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Review Article

# Collision Lesions in Head and Neck Area: A Retrospective Study of 97 Reported Cases

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**Abstract**

**Background:** Collision (hybrid) lesions consist of two or more distinct histologic types occurring separately but within the same area. This review focuses on discussing all related and available case reports of hybrid lesions in the head and neck area.

**Methods:** The literature was searched using several databases, including Scopus, Web of Science, PubMed, and Google Scholar. Papers published from 1990 to 2022 with full text available were considered for inclusion in the study. For a more comprehensive understanding, all lesions were categorized into cutaneous, jaw, and salivary gland groups and lesions in miscellaneous tissues.

**Results:** In general, 97 reported collision lesions developed in the head and neck region. Among them, 31 (32%) and 34 (35%) cases were cutaneous hybrid lesions and in the jaws, respectively. In addition, 27 (27.8%) and 5 (5.1%) cases were in salivary glands and miscellaneous tissues, respectively.

**Conclusion:** While the presence of true hybrid tumors is well-established, some reported cases have prompted researchers to categorize them as biphasic differentiations. The rarity of these cases contributes to limited knowledge regarding their clinical behavior, prognosis, and optimal treatment. It is crucial to emphasize the characteristic and potentially perplexing histological appearances.

**Keywords:** Hybrid, Jaw, Odontogenic, Salivary glands, Skin

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## Background

Collision (hybrid) lesions comprise two or more distinct histologic types separately occurring but within a similar area. While these lesions occur only occasionally, their occurrences have been well recognized. They may manifest as either benign conditions or malignant tumors (1). Biphasic differentiated lesions present as mixtures of two or more cellular patterns corresponding to lesion classification (2). There is a limited body of research about the epidemiology of collision lesions. However, a retrospective observational study of 77 proven hybrid skin lesions, collected from nine countries, revealed collision lesions on the head and neck area (24.7%), the trunk (48.1%), the lower extremities (11.7%), and the upper extremities (5.2%) (3). A previous systematic review reported only 203 cases of reported hybrid odontogenic lesions in the literature (4). In addition, it has been shown that hybrid salivary gland neoplasms comprise 0.1% of all salivary gland tumors (5).

The pathogenesis of collision lesions remains a controversial issue, and three theories may explain the co-existence of two or more pathological entities (tumors/lesions) simultaneously. Firstly, they may result from the proliferation of two different cell lines. Secondly, they might share a common origin in a totipotent cell, which differentiates into two different cell lines. The third theory involves the sarcomatous conversion of an epithelial tumor (6). Regarding collision tumors, it has been suggested that pathogenic mechanisms such as the "random collision effect", "field cancerization", and "tumor-to-tumor carcinogenesis" are involved in this phenomenon (7). Additionally, composite tumors have been explained in the literature. These tumors develop due to a common driver mutation with different histological features from a common tumor source (8). Both composite and collision tumors have distinct morphological and histological features coexisting within the same organ. However, the intermingling of cells detected in composite lesions

cannot be observed in collision lesions (8).

The simultaneous occurrence of lesions in different tissues has been documented, emphasizing the importance of reviewing reported cases of collision lesions, especially since the treatment protocol for each entity varies (9). This review discusses all related and available case reports of hybrid lesions in the head and neck area.

## Materials and Methods

The literature was searched using the Scopus database, Web of Science, PubMed, and Google Scholar. Papers published from 1990 to 2022 with full texts were considered for inclusion in the study. For a more comprehensive understanding, all lesions were classified into salivary gland lesions, jaw lesions, cutaneous lesions, and lesions in miscellaneous tissues.

## Results

In general, 97 reported collision lesions developed in the head and neck regions. Among the cases, 31 (32%) were cutaneous hybrid lesions. The most common cutaneous lesion combined with other lesions was basal cell carcinoma (BCC), with 17 cases (17.5%), followed by malignant melanoma (n = 13, 13.4%). Table 1 summarizes all documented cutaneous collision lesions.

