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Editorial

The Omnipotent Halcyon-Glycogen Rich Invasive Carcinoma Breast

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Glycogen rich invasive carcinoma breast is configured of an infiltrative carcinoma preponderantly (>90%) comprised of neoplastic cells imbued with abundant, clear, glycogen-rich cytoplasm. Initially scripted in 1981, the exceptionally encountered neoplasm configures up to 3% of invasive breast carcinoma variants. Additionally, glycogen rich carcinoma may emerge as a variant of apocrine carcinoma.

Generally a disease of elderly, postmenopausal population, tumefaction commonly emerges in women > 65 years.

Genetic, hormonal or environmental factors contribute to emergence of invasive ductal carcinoma. Hormonal factors engendering ductal carcinoma are early menarche', delayed menopause, nulliparous state, primigravida > 30 years, obesity with postmenopausal state, occurrence of oestrogen- producing ovarian neoplasms or administration of oestrogens or combined hormone- replacement therapy with progestogens [1,2].

Additionally, conditions such as proliferative breast disease, ductal carcinoma in situ, concurrent multiple, non-proliferative or proliferative benign breast lesions, endometrial carcinoma, carcinoma of contralateral breast, previous breast tissue sampling, radiation exposure in young women < 30 years as encountered with supra-diaphragmatic radiotherapy adopted for Hodgkin's lymphoma, inherent mammographic density with carcinomas occurring at ≤ 50 years or birth weight > 3 kilograms can contribute to emergence of invasive ductal carcinoma breast [1,2].

Factors decimating possible occurrence of carcinoma breast are enhanced physical activity, consumption of dietary soy or carotenoids, primigravid state <18 years, oophorectomy <35 years, irradiated ovaries or obese individuals <40 years with an-ovulatory menstrual cycles and decimated premenstrual progesterone levels [1,2].

Cytological smears appear hyper-cellular wherein tumour cells appear to configure loosely cohesive cellular clusters or syncytial groups along with disseminated singular cells. Majority of neoplastic cells are impregnated with abundant, eosinophilic, finely granular, foamy or clear cytoplasm with well defined cytoplasmic outline and centric, spherical to ovoid nuclei with prominent nucleoli. Nuclear pleomorphism is moderate to marked. Tumour cells may be appropriately subjected to Periodic acid Schiff's (PAS) stain [2,3].

Grossly, a firm to hard, poorly circumscribed tumefaction of cartilaginous consistency appears to recede from circumscribing mammary tissue. Scraping of tumour creates a grating sensation. Cut surface may or may not exhibit chalky- white, linear streaks with focal elastosis extending into enveloping stroma with configuration of "crab like" foci of calcification [3,4].

Upon microscopy, tumefaction expounds a solid or solid-papillary configuration. Tumour cells appear enlarged, clear and demonstrate a distinct cellular perimeter. Majority (>90%) of cells are permeated with glycogen. Tumour cells are pervaded with clear to granular cytoplasm with minimal intracellular mucin. Cytoplasmic vacuoles are absent. Frequently, tumefaction expounds a variable intra-ductal component. Tumour cells may commonly depict apocrine features and are pervaded with abundant, granular, eosinophilic cytoplasm with apocrine snouts [3,4].

Ultrastructural examination exemplifies non membrane bound glycogen molecules and vacant glycogen lakes. Besides, dense junctions between tumour cells may be discerned. Desmosomes are immature and occasional, short microvilli may be encountered [3,4].

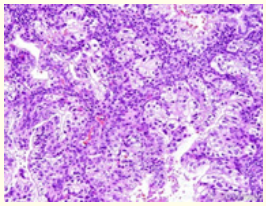

Figure 1: Glycogen rich invasive carcinoma breast exemplifying intra-ductal aggregates of tumour cells pervaded with abundant, clear, vacuolated cytoplasm and centric, hyperchromatic nuclei. Infiltration into surrounding breast parenchyma is observed [9].

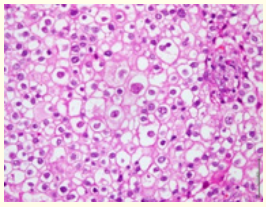

Figure 2: Glycogen rich invasive carcinoma delineating aggregates of tumour cells pervaded with abundant, vacuolated cytoplasm with centric, hyperchromatic nuclei. Tumour infiltration into surrounding stroma is observed [10].

Staging of carcinoma breast as per American Joint Committee on Cancer (AJCC) 8th edition [3,4]

Primary tumour

- TX: Primary tumour cannot be assessed
- T0: No evidence of primary tumour
- Tis: Tumour appearing as ductal carcinoma in situ, Paget's disease, encapsulated papillary carcinoma and solid papillary carcinoma
  - Tis (DCIS) appearing as ductal carcinoma in situ devoid of invasive carcinoma
  - Tis (Paget) appearing as Paget's disease devoid of invasive carcinoma
- T1mi: Tumour ≤ 1 millimetre magnitude
- T1a: Tumour > 1 millimetre and ≤ 5 millimetre magnitude
- T1b: Tumour > 5 millimetre and ≤ 10 millimetre magnitude
- T1c: Tumour > 10 millimetre and ≤ 20 millimetre magnitude
- T2: Tumour > 20 millimetre and ≤ 50 millimetre magnitude
- T3: Tumour > 50 millimetre magnitude
- T4a: Tumour extension into chest wall and devoid of infiltration into pectoralis muscle
- T4b: Tumour associated with oedema as peau d'orange, cutaneous ulceration and ipsilateral satellite cutaneous nodules
- T4c: Tumour demonstrating features of T4a and T4b
- T4d: Tumour demonstrating inflammatory carcinoma which implicates > 1/3 of cutaneous surface of breast and is discerned upon clinical examination

