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Evaluation of early malignant Changes of dentigerous Cyst into central mucoepidermoid carcinoma by immunohistochemical of Ki67, p53, bcl2 and periodic acid Schiff mucin stain

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Abstract

Odontogenic cysts, such as dentigerous cysts, are common jaw lesions, often associated with impacted teeth and presenting as asymptomatic radiolucencies. While typically benign, they may undergo neoplastic transformation into aggressive lesions like ameloblastomas or central mucoepidermoid carcinomas (MEC). This study analyzed a histopathological lesion initially diagnosed as a dentigerous cyst using H&E, p53, Ki-67, Bcl-2, PAS, and Mucicarmine staining to assess cellular composition and malignant potential. Histological findings revealed a stratified squamous epithelial lining with mucous metaplasia, chronic inflammation, and an intact basement membrane.

Immunohistochemistry showed low p53 expression, basal-layer Ki-67 positivity, and localized Bcl-2 staining, suggesting low proliferative activity. PAS staining confirmed basement membrane integrity, while Mucicarmine detected focal mucous cell metaplasia. The presence of mucous, epidermoid, and intermediate cells raised suspicion of early-stage MEC; however, the absence of significant atypia or diffuse proliferative markers supported the diagnosis of a dentigerous cyst with mucous metaplasia. A rare case of low-grade intraosseous MEC was identified in a 47-year-old woman, progressing untreated for two years before symptomatic presentation. Surgical enucleation and histopathological analysis confirmed epithelial dysplasia and mucous metaplasia, consistent with MEC. A four-year follow-up showed complete bone regeneration without recurrence. This case emphasizes the necessity of early diagnosis, routine monitoring, and comprehensive histopathological evaluation of odontogenic cysts to prevent malignancy. Conservative management is effective for benign lesions, while molecular studies (MAML2, P63, Bcl-2 gene rearrangement) are recommended for cases with neoplastic potential.

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1. Introduction

The mandibular and maxillary jaws act as hosts for cysts and tumors due to tissue variations involved in tooth formation.[1] Odontogenic cysts are pathological cavities lined with epithelium and filled with fluid, semisolid, or gaseous material, which develop from remnants of the odontogenic apparatus, while odontogenic tumors are solid tissue masses that are not necessarily neoplastic.[2,3] Approximately 25% of all odontogenic cysts of the jaws are dentigerous cysts.[4] Radiographically, they are represented by a well-defined unilocular radiolucent area, usually involving the crown of an impacted tooth. They are primarily associated with the mandibular third molar, followed by the maxillary canines, mandibular premolars, maxillary third molars, and supernumerary teeth.[5,6] The peak incidence occurs in the second and third decades of life.[6,7] These cysts are usually asymptomatic and are detected during routine radiographic examinations. The occurrence of this cyst in the first decade of life is relatively low, at approximately 4 to 7%. Histologically, they are characterized by a cavity lined with nonkeratinizing thin epithelium without rete pegs.[6,8] Dentigerous cysts, along with other odontogenic cysts, despite their high prevalence, have the potential to transform into neoplastic lesions such as ameloblastomas, adenomatoid odontogenic tumors, and even non-odontogenic malignant tumors.[9] However, the frequency of these neoplastic transformations is known to be low.[9] Dentigerous cysts also have the potential to transform into mucoepidermoid carcinoma of the jaws.[10,11] Mucoepidermoid carcinoma (MEC) is the most common malignant salivary gland neoplasm, accounting for 2.8% to 15% of all salivary gland tumors.[12] Aberrant salivary gland neoplasms arising within the jaws as primary central bony lesions are extremely rare, with central mucoepidermoid carcinoma being the most common.[9] Several case reports have been recorded in the literature regarding the neoplastic transformation of the epithelial lining of odontogenic cysts.[13,14] The pathogenesis of central mucoepidermoid carcinoma (CMEC) is still unknown. However, it has been suggested that the origin may be from ectopic salivary glands, metaplastic transformation of odontogenic epithelium, or neoplastic transformation of the epithelial lining of odontogenic cysts.[15,16] This lesion is more frequent in the mandible than in the maxilla, and it typically occurs in the 4th and 5th decades of life.[13,17] It affects females more frequently than males.[14] Histopathologically, MEC contains three main cell types: mucin-producing, epidermoid, and intermediate cells.[17,18] In this article, we report a rare case of central mucoepidermoid carcinoma (CMEC) arising from the malignant transformation of the epithelial lining of a dentigerous cyst in the posterior region of the mandible.

2. Case report

This study presents a rare and clinically significant case of a dentigerous cyst (DC) undergoing malignant transformation into low-grade intraosseous mucoepidermoid carcinoma (MEC). This phenomenon, though uncommon, underscores the importance of early detection and meticulous histopathological and immunohistochemical evaluation in managing long-standing odontogenic cysts .

3. Clinical Background: A 47-year-old woman was referred for an orthodontic consultation. She had multiple missing teeth and severe crowding in the anterior teeth. After clinical and radiographic examination, including an orthopantomogram (OPG) and lateral cephalogram, a single, unilateral, well-defined radiolucent area was observed extending to the cemento-enamel junction of the right mandibular impacted third molar. The cystic lesion measured approximately 2 cm, consistent with the appearance of a dentigerous cyst. The crown of the unerupted tooth was in a distoangular position [Figures-1: a&b]. The patient was referred to the oral medicine and maxillofacial department for the surgical removal of the cystic lesion and the associated impacted tooth before orthodontic treatment; however, she refused the surgical intervention.

