must be made between these characteristics and the histopathological presentation of the lesion. Therefore, a biopsy is essential for the diagnosis of OF. Histological examinations of conventional OF reveal cells resembling fibroblasts that are associated with the deposition of cementicles, osteoid and woven bone. Frequently, a prominent osteoblastic rim is observed adjacent to this mineralized substance. The pathological specimen contains fragments of a fibrous capsule. In cases of conventional OF, curettage and enucleation are recommended, with low rates of recurrence reported after these surgical procedures. Surgical resection may be the treatment of choice for juvenile OF. In cases involving the sinuses, lesions are incompletely excised, resulting in increased recurrence rates.

Cementoblastoma is a rare mesenchymal/ectomesenchymal benign odontogenic tumor. This tumor primarily affects adults in their second and third decades of life, regardless of gender. Cementoblastoma is associated with the root of a tooth, primarily in the mandibular permanent first molars. Nonetheless, it may also be found in other quadrants. This condition is characterized clinically by pain and swelling of the buccal and lingual aspects of the alveolar ridges. Although vitality tests may indicate that the affected tooth is alive, percussion may cause discomfort. Cementoblastoma radiographically manifests as a round, well-defined, radiopaque mass connected to a tooth by a thin radiolucent rim. This lesion may also be associated with root resorption, root canal invasion, tooth displacement and periodontal ligament space obliteration. Microscopically, cementoblastoma presents dense masses of acellular cementum-like material with basophilic reversal lines in a fibrous stroma. The mass of the lesion fuses with the tooth's root. Cementoblastoma must be distinguished from other mineralized tumors based on correlation with clinical and radiographic findings as well as a biopsy. Surgical excision and tooth extraction are recommended because of the fusion of the lesion to the root cementum. Cementoblastoma typically has a low recurrence risk. In the event of an early diagnosis, root canal therapy and apicectomy of the affected root may be considered to save the tooth.

Calcifying Cystic Odontogenic Tumor (CCOT) (calcifying odontogenic cyst) (calcifying odontogenic cyst) (Gorlin cyst) was reclassified according to the 2005 World Health Organization classification of odontogenic tumors as a rare benign odontogenic neoplasm. This condition is typically diagnosed in the second and third decades of life, regardless of gender. CCOT can be found in the maxilla and mandible, with a preference for the anterior region, particularly the incisor and canine regions. This tumor is typically a painless, slow-growing, bone-expanding lesion that may be associated with an unerupted tooth due to its occasional involvement with the crown of a tooth. On radiographs, CCOT may appear as a well-defined unilocular radiolucent lesion with irregular radiopaque material of varying radiodensities, described as a salt-and-pepper pattern of flecks. In some instances, CCOT can mimic apical periodontitis by manifesting as a well-defined radiolucent periapical round lesion. These lesions, unlike apical periodontitis, are unrelated to pulp necrosis, but may be associated with an infected root canal. CBCT can be a useful diagnostic tool for CCOT because the mineralized content of this tumor can be observed in CBCT examinations of the lesion's periphery. A biopsy is required to diagnose CCOT. The histopathological examination reveals that the cyst wall is composed of connective tissue and lined with thin odontogenic epithelial cells with ameloblastomatous characteristics. These cells are frequently associated with the formation of ghost cells and in some areas, calcification. In some lesions, eosinophilic dentinoid or dysplastic dentin can be observed. The enucleation of CCOT is advised and few recurrence are reported.

