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Hypertension, Lifestyle Factors, and Breast Cancer Outcomes: A Decade-Long Retrospective Cohort Study

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ABSTRACT

Background & Objective: Breast cancer is the second leading cause of cancer-related mortality in women and is influenced by genetic, environmental, and lifestyle factors. This study aimed to evaluate the prevalence of risk factors in breast cancer patients and their association with disease progression and survival.

Materials & Methods: A retrospective analysis was conducted on 400 breast cancer patients treated between 2011 and 2021 at Sina and Farmaniyeh hospitals in Tehran. The study evaluated clinical factors like hypertension, diabetes, and hyperlipidemia, alongside reproductive histories and lifestyle factors such as smoking and oral contraceptive use. Tumor characteristics and 5-year survival outcomes were analyzed using logistic regression and Kaplan-Meier methods.

Results: Hypertension was correlated with advanced tumor stages ($P = 0.048$) and higher grades ($P = 0.025$). Early menarche is associated with lymphovascular involvement ($P = 0.005$) and an increased risk of metastasis ($P = 0.049$). Additionally, smoking significantly raised the risk of lymphovascular invasion ($P < 0.001$). Survival analysis revealed a non-significant trend toward reduced 5-year survival in hypertensive patients (log-rank $P = 0.288$), with cumulative hazard curves indicating higher mortality rates. No significant associations were found for diabetes, family history, or contraceptive use.

Conclusion: The results indicate that hypertension is linked to aggressive tumor characteristics, potentially affecting disease progression. Although the connection to survival was not statistically significant, the increased risk in hypertensive patients calls for further research. Additionally, early menarche and smoking correlate with negative tumor features, emphasizing the need to monitor hypertension and address lifestyle factors in breast cancer management.

Keywords: Breast Neoplasms, Disease Progression, Hypertension, Survival Analysis

1. Introduction

Breast cancer represents the second leading cause of cancer-related mortality among women worldwide. In 2020, global epidemiological data documented over 2.3 million incident cases of breast cancer, accounting for approximately 685,000 mortalities (1). Advances in routine screening protocols have facilitated the early detection of disease in contemporary clinical practice, enabling therapeutic intervention before disease metastasis. Consequently, a marked reduction in breast cancer-associated mortality rates has been observed in recent decades compared to historical trends (2).

The clinical trajectory of breast cancer is modulated by a multifactorial interplay of genetic predispositions, environmental determinants, and comorbid health

conditions, which collectively influence therapeutic responses and prognostic outcomes (3). Beyond established risk factors such as age at menarche, age of first pregnancy, number of pregnancies, breastfeeding history, menopausal status, the use of oral contraceptive pills (OCP), and lifestyle variables including smoking emerging evidence highlights the role of metabolic disorders (e.g., hyperlipidemia, diabetes) and cardiovascular comorbidities in shaping breast progression (4-8). Notably, hypertension, clinically defined as a persistent elevation of arterial blood pressure (≥140/90 mmHg), constitutes a pervasive public health challenge, affecting an estimated 1.28 billion adults globally and contributing significantly to premature morbidity and mortality (9, 10). Its frequent coexistence with breast cancer in aging women underscores the clinical urgency of elucidating their interaction (11, 12).

Demographic analyses reveal a pronounced overlap in breast cancer incidence and hypertension among postmenopausal populations, a cohort also characterized by elevated risks linked to delayed age at first pregnancy, nulliparity, and metabolic syndrome (13, 14). Shared molecular mechanisms between hypertension and breast carcinogenesis further suggest pathophysiological crosstalk. For instance, polymorphisms in BRCA2 and GRK4 genes, implicated in oncogenesis and hypertensive pathogenesis, may reflect overlapping genetic susceptibilities (15, 16). Hypertension-driven chronic inflammation, characterized by sustained pro-inflammatory cytokine release, may synergize with tumorigenic pathways, a hypothesis supported by established links between inflammatory mediators and carcinogenesis (16, 17). Concurrently, hyperlipidemia and diabetes may exacerbate oxidative stress and hormonal dysregulation, potentially accelerating breast cancer progression (18, 19).

