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Original Research Article

CORRELATION BETWEEN KI67 AND TUMOR GRADE IN BREAST CARCINOMA

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ABSTRACT

Background: Breast carcinoma is a heterogeneous malignancy with variable clinical behavior and prognosis. Histological tumor grade and Ki-67 proliferation index are both established indicators of tumor aggressiveness, with Ki-67 serving as a quantitative marker of proliferative activity. Assessing the correlation between these parameters can improve prognostic accuracy and guide treatment planning. The aim is to evaluate the correlation between Ki-67 proliferation index and histological tumor grade in invasive breast carcinoma.

Materials and Methods: This cross-sectional observational study was conducted at a tertiary care hospital on 70 consecutive cases of histologically confirmed invasive breast carcinoma. Tumor grading was performed using the Nottingham modification of the Scarff-Bloom-Richardson system. Ki-67 expression was assessed immunohistochemically, and the labelling index was categorized as low (<15%), intermediate (15–30%), or high (>30%). Statistical analysis was performed using SPSS version 26.0, with Spearman's rank correlation coefficient, Chi-square test, and one-way ANOVA applied as appropriate. A p-value <0.05 was considered statistically significant.

Results: The most common age group was 41–50 years (31.43%). Grade III tumors were most frequent (42.86%), followed by Grade II (40.00%) and Grade I (17.14%). High Ki-67 expression was observed in 60.00% of cases, intermediate in 25.71%, and low in 14.29%. A significant association was found between tumor grade and Ki-67 category ($p < 0.001$), with 86.67% of Grade III tumors showing high Ki-67 and none showing low Ki-67. Mean Ki-67 values increased with grade: Grade I (12.45%), Grade II (32.86%), and Grade III (54.73%), with a significant difference across grades ($p < 0.001$).

Conclusion: There is a strong, statistically significant positive correlation between histological tumor grade and Ki-67 proliferation index in invasive breast carcinoma. Higher tumor grades are associated with higher Ki-67 expression, reflecting more aggressive tumor biology. Ki-67 can serve as a valuable adjunct to histological grading in prognostic evaluation and treatment decision-making.

Keywords: Breast carcinoma, Ki-67, Tumor grade, Proliferation index, Prognostic marker.

INTRODUCTION

Breast carcinoma is the most frequently diagnosed malignancy and a leading cause of cancer-related mortality among women worldwide. Despite advances in screening, diagnosis, and treatment, it remains a heterogeneous disease with variable biological behavior and clinical outcomes. Accurate prognostication is therefore critical to guide therapeutic decisions and improve survival. Traditional prognostic parameters such as tumor size, lymph node status, and histological grade continue to play an important role; however, they may not fully capture the intrinsic biological aggressiveness of the tumor. In recent years, proliferation markers, particularly Ki-67, have emerged as valuable tools in

