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Editorial

Infinitesimal and Jammed - Microglandular Adenosis - Breast

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Microglandular adenosis emerges as an infrequently discerned, benign tumefaction of breast parenchyma. Infrequently denominated as microglandular hyperplasia or microglandular adenoma, neoplasm configures as a potential precursor of triple negative invasive carcinoma breast. Lesion is constituted of haphazard, irregular dissemination of miniature, uniform, spherical, open glandular articulations impregnated with eosinophilic secretions.

Neoplasm appears reminiscent of invasive carcinoma breast associated with tumour infiltration into surrounding stroma and delineates dispersed singular layer of epithelial cells in the absence of myoepithelial cells. The disorderly proliferation of miniature, open glandular structures constituted of bland epithelial cells is encompassed within a distinct basement membrane devoid of myoepithelial cell layer.

Tumefaction may invade circumscribing adipose tissue and fibrous tissue stroma in the absence of a distinct stromal reaction. Tumour cells appear immune reactive to S100 protein and immune non reactive to oestrogen receptors (ER) and progesterone receptors (PR).

The exceptionally encountered microglandular adenosis is devoid of specific association with cogent epidemiological factors. Generally, neoplasm emerges within female subjects and no age of disease emergence is exempt. Commonly confined to mammary tissue, a specific localization with diverse breast quadrants is absent [1,2].

Of obscure aetiology, neoplasm exhibits gain of somatic mutations within genes as TP53. Besides, molecules associated with PIK3CA pathway and tyrosine kinase receptor signalling elated genes may appear within microglandular adenosis and microglandular adenosis associated triple negative carcinoma breast, thereby indicating a contributory effect as a non-obligate precursor of carcinoma breast [1,2].

Few instances of microglandular adenosis appear clone specific. Array comparative genomic hybridization (aCGH) and next generation sequencing (NGS) demonstrates molecular progression of tumour cells into concordant invasive carcinoma breast. Thus, it is posited that microglandular adenosis contributes as a non-obligate precursor of triple negative invasive carcinoma breast. Genomic mutations occurring within PI3K pathway with the involvement of PIK3CA, PTEN, INPP4B and BRCA1 genes may ensue in lesions of microglandular adenosis and atypical microglandular adenosis [1,2].

Microglandular adenosis may represent as a thickened area or palpable tumour mass. Besides, tumefaction may be discovered incidentally upon clinical examination or employment of various imaging modalities [2,3].

Cytological examination displays sparsely cellular smears composed of monotonous population of cells of intermediate magnitude. Tumour cells are permeated with clear to vacuolated cytoplasm, spherical, uniform nuclei and miniature nucleoli. Singularly dispersed or clusters of clear cells abutting spindle shaped fibroblasts may be discerned [2,3].

Grossly, tumefaction expounds inadequately defined tissue perimeter and melds into circumscribing breast tissue. Tumour mass or nodule frequently coexists with foci of invasive ductal carcinoma [2,3].

Upon microscopy, tumefaction exhibits haphazard amalgamation of miniature, uniform, spherical, open glands impregnated with eosinophilic secretions. The glandular structures are irregularly disseminated and appear to infiltrate circumscribing fibrous tissue or adipose tissue.

Neoplastic glands are layered by singular layer of cuboidal or flattened epithelial cells pervaded with vacuolated or granular cy-

toplasm, bland nuclei and absent nucleoli. Cytoplasmic apocrine snouts or a distinct myoepithelial layer appear absent although a thickened basement membrane may be discerned [3,4].

Atypical microglandular adenosis demonstrates enhanced glandular complexity with cribriform architecture and is constituted of glandular articulations coated with multi-layered epithelium. Besides, morphological features as fused glandular units and intra-luminal bridging may appear. Tumour cell nuclei appear hyperchromatic and are pervaded with prominent nucleoli. Cytological atypia is mild and mitotic figures are occasional [3,4].

Ultrastructural examination demonstrates thickened basement membrane circumscribing epithelial cell clusters. Tumour cells are permeated with electron lucent cytoplasm and sparse organelles [3,4].

Microglandular adenosis appears immune reactive to CAM5.2, AE1, S100 protein, p63, CK8/CK18 and epithelial growth factor receptor (EGFR). Variable immune reactivity to smooth muscle actin (SMA),vimentin, type IV collagen and laminin is observed. Intraglandular secretions may be highlighted by Periodic acid Schiff's (PAS) stain with diastase resistance [5,6].

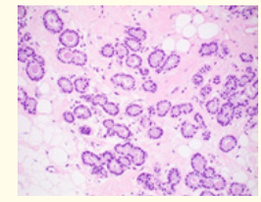

Figure 1: Microglandular adenosis delineating irregular distributed small, uniform glandular structures lined by cuboidal epithelial cells imbued with moderate cytoplasm, bland nuclei and indistinct nucleoli. Infiltration into surrounding fibrous tissue and adipose tissue is observed [8].

