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Commentary

# Sap and Glop-Secretory Carcinoma Salivary Gland

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Secretory carcinoma of salivary gland is an infrequently observed, primary salivary gland tumour. Upon morphological assessment, immunohistochemistry or molecular characteristics, tumefaction simulates secretory carcinoma of breast. Secretory carcinoma of salivary gland was previously designated as mammary analogue secretory carcinoma (MASC).

Specifically, tumefaction harbours ETV6 genetic rearrangement along with the commonly discerned ETV6-NTRK3 genetic translocation t(12;15)(q13;q25).

Neoplasm preponderantly incriminates adult subjects and enunciates diverse histological configurations as cystic, solid, tubular, papillary or lobular. Tumefaction may configure cysts permeated with intraluminal colloid-like secretions.

Tumour cells appear pervaded with bubbly cytoplasm, monomorphic, spherical nuclei and miniature, distinct nucleoli. Upon immunohistochemistry, tumour cells express GATA3, S100 protein, NTRK3, MUC4 or mammaglobin.

Generally, the low grade neoplasm represents with indolent clinical course. However, localized or regional lymph node metastasis may ensue in up to 22% tumours.

Exceptionally occurring high grade neoplastic transformation may be accompanied by aggressive clinical behaviour and emergence of distant metastasis.

Inadequate surgical extermination of the neoplasm is accompanied by locally aggressive biological behaviour and adverse prognostic outcomes. Neoplasms depicting high grade transformation and aggressive clinical course may be accompanied by distant metastasis.

Neoplasm exhibits a mild male predominance with male to female proportion of 1.4:1. Mean age of disease emergence is 49.9 years although tumefaction may arise within a comprehensive range of 5 years to 87 years [1,2].

Of obscure aetiology, secretory carcinoma of salivary gland exhibits repetitive chromosomal translocation t(12;15)(p13;q24) with consequent occurrence of genomic fusion within ETV6 gene situated upon chromosome 12 and NTRK3 gene confined to chromosome 15. Alternate genetic fusion partners as ETV6-RET or ETV6-MET may be exemplified. Additionally, non-ETV6 chromosomal translocations as VIM-RET and CTNNA1-ALK are documented [2,3].

Secretory carcinoma of salivary gland preponderantly incriminates parotid gland (76%), minor salivary glands of oral cavity or submandibular gland. Exceptionally, tumefaction may be confined to nasal cavity, cutaneous surfaces, vulva, pulmonary parenchyma, thyroid gland or lacrimal gland [2,3].

Secretory carcinoma of salivary gland represents as a painless, gradually progressive tumefaction. Mean duration of tumour emergence is one year [2,3].

Upon initial tumour discernment, neoplasm appears within stage T1 or stage T2. With an indolent clinical course, proportionate tumour reoccurrence appears beneath < 10%. Comprehensive, macroscopic tumour resection is associated with overall survival of ~95%.

Incrimination of localized or regional lymph nodes occurs in up to 22% neoplasms whereas distant metastasis ensues in < 5% lesions [2,3].

Cytological examination appears non specific with variable diagnostic outcomes. Cellular smears indicative of the neoplasm and cell block preparations are optimally necessitated for appropriate tumour discernment and cogent immunohistochemistry. Segregation of secretory carcinoma from various benign or low grade, malignant salivary gland neoplasms may be challenging [2,3].

Smears are comprised of cohesive clusters or sheets of epithelial cells permeated with variable quantities of eosinophilic, granular to vacuolated cytoplasm and uniform nuclei with singular nucleoli. Frequently, papillary fragments or acinar configurations are encountered. Cystic lesions exhibit an intermingling of cystic debris. Cell block preparations may be suitably adopted for precise immunohistochemistry with markers such as S100 protein, GATA3 or pan-TRK [2,3].

Grossly, a well circumscribed, non encapsulated, solid or cystic lesion with median magnitude of 20 millimetres and diameter varying from 6 millimetres to 30 millimetres may be delineated. Nevertheless, tumour magnitude of 70 millimetres is documented [3,4].

Upon microscopy, the well circumscribed tumefaction demonstrates an infiltrative perimeter. Although tumour architecture is lobulated, configurations as macro-cystic, micro-cystic, solid, tubular, follicular or papillary-cystic patterns may be exemplified.

Intra-luminal secretions appear as pale, eosinophilic, colloid-like wherein secretions may be highlighted with periodic acid Schiff's (PAS) stain with diastase resistance [3,4].

Tumour cells appear pervaded with eosinophilic or vacuolated cytoplasm and monomorphic, spherical, vesicular nuclei demonstrating miniature, distinctive nucleoli.

Occasionally, foci of lymphatic and vascular invasion or perineural invasion may be enunciated [3,4].

High grade neoplastic transformation may ensue with the emergence of a distinct population of tumour cells exemplifying solid or trabecular configuration, pleomorphic nuclei, comedo-like necrosis, infiltrative tumour perimeter, encompassing desmoplastic stroma, elevated mitotic activity, decimation of secretory activity or perineural invasion. A distinctive component of low grade, conventional secretory carcinoma may be admixed with high grade transformation.