There were 34 cases (35%) of hybrid lesions in the jaws. The most common lesions in the jaws were odontogenic lesions (n = 33, 34%). An adenomatoid odontogenic tumor (AOT) was the most common odontogenic lesion combined with other lesions (n = 13, 13.4%), followed by central odontogenic fibroma (COF; n = 10, 10.3%). Considering non-odontogenic lesions, central giant cell granuloma (CGCG) was the most common hybrid lesion in the jaws (n = 10, 10.3%). CGCG was combined with COF in 8 cases and odontogenic keratocyst (OKC) in 2 cases. The mandible was the most frequently affected site, with 24 cases (24.7%). Table 2 provides information about hybrid lesions in the jaws.

There were 27 documented cases (27.8%) of salivary gland collision lesions. The most common combined salivary gland lesion was adenoid cystic carcinoma (AdCC) with 13 cases (13.4%), followed by salivary duct carcinoma (SDC) with 11 cases (11.3%). The parotid gland was the most affected salivary gland, with 19 cases (19.5%). Table 3 lists all reported hybrid salivary gland lesions. In addition, there were 5 cases (5.1%) of collision lesions in miscellaneous tissues (Table 4).

## Discussion

A collision lesion refers to the simultaneous presence of two distinct primary lesions in a single anatomical site without significant histological admixture at the interface (59). This occurrence may be coincidental, or there could be a related pathogenesis (59). It is believed that two independently arising primary lesions converge in the same location, or that the development of one lesion modifies the surrounding microenvironment, allowing a second lesion to emerge in the same area (73).

The coexistence of BCC, the most common skin cancer, with other skin lesions such as melanocytic nevus, seborrheic keratosis, and malignant melanoma has been previously reported (1,74). In the current review, 54.8% of cutaneous hybrid lesions were BCCs. However, a prior paper detected BCCs in 34.3% of all skin hybrid cases in head and neck areas (3). The hypothesis suggests that the presence of one tumor may induce epithelial or stromal changes, facilitating the development of a second lesion. Skin damage due to chronic exposure to sunlight is considered the primary etiologic factor for BCC, malignant melanoma, and some other cutaneous lesions (20). Some studies have demonstrated that BCCs originate from the basal layer of the epidermis and the hair follicle outer root sheath. Consequently, normal melanocytes are probably entrapped as the tumor grows (12,75). Another study has revealed that BCC contains HMB45-positive melanocytes. As a result, the abnormal stimulation of these melanocytes may lead to the development of malignant melanoma within BCC (20).

In total, 25 cases (25.7%) of odontogenic lesions combined with other lesions were noted in the current review. The mechanism of the association of two different odontogenic lesions is unclear, but it is believed that the second lesion is a secondary phenomenon of a pre-existing tumor. A growing body of research shows that the hybrid odontogenic lesion is the result of the pluripotent characteristics of the odontogenic epithelium. It means that both lesions develop from a common source (46,76,77). Different sources for the odontogenic epithelium have been demonstrated. The rests of dental lamina (in the alveolar bone), the epithelial cell rests of Malassez (Hertwig's root sheath remnants) in the periodontal ligament, the rests of the enamel organ and reduced enamel epithelium (deep in the body of jaw bones), the rests of Serres (in the gingiva), and the pericoronal follicular tissue are considered the main sources of odontogenic epithelial stem cells (78-81). A previously published study revealed that the embryonic residues of the odontogenic epithelium within the jaws are capable of proliferating and developing different odontogenic lesions (79). Notably, the majority of odontogenic lesions are associated with an impacted tooth (36). In a recent systematic review, 24.1% of cases were AOT combined with a calcifying epithelial odontogenic tumor (CEOT) or dentigerous cysts (12.8% vs. 11.3%) (4). Our findings demonstrated AOT in 39.3% of cases combined with different odontogenic and non-odontogenic lesions (Table 2). The pathogenic mechanism is unclear; however, it has been suggested that it is a collision of two separate lesions or a transformation of one lesion into another. For example, the solid portion of CCOT and COC may have some characteristics of odontogenic tumors, such as ameloblastoma, AF, and AFO. In addition, it is believed that the proliferation of odontogenic epithelium in COC might induce the adjacent mesenchymal tissue to develop

