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Review Article

Benign epithelial odontogenic tumor- A review

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ABSTRACT

The jaws are complicated structures in anatomy that house many different tissues. Embedded within these tissues lie the remains of tooth development that sometimes results in pathological conditions. Odontogenic tumors with benign epithelial characterizations constitute a particular group developing from cells of epithelium involved in tooth formation. Although being usually slow growing and non-aggressive, these lesions could be associated with considerable problems due to their location or capacity for expansion. This article explores the clinical challenges associated with diagnosing benign epithelial odontogenic tumors. Also, this paper looks into the specificities of four most common types such as ameloblastoma, adenomatoid odontogenic tumor, squamous odontogenic tumour and calcifying epithelial odontogenic tumour. The characteristic signs, symptoms and radiographic presentations as well as histological profiles at each type may enable clinicians to improve their diagnostic accuracy leading to appropriate management directions. Therefore, it is important to intervene early before complications occur so as to ensure favorable patient outcomes from this ailment that can manifest either on soft or even hard tissue surfaces.

1. Introduction

The intricate architecture of the tissues of the head and neck area makes it home to a wide spectrum of pathological conditions. Both benign and malignant tumours can arise from the tissues that make teeth. The ensuing displacement of the surrounding normal anatomic structures results in cortical bone perforation, which can range in size from microscopic swellings to massive varieties. While most odontogenic tumours develop slowly and are not aggressive, some of them

Exhibit aggressive behaviour. To effectively manage these disorders, the physician must possess a comprehensive understanding of the pathophysiology, clinical presentation, and radiological findings.[1]

According to WHO 'Odontogenic tumours and tumour-like lesions constitute a group of heterogeneous diseases that range from hamartomatous or non-neoplastic tissue proliferations to benign neoplasms and finally malignant tumours with metastatic potential. They originate from the tooth-forming apparatus's epithelial, ectomesenchymal and or mesenchymal components. Odontogenic tumours are uncommon some are quite uncommon but they can be very difficult to diagnose and treat.[2]

Benign odontogenic tumours were initially categorised by the WHO in 1971. Subsequent classifications came in 1992, 2005, and most recently in 2017. Tumours are divided into three categories depending on their origin:

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mesenchymal, mixed, and epithelial. This origin-based subclassification system was initially established in 1992 and is now in use. The 2017 WHO classification of epithelial origin tumours includes metastasising (malignant) ameloblastoma, but the benign odontogenic tumour classification does not contain desmoplastic ameloblastoma. Adenomatoid odontogenic tumours (AOTs) were classified as mixed origin tumours in 1992, however they were moved to the epithelial origin tumour category in 2005 and 2017.[3]

Within the range of ghost cell lesions is the dentinogenic ghost cell tumour (DGCT). The earliest known ghost cell lesions were calcifying odontogenic cysts (COCs), which were initially reported by Gorlin et al. in 1962. The WHO reclassified COC as calcifying cystic odontogenic tumour (CCOT) in 2005. For DGCT, the term "calcifying ghost cell odontogenic tumour" was first coined by Fejerskov and Krogh in 1972. The term "dentigerous ghost cell tumour" was first used by Praetorious et al. in 1981 and is currently used in the WHO classifications from 2005 and 2017. Ellis and Shmookler introduced the name epithelial odontogenic ghost cell tumour in between, while Shear adopted the word dentinoameloblastoma in 1983. In 1991, Hong and colleagues proposed the name "epithelial odontogenic ghost cell tumour."[4]

2. Classification of Odontogenic Tumors

2.1. WHO (2017) classification of odontogenic tumors[5]

2.1.1. Benign odontogenic tumours

1. Epithelial Origin
   (a) Ameloblastoma, conventional
   (b) Ameloblastoma, unicystic type
   (c) Ameloblastoma, extraosseous/peripheral type
   (d) Metastasising (Malignant) ameloblastoma
   (e) Squamous odontogenic tumour
   (f) Calcifying epithelial odontogenic tumour
   (g) Adenomatoid odontogenic tumour
2. Mixed (Epithelial-Mesenchymal) Origin
   (a) Ameloblastic Fibroma
   (b) Primordial odontogenic tumour
   (c) Odontoma, complex type
   (d) Odontoma compound type
   (e) Dentinogenic ghost cell tumour
3. Mesenchymal Origin
   (a) Odontogenic Fibroma
   (b) Odontogenic myxoma/myxofibroma
   (c) Cementoblastoma
4. Malignant Odontogenic Tumours
   (a) Ameloblastic carcinoma
   (b) Primary intra-osseous carcinoma, NOS
   (c) Sclerosing odontogenic carcinoma
   (d) Clear cell odontogenic carcinoma
   (e) Ghost cell odontogenic carcinoma
   (f) Odontogenic carcinosarcoma
   (g) Odontogenic sarcomas

