EPITHELIAL PROLIFERATION AND LEUKOCYTE INFILTRATION IN CHRONIC BREAST INFLAMMATION

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Abstract. Objective: To describe the relationship between epithelial proliferation and leukocyte infiltration in chronic breast inflammation using Ki-67 and CD45 immunohistochemistry. The source study included 63 observations, comprising 50 cases of chronic inflammation and 13 morphologically normal controls. Tissue morphology and digitally quantified immunostaining were assessed together. In the illustrated 34- and 63-year-old inflammatory cases, Ki-67 positivity was 5.33% and 4.47%, whereas CD45 positivity was 21.98% and 23.58%, respectively. Corresponding illustrated control values were 10.82% and 8.97% for Ki-67 and 7.61% and 9.98% for CD45. These case-level findings describe prominent leukocyte infiltration with relatively low proliferative activity. They support integrated interpretation of morphology and immunohistochemistry but do not establish malignant transformation or predict future breast cancer.

Keywords. Breast tissue, chronic inflammation, Ki-67, CD45, immunohistochemistry, digital pathology.

Introduction

Chronic inflammation of the breast involves changes in both the epithelial compartment and its stromal environment. Histological assessment can be supplemented by immunohistochemistry to distinguish the distribution of leukocytes from epithelial proliferative activity. In the source investigation, Ki-67 was used to assess proliferation, while CD45 characterised the leukocyte component of stromal and periepithelial infiltrates [1, 2].

The objective was to describe Ki-67 and CD45 expression in chronic breast inflammation in relation to tissue morphology and the age of the illustrated cases.

Materials and methods

The comparative descriptive and analytical morphological study examined biopsy and surgical specimens and archival paraffin blocks from 63 observations: 50 with confirmed chronic inflammation and 13 assessed as morphologically normal. The main comparative statistical subset comprised 37 observations aged 30–35 years, including 25 inflammatory cases and 12 controls. The remaining 26 observations were used for descriptive assessment of age-related morphology [1].

Formalin-fixed paraffin sections were examined by routine histology and immunohistochemistry with positive and negative controls. Digital image analysis included QuPath. Ki-67 nuclear immunoreactivity and the proportion and distribution of CD45-positive cells were evaluated alongside epithelial and stromal morphology. Acute suppurative inflammation, extensive necrosis, invasive carcinoma, carcinoma in situ and marked atypical proliferation were excluded. This report presents the individual 34- and 63-year-old examples documented in the dissertation abstract; it does not treat their values as group means.

Results.

In the illustrated 34-year-old control specimen, 282 of 2,606 analysed cells were Ki-67 positive (10.82%). In the inflammatory specimen of the same reported age, 158 of 2,965 cells were positive (5.33%). In the 63-year-old examples, Ki-67 positivity was 211 of 2,353 cells (8.97%) in the control and 137 of 3,063 cells (4.47%) in the inflammatory specimen. Immunoreactivity was described principally in epithelial nuclei [1].

CD45 showed a different pattern. In the 34-year-old examples, positivity was 146 of 1,918 cells (7.61%) in the control and 747 of 3,399 cells (21.98%) in the inflammatory specimen. At age 63, the respective values were 373 of 3,737 cells (9.98%) and 324 of 1,374 cells (23.58%). CD45-positive cells accumulated in stromal, periductal and perilobular regions. Histological examination also identified lymphoplasmacytic and macrophage infiltration, reactive epithelial changes and stromal fibrosis [1].

Discussion

The illustrated cases show that substantial leukocyte infiltration can coexist with lower Ki-67 positivity than that observed in the respective control examples. Interpreting both markers with the histological architecture provides a more informative description of chronic inflammation than either percentage alone. The distribution of CD45-positive cells helps localise the inflammatory component, while Ki-67 adds information on proliferative activity [1, 2].

These findings require case-level interpretation. The reported cell counts are image-analysis denominators rather than numbers of independent patients. They cannot be used as independent observations to infer group significance. The abstract also does not establish a consistently epithelial-only denominator for every displayed Ki-67 percentage. Consequently, these values should not be presented as validated epithelial proliferation thresholds. The selected benign inflammatory material and absence of longitudinal cancer outcomes preclude conclusions about malignant progression.

Conclusion

Joint assessment of Ki-67, CD45 and tissue morphology characterises the coexistence of leukocyte infiltration, stromal remodelling and epithelial proliferative changes in chronic breast inflammation. The illustrated findings support an integrated pathological description. Patient-level analyses with standardised cell selection and longitudinal follow-up are needed before any prognostic application.

References

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