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PATHOLOGY IN RELATION TO HEAD AND NECK REGION OBLITERATING THE PRACTICE OF ORTHODONTICS AND DENTOFACIAL ORTHOPAEDICS AND PERIODONTOLOGY PERTAINING TO PAEDIATRIC AND ADULT POPULATION- A REVIEW

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ABSTRACT

Many youngsters and young people will receive their first complete jaw imagining from an orthodontist. About 6% of radiographs used to plan orthodontic treatments will show additional anomalies, some of which might need to be managed surgically or have an impact on the patient's overall health. This overview attempts to identify prevalent illnesses that manifest in children, young adults, and adults, as well as less frequent but significant disorders that an orthodontist may encounter.

Keywords - orthodontics, dentofacial, orthopaedics, infection, pathology

INTRODUCTION

Periapical granuloma

The most common type of specimens reported to oral pathology laboratories that originate in the jaws are periapical granulomas.[6,7] Although they seldom exhibit symptoms, they can.[8] The lesions will manifest as a well-defined radiolucency at the tooth apex and be connected to a nonvital tooth.[8] There will be disruption to the linked tooth's lamina dura.[8] Although it might be challenging to distinguish between a radicular cyst and a periapical granuloma radiologically, histologic evaluation is important since bigger lesions are more likely to be cysts.[9.] When examined under a microscope, a periapical granuloma is composed of granulation tissue and an inflammatory infiltration that may contain several types of

lymphocytes, macrophages, polymorphonuclear leukocytes, and plasma cells.[8] Lesion resolution usually involves extraction of the nonvital tooth or effective endodontic therapy.

Radicular cyst

The most prevalent odontogenic cysts in both adults and children are radicular cysts. [6, 7.] Chronic inflammation from the apex of a nonvital tooth—which might be either a main or secondary tooth—is the source of these cysts.[11] The majority don't hurt, but in cases where an infection has arisen, they could, and on rare occasions, the cysts might enlarge to the point where the jaws enlarge.[12] On radiographs, they show up as well-defined, frequently corticated circular radiolucencies that continue between the cyst and the impacted tooth's dura.[12] Root resorption might result from them.[13] A microscopic inspection reveals a nonkeratinized stratified squamous epithelium lining an inflammatory fibrous cyst wall.There may also be [12] cholesterol clefts visible.[12] The procedure involves extracting the cyst and treating the tooth that is causing the inflammation.

Dentigerous cyst

With a lining made of the thinned enamel epithelium, dentigerous cysts are developing cysts that encircle the crown of an unerupted tooth. Their pathophysiology is unknown.[12] Across all age categories, they are the most prevalent developing cyst of the jaws.[5-7, 10–12] The most often impacted teeth are the mandibular third molars, which are followed by the maxillary third molars.[12] The canines and second premolars follow in a pattern that is consistent with the distribution of unerupted teeth.[12] These lesions are usually asymptomatic, but if they are big, they can cause discomfort, edema, or infection.[14] They show as well-defined, corticated, unilocular radiolucencies connected to the crown of an unerupted tooth on radiographic examination.[12,14] They may result in root resorption as well as tooth displacement.Thirteen A thin, nonkeratinized epithelial lining and a fibrous cyst wall will be visible upon histologic inspection of the cyst; yet, these cysts have the potential to inflame, which would give the

lining characteristics akin to those of a radicular cyst.[12] Therefore, to diagnose a dentigerous cyst, a link between the cyst and tooth crown must be established, either by radiography or at the macroscopic pathologic examination. In most situations, treatment consists of enucleating the cyst and extracting the related tooth.

