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Ameloblastoma

A Project Submitted to The Council of the College of Dentistry at the University of Baghdad, Department of Oral Diagnosis in Partial Fulfillment of the Requirements for the Degree of B.D.S

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Certification of Supervisor

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Dedications

To my dreams, my fantasies, my weakness and my strength, I think this is the beginning of our path.

Acknowledgment

To Dr. Raghad Al-Hashimi, the Dean of the college of Dentistry/Baghdad University

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Introduction

Odontogenic tumors (OT) are a heterogeneous group of lesions of diverse clinical behavior and histopathologic types, ranging from hamartomatous lesions to malignancy. Therefore OT are found within the jaw bones (central types) or in the mucosal tissue overlying tooth-bearing areas (peripheral types). OT are basically divided into two primary categories; malignant and benign but the etiology is widely unknown. Ameloblastoma is a rare, benign, slow-growing but locally invasive neoplasm of odontogenic origin involving the mandible (80 %) and maxilla; conservative treatment results in a high recurrence rate. It belongs to the group of tumors arising from odontogenic epithelium with mature fibrous stroma without odontogenic ectomesenchyme according to the World Health Organization (WHO) Classification of Odontogenic Tumors in 2005. benign ameloblastoma is divided into: solid /multicystic, extra-osseous/peripheral, desmoplastic, and unicystic. The most common ameloblastoma is the solid/ multicystic/ conventional type. Trauma, poor nutrition, inflammation or oral infections, and irritation from tooth extractions were previously considered triggers for ameloblastoma but now the research focus changed. The most common presentation for ameloblastoma is a painless swelling of the mandible or maxilla, though in 35 % had their lesion identified as an incidental finding on imaging. The standard method of treatment is surgery either radical or conservative. The rate of recurrence and prognosis depend on the technique of surgery.

Odontogenic tumors (OT) are a broad groupe of lesions that range from hamartomatous lesions to malignancy in terms of clinical behavior and histopathologic characteristics. OT are derived from the tooth-forming apparatus' ectomesenchymal and/or epithelial tissues. Odontogenic tumors, like normal odontogenesis, are the result of inductive interactions between odontogenic ectomesenchyme and epithelium. As a result, OT

can be located in the mucosal tissue above tooth-bearing areas (central types) or within the jaw bones (central types) (peripheral types). The genesis of OT is unknown, but it is categorized into two primary categories: malignant and benign (Bilodeau and Collins, 2017).

Varying odontogenic tumors have different incidences in different countries. The most frequent odontogenic tumor is ameloblastoma, which accounts for 30–35 percent of all cases (Wright and Vered, 2017).

Most benign odontogenic tumors have vague clinical characteristics. In the early stages, odontogenic tumors may appear as a modest extension of the maxilla or mandible, but they can grow to be quite enormous. Odontogenic tumors are slow-growing tumors that have been linked to unerupted teeth, enlargement of the alveolar process, tooth loosening, and occlusion alterations. Pain, parathesia, ulceration of the oral mucosa, tooth movement, and quick onset of occlusion alterations are all common symptoms of malignant odontogenic tumors (MacDonald, 2016).

Aim of study

In this study we will demonstrate the incidence, possible etiology, pathology, clinical features and management of ameloblastoma.

Review of literature

Ameloblastoma is a rare, benign, slow-growing but locally invasive neoplasm of odontogenic origin involving the mandible (80 %) and ,maxilla. The neoplasm was first described by Cusack in 1827 (Cusack Etymologically, the name derives from the old French word .(1827 amel,'' which means enamel, and the Greek word ''blastos,'' meaning '' germ or bud. Over time, this tumor has been referred to by many different '',names including ''cystosarcoma,'' ''adamantine epithelioma adamantinoma,'' and finally ''ameloblastoma'')Brazis et al.,1995)''.

It belongs to the group of tumors arising from odontogenic epithelium with mature fibrous stroma without odontogenic ectomesenchyme according to the World Health Organization (WHO) Classification of Odontogenic Tumors in 2005. Ameloblastoma is divided into 4 types: unicystic, solid/ multicystic, desmoplastic, and peripheral. It is believed to originate from remnants of tooth-forming apparatus, such as developing enamel organ, odontogenic rests, reduced enamel epithelium and the epithelial lining of odontogenic cysts, especially dentigerous cysts, or from the basal epithelial cells of the oral mucosa (Ide et al.,2009).

1. Epidemiology

In Asia and Africa, ameloblastoma is the most frequent odontogenic tumor, whereas in Europe and America, odontoma is the most prevalent odontogenic tumor. Because most patients in impoverished countries do not seek medical attention until their diseases become symptomatic or grossly visible, the fact that odontomas are detected during regular radiography scans and do not generate clinical symptoms may account for odontoma underreporting. The average age of ameloblastoma patients in developed countries was 39.1 years, while it was 27.7 years in poor

countries. (Jing et al., 2007, Sriram and Shetty, 2008). Several studies revealed no significant gender predilection in the prevalence of ameloblastoma (Adeline et al., 2008).

The reported cases of ameloblastoma occurred over a wide range of ages, 10 to 90 years has been reported and highest incidence was recorded in the 3rd to 4th decade of life. Average age of diagnosis is 33 to 39 years, but most cases are reported between 20 to 60 years (Shafer et al., 2006).

Ameloblastoma of the jaws is the most commonly encountered odontogenic tumour in Africa and Asia but the second most common odontogenic tumour in North and South America. There is conflicting reports on the incidence rate in different races. Some studies have suggested a greater frequency of ameloblastoma in individuals of African descent; however, other studies have demonstrated the absence of a racial predilection (Ademola and Madukwe, 2018).

According to Hendra et al., the global incidence of ameloblastoma is person per million (Hendra et al., 2020) 0.92.

2. Classification

In the 2005 World Health Organization classification the benign ameloblastoma is divided into 1) solid/multicystic, 2) extraosseous/peripheral, 3) desmoplastic, and 4) unicystic (3). The solid/ multicystic ameloblastoma can histopathologically be divided into a follicular and a plexiform type; the follicular type can be further subdivided into a spindle cell type, an acanthomatous type, a granular type and a basal cell type. The plexiform type contains basal cells arranged in anastomosing strands with an inconspicuous stellate reticulum. The stroma is usually delicate, often with cystlike degeneration. The unicystic ameloblastoma represents an ameloblastoma variant that on gross

examination, and not based on the appearance on the radiograph, presents as a cyst. Two histopathological variants are recognized, being the luminal variant and the mural variant. The extraosseous type shows the histopathogical cell types and patterns as seen in the solid/multicystic type. In the desmoplastic type the stromal component dominates, compressing the odontogenic epithelial components (Hertog et al., 2012). Table 1 show the classification of ameloblastoma.

