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Editorial

# Estuary and Trench-Canalicular Adenoma Salivary Gland

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Canalicular adenoma of salivary gland emerges as a benign epithelial neoplasm constituted of branching, interconnected cords of cuboidal to columnar epithelial cells circumscribed by a pauci-cellular and vascularized stroma. Initially scripted by McFarland in 1942, the neoplasm was contemplated as a variant of monomorphic or basal cell adenoma. Notwithstanding, World Health Organization categorized the neoplasm as a distinct entity in 1991.

The nomenclature of canalicular tumour, canalicular mixed tumour, monomorphic adenoma, canalicular subtype, cystic adenoma or adenomatosis of accessory salivary glands appears obsolete.

The benign epithelial neoplasm is pre-eminently composed of monomorphic epithelial cells and is devoid of a distinct basal or myoepithelial cell layer. Tumefaction is frequently confined to minor salivary glands wherein multifocal lesions may be exemplified.

Canalicular adenoma commonly emerges within the adult population >50 years with peak age of disease occurrence at seventh decade. A female predominance is observed. Non Asian ethnic groups are frequently implicated [1,2].

Canalicular adenoma configures up to 3% of salivary gland tumours and 4% to 6% of minor salivary gland tumours [1,2].

Canalicular adenoma of salivary gland occurs within the upper lip (80%) followed by buccal mucosa, lower lip and hard and soft palate.

Tumour emergence within major salivary glands as the parotid is extremely exceptional wherein a pleomorphic adenoma demonstrating HMGA2-WIF1 genetic fusion may require distinction [1,2].

Of obscure aetiology, canalicular adenoma is posited to arise from terminal ducts of salivary glands wherein intra-lobular ducts appear to simulate intercalated ducts [1,2].

Cytogenetic analysis depicts doubling of normal complement. Genetic fusions appear absent.

Clinically, tumefaction represents as a gradually progressive, painful or painless swelling or lesion. Singular or bilateral, multifocal neoplasms may be observed. Clinical symptoms as localized pressure or discomfort along with or devoid of prosthesis may ensue [2,3].

Ulceration of superimposed mucosa may be encountered. Neoplastic occurrence may be delayed to months or years. Occasionally, canalicular adenoma may configure as collision tumour or hybrid tumour. Canalicular adenoma of salivary gland may be appropriately

discerned by cogent clinical examination wherein a mobile, well circumscribed tumour nodule of magnitude varying from 0.5 centimetres to 2 centimetres is observed. Alternatively, bilateral or multiple nodules or an exophytic mass or swelling appears to be superimposed by ulcerated, blue-tinged mucosa [2,3].

Cytological smears display pseudo-papillary clusters of elliptical, spindle shaped or basaloid tumour cells pervaded with scanty or eosinophilic cytoplasm. Alternatively, isolated tumour cells permeated with bland nuclear chromatin and absent nucleoli are encountered. Intervening chondroid or myxoid substance appears absent [2,3].

Grossly, neoplasm is encapsulated. Singular or multifocal tumours may be encountered. Singular tumour nodule or fragmented tissue specimens may be obtained. Cut surface appears homogeneous or cystic spaces may be discerned [2,3].

Upon microscopy, the well vascularized neoplasm demonstrates dual cellular layers, strands, ribbons, anastomosing cords or branching tubules. Canalicular adenoma expounds cystic spaces intermingled between strands of cells or cellular trabeculae. A distinct 'beading' configuration or cellular cords with 'clubbed' terminals is exemplified. An extraneous layer of myoepithelial cells is absent.

Neoplastic cells may infiltrate circumscribing capsule with occurrence of extracapsular tumour islands, a feature expounded within normal salivary gland tissue or multiple tumours. Cystic alterations are frequently encountered [3,4].

A layer of columnar or cuboidal epithelial cells configures the neoplasm with foci of basaloid cells. Tumour cells are imbued with amphophilic to eosinophilic cytoplasm, uniform spherical to elliptical nuclei, focal nucleoli and basophilic nuclear chromatin. A population of apocrine cells or oncocytic cells and pigmented cells is observed.

Focal mucous or mucinous metaplasia may occur. Additionally, features as microliths, tyrosine crystals, morules and intraluminal squamous balls may be encountered [3,4].

Canalicular lumen or stroma may depict a population of foamy, lipofuscin or hemosiderin laden macrophages. Lumens may depict focal haemorrhage. Encompassing stroma is well vascularized and loose with occurrence of perivascular eosinophilic cuffs. Alternatively, enveloping stroma may be sclerotic. Focal degeneration or infarction of surrounding stroma is observed [3,4].

Mitotic figures and tumour necrosis are minimal to absent. Cellular or nuclear atypia appears absent. Malignant transformation remains undocumented [3,4].

Ultrastructural examination depicts dual cellular layer composed of tall columnar cells circumscribing canalicular lumens and conical cells disseminated amidst columnar cells wherein cellular apposition with stromal connective tissue is maintained. A component of interdigitating cells is encountered.

Canalicular lumen is impregnated with minimal quantities of cellular debris whereas mucoid substance is absent. Few desmosomes are discerned.

Tumour cells appear imbued with ovoid nuclei and prominent nucleoli along with focal condensation of nuclear chromatin [4,5].