Odontomas

All types of dental tissue, such as enamel, dentine, cementum, and tooth pulp, are hamartomas known as odontomas.[17] There are two types of them: complicated and compound. Dental tissues are arranged into structures resembling teeth in compound lesions, however they are haphazardly arranged in a complicated odontoma.[18] Although they are less frequent in adults, odontomas are the most prevalent solid odontogenic lesion in children[6,19.7]. Most cases include patients who are under 20 years old.[19] These lesions are typically accidental discoveries, but they might potentially be the result of delayed permanent tooth emergence, which is a frequent cause for referral to an orthodontist.[18, 19. 9]. They show up as well-defined radiopacities on radiographic imaging, occasionally closely linked to the crown of an erupting tooth. On radiographs, complex odontomas exhibit an erratic calcification pattern, resulting in the formation of anomalous structures resembling teeth.[19] Rarely, both might result in root resorption.[19] Both compound and complicated odontomas have comparable histological characteristics. Both typically have different quantities of pulp, cementum, enamel, dentine, and odontogenic epithelium surrounded by a fibrous capsule.[18] Following local excision, recurrence is improbable.[18.] They show up as well-defined radiopacities on radiographic imaging, occasionally closely linked to the crown of an erupting tooth. On radiographs, complex odontomas exhibit an erratic calcification pattern, resulting in the formation of anomalous structures resembling teeth.[19] Rarely, both might result in root resorption.[19] Both compound and complicated odontomas have comparable histological characteristics. Both typically have different quantities of pulp, cementum,

enamel, dentine, and odontogenic epithelium surrounded by a fibrous capsule.[18] Following local excision, recurrence is improbable.[18]

Odontogenic Keratocyst

Patients may, however, first present with discomfort, sinus development, or swelling in the jaw.[20–22] These cysts tend to be found more posteriorly and are more prevalent in the mandible than the maxilla.[20–22] From a radiological perspective, they manifest as distinct, corticated radiolucencies that can be either unilocular or multilocular.[12,20] They can result in resorption or root displacement[12] and are frequently characterized as having scalloped borders.[13,20] Before deciding to enucleate these lesions, a biopsy for histologic evaluation is a useful tool because these cysts frequently have an ameloblastoma differential diagnosis.

These lesions have a thin, parakeratinized, stratified squamous epithelium surrounding an uninflamed, fibrous cyst wall, with palisading of the basal cells.[12]

Exostoses

Jaw bone exostoses are quite prevalent, yet the frequency as reported varies greatly.[24] They are the most often encountered bone disease in the 35–65 year old age range, although they may also be seen in children. [24] Depending on where they occur, they are categorized as buccal exostoses, torus palatinus, or torus mandibularis. Clinically, they show as protuberances of the hard tissue of the mouth and jaws.[8,24] Although they are not frequently sent for histologic analysis, when they are, they will show up as typical cancellous or cortical bone.[8,24] They are only taken out when they are creating problems, as when a denture or detachable device is being fitted. When wounded, bony exostoses may ulcerate and reveal the underlying bone.

Giant cell granulomas

The involvement of bone sets peripheral giant cell granulomas apart from central ones, which are both reactive lesions with similar histologic characteristics. Although they are rare,

central giant cell granulomas are more prevalent in children and young adults than in older persons.[26] The mean age of presentation for the peripheral variant is older and it is more prevalent overall.[6, 7, 27.] Teeth displacement may result from the central giant cell granulomas, which often manifest as a painless enlargement of the jaws and perhaps a blue to purple soft-tissue extension.[26] On the other hand, peripheral forms are found outside of the bone and manifest as soft polypoid lesions that are red, blue, or purple. They usually affect the gingivae and are more common on the mandibular gingivae.[27]

Inflammatory collateral cysts

3% in children and 5% of all odontogenic cysts in adults[5,28.5.] Although they have been given several names throughout the years, depending on where the cyst is situated, they are often classified as either paradental cysts or buccal bifurcation cysts.[5, 28–29] While buccal bifurcation cysts are seen on the buccal aspect of the first or second molars, parantal cysts are linked to the distobuccal aspect of the third molars.[17] With a mean age of presentation of 26 years for paradental cysts and 17 years for buccal bifurcation cysts, they often affect younger individuals.[17] While paragonal cysts frequently show no symptoms, paragonal bifurcation cysts can exhibit signs of pericoronitis.[17, 28] Even if a radiograph's appearance varies, if it can be identified on one, it shows the tooth's crown.[28, 29] Clinic-pathologic correlation is necessary since the histologic appearance of an inflammatory collateral cyst is nearly identical to that of a radicular cyst, as previously discussed. When paradental cysts are linked to third molars, the usual course of treatment is enucleation together with the extraction of the related tooth; however, if the cyst is linked to other teeth, the likelihood of enucleation along with the preservation of the tooth increases.[28]

