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Editorial

The Husky Eaves-Solid Papillary Carcinoma Breast

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Solid papillary carcinoma emerges as a subtype of ductal carcinoma of breast. Characteristically, neoplasm denominates well circumscribed, solid areas of cellular tumour nodules traversed by attenuated cores of delicate fibro-vascular tissue. Frequently, tumefaction expounds foci of neuroendocrine differentiation. Nuclear atypia appears low grade or intermediate grade.

Additionally designated as neuroendocrine carcinoma breast *in situ*, spindle cell ductal carcinoma *in situ*, neuroendocrine ductal carcinoma *in situ* or endocrine ductal carcinoma *in situ*, neoplasm may emerge as an *in situ* or invasive neoplasm. Emergence of an *in situ* or invasive neoplastic component necessitates definitive categorization.

In situ carcinoma expounds well defined, solid nests of tumour cells simulating cellular distribution pattern concordant with an intra-ductal neoplasm. Peripheral aggregation of myoepithelial cells may or may not be discernible.

Invasive solid papillary carcinoma demonstrates lack of myoepithelial cells along with definitive foci of tumour infiltration. Tumour cells appear immune reactive to oestrogen receptors (ER+) with variable immune reactivity to progesterone receptors (PR) and immune non reactive HER2-.

Solid papillary carcinoma commonly arises within postmenopausal women with median age of disease emergence at 73 years [1,2].

Of obscure aetiology and pathogenesis, tumefaction is commonly confined to centric or sub-areolar region of the breast. However, no zone to breast parenchyma is exempt from tumour emergence [1,2].

Luminal phenotype of the neoplasm demonstrates simple genomes and few copy number aberrations. Tumour cells expound loss of chromosome 16q or gains within chromosome 16p and 1q.

Neuroendocrine differentiation with RET, ASCL1 and DOK7 genes are significantly expressed within solid papillary carcinoma, in contrast to encapsulated papillary carcinoma [1,2].

Clinically, neoplasm may be discerned as an aberrant morphological feature upon mammography. Alternatively, a palpable tumour mass or blood-stained nipple discharge may emerge [2,3].

Cytological smears are hyper-cellular and composed of miniature to enlarged, dis-cohesive clusters of ductal epithelial cells commingled with innumerable singularly disseminated cells.

Tumour cells are imbued with miniature, eccentric, bland, spherical to ovoid nuclei pervaded with fine, granular nuclear chromatin and inconspicuous nucleoli. Constituent cells delineate a decimated nucleocytoplasmic ratio.

Generally, naked nuclei of myoepithelial cells are absent. Aggregates of mucin and capillary-sized vascular articulations may be infrequently observed [2,3].

Grossly, a well circumscribed, firm to soft tumefaction may be observed. Cut surface is tan, grey/white or pink. Solid papillary carcinoma *in situ* with minimal neoplastic confluence may exhibit firm, scattered nodules.

Upon microscopy, tumour nodules are solid, expansible and comprised of amalgamates of proliferating ductal epithelial cells traversed by core of delicate, fibro-vascular tissue [3,4].

Tumour cells appear monomorphic, spherical, ovoid, plasmacytoid or spindle shaped and are permeated with pale, eosinophilic to amphophilic, granular cytoplasm with mildly enlarged, spherical to elliptical nuclei, fine nuclear chromatin and indistinct nucleoli. Few tumour cell nuclei depict open, granular chromatin and miniature nucleoli. Occasionally, conspicuous nuclear grooves may be discerned. Nuclear atypia is minimal to moderate [3,4].

Intracellular and extracellular mucin is commonly encountered with infrequent signet ring cells. Mitotic figures are variable.

Neoplasm delineates a solid pattern of epithelial cell proliferation traversed by attenuated cores of fibro-vascular tissue circumscribed by palisades of tumour cells. Neoplasms constituted of spindle shaped cells expound a 'streaming' tumour pattern. Occasional micro-cystic spaces are discerned. Fibro-vascular cores may exemplify focal hyalinization [3,4].