**Table 1.** Collision Cutaneous Lesions in the Head and Neck Area

| First Lesion | Second Lesion | Site | Gender | Age Range (y) | Duration | *Survival Status After Diagnosis Until the Report of Case* | Reference |
|---|---|---|---|---|---|---|---|
| BCC | Atypical fibroxanthoma | Nose | F | 85 | 3 years | Alive | (10) |
| BCC | Lentigo maligna | Nose | M | 78 | 2 months | Alive | (11) |
| BCC | Lentigo maligna | Nose | F | 82 | No data | Alive | (12) |
| BCC | Seborrheic keratosis | Nose | F | 62 | No data | Alive | (13) |
| BCC | Seborrheic keratosis | Occipital | F | 78 | A few months | Alive | (14) |
| BCC | Seborrheic keratosis | Auricle | F | 89 | No data | Alive | (15) |
| BCC | Seborrheic keratosis | Cheek | M | 82 | Several years | Alive | (16) |
| BCC | Seborrheic keratosis | Temple | M | 68 | 12 years | Alive | (17) |
| BCC | Melanocytic nevus | Neck | M | 63 | A few months | Alive | (17) |
| BCC | Superficial spreading Melanoma | Nose | M | 76 | No data | Alive | (18) |
| BCC | Keratoacanthoma | Cheek | M | 46 | 6 weeks | Alive | (19) |
| BCC | Melanoma | Neck | F | 68 | Several years | Alive | (20) |
| BCC | Melanoma | Temple | M | 79 | Several months | Died 3 months later | (21) |
| BCC | Melanoma | Scalp | M | 78 | No data | Alive | (22) |
| BCC | Melanoma | Canthus | M | 91 | No data | Alive | (23) |
| BCC | Sclerosing blue nevus | Scalp | F | 67 | A few months | Alive | (24) |
| BCC | Signet-ring SCC | Eyelid | F | 93 | 6 months | Alive | (25) |
| Atypical fibroxanthoma | Melanoma | Scalp | M | 76 | 3 months | Alive | (26) |
| Chondroid syringomas | Intradermal melanocytic nevus | Nose | F | 70 | 6 months | Alive | (27) |
| desmoplastic trichoepithelioma | Melanocytic nevus | Cheek | F | 27 | 2 years | Alive | (28) |
| Invasive SCC | Invasive melanoma | Scalp | M | 98 | 1 year | Alive | (1) |
| Acantholytic SCC | Atypical fibroxanthoma | Cheek | M | 60 | Several years | Alive | (29) |
| SCC | Melanoma | Cheek | F | 84 | No data | Alive | (30) |
| SCC | Melanoma | Lower lip | M | 46 | No data | Alive | (31) |
| Trichoblastoma | BE/F neoplasm | Cheek | M | 69 | No data | Alive | (32) |
| Trichoblastoma | Inverted follicular keratosis | Chin | M | 40 | No data | Alive | (32) |
| Trichoblastoma | BE/F neoplasm | Forehead | M | 49 | No data | Alive | (32) |
| Trichoblastoma | BE/F neoplasm | Occipital | M | 27 | No data | Alive | (32) |
| Trichoblastoma | Seborrheic keratosis | Scalp | F | 28 | 2 months | Alive | (33) |
| BSCC | Melanoma | Frontal | M | 60 | 25 years | Alive | (34) |
| Merkel cell carcinoma | Lentigo maligna Melanoma | Cheek | M | 79 | No data | Alive | (35) |

*Note*. BCC: Basal cell carcinoma; SCC: Squamous cell carcinoma; BE/F: Benign epidermal/follicular; BSCC: Basosquamous cell carcinoma.