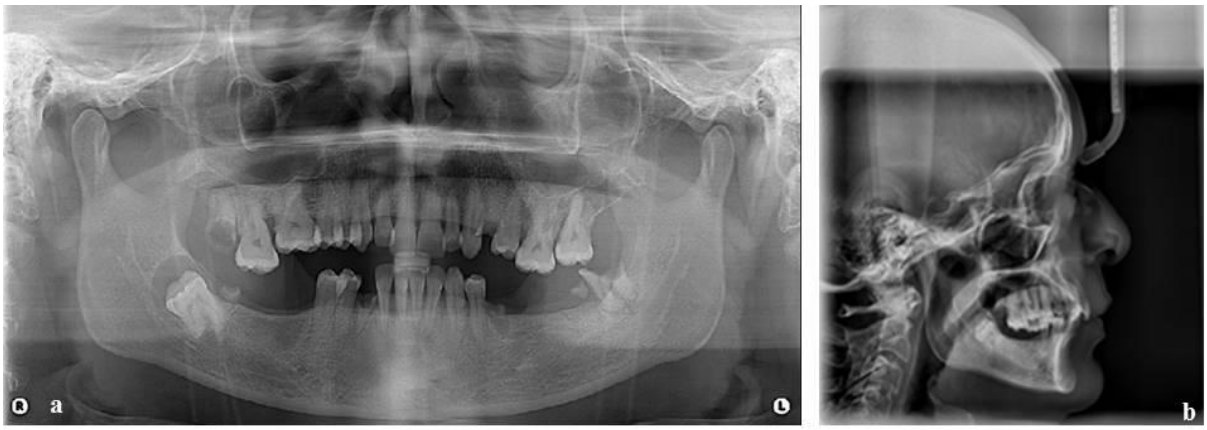

Figure-1: Initial radiographic appearance of cystic lesion (a) Panoramic X-ray. (b) lateral cephalometic

4. Clinical Update: After two years, the patient presented to the Department of Oral Medicine and Maxillofacial Surgery at Misurata's Faculty of Dentistry with the chief complaint of mild discomfort and an associated intraoral swelling in the right posterior region of the mandible, near the impacted third molar. The swelling had been present for one month. The patient had no significant medical issues, drug allergies, family history, or tobacco use. On extraoral examination, there was no facial asymmetry, and the skin appeared normal. The submandibular, submental, and cervical lymph nodes were neither palpable nor tender. Intraoral examination revealed a small, smooth, slightly firm, and tender swelling in the posterior right part of the mandible. The cortical bone was not involved, there was no tooth mobility, and the overlying mucosa was intact. The right mandibular third molar was clinically absent. Additional diagnostic tests, such as radiographic imaging, were recommended to refine the diagnosis.

5. Radiological Investigations: The orthopantomogram and lateral cephalogram radiographs taken two years ago revealed a well-defined unilocular radiolucency surrounding the crown at the cervical region of the distally inclined impacted right mandibular third molar, as shown previously in (Figures-1: a&b), which were taken during the orthodontic consultation.

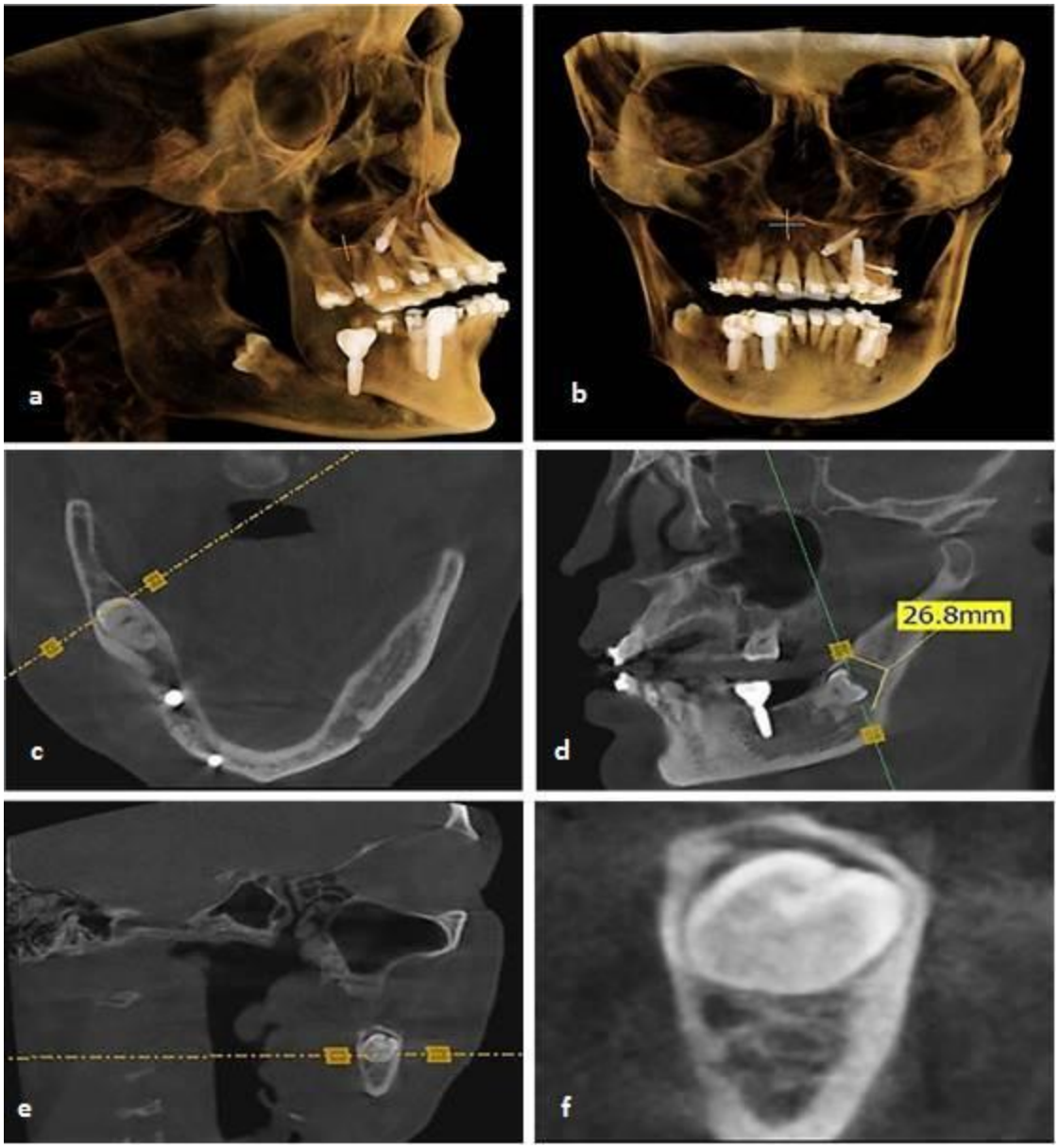

Figure-2: Cone beam volumetric tomography showing the lesion boundariesa, b, c, d, e, and f show the extension of unilocular radiolucency surrounding the impacted right mandibular third molar, indicating a dentigerous cyst, with no cortical perforation.