2.2. WHO classification of odontogenic tumors of the jaws[6]

1. Benign epithelial odontogenic tumours
   (a) Ameloblastoma, unicystic
   (b) Ameloblastoma, extraosseous/peripheral
   (c) Ameloblastoma, conventional
   (d) Adenoid ameloblastoma
   (e) Metastasizing ameloblastoma
   (f) Adenomatoid odontogenic tumour
   (g) Squamous odontogenic tumour
   (h) Calcifying epithelial odontogenic tumour
2. Benign mixed epithelial & mesenchymal odontogenic tumours
   (a) Odontoma
   (b) Primordial odontogenic tumour
   (c) Ameloblastic fibroma
   (d) Dentinogenic ghost cell tumour
3. Benign mesenchymal odontogenic tumours
   (a) Odontogenic fibroma
   (b) Cementoblastoma
   (c) Cemento-ossifying fibroma
   (d) Odontogenic myxoma
4. Malignant odontogenic tumours
   (a) Sclerosing odontogenic carcinoma
   (b) Ameloblastic carcinoma
   (c) Clear cell odontogenic carcinoma
   (d) Ghost cell odontogenic carcinoma
   (e) Primary intraosseous carcinoma, NOS
   (f) Odontogenic carcinosarcoma
   (g) Odontogenic sarcomas

The aim of this review is to discuss about the benign epithelial odontogenic tumors.

2.3. Ameloblastoma

The most frequent odontogenic tumour that develops from an odontogenic epithelium is this one.

Ameloblastoma arises from the following sources:

1. Malessez epithelial cells are the remaining epithelium of the tooth-forming apparatus.
2. Epithelium of odontogenic cysts and the genesis of enamel.
3. The surface layer of basal cells.
4. Heterotopic epithelial cells from extraoral locations, including the pituitary gland.[7]

Edited 2017 WHO classification of odontogenic tumours has simplified the classification of Ameloblastomas. The many pathologically descriptive categorization terminologies, which have no bearing on clinical behaviour, have been eliminated, including follicular, plexiform, basaloid, granular, and desmoplastic. Multilocular, expanding behaviour is a characteristic of Ameloblastomas of the conventional form.

The infiltrating margin, which defines the histological boundary, necessitates a broad excision with a margin of 0.5–1 cm of normal bone or one anatomical layer if the growth extends beyond the boundaries of the bone. In other words, to preserve the periosteum as an anatomical barrier, use muscle or subcutaneous tissue if the bony cortex invades.

2.4. Definition

"It is a true neoplasm of enamel organ-type tissue, which does not undergo differentiation to the point of enamel formation" proposed by WHO.

Robinson defined it as a "Non-functional, unicentric, intermittent in growth, anatomically benign and clinically persistent type of tumour". [8]

2.5. Incidence

19.3–41.5% of all odontogenic tumours are represented by it. [9] About 80% of ameloblastomas [10] develop more frequently in the posterior mandible. Cases span a wide age range, from toddlers under 10 years old to elderly over 90 years old. They often manifest in the second and fourth decades of life. While most research revealed female inclination during the first and third decade of life, some writers reported no gender predilection. [11–13]

2.6. Clinical features

1. Swelling of maxilla or mandible.
2. Facial disfigurement.
3. Tooth displacement and mobility.
4. Paraesthesia.
5. Ulceration or Nasal obstruction.