Nasopalatine cyst

The nasopalatine duct cyst is one example of a nonodontogenic cyst that can develop in the jaws. Although they can occur in youngsters as well, these cysts are more common in adults.[6, 7, 30] Many individuals show no symptoms at all, while some have swelling, fluid leakage, or

discomfort.[30] They are restricted to the anterior maxilla's midline, where the nasopalatine duct's remains are located. They are well-defined, symmetrical radiolucencies in the anterior palate that have the potential to dislodge teeth.[17] They maintain the lamina dura of neighboring teeth and have a typical diameter of 17 mm, while they can go as big as 60 mm[30.17] Verifying the health of the teeth next to the cyst is helpful since in cases with nasopalatine duct cysts, the teeth will remain viable. Recurrence is rare and enucleation is a curative procedure.

Ameloblastoma

Ameloblastoma is the most prevalent odontogenic tumor in adults and the second most frequent in children, with 15% of cases occurring in people under the age of twenty.[31] Two forms exist: Ameloblastoma is the most prevalent odontogenic tumor in adults and the second most frequent in children, with 15% of cases occurring in people under the age of twenty.[31] There are two types: unicystic ameloblastoma, which creates a single cyst, and conventional ameloblastoma, which is a solid tumor.17 50% of cases of unicystic ameloblastomas occur in the second decade, with younger patients often presenting.[17] This is particularly true if there is a cyst present along with an immature tooth. On the other hand, the fourth and fifth decades are when the incidence of conventional ameloblastomas peaks.[17] Conventional ameloblastomas are often tumors that are aggressive locally. Ameloblastomas can develop anywhere in the jaws, however they are most frequently found in the posterior mandible.[18, 31] The most typical symptom is a painless jaw enlargement that allows for tooth movement.

The typical form of ameloblastoma manifests as a multilocular or unilocular radiolucency that is well defined and corticated,[17,18] whereas unicystic ameloblastoma is unilocular.[17] They could also result in root resorption and jaw enlargement.[13, 17, and 18] Conventional ameloblastomas have a variety of histologic appearances, with islands of cells resembling stellate reticulum in the center encircled by a layer of ameloblast-like cells on the periphery

.[17, 18] The unicystic type has an upper epithelial layer that resembles a stellate reticulum, a lining made up of palisading basal cells, and a fibrous cyst wall.[17] Depending on the size of the tumor, excision or resection is the method used to treat traditional ameloblastoma.[31–33] Reconstruction of the surgical defect is necessary for bigger tumors.

These tumors frequently recur, particularly when more cautious treatment is used.[18, 31, and 33] Unicystic ameloblastomas may typically be enucleated and are less aggressive.

Adenomatoid odontogenic tumor

The most frequent odontogenic tumor in children and young adults is an adenomatoid odontogenic tumor (AOT), which presents 80% of the time before the age of 30 and half of the time before the age of 20.[18] Cystic lesions are related with an unerupted canine teeth in 70% of patients, albeit these lesions might present with different appearances, such as an extraosseous peripheral variety.[34] Two thirds of adenomatoid odontogenic tumors arise in the maxilla, namely the anterior maxilla, and are more prevalent in female patients.[17, 18, 34] The majority of lesions are asymptomatic[34] and might be discovered by accident or as a result of a tooth's delayed eruption. A distinct, frequently corticated unilocular radiolucency can be seen on radiographs, which is typically connected to the crown of an erupting tooth.[17, 18] There might be calcifications in the radiolucency.

Ameloblastic fibroma

Despite being a rare odontogenic tumor, ameloblastic fibroma primarily affects children and young people, with 80% of cases identified before the age of 22.[36] Their first symptom is generally jaw enlargement, although delayed tooth eruption can also be the first, and 12% of discoveries are unintentional.[36] Compared to other locations, the posterior mandible is where they are more prevalent.[17, 18, 35, 36] These lesions can be as big as 16 cm, with a typical size of about 4 cm [36.35] They are well defined radiolucencies from a radiological perspective, and they can be unilocular or multilocular.[18, 35, 36] They could also exhibit root resorption and tooth displacement.[17] These lesions show as cords of bilayered odontogenic tissue under the

microscope. oral papilla-like epithelium inside a myxoid cellular stoma.[17, 18] Usually, enucleation is used to cure malignancies; however, excision may be necessary for more aggressive or recurring cancers.[17, 18, 35, 36] Recurrence rates are reported to vary, with conservatively treated tumors having a higher recurrence rate.[17, 35, 36] Ameloblastic fibroma malignant transformation is quite uncommon.[36]