Regional lymph nodes

- NX: Regional lymph nodes cannot be assessed
- N0: Regional lymph node metastasis absent
- N0 (i-): Regional lymph node metastasis absent upon histological assessment or immunohistochemistry
- N0 (i+): Regional lymph nodes depicting isolated tumour cells or a cluster of tumour cells ≤ 0.2 millimetre diameter or < 200 cells
- N0 (mol+): Regional lymph nodes delineating tumour cells upon reverse transcriptase polymerase chain reaction (RT-PCR) and non discernible upon light microscopy
- N1mi: Regional lymph nodes with micro-metastasis or tumour deposit > 0.2 millimetre and ≤ 2.0 millimetre or ≤ 0.2 millimetre and > 200 cells
- N1a: Regional lymph node metastasis within one to three 3 axillary lymph nodes with minimally a singular tumour deposit > 2.0 millimetre diameter
- N1b: Regional lymph node metastasis into internal mammary sentinel lymph node with tumour deposit > 2.0 millimetre diameter
- N1c: Is constituted of combined N1a and N1b
- N2a: Regional lymph node metastasis into 4 to 9 axillary lymph nodes with minimally a singular tumour deposit > 2.0 millimetre diameter
- N2b: Regional lymph node metastasis within clinically palpable internal mammary lymph nodes and axillary lymph nodes devoid of tumour deposits
- N3a: Regional lymph node metastasis into ≥ 10 axillary lymph nodes with minimally a singular tumour deposit > 2.0 millimetre magnitude or metastasis into infra-clavicular lymph nodes
- N3b: Regional lymph node metastasis into internal mammary lymph nodes as discerned upon imaging and tumour stage appearing as N1a or N1b
- N3c: Regional lymph node metastasis into ipsilateral supraclavicular lymph nodes

Distant metastasis (M)

- M0: Distant metastasis absent
- M1: Distant metastasis present with magnitude of histological tumour deposits > 0.2 millimetres
- y: Adoption of preoperative radiotherapy or chemotherapy
- r: Recurrent tumour stage

Glycogen rich invasive carcinoma breast can be appropriately highlighted with Periodic acid Schiff's (PAS) stain with sensitivity to diastase.

Tumour cells appear immune non reactive to CK7 and various lipid discerning stains [5,6].

Glycogen rich invasive carcinoma breast requires segregation from neoplasms as apocrine carcinoma, clear cell sugar tumour, lipid rich carcinoma, secretory carcinoma or various myoepithelial lesions [5,6].

Glycogen rich invasive carcinoma breast may be suitably subjected to surgical extermination of the lesion. Besides, breast conserving surgical modalities as lumpectomy or mastectomy may be adopted in concurrence with or in the absence of axillary lymph node dissection. Following lumpectomy, localized radiotherapy of the breast may be optimally employed. Subsequent to surgical procedures as mastectomy, radiotherapy to the chest wall may be adopted in order to decimate possible localized tumour reoccurrence.

Additionally, neoadjuvant or adjuvant systemic chemotherapy may be optimally employed.

Targeted therapies as anti-endocrine therapy or anti-HER2 directed therapy appears beneficial [6,7].

Appropriate selection of various therapeutic strategies as diverse surgical procedures, commencement of radiotherapy or cogent systemic therapies is contingent to multiple contributory factors as anatomic localization of the neoplasm, age of implicated subject, tumour stage, tumour magnitude, histological grade, status of immune biomarkers, outcomes of genomic risk assessment as Oncotype DX Recurrence Score ®, proportionate occurrence of heritable carcinoma breast as status of BRCA genes, associated comorbid conditions, preceding exposure to chemotherapy or radiation therapy along with preferential adoption of surgical manoeuvers and cosmetic outcomes [7,8].

Akin to invasive ductal carcinoma of no special type (NST), prognostic outcomes are contingent to contributory factors as age of implicated individual or tumour stage as assessed by tumour magnitude and status of regional lymph nodes or histological grade. Tumour infiltration into regional lymph nodes and vascular structures is associated with enhanced possible emergence of localized tumour reoccurrence and neoplastic reappearance upon distant sites [7,8].

Expression of hormone receptors is associated with superior short term survival. Expression of HER2 is accompanied by ameliorated survival, especially in instances subjected to anti-HER2 therapy. Genetic expression signature is associated with elevated overall survival in luminal A subtype and decimated survival in basal-like subtype of glycogen rich invasive carcinoma breast [7,8].

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9. Image 1 Courtesy: Dovemed.com.

10. Image 2 Courtesy: Europe PMC.