refining prognostic assessment and informing treatment planning in breast cancer.[1] Ki-67 is a nuclear protein expressed in all active phases of the cell cycle except the resting phase (G0). It has become one of the most widely used immunohistochemical markers for evaluating tumor proliferative activity.[2] The proportion of tumor cells expressing Ki-67, known as the Ki-67 labeling index, serves as a quantitative measure of cell proliferation. A higher Ki-67 index generally indicates increased mitotic activity and aggressive tumor behavior. Assessment of Ki-67 has been incorporated into several prognostic and predictive models, and it is used to aid in the molecular subtyping of breast cancer, particularly in differentiating luminal A from luminal B subtypes.[2] The concept of assessing proliferative activity in breast tumors has evolved over decades. Early studies used methods such as thymidine labeling index and flow cytometric S-phase fraction to quantify proliferation. However, these techniques required fresh or frozen tissue, limiting their routine applicability. The development of the monoclonal antibody Ki-67 revolutionized proliferation assessment, enabling accurate in situ detection of dividing cells in formalin-fixed, paraffin-embedded tissues.[3-5] Since then, Ki-67 immunohistochemistry has become a mainstay in histopathology laboratories due to its relative simplicity, reproducibility, and cost-effectiveness compared with molecular assays.[3,4] Histological tumor grade, determined by systems such as the Nottingham modification of the Scarff-Bloom-Richardson method, remains a key prognostic factor in breast carcinoma. It reflects the degree of tumor differentiation by evaluating tubule formation, nuclear pleomorphism, and mitotic count. Tumor grade is strongly associated with disease outcome, with high-grade tumors generally exhibiting poorer prognosis and higher recurrence rates. Among its components, mitotic count is directly linked to proliferative activity, suggesting a biological relationship between histological grade and Ki-67 expression. Indeed, tumors with higher grades often display higher Ki-67 labeling indices, indicating that both measures capture aspects of the same underlying biological process—tumor cell proliferation.[5] While histological grading is an established prognostic tool, it is inherently subjective, with inter-observer variability affecting reproducibility. Ki-67, being a quantifiable biomarker, offers the potential to complement and refine prognostic assessment. Several studies have explored the correlation between Ki-67 and tumor grade, reporting a significant positive association between the two parameters. High-grade tumors tend to have higher Ki-67 indices, whereas low-grade tumors usually exhibit lower proliferative activity. This correlation reinforces the role of Ki-67 as a surrogate marker of histological grade and supports its use in prognostic stratification, especially in cases where grading may be equivocal or borderline.[6,7] Beyond its prognostic value, Ki-67 is increasingly recognized for its predictive role in breast cancer management. It can help identify patients who are more likely to benefit from adjuvant chemotherapy, as rapidly proliferating tumors often respond better to cytotoxic agents. In hormone receptor-positive, HER2-negative breast cancers, Ki-67 levels are used to distinguish between luminal A and luminal B molecular subtypes, with the latter characterized by higher proliferation and poorer prognosis.[2] Multigene assays such as Oncotype DX and MammaPrint incorporate proliferation-related genes, including those related to Ki-67, further validating its clinical relevance.[6,8] However, these genomic assays are costly and not widely accessible in resource-limited settings, making Ki-67 immunohistochemistry a practical alternative. Genomic and molecular profiling have significantly advanced breast cancer classification, but immunohistochemical markers such as Ki-67 continue to serve as essential, cost-effective surrogates for tumor biology. In settings where genomic assays are unavailable, Ki-67, along with estrogen receptor, progesterone receptor, and HER2 status, provides a reliable basis for molecular subtype assignment.[7] Furthermore, Ki-67 has been shown to reflect the “hallmarks of cancer” described in molecular oncology, including sustained proliferative signaling and evasion of growth suppression.[8] Despite its widespread use, the assessment of Ki-67 is not without challenges. Variability in pre-analytical, analytical, and interpretive factors can affect results, leading to inconsistent reporting between laboratories. The International Ki-67 in Breast Cancer Working Group has issued updated recommendations to standardize assessment, including guidance on tissue handling, staining protocols, scoring methods, and reporting formats.[2] These efforts aim to improve inter-observer reproducibility and facilitate broader adoption of Ki-67 in clinical practice. The relationship between Ki-67 and tumor grade is of particular interest because both are proliferation-related parameters obtained through routine pathological evaluation. Ki-67 provides an objective measure of proliferative fraction, whereas tumor grade offers a morphological context to tumor aggressiveness. Correlating these two parameters can yield important insights into tumor biology, enhance prognostic accuracy, and potentially inform treatment decisions. A strong correlation would support the use of Ki-67 as a complementary or even surrogate marker for histological grading in certain scenarios. In addition, understanding this correlation can have implications for tailoring treatment. For instance, patients with low-grade tumors but unexpectedly high Ki-67 may require closer surveillance or consideration for more aggressive therapy. Conversely, high-grade tumors with low Ki-67 could suggest a more indolent course than expected, possibly influencing treatment de-escalation in selected cases. Such nuanced interpretation aligns with the trend toward personalized medicine in oncology, where treatment

is increasingly based on a composite of clinical, histological, and molecular features rather than on single parameters. Given the clinical importance of both Ki-67 and histological grade, evaluating their relationship in a defined patient cohort can contribute to more precise prognostication and better-informed treatment planning. In resource-limited settings, where advanced molecular tests are often unavailable, a robust correlation between these two parameters could strengthen the utility of conventional pathology-based prognostic tools.

