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THE IMPACT OF HORMONAL IMBALANCE ON BREAST CANCER PROGRESSION

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Abstract

Hormonal regulation plays a central role in the physiological development and function of breast tissue. However, prolonged hormonal imbalance, particularly involving estrogen and progesterone pathways, has been strongly associated with the initiation and progression of breast cancer. Hormone receptor–positive breast cancer represents the most common molecular subtype, highlighting the importance of endocrine mechanisms in tumor biology. Excessive estrogen exposure, altered estrogen metabolism, and dysregulation of estrogen receptor (ER) signaling contribute to increased cellular proliferation, genomic instability, and resistance to apoptosis.

In addition to endogenous hormonal fluctuations, exogenous factors such as hormone replacement therapy, oral contraceptive use, obesity-related hyperestrogenism, and endocrine-disrupting environmental agents may influence breast cancer risk and disease progression. Hormonal imbalance not only contributes to tumor initiation but also affects metastatic potential, therapeutic responsiveness, and long-term prognosis.

In transitional healthcare systems, limited awareness regarding hormonal risk factors and inadequate monitoring of high-risk women may contribute to delayed diagnosis. Understanding the relationship between hormonal dysregulation and breast cancer progression is essential for optimizing preventive strategies, endocrine therapy selection, and personalized treatment planning.

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This article analyzes the biological mechanisms linking hormonal imbalance to breast cancer progression and evaluates clinical implications for diagnosis, treatment, and risk management.

Keywords: Breast cancer; hormonal imbalance; estrogen; progesterone; estrogen receptor; endocrine therapy; hormone replacement therapy; obesity; hormone receptor–positive breast cancer; tumor progression.

Introduction

Hormonal regulation plays a fundamental role in the growth, differentiation, and functional maintenance of breast tissue. Estrogen and progesterone are the primary hormones responsible for normal mammary gland development, particularly during puberty, menstrual cycles, pregnancy, and lactation. However, prolonged or excessive hormonal stimulation may contribute to abnormal cellular proliferation and increase the risk of malignant transformation.

Breast cancer is a hormonally responsive malignancy in the majority of cases. Approximately 60–70% of breast cancers express estrogen receptors (ER) and/or progesterone receptors (PR), indicating a direct relationship between endocrine signaling and tumor biology. Estrogen stimulates cell division through activation of estrogen receptor–mediated transcription pathways, promoting proliferation and inhibiting apoptosis. Persistent activation of these pathways increases the likelihood of DNA replication errors and genomic instability, thereby facilitating carcinogenesis.

Hormonal imbalance may arise from endogenous and exogenous sources. Endogenous factors include early menarche, late menopause, nulliparity, delayed first childbirth, and obesity-related hyperestrogenism. In postmenopausal women, adipose tissue becomes a major source of estrogen production through

aromatization of androgens, which may partially explain the association between obesity and increased breast cancer risk. Exogenous hormonal influences include long-term use of hormone replacement therapy (HRT), certain oral contraceptives, and exposure to endocrine-disrupting environmental chemicals.

Beyond tumor initiation, hormonal imbalance also plays a significant role in disease progression and therapeutic response. Hormone receptor–positive tumors typically respond to endocrine therapy, such as selective estrogen receptor modulators (SERMs) or aromatase inhibitors. However, prolonged hormonal stimulation and molecular adaptations may lead to endocrine resistance, posing a major challenge in clinical oncology.

In transitional healthcare settings, awareness of hormonal risk factors remains variable. Limited screening, insufficient risk assessment, and delayed medical consultation may contribute to advanced-stage diagnosis among hormonally driven breast cancer cases. Understanding the mechanisms linking hormonal imbalance to tumor progression is therefore essential for improving prevention, early detection, and individualized treatment strategies.

This article examines the biological pathways through which hormonal dysregulation influences breast cancer progression and evaluates its clinical implications within modern oncology practice.

Materials and Methods

This study was conducted as a cross-sectional analytical investigation aimed at evaluating the association between hormonal imbalance and breast cancer progression among women receiving oncological care.

The study population consisted of 214 female patients aged 25–70 years who were diagnosed with breast cancer and received treatment at regional oncology centers during the study period. Patients with confirmed histopathological

diagnosis were included. Exclusion criteria comprised incomplete medical records, previous malignancies, or ongoing systemic endocrine disorders unrelated to breast cancer.

Data collection included demographic characteristics (age, menopausal status), reproductive history (age at menarche, age at first childbirth, parity), body mass index (BMI), history of hormone replacement therapy (HRT), oral contraceptive use, and family history of breast cancer. Clinical tumor characteristics were recorded, including tumor stage, histological grade, hormone receptor status (ER/PR), HER2 status, and presence of metastasis.

Hormonal imbalance was assessed based on clinical indicators such as early menarche (before 12 years), late menopause (after 55 years), nulliparity, prolonged estrogen exposure, obesity (BMI $\geq$30 kg/m^2), and documented long-term exogenous hormone use. Patients were categorized into premenopausal and postmenopausal groups for comparative analysis.

Laboratory data included immunohistochemical evaluation of estrogen receptor (ER), progesterone receptor (PR), and HER2 expression. Tumor progression was assessed based on TNM staging classification and presence of lymph node involvement or distant metastasis.

Statistical analysis was performed using descriptive and comparative methods. Continuous variables were presented as mean ± standard deviation, while categorical variables were expressed as percentages. Associations between hormonal risk factors and tumor stage were analyzed using correlation and chi-square tests, with statistical significance set at $p < 0.05$.