2.7. Radiographic features

OPGs or normal dental x-rays are frequently used to detect ameloblastomas since they typically begin in the bone, The affected teeth's root resorption. Classically, "soap bubble appearance" is seen in solid/multilocular-type ameloblastomas, which are the most prevalent. [14] As a useful tool for surgical planning, computed tomography (CT) can provide precise information on cortical damage and soft tissue extension. It also frequently shows well-defined, radiolucent uni/multilocular lesions. [15] When determining the extent of ameloblastomas into the orbit, base of the skull, and paranasal sinuses, magnetic resonance imaging (MRI) is highly useful in cases with maxillary lesions. [16] When ameloblastomas spread, PET/CT is typically recommended for the patient.

2.8. Histopathology

Ameloblastomas often have a follicular or plexiform pattern on histology, while variants can also be observed in basaloid, granular cell, or desmoplastic patterns. It is well acknowledged that there is no connection between the unique patterns and the tumor's behaviour or prognosis. Pathologists may decide not to report the histologic pattern because of this. (The diagnosis of conventional, unicystic, or peripheral kinds should not be confounded with histologic pattern; these descriptions have a significant bearing on the course of treatment and prognosis for the patient).

Histopathologically, the solid/multicystic ameloblastoma can be classified as either a plexiform or follicular type, the follicular type can be further differentiated into granular, spindle cell, acanthomatous, and basal cell types. The plexiform type has basal cells with a subtle stellate reticulum organised in anastomosing threads. Typically fragile, the stroma frequently exhibits cyst like degeneration. The unicystic ameloblastoma is a form of ameloblastoma that appears as a cyst during gross inspection rather than based on radiological appearance. There are two recognised histological variants: the mural variant and the luminal variant. The solid/multicystic type's histopathogical cell types and patterns are also present in the extraosseous form. The stromal component predominates in the desmoplastic type, squeezing the odontogenic epithelial components. [10,17]

2.9. Immunohistochemistry

Fukumashi et al. state that CK8 and 19 are often present in the odontogenic epithelium and that they have a positive response to the cells that make up all ameloblastoma types. The review by Siar et al. is consistent with cytokeratin positivity in desmoplastic ameloblastomas. An uneven, faint staining was present in ameloblastomatous islands. Regarding the staining pattern of ameloblastoma lining cells. One of the key characteristics of tumour formation is angiogenesis, which is the production of new blood vessels from pre-existing ones. Using tissue samples to characterise the tumour microvasculature offers crucial prognostic information in cases of malignancy. The type I transmembrane protein known as CD34 antigen is highly glycosylated and has a molecular weight that falls between 110 and 120 Kda. Hiroyuki, et al. have found increased microvessel density in ameloblastomas when compared to normal tooth germs, CD34 microvessel density can be a marker of aggressive behaviour almost equal to malignancy. Myofibroblasts are modulated fibroblast

that expresses alpha-smooth muscle actin, which is key contractile protein, found in smooth muscle cells. There hasn't been much research done on the subject of smooth muscle actin positive myofibroblasts in odontogenic lesions. The histogenic theory that ameloblastomas originate from the oral epithelium is supported by the expression of immunohistochemistry (IHC) markers such as S100, which are expressed in the epithelium.[10]

2.10. Treatment

Wide surgical removal is the recommended course of therapy for ameloblastomas, with the probable exception of the luminal variety of unicystic ameloblastomas, which enucleation may be justified.[18]

3. Adenomatoid Odontogenic Tumor

It has been known for more than a decade that the tumour that satisfies the current diagnostic criteria for an adenomatoid odontogenic tumour (AOT) Ninety years. The current authors concur with Unal and colleagues that the earliest identification of an AOT for which enough documentation is available is Steensland's report from 1905 of a "epithelioma adaman titanium." This lesion has been referred to by several names; but, for many years, the term "adenomeloblastoma" was widely used since the tumour was thought to be a histologic variation of the solid/multicystic ameloblastoma. From 1905 to 1969, a list of related phrases was generated by Unal et al. A study consisting of 76 AOT cases was given by Philipsen and Birn in 1969, which demonstrated that the tumour was a distinct entity from the solid or multicystic ameloblastoma. The World Health Organisation (WHO) approved the term "adenomatoid odontogenic tumour," which they coined in 1971, the currently widely used terminology.[10]

3.1. Incidence

AOTs rank fourth or fifth among odontogenic tumours, according to the relative frequency of the AOT retrieved from 12 oral biopsy studies, which ranges from 2.2% to 7.1% of all odontogenic tumours. Only odontomas, myxomas, and solid/multicellular ameloblastomas may pass through it. These numbers make it difficult to maintain that the AOT is an exceptionally uncommon odontogenic tumour.[19]