MATERIALS AND METHODS

This cross-sectional observational study was conducted at tertiary Care Hospital. A total of 70 consecutive cases of invasive breast carcinoma diagnosed on histopathological examination were included. Ethical approval was obtained from the Institutional Ethics Committee, and informed consent was obtained from all participants.

Inclusion Criteria

- Female patients with histologically confirmed invasive breast carcinoma.
- Adequate tissue sample available for immunohistochemical (IHC) analysis.
- No prior chemotherapy, radiotherapy, or hormonal therapy before biopsy or surgery.

Exclusion Criteria

- Patients with recurrent breast carcinoma.
- Cases with insufficient tumor tissue or poor tissue preservation.
- Metastatic lesions from non-breast primaries.

Histopathological Evaluation: Formalin-fixed, paraffin-embedded (FFPE) tissue blocks from breast carcinoma specimens were retrieved for analysis. Hematoxylin and eosin (H&E)-stained sections of 4 µm thickness were prepared for morphological examination under a light microscope. Tumor grading was carried out according to the Nottingham modification of the Scarff-Bloom-Richardson (SBR) grading system. This method evaluates three histological parameters: tubule formation, nuclear pleomorphism, and mitotic count. Each parameter was assigned a score, and the total score was used to classify tumors into Grade I (well-differentiated), Grade II (moderately differentiated), or Grade III (poorly differentiated).

Immunohistochemistry for Ki-67

Sections of 4 µm thickness were cut from FFPE tissue blocks and mounted on poly-L-lysine-coated glass slides. These slides were deparaffinized in xylene and rehydrated through graded alcohol series. Antigen retrieval was performed using citrate buffer (pH 6.0) in a microwave oven for 15 minutes at 800 W. Endogenous peroxidase activity was blocked by treating the sections with 3% hydrogen peroxide for 10 minutes. The slides were then incubated with the primary antibody against Ki-67 (clone MIB-1, [manufacturer], dilution 1:[x]) for one hour at room temperature. Detection was achieved using a polymer-based detection system ([e.g., Dako EnVision]) with diaminobenzidine (DAB) as the chromogen. Hematoxylin was used for counterstaining. Appropriate positive and negative controls were included with each batch to ensure staining reliability.

Scoring of Ki-67

The Ki-67 labeling index (LI) was assessed by counting the percentage of positively stained tumor cell nuclei among at least 500 invasive tumor cells in areas showing the highest nuclear staining activity ("hot spots"). Counting was performed using high-power fields (×400 magnification). The Ki-67 LI was documented as a continuous variable expressed in percentage. For analytical purposes, the results were further categorized into three proliferation groups: low proliferation (<15%), intermediate proliferation (15–30%), and high proliferation (>30%).

Statistical Analysis

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The correlation between Ki-67 LI and tumor grade was assessed using Spearman's rank correlation coefficient. Associations between categorical variables were tested with the Chi-square test or Fisher's exact test where applicable. A p-value <0.05 was considered statistically significant.

RESULTS

[Table 1] shows the age-wise distribution of the 70 patients included in the study. The most common age group was 41–50 years, comprising 22 patients (31.43%), followed by 51–60 years with 18 patients (25.71%). Patients aged more than 60 years accounted for 16 cases (22.86%), while the youngest group, ≤40 years, represented 14 cases (20.00%). This indicates that invasive breast carcinoma in this study population was most frequently diagnosed in middle-aged women, though cases spanned across all adult age groups.

[Table 2] presents the histological grading of the tumors according to the Nottingham modification of the Scarff-Bloom-Richardson system. The majority of cases were Grade III tumors (30 cases, 42.86%) (Fig.1), indicating poor differentiation. Grade II tumors (moderately differentiated) (Fig.2) were seen in 28 cases (40.00%), while Grade I (well-differentiated) tumors were the least common, occurring in 12 cases (17.14%). This distribution suggests a predominance of higher-grade tumors in the study cohort.

[Table 3] outlines the distribution of Ki-67 proliferation index categories. A high Ki-67 index (>30%) (Fig.3) was observed in 42 cases (60.00%), making it the most common category, followed by the intermediate group (15–30%) (Fig.4) with 18 cases (25.71%). Only 10 patients (14.29%) had a low Ki-67 index (<15%) (Fig.5). The predominance of high proliferation indices suggests a trend toward

biologically more aggressive tumors in this population.

