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Commentary

Spittle and Aqueduct-Salivary Duct Carcinoma

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Received: July 01, 2023; **Published:** July 24, 2023

Salivary duct carcinoma represents as a malignant, high grade salivary gland neoplasm which morphologically simulates invasive ductal carcinoma of breast and manifests an *in situ* and invasive component. Salivary duct carcinoma was initially scripted by Kleinsasser in 1968.

As a malignant, high grade neoplasm of salivary glands delineating histological features akin to invasive ductal carcinoma of breast, tumefaction is predominantly (> 90%) immune reactive to luminal androgen receptors (AR). Besides, foci of *in situ* duct carcinoma frequently display comedo necrosis.

Additionally designated as high grade ductal carcinoma, salivary duct carcinoma represents ~10% of malignant salivary gland neoplasms. Tumefaction is accompanied by inferior prognostic outcomes.

Salivary duct carcinoma commonly incriminates subjects within 6th decade to 7th decade with mean age of disease emergence at 67.4 years. Nevertheless, neoplasm may arise within a comprehensive age range of 33 years to 92 years. A male predilection is encountered with male to female proportion of ~2.4:1 [1,2].

Salivary duct carcinoma preponderantly incriminates parotid gland. Besides, submandibular gland or minor salivary glands may be implicated.

Diverse tumour suppressor genes as TP53, HRAS, PIK3CA, PTEN or BRAF may contribute to genesis of salivary duct carcinoma.

Of obscure aetiology, salivary duct carcinoma may arise de novo in around ~60% instances. Nearly ~40% neoplasms may emerge as salivary duct carcinoma ex pleomorphic adenoma [1,2].

Salivary duct carcinoma manifests as a rapidly progressive tumefaction. Typically, neoplasm is confined to cervical region abutting the parotid gland. Clinical symptoms as facial pain, facial weakness or paralysis may ensue.

Neoplasm is associated with regional lymph node metastasis or distant metastasis within sites as pulmonary parenchyma, bone, brain or diverse sites [1,2].

Cytological examination of the high grade tumefaction exhibits cellular smears comprised of clusters of epithelial cells pervaded with abundant cytoplasm and pleomorphic nuclei. Cellular component is enmeshed within a dirty background. Necrotic substance and apoptotic debris may be discerned. Mitotic figures can be encountered.

Pertinent immunohistochemistry can be beneficially employed upon cell blocks and air dried or alcohol fixed smears for diagnostic confirmation, as neoplastic cells appear immune reactive to androgen receptors (AR). Besides, precise immunostaining can be adopted to exclude malignant metastasis from various high grade, primary neoplasms of the salivary gland [1,2].

Typically, frozen section examination appears superfluous as cytological assessment is confirmatory of a high grade, malignant salivary gland neoplasm. Focal necrosis and cellular pleomorphism are frequently enunciated [1,2].

Macroscopically, mean gross dimension emerges at 3.2 centimetres. Tumour magnitude varies from 0.5 centimetres to 9 centimetres. Characteristically, neoplasm depicts an inadequately defined perimeter although a well circumscribed countenance may be encountered.

Cut surface is heterogeneous and exemplifies foci of gross tumour necrosis. Regional lymphadenopathy is commonly delineated [2,3].

Upon microscopy, the high grade, infiltrative neoplasm configures cords, nests or cribriform glandular articulations encompassed within a desmoplastic stroma.

Tumour cells are incorporated with abundant eosinophilic or oncocytic cytoplasm with significant nuclear pleomorphism, anisocytosis and enhanced nucleo-cytoplasmic ratio. Frequently, *in situ* neoplastic component is associated with centric tumour necrosis [2,3].

Secondary salivary duct carcinoma may demonstrate foci of pleomorphic adenoma intensely concurrent with the malignant component. Around ~40% of salivary duct carcinomas appear as secondary neoplasms.

Salivary duct carcinoma exhibits histological variants which enunciate focal areas demonstrating conventional histological features. Aforesaid variants delineate morphological features of mucin rich, micro-papillary, sarcomatoid, rhabdoid or basal-like salivary gland tumours [2,3].

Salivary duct carcinoma is preponderantly (> 90%) immune reactive to androgen receptors, GATA3, cytokeratin AE1/AE3, CK7, 34βE12 or epithelial membrane antigen (EMA). Majority (~80%) of lesions appear immune reactive to gross cystic disease fluid protein-15 (GCDFP-15). Besides, neoplastic cells are immune reactive to prostate specific antigen (PSA) or HER2. Mucin rich variant appears immune reactive to mucicarmine [3,4].

Salivary duct carcinoma appears immune non reactive to S100 protein, DOG1 or SOX10. Majority of neoplasms are immune non reactive to p63, CK5/6, oestrogen receptor (ER) or progesterone receptor (PR).

Salivary duct carcinoma exhibits genetic alterations within PLAG1, HMGA2, PIK3CA, HRAS, TP53, ERBB2 or BRAF genes [3,4].

Salivary duct carcinoma requires segregation from neoplasms such as metastatic adenocarcinoma occurring due to primaries from invasive ductal carcinoma breast, pulmonary adenocarcinoma, prostatic adenocarcinoma, metastatic squamous cell carcinoma, high grade mucoepidermoid carcinoma, epithelial-myoepithelial carcinoma, myoepithelial carcinoma, adenoid cystic carcinoma, adenosquamous carcinoma or intra-ductal carcinoma (cribriform cystadenocarcinoma) [3,4].

