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# Odontogenic Neoplasms with Infrequent Microscopic Features: A Retrospective Study

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## Abstract

**Objectives:** Odontogenic tumors are lesions with unique microscopic patterns and clinical behavior. They are divided into three groups: epithelial, mesenchymal, and mixed. These neoplasms have several unique microscopic patterns; however, limited information exists on their infrequent features. This study aimed to enquire into the infrequent histopathologic features of odontogenic neoplasms in an oral pathology referral center in Iran.

**Methods:** The archives of oral pathology department, Shahid Beheshti University of Medical Sciences, were investigated retrospectively over a 30-year period. The files with a diagnosis of odontogenic tumors were selected. All microscopic slides were re-evaluated, and cases with infrequent features were extracted. Finally, demographic data were extracted and analyzed for possible correlations with pathologic features.

**Results:** Over 30 years, 503 odontogenic tumors were diagnosed, of which 30 (5.96%) showed infrequent microscopic features. The mean age of patients was 33.7, ranging from 15 to 69 years. Mandibular predilection (80%) was the most prominent feature, and the male-to-female ratio was 0.87. Approximately 80% of the lesions displayed painless swelling. In solid ameloblastoma, microscopic features included adenoid appearance, hyalinization, keratin pearls, dyskeratosis, and hemangiomatous appearance. Papillary patterns, pure granular cells, clear cells, hypercellular whorls, and ghost cells were also observed, while ameloblastic carcinomas most commonly showed mucous cells, keratin pearls, and sheets of spindle cells.

**Conclusion:** Although odontogenic tumors are mostly a homogeneous group of tumors, they sometimes show diversity in several features, many of which indicate the multipotentiality of the odontogenic epithelium. In this study, although all the criteria of adenoid tumors were not observed, five samples showed some features of adenoid ameloblastoma.

**Keywords:** Ameloblastoma; Adenoid; Odontogenic Tumor; Mandible

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## Introduction

Odontogenic tumors are divided into three groups: epithelial, mesenchymal, and mixed.[1] Ameloblastoma is the most clinically significant odontogenic neoplasm of the epithelial origin and shows several different histological variants[2], including the solid/multicystic/conventional, unicystic, and peripheral forms. Solid ameloblastoma is a slow-growing, locally aggressive tumor of the jaws with a tendency to the posterior mandible.[3,4] This tumor can be seen in a wide age range, with an average age of 33 years and no gender predilection. [5] In terms of histopathologic features, this tumor is characterized by invagination of the odontogenic epithelium in a fibrous stroma. Epithelial nests contain a core of loosely arranged angular cells and peripheral ameloblast-like cells with reverse polarity. Other histologic subtypes of ameloblastoma, such as follicular, plexiform, acanthomatous, granular cells, desmoplastic, and basal cells, have also been recognized.[3] Other less commonly encountered microscopic variants include keratoameloblastoma (KA), hemangiomatous (HA), adenoid ameloblastoma (AdAM), and mucous cell differentiation.[3,6] Although variations in these histomorphologic patterns generally have little impact on the behavior of tumors[3], failure to recognize these patterns causes confusion for pathologists in the accurate diagnosis of these lesions.

This study aimed to assess the infrequent histopathologic characteristics of odontogenic tumors diagnosed in an oral pathology referral center in Iran over a period of 30 years.

## Methods

This study was approved by the ethics committee of Shahid Beheshti University of Medical Sciences (IR.SBMU.DRC.REC.1401.019). The archives (1993 to 2022) available in the oral pathology department at Shahid Beheshti University of Medical Sciences, served as the data source for this retrospective study. The files with a diagnosis of odontogenic tumors were selected. An experienced oral pathologist re-evaluated all histopathologic slides, and cases with infrequent features were extracted. Then demographic data, such as age and gender, histopathologic features, such as the location of lesions, clinical features, and radiographic findings were recorded and analyzed using descriptive statistics in SPSS software.