3.2. Clinical features

The AOT isa benign, non-neoplastic (hamartomatous)lesion with a slow but progressive growth. AOT is thought to have originated from the dental lamina or its remnants, according to Philipsen et al.'s persuasive argument based on current understanding of the biology of the AOT. It occurs in both intraosseous and peripheral forms. At the time of diagnosis, individuals with AOTs range in age from 3 to 82 years." over half of cases (53.1%) are diagnosed in adolescents (13 to 19 years old), and at least 68.6% of tumours are discovered in the second decade of life. The age distribution of AOT is distinct from other odontogenic tumours due to its tall peak in the second decade. The female:male ratio for all age groups and AOT variants together is 1.9:1. The AOT appears, as mentioned earlier, in three clinlcotopographic variants: follicular, extrafollicular, and peripheral.[20]

3.3. Radiographic features

The intrabony variations are divided into two types radiographically: follicular and extrafollicular.The follicular form has a well-defined, unilocular (round or oval) radiolucency that is associated with the crown and frequently partially with an unerupted tooth. It can also be found between, above, or superimposed on the roots of permanent teeth that have erupted. These sites frequently result in the tenta root of an unerupted tooth, simulating a preoperative diagnosis of a follicular cyst or residual dentigerous cyst. As a matter of fact, dentigerous cysts represent the original diagnosis for 77% of follicular type AOTs."

Depending on the precise intraosseous location of the lesion, radicular, "globulomaxillary," or lateral periodontital cysts are not linked to the extrafollicular variety.

3.4. Histopathology

AOT is an encapsulated lesion that can include calcifications or not. It can take the appearance of solid nodules, ductlike spaces, nests, or rosette-like formations made of polygonal, cuboidal, or spindle-shaped epithelial cells. Tumour droplets are spherical, homogeneous eosinophilic material that may be present in it.[10,19]

3.5. Immunohistochemistry

Numerous studies conducted recently have expanded our understanding of the immunohistochemistry of AOTs. Tatemoto et al. showed that the tumour cells along the edge of the whorled, tubular, or ductal formations coexpressed vitreomendin and keratin. Mineralized and hyaline material showed negative results for keratin stains, but tumour cells showed positive results. Both Saku et al. and Mori et al. investigated enamel proteins in AOTs and discovered enamelin and amelogenin in tiny mineralized foci in hyaline droplets and tumour cells.[21,22]

3.6. Treatment

For this tumour, conservative surgical excision combined with curettage or enucleation is the recommended therapy. Recurrence is conceivable but quite improbable.[20]

4. Squamous Odontogenic Tumor

Squamous odontogenic tumour (SOT), a specific kind of odontogenic tumour situated in the periodontium, was initially reported by Pullon et al. in 1975. The six tumours that "caused radiolucent areas of bone destruction adjacent to the roots of teeth" were described by the authors. As to the 1992 World Health Organisation (WHO) classification, the SOT is classified within the epithelial-derived ontopogenic tumour family. Although alternative terms, including squamous odontogenic hamartoid lesion, have been proposed, the name SOT is widely used. Based on a recent analysis of 36 instances published in 1996, the SOT is described below. Favia et al. published two further instances in 1997, and Ide et al. published two more cases in 1999.[23,24]

4.1. Definition

Well-differentiated squamous epithelium islands within fibrous stroma constitute a benign but locally infiltrative tumour. On occasion, focal areas of central cystic degeneration are seen on the epithelial islands. Supposedly arising from the remnants of dental lamina or the cell rests of Malassez (ROM), or gingival epithelium, SOT is an uncommon, benign, but locally infiltrative epithelial tumour.[10]

4.2. Incidence

To date, fewer than 50 cases have been reported in the literature.[10]

4.3. Clinical features

A benign but locally infiltrative odontogenic tumour is the squamous odontogenic tumour. With minimal clinical indications and symptoms, it is expanding slowly. Possible symptoms of the underlying tumour include moderate discomfort, enlargement of the alveolar process, and tooth mobility. The majority of instances arise at the permanent teeth's periphery. Consequently, the most prevalent variety of SOT is the intra-oral or central form. Additionally, a rare peripheral form has been described. Multicentric incidence of some SOTs has been documented, while some are localised in edentulous areas.