the characteristics of other odontogenic tumors (47,80). It has been shown that AOT arises from epithelial remnants such as dental lamina, Serres pearls, or Hertwig's epithelial root sheet and the enamel organ (52). It has been demonstrated that some odontogenic cysts occur in association with odontogenic lesions, including AOT. For instance, an association of AOT with dentigerous cysts has been reported (82). Apparently, both AOT and the dentigerous cyst are associated with an impacted tooth and are both common in younger patients and in the anterior maxilla (82). Histologically, AOT is partly cystic in some cases; therefore, it is unclear whether the lining of the associated cyst is a true dentigerous cyst or is only a secondary cystic change within an AOT (83). Even the radiographic findings in AOT are frequently similar to those of other odontogenic lesions, such as the dentigerous cyst and COC (42). A combination of CCOT or COC with other odontogenic lesions such as ameloblastoma, AF, and AFO had been reported as well (9). CEOT is also associated with AOT (84). The mechanism is unclear, but some authors suggest that CCOT may contribute to the development of other types of odontogenic tumors (36). Nevertheless, some other authors believe that the second lesion is a secondary phenomenon of a pre-existing

**Table 2.** Collision Lesions in Jaws

| First Lesion | Second Lesion | Site | Gender | Age Range (y) | Duration | Survival Status After Diagnosis Until the Report of Case | Reference |
|---|---|---|---|---|---|---|---|
| CCOT | Ameloblastoma | Mandible | M | 17 | 1 month | Alive | (36) |
| CCOT | AFO | Mandible | F | 4 | No data | Alive | (37) |
| CCOT | AOT and ameloblastoma | Mandible | F | 64 | 16 months | Alive | (38) |
| CCOT | AOT | Maxilla | F | 2 | No data | Alive | (39) |
| CEOT | AOT | Mandible | F | 17 | No data | Alive | (40) |
| CEOT | AOT | Maxilla | F | 16 | No data | Alive | (41) |
| CEOT | AOT | Maxilla | F | 23 | No data | Alive | (42) |
| COC | AFO and AOT | Mandible | M | 7 | No data | Alive | (43) |
| COC | AF | Maxilla | M | 13 | No data | Alive | (44) |
| COC | AF | Mandible | M | 22 | 3 weeks | Alive | (44) |
| COC | AF | Mandible | F | 6 | No data | Alive | (44) |
| COC | AF | Mandible | F | 14 | 10 days | Alive | (45) |
| COC | AF | Mandible | M | 10 | No data | Alive | (46) |
| COC | AF | Maxilla | F | 22 | 3 months | Alive | (47) |
| OKC | Ameloblastoma | Mandible | F | 17 | 3 months | Alive | (46) |
| OKC | CGCG | Mandible | M | 29 | No data | Alive | (48) |
| OKC | CGCG | Mandible | M | 10 | 3 months | Alive | (49) |
| BPNST | Peripheral osteoma | Palate | M | 61 | 6 months | Alive | (50) |
| AOT | Ameloblastoma | Mandible | F | 31 | No data | Alive | (51) |
| AOT | UA | Mandible | F | 31 | No data | Alive | (9) |
| AOT | FCOD | Maxilla | F | 24 | 9 months | Alive | (52) |
| AOT | FOL | Maxilla | F | 22 | 1 month | Alive | (53) |
| AOT | FOL | Maxilla | F | 34 | No data | Alive | (54) |
| AOT | COF | Maxilla | F | 23 | No data | Alive | (54) |
| AOT | COF | Mandible | F | 30 | 4 months | Alive | (55) |
| COF | CGCG | Mandible | F | 57 | No data | Alive | (56) |
| COF | CGCG | Mandible | F | 15 | No data | Alive | (57) |
| COF | CGCG | Mandible | M | 18 | No data | Alive | (57) |
| COF | CGCG | Mandible | M | 25 | No data | Alive | (57) |
| COF | CGCG | Mandible | M | 50 | No data | Alive | (57) |
| COF | CGCG | Mandible | M | 59 | No data | Alive | (57) |
| COF | CGCG | Mandible | M | 73 | No data | Alive | (57) |
| COF | CGCG | Mandible | M | 19 | No data | Alive | (57) |
| Ameloblastoma | Hemangioma | Mandible | M | 42 | 6 months | Alive | (58) |

*Note*. CCOT: Calcifying cystic odontogenic tumor; COC: Calcifying odontogenic cyst; CEOT: Calcifying epithelial odontogenic tumor; OKC: Odontogenic keratocyst; BPNST: Benign peripheral nerve sheath tumor; AOT: Adenomatoid odontogenic tumor; COF: Central odontogenic fibroma; AFO: Ameloblastic fibro-odontoma; AF: Ameloblastic fibroma; CGCG: Central giant cell granuloma; UA: Unicystic ameloblastoma; FCOD: Focal cemento-osseous dysplasia; FOL: Fibro-osseous lesion; COF: Cemento-ossifying fibroma.