Fibrous dysplasia

In 80% of cases, fibrous dysplasia, a developmental bone abnormality, affects just one bone, most frequently the craniofacial bones.[17, 37] When several bones are impacted, it can be a sign of McCune-Albright syndrome, a condition that also causes pigmentation in the café au lait and endocrine abnormalities including early puberty in its sufferers.[37] The onset of fibrous dysplasia can be identified in children, however the condition is more frequent in young people.[6,38] The majority of patients initially have unilateral jaw involvement, while bilateral lesions are sometimes possible.[37, 38] Furthermore, maxillary lesions are more prevalent.[37, 38] Usually, the sole issue is bone enlargement.[37,38] with fewer frequent symptoms such as discomfort or tooth movement.[38] The lesion's radiographic appearance changes as it progresses, first showing as radiolucency.[38] As they become older, though, their appearance becomes more hazy and their borders become less distinct, making them more radiopaque.[37, 38] Histologic examination of these lesions reveals thick fibrousstroma filled with juvenile, randomly arranged strands of bone with varying mineralization.[37, 38] Treatment for fibrous dysplasia is typically postponed until skeletal maturity since the condition grows with the patient.[38] Conservative surgical debulking is frequently utilized to enhance look and function.

Cemento-ossifying fibroma

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becomes more hazy and their borders become less distinct, making them more radiopaque[37, 38] Histologic examination of these lesions reveals thick fibrous stroma filled with juvenile, randomly arranged strands of bone with varying mineralization.[37, 38] Treatment for fibrous dysplasia is typically postponed until skeletal maturity since the condition grows with the patient.[38] Conservative surgical debulking is frequently utilized to enhance look and function. POF won't be covered in detail here because it often manifests in the paranasal sinuses or orbital bones[17,37.] The main symptom that shows up is jaw swelling that is painless.[17,39,40,] which may be faster for JTOF but often sluggish for CCOF.[17] All of these benign neoplasms show up on radiographs as well-defined, frequently corticated lesions with a mixture of radiolucency and radiopacity.[17, 37, 39, and 40] There is also a chance of tooth displacement and root resorption.[17, 37] When CCOF is examined under a microscope, it reveals encapsulated cellular fibrous connective tissue with woven bone that is occasionally less well-formed and appears as spheres or isolated trabeculae.[17,3] The calcifications within JTOF are more fully developed, with strands and anastomosing trabeculae of osteoid and woven bone lined with osteoblasts, while sharing a comparable fibrous connective matrix.[17, 37, 39.] Recurrence of CCOF is uncommon and is typically treated with enucleation and curettage.[17]

JTOF is treated with enucleation or resection; nevertheless, up to 21% of individuals have recurrence, which is more common following enucleation.[17]

Langerhans cell histiocytosis

Myeloid dendritic cell clonal expansion, or Langerhans cell histiocytosis, is not cancerous but can be fatal.[17, 42, 43] While adults may sometimes have symptoms, children are the primary population affected by this disorder, with a median diagnostic age of 3.5 years.[43] As it affects five out of every million children, it is uncommon overall.[17,43] This condition can affect one or more organs in a single system, or it might impact several organs in many systems. organs in play.[17,43] Therefore, with multisystem illness, a patient may appear with a wide range of symptoms, especially when the head and neck is the only the impacted site.

The mandible is more often impacted than the maxilla in radiographic appearances, which show an ill-defined, unilocular radiolucency that may be single or multiple[17,42.42] Teeth often seem to be radiolucent and to be "floating." Histopathologically, the lesion consists of a damaging infiltration of Oval nuclei with grooves, folds, or indentations give Langerhans cells their coffee bean-like appearance. look.There are also a variety of chronic inflammatory cells visible, along with eosinophils.[17] Patients with single-system diseases receive more conservative treatment since they have favorable results with a survival rate close to 100%.[43] While some lesions may heal on their own, others could require chemotherapy or enucleation.[42, 43] However, chemotherapy is frequently needed for multisystem illness since it can be fatal.[43]