## Results

In this study, 30 odontogenic tumors with infrequent histopathologic features were identified. These tumors belonged to 14 males and 16 females (M/F=0.87/1) with a mean age of 33.7 years. The most common location was the mandible (mandible/maxilla=4) (Table 1), and most of the lesions showed radiolucent (93.33%) and multilocular

(66.66%) patterns (Table 2). In most cases, the expansion was painless (80%) (Table 3).

**Table 1-** Demographic information of odontogenic neoplasms with infrequent microscopic features

| Histopathologic diagnosis | N | Mean age (range) | Gender | | Lesion location | |
|---|---|---|---|---|---|---|
| | | | Male | Female | Mandible | Maxilla |
| Solid Ameloblastoma | 18 | 35.27 (16-59) | 7 | 11 | 16 | 2 |
| Unicystic ameloblastoma | 1 | 28 | 1 | - | - | 1 |
| Ameloblastic carcinoma | 3 | 45.66 (20-63) | 2 | 1 | 3 | - |
| Adenomatoid odontogenic tumor | 5 | 22 (14-28) | 2 | 3 | 3 | 2 |
| Central odontogenic fibroma | 2 | 42 (15, 69) | 1 | 1 | 1 | 1 |
| Ameloblastic fibro-odontoma | 1 | 17 | 1 | - | 1 | - |
| **Total** | 30 | | 14 | 16 | 24 | 6 |

**Table 2-** Radiographic information of odontogenic neoplasms with infrequent microscopic feature

| Histopathologic diagnosis | N | Internal structure | | Locularity | | Clinical manifestation | | |
|---|---|---|---|---|---|---|---|---|
| | | Lucent | Mixed/opaque | uni | multi | asymptomatic | Painless swelling | Painful swelling |
| Solid Ameloblastoma | 18 | 18 | - | 3 | 15 | 1 | 14 | 3 |
| Unicystic Ameloblastoma | 1 | 1 | - | 1 | - | - | 1 | - |
| Ameloblastic carcinoma | 3 | 2 | 1 | - | 3 | - | 3 | - |
| Adenomatoid odontogenic tumor | 5 | 5 | - | 5 | - | 1 | 4 | - |
| Central odontogenic fibroma | 2 | 2 | - | 1 | 1 | 1* | 1 | - |
| Ameloblastic fibro-odontoma | 1 | - | 1 | - | 1 | - | 1 | - |
| **Total** | 30 | 28 | 2 | 10 | 20 | 3 | 24 | 3 |

*There was no evidence of clinical sign except for an indentation in the palatal mucosa in the alignment of left first maxillary molar.

**Table 3-** Infrequent microscopic features of odontogenic tumors

| Histopathologic diagnosis | N | Infrequent microscopic features |
|---|---|---|
| Solid Ameloblastoma | 18 | significant keratin pearl (n=3), adenoid structures (n=1), adenoid structures /hyalinization (n=1), ), adenoid structures/ papillary pattern (n=1), hypercellular whorls (n=2), hyalinization (n=5), hemangiomatous pattern (n=2), , individual cell keratinization (n=1), pure granular cells (n=1), presence of ghost cells (n=1), clear cells (n=6) |
| Unicystic Ameloblastoma | 1 | Many giant cells (CD68 +) in underlying connective tissue |
| Ameloblastic carcinoma | 3 | Mucous cells (n=1), significant keratin pearls (n=2), sheets of spindle cells (n=1) |
| Adenomatoid odontogenic tumor | 5 | Predominantly cystic lesion (n=3), clear cells (n=2) |
| Central odontogenic fibroma | 2 | Pindborg like areas, signet ring cells in odontogenic islands, calcification in whorls (n=1)/ massive eosinophilic material negative for amyloid (n=1) |
| Ameloblastic fibro-odontoma | 1 | Squamous metaplasia, microcysts formation |

