Online Journal of Gynecology, Obstetrics and Maternity Care
ISSN: 2642-0163

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Editorial *Volume 6 Issue 1*

The Lanky Antipode-Tall Cell Carcinoma with Reverse Polarity

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Received Date: February 17, 2024; **Published Date:** March 05, 2024

Editorial

Tall cell carcinoma with reverse polarity is an exceptional subtype of papillary carcinoma of breast. Neoplasm characteristically displays IDH2 p.Arg172 hotspot genetic mutation and enunciates minimal malignant metamorphosis. Cytological examination singularly disseminated or nests and papillary configurations of tumour cells impregnated with grooved nuclei. Microscopically, solid aggregates, circumscribed nodules or attenuated, abridged papillae composed of cuboidal, columnar or tall, columnar cells pervaded with abundant, eosinophilic cytoplasm and bland nuclei situated upon the apical pole are observed. Tumour cells appear immune reactive to high molecular weight and low molecular weight keratin CK5/6, CK7, IDH1/1DH2, E- cadherin, MUC1, GATA3, gross cystic disease fluid protein 15(GCDFP-15), mammaglobin or calretinin. Tall cell carcinoma with reverse polarity requires segregation from neoplasms as solid papillary carcinoma, encapsulated papillary carcinoma, intraductal papilloma with usual ductal hyperplasia or metastatic thyroid carcinoma. Ultrasonography may delineate a solid, hypoechoic tumefaction along with or devoid of posterior shadowing. Neoplasm may be optimally subjected to surgical extermination.

Tall cell carcinoma with reverse polarity is an exceptionally discerned subtype of papillary carcinoma of breast associated with minimal possible occurrence of malignant metamorphosis. Initially scripted in 2003, tumefaction is constituted of solid tumor nodules composed of columnar epithelial cells. Morphologically, neoplasm is reminiscent of tall cell variant of papillary thyroid carcinoma and frequently expounds distinctive hotspot mutations within IDH2 R172 gene. Several tumor nodules are impregnated with attenuated fibro-vascular cores with consequent occurrence of solid nests or papillary architecture Constituent epithelial cells are pervaded with abundant, glassy eosinophilic cytoplasm with anomalous, apical nuclei, thereby inducing a 'reverse' nuclear polarity. Tumefaction expresses a triple negative immune phenotype with low malignant potential.

Additional nomenclature as solid papillary carcinoma with reverse polarity (SPCRP), breast tumor simulating tall cell variant of papillary thyroid carcinoma or solid papillary breast carcinoma resembling tall cell variant of papillary thyroid neoplasm are not recommended. Commonly implicating the mammary tissue, tall cell carcinoma with reverse polarity is an exceptionally encountered neoplasm arising within postmenopausal female subjects. Median age of disease emergence is 64 years and the neoplasm may appear within 39 years to 89 years [1,2].

Of obscure pathogenesis and physiology, majority (~84%) of neoplasms characteristically display IDH2 p.Arg172 hotspot genetic mutation. Majority (~100%) of neoplasms expound hotspot mutations within IDH2 R172. Nearly two thirds (~67%) of neoplasms depict hot spot mutations within PIK3CA gene. Besides, TET2 truncating mutation, PRUNE2 chromosomal mutation and ATM, KIT or MET genetic alterations may be encountered. Neoplasm appears devoid of BRAF genetic mutation and RET / PTC genomic rearrangements [1,2]. Cytological examination exhibits significantly cellular smears. Tumour cells appear singularly disseminated or may configure nests and papillary configurations. Tumour cells are impregnated with grooved nuclei [2,3]. Grossly, a well circumscribed, firm, grey/ white tumefaction is observed. Median tumour magnitude appears at 1.5 centimetres with tumour magnitude varying from 0.6 centimetres to 5 centimetres. Occasionally, neoplasm may be cystic and demonstrates translucent, colloid-like areas reminiscent of thyroid tissue [2,3].

Upon microscopy, solid aggregates, circumscribed nodules or attenuated, abridged papillae configured of epithelial cells are observed. The cellular component is encompassed within a dense, fibrous tissue stroma. Tumefaction is constituted of cuboidal, columnar or tall, columnar cells pervaded with abundant, eosinophilic cytoplasm and bland nuclei situated upon the apical pole, thereby configuring a 'reverse' nuclear polarity [3,4]. Tumour cells variably express nuclear grooves with intra-nuclear pseudo-inclusions. Besides, true papillae and cystic spaces impregnated with amphophilic, colloid-like secretions may be discerned. Aggregates of foamy histiocytes may appear confined to fibro-vascular cores. Mitotic figures are exceptionally discerned [3,4] (Figures 1 & 2, Table 1).

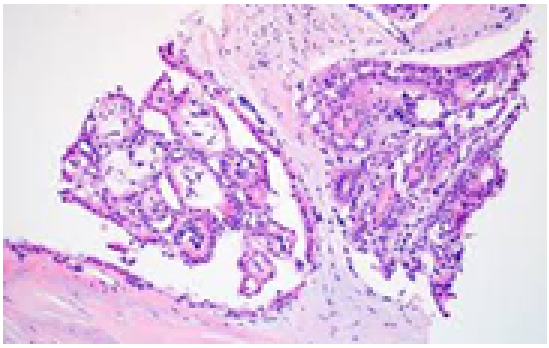

Figure 1: Tall cell carcinoma with reverse polarity demonstrating papillary structures layered by tall columnar epithelial cells pervaded with abundant eosinophilic cytoplasm and apical nuclei [5].