Table 1. Classification ofAmeloblastoma . (Effiom et al., 2018).

Clinicohistologic types of ameloblastoma

Types of ameloblastoma	*Synonyms*	*Salient features*	*Conventional radiographic features*	*Histopathological variants*
Benign				
Solid/multicystic	Conventional/Classical ameloblastoma	Mean age: 36 years Male > female Slightly higher in mandible	Unilocular radiolucency Multilocular radiolucency Unerupted tooth Root resorption	Cystic, acanthomatous, granular, basaloid, spindle, clear cell, hemangiomatous
Unicystic	Cystogenic ameloblastoma	Dentigerous type: Mean age: 16.5 years Male > female Non-dentigerous type: Mean age: 35.2 years Female > male Slightly higher in mandible (posteriorly)	Unilocular radiolucency Multilocular radiolucency Unerupted tooth Unilocular radiolucency Multilocular radiolucency	Luminal (plexiform unicystic, intraluminal), mural
Peripheral	Extraosseous/soft tissue ameloblastoma	Mean age: 51 years Male > female Slightly higher in mandible Exophytic Mean size 1.3 cm	Saucerization	Not applicable
Desmoplastic	Ameloblastoma with pronounced desmoplasia	Mean age: 41.6 years Female = male Maxilla = mandible	Mixed radiolucent/ radiopaque Root resorption	Hybrid Desmoplastic ± osteoplasia
Malignant				
Metastasizing	Malignant ameloblastoma	Mean age: 34.4 years Male > female Slightly higher in mandible Distant sites: lungs and other areas	Same as solid/multicystic	Same as solid/multicystic
Primary ameloblastic carcinoma	Not applicable	Mean age: 53 years Male > female Higher in mandible (posteriorly)	Ill-defined multilocular radiolucency Foci of calcification	Not applicable
Secondary ameloblastic carcinoma (intraosseous)	Carcinoma ex intraosseous ameloblastoma	Rapid growth, 7th decade Male > female Slightly higher in mandible	Ill-defined multilocular radiolucency Foci of calcification	Not applicable
Secondary ameloblastic carcinoma (peripheral)	Carcinoma ex peripheral ameloblastoma	Male = female Alveolar bone resorption	Interradicular radiolucency	Not applicable

The new version simplified classification into 3 types: conventional, unicystic and peripheral. The solid/multicystic term was discarded, as it

could be confused with the unicystic type. Desmoplastic ameloblastoma was also reclassified as a histological subtype and not as a clinicalpathological entity, based on the fact that it behaves like any conventional ameloblastoma, although its clinical and radiographic characteristics are peculiar (Speight and Takata, 2018).

2.1 Conventional ameloblastoma

The most common ameloblastoma is the solid/ multicystic/ conventional type, making up about 91% of all cases of ameloblastoma. It is slow growing and runs a benign course. Histologically, the solid/multicystic/conventional ameloblastoma displays two distinct histological patterns: the follicular and plexiform types. The follicular type displays proliferating odontogenic epithelial cells arranged in islands, while plexiform type displays epithelial cells arranged in continuous anastomosing strands. It is not uncommon for an ameloblastoma to display both histological patterns. In addition to these two histological types, cystic, granular, acanthomatous, spindle cell, basal cell, clear cell, and other microscopic subtypes have been reported (Effiom et al., 2018).

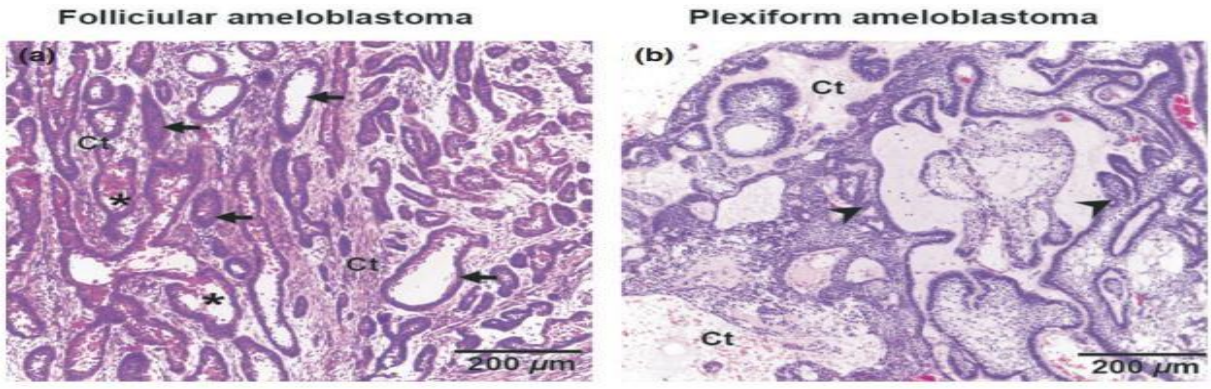
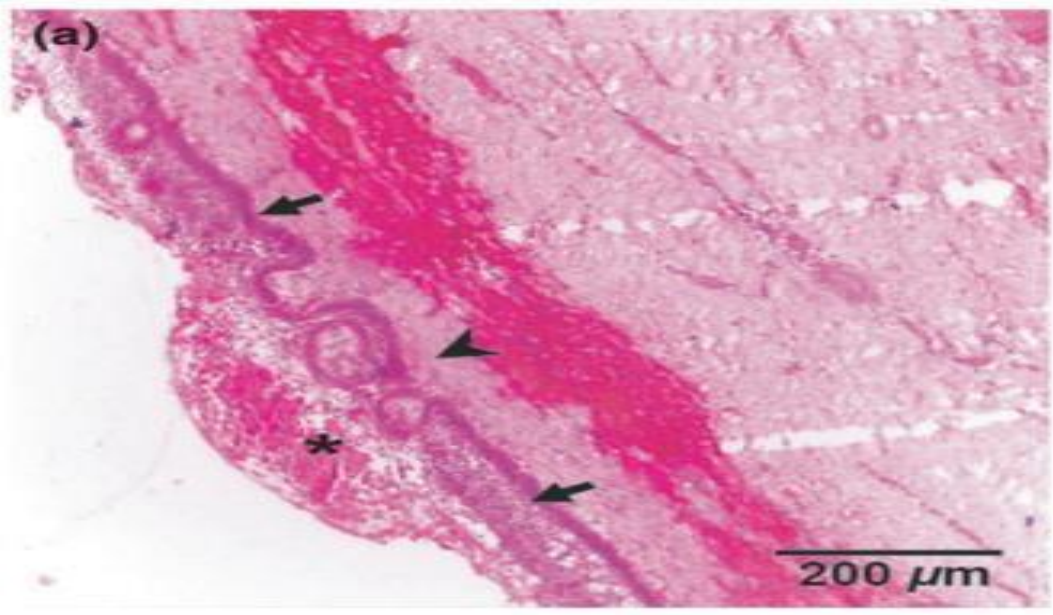