Characteristically, tumefaction is confined to cervical region and may be appropriately accessible to imaging studies and fine needle aspiration cytology (FNAC). Incriminated subjects demonstrate occurrence of pleomorphic adenoma for an extended duration which progresses rapidly [4,5].

Fluorescent *in situ* hybridization (FISH) exhibits genetic amplification of HER2. Cogent immunohistochemistry for HER2 may be advantageously adopted for neoplastic discernment wherein tissue samples appear amplified in instances where average copy number ratio (HER2/CEP17) is ≥ 2.0.

Upon computerized tomography (CT), tumefaction with characteristic heterogonous image enhancement is encountered. Foci of calcification may be detected [4,5].

Magnetic resonance imaging (MRI) exemplifies an invasive neoplasm with inadequately defined tumour margin. Few (~15%) neoplasms may appear as well circumscribed.

T1 weighted magnetic resonance imaging depicts a hypo-intense neoplasm with decimated signal intensity, in contrast to circumscribing salivary gland tissue. Tumefaction appears isointense as compared to adjoining skeletal muscle [4,5].

T2 weighted magnetic resonance imaging displays a hyper-intense tumefaction with enhanced signal intensity, in contrast to contralateral parotid gland. Cellular neoplastic components depict preliminary image enhancement with elevated washout ratio [4,5].

Fibrotic areas and zones with decimated cellularity enunciate gradual, upward image enhancement. Cellular tumour zones with focal necrosis display preliminary image enhancement and inadequate washout. Salivary duct carcinoma can be appropriately treated with total parotid resection along with dissection of incriminated cervical lymph nodes.

Adjuvant radiotherapy appears beneficial as a therapeutic strategy for treating high grade salivary duct carcinoma [4,5].

Advanced disease can be subjected to chemotherapy with agents as antiandrogens, trastuzumab, anti-BRAF or anti-PIK3CA molecules. Salivary duct carcinoma is associated with inferior prognostic outcomes. Overall survival appears at ~35%. Median overall survival is contemplated to be ~3.1 years [4,5].

Factors contributing to adverse prognostic outcomes emerge as

- Advanced tumour stage, especially neoplasms associated with regional lymph node metastasis ≥ N2.
- Perineural invasion, particularly incrimination of facial nerve which may be resected during therapeutic surgical intervention.
- Extra-nodal tumour extension.
- Age of incriminated subject ≥ 50 years.

Proportionate 5 year survival for stage I salivary duct carcinoma emerges at 42% and 23% for stage IV tumours [4,5].

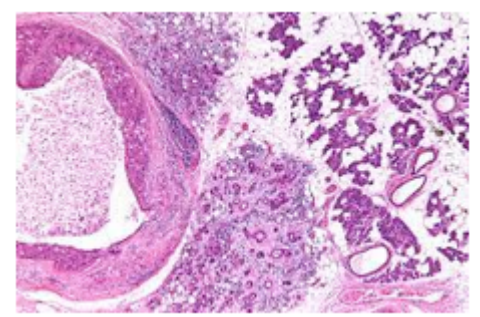

Figure 1: *Salivary gland carcinoma depicting cords, nests and cribriform structures lined by epithelial cells incorporated with abundant, eosinophilic cytoplasm and pleomorphic nuclei with enhanced nucleo-cytoplasmic ratio. Surrounding stroma is desmoplastic [6].*

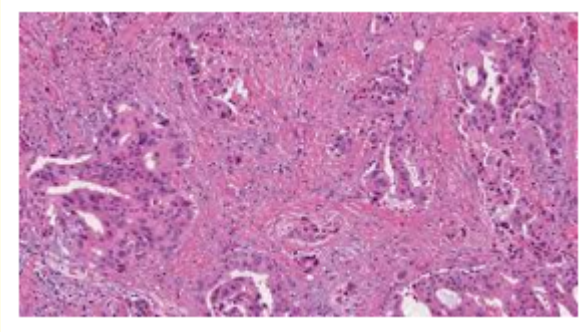

Figure 2: *Salivary duct carcinoma exemplifying cords, nests and trabeculae lined by epithelial cells permeated with abundant, eosinophilic cytoplasm and pleomorphic nuclei with enhanced nucleo-cytoplasmic. Tumour cell aggregates appear enmeshed within a desmoplastic stroma [7].*

Bibliography

1. Liu F., *et al*. "Comprehensive diagnosis and treatment of ductal carcinoma of the submandibular gland: case report and literature review". *International Journal of Clinical and Experimental Pathology* 16.3 (2023): 67-75.
2. Young A and Okuyemi OT. "Malignant Salivary Gland Tumors". Stat Pearls International. Treasure Island, Florida (2023).
3. Bou Zerdan M., *et al*. "Molecular Targets in Salivary Gland Cancers: A Comprehensive Genomic Analysis of 118 Mucoepidermoid Carcinoma Tumors". *Biomedicines* 11.2 (2023): 519.
4. Laughlin BS., *et al*. "Clinicopathologic Factors and Their Association with Outcomes of Salivary Duct Carcinoma: A Multicenter Experience". *Advances in Radiation Oncology* 8.4 (2023): 101204.
5. Cunha JL., *et al*. "Salivary gland tumors: A 13-year clinicopathologic retrospective study in a Brazilian northeast population". *Journal of Clinical and Experimental Dentistry* 15.2 (2023): e88-e95.
6. Image 1 Courtesy: Wikipedia
7. Image 2 Courtesy: My pathology report.

Volume 22 Issue 8 August 2023