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Breast Edema Score on Magnetic Resonance Imaging and Its Association with Molecular Subtypes of Breast Cancer

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Abstract

Breast edema is an important magnetic resonance imaging (MRI) finding in breast cancer, reflecting alterations in lymphatic drainage, microvascular permeability, and tumor-associated inflammatory changes. This review summarizes the mechanisms and MRI appearance of breast edema, the breast edema score (BES), and its relationship with the molecular subtypes of breast cancer. On T2-weighted imaging, edema may appear in peritumoral, prepectoral, subcutaneous, or diffuse patterns, and increasing edema extent may indicate more aggressive tumor biology. Available evidence suggests significant associations between BES and molecular phenotype, with higher edema scores reported more frequently in HER2-positive and triple-negative tumors, whereas lower scores are more often associated with luminal subtypes. Breast edema and peritumoral edema have also been linked to lymphovascular involvement, axillary nodal disease, inflammatory changes, and adverse prognostic features. Multiparametric breast MRI, combining T2-weighted imaging with dynamic contrast-enhanced and diffusion-weighted sequences, provides complementary structural and functional information. Overall, BES represents a promising non-invasive imaging biomarker that may contribute to molecular subtype prediction, risk stratification, and preoperative assessment, although standardized scoring criteria and prospective validation remain necessary before routine clinical implementation.

Keywords: Breast cancer; Breast edema score; Magnetic resonance imaging; Molecular subtypes; T2-weighted imaging.

Introduction

Breast edema in breast cancer: definition and radiologic perspective

Breast edema refers to abnormal accumulation of interstitial fluid in the breast, typically due to impaired lymphatic/venous drainage or tumor-related inflammation. (Verbelen et al., 2019).

Definitions and Clinical Criteria

Common clinical features include breast swelling, heaviness, peau d'orange, skin thickening, redness, pitting, enlarged pores, hardness and pain Breast edema is often considered a form of breast lymphedema after breast-conserving therapy. Lack of a uniform definition has produced incidence ranges from 0–90% after breast-conserving surgery and radiotherapy . (Verbelen et al., 2019).

Mechanism of breast edema

1. Tumor obstruction of lymphatic drainage

Malignant cells infiltrate and compress intra-mammary lymphatic vessels and regional nodes, reducing lymph outflow from the breast parenchyma and skin. This causes accumulation of protein-rich interstitial fluid and edema confined to the breast . (Kataru, 2019).

In breast cancer, the lymphatic system is the main route of regional metastases. The collection of peritumoral fluid is strongly related to the malfunctioning of lymphatic vessels, which are essential for controlling tumour development and metastasis (Kataru, 2019).

2. Tumor-driven microvascular changes

Tumors secrete angiogenic and permeability factors (e.g., VEGF-family), increasing capillary leak and local fluid load that must be cleared by lymphatics; when lymphatics are blocked, this excess fluid amplifies breast edema . (De Paz Linares et al., 2021).

Inflammatory cytokines from tumor and stromal cells further increase endothelial permeability and tissue water. (De Paz Linares et al., 2021).

3. Inflammatory and fibrotic remodeling of breast tissue

Persistent stasis of protein-rich lymph provokes chronic inflammation and fibroblast activation, leading to dermal and subcutaneous thickening and sometimes peau-d'orange, even without prior surgery or radiotherapy . (Jahan et al., 2025).

Radiologic Definition and Patterns

On MRI, edema is defined as high T2-weighted signal intensity (water-like) in or around the breast parenchyma (Santucci et al., 2021). Focal edema linked to malignancy is usually classified into patterns: (Santucci et al., 2021).

- Peritumoral edema (around the mass) (Harada et al., 2021).
- Prepectoral edema (retro-mammary) (Harada et al., 2021).
- Subcutaneous edema (Harada et al., 2021).
- Diffuse edema, often seen in inflammatory breast cancer or severe lymphatic obstruction. (Cardoso, F.2019).

These patterns are often combined into a breast edema score (BES 1–4/5), from no edema to subcutaneous/diffuse edema highly suggestive of occult inflammatory breast cancer (Harada et al., 2021).

Relation between breast cancer aggressiveness and vascular and lymphatic infiltration and inflammatory response:

Breast cancer aggressiveness is closely linked to vascular and lymphatic infiltration as well as the inflammatory response within the tumor microenvironment. Increased angiogenesis and lymph angiogenesis facilitate tumor growth and metastasis, with inflammation playing a key role in promoting these processes through pathways such as COX-2/EP4 signaling, which enhances cancer cell migration, invasion, and stem-like properties (De Paz Linares et al., 2021). Tumors with high microvessel density and vascular invasion tend to be more aggressive and are associated with poorer survival outcomes . (De Paz Linares et al., 2021).