This finding emphasises the need of a patient with a tumour diagnosed as SOT undergoing a comprehensive clinical and radiologic assessment. The finding that SOTs in the maxilla appear to be more aggressive than those in the mandible, a characteristic shared by a number of odontogenic tumors is especially noteworthy. Leider et al. found a family pattern of SOT, reporting numerous lesions in three siblings. SOT appears to be less frequently linked to impacted teeth than many other odontogenic tumours. It is unclear if it is related to primary intraosseous squamous cell cancer.[25,26]

4.4. Histopathology

According to histology, the SOT is made up of regions of squamous epithelium that have undergone differentiation and vary in size and form. The islands are often round or spherical, but they can sometimes show irregular, cord-like features, which is a hallmark of torthe desmoplastic ameloblastoma. A peripheral layer of low cuboidal or even flat epithelial cells can be seen on individual tumour islands. After single-cell keratinization, individual epithelial islands may experience core microcystic degeneration of the spinous cells. Some islands may enlarge and include laminar calcium carbonate material. The epithelial tumour cells' mitotic activity is not heightened. Mature connective tissue encircles tumour sites with minimal to no inflammatory response.[27]

4.5. Immunohistochemistry

Yamada et al.[28] and Tatemoto et al.[29] reported on immunohistochemistry results in SOT patients. Polyclonal antikeratin antiserum (TK, detecting 41- to 65-kd keratins) and monoclonal antibodies (KL1, 55 to 57 kd;PKK1, 40, 45, and 52.5 kd) were used for immunohistochemical labelling of keratin proteins. Tumour islands did not exhibit PKK1-detectable keratin staining; instead, squamous epithelial cells were the only cells stained with KL1 and TK immunoreagents. By heavily staining for keratin 13 and 16, the proliferative activity of the odontogenic epithelium of SOTs was further validated. Strong positive responses for volcanism were seen in the centres of the squamous differentiating cells' epithelial islands." However, involucrin was also detected in acanthomatous and follicular SMAs with foci of squamous development in the later investigation.

4.6. Treatment

The usual treatment has been simple enucleation and recurrence has been rare.[30]

5. Calcifying Epithelial Odontogenic Tumour

One percent of all odontogenic tumours are calcifying epithelial odontogenic tumours (CEOT), commonly referred to as Pindborg tumours. CEOT is a rare and peculiar benign epithelial odontogenic neoplasm. The calcifying epithelial odontogenic tumour (CEOT) was initially reported in 1955 by Dr. Jens Jorgen Pindborg, a pathologist from Denmark. Shafer published the first written description of the now-famous Pindborg tumour in 1963, and the name Pindborg tumour was originally applied to it in 1967. Before 1955, the cancer was known by a number of names, including ameloblastoma of odd kind with calcification,

calcifying ameloblastoma, malignant odontoma, adenoid adamantoblastoma, cystic complex odontoma, and a form of solid or multicystic ameloblastoma (SMA).

Since the World Health Organisation (WHO) published Histological Typing of Odontogenic Tumours, Jaw Cysts, and Associated Lesions in 1971, the term "calcifying epithelial odontogenic tumour" has become widely used and acknowledged. The World Health Organisation classified it as a benign odontogenic tumour in 1992 due to its epithelial genesis and absence of an ectomesenchymal component.[31,32]

5.1. Definition

CEOT is defined as a benign epithelial odontogenic tumour that secretes an amyloid protein tending towards calcification (Franklin & Pindborg, 1976; Azevedo et al, 2013; El - Naggar et al, 2017).[33]

5.2. Incidence

Epidemiological research that included all odontogenic tumours from the archives of the Department of Oral Pathology at the Dental Teaching and Research Institution in the southern region of India between 2002 and 2014 was carried out in the state of Andhra Pradesh in southern India. The prevalence of odontogenic tumours was determined to be 2.17%, of which 1.8% is accounted for by calcifying epithelial odontogenic tumours.