Figure 1. Conventional ameloblastoma histological types (Effiom et al., .(2018

2.2 Unicystic ameloblastoma

Unicystic ameloblastomas are a type of cystic lesion that grows slowly and is relatively aggressive locally. The lesions usually have large unilocular radiolucencies with a well-defined border on radiography. An of %impacted or unerupted tooth is responsible for around 50-80 instances. As a result, the clinical and radiological manifestations of unicystic ameloblastoma can be confused with those of dentigerous cysts (Zhang et al., 2010).

The degree of ameloblastomatous epithelial expansion is classified into three histological types: luminal, intraluminal, and mural. Their biological behavior, therapy, and prognosis are all influenced by this classification. Unicystic ameloblastomas are thought to be less aggressive than solid and multicystic ameloblastomas, and to respond better to conservative treatments such as enucleation, curettage, and marsupialization (Hsu et al., 2014).

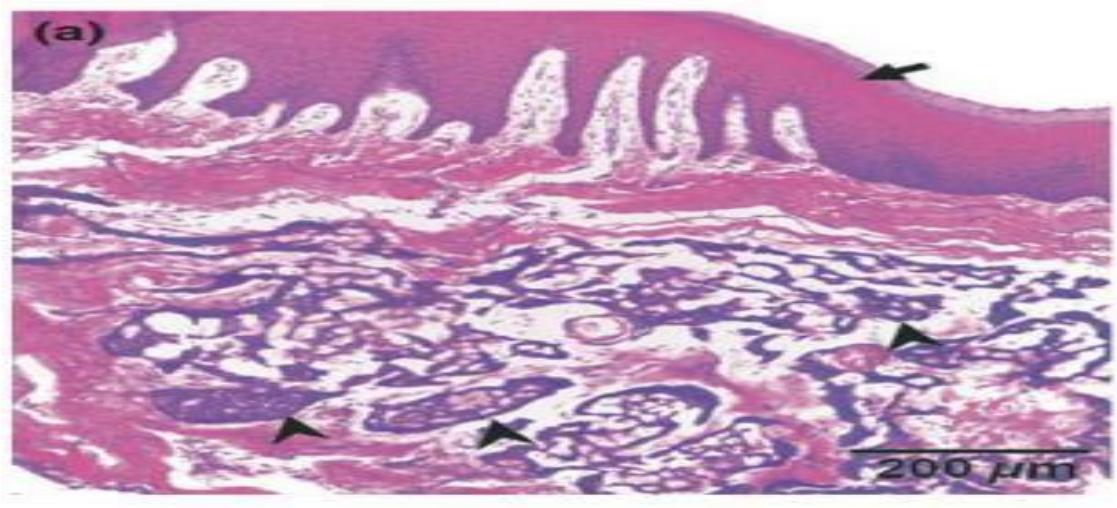


.**Figure 2. Unicystic ameloblastoma.** (Effiom et al., 2018)**.**

2.3Peripheral ameloblastoma

PA is defined as a “benign neoplasm or hamartomatous lesion confined to the soft tissues overlying the tooth-bearing areas of the jaws or alveolar mucosa in the edentulous area”. This definition has not included the extra gingival lesions that occur in the buccal mucosa, lips, palate, and floor of the mouth. It has been hypothesized that the buccal mucosal lesions have more chances of having its origin from the salivary excretory ducts. Odontogenic epithelial gingival hamartoma (OEGH) or odontogenic epithelial hamartoma (OEH) is another entity, which is similar to PA on clinical and histopathological grounds (Premalatha et al., (2013.

Peripheral ameloblastoma is the rarest type of ameloblastoma, accounting for only 1% of all ameloblastoma occurrences. Patients in their forties and fifties, on average, are the most afflicted. These lesions, which appear on the posterior gingiva or alveolar sulcus, are more prevalent in the mandible than in the maxilla. Peripheral ameloblastoma has the same histological pattern as solid/multicystic/conventional ameloblastoma and is made up of islands of ameloblastic epithelium (Siar et al., 2012).



.**Figure 3. Peripheral ameloblastoma** (Effiom et al., 2018)**.**

2.4 Desmoplastic ameloblastoma

Histologically, it is characterized with extensive stromal collagenisation or desmoplasia with small nests and strands of odontogenic epithelium. Additionally “hybrid” lesions showing some microscopic features of the desmoplastic variant together with typical areas of follicular or plexiform ameloblastoma have been described. Most diagnoses are made related to the histological specimen after the DA was already removed (Gardner et al., 2015).

The histological feature of extensive stromal dysplasia is pathognomonic. It consists of islands of odontogenic epithelium with variable shapes and sizes proliferating within a highly collagenous connective tissue. The thick collagen fibers tend to compress the odontogenic epithelial islands from the periphery, giving rise to the bizarre shapes and sizes. It is not uncommon for desmoplastic ameloblastoma to contain metaplastic bone formations (Sun et al., 2009).