Given the critical public health implications of these interactions and the paucity of conclusive evidence from prior studies, rigorous investigation into the synergistic effects of hypertension—with particular emphasis on its interplay with reproductive history, metabolic comorbidities, and lifestyle factors—on breast cancer progression is warranted.

2. Materials and Methods

2.1 Study Design and Population

This retrospective cohort study included female patients diagnosed with breast cancer at Sina and Farmaniyeh hospitals in Tehran, Iran, between January 2011 and December 2021. Patients were identified through hospital archives using International Classification of Diseases (ICD-10) codes for breast cancer (C50). Exclusion criteria comprised individuals with incomplete clinical, pathological, or follow-up records. A total of 400 patients met the eligibility criteria and were included in the final analysis.

2.2 Data Collection

Demographic, clinical, and pathological data were extracted from electronic health records and histopathology reports. Variables included age at diagnosis, tumor stage, comorbidities (hypertension, diabetes, hyperlipidemia), menopausal status, reproductive history (age at menarche, age at first pregnancy, breastfeeding status), lifestyle factors (smoking status), and survival outcomes.

2.3 Variables and Definitions

Breast Cancer Staging: Tumor-node-metastasis (TNM) classification was assessed according to the American Joint Committee on Cancer (AJCC) 7th edition guidelines (20).

Hypertension (HTN): Defined as systolic blood pressure (SBP) ≥140 mmHg, diastolic blood pressure (DBP) ≥90 mmHg, or current use of antihypertensive medication (21).

Diabetes Mellitus (DM): Diagnosed per American Diabetes Association (ADA) criteria: fasting plasma glucose ≥126 mg/dL, HbA1c ≥6.5%, or documented use of antidiabetic therapy (22).

Hyperlipidemia: Identified by LDL cholesterol ≥130 mg/dL, triglycerides ≥150 mg/dL, or lipid-lowering medication use, based on the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) guidelines (23).

Menopausal Status: Postmenopausal status was defined as ≥12 consecutive months of amenorrhea not attributable to other causes or follicle-stimulating hormone (FSH) levels >30 IU/L (24).

Smoking: Classified as smokers (current or former), or never smokers, with current smoking defined as ≥1 cigarette/day for ≥6 months.

Reproductive Factors: Age at menarche (first menstrual cycle), age at first pregnancy (primiparity), GRAVID status, breastfeeding duration (total months), the use of OCP, and age at menopause were self-reported or extracted from medical records.

2.4 Survival Analysis

Survival outcomes were assessed using Kaplan-Meier analysis to estimate the 5-year overall survival (OS) probabilities, with patients categorized based on their hypertension status. The survival curves were compared using the log-rank (Mantel-Cox) test, and any censored observations—such as patients lost to follow-up or those who remained event-free at the end of the study—were marked on the curves. Additionally, cumulative hazard functions were derived using the Nelson-Aalen estimator to quantify mortality risk over time. Survival duration was calculated in months, beginning from the date of diagnosis to the date of death or the last follow-up. Some patients were monitored for up to 60 months, and a subset completed the full 5-year follow-up, which allowed for a robust estimation of overall survival.

2.5 Statistical Analysis

Data were analyzed using IBM SPSS Statistics 26 (IBM Corp., Armonk, NY). Continuous variables were reported as mean ± standard deviation (SD) or median (interquartile range, IQR), and categorical variables as frequencies (%). Correlations between variables were assessed using Pearson's (normally distributed data) or Spearman's (non-parametric data) tests Chi-square tests were used to evaluate associations between categorical variables. A two-tailed P-value <0.05 was considered statistically significant.

3. Result

3.1 Baseline Characteristics of the Study Population

A total of 400 Iranian ethnicity patients diagnosed with breast cancer, aged between 22 and 86 years, were included in the study. The median age of the patients was 51.32 years, with a standard deviation of ± 0.549 years. Table 1 presents the demographic features of the tumors as well as the characteristics of the patients included in the study. This investigation aimed to determine the prevalence of hypertension, hyperlipidemia, and diabetes mellitus among individuals diagnosed with breast cancer. The findings of this study are detailed in Table 1.