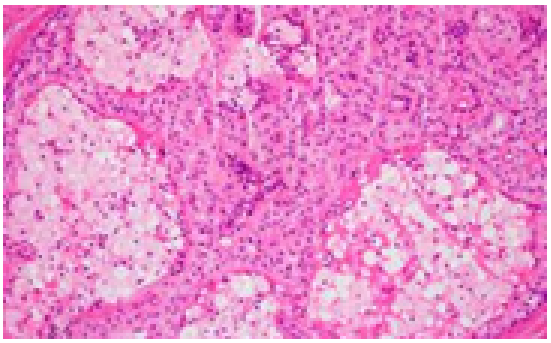

Figure 2: Tall cell carcinoma with reverse polarity delineating papillary articulations layered by tall columnar epithelial cells permeated with abundant eosinophilic cytoplasm and apical nuclei [5].

Feature	Apocrine Carcinoma	Secretory Carcinoma	Solid Papillary Carcinoma	Encapsulated Papillary Carcinoma	Papillary Thyroid Carcinoma	TCCRP
Papillary architecture	Uncommon, focal	Uncommon	Common, solid papillary enclosing sinusoidal vessels	Common, well developed papillary architecture with cystic change and haemorrhage	Common	Common
Secretion	Absent	Common including intra and extracellular PAS+ secretion	Extracellular mucin is common	Absent	Common, follicles with colloid	Colloid-like

Cells	Large with vesicular nuclei and ample granular eosinophilic cytoplasm. Can be low, intermediate or high grade	Low to intermediate grade nuclei, inconspicuous nucleoli, prominent secretions	Low to intermediate grade nuclei, inconspicuous nucleoli	Low to intermediate grade nuclei, infrequent mitoses	Ovoid crowded pale nuclei with nuclear grooves and pseudo inclusions	Pale cells, low to intermediate grade nuclei, inconspicuous nucleoli
Nuclear polarity	Normal	Normal	Normal	Normal	Normal	Reverse
Immunohisto chemistry	GCDFP-15, GATA-3, AR, HER2, reactive ER/PR non reactive	Triple negative, NTRK , alpha1 antitrypsin reactive	Neuroendocrine marker reactive, myoepithelial staining in and around lesion are absent	Myoepithelial staining in and around lesion absent Neuroendocrine marker s non reactive	TTF1, thyroglobulin, PAX8, CK7, AE1/3, CAM5.2 reactive	Triple negative, low and hi gh molecular weight cytokeratin, GATA-3, antimitochondrial antibody IDH2 reactive
Molecular profile	Non specific	ETV6-NTRK3 genetic fusion	PIK3CA (~45%)	Non specific	BRAF, RET mutations	IDH2 p.Arg172 mutations

Table 1: Histological Features, Molecular Characterization and Differential Diagnosis of Tall Cell Carcinoma with Reverse Polarity [1].

Tall cell carcinoma with reverse polarity appears immune reactive to high molecular weight and low molecular weight keratin CK5/6, CK7, IDH1/1DH2, E- cadherin, MUC1, GATA3, gross cystic disease fluid protein 15(GCDFP-15), mammaglobin or calretinin. Ki67 proliferative index is minimal and appears <5% [6,7]. Sensitivity to monoclonal antibody 11C8B1 (93%) and specificity for IDH2 R172 (100%) hotspot chromosomal mutations is encountered. Tumour cells expound a triple negative phenotype with hormone receptors, HER2, oestrogen receptors(ER) or progesterone receptors (PR). Tumour cells appear immune non-reactive to myoepithelial markers as p63, SMMHC, calponin, thyroid transcription factor 1(TTF1), thyroglobulin, chromogranin A or synaptophysin [6,7].

Tall cell carcinoma with reverse polarity requires segregation from neoplasms as solid papillary carcinoma, encapsulated papillary carcinoma, intraductal papilloma with usual ductal hyperplasia or metastatic thyroid carcinoma [6,7]. Typically, neoplasm may represent as a tumefaction discerned upon mammography or as a palpable mammary mass. Upon mammography or ultrasonography, a solid tumefaction is observed. Ultrasonography may delineate a hypoechoic tumefaction along with or devoid of posterior shadowing. Tumefaction may be appropriately discerned with cogent tissue sampling or surgical excision specimens who demonstrate pathognomonic histological features and reactivity upon precise immunohistochemistry. Tumour cells expound IDH2 or TET2 genetic mutations. Exclusion of distant metastasis from neoplasms as carcinoma thyroid is necessitated [6,7]. Neoplasm may be optimally subjected to surgical extermination. However, procedures as sentinel lymph node evaluation, radiation therapy or systemic chemotherapy appear superfluous. Tumefaction is associated with superior prognostic outcomes. Distant metastasis into sites as bone or regional lymph nodes are exceptionally encountered

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