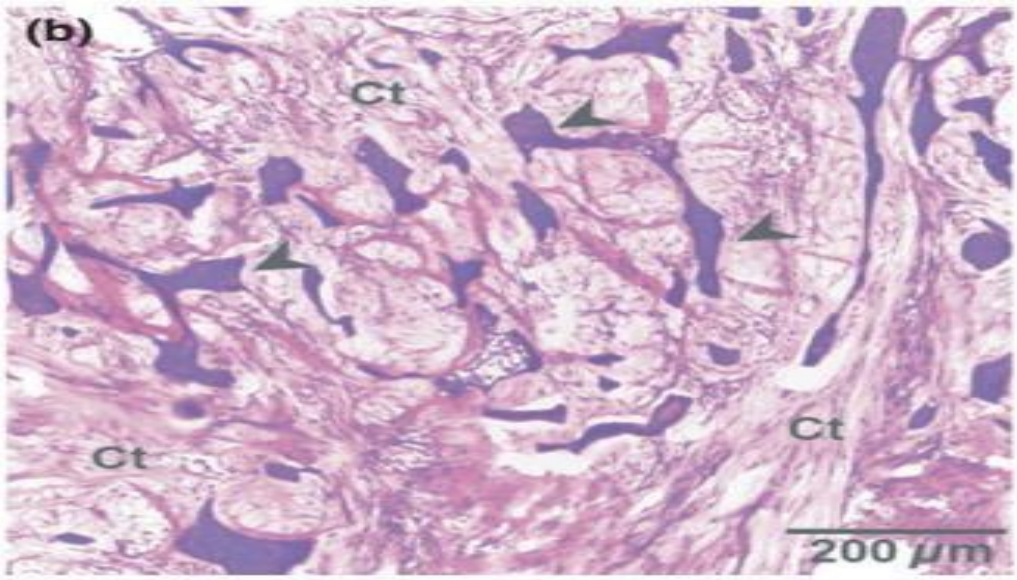


Figure 4. Desmoplastic ameloblastoma (Effiom et al., 2018).

2.5. Malignant ameloblastoma and ameloblastic carcinoma

Ameloblastoma rarely evolves into malignancy and develops hematogenous spread; although the benign histology of ameloblastoma is the same, the malignant histology is characterized by the presence of metastases and is associated with cytologic atypia with or without metastases (Mogollon-reyes, 2019).

The terminology for these lesions is somewhat confusing, but should not be considered controversial. The term *malignant ameloblastoma* is used for a tumor that shows the histopathologic features of ameloblastoma, both in the primary tumor and in the metastatic deposits. This is a very rare neoplasm, with fewer than 30 well-documented cases described in the literature. The term *ameloblastic carcinoma* should be reserved for an ameloblastoma that has cytologic features of malignancy in the primary tumor, in a recurrence, or in any metastatic deposit. This is also a rare condition, although approximately 200 cases have been reported. These lesions may follow a markedly aggressive local course, but metastases do not necessarily occur (Sciubba et al., 2005; Neville, 2009).

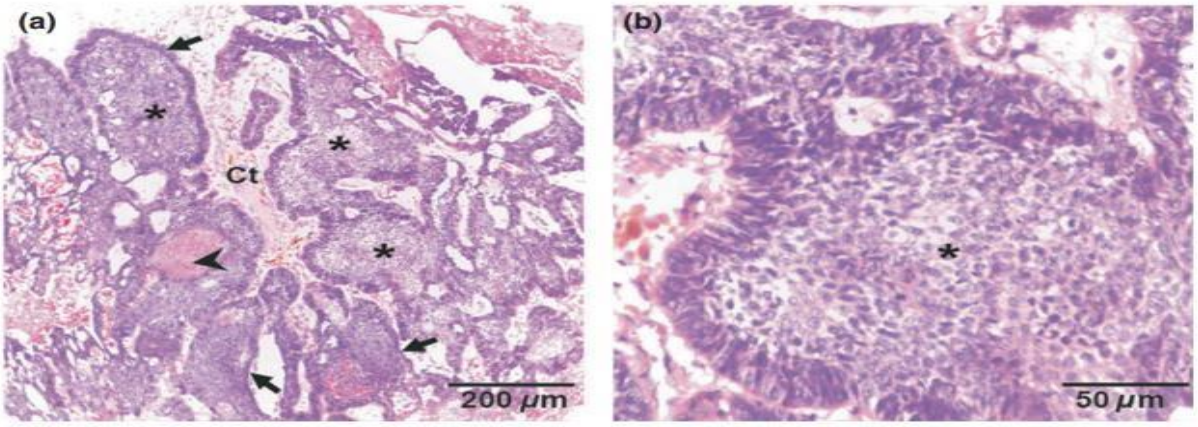


Figure 5. Ameloblastic carcinoma. (a) At low magnification, (b) At higher magnification, (Effiom et al., 2018).

3. Etiology

Ameloblasts are ectodermal cells that originated from the oral epithelium. The cells that deposit tooth enamel, which creates the crown's outer surface, are only present during tooth formation. Only until odontoblasts have formed the primary layer of dentin do ameloblasts become functional (the layer beneath enamel). Before or after tooth eruption, the cells become part of the enamel epithelium and finally experience apoptosis (cell death). Cell rests of Malessez and cell rests of Serres are deposits of these cells in the structures in and around the tooth. Ameloblastomas are thought to develop from either the cells listed above or other ectodermal cells, such as those connected with the enamel organ. Palanisamy and jenzer, 2022).

Ameloblastoma was originally thought to be caused by trauma, poor nutrition, inflammation or oral infections, and irritation from tooth extractions. The discovery of remains of the migrating epithelium at the enamel organ's cervical loop shifted the focus of ameloblastoma research in a new direction. The expression similarity of cytokeratin and vimentin between growing tooth germ and ameloblastoma further validated our findings (Brown and Betz, 2015).

4. Clinical features

A painless swelling of the mandible or maxilla is the most common symptom of ameloblastoma, however up to 35% of patients had their lesion found as an accidental finding on imaging. Pain is infrequent, however it can develop as a result of bleeding, particularly after a fine needle aspiration (FNA) (Wenig, 2007). Ameloblastic carcinoma is a rare malignant tumor that causes pain and rapid growth. Tooth displacement and root resorption are uncommon in desmoplastic ameloblastomas, however they have been documented in up to 25% of cases. Paresthesias

are uncommon, and just a few occurrences of perineural invasion have been described (Schafer et al., 1998).

Up to 80 % of ameloblastoma cases occur in the mandible, with a predilection for the posterior mandibular region. Rare cases have been reported as primary to the sinonasal cavities. Ameloblastoma can be associated with unerupted third molar teeth, particularly in the unicystic type. Desmoplastic ameloblastomas often occur in the anterior or premolar regions of the mandible or maxilla. Ameloblastic carcinomas also favor the mandible over the maxilla. Maxillary ameloblastomas also mostly occur in the posterior molar region (Becelli, 2002).