We decided to remove the dentigerous cyst. The patient underwent surgical enucleation of the cyst, along with the unerupted third molar, under local anesthesia. Enucleation of the cystic

The CBCT scan showed that the cyst had slightly enlarged, measuring 2.68 cm, extending across To further evaluate the expansion of the lesion, cone beam computed tomography (CBCT) was recommended. the alveolar crest. However, there was no evidence of cortical destruction or lateral perforation of the mandibular bone (Figures- 2: a, b, c, d, e, f).

and radiographic findings, a provisional diagnosis of a dentigerous cyst was made. The excised tissue was sent for histopathological examination for further evaluation. lesion was performed with adequate surgical margins (Figures 3a, b, c). Based on the clinical and radiographic findings, a provisional diagnosis of a dentigerous cyst was made. The excised tissue was sent for histopathological examination for further evaluation.

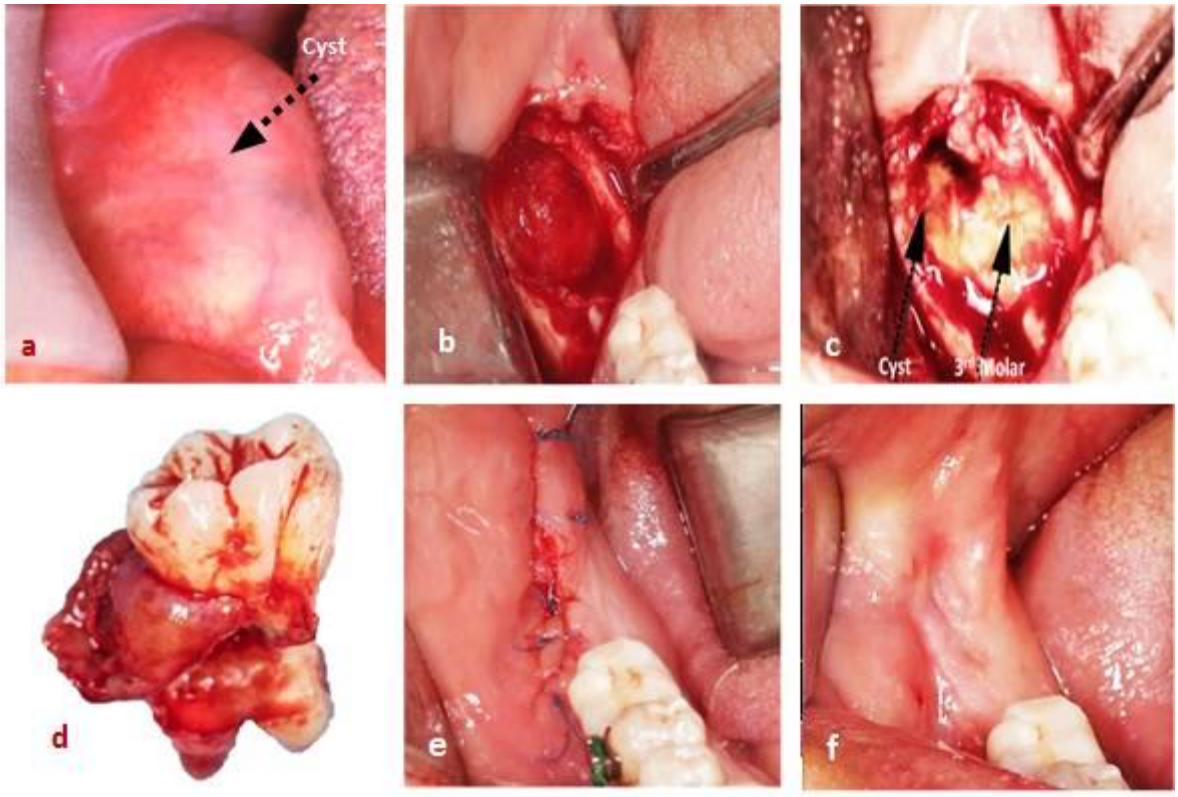

Figure-3: (a) Cyst (arrow pointing to the cyst). **(b**) Exposed cyst. **(c)** Cyst and 3rd mola**r** (arrows pointing to each structure). **(d)**Cyst with extracted third molar. **(e)** Surgical site with sutures. **(f)** Healed surgical site.

The patient was followed up clinically and radiographically every three months for one year (Figure-3-d), then annually for four years. (Figure-4) shows the final clinical and radiographic appearance four years after surgery, with complete new bone formation observed. After this follow-up period, the patient was discharged, as there was no clinical or radiographic evidence of recurrence.