[Table 4] examines the correlation between tumor grade and Ki-67 proliferation category. Among Grade I tumors, most cases (58.33%) fell into the low Ki-67 group, and only one case (8.33%) showed a high Ki-67 index. In contrast, Grade II tumors showed a shift toward higher proliferation, with 53.57% in the high Ki-67 category. The pattern was most striking in Grade III tumors, where 86.67% had a high Ki-67 index, and none were in the low Ki-67 group. The association between tumor grade and Ki-67 index was found to be statistically significant ($p < 0.001$, Chi-square test), indicating that higher-grade tumors tend to have a higher proliferation rate.

[Table 5] provides the mean Ki-67 labeling index across the tumor grades. The mean Ki-67 index increased progressively with tumor grade: Grade I had a mean of 12.45% (SD 3.28), Grade II had 32.86% (SD 6.14), and Grade III had 54.73% (SD 9.21). The difference in mean Ki-67 indices among the three grades was statistically significant ($p < 0.001$, one-way ANOVA test). This finding reinforces the strong positive correlation between tumor grade and cellular proliferation as measured by Ki-67. Overall, the results demonstrate a clear relationship between histological tumor grade and Ki-67 proliferation index, with higher tumor grades showing significantly higher Ki-67 expression, both in categorical and mean value analyses.

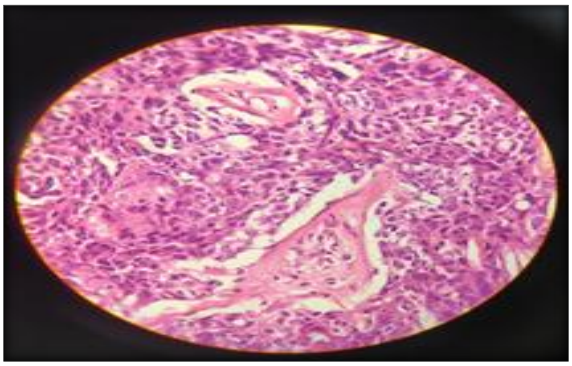

Figure 1: Invasive Ductal Carcinoma (Grade III, H&E 400X)

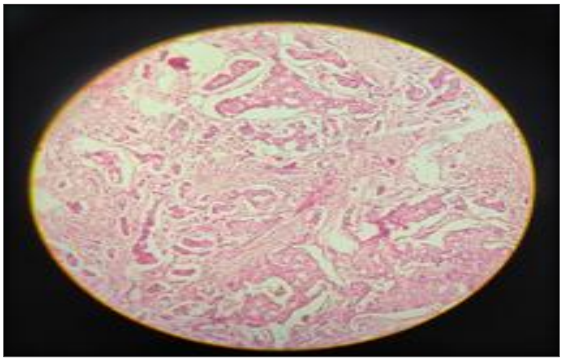

Figure 2: Invasive Ductal Carcinoma (Grade II, H&E 100X)

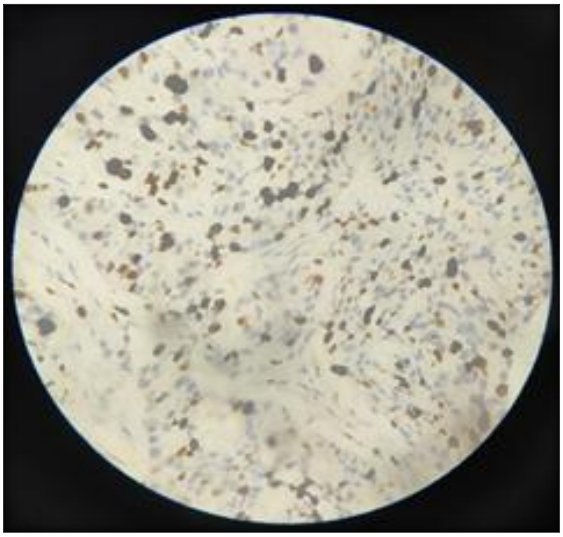

Figure 3: Ki67 proliferation index – 50-60% (IDC – Grade III)

