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Short Communication

Baroque and Ornate-Florid Ductal Hyperplasia Breast

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Florid usual epithelial hyperplasia emerges as a benign lesion comprised of intra-ductal proliferation of progenitor epithelial cells. Tumefaction demonstrates a variable proportion of solid or fenestrated pattern of neoplastic growth.

Neoplasm manifests as a component of fibrocystic change within breast tissue. Lesion expounds mild cytological variation, a 'streaming' pattern of tumour evolution with fenestrated spaces and absence of cellular polarity. Neoplastic cells appear immune reactive to high molecular weight cytokeratin.

Additionally designated as epithelial hyperplasia, intra-ductal hyperplasia, hyperplasia of usual type and ductal hyperplasia without atypia or epitheliosis, tumefaction is associated with possible emergence of subsequent carcinoma breast of ~ 1.5 times to 2 times.

Mean age of disease occurrence is 54 years. Neoplasm is encountered within 20% of surgical samples of benign breast lesions [1,2].

Florid usual ductal hyperplasia commonly arises within terminal duct lobular units wherein extra-lobular mammary ducts may be infrequently implicated.

Although specific aetiological features appear absent, tumefaction is posited to arise on account of proliferation of CK5+ progenitor cells which may differentiate along glandular lineage or myoepithelial lineage. Glandular progenitor cells appear preponderant and demonstrate intermediate cellular differentiation [1,2].

Florid usual ductal hyperplasia demonstrates activating mutations within PI3K/AKT/mTOR pathway which contribute to disease pathogenesis.

Clinically, neoplasm is devoid of specific symptoms [1,2].

Cytological smears appear moderately to significantly cellular and are composed of sheets, clusters and cohesive aggregates of bland, uniformly spaced ductal epithelial cells with accompanying myoepithelial cells. Tumour cells are devoid of significant nuclear overlapping or nuclear crowding. Nuclei of ductal epithelial cells are pervaded with finely granular nuclear chromatin and inconspicuous, miniature nucleoli. Tumour cell aggregates lack dis-cohesive morphological features. The cellular component may be commingled with naked nuclei of myoepithelial cell origin [2,3].

Grossly, neoplasm is devoid of specific macroscopic features [2,3].

Upon microscopy, proliferation of luminal epithelial cells and myoepithelial cells is observed. Infrequently, aforesaid cellular lineages are intermingled with a component of apocrine cells. Tumour cells expound minimal variation of cellular and nuclear magnitude or outline. Tumour cell nuclei appear as miniature, ovoid to frequently elongated or asymmetric, tapered or pear shaped and demonstrate miniature nucleoli. Nuclear chromatin is lightly stained, granular and eu-chromatic.

Frequently, tumour cell nuclei exhibit longitudinal nuclear grooves and appear as coffee bean-like with occasional nuclear pseudo-inclusions [3,4].

Several neoplasms delineate cellular maturation and cellular shrinkage with basal nuclei progressing into centric zone of cellular proliferation and emergence of distinctive miniature, pyknotic nuclei [3,4].

Tumour cells are impregnated with eosinophilic, moderate to minimal cytoplasm and enunciate an indistinct cellular perimeter.

The cellular lesion expounds architectural features as cohesive proliferation with haphazard configuration of cells or a 'streaming' pattern of tumour evolution. Neoplasm may exhibit fenestrated, solid or infrequent micro-papillary articulations. Commonly, irregular, slit-like fenestrations are disseminated along tumour periphery.

Tumour cells are aligned parallel to edges of secondary spaces and appear non polarized, in contrast to cellular orientation en-

countered within lesions of atypical ductal hyperplasia or ductal carcinoma in situ [3,4].