Ameloblastic Fibro-Odontoma (AFO) is a rare, benign, slow-growing tumor composed of odontogenic ectomesenchyme resembling dental papilla and containing epithelial strands and nests resembling dental lamina, enamel organ, dentine and enamel. This tumor typically affects young patients between the ages of 5 and 17, with no apparent gender preference. AFO is a clinically painless swelling that causes a delay in tooth eruption, tooth displacement or even tooth loss. This tumor typically affects the posterior mandible or posterior portion of the maxilla and may result in an obvious bony expansion in these areas. On conventional radiographs, AFO appears as a well-defined radiolucent area containing radiopaque material of varying sizes and shapes, affecting primarily the crown of an unerupted tooth or the periapical region. A large amount of radiopaque material may be present and an AFO may closely resemble an odontoma. AFO is diagnosed based on the presence of a cell-rich primitive ectomesenchyme resembling dental papilla, permeated cords and nests of odontogenic epithelium with ameloblastic differentiation. Typically, mesenchymal cells have a stellate or spindle-like appearance. AFO specimens display dentinoid and enamel matrix structures with varying degrees of maturation. Due to the rarity of this entity, there is no consensus regarding its management. Apparently, curettage of the affected tooth is sufficient for the treatment of small tumors. Curettage or enucleation may be indicated for the treatment of large tumors, particularly when it is possible to preserve the basal cortical bone and recurrences are not often reported.

Adenomatoid Odontogenic Tumor (AOT) is an uncommon, benign, slow-growing odontogenic tumor composed of odontogenic epithelium. AOT is typically asymptomatic and identified primarily in the second decade of life, with half of cases occurring during adolescence and there is a slight female predilection. This tumor is associated with swelling, tooth impaction and tooth displacement. Three clinicopathologic variants of AOT exist: Follicular, extrafollicular and peripheral extraosseous type. The follicular

type is linked to the crown of an unerupted tooth, particularly the maxillary canine. In the periapical region of the anterior mandible, the extrafollicular type may exist and tooth displacement may occur. Initial AOT may appear on radiographs as a well-defined unilocular radiolucency, which may be associated with the crown or present in the interradicular region of a tooth, lateral to the root or apex region, mimicking apical periodontitis. Despite this condition's apparent similarity to apical periodontitis, AOT typically displays an intact periodontal ligament space and lamina dura, which could be a useful parameter given that inflammatory periapical lesions typically result in periapical alterations. Depending on the amount of intralesional calcified deposits and the radiographic technique used, the presence of fine calcifications in the interior of a radiolucent lesion may be observed. This tumor is composed of odontogenic epithelial cells with cuboidal or columnar morphology that form nests or rosette-like structures on a histological level. Some of these tumors may have a partially cystic and partially solid epithelium. Visible are spaces resembling conduits lined by a row of columnar cells with polarized nuclei and amorphous nodules containing amyloid-like and calcified substances. In these lesions, dysplastic structures or calcified osteodentin may be present. After histopathological confirmation, AOT must be treated with surgical excision and the prognosis is typically favorable.

Osteosarcoma is a primary malignant bone tumor characterized by osteoid or bone matrix production. The majority of osteosarcomas affect long bones, with only 5% to 13% affecting the jaws. Maxilla and mandible are equally affected, with a predominance among patients between the ages of 10 and 20 and a slight male predilection. Osteosarcomas of the maxilla and mandible typically develop one to two decades later than osteosarcomas of the long bones and mandibular tumors are more prevalent in the molar region. Clinically, these lesions are distinguished by an obvious swelling of the affected area, with some cases accompanied by pain, tooth loosening or paresthesia. Some osteosarcoma patients have a history of increased tumor growth after tooth extraction or a history of preexisting conditions such as prior radiation, fibrous dysplasia, Paget's disease and chronic osteomyelitis. In radiographic examinations, osteosarcoma may reveal an image that ranges from sclerotic to radiolucent/radiopaque as the density varies based on the degree of tumor differentiation or disease stage. The alveolar ridge surrounding the root of a mandibular molar exhibited a well-defined, high-density alteration in a recent case of osteosarcoma resembling benign cementoblastoma. In the early stages of osteosarcoma, a widened periodontal ligament may be the only radiographic sign of the lesion. However, some osteosarcomas are associated with the loss of periodontal space visualization and the destruction of the cortical plate. The osteosarcoma may exhibit a sunray pattern, which may correspond to a parosteal osteogenic reaction to the tumor's spread however, this sign is not diagnostic. Due to the osteosarcoma's variable radiographic characteristics, histopathological examination is essential for its diagnosis. Osteosarcomas are classified microscopically as osteoblastic, chondroblastic and fibroblastic subtypes. Osteoblastic osteosarcoma consists of an osteoid surrounded by fibroblast-like cells arranged irregularly. A chondroblast subtype is characterized by lobules of feathery-looking tumor cells and atypical chondrocytes in the lacunae. There are spindle-shaped tumor cells arranged in a herringbone pattern and areas of osteoid formation in fibroblastic osteosarcoma. No consensus exists regarding the treatment protocol for osteosarcoma of the jaw, however, surgical resection with tumor-free margins appears to be the primary modality associated with improved survival rates whereas the benefits of radiochemotherapy are debated. In comparison to its manifestation in long bones, osteosarcoma of the jaw has significantly higher survival rates and distant metastases occur less frequently.