Inflammatory breast cancer (IBC), a highly aggressive subtype, shows extensive dermal lymphatic invasion and macrophage infiltration driven by chemokines like CCL2, which promote tumor proliferation and metastasis .

The immune infiltrate composition also influences prognosis; for example, higher lymphocytic infiltration correlates with improved survival, while macrophage-rich environments often support tumor progression 4. Additionally, aggressive subtypes such as triple-negative breast cancer exhibit increased mast cell infiltration, inflammation, epithelial-mesenchymal transition (EMT), and angiogenesis, all contributing to their invasive phenotype . Overall, the interplay between vascular/lymphatic invasion and inflammatory responses shapes breast cancer aggressiveness and metastatic potential through complex cellular and molecular mechanisms

Relation between breast edema and molecular classification :

Tumour-related breast edema MRI characteristics may be useful in distinguishing molecular subtypes of breast cancer and could be used as a promising feature to improve the predictive performance of pathological

axillary lymph nodes in patients with breast cancer, contributing to preoperative treatment planning and prognostic outcome. (Abdelbary et al., 2024).

There was a highly significant difference between BES in terms of molecular subtypes of breast cancer ($p < 0.0001$), BES 2; was seen more frequently in the HER2 + subtype in 25 masses (83.3% of HER2 + masses) , while BES 4; was seen more in the TN subtype in 21 masses (58.3% of TN masses) , followed by the luminal B subtype (40.5% of LB masses), which was found to be the most predominant type of edema among luminal B masses. BES 1 is not found at all in both luminal B and TN subtypes, indicating that these two subtypes must have breast edema regardless their score. (Abdelbary et al., 2024).

Another study reveal that Invasive ductal cancers were the most common cancers in the study as well as in both the groups. Invasive lobular cancer was seen in five cases and rest were other subtypes that included mucinous, tubular, metaplastic, papillary and medullary varieties). No statistically significant correlation was found between the histological tumor type and the PE in two groups. Luminal B were the most common molecular subtype as well as in both the groups separately followed by triple negatives, Her2 enriched and luminal A cancers. Majority of the triple negative tumors were associated with PE, while the majority of the luminal A subtypes did not show edema. There was statistically significant correlation present ($p<.05$) between various molecular subtype and two groups . (Kushvaha & Renganathan, 2022).

Molecular subtype showed a significant association with BES categories ($P < 0.001$). Luminal A and luminal B (HER2-) were significantly more prevalent in lesions with BES-1. BES-2 was more frequent in luminal B (HER2-) and triple-negative breast cancer (TNBC). Lesions with BES-3 were mostly characterized by luminal B (HER2+) and TNBC, whereas lesions with BES-4 were mostly distinguished by the absence of luminal A and the presence of all other subtypes.

Molecular Classification

We now know that breast cancer represents a biologically and phenotypically heterogeneous collection of diseases with different clinical and treatment response behaviours (Feng et al., 2018).

In this era of modern medicine, only the morphological classification (nuclear grade, tubular grade, mitotic index, histological grade, and architectural characteristics) and the clinical pathological parameters (tumour size, lymph node involvement, metastasis), are insufficient to predict the real behaviour of breast tumor pathophysiology (Nascimento and Otoni, 2020).

Based on comprehensive gene expression profile studies, four clinically relevant molecular subtypes were revealed: Luminal A, Luminal B, enriched HER2 (HER2+), and Triple Negative (TN) (Harbeck et al., 2019).

The groups of genes responsible mainly for the segregation of the molecular subtypes of breast cancer are genes related to the expression of estrogen receptors (ER), progesterone receptors (PR), HER2 (Human epidermal growth factor receptor 2), and cell proliferation regulator (Ki- 67) (Lukong, 2017).

The Immunohistochemical (IHC) panel with these four biomarkers (ER/PR/HER2/Ki-67) has been considered efficient and significant in the stratification of these molecular entities (Tsang et al., 2020).

They are prognostic markers and important predictive factors for hormonal and anti-HER2-targeted therapy. ER and PR are nuclear sex steroid receptors that stimulate the growth of normal and neoplastic breast epithelium. They are expressed in ~75% of all breast cancers. When present, they are bona fide indicators for responsiveness to hormonal therapy. ER and PR are assessed immunohistochemically with a 1% nuclear expression cut off. ER/ PR-positive cancers are usually low grade and less aggressive (Tsang et al., 2020).