**Solid Ameloblastoma**

The mean age of patients was 35.27 years, with a male-to-female ratio of 0.63/1. Mandible (88.88%) was the most frequently involved site, followed by the maxilla (11.11%) (ratio: 8/1). Bone expansion was observed in 77.77% of the cases. Infrequent microscopic features included significant keratin pearls (n=3), adenoid structures (n=1), adenoid structures /hyalinization (n=1), adenoid structures/papillary pattern (n=1), significant hyalinization (n=5), hemangiomatous appearance (n=2), and individual cell keratinization (n=1), as well as the presence of pure granular cells (n=1), ghost cells (n=1), hypercellular whorls (n=2), and clear cells (n=6) (Figures.1, 2).

**Unicystic Ameloblastoma**

There was a 28-year-old male with a painless expansion of the mandible. Histopathologic sections showed a cystic lesion lined by ameloblastic epithelium. The underlying fibrous connective tissue revealed many multinucleated giant cells that were positive for CD68 (Figure 3).

**Ameloblastic Carcinoma**

For this type of tumor, a male predilection was noticed with a mean age of 45.66 years. All three cases occurred in

the mandible, and infrequent histopathologic features included the presence of mucous cells (n=1), significant keratin pearls (n=1), and sheets of spindle cells (n=1) (Figure 4).

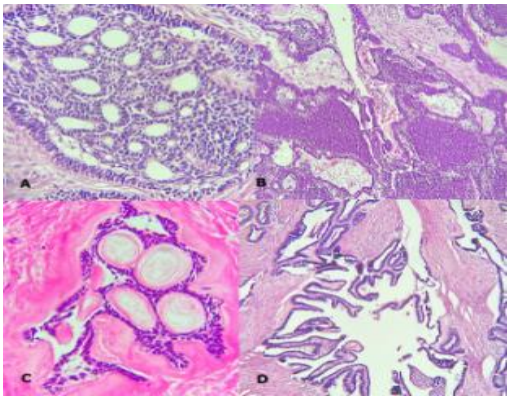

**Figure 1: Ameloblastoma. A: adenoid feature (H&E, ×400). B: hypercellular masses or whorls (H&E, ×100). C: hyalinization of stroma leading to adenoid appearance (H&E, ×400). D: papillary structures (H&E, ×400).**

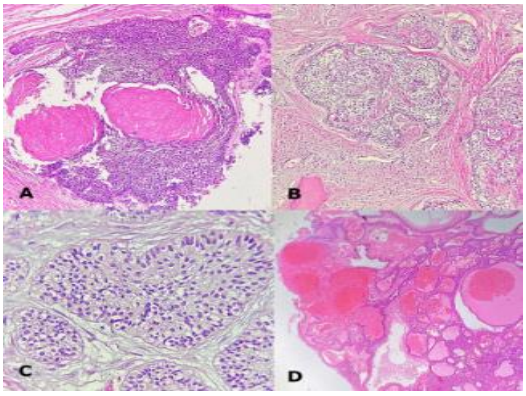

**Figure 2: Ameloblastoma. A: ghost cells (H&E, ×400). B: clear cells (H&E, ×100). C: pure granular cells (H&E, ×400). D: hemangiomatous variant (H&E, ×100).**

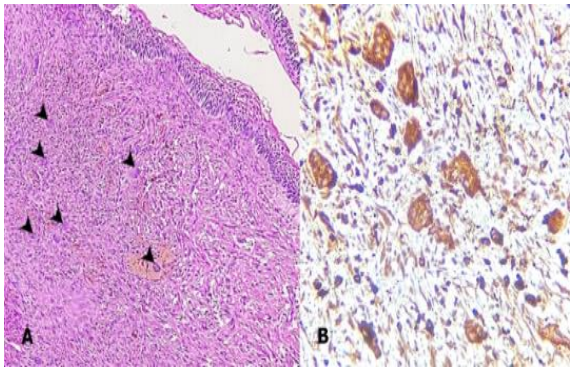

**Figure 3: Unicystic Ameloblastoma. A: ameloblastic epithelium and underlying connective tissue with many giant cells (black arrowheads) (H&E, ×100). B: giant cells showed immunoreactivity with CD68 protein (IHC, ×400).**