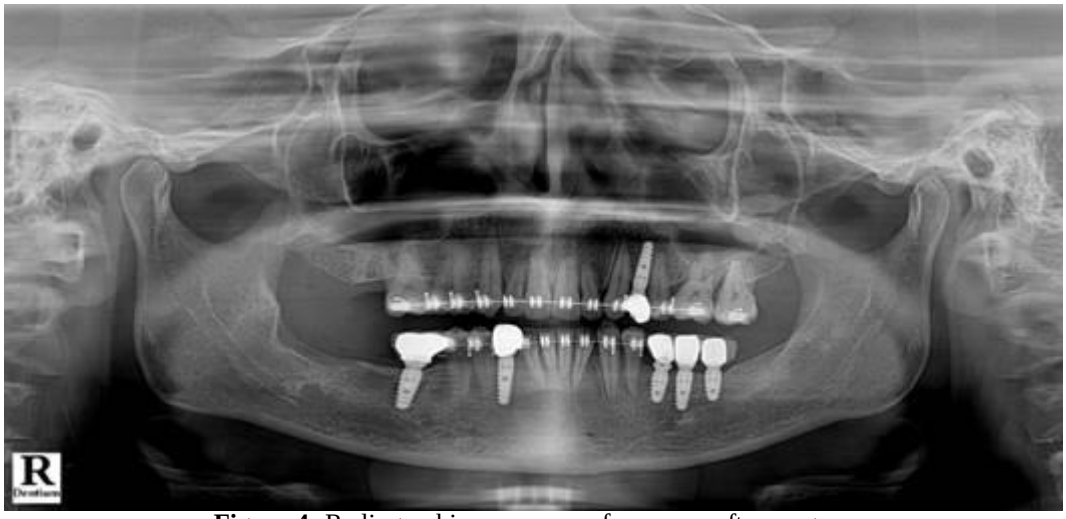

Figure 4: Radiographic appearance four years after surgery.

6. Histopathological Findings: Microscopic analysis of the cystic specimen section, stained with **Hematoxylin and Eosin (H&E)** (Figure-5), revealed that the epithelial lining consists of non-keratinized stratified squamous epithelium. This epithelium appears thin and uniform, typically comprising 2–4 cell layers in most areas. Evidence of focal hyperplasia or epithelial thickening is observed, likely as a response to prolonged chronic inflammation. The basal layer of the epithelium exhibits palisading with cuboidal to low columnar cells. The nuclei are hyperchromatic, reflecting active cellular processes. The subepithelial connective tissue contains a mix of loose to dense collagen fibers. Chronic inflammation is evident, characterized by the presence of lymphocytes and plasma cells scattered within the stroma. Vascular proliferation, indicated by capillaries, is also noted. Additionally, mucous metaplasia (goblet cells) is observed. In rare cases, mucous metaplasia within the epithelial lining may predispose the lesion to malignant transformation, such as mucoepidermoid carcinoma. Furthermore, epithelial thickening can lead to the formation of rete pegs. There is focal evidence of mucous cells coexisting with epidermoid and possibly intermediate cells, suggesting a combination of cell types. The presence of all three cell types raises the suspicion of mucoepidermoid carcinoma (MEC). Other cystic changes include the presence of empty or clear spaces and white areas (marked with black arrowheads), which may represent areas of degeneration, edema, or remnants of cystic fluid. These histological observations collectively suggest a low-grade lesion with potential dysplastic transformation. Further supplemental evaluation using immunohistochemical markers (e.g., Ki-67, p53, Bcl-2, PAS, and Mucicarmine stains) is essential to confirm or rule out the diagnosis.