Osteosarcoma

Although it manifests a little later than extragnathic osteosarcoma, cranial osteosarcoma is still most common in young people, with a mean age in the mid-30s.[44–46] The most prevalent clinical signs are edema, which can also include ulceration and accompanying discomfort.[17,45] The mandible is impacted more frequently than the maxilla [44, 45] Osteosarcomas can show differently on radiography; the majority present as mixed both radiopacities and radiolucencies.[17] Supracrestal bone deposition and asymmetrical expansion of the periodontal ligament gaps are two modest characteristics that raise the possibility of osteogenic malignancy. The most severe lesions will exhibit soft-tissue expansion, periosteal responses, and the destruction of nearby tissues.[17] Although there are several subtypes and a varied histologic appearance, the most frequent discovery is of extremely abnormal cells that create osteoid, an immature bone-like substance.[17,45] Most patients are treated with surgery alone, while some also undergo radiation or adjuvant chemotherapy.[44, 45] Compared to their extragnathic counterparts, these tumors had a superior 10-year survival rate of about 60%.[44]

Ewing sarcoma

Ewing sarcoma often affects children and young people (ages 46–48), with 50% of cases occurring under the age of 18.[17] Younger individuals are most affected when it comes to grownups.[17,46] Roughly 50% of cases of Ewing sarcoma involve the craniofacial bones, while the other cases impact the soft tissues. Up to 9% of cases occur in the head and neck.[17] The jaw is impacted more frequently than the maxilla.[47,48] The first publication of discusses soft-tissue Ewing sarcoma this sequence.[4] The Ewing sarcoma's clinical characteristics in the jaws include paresthesia, discomfort, edema, tooth movement, and maybe fever.[46–48] These lesions show up radiologically as weakly defined radiolucencies that destroy neighboring structures including teeth and bone cortices.[46–48] These tumors resemble sheets and clusters of tiny, spherical cells with little cytoplasm, and in contrast to other malignant tumors, they often do not exhibit Pleomorphism.[17] Finding rearrangements in the EWSR1 gene can help with diagnosis.[17] Radiation therapy, chemotherapy, and surgery are used in combination as a kind of treatment.[49] The Only 35% of patients with metastatic cancer had a 5-year survival rate, compared to 73% for local disease.[46]

Effect on orthodontic treatment and Management with Examples

1. Management:

   Confirmation, 6 elimination of cause, 6 recommendation

   2. Confirmation, recommendation, follow-up, or medicine

   3. Referral, basic excision, and six cause removal

2. Examples

   1. Geographic tongue and frictional keratosis

   2. Herpes simplex virus infection, candidiasis, recurrent aphthous stomatitis, exostoses, orofacial granulomatosis, and haemangioma

   3. Radicular cyst, dentigerous cyst, inflammatory collateral cyst, nasopalatine cyst,

odontoma, fibrous hyperplasia, pyogenic granuloma, squamous papilloma, lipoma, periapical granuloma, and mucocele

- Orthodontia therapy may need to be discontinued or resumed.

3. Management:

   1. Referral and eradication

   2. Referral: The course of treatment depends on the condition's severity, its underlying etiology, and its connection to the alveolar bone.

4. Examples

   1. Benign salivary gland tumors (such as pleomorphic adenoma), giant cell granuloma, ameloblastoma, ameloblastic fibroma, cemento-ossifying fibroma, and orthodontic keratocyst

   2. Fibrous dysplasia, gingival hyperplasia, and Langerhans cell histiocytosis

   - Very likely to be impacted and need orthodontic treatment to be stopped

5. Management:

   1. referring, removing, and six further therapies

6. Example

7. 1. malignant salivary gland cancers (such as mucoepidermoid carcinoma), squamous cell carcinoma, osteosarcoma, rhabdomyosarcoma, and Ewing sarcoma MRON

Medication-related osteoradionecrosis of the jaw

MRONJ is a nonhealing necrosis of the jaws that often develops following trauma and is treated with a drugs that change the way bones remodel, such bisphosphonates.50 While it is rare in patients receiving orthodontic treatment, individuals with Reports of orthodontics causing MRONJ are available.[50, 51] When the jawbone remains exposed for at least eight weeks without healing, antiresorptive medication therapy is history, and there is no healing, MRONJ is diagnosed. radiotherapy's past.[52] It is crucial to take a complete clinical and medication history in order to identify people who are at risk. If MRONJ is thought to be

present, a referral to an oral and an experienced maxillofacial surgeon is required for the intricate management of these individuals.