3.2 Analyzing the Correlation Among Variables

The study aimed to establish a relationship between patient characteristics and breast cancer demographics. Our research revealed a significant correlation between hypertension and the stage and grade of breast cancer. However, we did not find any significant correlations between the age of first pregnancy, age of menopause, breastfeeding, diabetes mellitus, gravid status, hyperlipidemia, menopause status, and the use of OCP with the characteristics of breast cancer. We did discover a significant correlation between the age at which patients experienced menarche and both lymphovascular involvement and metastasis. Additionally, there is a significant positive association between cigarette smoking and lymphovascular invasion in breast cancer. For specific details on these correlations, please refer to Table 2.

3.3 Survival Analysis

Survival analysis indicated that hypertensive breast cancer patients experienced significantly poorer 5-year OS compared to their non-hypertensive counterparts, with Kaplan-Meier probabilities of 70% versus 85%, respectively (log-rank chi-square: 1.128; P-value = 0.288; see Figure 1a). The association was not significant; however, the survival curves diverged notably after 15 to 20 months post-diagnosis, indicating a progressive decline in overall survival among patients with hypertension. This trend suggests a cumulative adverse effect of hypertension on long-term outcomes. Cumulative hazard analysis further supported these findings, showing that the mortality risk in the hypertensive cohort was nearly doubled (Nelson-Aalen cumulative hazard: 0.32 compared to 0.18 at 5 years; refer to Figure 1b). These results emphasize hypertension as a significant predictor of reduced survival.

Table 1. The clinical and pathological features of breast cancer patients.

Characteristics		% of total patients
Age	22-29 years old	1.0
	30-39 years old	15.1
	40-49 years old	30.8
	50-59 years old	28.8
	60-69 years old	15.5
	70 ≤ years old	8.1
Breast cancer type	Invasive ductal carcinoma	93.6
	Other types	6.4
Breast cancer grade	I	20.2
	II	51.0
	III	28.8
Breast cancer stage	I	26.5
	II	51.9
	III	18.6
	IV	3.0
Metastasis	Positive	12.1
	Negative	87.9

Characteristics		% of total patients
Lymphovascular invasion	Positive	33.5
	Negative	66.5
Smoker patients	Positive	5.7
	Negative	94.3
Breastfeeding	Positive	13.1
	Negative	86.9
Family history of breast cancer	First degree relative	14.3
	Second degree relative	11.3
	Negative	74.5
Family history of cancer other than breast	Positive	19.0
	Negative	81.0
Patients with hypertension	Positive	18.7
	Negative	81.3
Patients with diabetes mellitus	Positive	11.1
	Negative	88.9
Patients with Hyperlipidemia	Positive	8.1
	Negative	91.9
OCP consumption	<1 years old	5.4
	1-2 years old	7.4
	2< years old	13.8
	Negative	73.4
Age at first pregnancy	<18 years old	20.5
	18-35 years old	74.8
	35< years old	0.7
	Single or Infertile	4.0
GRAVID	1-3	59.4
	4 ≤	31.9
	Single or Infertile	8.7
Menopause status	Pre menopause	50.9
	Post menopause	49.1
Age of Menopause	30-34 years old	1.2
	34-39 years old	3.6
	40-44 years old	12.2
	45-49 years old	26.8
	50-54 years old	48.8
	55-59 years old	6.0
	60 ≤ years old	1.2
Menarche age	7-11 years old	11.7
	12-13 years old	45.6
	14 ≤ years old	42.1
	Amenorrhea	0.6

Table 2. The associations between the characteristics of patients and the features of tumors (r: the correlation coefficient; the significant associations were bolded).