Solid papillary carcinoma *in situ* enunciates tumour nodules with smooth, rounded contour. Neoplastic dissemination is concordant with pattern configured by subjacent glandular tree. Tumefaction depicts absence, attenuation or retention of myoepithelial cells encompassing perimeter of tumour nodules. Frequently, myoepithelial cells appear attenuated or absent along fibro-vascular cores intrinsic to tumour parenchyma.

Simulating an intra-ductal tumour cell morphology, *in situ* carcinoma (pTis) may be categorized in the absence of myoepithelial cell layer. Meticulous histological assessment of tumour dissemination and implication of surgical margins is necessitated in lesions devoid of an identifiable myoepithelial cell layer [3,4].

Tumour grading is contingent to nuclear morphology of tumour cells. Solid papillary carcinoma accompanied with neoplastic invasion or invasive carcinoma concurrent with solid papillary carcinoma *in situ* may emerge as:

- Invasive solid papillary carcinoma.
- Mucinous carcinoma wherein type B cellular mucinous carcinoma is commonly observed.
- Conventional pattern of invasive carcinoma or invasive ductal carcinoma of no special type (NST).
- An admixture of aforesaid tumour configurations [3,4].

Invasive solid papillary carcinoma is constituted of solid papillary carcinoma demonstrating focal tumour induced cellular destruction within extra-lobular stroma.

Tumour cell nests appear angulated with ragged contours or configure irregularly anastomosing islands of tumour cells. Foci of jigsaw-like appearance are enunciated.

Neoplastic dissemination appears confluent and haphazard, indicative of tumour emergence within subjacent glandular tree. Myoepithelial cells are absent. Additionally, tumour infiltration is associated with desmoplastic stroma and entrapment of normal glandular articulations or adipose tissue. Focal vascular invasion is encountered. Exceptionally, tumour cells nests with invasion expound spherical outline although features as geographic confluence, expansible tumour mass, random dissemination of tumour nodules and concordant reactive stroma appear suggestive of tumour extension beyond the ductal tree [3,4].

Staging of invasive tumours is obtained with Nottingham grade and immune reactivity to oestrogen receptors (ER), progesterone receptors (PR) and HER2. Precise tumour staging is contingent to singular feature as magnitude of invasive component.

Ultrastructural examination expounds diverse granule subtypes as mucinous, small dense core and large serous-like granules impregnated within the tumour cells [3,4].

Nottingham Bloom-Richardson grading system is contingent to:

- Configuration of tubules by the tumour.
- Quantifiable mitotic figures per 10 high power fields as exemplified within actively proliferating, cellular areas.
- Occurrence of nuclear pleomorphism.

Configuration of tumour tubules contributes to pertinent scoring and is classified as:

- 1 point: Tubules representing > 75% of tumefaction.
- 2 points: Tubules articulating 10% to 75% of tumefaction.
- 3 points: Tubules manifesting < 10% of tumefaction.

Mitotic figures are appropriately evaluated upon tumour periphery and are aptly quantified within mitotically active areas. Estimation of mitotic figures confined within 10 high power fields (hpf) constituting a singular, non contiguous neoplastic area is optimal.

Nuclear pleomorphism is classified as:

- 1 point: Neoplasms depicting minimal variation of nuclear magnitude and outline with configuration of miniature, regular, uniform neoplastic cells.
- 2 points: Neoplasms delineating moderate variation in nuclear magnitude and outline.
- 3 points: Neoplasms demonstrating significant variation in nuclear magnitude and outline.

Carcinoma breast is graded and scored as:

- 3 - 5 points: accumulated by well differentiated, grade I carcinoma breast.
- 6 - 7 points: accumulated by moderately differentiated, grade II carcinoma breast.
- 8 - 9 points: accumulated by poorly differentiated, grade III carcinoma breast [3,4].

Solid papillary carcinoma is intensely and diffusely immune reactive to oestrogen receptors (ER) or progesterone receptors (PR). Besides, immune reactivity to chromogranin, synaptophysin or insulinoma-associated protein 1 (INSM1) is observed.