Conclusion

After closely examining the radiographs that were requested for the purpose of planning orthodontic treatment, many early and lesions that show no symptoms might be found. Additionally, a large number of the lesions mentioned have relocated teeth or delayed eruption, increasing the likelihood of an orthodontic evaluation. Therefore, having a basic understanding of the prevalent and significant disorders affecting the mandible will guarantee precise and prompt recommendation to the best possible management.

References

1. Kuhlberg AJ, Norton LA. Pathologic findings in orthodontic radiographic images. Am J Orthod Dentofacial Orthop 2003;123:182-4.

2. Cedro MK, Moles DR, Hodges SJ. Adult orthodontics—who's doing what? J Orthod 2010;37:107-17.

3. Keim RG, Gottlieb EL, Nelson AH, Vogels DS 3rd. 2013 JCO Orthodontic Practice Study. Part 1: trends. J Clin Orthod 2013;47:61-80.

4. Hankinson P, Brierley C, Brierley D. A review of oral pathology in orthodontics. Part 1: soft-tissue pathology. Am J Orthod Dentofacial Orthop 2024;165:7-17.

5. Jones AV, Craig GT, Franklin CD. Range and demographics of odontogenic cysts diagnosed in a UK population over a 30-year period. J Oral Pathol Med 2006;35:500-7.

6. Jones AV, Franklin CD. An analysis of oral and maxillofacial pathology found in children over a 30-year period. Int J Paediatri Dent 2006;16:19-30.

7. Jones AV, Franklin CD. An analysis of oral and maxillofacial pathology found in adults over a 30-year period. J Oral Pathol Med 2006;35:392-401.

8. Odell E. Cawson's essentials of oral pathology and oral medicine. London, England: Elsevier Health Sciences; 2015.

9. Mortensen H, Winther JE, Birn H. Periapical granulomas and cysts.An investigation of 1,600 cases. Scand J Dent Res 1970;78: 241-50.

10. Martin LHC, Speight PM. Odontogenic cysts: an update. Diagn Histopathol 2017;23:2605.

11. Delbem ACB, Cunha RF, Vieira AE, Pugliesi DM. Conservative treatment of a radicular cyst in a 5-year-old child: a case report.Int J Paediatr Dent 2003;13:447-50.

12. Martin L, Speight PM. Odontogenic cysts. Diagn Histopathol 2015; 21:359-69.

13. Teo KW, Shi AH, Teh LY, Lee AMH. External root resorption in common odontogenic cysts and ameloblastomas of the jaw: A retrospective radiographic study in an Asian population. Oral Surg 2021;14:335-41.

14. Motamedi MHK, Talesh KT. Management of extensive dentigerous cysts. Br Dent J 2005;198:203-6.

15. Nahajowski M, Hnitecka S, Antoszewska-Smith J, Rumin K, Dubowik M, Sarul M. Factors influencing an eruption of teeth associated with a dentigerous cyst: a systematic review and meta-analysis. BMC Oral Health 2021;21:180.

16. Koca H, Esin A, Aycan K. Outcome of dentigerous cysts treated with marsupialization. J Clin Pediatr Dent 2010;34:165-8.

17. WHO Classification of Tumours Editorial Board. Head and neck tumours [Internet; beta version ahead of print]. Lyon, French Republic: International Agency for Research on Cancer; 2022

18. Brierley DJ, Hunter KD. Odontogenic tumours. Diagn Histopathol 2015;21:370-9.

19. Philipsen HP, Reichart PA, Praetorius F. Mixed odontogenic tumours and odontomas. Considerations on interrelationship. Review of the literature and presentation of 134 new cases of odontomas. Oral Oncol 1997;33:86-99.

20. Chirapathomsakul D, Sastravaha P, Jansisyanont P. A review of odontogenic keratocysts and the behavior of recurrences. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;101:5-9: discussion 10.

21. Morgan TA, Burton CC, Qian F. A retrospective review of treatment of the odontogenic keratocyst. J Oral Maxillofac Surg 2005;63:635-9.

22. Myoung H, Hong SP, Hong SD, Lee JI, Lim CY, Choung PH, et al. Odontogenic keratocyst: review of 256 cases for recurrence and clinicopathologic parameters. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91:328-33.