### Adenomatoid Odontogenic Tumor (AOT)

The mean age of patients with AOT was 22 years with a female predilection (1.5/1). There was also a mandibular tendency (1.5/1), and infrequent histopathologic features comprised of pure cystic structures (i.e., adenomatoid odontogenic cysts or AOC) and the presence of clear cells.

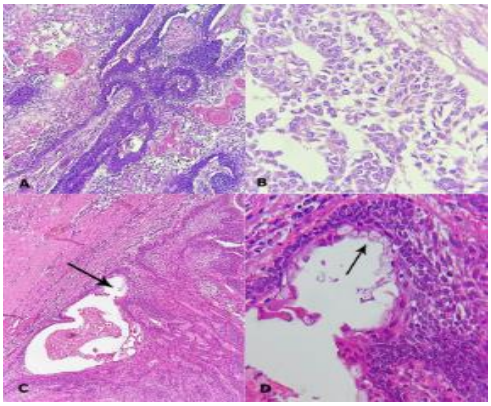

**Figure 4: Ameloblastomic carcinoma. A: keratin pearl formation (H&E, ×100). B: spindle cell proliferation (H&E, ×400). C, D: mucous cells (black arrows) (H&E, ×100, ×400).**

### Central Odontogenic Fibroma (COF)

The mean age of patients with COF was 42 years with no sex or site predilection. One case showed Pindborg-like areas, signet ring cells in odontogenic islands, and calcification in whorls, and the other patient revealed a vast eosinophilic material (negative for amyloid) with scant odontogenic epithelium.

### Ameloblastic Fibro-odontoma (AFO)

A 17-year-old male was diagnosed with AFO and painless expansion of the mandible. Histopathologic sections showed cords and islands of ameloblastic epithelium in the dental papilla-like stroma. Some islands displayed cystic formation and squamous metaplasia.

## Discussion

In the present study, ameloblastoma tumors showed a slight female predilection while conventional types of these tumors show no gender tendency.[7] Mandible was the most common location, and most patients were diagnosed in fourth decade of life. The most common presentation was painless expansion, which agreed with previous reports of common tumor types.[7] Among important infrequent histopathologic presentations were massive keratin pearl formation and individual cell keratinization. Keratoameloblastoma (KA) is a rare subtype of ameloblastoma.[2] Whitt et al. [8] categorized KA into four histopathological groups: [a] Papilliferous, in which the odontogenic epithelium shows papillary projections into cystic spaces; [b] Simple, in which epithelial follicles are lined by ameloblast-like cells with reverse polarity and filled with parakeratin or orthokeratin; [c] Simple with odontogenic keratocyst (OKC)-like structures, mimicking the features of the simple subtype and conventional OKC; and [d] Complex, where epithelial follicles are packed with

parakeratin/orthokeratin and keratin masses extruding into connective tissue stroma and forming Pacinian like stacks. Moreover, cementum or woven bone may be seen in KA[2,8], which can display stellate reticulum, squamous, granular, or basal cell differentiation with considerable keratin pearl deposition in the stroma.[2] Both acanthomatous ameloblastoma and KA are associated with keratinization; however, keratinization is rare in the acanthomatous variant, in which squamous metaplasia is more common. In addition, KA shows keratin deposition in the stroma[2,9]; nevertheless, since a small number of KA cases have been reported so far, it is difficult to verify if the biologic behavior of KA differs from other histologic subtypes of ameloblastoma.[2] Regarding the nonencapsulated nature and locally infiltrative growth pattern of KA, the therapeutic intervention recommended for this type of tumor includes en-block resection along with sufficient safety margins and close clinical follow-up.[2,8,9] During the follow-up period, it is critical not to confuse recurring lesions with the postoperative bone healing process (due to the presence of radiopaque materials).[9] Although marked keratin pearls in the stellate reticulum area were noticed in patients of the present study, considering the previous explanations, these were not classified as either KA or the usual acanthomatous variant.