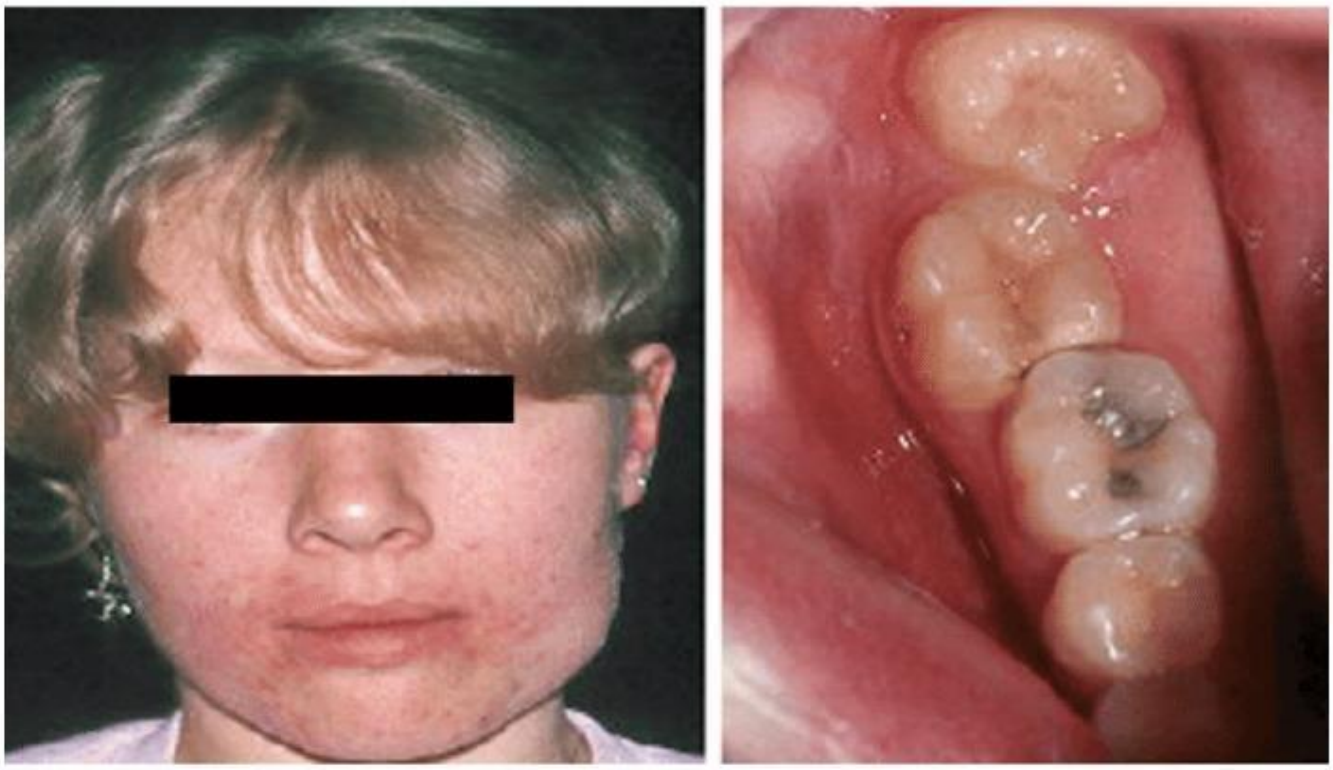


Figure 6. Clinical presentation of ameloblastoma (Becelli, 2002).

5. Diagnosis

5.1 Imaging

Imaging and possible biopsy are part of the preoperative diagnostic examination. Apart from the peripheral subtype, which arises in the gingiva or buccal mucosa, ameloblastomas arise in bone and are thus frequently detected incidentally on dental X-rays (pantomography) or plain films; Xrays typically show a lytic lesion with scalloped margins, resorption of tooth roots, and impacted molars (unicystic) (Singer et al., The most common ameloblastoma, the multilocular/solid variety, .(2009 has the distinctive "soap bubble" look. Plain X-rays, while sometimes sufficient for a thorough examination, lack sensitivity and specificity for determining the degree of bone and soft tissue invasion. The most helpful diagnostic imaging modality is computed tomography (CT), which often shows welldefined radiolucent uni/multilocular expansile lesions (Underhill et al., 1992).

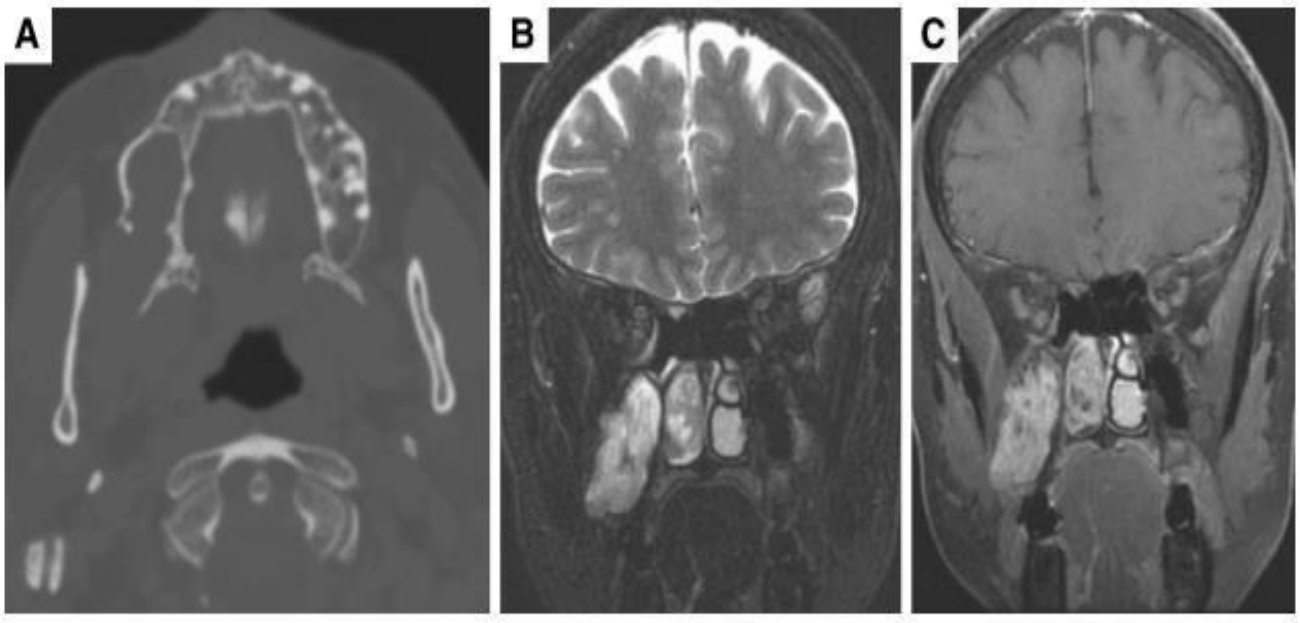


Figure 7. Ameloblastoma as shown in CT (A), MRI (B,C) (Fujita et al., 2013).