Chondrosarcoma is a malignant disease with a cartilaginous formation that accounts for 10%-20% of all primary malignant bone tumors, making it, after osteosarcoma, one of the most prevalent primary malignant bone tumors. Less than one percent of chondrosarcomas involve the jaws. It is more prevalent in men, typically in their forties. Chondrosarcoma of the jaw manifests clinically as a slow-growing, painless swelling that occurs most frequently in the anterior maxilla and mandible, particularly in the symphysis, coronoid and condylar processes. Chondrosarcoma radiographic characteristics may range from a completely osteolytic lesion to a poorly defined radiolucent area with calcified radiopaque foci. The calcified regions may have a mottled appearance with varying densities. Usually, this condition exhibits characteristics consistent with malignancy, particularly in terms of its ill-defined borders. In initial cases of chondrosarcoma in tooth-bearing areas, a widened periodontal space mimicking periapical inflammatory conditions and even osteosarcoma may be observed. A CT scan may be useful for assessing tumor extension and analyzing cortical and soft tissue degeneration. Chondrosarcoma must be diagnosed with the aid of a histopathological examination and a biopsy. On the basis of cell density and nuclear changes in neoplastic chondrocytes, conventional chondrosarcomas are classified as grade I, grade II or grade III. These tumors consist of round to oval cells in lacunae permeating a chondroid matrix with potential myxoid alterations. Despite the histological diversity of chondrosarcomas, conventional and mesenchymal subtypes are regarded as the most prevalent in the jaws. Hyaline cartilage and small round to oval cells are frequently arranged in a pericytomatous vascular configuration in mesenchymal chondrosarcomas. Surgical resection with a wide margin devoid of cancer is regarded as the most effective treatment for this tumor. Typically, radiotherapy and chemotherapy are used to treat high-grade cases, but they do not appear to have a significant impact on survival rates.

Head-and-neck metastases constitute 1% of oral cancers. Metastatic tumor cells rarely colonize the mouth and when they do, this indicates widespread disease. Oral metastases were the first sign of metastatic spread in 25% of cases and constitute a distant cancer with undiagnosed primary site in 23%. Oral metastatic lesions can affect any site in the oral

cavity, but in the jawbones, the mandible is more commonly affected than the maxilla, with the molar region being the most common site, followed by the premolar and angle-ramus. The mandible may be preferred because it has more hematopoietic tissue with sinusoidal vascular spaces that allow tumor cell access. The mandible's blood supply relative to the maxilla may also cause mandibular preference. Hematopoietic bone marrow, branching blood vessels and slow blood flow make jawbones susceptible to neoplastic cell deposition. Oral metastatic lesions have gender-specific primary tumors. Lungs, kidneys, livers and prostates are men's primary sites whereas female primary sites include the breast, genital organs, kidney and colorectum. Prostate and breast cancer metastasize to the jawbone. The clinical manifestations of oral cavity metastatic tumors include jaw pain, exophytic lesion (swelling or mass that may be ulcerated), paresthesia and numbness, as well as deceptive manifestations such as toothache, dentoalveolar swelling and loose tooth. The latter signs and symptoms may prompt clinicians to consider the possibility of an odontogenic disease. The radiographic signs vary and may combine both radiolucent and radiopaque elements, thus rendering difficult to place a definite diagnosis without a biopsy. Metastatic lesions lack radiographic pathognomonic features. Metastatic disease appears as a radiolucency "moth-eaten" by destructive cells on radiographs. Pure and mixed osteolytic-osteoblastic metastases are rare. Certain tumors can induce reactive new bone formation and create a mixed radiopaque (osteoblastic metastasis) and radiolucent (fibro-osseous) lesion. Metastatic breast and prostate cancer exhibit this pattern. New bone spicules radiate away from the cortex in the sunburst subtype of periosteal reaction. Periosteal reaction depends on pathology severity, virulence and duration. The periosteum reacts continuously and solidly to slow-growing diseases. The periosteum cannot produce new bone as fast as a rapidly growing bone lesion. Thus, bone formation is discontinuous. The rapid growth of pathologic processes in the bone matrix subperiosteal space hinders confluent cell element maturation. Matrix deposits along Sharpey's fibers along periosteal vessels support the periosteum and maintain its relationship with the host bone cortex. Sunburst periosteal reactions can be caused by osteosarcoma, metastasis (especially from the sigmoid colon and rectum), Ewing's sarcoma, haemangioma, meningioma, tuberculosis, tropical ulcer or fibrous dysplasia.