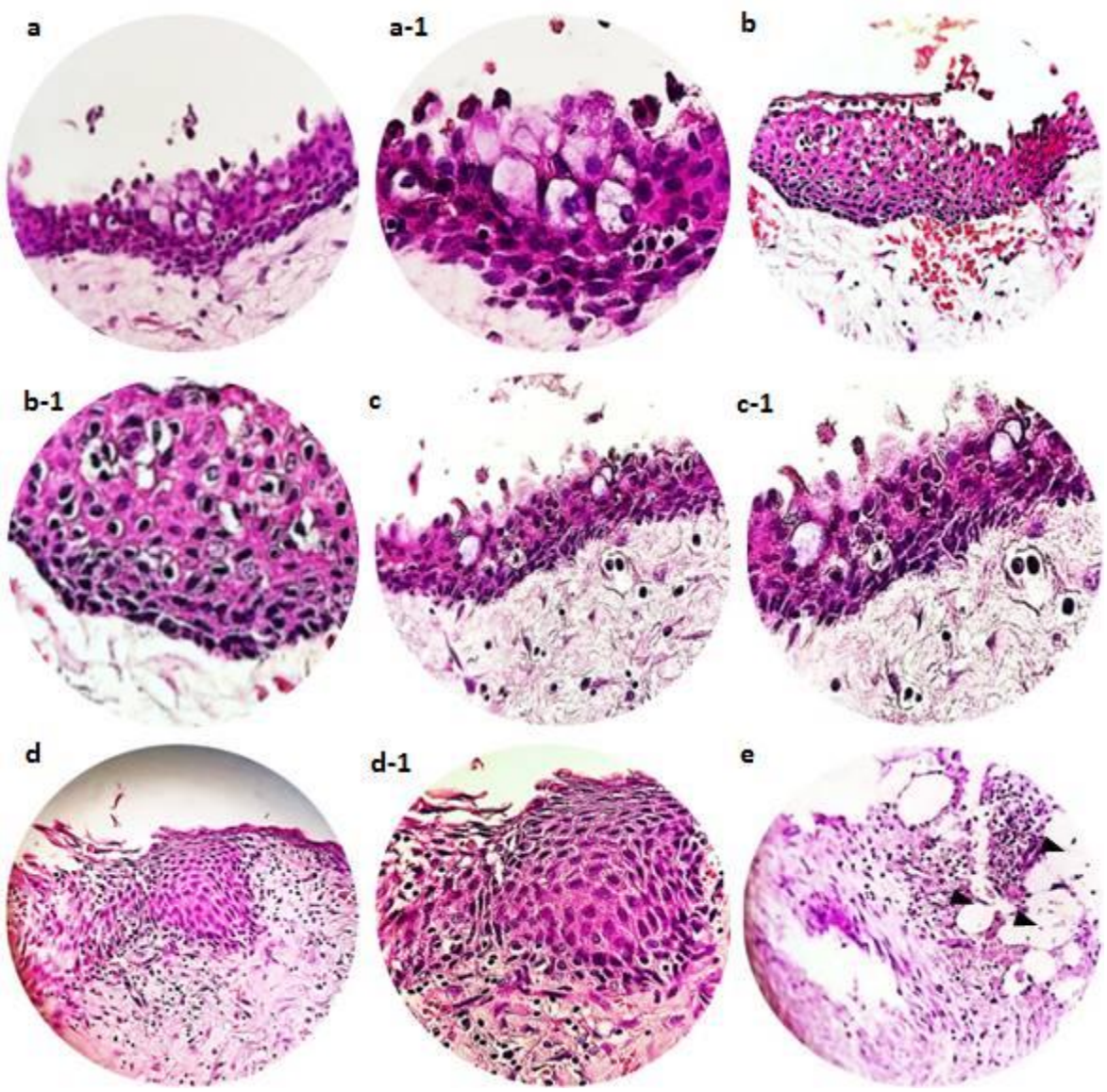

Figure-5: Histological images is identified a dentigerous cyst lesion, stained with H&E stain, reveal **(a)** Low-power view of the cystic lining showing Thin, non-keratinized stratified squamous epithelium lining the dentigerous cyst, with a flat interface between epithelium and connective tissue, there is evidence of mucous cell metaplasia interspersed within the basal and parabasal layers. **(a-1)** High-power magnification of **(a)** highlighting mucous cell clusters with pale, vacuolated cytoplasm indicative of mucous cell metaplasia, alongside basal cell hyperplasia. **(b)** Low-power Hyperplastic epithelial lining, Chronic inflammatory cell infiltration in the cyst wall, with evidence of vascular congestion. **(b-1)** High-power magnification of **(b)** showing a epidermoid, intermediate cells and clear cluster of mucous cells with intracellular mucin accumulation, along with a dense inflammatory infiltrate in the underlying connective tissue. **(c)** Stratified squamous epithelium exhibiting focal vacuolar degeneration and mucous cells interspersed among basal and parabasal layers, indicating progressive metaplastic changes. **(c-1)** High-power view of **(c)** showing well-defined mucous cells with mucin-rich cytoplasm and mild surrounding inflammation. **(d)** Fibrous connective tissue wall with chronic inflammatory infiltrates and reactive epithelial hyperplasia, including mucous cell metaplasia. **(d-1)** High-power view of **(d)** showing scattered mucous cells with intracellular mucin and focal loss of basal cell polarity. **(e)** Cystic cavity showing necrotic debris, disrupted epithelium, and mucous cell-rich areas.

Mucicarmine and PAS stains demonstrate mucous production, consistent with mucous metaplasia or mucin-producing neoplasms (Figure-6-(a),(a-1),(b),(b-1))**.** Periodic Acid-Schiff (PAS) Staining images shows positive PAS (magenta areas) highlights glycogen, mucopolysaccharides, or basement membranes. Cytoplasmic positivity in certain cells and extracellular material suggests the presence of mucins or other glycogen-rich substances (Figure-6-(c),(c-1),(d),(d-1),e). That indicate mucous production, consistent with mucous metaplasia or mucin-producing neoplasms.