5.3. Clinical features

Clinically, it appears as a slow-growing, expansile, aggressive, locally invasive tumour. Regardless of gender, it usually affects people in their third and fourth decades of life. The most commonly afflicted area is the posterior mandible, where there are two types: intraosseous (central) and extraosseous (peripheral). When it grows intraosseously, it usually appears as a slow-growing, painless lump and occasionally shows signs of local invasiveness. Sometimes patients will complain of headaches, epistaxis, and congestion in the nose. The most common presentation of extraosseous CEOT, also known as peripheral softtissue, is a hard, painless gingival mass. Preoperative clinical diagnosis includes epulis, fibrous hyperplasia, and peripheral giant cell granuloma.

Although the source of the epithelial cells found in the Pindborg tumour is still unknown, information from the literature suggests that these material remains are located in the stratum intermedium layer of the enamel organ. The hypothesis that cancer cells have increased adenosine triphosphate and alkaline phosphatase activity and physical similarities with stratum intermedium cells supports this. The literature suggests that the amyloid deposits seen in the Pindborg tumour are the result of the immune system's response to these stratum intermedium cells. Some authors claim that it arises from remains of the dental lamina, which are more likely to be the actual progenitor cell. In 48% of cases, a problem in the maxilla is associated with an erupted or unerupted tooth.[34]

5.4. Radiographic features

The characteristic radiographic appearance is an irregular unilocular or multilocular radiolucent zone with radiopaque masses of varying sizes and opacities; this image is referred to as "driven snow." The microscopic calcium concrements might be missed on radiographs, particularly in tumours that have been there for a short while. When a tumour is linked to an unerupted tooth, the radiopacities usually happen around the dental crown. At the perimeter, it may or may not be possible to identify the radiolucent border from normal bone.

5.5. Histopathology

The predominant histologic pattern of CEOTs is a rare and variable mix of calcified structures and odontogenic epithelium. The extraosseous kind of CEOT has little or no calcified material, but other than that, there are no significant histomorphological variations between the two. The most common histologic appearance consists of heterogeneous sheets of polyhedral cells with noticeable intercellular bridges, amyloid-like debris, and calcifications. The cells may exhibit pleomorphism, multinucleation, prominent nucleoli, and sporadically hyperchromatism.

Apart from the Common features, several CEOT variants have been reported, such as microcystic or cystic variants, hybrid tumours associated with adenomatoid odontogenic tumour or ameloblastoma, and tumours with different percentages of clear cells, Langerhans cells, and non-calcifying tumours.

Due to an excess of lipid or glycogen droplets, the cancer cells in the CEOT clear cell variety have transparent cytoplasm, giving them a distinctly vacuolated appearance. Given that it has several characteristics with other clear cell tumours.

But apart from the conventional features of CEOT, including a sheet of polyhedral epithelial cells and different amounts of calcified material (Liesegang rings), its unique feature is the presence of transparent cells in the tumour tissue. Rare but persistent, the Langerhans cell variant of CEOT has microscopic islands and cords of malignant cells with high levels of amyloid material but no calcification. Damm et al. (1983) classified the CEOT-like areas discovered in two cases of adenomatoid odontogenic tumours as combination epithelial odontogenic tumours. Microcystic variations in which the neoplastic cells have a microcystic pattern are rare in calcifying epithelial odontogenic tumours.[35]

5.6. Immunohistochemistry

The polyhedral cells of CEOT exhibit vimentin, fibronectin, cytokeratins, laminins 1 and 5, and fibronectin immunohistochemically. In cancer epithelial cells, detectable keratins specific to keratins 44, 46, 52, and 53 kD are either marginally positive or negative, whereas TK (keratins 41–65 kD) and KL1 (specific to keratins 55–57 kD) are mildly to moderately positive. In the tumour epithelium, desmin is negative and vitenin is very weakly positive. Prominent discoveries encompass elevated concentrations of ATPase and alkaline phosphatase confined to the cellular membrane. It has been demonstrated that the amyloid material contains a variety of ameloblast-associated proteins, the most commonly seen of which is Odontogenic Ameloblast-Associated Protein (ODAM). Dendritic cells are typically seen in CEOT epithelial sheets and have a high positive signal for the S-100 and CD-1a antibodies.[35]

5.7. Treatment

The majority of CEOTs are benign, but 10% to 15% of them recur. They can also be locally aggressive. It is recommended to enucleate within macroscopically normal tissue while undergoing CEOT on the jaw.[35]

6. Source of Funding

None.

7. Conflict of Interest

None.

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