Ultrastructural examination is generally not recommended. However, upon electron microscopy, tumour cells display features of intercalated duct or striated duct cell differentiation [3,4].

Secretory carcinoma of salivary gland appears immune reactive to CK7, GATA3, S100 protein, SOX10, MUC4, mammaglobin or PAN-TRK.

Tumour cells appear immune non reactive to DOG1, NR4A3, p63, CK5/6, calponin, androgen receptor (AR), oestrogen receptor (ER), progesterone receptor (PR) or thyroid transcription factor 1 (TTF1) [5,6].

| Low grade | Intermediate grade | High grade | Variable grade |
|---|---|---|---|
| Acinic cell carcinoma | Myoepithelial carcinoma | Salivary duct carcinoma | Mucoepidermoid carcinoma |
| Basal cell adenocarcinoma | Sebaceous adenocarcinoma | Squamous cell carcinoma | Adenoid cystic carcinoma |
| Epithelial-myoepithelial carcinoma | Lymphoepithelial carcinoma | Small cell carcinoma | Salivary carcinoma NOS |
| Secretory carcinoma | | Large cell neuroendocrine carcinoma | Intraductal carcinoma |
| Polymorphous adenocarcinoma | | Large cell undifferentiated carcinoma | Carcinoma ex pleomorphic adenoma |
| Hyalinising clear cell carcinoma | | Carcinosarcoma | |
| Mucinous adenocarcinoma | | Salivary gland carcinomas with high grade transformation | |
| Micro-secretory adenocarcinoma | | | |
| Sclerosing microcystic adenocarcinoma | | | |
| Sialoblastoma | | | |
| Metastasizing pleomorphic adenoma | | | |

***Table:*** *Histopathological stratification of salivary gland malignancies [3].*

*NOS: Not Otherwise Specified.*

Secretory carcinoma of salivary gland requires segregation from neoplasms such as acinic cell carcinoma, intraductal carcinoma, polymorphous adenocarcinoma or mucoepidermoid carcinoma [5,6].

Secretory carcinoma of salivary gland may be appropriately discerned with imaging modalities as ultrasonography, computerized tomography (CT) or magnetic resonance imaging (MRI).

Cogent discernment upon cytology necessitates confirmatory evidence of suspicious neoplastic emergence and cell block for precise immunohistochemistry.

Radiographic features appear nonspecific. Besides, neoplasms frequently depict a cystic component [5,6].

Primary, comprehensive surgical extermination of the neoplasm is an optimal, recommended therapeutic strategy. Additionally, dissection of regional cervical lymph nodes or adjuvant radiotherapy may be employed for tumours associated with regional lymph node metastasis and concurrent adverse prognostic factors [5,6].

Although unestablished, TRK inhibitors may be adopted to treat lesions delineating ETV6-NTRK3 genetic rearrangements. Nevertheless, initial therapeutic response followed by acquired drug resistance may occur when employed to alleviate advanced disease.

Tumour stage III or stage IV and appearance of high grade morphological features are intensely associated with inferior clinical outcomes [5,6].

Factors contributing to unfavourable prognostic outcomes appear as occurrence of pleomorphic tumour cell nuclei, mitotic figures or foci of comedonecrosis. The exceptionally encountered, high grade neoplastic transformation with aggressive clinical behaviour may be accompanied by distant metastasis [5,6].

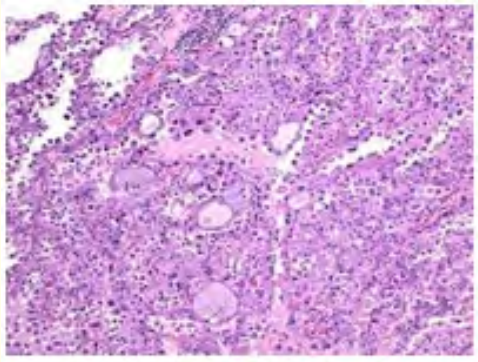

***Figure 1:*** *Secretory carcinoma depicting nests, cords, trabeculae and micro-cysts composed of epithelial cells imbued with eosinophilic cytoplasm and monotonous, spherical nuclei with single nucleolus. Encompassing stroma is desmoplastic [7].*

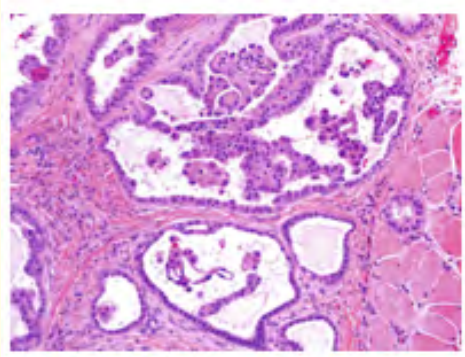

***Figure 2:*** *Secretory carcinoma delineating macro-cysts and trabecular articulations lined by epithelial cells pervaded with eosinophilic cytoplasm, monomorphic spherical nuclei and singular nucleolus. Surrounding stroma is desmoplastic and abundant [8].*

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7. Image 1 Courtesy: Libre pathology.

8. Image 2 Courtesy: Springer link.

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