**Table 3.** Collision salivary gland lesions

| First Lesion | Second Lesion | Site | Gender | Age Range (Years) | Duration | Survival Status After Diagnosis Until the Report of Case | Reference |
|---|---|---|---|---|---|---|---|
| Warthin tumor | Small lymphocytic lymphoma | Parotid | M | 60 | 3-4 years | Alive | (59) |
| Warthin tumor | Sebaceous adenoma | Parotid | M | 60 | A few months | Alive | (5) |
| AdCC | Basal cell adenoma | Parotid | M | 62 | A few months | Alive | (5) |
| AdCC | MEC | Palate | F | 49 | No data | Alive | (60) |
| AdCC | EMC | Palate | M | 71 | No data | Alive | (60) |
| AdCC | MEC | Parotid | M | 53 | No data | Alive | (61) |
| AdCC | EMC | Parotid | M | 71 | No data | Alive | (61) |
| AdCC | EMC | Palate | F | 66 | No data | Alive | (5) |
| AdCC | EMC | Maxillary sinus | F | 26 | 3 months | Died after 8 years | (62) |
| AdCC | SDC | Palate | M | 51 | No data | Alive | (61) |
| AdCC | SDC | Parotid | M | 40 | No data | Alive | (6) |
| AdCC | Mucous retention cyst | Parotid | F | 14 | No data | Alive | (63) |
| AdCC | SDC | Sublingual gland | F | 59 | No data | Alive | (64) |
| AdCC | SDC | Submandibular | F | 81 | No data | Died | (6) |
| EMC | SDC | Parotid | M | 28 | Alive | Alive | (61) |
| EMC | AdCC and BCAC | Parotid | M | 51 | 10 years | Alive | (65) |
| EMC | SDC | Parotid | M | 74 | No data | Alive | (66) |
| EMC | BCAC | Parotid | F | 74 | No data | Alive | (6) |
| EMC | BCAC | Parotid | M | 56 | No data | Alive | (6) |
| EMC | Keratinizing SCC | Parotid | F | 73 | No data | Alive | (6) |
| Keratinizing SCC | SDC | Parotid | M | 66 | No data | Alive | (6) |
| Keratinizing SCC | SDC | Lacrimal gland | F | 64 | No data | Alive | (6) |
| ACC | SDC | Parotid | M | 53 | A few weeks | No data | (5) |
| MC | SDC | Parotid | M | 65 | No data | Alive | (6) |
| ACC | SDC | Parotid | M | 42 | No data | Died | (6) |
| Basal cell adenoma | Canalicular adenoma | Parotid | M | 70 | 15-20 years | Alive | (5) |
| Neurofibroma | Schwannoma | Intraparotid facial nerve | F | 29 | 2 years | Alive | (67) |

*Note*. AdCC: Adenoid cystic carcinoma; EMC: Epithelial-myoepithelial carcinoma; ACC: Acinic cell carcinoma; SDC: Salivary duct carcinoma; BCAC: Basal cell adenocarcinoma; MC: Myoepithelial carcinoma.

**Table 4.** Miscellaneous collision lesions in different sites

| First Lesion | Second Lesion | Site | Gender | Age Range (Years) | Duration | Survival Status After Diagnosis Until the Report of Case | Reference |
|---|---|---|---|---|---|---|---|
| OSCC | Papillary thyroid carcinoma | Cervical lymph Node | M | 47 | 2 months | Alive | (68) |
| SCC | Small cell carcinoma | Hypopharynx | M | 74 | 3 months | Alive | (69) |
| SCC and adenocarcinoma | SNEC | Maxillary sinus | F | 52 | 1 month | Alive | (70) |
| SCC | Osteosarcoma | Maxillary sinus | M | 24 | 6 months | Alive | (71) |
| ITAC | PDNC | ethmoid | No data | 79 | No data | 26 months | (72) |

*Note*. SNEC: Small cell neuroendocrine carcinoma; ITAC: Sinonasal intestinal-type adenocarcinoma; PDNC: Poorly differentiated neuroendocrine carcinoma.

tumor (85, 86). Interestingly, CEOT is a benign, slow-growing odontogenic tumor (87), but AOT is considered a hamartoma with a limited growth rate (80). It is hypothesized that neoplastic and hamartomatous lesions can occur at any stage of odontogenesis (82).