Parameters	Normal values			
	Tumor grade	Tumor stage	Lymphovascular invasion	Metastasis
Age of first pregnancy	r:-0.085 P-Value:0.315	r:0.043 P-Value:0.598	r:0.026 P-Value:0.754	r:0.028 P-Value:0.0729
Age of menarche	r:-0.079 P-Value:0.318	r:0.107 P-Value:0.166	r:0.217 P-Value:0.005	r:0.151 P-Value:0.049
Age of menopause	r:0.031 P-Value:0.786	r:0.028 P-Value:0.404	r:0.001 P-Value:0.992	r:0.170 P-Value:0.126
Breastfeeding	r:0.002 P-Value:0.974	r:-0.033 P-Value:0.599	r:-0.050 P-Value:0.441	r:0.112 P-Value:0.076
Diabetes Mellitus	r:0.024 P-Value:0.644	r:0.014 P-Value:0.778	r:-0.076 P-Value:0.134	r:-0.019 P-Value:0.695
GRAVID	r:0.047 P-Value:0.472	r:0.007 P-Value:0.912	r:0.118 P-Value:0.065	r:0.047 P-Value:0.455
Hyperlipidemia	r:0.030 P-Value:0.554	r:0.016 P-Value:0.753	r:-0.049 P-Value:0.331	r:-0.003 P-Value:0.954
Hypertension	r:0.115 P value:0.025	r:0.098 P value:0.048	r:0.060 P value:0.238	r:0.092 P value:0.063
Menopause status	r:-0.026 P value:0.595	r:0.031 P value:9,514	r:0.018 P value:0.709	r:0.080 P value:0.090
OCP consumption	r:0.046 P value:0.429	r:0.001 P value:0.986	r:0.023 P value:0.667	r:0.001 P value:0.988
Smoking	r:0.031 P value:0.594	r:0.003 P value:0.953	r: 0.184 P value: 0.001	r:0.042 P value:0.457

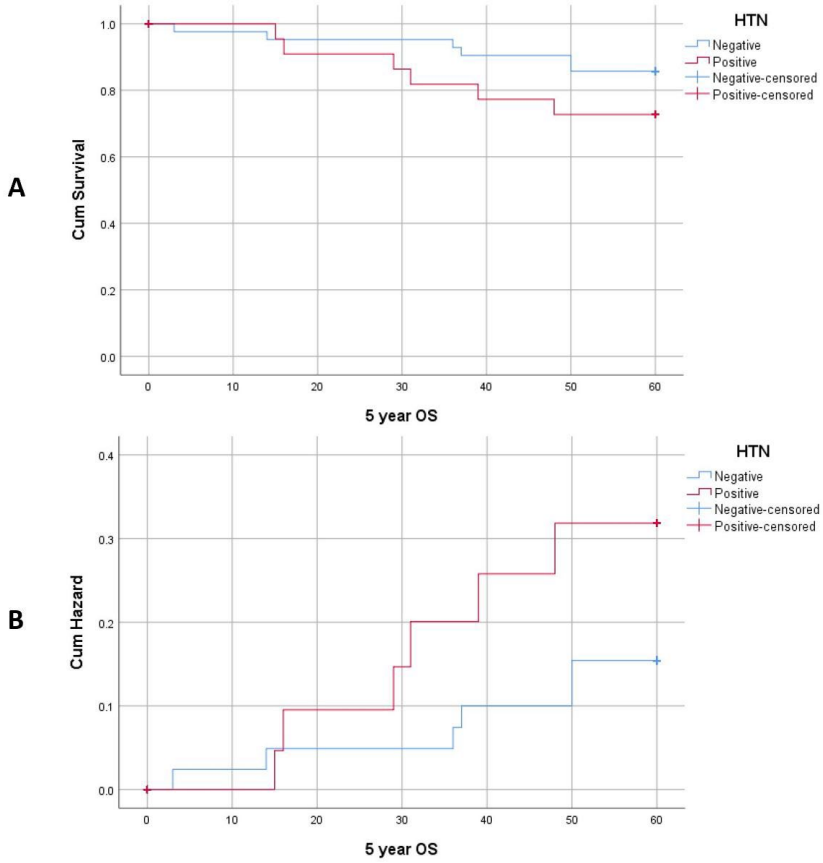

Figure 1. Survival and cumulative hazard analysis based on hypertension status in breast cancer patients. (A) The Kaplan-Meier survival curves show a significantly lower 5-year overall survival rate for hypertensive (HTN-positive) patients, with a survival probability of 70%, compared to non-hypertensive (HTN-negative) patients, who have a survival probability of 85% (log-rank chi-square: 1.128; P-value = 0.288). Survival divergence begins to appear 15–20 months after diagnosis, with a continued decline in the HTN-positive group. Censored observations, indicated by vertical ticks, represent patients who were lost to follow-up or remained event-free at the end of the study. (B) The Nelson-Aalen cumulative hazard curves demonstrate a higher mortality risk among HTN-positive patients, with a cumulative hazard of 0.32 at 5 years, compared to 0.18 for HTN-negative patients. (**Designed by Authors, 2025**).