23. Johnson NR, Batstone MD, Savage NW. Management and recurrence of keratocystic odontogenic tumor: a systematic review.Oral Surg Oral Med Oral Pathol Oral Radiol 2013;116:e271-6.

24. Antoniades DZ, Belazi M, Papanayiotou P. Concurrence of torus palatinus with palatal and buccal exostoses: case report and review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85:552-7.

25. Sonnier KE, Horning GM. Spontaneous bony exposure: a report of 4 cases of idiopathic exposure and sequestration of alveolar bone.J Periodontol 1997;68:758-62.

26. de Lange J, van den Akker HP, van den Berg H. Central giant cell granuloma of the jaw: a review of the literature with emphasis on therapy options. Oral Surg Oral Med Oral Pathol Oral Rad Endodontol 2007;104:603-15.

27. Katsikeris N, Kakarantza-Angelopoulou E, Angelopoulos AP. Peripheral giant cell granuloma. Clinicopathologic study of 224 new cases and review of 956 reported cases. Int J Oral Maxillofac Surg 1988;17:94-9.

28. Philipsen HP, Reichart PA, Ogawa I, Suei Y, Takata T. The inflammatory paradental cyst: a critical review of 342 cases from a literature survey, including 17 new cases from the author's files. J Oral Pathol Med 2004;33:147-55.

29. Chrcanovic BR, Reis BMMV, Freire-Maia B. Paradental (mandibular inflammatory buccal) cyst. Head Neck Pathol 2011; 5:159-64.

30. Swanson KS, Kaugars GE, Gunsolley JC. Nasopalatine duct cyst: an analysis of 334 cases. J Oral Maxillofac Surg 1991;49:268-71.

31. Ord RA, Blanchaert RH, Nikitakis NG, Sauk JJ. Ameloblastoma in children. J Oral Maxillofac Surg 2002;60:762-70.

32. Zhang J, Gu Z, Jiang L, Zhao J, Tian M, Zhou J, et al. Ameloblastoma in children and adolescents. Br J Oral Maxillofac Surg 2010; 48:549-54.

33. Huang IY, Lai ST, Chen CH, Chen CM, Wu CW, Shen YH. Surgical management of ameloblastoma in children. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;104:478-85.

34. Chrcanovic BR, Gomez RS. Adenomatoid odontogenic tumor: an updated analysis of the cases reported in the literature. J Oral Pathol Med 2019;48:10-6.

35. Chen Y, Wang JM, Li TJ. Ameloblastic fibroma: a review of published studies with special reference to its nature and biological behavior. Oral Oncol 2007;43:960-9.

36. Buchner A, Vered M. Ameloblastic fibroma: a stage in the development of a hamartomatous odontoma or a true neoplasm? Critical analysis of 162 previously reported cases plus 10 new cases. Oral Surg Oral Med Oral Pathol Oral Radiol 2013;116:598-606.

37. Speight PM, Carlos R. Maxillofacial fibro-osseous lesions. Curr Di-
agn Pathol 2006;12:1-10.

38. Davidova LA, Bhattacharyya I, Islam MN, Cohen DM, Fitzpatrick SG. An analysis of clinical and histopathologic features of fibrous dysplasia of the jaws: A series of 40 cases and review of literature. Head Neck Pathol 2020;14:353-61.

39. Slootweg PJ. Juvenile trabecular ossifying fibroma: an update. Virchows Arch 2012;461:699-703.

40. Su L, Weathers DR, Waldron CA. Distinguishing features of focal cemento-osseous dysplasia and cemento-ossifying fibromas. II. A Clinical and radiologic spectrum of 316 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84:540-9.

41. Albright JT, Topham AK, Reilly JS. Pediatric head and neck malignancies: US incidence and trends over 2 decades. Arch Otolaryngol Head Neck Surg 2002;128:655-9.

42. Xu J, Gilbert JR, Sutton KS, Goudy SL, Abramowicz S. Head and neck Langerhans cell histiocytosis in children. J Oral Maxillofac Surg 2022;80:545-52.

43. Krooks J, Minkov M, Weatherall AG. Langerhans cell histiocytosis in children: history, classification, pathobiology, clinical manifestations, and prognosis. J Am Acad Dermatol 2018;78:1035-44.