Florid usual ductal hyperplasia exemplifies variant neoplastic patterns as

- Micropapillary pattern which typically appears as a focal configuration within lesions delineating conventional pattern of usual ductal hyperplasia breast.
- Mild duct dilation constituted of abridged, stubby papillae of nearly uniform dimensions delineating cytological features of usual ductal hyperplasia. Tumefaction exhibits cellular maturation wherein tips of papillae articulate intense knots of mature epithelial cells devoid of polarization
- Immature cellular pattern is an uncommon variant preponderantly constituted of enlarged, immature, hyperplastic basal epithelial cells which may expand beyond the normal one to two cell layer and appear thickened to several cellular layers. Besides, foci of cellular maturation are observed. Majority of cells frequently configure fibro-epithelial lesions demonstrating significant cellular stroma
- Necrotic pattern wherein lesions of florid usual ductal hyperplasia exceptionally demonstrate centric necrosis. Characteristically, tumefaction concurs within a radial scar, complex sclerosing lesion, nipple adenoma or juvenile papillomatosis. Cells constituting radial scar exhibit mild nuclear enlargement.

Florid usual ductal hyperplasia emerging within radial scar or complex sclerosing lesions may occasionally depict foci of active nuclei with mild nuclear enlargement. Besides, discernible cytological and architectural features of usual ductal hyperplasia remain unaltered [3,4].

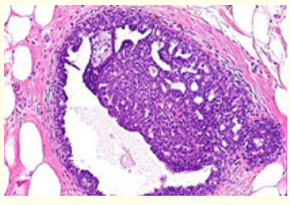

Figure 1: Florid ductal hyperplasia delineating solid sheets and papillae of luminal epithelial cells and myoepithelial cells with parallel cellular orientation. Tumour cells depict moderate cytoplasm and uniform, ovoid nuclei with absent cellular polarity [7].

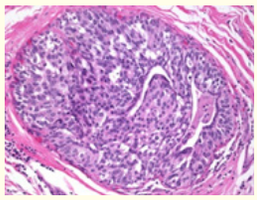

Figure 2: Florid ductal hyperplasia delineating solid sheets and papillae of parallel luminal epithelial cells and myoepithelial cells delineating moderate cytoplasm and uniform, ovoid nuclei with absent cellular polarity [8].

Precursor lesion
Atypical ductal hyperplasia
Flat epithelial atypia
Ductal carcinoma *in situ*
Non invasive lobular neoplasia
Atypical lobular hyperplasia
Lobular carcinoma *in situ*
Classic lobular carcinoma *in situ*
Pleomorphic lobular carcinoma *in situ*
Florid lobular carcinoma *in situ*

Table 1: Histopathological classification of precursor lesions of breast [1].

Florid usual ductal hyperplasia breast appears immune reactive to high molecular weight cytokeratin as CK5, CK14, 34 β E12, oestrogen receptor, E-cadherin and low molecular weight cytokeratin as CK7, CK8 or CK18 [5,6].

Florid usual ductal hyperplasia breast requires segregation from neoplasms as atypical ductal hyperplasia/ low grade ductal carcinoma in situ, micro-papillary ductal carcinoma in situ, intermediate grade ductal carcinoma in situ or solid papillary carcinoma [5,6].

Florid usual ductal hyperplasia may be appropriately discerned upon histological assessment of surgical tissue samples obtained with surgical excision or biopsy procedures [5,6].

Upon mammography, lesion appears devoid of specific features. Occasionally, neoplasm may demonstrate micro-calcifications.

Florid usual ductal hyperplasia may concur with a pre-existing lesion as radial scar or papilloma which may be discernible upon imaging.

Magnetic resonance imaging (MRI) expounds image enhancement of the neoplasm [5,6].

Generally, therapeutic intervention of florid usual ductal hyperplasia appears superfluous. Florid usual ductal hyperplasia appears to be associated with 1.5 times to 2 times enhanced possible emergence of subsequent carcinoma breast [5,6].

Tumour is accompanied by augmented possible occurrence of malignant metamorphosis within subjects demonstrating a family history of carcinoma breast.

Lesion manifests as an indicator of generalized carcinoma breast, in contrast to emerging as a direct precursor lesion [5,6].

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7. Image 1 Courtesy: Libre pathology.

8. Image 2 Courtesy: Webpathology.com.