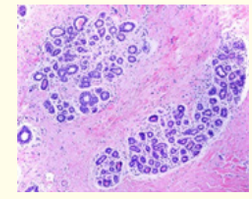

Figure 2: Microglandular adenosis exhibiting irregularly disseminated small, uniform glandular structures lined by cuboidal epithelial cells impregnated with moderate cytoplasm, bland nuclei and indistinct nucleoli. Infiltration into surrounding fibrous tissue and adipose tissue may occur [9].

	MGA	Atypical MGA	MGA-associated invasive carcinoma	Well differentiated invasive ductal carcinoma
Collagen IV	+	+	-	-
S100 protein	+	+	+/-	-
ER/PR	-	-	-	+(diffuse)
Myoepithelial markers	-	-	-	-
EMA	-	-	-	+
Desmoplasia	Absent	Absent	Present	Present
Cytological atypia	Minimal	Minimal to mild	Moderate to severe	Minimal to mild
Growth pattern	Infiltrative	Infiltrative	Infiltrative	Infiltrative
Gland shape	Round with secretions	Variable(fused, round, angulated, complex)	Irregular	Variable(round, angulated, complex)
Apical cytoplasmic snouts	Absent	Absent	Absent	Absent

Table 1: Differential Diagnosis of Microglandular Adenosis [3].

ER: Oestrogen receptor, PR: Progesterone receptor, MCA: Microglandular adenosis, EMA: Epithelial membrane antigen.

Tumour cells are triple negative and appear immune non reactive to oestrogen receptors (ER), progesterone receptors (PR) and HER2. Besides, tumour cells appear immune non reactive to myoepithelial markers as actin, calponin, p63, smooth muscle myosin heavy chain (SMMHC), epithelial membrane antigen (EMA), gross cystic disease fluid protein-15 (GCDFP-15) or p53. Ki67 proliferative index is minimal [5,6].

Microglandular adenosis breast requires segregation from neoplasms as well differentiated invasive ductal carcinoma, tubular carcinoma, microglandular adenosis associated invasive carcinoma, apocrine adenosis, sclerosing adenosis or in situ carcinoma [5,6].

Mammographic assay depicts density of breast tissue or focal calcification.

Microglandular adenosis may be appropriately ascertained with histological assessment of implicated breast tissue with cogent immunohistochemistry [6,7].

Microglandular adenosis may be suitably treated by comprehensive surgical excision of the neoplasm. Exclusion of tumour infiltration is necessitated. Additionally, meticulous monitoring is mandated, especially within lesions of atypical microglandular hyperplasia.

Incomplete surgical extermination of the neoplasm is associated with tumour reoccurrence [6,7].

Surgical eradication with broad perimeter of normal tissue and tumour free surgical margins is optimally adopted for treating lesions of atypical microglandular adenosis with curative intent.

The essentially benign microglandular adenosis may be associated with invasive carcinoma breast in roughly one fourth (~27%) of instances. Malignant transformation into invasive carcinoma breast may represent with immunohistochemistry identical to lesions of microglandular adenosis [6,7].

Microglandular adenosis appears concordant with variants of invasive carcinoma breast as adenoid cystic carcinoma, secretory carcinoma, squamous carcinoma, matrix producing carcinoma breast and triple negative, basal-like carcinoma breast.

Atypical microglandular adenosis is associated with enhanced possible progression into invasive carcinoma breast [6,7].

Bibliography

1. Jaramillo C., *et al*. "Luminal-Type Invasive Carcinoma in Association With Microglandular Adenosis/Atypical Microglandular Adenosis: A Case Report and Molecular Comparison". *Cureus* 15.4 (2023): e-37198.

2. Thu Huong N., *et al*. "Ductal carcinoma in situ arises from microglandular adenosis and atypical microglandular adenosis in a young woman". *Journal of Clinical Imaging Science* 13 (2023): 15.

3. Almahari SAI., *et al*. "Deceptive Triple-Negative Breast Cancer of Intermediate Grade: A Case of Rare Microglandular Adenosis-Associated Carcinoma". *Cureus* 15.5 (2023): e39531.

4. Hammad MF and Alkhayyat R. "Clinicopathological and radiological features of triple negative breast cancer in Bahrain". *Journal of Cancer Diagnosis* 5 (2021): 3.

5. Kravtsov O and Jorns JM. "Microglandular adenosis and associated invasive carcinoma". *Archives of Pathology and Laboratory Medicine* 144 (2020): 42-46.

6. Schwartz CJ., *et al*. "Microglandular adenosis is an advanced precursor breast lesion with evidence of molecular progression to matrix-producing metaplastic carcinoma". *Human Pathology* 85 (2019): 65-71.

7. Sugino T., *et al*. "A case of extensively spreading acinic cell carcinoma of the breast with microglandular features". *Pathology International* 73.1 (2023): 45-51.

8. Image 1 Courtesy: Pathology outlines.

9. Image 2 Courtesy: Science photo library.