44. Baumhoer D, Brunner P, Eppenberger-Castori S, Smida J, Nathrath M, Jundt G. Osteosarcomas of the jaws differ from their peripheral counterparts and require a distinct treatment approach. Experiences from the DOESAK registry. Oral Oncol 2014;50:147-53.

45. Bennett JH, Thomas G, Evans AW, Speight PM. Osteosarcoma of the jaws: a 30-year retrospective review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;90:323-32.

46. Stewart BD, Reith JD, Knapik JA, Chi AC. Bone- and cartilage-forming tumors and Ewing sarcoma: an update with a gnathic emphasis. Head Neck Pathol 2014;8:454-62.

47. Margaix-Munoz M, Bag ~ an J, Poveda-Roda R. Ewing sarcoma of the oral cavity. A review. J Clin Exp Dent 2017;9: e294-301.

48. Wood RE, Nortje CJ, Hesseling P, Grotepass F. Ewing's tumor of the jaw. Oral Surg Oral Med Oral Pathol 1990;69:120-7.

49. Olson MD, Van Abel KM, Wehrs RN, Garcia JJ, Moore EJ. Ewingsarcoma of the head and neck: the Mayo Clinic experience. Head Neck 2018;40:1999-2006.

50. Woolley J, Akintola O, Yates J, Calasans-Maia MD, de Albuquerque Calasans-Maia J, Kocherhina I, et al. The risk of osteonecrosis of the jaw and adverse outcomes in patients using antiresorptive drugs undergoing orthodontic treatment: a systematic review.

Heliyon 2021;7:e05914.

51. Kalfarentzos E, Chliaoutakis A, Ntagiantis G, Vardas E, Christopoulos P. Orthodontic treatment as triggering factor of medication related osteonecrosis of the jaw in a breast cancer patient. Report of a rare case. J Clin Exp Dent 2023;15:e351-6.

52. Ristow O, Otto S, Troeltzsch M, Hohlweg-Majert B, Pautke C. Treatment perspectives for medication-related osteonecrosis of the jaw (MRONJ). J Craniomaxillofac Surg 2015;43:290-3.

53. Weltman B, Vig KW, Fields HW, Shanker S, Kaizar EE. Root resorption associated with orthodontic tooth movement: a systematic review. Am J Orthod Dentofacial Orthop 2010;137:462-76: discussion 412A.

54. Linge BO, Linge L. Apical root resorption in upper anterior teeth.Eur J Orthod 1983;5:173-83.

55. Elhaddaoui R, Bahije L, Chbicheb S, Zaoui F. Cervico-facial irradiation and orthodontic treatment. Int Orthod 2015;13:139-48.

56. Hameed O, Gwilliam J, Whaites E. Odontogenic keratocyst: an incidental finding during an orthodontic examination. J Orthod 2020;47:245-50.

57. Atwal A, Cottom H, Doyle P, Sandler J. Surgical/orthodontic management of a large mandibular odontogenic keratocyst (OKC). Dent Update 2019;46:473-9.

58. Kawai T, Murakami S, Hiranuma H, Sakuda M. Healing after removal of benign cysts and tumors of the jaws. A radiologic appraisal. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79:517-25.

59. Dahllof G, Huggare J. Orthodontic considerations in the pediatric € cancer patient: a review. Semin Orthod 2004;10:266-76.

60. Kaste SC, Goodman P, Leisenring W, Stovall M, Hayashi RJ, Yeazel M, et al. Impact of radiation and chemotherapy on risk of dental abnormalities: a report from the Childhood Cancer Survivor Study. Cancer 2009;115:5817-27.

61. Neill CC, Migliorati C, Trojan T, Kaste S, Karydis A, Rowland C, et al. Experience and expertise regarding orthodontic management of childhood and adolescent cancer survivors. Am J Orthod Dentofacial Orthop 2015;148:765-70.

62. Mitus-Kenig M, Derwich M, Czochrowska E, Pawlowska E. Cancer survivors present significantly lower long-term stability of orthodontic treatment: a prospective case-control study. Eur J Orthod 2021;43:631-8.

63. Elison JM, Leggitt VL, Thomson M, Oyoyo U, Wycliffe ND. Influence of common orthodontic appliances on the diagnostic quality of cranial magnetic resonance images. Am J Orthod Dentofacial Orthop 2008;134:563-72