Finally, renal osteodystrophy (osteitis fibrosis) is a metabolic bone disorder caused by mineral and bone disorders associated with end-stage renal disease, secondary hyperparathyroidism and calcitriol deficiency. These metabolic alterations result in a broad clinical syndrome characterized by abnormalities in calcium, phosphorus, PTH and vitamin D metabolism, bone turnover and mineralization. The involvement of the jaw is relatively common and one of the earliest indicators of renal disease. Clinically, renal osteodystrophy is characterized by a painless, diffuse or localized enlargement of the maxilla and mandible, which is characterized radiographically by a mixed radiolucent/radiopaque lesion with an abnormal trabecular configuration, a diffuse ground-glass pattern and loss of lamina dura. Combining clinical, radiographic and histopathological findings is required to diagnose renal osteodystrophy. This condition is characterized histologically by trabeculae of woven bone of varying sizes, numerous osteoblasts on the bone surface and an abundance of osteoclastic resorption lacunae. Additionally, a prominent fibrous tissue is visible surrounding the trabecular bone. The most common treatment option for renal osteodystrophy is surgical recontouring. Additionally, parathyroidectomy has been described as an effective treatment for renal osteodystrophy.

The effectiveness of root canal therapy is measured by the absence of pain, the absence or reduction of periapical lesions and the restoration of tooth function In endodontic practice, the diagnosis of periapical lesions is difficult because many of these lesions can mimic endodontic lesions, even when derived from different sources. Inflammatory alterations in the periapical bone structure may result from an infection of the root canal system and osteoclastic or osteoblastic activity. The presence of radiolucent or radiopaque images in radiographic examinations enables the detection of these alterations. Similarly, lesions of inflammatory, neoplastic, dysplastic or metabolic origin may also exhibit such alterations in bone. Although radiolucent lesions are more commonly associated with root canal infection, radiopaque lesions are also associated with equally speculative causes and require the same care to avoid misdiagnosis. Clinical factors, such as pulp sensitivity and vitality tests, are typically useful in determining whether periapical changes are inflammatory or non-inflammatory in nature.

CONCLUSION

For the diagnosis of periapical lesions in this scenario, a comprehensive clinical examination, in conjunction with clinical and radiographic aspects of the disease, is essential. Even though the absence of pulp response suggests that a periapical lesion may be of endodontic origin, this finding may coincide with the presence of a non-endodontic lesion in some instances. When clinical and radiographic findings are insufficient to identify the origin of a periapical lesion, a biopsy followed by histopathological analysis is necessary. CBCT is also an important diagnostic tool in dentistry as it allows for more precise detection of periapical lesions than periapical and panoramic radiography. The characteristics of CBCT and its map-reading strategy may allow for a comprehensive examination of a multidimensional structure, thereby elucidating the precise location of the periapical lesion, the bone resorption or bone formation characteristics and the presence, absence or regression of the lesion.

In conclusion, the endodontist must know how to diagnose radiopaque periapical lesions. Knowing how to differentiate between inflammatory, non-inflammatory, neoplastic and metabolic lesions expedites diagnosis and ensures the selection of the appropriate treatment.

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