Tumour cells appear immune non reactive to HER2 (ERBB2) and high molecular weight cytokeratin as CK5/6, CK14 or 34β E12. Ki67 proliferative index is minimal or intermediate [5,6].

Solid papillary carcinoma requires segregation from neoplasms as usual ductal hyperplasia, conventional ductal carcinoma *in situ*, encapsulated papillary carcinoma, tall cell carcinoma with reverse polarity and neuroendocrine tumour or neuroendocrine carcinoma [5,6].

Upon mammography, solid papillary carcinoma emerges as a lobulated, circumscribed tumefaction along with or devoid of calcification. Invasive neoplasms depict indistinct tumour perimeter.

Ultrasonography expounds a hypoechoic or heterogeneous, solid tumefaction.

Magnetic resonance imaging (MRI) displays image enhancement of an ovoid or irregular tumour mass with circumscribed border delineating heterogeneous signal intensity. Initial phase with contrast administration depicts rapid image enhancement.

Solid papillary carcinoma breast may be appropriately discerned by fine needle aspiration cytology (FNAC) or cogent histological assessment of tumour tissue sampled by biopsy or surgical excision [5,6].

Solid papillary carcinoma *in situ* emerges as a variant of ductal carcinoma *in situ* and may be managed with surgical extermination of the neoplasm. Modalities of breast conserving surgical excision may be accompanied by cogent radiation therapy.

Precise endocrine therapy may be beneficially employed for decimation of possible emergence of invasive carcinoma.

Invasive solid papillary carcinoma may be subjected to therapeutic strategies applicable to invasive carcinoma breast as surgical eradication of the neoplasm with assessment of sentinel lymph node. Modalities of breast conserving surgical excision may be accompanied by cogent radiation therapy.

Endocrine therapy is characteristically recommended as the neoplasms are intensely immune reactive to oestrogen receptors (ER) [5,6].

Prognostic outcomes are excellent. Systemic therapy may be efficaciously adopted as the specific histological subtype is associated with superior prognostic outcomes.

Regional lymph node metastasis into axillary nodes (6%) and distant metastasis are exceptionally encountered and discerned within neoplasms with overt tumour invasion [5,6].

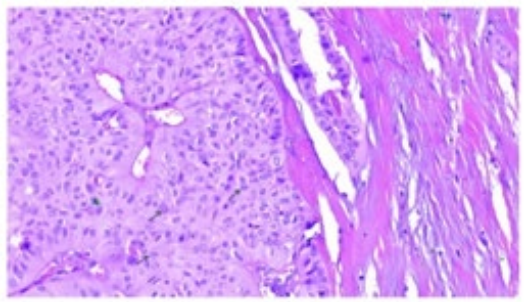

Figure 1: *Solid papillary carcinoma delineating nests of tumour cells imbued with pale, amphophilic cytoplasm, miniature, uniform nuclei and indistinct nucleoli, traversed by delicate fibro-vascular cores. Myoepithelial cell layer is absent [7].*

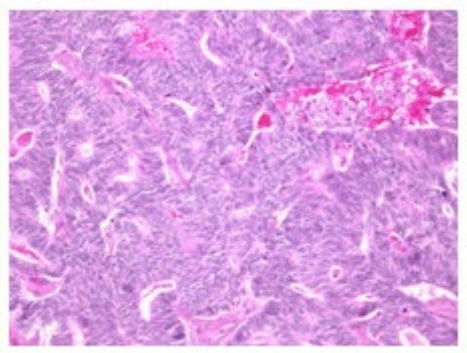

Figure 2: *Solid papillary carcinoma demonstrating nests of tumour cells pervaded with pale, amphophilic cytoplasm, miniature, uniform nuclei and inconspicuous nucleoli, traversed by delicate fibro-vascular cores. Myoepithelial cell layer is absent [8].*

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7. Image 1 Courtesy: Nature.com.

8. Image 2 Courtesy: Webpathology.com.

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