Adenoid ameloblastoma (AdAM) shows diverse microscopic characteristics reminiscent of adenomatoid odontogenic tumors (AOT) and ameloblastoma with hard tissue (dentinoid) deposition.[6] This tumor is neither encapsulated nor circumscribed and manifests with follicular, plexiform, or solid arrangements, as well as ameloblast-like peripheral columnar cells. Hypercellularity, foci of whorled structures, duct-like spaces, and hyaline droplets (like AOT) may be seen in follicular central zones and plexiform arrangements. Moreover, dentinoid material in association with epithelial cells, Papillary-like patterns, clusters of ghost cells, concentric lamellar basophilic calcified nodules, follicles similar to conventional ameloblastoma, inflammatory cell infiltration, and multinucleated giant cells may be evident. Increased mitotic activity and mild nuclear hyperchromatism have been reported as well.[6] Adorno-Farias et al.[10] mentioned that immunohistochemistry could not help much to distinguish AdAM from conventional ameloblastomas, but microscopic features such as pseudo ducts, squamous metaplasia, nuclear hyperchromatism, clear cells, whorled epithelial structures, cribriform growth pattern, proliferation of spindle cells, and deposition of extracellular eosinophilic materials may be helpful in this regard. Dentinogenic ghost cell tumor (DGCT) is differentiated from AdAM and shows more ghost cells and dysplastic dentin without adenoid differentiation.[6] According to the WHO classification, important microscopic features of AdAM include an ameloblastoma-like component, duct-like structures, whorled cellular condensations, and cribriform architecture. Approximately two-thirds of these neoplasms include varying amounts of dentinoid materials.[11] Interestingly, BRAFp. V600E mutation, which is commonly identified in conventional and unicystic ameloblastoma, is absent in AdAM. Whether AdAM is a unique neoplasm or a microscopic variant of ameloblastoma needs further exploration.[11] This tumor displays an aggressive behavior with a high ki67 proliferation index and a recurrence rate between 45.5% and 70% [11], and its treatment of choice is radical surgery.[6] In the present study, adenoid formation (n=1), adenoid and papillary areas (n=1), adenoid plus hyalinization (n=1), and hypercellular whorls (n=2) were seen in five patients, who might be classified as AdAM (Figure 2) despite not presenting all diagnostic criteria of typical AdAM.

In HA tumors, numerous blood-filled spaces or large endothelial-lined capillaries in the connective tissue are characteristic.[12] Kasangari et al.[12] summarized various theories about the pathogenesis of HA as follows: 1. Angiogenesis during odontogenesis, 2. Development of a separate tumor, 3. Hamartomatous abnormality, 4. Traumatic event (formation of the granulation tissue), 5. Secondary changes, and 6. Formation of a collision neoplasm. Most HAs depict microscopic features of plexiform ameloblastoma (anastomosing cords) with prominent vascular components, and the epithelium is surrounded by columnar or cuboidal ameloblast-like cells surrounded with stellate reticulum-like areas.[3,12] Cystic transformation and necrotic areas may be seen in the stroma.[3] In the present study, two HA cases showed plexiform features.

Granular cytoplasmic changes in the stellate reticulum of ameloblastoma are well-characterized, but rarely, granular changes can also be seen in peripheral ameloblastic cells. In these cases, microscopic diagnosis is problematic and thought-provoking because of its close resemblance to salivary gland tumors.[13,14] One of the patients in the present study showed pure granular cells with an immense cystic formation that, at first glance, mimicked salivary tumors such as oncocytoma.