MRI provides potentially more complete information than CT about soft tissue extension and marrow extension beyond the lytic bone cavity. MRI is particularly useful for ameloblastomas arising from the maxilla, as it helps to characterize extension to the orbit, paranasal sinuses, and skull base. MRI should be considered in desmoplastic ameloblastomas because they have poorly defined soft tissue borders and are often misdiagnosed as a fibro-osseous lesion. PET-CT is generally reserved for metastatic ameloblastoma, where it may aid with staging of the distant metastasis (Fujita et al., 2013).

5.2 Biopsy

Biopsy may be useful prior to treatment to avoid unnecessary operations on lesions of alternative etiology, such as osteomyelitis, cystic fibrous dysplasia, giant cell tumor, ossifying fibroma, multiple myeloma, and rare sarcomas, which should be alternatively treated or simply observed. In malignant ameloblastomas, biopsy also allows for correct preoperative staging (Dunfee et al., 2006).

A biopsy should be done at the start of the case to sort this out. Maxillary ameloblastomas often present with involvement of adjacent soft tissue, resembling adenocarcinomas and squamous cell carcinomas. Fine needle aspiration can be acquired via a window of cortical erosion as identified by imaging or from the dental socket. Incisional biopsy can provide a more accurate diagnosis but requires disruption of the mucosa which will ultimately need to be removed at surgery. Peripheral ameloblastomas are not covered by bone and can be biopsied without difficulty (McClary et al., 2016).

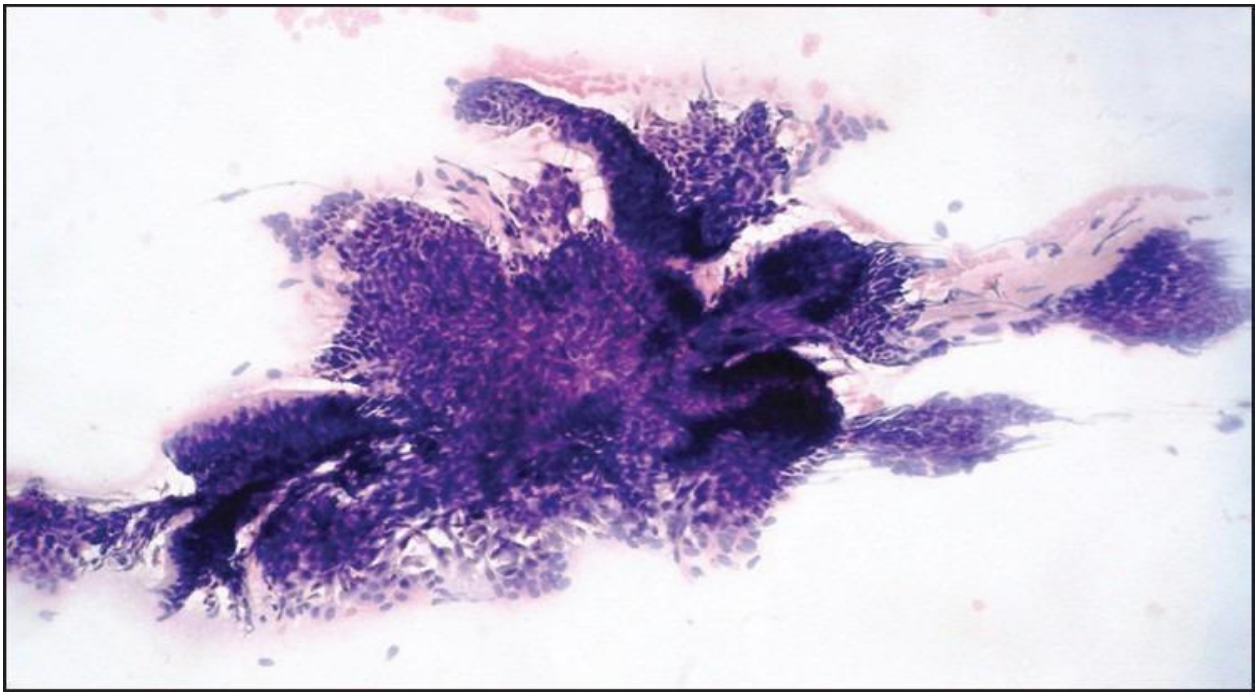


Figure 8. FNA smear of ameloblastoma (McClary et al., 2016).

6. Treatment

If the recurrence rate is a priority, a wide excision of the jaw is usually the preferred treatment for ameloblastoma. Radical surgery, on the other hand, frequently results in major consequences, such as facial deformities, masticatory dysfunction, and aberrant jaw movement. Given the features highly rare of ameloblastoma as a locally invasive, slow-growing, and metastasizing benign tumor, the treatment technique should be prioritized based on the patients' morbidity and quality of life, emphasizing that the recurrence rate is not always the most important aspect (Dandriyal, 2011).

There are two therapy strategies for ameloblastoma: a conservative way of treatment and radical procedures. Non-radical surgical procedures like enucleation and curettage, combined with liquid nitrogen spray cryosurgery, or just drilling of the perilesional bone are mentioned to be useful in unicystic ameloblastomas, especially in children and young patients. Other authors show high rates of reccurence of ameloblastoma

after conservative treatment protocols and therefore recommend radical surgical treatment (Pinsolle et al., 1995).

The location of the tumor, the type of ameloblastoma, and the patient's age all play a role in determining the best treatment option. Ultimately, the goal is to achieve a tumor-free patient with a low risk of recurrence. Treatment options are always weighed individually based on the patient's age, condition, and tumor location, with the patient's wants and expectations taken into account (Arotiba et al., 2005).

According to the literature, unicystic ameloblastomas can be treated conservatively with curettage, enucleation, and cryosurgery. Ameloblastoma can reach the cancellous bone at a mean of 4.5 mm but up to 8 mm beyond the radiological boundary. Therefore, a radical approach to nonunicystic ameloblastomas through segmental or marginal resections with tumor-free margins is the preferred treatment modality to prevent recurrence (Simon et al., 2013). An accurate description of the ameloblastoma type, growth pattern, and radiological diagnoses is needed to determine the best treatment option. The longer the tumor persists, the more frequently recurrence occurs; and the more conservative treatment procedures the patient has undergone, the greater the risk of malignant transformation. In cases of solid or multicystic ameloblastomas, radical surgery (marginal or segmental resection) is preferred, requiring a plate reconstruction or considerable reconstructive surgery (Hasegawa et al., Following mandibular reconstruction, oral function rehabilitation .(2013 is facilitated using dental implants and restorative dentistry. Primary and recurrent peripheral ameloblastomas are excised through peripheral ostectomy. Peripheral ameloblastomas rarely recur following conservative surgery (Hertog et al., 2012).

