Switch and Variance-Warthin’s Tumour

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Warthin’s tumour is a benign salivary gland neoplasm commonly incriminating parotid gland. Tumefaction enunciates cystic configurations coated with a dual epithelial cell layer with subjacent dense, lymphoid stroma. Additionally designated as papillary cystadenoma lymphomatosum papilliferum or adenolymphoma, Warthin’s tumour is frequently expounded and follows pleomorphic adenoma in frequency. Generally, Warthin’s tumour occurs due to incorporation of lymphoid tissue within the parotid gland. Alternatively, inflammatory infiltrate may induce cystic and oncocytic modifications within pertinent salivary gland. Neoplasm may be concordant with tubercular infection induced by Brucella melitensis [1, 2]. Warthin’s tumour is a non clonal, diploid neoplasm. However, neoplasms concurrent with muco-epidermoid carcinoma appear associated with chromosomal translocation t(11;19) and fusion transcript of CRTC1/MAML2 genes. Besides, deoxy-ribonuclease (DNA) of human herpesvirus 8 (HHV8) may accompany Warthin’s tumour [1, 2].

Warthin’s tumour is commonly discerned in adults > 40 years. A male preponderance is observed. Cigarette smoking may contribute to disease emergence [1, 2].

Warthin’s tumour is preponderantly confined to the parotid gland and configures an estimated 10% of neoplasms incriminating parotid gland. An estimated ~15% neoplasms emerge as multifocal or bilateral lesions. Around 70% of bilateral salivary gland neoplasms are comprised of Warthin’s tumour. Occasionally, tumefaction may appear within oral cavity, larynx or cervical lymph nodes [1, 2].

Grossly, Warthin’s tumour is an encapsulated, lobulated neoplasm. Cut surface appears pale grey. The multi-cystic tumefaction is imbued with mucinous or serous secretions [1, 2]. Tumour magnitude varies between 2 centimetres to 5 centimetres. Tumefaction may be adherent to superimposed cutaneous surface. Neoplasm may undergo haemorrhagic infarction, especially following manoeuvres such as fine needle aspiration [1, 2].

Cytological examination exhibits degenerated oncocyes preponderantly depicting a ‘squamous’ countenance.

Sheets of oncocyes appear admixed with aggregates of chronic inflammatory cells as lymphocytes characteristically disseminated within a ‘dirty’ proteinaceous substance [1, 2].

Clusters and aggregates of oncocyes demonstrate an organized ‘honeycomb’ pattern or papillary configurations. Upon Papanicolaou stain, cell clusters typically depict peripheral cyanophilia with a centric, bright orange zone [1, 2].

Oncocytes Are permeated with dense, granular cytoplasm, centric nuclei and miniature nucleoli. Intermingled lymphocytes configure as miniature, mature cells [1, 2].

Upon microscopy, neoplasm depicts a dual layer of epithelial cells with subjacent dense, lymphoid stroma incorporated with variable germinal centres. Cystic tumour spaces appear confined by polypoid projections constituted of admixed lymphoid and epithelial
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cellular constituents. Oncocytes and layering columnar epithelial cells depict palisading of superficial cells with discontinuity of subjacent basal cells [1, 2]. Cellular features such as cilia, squamous metaplasia associated with infarct-like necrosis, mast cells, dendritic cells, mucin secreting cells or sebaceous cells are occasionally discerned. Signet ring cells are extremely exceptional. A distinctive myoepithelial cell layer is absent.

Upon ultrastructural examination, oncocytes appear permeated with ‘cup’ shaped mitochondria or mitochondrial organelles demonstrating ‘concentric rings’. However, mitochondria are devoid of partitions [1, 2].

**Figure 1:** Warthin’s tumour demonstrating aggregates of mature lymphocytes configuring germinal centres superimposed with dual epithelial cell layer delineating significant oncocytic change [5].

**Figure 2:** Warthin’s tumour depicting lymphoid rich stroma surrounding salivary glandular articulations layered by dual layer of epithelium enunciating significant oncocytic change [6].
Warthin’s tumour is immune reactive to keratin, CK7, CK8/18, CK19 and mitochondrial markers. Focal immune reactivity to carci-noembryonic antigen (CEA), CK5/6, CK10, CK14, p63 or 34βE12 is observed.

Warthin’s tumour is immune non reactive to amylase, vimentin, desmin, smooth muscle actin (SMA), Epstein Barr virus (EBV) or human herpesvirus 8 (HHV8) [3, 4].

Warthin’s tumour requires segregation from neoplasms such as acinic cell carcinoma, squamous cell carcinoma, metastasis into regional lymph nodes, sebaceous lymphadenoma, cystadenomas of salivary glands or lympho-epithelial cysts [3, 4]. Warthin's tumour can be appropriately subjected to surgical exetermination of the neoplasm. An estimated 2% neoplasms reoccur following comprehensive surgical resection [3, 4]. Warthin's tumour may appear synchronous with pleomorphic adenoma and salivary duct carcinoma. Nearly 1% tumefaction undergo malignant metamorphosis which may manifest as lymphoma, Merkel cell carcinoma, adenocarcinoma-not otherwise specified (NOS), basal cell carcinoma, mucoepidermoid carcinoma, oncocytic carcinoma, salivary duct carcinoma, squamous cell carcinoma or Warthin’s adenocarcinoma [3, 4].

References
5. Image 1 Courtesy: Libre Pathology.