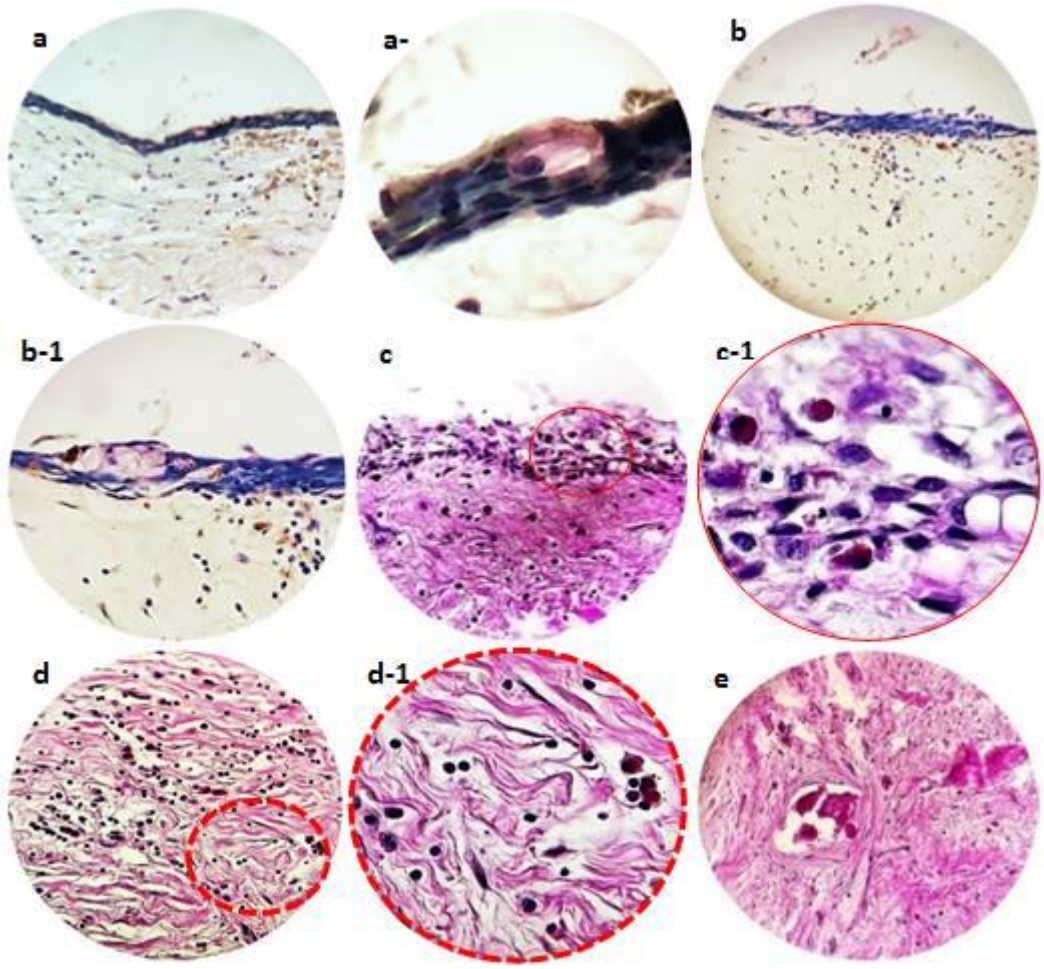

Figure-6: The images are stained with mucicarmine and PAS (Periodic Acid-Schiff) show respectively, **(a)** Low-power mucicarmine stain showing epithelial lining of the cyst with faint mucin positivity in the basal and parabasal layers. **(a-1)** Higher magnification of (a) showing intracellular mucin accumulation within the epithelial lining. **(b)** Mucicarmine stain revealing connective tissue with scattered areas of mucin-rich cells and minimal inflammatory infiltrates. **(b-1)** Higher magnification of (b) highlighting mucin-producing cells within the epithelial lining. **(c)** PAS stain showing strong positivity in the epithelial basement membrane and mucous cells. **(c-1)** Higher magnification of (c) revealing PAS-positive mucin granules in epithelial mucous cells. **(d)** PAS stain showing fibrous connective tissue with PAS-positive deposits, indicating glycogen-rich structures. **(d-1)** Higher magnification of (d) showing clearly defined PAS-positive mucopolysaccharides deposits in the connective tissue. **(e)** PAS stain demonstrating cystic cavity contents with necrotic debris and scattered mucinous material.

7. Immunohistochemistry Findings:

- P53 immunohistochemical staining shows positive nuclear staining, where the nuclei of certain cells are stained brown, that indicating P53 expression. Strong and diffuse staining in some areas, and other areas show lighter or absent staining, which could represent regions of normal or non-neoplastic tissue with no significant P53 alterations. The cytoplasm and extracellular matrix show minimal to no staining, which is expected, as P53 expression is typically confined to the nucleus. Positive nuclear staining is evident in specific areas, possibly indicating areas of dysplasia, or reactive changes (Early neoplastic transformation). Areas with minimal staining may represent non-dysplastic or reactive regions (Figure- 7-(a),(a-1),(b),(b-1)).

- Ki67 (Proliferation Marker) shows nuclear staining in a number of cells is evident, marking proliferative activity. Ki67 staining intensity and distribution are uneven, suggesting focal areas of high proliferation. The strong nuclear positivity in basal and parabasal layers indicates increased turnover, supporting a diagnosis of epithelial dysplasia (Figure- 7- (c),(c-1),(d),(d-1))**.**

- Bcl-2 (Anti-apoptotic Marker) shows positive brown staining in the cytoplasm of certain cells indicates expression of the anti-apoptotic protein Bcl-2. That is more pronounced in the basal layer or specific cell clusters, this indicate resistance to apoptosis which can be a feature of neoplastic or dysplastic tissues, as it supports cell survival (Figure- 7- (e),(e-1),(f),(f-1)).

To conclude the observations of H&E, mucicarmine, PAS, and immunohistochemical staining which showed positive markers with P53 (2.1%), Ki67 (10.6%), and Bcl2 (15.8%), confirming a final diagnosis of early and low-grade intraosseous mucoepidermoid carcinoma (MEC) of the mandible arising from a long standing dentigerous cyst (DC).