Postsurgical defects in the maxillary region predispose the patient to hypernasal speech, fluid leakage into the nasal cavity, impaired masticatory function, and in some patients, various degrees of cosmetic deformity. Mandibular resection can also prove devastating to mastication, deglutition, phonation, and oral competence. Moreover, the mandible frames the lower third of the face and represents a major component of the human appearance. Satisfactory reconstruction of complex jaw defects, especially in a single-step procedure, is therefore a surgical challenge. For benign tumors, the bone grafts have become a reliable source during the last few years in osseous reconstruction. The fibula, scapula and iliac crest are the commonly chosen donor sites to reconstruct mandibular or maxillary defects (Dandriyal, 2011).

The most appropriate treatment for young, growing patients remains controversial. Some studies have tried to address this issue. Takahashi et al. studied a population of 27 patients with a mean age of 12.3 years, in whom they recommended conservative treatment because of an often less aggressive histological type (plexiform), in order to allow continued mandibular growth and consequently limit the major cosmetic and functional sequelae observed at this age. Secondary resection in the event of recurrence proved to be effective and allowed less mutilating treatment Takahashi et al, 1998).

7. Recurrence

Several studies have reported a higher recurrence rate after conservative treatment compared to radical treatment. Ameloblastomas have a high tendency to recur. A meta-analysis found a pooled recurrence rate of 8% for ameloblastomas treated radically and 41% when treated conservatively. The respective values for unicystic ameloblastoma

recurrence were 3% and 21%. They also found that conventional ameloblastomas recurred more often than unicystic ameloblastomas despite treatment modality, indicating a more aggressive behavior among ameloblastoma compared to unicystic ameloblastomas. Current thinking indicates that a segmental resection with sufficient, healthy margins is the predominant choice for treatment regardless of type (Hendra et al., 2019).

8. Prognosis

Although it is considered a benign tumor, ameloblastoma has aggressive behaviors including local recurrence, cancerization or even distant metastasis. Therefore, many surgeons tended to take a radical surgery when facing this disease. more than 50% patients receiving the conservative treatment had good prognosis without any recurrence. In another word, more than 50% patients could be treated by a conservative method, and maintain the continuity of their jaws to have a better quality of life. Obviously, the radical surgery is an overtreatment that have been chosen as a routine way (Li et al., 2012).

Many factors were related with the prognosis of ameloblastoma. Some scholars believed that a radical surgery should be used for the multicystic ameloblastoma to prevent the recurrence. From the pathological aspect, the follicular ameloblastomas were thought to have a higher recurrence rate than plexiform or unicystic (Rapidis et al., 2004).

9. Conclusion

Ameloblastoma is benign tumor of jaw with invasive behaivour and low potency for malignant transformation. Unfortunately its usually asymptomatic so there is delay in diagnosis and management. So recommend the health authorities to immediately start campaigns about the annul screening and about how serious the problem is.

References

1. Adeline VL, Dimba EA, Wakoli KA, Njiru AK, Awange DO, Onyango JF, Chindia ML. Clinicopathologic features of ameloblastoma in Kenya: a 10-year audit. Journal of Craniofacial Surgery. 2008 Nov 1;19(6):1589-93.
2. Ademola J, Madukwe IU. Epidemiology of ameloblastoma. Nigerian Journal .of Dental Sciences. 2018 Dec 20;1(1&2):14-31.
3. Arotiba GT, Ladeinde AL, Arotiba JT, Ajike SO, Ugboko VI, Ajayi OF. Ameloblastoma in Nigerian children and adolescents: a review of 79 cases. Journal of oral and maxillofacial surgery. 2005 Jun 1;63(6):747-51.
4. Becelli R, Carboni A, Cerulli G, Perugini M, Iannetti G. Mandibular ameloblastoma: analysis of surgical treatment carried out in 60 patients between 1977 and 1998. Journal of Craniofacial Surgery. 2002 May 400-395:(3)13;1.
5. Bilodeau EA, Collins BM. Odontogenic cysts and neoplasms. Surgical pathology clinics. 2017 Mar 1;10(1):177-222.
6. Brazis PW, Miller NR, Lee AG, Holliday MJ. Neuro-ophthalmologic aspects of ameloblastoma. Skull base surgery. 1995 Oct;5(04):233-44.
7. Brown NA, Betz BL. Ameloblastoma: a review of recent molecular pathogenetic discoveries. Biomarkers in cancer. 2015 Jan;7:BIC-S29329.
8. Cusack JW. Report of the amputations of the lower jaw. Dubliln Hop Rec .38–4:1 ;1827.
9. Dandriyal R, Gupta A, Pant S, Baweja HH. Surgical management of ameloblastoma: Conservative or radical approach. National journal of maxillofacial surgery. 2011 Jan;2(1):22.
10. Dunfee BL, Sakai O, Pistey R, Gohel A. Radiologic and pathologic characteristics of benign and malignant lesions of the mandible. Radiographic. Nov;26(6):1751-68 2006.
11. Effiom OA, Ogundana OM, Akinshipo AO, Akintoye SO. Ameloblastoma: current etiopathological concepts and management. Oral diseases. 2018 Apr;24(3):307-16.
12. Fujita M, Matsuzaki H, Yanagi Y, Hara M, Katase N, Hisatomi M, Unetsubo T, Konouchi H, Nagatsuka H, Asaumi JI. Diagnostic value of MRI for odontogenic .tumours Dentomaxillofacial .Radiology 2013

May;42(5):20120265.