The presence of mucous cells is an exceedingly rare finding in ameloblastoma.[15] Mucous cells are organized in several arrangements. For example, they may form glands, be dispersed singly in nests among the central cells of ameloblastoma follicles, be present in areas of cystic degeneration lining cyst cavities or be seen as linear arrays in the areas of cystic degeneration and as clumps in solid ameloblastoma islands.[5] The presence of mucous cells in ameloblastoma is believed to represent the multipotentiality of the odontogenic epithelium.[5] In the present study, one patient diagnosed with ameloblastic carcinoma showed

mucous cell differentiation in cystic spaces.

Ameloblastoma is rarely associated with the presence of ghost cells. In this regard, DGCTs are composed of ameloblastomatous epithelial islands with areas of ghost cell formation and dentinoid material deposition.[16] Two distinctive features that differentiate DGCTs from ameloblastoma include the presence of numerous ghost cells and masses of dentinoid materials in the former.[17]

Clear cell ameloblastoma (CCAM) is histologically identified as ameloblastoma with an extensive clear cell component.[18] Due to the reported metastatic potential of this entity, it seems that this tumor bears a cancerous potential, and the term CCAM may be misleading.[18] Some have suggested this tumor as a subset of clear cell odontogenic carcinoma (COdC) rather than a separate entity.[19] In none of our patients, the presence of clear islands was enough to diagnose COdC. The ratio of clear cells to distinguish between these two entities has not been determined. Braunshtein et al.[18] stated that COdC and CCAM should be regarded as low-grade malignancies and could represent a clinicopathologic spectrum of a single entity rather than two separate lesions. It should be noted that COdC is characterized by EWSR1 gene rearrangements in about 80% of cases.[11] This mutation is not present in ameloblastoma. In the present study, none of the cases showed histopathological features such as cellular atypia or increased mitosis or necrosis, while the ameloblastic pattern was the predominant feature, which could help discern this entity from carcinomas. Another issue to be considered was the possibility of clear cell changes due to improper fixation.

Multinucleated giant cells are well-recognized in various malignancies but have rarely been defined in odontogenic neoplasms. Giant cells associated with tumors are divided into neoplastic and nonneoplastic categories. Giant cells in our patients were positive for CD68, which was in agreement with previous reports.[7,20] Sekhar et al.[20] mentioned that the presence of giant cells in close proximity to the tumor might show that these giant cells are parts of a foreign-body giant cell response. To date, only three sporadic cases of unicystic ameloblastoma with stromal giant cells have been reported in the literature, and the patient reported in this study is the fourth.[7,20]

The spindle cell variant of ameloblastic carcinoma is an exceedingly rare neoplasm but is easily distinguishable by detecting ameloblastic islands. One of the leading criteria to differentiate the spindle cell variant of ameloblastic carcinoma from odontogenic carcinosarcoma is the lack of ameloblastic fibrosarcoma-like patterns in the former.[21] In the present study, the diagnosis was easily established due to the presence of ameloblastic islands.

Pure cystic AOT was also diagnosed in the present study. As a non-aggressive epithelial odontogenic neoplasm, AOT presents with diverse histopathologic characteristics. The cut surface of AOT is mainly solid or may show some degrees of cystic changes. In some cases, the solid part may be seen only as masses in the connective tissue wall of a cyst.[22] Some studies have reported cases of purely cystic AOT[23,24], and some authors have preferred the name AOC[23], while others have reiterated its tumoral nature.[25,26] Clear cell changes may also be seen in AOT, which can signify cellular degeneration or the odontogenic epithelial origin of the lesion.[27] In this study, one patient showed the presence of numerous clear cells, mimicking clear cell carcinoma, but the thick capsule and duct-like areas suggested the diagnosis of AOT.

COF is a mesenchymal odontogenic tumor.[28] In this study, a long-lasting case of COF was encountered in a 69-year-old male with radiolucency of the left maxilla. There were no clinical signs except for an indentation in the palatal mucosa in the alignment of the maxillary left first molar. Histopathologic sections showed numerous amyloid-negative eosinophilic materials along with scattered odontogenic islands. Another patient with COF revealed Pindborg-like areas, signet ring cells in odontogenic islands, and extensive whorl calcification.