4. Discussions

Breast cancer prognosis is influenced by a complex interaction of genetic, environmental, and comorbid factors, with hypertension emerging as a significant factor affecting disease aggressiveness **(25, 26)**. Our findings indicate that breast cancer patients with hypertension tend to have more advanced tumor stages (P=0.048) and higher histologic grades (P=0.025). This aligns with previous research linking hypertension to oncogenic pathways such as chronic inflammation, oxidative stress, and dysregulated angiogenesis **(7, 27)**. Han, Guo **(7)** corroborated this association through a meta-analysis, identifying elevated blood pressure as a risk factor for breast cancer incidence. Furthermore, Powe, Voss **(28)** reported that beta-blocker therapy—a cornerstone antihypertensive treatment—reduces metastasis and recurrence rates, potentially by antagonizing adrenergic signaling pathways that promote tumor proliferation and immune evasion **(29, 30)**. In another study, Jung, Rosenzweig **(31)** found that patients with a history of hypertension had significantly worse outcomes when diagnosed with metastatic breast cancer, which aligns with our results. These findings suggest that managing hypertension could offer both cardiovascular and cancer-protective benefits, although further studies are needed to clarify the underlying mechanisms.

Moreover, our study identified early menarche (≤ 12 years) as a predictor of lymphovascular invasion (P = 0.005) and metastatic progression (P = 0.049), highlighting the role of prolonged estrogen exposure in breast cancer development **(32-35)**. However, the relationship between the age of menarche and tumor behavior remains a topic of debate. For instance, Orgéas, Hall **(36)** observed an inverse association between menarche age and lymph node invasion, which contrasts with our results. This discrepancy may reflect differences in study populations, tumor subtypes, or methodologies, underlining the need for standardized protocols in assessing reproductive risk factors.

While hypertension did not significantly impact 5-year survival (log-rank P = 0.288), the divergence in cumulative hazard curves after 15–20 months post-diagnosis implies a latent detrimental effect, potentially mediated by treatment-related cardiotoxicity or comorbid complications **(37, 38)**. The lack of associations between diabetes, family history, or contraceptive use and tumor

progression further highlights the multifactorial nature of breast cancer pathogenesis. This underscores the importance of developing population-specific risk stratification models (39, 40).

5. Conclusion

This study highlights hypertension as an important factor influencing tumor aggressiveness, with a correlation observed between hypertension and advanced cancer stage as well as higher tumor grade. Additionally, early menarche and smoking are linked to negative histopathological features. While hypertension did not have a significant impact on survival rates, trends in cumulative hazard suggest it may have prognostic importance, indicating the need for further research. These findings support the integration of hypertension management and lifestyle interventions into breast cancer treatment protocols to help reduce disease progression.

6. Declarations

6.1 Acknowledgments

The authors express their gratitude to all staff at Sina and Farmaniyeh hospitals in Tehran for their valuable collaboration in conducting this study.

6.2 Ethical Considerations

This study was approved by the Ethics Committee of Tehran University of Medical Sciences (IR.TUMS.SPH.REC.1400.011). Patient confidentiality was maintained through anonymization of identifiers during data extraction.

6.3 Authors' Contributions

F.S., M.M.A., and M.H.M. conceived and designed the study. L.G. assisted with patient recruitment, and M.H.M. carried out data acquisition. S.F., R.H.M., and N.A. conducted statistical analyses and interpreted the results. M.H.M. wrote the initial manuscript, which was critically revised for intellectual content by F.S., M.M.A., and L.G. All authors reviewed, edited, and approved the final version of the manuscript.

6.4 Conflict of Interest

The author has no financial conflicts of interest and no funding sources to disclose.

6.5 Fund or Financial Support

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6.6 Using Artificial Intelligence Tools (AI Tools)

The authors affirm that no AI tools were used in the design, execution, analysis, or writing of this research. All data collection, interpretation, and manuscript preparation were conducted manually by the authors without the assistance of generative AI or automated content creation technologies.

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