The majority of ER-positive cancers are also PR+. However, a small percentage of breast cancers show single hormone receptor positivity. These tumours seem to be more aggressive and less responsive to hormonal therapy compared with ER/ PR-positive cancers. Approximately 15% of breast cancers overexpress HER2 with amplification of the corresponding gene at 17q12. HER2 status is tested using a combination of IHC and DNA in situ hybridization techniques (Wolff et al., 2018).

Currently, HER2-positive tumours are defined by >10% of cells with strong circumferential staining or HER2:CEP17 ratio ≥2. HER2 overexpression is associated with aggressive clinical course and poor prognosis but is also predictive of response to anti-HER2 targeted treatments (Tsang et al., 2020).

The remaining 10% to 15% of breast cancers that express none of these 3 markers are termed triple negative breast cancers (TNBC). TNBC are in general high grade and associated with a poor prognosis. Patients with TNBC do not benefit from the current targeted therapies (Yam et al., 2017).

Luminal A

This molecular subtype is the most common and comprises approximately half of newly diagnosed breast cancer cases. According to the last update of St. Gallen guidelines in 2013, the immunohistochemical profile of this subtype was defined as: ER+ (≥ 1%), high expression of PR (≥ 20%), HER2- (≤ 10%), and low levels of Ki-67 (< 14%) (Tsang et al., 2020).

This subtype has been associated with a highly favorable prognosis, with a more indolent clinical course, and generally shows less lymph node involvement. Nonetheless, due to the positive status of hormone receptors, patients benefit from endocrine therapies, either with selective estrogen receptor modulators (tamoxifen) or with aromatase inhibitors (anastrozole) (Table 2) (Tsang et al., 2020).

Luminal B

Responsible for approximately 20% to 30% of invasive breast cancer cases. This subtype can be categorized immunophenotypically into Luminal B (HER-): ER+ (≥ 1%), PR- or < 20%, HER2- (≤ 10%) and high levels of Ki-67 (≥ 20%); or Luminal B (HER2+): ER+ (≥ 1%), HER2+ (> 10%) and any level of PR and Ki-67. The expression of low molecular weight cytokeratin's from luminal epithelial cells is a rule (Provenzano et al., 2018).

This molecular entity generally presents a moderate histological grade, including most of the IDC-NST and associated with an intermediate prognosis, with greater likelihood of locoregional recurrence when compared to Luminal A (Hashmi et al., 2018).

Luminal B subtype is understood as the most aggressive form of hormone-dependent breast cancer cases, requiring additional treatments to hormonal therapy, such as chemotherapy (when HER2 +/-) or targeted target therapy (when HER2 +) (Table 2) (Fragomeni et al., 2018).

Human Epidermal growth factor (HER2+)

It represents 15% to 20% of newly diagnosed breast cancer cases. This subtype is characterized by a high expression of HER2 (> 10%), negativity for ER (< 1%) and PR (< 20%), and high expression of Ki-67 (> 20%). In addition to the immunophenotypic characterization routinely used to assess the status of HER2 in breast cancer, the FISH (Fluorescence in situ hybridization) technique has also been employed to assess gene amplification (Desai et al., 2018).

It must be clarified that the HER2-enriched subtype is not synonymous with clinically HER2-positive breast cancer. While about 50% of clinical HER2-positive breast cancers are HER2-enriched, the remaining 50% can include any molecular subtype but are mostly HER2- positive luminal subtypes (Marrazzo et al., 2020).

These tumours are generally more prevalent in patients with BRCA1 mutations and young women, with a higher histological grade, risk of loco-regional recurrence, contralateral disease and systemic relapse (Kumar and Aggarwal, 2016).

Many gene expression profile studies have been carried out to better understand the heterogeneity of this particularly aggressive form of breast cancer (Nascimento and Otoni, 2020).

Despite its simple definition, this subtype has been a challenge for the clinic, due to its morphological, molecular and clinical heterogeneity and the lack of targeted therapies (Russnes et al., 2017). Non-surgical

treatment of the TN subtype has been limited to platinum-based chemotherapy and PARP (Poly ADP-ribose polymerase) inhibitors for patients with BRCA1 and 2 mutations (Harbeck et al., 2019).

MRI Technique and Imaging Protocol of the Breast

Pre-contrast sequences:

1. T1-weighted sequences are useful for detecting the presence of a fatty component within a lesion, which is also a major aspect predicting its benign nature. T1 sequences are therefore performed without fat saturation. They also allow met al., markers to be detected which may have been positioned at the end of biopsy (Thomassin-Naggara et al., 2012).