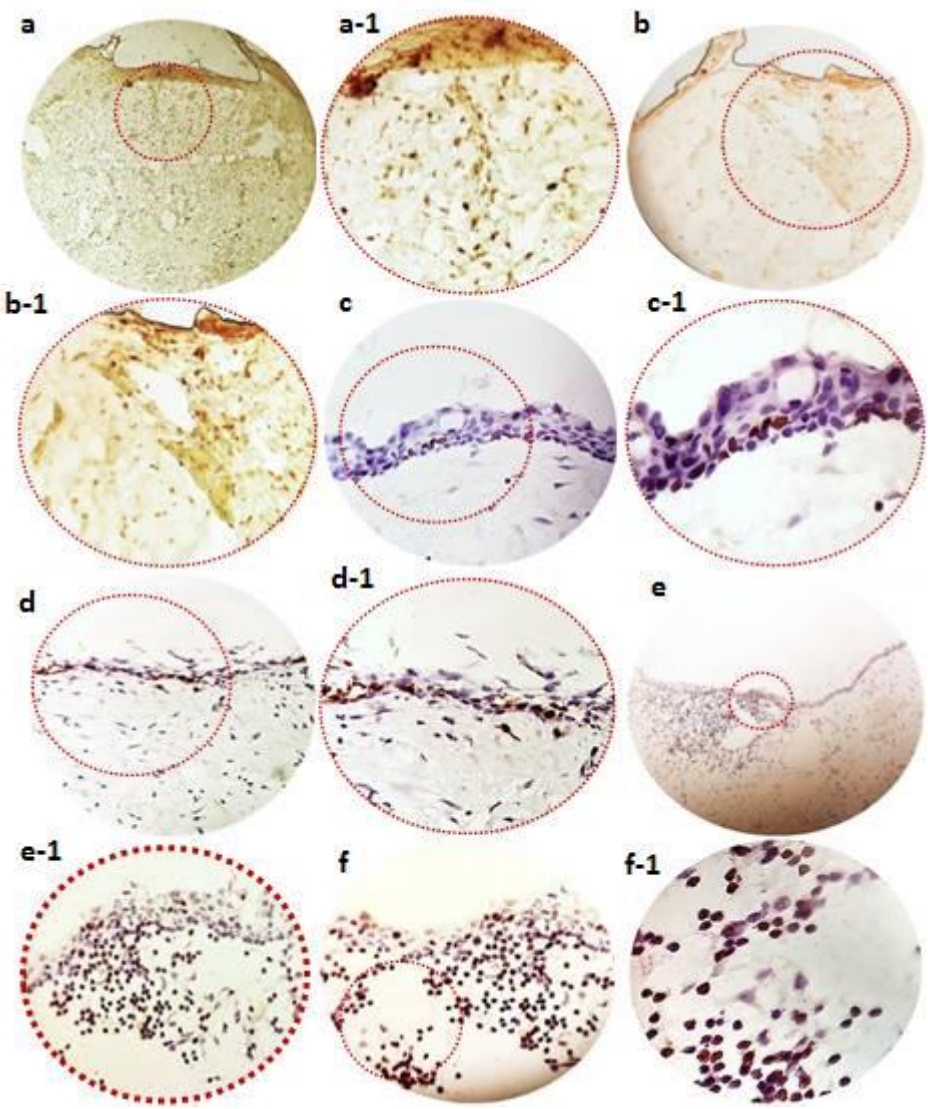

Figure-7: The images of (IHC) staining for **p53**, **Ki-67**, and **Bcl-2** show: **(a)** Low-power view of p53 immunostaining showing patchy positivity in the epithelial lining of the cyst. **(a-1)** High-power view of **(a)** highlighting nuclear positivity for p53 in basal and parabasal epithelial cells. **(b)** Another section showing p53 expression with focal staining in the epithelium. **(b-1)** High-power magnification of **(b)** demonstrating strong nuclear staining for p53, indicating potential dysplastic changes. **Ki-67 Stain (Proliferation Marker): (c)** Ki-67 immunostaining at low power, showing nuclear positivity in the basal and parabasal layers of the epithelial lining. **(c-1)** High-power view of **(c)** showing increased Ki-67 expression in proliferative basal epithelial cells, indicating cellular proliferation. **(d)** Ki-67 staining with scattered positivity in the basal epithelial layers and occasional subepithelial inflammatory cells. **(d-1)** High-power magnification of **(d)** showing strong nuclear staining in actively proliferating cells. **Bcl-2 Stain (Anti-apoptotic Marker): (e)** Low-power view of Bcl-2 immunostaining showing diffuse positivity in the basal epithelial layers of the cyst lining. **(e-1)** High-power view of **(e)** highlighting cytoplasmic Bcl-2 expression in basal cells, indicating increased anti-apoptotic activity. **(f)** Another section stained with Bcl-2 showing diffuse staining in the epithelial and subepithelial regions. **(f-1)**

High-power magnification of **(f)** revealing strong cytoplasmic staining for Bcl-2 in basal epithelial cells, consistent with enhanced cell survival.

8. Final Diagnosis: Long-standing Dentigerous Cyst with Reactive and Dysplastic Changes Based on the provided histological and immunohistochemical findings respectively : Non-keratinized stratified squamous epithelial lining with hyperplasia and occasional mucous cell metaplasia. Presence of chronic inflammatory infiltrates and fibrosis in the connective tissue wall. Degeneration of epithelial lining in areas, with occasional necrotic debris. Immunohistochemical Findings: p53: Patchy nuclear positivity, indicating reactive dysplastic changes or potential early transformation. Ki-67: Increased nuclear positivity in basal and parabasal layers, suggesting active cellular proliferation. Bcl-2: Diffuse cytoplasmic positivity in basal epithelial cells, indicating enhanced anti-apoptotic activity. These findings suggest a long-standing dentigerous cyst with evidence of inflammation, hyperplasia, and reactive dysplasia, but without overt malignancy. However, the presence of increased p53 and Ki-67 expression warrants close monitoring for potential malignant transformation (e.g., Early mucoepidermoid carcinoma).

9. Discussion:

The case highlights the rare occurrence of low-grade intraosseous mucoepidermoid carcinoma (MEC) arising from a dentigerous cyst (DC). Low-grade central mucoepidermoid carcinoma (CMEC) and glandular odontogenic cyst (GOC) exhibit significant histological overlap. In our case, pseudo-glandular structures with PAS-positive mucous cells, focal epithelial thickenings, and glycogen-rich clear cells were observed, resembling features of GOC [17, 18]. However, the absence of ciliated epithelium and duct-like spaces with mucous cells differentiates this case, confirming an early CMEC diagnosis. This transformation is supported by histopathological, histochemical, and immunohistochemical findings, which provide valuable insights into its biological behavior and diagnostic approach. The lesion presents as a unilocular cyst containing epidermoid, mucous, and intermediate cells—a combination characteristic of MEC, commonly observed in transformations from dentigerous cystic lesions. [19] Thickening of the cystic lining, along with chronic inflammation in the stroma, is evident. Chronic inflammation is a well-known factor contributing to mucous metaplasia in the cystic wall, a recognized precursor for malignant changes in dentigerous cysts. [20,21] The presence of microcysts, mucous goblet cells, and mucin accumulation, confirmed by mucicarmine and PAS staining, is critical for identifying mucous metaplasia and diagnosing MEC. [22] The prolonged persistence of a dentigerous cyst, in combination with chronic inflammation, may predispose the lesion to oncogenic events. The transition from a benign cyst to MEC involves molecular alterations, including changes in p53, Ki-67, and Bcl-2 expression. [22,23] This underscores the importance of regular monitoring of cystic lesions, particularly those that persist over extended periods.

Immunohistochemical Markers and Their Diagnostic Significance

- **p53 positivity (2.1%)**: Low p53 positivity corresponds to minimal disruption of tumor suppressors, which is typical in low-grade MEC. Aberrant p53 expression is often

associated with early-stage malignancy. [24] Weber et al. concluded that p53-positive tumors have an increased potential for malignant transformation of benign tumors. [25]

- **Ki-67 index (10.6%)**: Ki-67 is the most frequently reported prognostic factor in mucoepidermoid carcinoma. [26] Our finding demonstrates mild proliferative activity consistent with a low-grade tumor. Ki-67 is a crucial marker for assessing cell proliferation and distinguishing malignancy grades. [27] Literature supports the idea that both p53 and Ki-67 have prognostic significance and correlate with survival rates in MEC and ACC. [28]
- **Bcl-2 positivity (15.8%)**: Bcl-2 expression indicates resistance to apoptosis and supports malignant transformation. Elevated Bcl-2 levels are commonly observed in MEC and are associated with malignant salivary gland tumors and aggressive behavior. [29] This finding is consistent with the study by Al-Rawi et al., who found that all cases of malignant salivary gland tumors were positive for Bcl-2. The highest recorded score was observed in ACC, while the lowest was noted in both low-grade PA carcinoma and low-grade MEC. [30]