Finally, a patient with aggressive AFO and extensive bone destruction was described in this study, accompanied by squamous cell metaplasia and microcyst formation in ameloblastic islands. Currently, AFOs are not considered separate entities in the WHO classification, and they are placed in the spectrum of histopathologic changes observed in a developing odontoma. Nevertheless, AFOs may show large sizes and can arise in age groups inconsistent with hamartoma.[29] Soluk-Tekkesin et al.[30] mentioned that the combination of age and lesion size may be helpful in discerning lesions with a true neoplastic nature (e.g., AFO) from hamartomatous masses (i.e., odontoma), suggesting that at least some AFOs, especially those larger than 2.1 cm detected in individuals younger than 13.5 years, should be considered true tumors.

## Conclusion

The infrequent microscopic patterns of common odontogenic tumors were described in this study. It is essential for pathologists to be familiar with these features to reach a reliable and proper diagnosis before administering efficient treatments. In this study, although all diagnostic criteria of adenoid tumors were not met, at least some features of AdAM were observed in five patients.

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## References

1. Wright JM, Soluk Tekkesin M. Odontogenic tumors: where are we in 2017? J Istanb Univ Fac Dent. 2017;51(3 Suppl 1):S10-30.
2. Palaskar SJ, Pawar RB, Nagpal DD, Patil SS, Kathuriya PT. Keratoameloblastoma a rare entity: a case report. J Clin Diagn Res. 2015;9(3):ZD05-7.
3. van Rensburg LJ, Thompson IO, Kruger HE, Norval EJ. Hemangiomatous ameloblastoma: clinical, radiologic, and pathologic features. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;91(3):374-80.
4. Atarbashi Moghadam S, Atarbashi Moghadam F, Mokhtari S, Eini E. Immunohistochemical analysis of P63 expression in odontogenic lesions. Biomed Res Int. 2013;2013:624176.
5. Punnya AV, Rekha K. "Ameloblastoma with mucous cells": review of literature and presentation of 2 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;106(6):e20-6.
6. Loyola AM, Cardoso SV, de Faria PR, Servato JPS, Eisenberg ALA, Dias FL, et al. Adenoid ameloblastoma: clinicopathologic description of five cases and systematic review of the current knowledge. Oral Surg Oral Med Oral Pathol Oral Radiol. 2015;120(3):368-77.
7. Robinson L, Fonseca FP, Lacerda JCT, van Heerden WF. Unicystic ameloblastoma with stromal giant cells: a case series of a rare entity. Oral Surg Oral Med Oral Pathol Oral Radiol. 2022;133(1):e20-5.
8. Whitt JC, Dunlap CL, Sheets JL, Thompson ML. Keratoameloblastoma: a tumor sui generis or a chimera? Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007;104(3):368-76.
9. Lee C, Park BJ, Yi WJ, Heo MS, Lee SS, Huh KH. Keratoameloblastoma: a case report and a review of the literature on its radiologic features. Oral Surg Oral Med Oral Pathol Oral Radiol. 2015;120(5):e219-25.
10. Adorno-Farias D, Muniz VRVM, Soares AP, Cury PR, Rabelo RG, Fernández-Ramires R, et al. Ameloblastoma with adenoid features: a series of eight cases. Acta Histochem. 2018;120(5):468-76.
11. Vered M, Wright JM. Update from the 5th edition of the world health organization classification of head and neck tumors: odontogenic and maxillofacial bone tumours. Head Neck Pathol. 2022;16(1):63-75.