Several published reports have documented the coexistence of OKC with other lesions, including CGCG and ameloblastoma (46,48,49). Numerous studies have revealed the association of CGCG with other lesions, such as OKC, COF, and the aneurysmal bone cyst (57,88,89). Three mechanisms have been suggested to explain the coexistence of an odontogenic tumor/lesion and CGCG. Firstly, this combination may result from a "collision tumor/lesion", representing the synchronous occurrence of an odontogenic lesion and CGCG. Secondly, the giant cell reaction induces odontogenic epithelium and ectomesenchyme, typically found in the tooth-bearing areas of the jaws, through the secretion of growth factors (56). The third theory proposes that an odontogenic lesion, such as COF, is a primary lesion inducing a reactive CGCG in the presence of a stimulus, such as trauma (90).

Collision (hybrid) tumors of the salivary glands are very rare and are composed of two or more different entities in a single neoplasm. Salivary gland tumors are made up of biologically and histologically diverse lesions (91, 92). These types of tumors originate in different regions but coalesce in a particular area. Histologically, there is a transitional zone between two entities. Salivary duct stem cells or reserve cells are suggested as the sources of these types of lesions (60,93). The parotid gland is the most common site for salivary gland tumors (91,94). In the present review, parotid tumors were the most common type of salivary gland tumors, and AdCC was the most common salivary gland tumor combined with other lesions (n = 13, 13.4%). In a previous study, SDC was the most common salivary gland tumor combined with other tumors (66).

Overall, a precise diagnosis to distinguish between two different tumors and between benign and malignant lesions in the same area plays an important role in appropriate patient treatment. The treatment plan and prognosis are different and are based on the accompanying tumor. It is also believed that the predominant component determines the biological behavior of the lesion and treatment plan (60). For example, melanomas arising within less aggressive lesions behave less aggressively (12). Furthermore, AOT is typically managed through surgical excision. However, a more extensive radical excision is required when it is associated with other lesions, such as ameloblastoma. Long-term follow-up is crucial in such cases, as ameloblastoma has the potential to metastasize to cervical lymph nodes and distant sites (9). Interestingly, the presence of giant cell granuloma-like areas in COF-like lesions increases the risk of recurrence following curettage (89). Additionally, the prognosis of hybrid cases of COF with a GCG-like component seems to be a locally aggressive course; however, reactive GCG-like lesions and a typical COF have better biological behavior and prognosis (52).

## Conclusion

While the presence of true hybrid tumors is well-established, some reported cases have prompted the researchers to categorize them as biphasic differentiations. The rarity of these cases contributes to limited knowledge regarding their clinical behavior, prognosis, and optimal treatment. It is crucial to emphasize the characteristic and potentially perplexing histological appearances. Molecular analysis proves beneficial in many instances. Hence, a meticulous examination of surgical and histologic specimens is imperative for determining appropriate treatment plans and predicting prognosis.

### Authors' Contribution

**Data curation:** Soussan Irani, Alfred K. Lam.
**Formal analysis:** Soussan Irani.
**Investigation:** Soussan Irani.
**Methodology:** Alfred K. Lam, Soussan Irani.
**Project administration:** Soussan Irani, Alfred K. Lam.
**Supervision:** Soussan Irani.
**Validation:** Soussan Irani.
**Writing–original draft:** Soussan Irani.
**Writing–review & editing:** Soussan Irani, Alfred K. Lam.

### Competing Interests

The authors declare that they have no conflict of interests.

### Ethical Approval

Not applicable.

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