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Editorial

Fuchsia and Roseate - Hürthle Cell Adenoma

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Hürthle cell adenoma is a thyroid neoplasm demonstrating follicular architecture with > 75% tumour cells configured as oncocytes which demonstrate intracytoplasmic accumulation of dysfunctional mitochondria. Morphological features as tumour magnitude, nuclear atypia, multinucleated cells, cellular or nuclear pleomorphism, enhanced mitotic activity or definitive histologic configurations appear non indicative of malignant metamorphosis.

Additionally designated as oncocytic cell adenoma, Askanazy cell adenoma or oxyphilic cell adenoma, exogenous factors influencing emergence of oncocytic neoplasms remain undefined [1,2].

Hürthle cell carcinoma is a malignant, follicular, oncocytic neoplasm delineating capsular or vascular invasion. Malignant neoplasms are preponderantly associated with haematogenous tumour dissemination with few metastases occurring within regional lymph nodes. In contrast to conventional follicular carcinoma, Hürthle cell carcinoma demonstrates aggressive biological behaviour [1,2].

Oncocytic neoplasms are commonly associated with aneuploidy and chromosomal gains or losses. Genomic mutations of PTEN and TP53 are delineated [1,2].

Generally, genomic deletions or mutation of mitochondrial DNA (mtDNA), which encodes for oxidative phosphorylation (OXPHOS) proteins, is observed. Consequently, deficient energy production and compensatory mitochondrial proliferation ensues [1,2].

Loss of functional mutation of MEN1 gene is infrequently observed.

Hürthle cell carcinoma is frequently discerned within elderly male population with a mean age of disease emergence at 57 years.

Upon gross examination, tumefaction is solitary, solid, encapsulated and lobulated with a centric scar [1,2].

Majority of neoplasms exceed > 2 centimetre diameter and appear bright to mahogany brown. Hürthle cell adenoma is prone to infarction, particularly subsequent to an invasive procedure as fine needle aspiration or core needle biopsy [1,2].

Tumours with significant vascular or lymphatic invasion appear multinodular with satellite nodules and demonstrate an irregular perimeter [1,2].

Predominantly cellular cytological aspirate is composed of ≥ 75% enlarged, pleomorphic, dis-cohesive Hürthle cells demonstrating abundant, granular cytoplasm and spherical nuclei with intracytoplasmic lumina or vacant vacuoles. Upon staining with Diff quik, aforesaid vacuoles appear magenta or represent as green vacuoles with Papanicolaou stain. Transgressing vascular articulations or capillaries appear to circumscribe clusters of Hürthle cells [1,2].

Generally, aspirate is devoid of colloid, inflammatory cells as lymphocytes, histiocytes or plasma cells or normal follicular epithelial cells. Malignant cells appear dis-cohesive, miniature or enlarged and exhibit significant dysplasia with nuclear crowding.

Distant, primary neoplasms metastatic to thyroid gland enunciate bland cytological features [1,2].

Upon microscopy, tumefaction is comprised of ≥75% oncocytic cells or enlarged cells with distinct cellular outline, granular,

intensely eosinophilic cytoplasm, enlarged nucleus with prominent nucleolus and an absence of cellular polarity. Nuclear grooves or nuclear pseudo-inclusions are occasional. Nuclear atypia is common and random, although non representative of malignant transformation. Tumour configurations as follicular, trabecular, solid or papillary patterns may be observed [1,2].

Follicular lumen is impacted with calcific concretions as non-laminated, psammoma body-like aggregates, as apposed to laminated, psammoma bodies confined to stroma of papillary thyroid carcinoma.

Hürthle cell carcinoma exhibits a solid or trabecular tumour configuration. Miniature tumour cells demonstrate enhanced nuclear/cytoplasmic ratio and focal clear cell change on account of distended mitochondria. Tumefaction is circumscribed by a dense capsule. Mitotic activity is increased [1,2].

Poorly differentiated oncocytic carcinoma commonly enunciates a magnitude > 4 centimetres with focal aggregates of miniature tumour cells. Besides, foci of tumour necrosis are observed. Mitotic activity is prominent [1,2].

Ultrastructural examination exhibits innumerable intracytoplasmic mitochondria [1,2].

Figure 1: Hurthle cell adenoma demonstrating enlarged, oncocytic cells with abundant, eosinophilic cytoplasm, moderate nuclear pleomorphism and traversing fibrous tissue septa. Atypia is minimal [5].

Figure 2: Hurthle cell carcinoma depicting enlarged oncocytes with abundant, granular eosinophilic cytoplasm, spherical nuclei with pleomorphism and inspissated colloid. Atypia and mitotic activity is minimal [6].

Hürthle cell adenoma is immune reactive to thyroglobulin, TTF1 or CK7. Tumefaction is immune non reactive to CK20.

Poorly differentiated oncocytic carcinoma is immune non reactive to TTF1 or thyroglobulin [3,4].

Hürthle cell adenoma requires segregation from disorders such as Hashimoto's thyroiditis, medullary carcinoma, nodular colloid goitre with prominent oncocytic cells, follicular thyroid carcinoma or papillary thyroid carcinoma with subtypes as oncocytic variant, Warthin-like variant or tall cell variant [3,4].

Upon imaging with radioactive iodine, Hürthle cell adenoma manifests as a 'cold' nodule. 'Warm' or 'hot' nodules are exceptional [3,4].

Hürthle cell carcinoma represents with characteristic histological features and decimated uptake of radioactive iodine upon imaging. Hürthle cell adenoma can be adequately treated with lobectomy of incriminated thyroid lobe. Hürthle cell carcinoma can be subjected to total thyroidectomy or radiofrequency ablation [3,4].

As compared to conventional follicular carcinoma, Hürthle cell carcinoma appears unresponsive to radioactive iodine [3,4].

The benign Hürthle cell adenoma is devoid of localized tumour reoccurrence following comprehensive surgical extermination of the neoplasm [3,4].

In contrast to conventional follicular carcinoma, Hürthle cell carcinoma is an aggressive lesion with frequent extra-thyroidal extension, localized tumour reoccurrence, metastasis to regional lymph nodes and proportionate mortality of up to 80% [3,4].

Inferior prognostic outcomes appear with tumours emerging within elderly population, tumour magnitude > 4 centimetres and extensive vascular invasion [3,4].

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5. Image 1 Courtesy: Webpathology.com.
6. Image 2 Courtesy: Pathology outlines.