12. Kasangari MD, Gundamaraju K, Jyothsna M, Subash AV, Aravind K. Hemangiomatous ameloblastoma-a case report of a very rare variant of ameloblastoma. J Clin Diagn Res. 2015;9(5):ZD08-10.
13. Argyris PP, McBeain MJ, Rake A, Pambuccian SE, Gopalakrishnan R, Koutlas IG. Recurrent ameloblastoma of the mandible with unusual granular cell component. Int J Surg Pathol. 2015;23(4):298-304.
14. Oza N, Agrawal K. Granular cell ameloblastoma mimicking oncocytoma. J Oral Maxillofac Pathol. 2012;16(3):446-9.
15. Wilson D, Walker M, Aurora N, Moore S. Ameloblastoma with mucous cell differentiation. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;91(5):576-8.
16. Mehendiratta M, Bishen KA, Boaz K, Mathias Y. Ghost cells: a journey in the dark. Dent Res J (Isfahan). 2012;9:S1-8.
17. Singhaniya SB, Barpande SR, Bhavthankar JD. Dentinogenic ghost cell tumor. J Oral Maxillofac Pathol. 2009;13(2): 97-100.
18. Braunshtein E, Vered M, Taicher S, Buchner A. Clear cell odontogenic carcinoma and clear cell ameloblastoma: a single clinicopathologic entity? a new case and comparative analysis of the literature. J Oral Maxillofac Surg. 2003;61(9):1004-10.
19. de Aguiar MC, Gomez RS, Silva EC, de Araújo VC. Clear-cell ameloblastoma (clear-cell odontogenic carcinoma): report of a case. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1996;81(1):79-83.
20. Sekhar MM, Kiran G, Reddy KR, Krishna A, Suri C. Multinucleated giant cells in unicystic ameloblastoma: a very rare case report and review of the literature. Adv Hum Biol?. 2015;5(1):56-9.
21. Matsushita Y, Fujita S, Yanamoto S, Yamada S, Rokutanda S, Yamashita K, et al. Spindle cell variant of ameloblastic carcinoma: a case report and literature review. Oral Surg Oral Med Oral Pathol Oral Radiol. 2016;121(3):e54-61.
22. Sargolzaei S, Atarbashi-Moghadam S, Aghdashi F, Mansouri Nejad S. Extrafollicular cystic adenomatoid odontogenic tumor of the maxilla: a rare challenging case report with review of the literature. J DENT MATER TECH. 2018;7(3):141-4.
23. Kumar R, Hashmi G, Amjad S, Aansari M. Adenomatoid odontogenic cyst of mandible: a rare case report. Int J Health Sci Res?. 2015;5(5):490-4.
24. Grover S, Rahim AM, Parakkat NK, Kapoor S, Mittal K, Sharma B, et al. Cystic adenomatoid odontogenic tumor. Case Rep Dent. 2015;2015: 503059.
25. Thakur A, Tupkari JV, Joy T, Hanchate AV. Adenomatoid odontogenic tumor: what is the true nature? Med Hypotheses. 2016;97:90-3.
26. Philipsen HP, Khongkhunthiang P, Reichart PA. The adenomatoid odontogenic tumour: an update of selected issues. J Oral Pathol Med. 2016;45(6):394-8.
27. Mohanty N, Routray S, Swain N, Ingale Y. Adenomatoid odontogenic tumor with clear cell changes. Indian J Pathol Microbiol. 2014;57(2):290-3.
28. Zhou CX, Li TJ. A clinicopathologic study on central odontogenic fibroma: with special reference to amyloid variant. Oral Surg Oral Med Oral Pathol Oral Radiol. 2018;126(6):513-20.
29. Atarbashi-Moghadam S, Ghomayshi M, Sijanivandi S. Unusual microscopic changes of ameloblastic fibroma and ameloblastic fibro-odontoma: a systematic review. J Clin Exp Dent. 2019;11(5): e476-81.
30. Soluk-Tekkesin M, Vered M. Ameloblastic fibro-odontoma: at the crossroad between" developing odontoma" and true odontogenic tumour. Head Neck Pathol. 2021;15(4):1202-11.