2. T2-weighted Imaging: Most breast MRI protocols include a T2-weighted unenhanced sequence, with or without fat suppression. T2-weighted images with fat suppression allow easier visibility of fluid intensity but at the expense of spatial resolution. T2-weighted images without fat suppression provide better depiction of the normal tissue architecture and lesion morphology. In T2-weighted fast spin echo images, water-containing lesions or edematous lesions have an intense signal, and therefore small cysts and myxoid fibroadenomas are very well identified (Fig.9). While most cases of cancer do not yield a high signal on T2- weighted images; so T2WI can be useful in the differentiation between benign and malignant lesions (Westra et al., 2014).

3. DWI Sequences: DWI quantifies the random movement of water molecules in tissue, which is influenced by tissue microstructure and cell density. Cancers display higher signal intensity at DWI because of increased cell density, which leads to decrease water diffusion. To obtain adequate DWI acquisitions, the selection of appropriate b values, adequate fat suppression, minimization of artifacts, and sufficient SNR are crucial (Partridge et al., 2013).

The apparent diffusion coefficient (ADC) is a quantitative measure of diffusivity derived from DWI. Values are usually expressed in 10−3 mm2 /sec. The mean ADCs are generally low (range, 0.8–1.3 × 10−3 mm2 /sec) in malignant lesions compared with those in benign lesions (range, 1.2–2.0 × 10−3 mm2 /sec) due to the hindered diffusion in malignant lesions. Consequently, cancers have a low signal intensity on the derived ADC maps (Fig. 10) (Mann et al., 2019).

The contrast of tumor to normal parenchyma increases with a b value from 0 to 1500 sec/mm2 and decreases with a b value greater than 1500 sec/mm2, but the higher b values lead to decreased SNR (Fig. 11). In addition, DWI performed by using a b value of less than 1000 sec/mm2 showed the greatest accuracy in distinguishing benign and malignant lesions. Consequently, b values of 0 and 800 sec/mm2 are recommended for clinical practice, providing a balance between sufficient diffusion weighting and acceptable SNR (Mann et al., 2019).

Higher b values emphasize contrast resolution between the lesions and normal breast tissue (Lee et al., 2021).

Dynamic Contrast-Enhanced MRI (DCE-MRI):

The basis for any MRI protocol is a dynamic T1-weighted contrast enhanced sequence T1-weighted Imaging: Images are usually acquired in the axial plane, which is faster than sagittal acquisition and provides a better overview of both breasts. A native T1-weighted acquisition should be obtained prior to contrast material administration. Contrast material should be administered at a maximum dose of 0.1 mmol per kg of body weight. Preferentially, a power injector should be used at a flow rate of 2 mL/sec. The contrast material bolus should be flushed with saline (a bolus of approximately 20 mL) (Carbonaro et al., 2011).

The sequence is called “dynamic” because it is first performed before contrast administration and is repeated multiple times after contrast administration. It is essential to obtain an image approximately 60–90 seconds after contrast material administration, as most breast cancers will show peak enhancement at that time. Lesion detection is primarily performed by using these postcontrast images. For images obtained without fat suppression, creating subtraction images from the pre- and postcontrast acquisitions is required. Subtraction images are also helpful for acquisitions with fat suppression because they help differentiate truly enhancing structures from lesions with native high signal intensity at T1 (Fig. 12) (Mann et al., 2019).

Dynamic Evaluation with Time–Signal Intensity Curves:

Dynamic analysis investigates the permeability of the vessels that supply a lesion. This is done by obtaining a series of T1-weighted acquisition between 5 and 7 minutes after contrast material administration. In the case of leaky vessels, the peak contrast material accumulation will have passed, and contrast material is being removed from the lesion. In lesions with less-permeable vessels, the contrast gradient over the vessel wall will still be positive, and therefore the enhancement of the lesion still increases (Partridge et al., 2014).

This is reflected in the shape of the time–signal intensity curves; a persistent increase is most commonly seen in benign lesions, whereas a decrease in the late phase is common in malignant lesions. To improve lesion classification, the most suspicious curve observed (washout >plateau > persistent). Approximately 85% of cancers manifest with a washout curve (Fig. 13). Persistent curves are rare in malignancies, although they may be present in ductal carcinoma in situ (DCIS) and more diffuse-growing invasive cancers, particularly lobular breast cancers (Abe et al., 2016 and Mann et al., 2019).

Multiparametric Breast MRI Protocol:

The state-of-the-art breast MRI protocol is a multiparametric examination consisting of dynamic contrast-enhanced (DCE) T1-weighted imaging, T2-weighted/bright fluid imaging, and oftentimes diffusion-weighted imaging (DWI). ultrafast imaging can be added to the standard imaging protocol. Such a multiparametric approach enables detailed tissue characterization in addition to improved discrimination between benign and malignant breast lesions (Fig 14) (Mori and Abe, 2022).

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