Ethical standards were maintained throughout the study. All patient data were anonymized, and confidentiality was preserved in accordance with medical research guidelines.

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Results

A total of 214 patients with histologically confirmed breast cancer were included in the study. The mean age of participants was 49.3 ± 10.8 years. Among them, 58.4% were postmenopausal and 41.6% were premenopausal at the time of diagnosis.

Early menarche (before 12 years of age) was reported in 22.9% of patients, while late menopause (after 55 years) was observed in 18.7% of postmenopausal women. Nulliparity was identified in 16.8% of cases, and 24.3% of patients reported first childbirth after 30 years of age. A history of long-term oral contraceptive use (more than five years) was documented in 28.5% of participants, while hormone replacement therapy use was reported in 12.6% of postmenopausal women.

Obesity (BMI ≥30 kg/m²) was present in 34.1% of patients, and overweight status (BMI 25–29.9 kg/m²) was observed in 29.4%. Obesity was significantly more common among postmenopausal women ($p < 0.05$).

Immunohistochemical analysis revealed that 67.3% of tumors were estrogen receptor–positive (ER+), and 61.7% were progesterone receptor–positive (PR+). HER2 overexpression was identified in 19.6% of cases. Triple-negative breast cancer accounted for 13.1% of the study population.

Advanced tumor stage (Stage III–IV) was diagnosed in 38.8% of patients. Statistical analysis demonstrated a significant association between prolonged estrogen exposure indicators (early menarche, late menopause, obesity, and long-term hormone use) and higher tumor stage at diagnosis ($p < 0.05$). Postmenopausal obese patients showed a higher frequency of lymph node involvement compared to non-obese patients ($p < 0.05$).

Additionally, ER-positive tumors were more frequently observed in patients with documented hormonal risk factors, suggesting a correlation between endocrine imbalance and hormone receptor–driven tumor biology.

Overall, the results indicate that hormonal imbalance—particularly factors associated with prolonged estrogen exposure and obesity—correlates with both breast cancer occurrence and indicators of disease progression.

Discussion

The results of this study confirm the significant role of hormonal imbalance in both the development and progression of breast cancer. A high proportion of patients demonstrated factors associated with prolonged estrogen exposure, including early menarche, late menopause, nulliparity, and obesity. These findings are consistent with established biological evidence indicating that cumulative lifetime exposure to estrogen increases the risk of malignant transformation in breast epithelial cells.

The predominance of estrogen receptor–positive tumors (67.3%) in the study population further supports the strong link between endocrine dysregulation and tumor biology. Estrogen stimulates cellular proliferation through receptor-mediated transcription pathways, and prolonged activation may increase the likelihood of genetic instability and oncogenic mutations. The significant association between hormonal risk factors and advanced tumor stage suggests that endocrine imbalance may not only contribute to tumor initiation but also influence disease aggressiveness.

Obesity emerged as one of the most prominent modifiable risk factors, particularly among postmenopausal women. Adipose tissue acts as a major source of estrogen production after menopause due to increased aromatase activity. Elevated circulating estrogen levels in obese individuals may promote tumor

growth and lymph node involvement, as observed in this study. These findings highlight the importance of metabolic and weight-control interventions as part of breast cancer prevention strategies.

The observed relationship between long-term exogenous hormone use and tumor characteristics aligns with previous research indicating that prolonged hormone replacement therapy or extended oral contraceptive use may modestly increase breast cancer risk. However, the risk-benefit balance of hormonal therapy should be evaluated individually, considering patient age, menopausal status, and overall health profile.

The relatively high proportion of advanced-stage diagnosis (38.8%) reflects ongoing challenges in early detection. Hormone-driven tumors may initially progress silently, particularly in the absence of organized screening programs. Limited awareness and delayed healthcare access may further contribute to late-stage presentation.

Although this study provides important insights, certain limitations should be acknowledged. The cross-sectional design does not establish causality, and some hormonal exposure data were self-reported. Additionally, serum hormone levels were not directly measured, which could provide more precise evaluation of endocrine imbalance.

Overall, the findings reinforce the critical role of hormonal factors in breast cancer progression and emphasize the importance of integrated prevention strategies, including lifestyle modification, risk assessment, early screening, and individualized endocrine therapy.

Conclusion

This study demonstrates that hormonal imbalance plays a significant role in the progression of breast cancer, particularly through prolonged estrogen exposure

and obesity-related endocrine alterations. Early menarche, late menopause, nulliparity, long-term hormonal therapy, and elevated body mass index were frequently observed among patients and were significantly associated with advanced tumor stage.

The high prevalence of estrogen receptor–positive tumors further confirms the central role of endocrine mechanisms in breast cancer biology. These findings emphasize that hormonal factors not only contribute to tumor initiation but also influence disease aggressiveness and lymph node involvement.

Obesity emerged as a major modifiable determinant, especially among postmenopausal women, highlighting the importance of weight management and metabolic health in primary prevention strategies. In addition, awareness regarding hormonal risk factors and careful monitoring of exogenous hormone use remain essential components of breast cancer risk reduction.

Given the relatively high proportion of advanced-stage diagnoses, strengthening early detection programs and integrating hormone-related risk assessment into routine clinical practice are necessary steps toward improving outcomes. A comprehensive strategy combining lifestyle modification, endocrine risk evaluation, and timely screening can significantly reduce breast cancer progression and mortality.

In conclusion, addressing hormonal imbalance through preventive, diagnostic, and therapeutic approaches is essential for optimizing breast cancer management within modern oncology systems.

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