These immunohistochemical findings align with those observed in our case. The biological behavior of the lesion suggests a low-grade tumor with minimal nuclear atypia and low mitotic activity, which is characteristic of the indolent behavior typical of low-grade MEC. [31] Low-grade MEC generally has a favorable prognosis with appropriate surgical management, as evidenced by the absence of recurrence or metastasis in our patient after four years. [32]

10. Conclusion

Dentigerous cysts, while often benign, carry the potential for malignant transformation, such as into mucoepidermoid carcinoma. This case emphasizes the importance of integrating histological and immunohistochemical evaluations, including markers like **p53**, **Ki-67**, and **Bcl-2**, to detect early dysplastic or malignant changes. These markers not only provide insights into the biological behavior of the cyst but also serve as prognostic indicators and guide surgical decision-making.

Early detection through clinical, radiographic, and pathological assessment, followed by appropriate surgical intervention, is crucial for reducing recurrence and achieving favorable outcomes. Regular postoperative monitoring and tailored management strategies informed by immunohistochemical findings further enhance the long-term prognosis, ensuring comprehensive care for patients with long-standing dentigerous cysts.

Recommendations:

1. Follow-Up: Regular radiographic monitoring (e.g., panoramic X-rays or CBCT) every 6–12 months after surgical removal of the cystic lesion is essential to check for recurrence. Clinical examinationis vital to detect signs of residual cyst or recurrence.

2. Pathological Review: Regular immunohistochemical analysis in recurrent or suspicious lesions monitor p53, Ki-67, MAML2, p63 and Bcl-2 expression, indicate cellular behavior and potential for transformation.
3. Adjunctive Therapy: Antibiotics and anti-inflammatory medications can manage secondary infection or inflammation if present. We consider using adjunctive treatments like corticosteroids if significant inflammation or postoperative swelling occurs.
4. Patient Education: We should advice the patient on symptoms of maintaining oral hygiene and regular dental visits. It is obligatory to educate the patient on symptoms of recurrence, such as swelling, pain, or changes in adjacent teeth.

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Declaration of Patient Consent

Informed consent was obtained from the patient included in this study. The patient was informed of the purpose, procedures, potential risks, and benefits of participating in the research. It was explained that participation is voluntary and that they could withdraw from the study at any time without any impact on their medical care. The patient was also assured that their personal data would remain confidential and that only anonymized information would be used for research publication. The authors confirm that all relevant ethical guidelines have been followed and that necessary approvals were obtained from the appropriate institutional review board.

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Nil.

Conflicts of interest

There is no conflict of interest.

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تقييم التغيرات الخبيثة المبكرة للكيس المتحدّر وتحوله إلى السرطان المخاطي البشروي المركزي باستخدام الكيمياء النسيجية المناعية للواسمات KI67 ، P53، BCL-2 وصبغة حمض الشيف الدوري للمخاط

الملخص

تُعتبر الأكياس السنية، مثل الكيس المتحدّر، من الآفات الشائعة في الفك، وترتبط غالبًا بالأسنان المنطمرة وتظهر كآفات شعاعية شفافة غير مصحوبة بأعراض. وعلى الرغم من كونها حميدة في العادة، إلا أنها قد تخضع لتحول ورمي إلى آفات عدوانية مثل الورم الميناني أو السرطان المخاطي البشروي المركزي.(MEC) . قامت هذه الدراسة بتحليل آفة نسيجية تم تشخيصها مبدئيًا ككيس متحدّر باستخدام تقنيات التلوين بـH&E ، والمناعية الكيميائية للبروتينات p53 ، Ki-67، Bcl-2، بالإضافة إلى تلوينات PAS و Mucicarmineلتقييم التركيب الخلوي وإمكانات التحول الخبيث. كشفت النتائج النسيجية عن بطانة حرشفية مطبقة مع تحولات مخاطية، والتهاب مزمن، وغشاء قاعدي سليم. أظهرت الفحوص المناعية الكيميائية تعبيرًا منخفضًا عنp53 ، وإيجابية Ki-67 في الطبقة القاعدية، وتلونًا موضعيًا لـ Bcl-2، مما يشير إلى نشاط تكاثري منخفض. أكّد تلوين PAS سلامة الغشاء القاعدي، في حين كشف تلوين Mucicarmine عن تحولات مخاطية بؤرية في الخلايا. أثار وجود الخلايا المخاطية والبشرانية والمتوسطة الشكوك حول مرحلة مبكرة منMEC ، إلا أن غياب اللانمطية الكبيرة أو العلامات التكاثرية المنتشرة دعم تشخيص الكيس المتحدّر مع تحولات مخاطية. تم التعرف على حالة نادرة من MEC داخل العظم منخفض الدرجة لدى امرأة تبلغ من العمر 47 عامًا، تطورت دون علاج لمدة عامين قبل ظهور الأعراض. أكد الاستئصال الجراحي والتحليل النسيجي وجود خلل تكاثري طلائي وتحولات مخاطية متوافقة مع .MEC أظهر المتابعة لمدة أربع سنوات تجددًا كاملًا للعظم دون نكس. تؤكد هذه الحالة أهمية التشخيص المبكر، والمراقبة الدورية، والتقييم النسيجي الشامل للأكياس السنية لمنع التحول الخبيث. يُعتبر التدبير المحافظ فعالًا للآفات الحميدة، فيما يُوصى بإجراء دراسات جزيئيةMAML2) ، P63، إعادة ترتيب جين (Bcl-2 للحالات ذات الإمكانية الورمية.

الكلمات المفتاحية:
الكيس المتحدّر, السرطان المخاطي البشروي, الكيمياء النسيجية المناعية, Ki67, P53, Bcl2, صبغة الحمض الدوري Pas

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