13. Gardner DG, Heikinheimo K, Shear M, Philipsen HP, Coleman HA. En: Barnes L, Eveson JW, Reichart P, Sidransky D.(Eds.): World Health Organization Classification of Tumours. Pathology and Genetics Head and Neck Tumors.
14. Hasegawa T, Imai Y, Takeda D, Yasuoka D, Ri S, Shigeta T, Minamikawa T, Shibuya Y, Komori T. Retrospective study of ameloblastoma: the possibility .of conservative treatment. Kobe J Med Sci. 2013 Nov 9;59(4):112-21.
15. Hendra FN, Natsir Kalla DS, Van Cann EM, de Vet HC, Helder MN, Forouzanfar T. Radical vs conservative treatment of intraosseous ameloblastoma: Systematic review and meta_analysis. Oral diseases. 2019 Oct;25(7):1683-96.
16. Hendra FN, Van Cann EM, Helder MN, Ruslin M, de Visscher JG, Forouzanfar T, de Vet HC. Global incidence and profile of ameloblastoma: A systematic review and meta_analysis. Oral Diseases. 2020 Jan;26(1):12-21.
17. Hertog D, Bloemena E, Aartman IH, van-der-Waal I. Histopathology of ameloblastoma of the jaws; some critical observations based on a 40 years single institution experience. Medicina oral, patologia oral y cirugia bucal. Jan;17(1):e76 2012.
18. Hsu MH, Chiang ML, Chen JK. Unicystic ameloblastoma. Journal of Dental .Sciences. 2014 Dec 1;9(4):407-11.
19. Ide F, Mishima K, Miyazaki Y, Saito I, Kusama K. Peripheral ameloblastoma in-situ: an evidential fact of surface epithelium origin. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 2009 Nov 7-763:(5)108;1.
20. Jing W, Xuan M, Lin Y, Wu L, Liu L, Zheng X, Tang W, Qiao J, Tian W. Odontogenic tumours: a retrospective study of 1642 cases in a Chinese population. International journal of oral and maxillofacial surgery. 2007 Jan 5-20:(1)36;1.
21. Li Y, Han B, Li LJ. Prognostic and proliferative evaluation of ameloblastoma based on radiographic boundary. International journal of oral science. 2012 Apr;4(1):30-3.

22. MacDonald D. Lesions of the jaws presenting as radiolucencies on cone-beam .CT. Clinical Radiology. 2016 Oct 1;71(10):972-85.
23. McClary AC, West RB, McClary AC, Pollack JR, Fischbein NJ, Holsinger CF, Sunwoo J, Colevas AD, Sirjani D. Ameloblastoma: a clinical review and trends in management. European Archives of Oto-Rhino-Laryngology. 2016 Jul;273(7):1649-61.
24. Mogollón-Reyes G. Malignant ameloblastoma: multiple local recurrence and .metastasis in the scalp. Case report. Case reports. 2019 Jun;5(1):36-45.
25. Neville BW. Oral and maxillofacial pathology. St. Louis, Mo: Saunders/ Elsevier. 2009.
26. :Palanisamy JC, Jenzer AC. Ameloblastoma. [Updated 2022 Jan 21]. In -StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan /Available from: https://www.ncbi.nlm.nih.gov/books/NBK545165 .
27. Pinsolle J, Michelet V, Coustal B, Siberchicot F, Michelet FX. Treatment of Head & Neck –ameloblastoma of the jaws. Archives of Otolaryngology .Surgery. 1995 Sep 1;121(9):994-6.
28. Premalatha BR, Patil S, Rao RS, Reddy NP, Indu M. Odontogenic tumor markers-an overview. Journal of international oral health: JIOH. 2013 .Apr;5(2):59.
29. Rapidis AD, Andressakis DD, Stavrianos SD, Faratzis G, Arnogiannaki-Liappi N, Lagogiannis GA, Valsamis SV, Apostolikas N. Ameloblastomas of the jaws: clinico-pathological review of 11 patients. European Journal of Surgical Oncology (EJSO). 2004 Nov 1;30(9):998-1002.
30. Schafer DR, Thompson LD, Smith BC, Wenig BM. Primary ameloblastoma of the sinonasal tract: a clinicopathologic study of 24 cases. Cancer: Interdisciplinary International Journal of the American Cancer Society. 1998 Feb 15;82(4):667-74.
31. Sciubba JJ, Eversole LR, Slootweg PJ. Odontogenic/ameloblastic carcinomas. Barnes L, Eveson JW, Reichart P, Sidransky D, eds. World Health Organization Classification Head and Neck Tumours. Lyon, France, IARC .Press. 2005.
32. .Shafer WG, Hine MK, Levy BM, Rajendran R, Sivapathasundharam B .Shafer's textbook of oral pathology. Diseases of the Skin. Rajendran R, editor 7-5:1103;2006.

33. Siar CH, Lau SH, Ng KH. Ameloblastoma of the jaws: a retrospective analysis of 340 cases in a Malaysian population. Journal of oral and maxillofacial surgery. 2012 Mar 1;70(3):608-15.
34. Simon EN, Merkx MA, Kalyanyama BM, Shubi FM, Stoelinga PJ. Immediate reconstruction of the mandible after resection for aggressive odontogenic tumours: a cohort study. International journal of oral and maxillofacial surgery. 2013 Jan 1;42(1):106-12.
35. Singer SR, Mupparapu M, Philipone E. Cone beam computed tomography findings in a case of plexiform ameloblastoma. Quintessence international. .Sep 1;40)8(2009.
36. Soluk-Tekkeşin M, Wright JM. The World Health Organization classification of odontogenic lesions: a summary of the changes of the 2017 (4th) edition. ..Turk Patoloji Derg. 2018 Jan 1;34(1):1-8
37. Speight PM, Takata T. New tumour entities in the 4th edition of the World Health Organization Classification of Head and Neck tumours: odontogenic and maxillofacial bone tumours. Virchows Archiv. 2018 Mar;472(3):331-9.
38. Sriram G, Shetty RP. Odontogenic tumors: a study of 250 cases in an Indian teaching hospital. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 2008 Jun 1;105(6):e14-21.
39. Sun ZJ, Wu YR, Cheng N, Zwahlen RA, Zhao YF. Desmoplastic ameloblastoma–A review. Oral oncology. 2009 Sep 1;45(9):752-9.
40. Takahashi K, Miyauchi K, Sato K. Treatment of ameloblastoma in children. .British Journal of Oral and Maxillofacial Surgery. 1998 Dec 1;36(6):453-6.
41. Underhill TE, Katz JO, Pope Jr TL, Dunlap CL. Radiologic findings of diseases involving the maxilla and mandible. AJR. American journal of .roentgenology
Aug;159(2):345-50 1992.
42. Wenig BM. Atlas of head and neck pathology, 2nd edn. Elsevier Saunders. 2007.
43. Wright JM, Vered M. Update from the 4th edition of the World Health Organization classification of head and neck tumours: odontogenic and .maxillofacial bone tumors. Head and neck pathology. 2017 Mar;11(1):68-77.
44. Zhang J, Gu Z, Jiang L, Zhao J, Tian M, Zhou J, Duan Y. Ameloblastoma in children and adolescents. British Journal of Oral and Maxillofacial Surgery.