Staging of malignant salivary gland tumours is designated as [5,6]:

- Stage 0: Tis, N0, M0.
- Stage I: T1, N0, M0.

- Stage II: T2, N0, M0.
- Stage III: T3, N0, M0 OR T0, T1, T2, T3, N1, M0.
- Stage IVA: T0, T1, T2, T3 or T4a, N2, M0 OR T4a, N0 or N1, M0.
- Stage IVB: Any T, N3, M0 OR T4b, any N, M0.
- Stage IVC: Any T, any N, M1.

Canalicular adenoma appears immune reactive to S100 protein, cytokeratin AE1/AE3, CK19, pancytokeratin, CK7, CK8, CK13, CK14, CD117 or BCL2. Focal immune reactivity to glial fibrillary acidic protein (GFAP) is encountered [4,5].

Variable immune expression of p63 with nuclear p63 within squamoid foci and absence of p63 within duct surrounding cells is observed. Nuclear immune reactivity to CAM5.2, SOX10 and nuclear and cytoplasmic immune reactivity to p16 and vimentin may ensue. Squamous cell balls appear immune reactive to p16 and CK5/6. Heterogeneous, intermediate to intense immune reactivity to cytoplasmic WT1 may emerge within periphery of ribbon-like structures, within micro-adenomas or accompanying cuboidal epithelial cells [4,5].

Surrounding stroma appears blue upon staining with Alcian blue, Periodic acid Schiff's stain and is immune reactive to CD15.

Tumour cells appear immune non reactive to E-cadherin, diverse myoepithelial cell markers as alpha smooth muscle actin (α SMA), smooth muscle myosin heavy chain, calponin, p40, DOG1, carcinoembryonic antigen (CEA) or epithelial membrane antigen (EMA) [5,6].

Canalicular adenoma of salivary gland requires segregation from neoplasms as adenocarcinoma not otherwise specified (NOS), adenoid cystic carcinoma, pleomorphic adenoma, polymorphous low grade adenocarcinoma or basal cell adenoma [5,6].

Clinically, demarcation from lesions such as mucocele, thrombosed blood vessels, lipoma or diverse salivary gland tumours is necessitated [5,6].

Precise biochemical or serological parameters indicative of the lesion appear absent.

Upon ultrasonography, a predominantly homogeneous, minimally echoic tumour mass impregnated with liquid substance of minimal viscosity is observed.

Upon adoption of magnetic resonance imaging (MRI), axial T1 weighted MRI delineates an isointense tumour mass. T2 weighted MRI is associated with enhanced signal intensity [5,6].

Canalicular adenoma may be appropriately subjected to precise surgical extermination. Nevertheless, multi-focal lesions appear to indicate tumour reoccurrence [5,6].

Comprehensive surgical resection of the tumour is associated with favourable prognostic outcomes. Neoplastic persistence may ensue on account of multi-focal lesions and cogent distinction from tumour reoccurrence may be challenging. An estimated 3% lesions display tumour reoccurrence whereas nearly 5% neoplasms may reappear following surgical intervention [5,6].

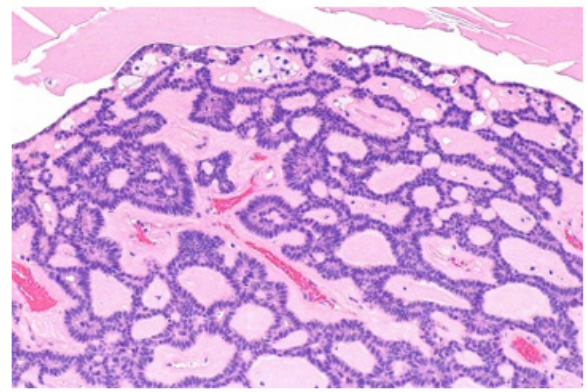

***Figure 1:*** *Canalicular adenoma demonstrating strands, cords, ribbons and canals layered by columnar to cuboidal cells surrounded by loose, vascularized stroma. Canalicular lumens are imbued with exfoliated squamous cells and red cells [7].*

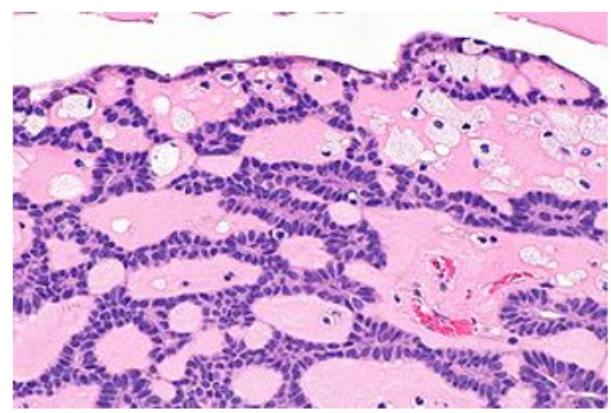

***Figure 2:*** *Canalicular adenoma delineating cords, strands, dual layers and ribbons layered by columnar to cuboidal cells surrounded by loose, vascularized stroma. Canalicular lumens are pervaded with exfoliated squamous cells and red cells [8].*

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7. Image 1 Courtesy: Wikimedia commons.

8. Image 2 Courtesy: Libre Pathology.

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