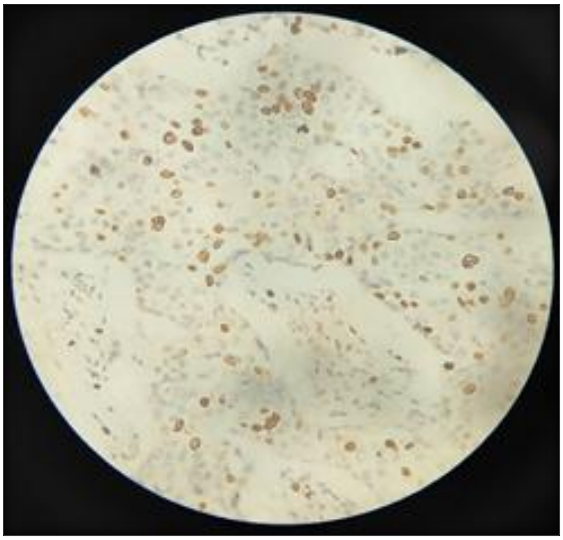

Figure 4: Ki67 proliferation index – 20-25% (IDC - Grade II)

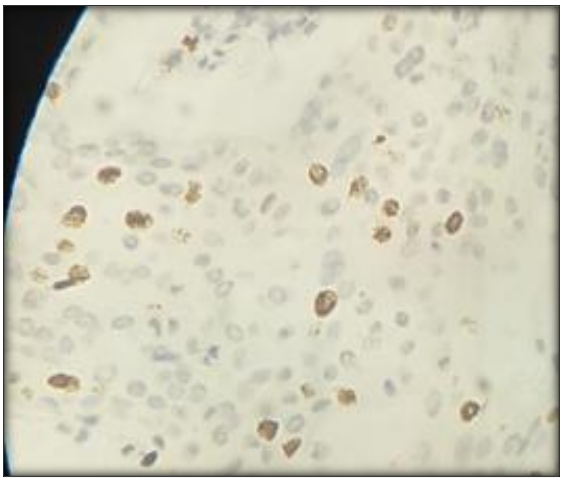

Figure 5: Ki67 proliferation index – 8-10% (IDC - Grade I)

Table 1: Distribution of Patients by Age Group

Age Group (years)	Number of Patients (n=70)	Percentage (%)
≤40	14	20.00
41–50	22	31.43
51–60	18	25.71
>60	16	22.86
Total	70	100.00

Table 2: Distribution of Tumor Grades

Tumor Grade	Number of Patients (n=70)	Percentage (%)
Grade I	12	17.14
Grade II	28	40.00
Grade III	30	42.86
Total	70	100.00

Table 3: Distribution of Ki-67 Proliferation Index Categories

Ki-67 Category	Number of Patients (n=70)	Percentage (%)
Low (<15%)	10	14.29
Intermediate (15–30%)	18	25.71
High (>30%)	42	60.00
Total	70	100.00

Table 4: Correlation Between Tumor Grade and Ki-67 Category

Tumor Grade	Low Ki-67 (<15%)	Intermediate (15–30%)	High (>30%)	Total	p-value
Grade I	7 (58.33%)	4 (33.33%)	1 (8.33%)	12	
Grade II	3 (10.71%)	10 (35.71%)	15 (53.57%)	28	
Grade III	0 (0.00%)	4 (13.33%)	26 (86.67%)	30	
Total	10 (14.29%)	18 (25.71%)	42 (60.00%)	70	<0.001

Table 5: Mean Ki-67 Labeling Index Across Tumor Grades

Tumor Grade	Mean Ki-67 (%)	Standard Deviation (SD)	p-value
Grade I	12.45	3.28	
Grade II	32.86	6.14	
Grade III	54.73	9.21	
Overall	—	—	<0.001

DISCUSSION

In this study, invasive breast carcinoma clustered in middle age, with 31.43% of patients aged 41–50 years, followed by 25.71% aged 51–60 years, while ≤40 and >60 years accounted for 20.00% and 22.86%, respectively. This middle-age peak is broadly consistent with regional series that place the highest burden around the peri-menopausal decades, as noted by Madani et al. (2016),[10] and aligns with contemporary reviews linking age patterns to screening uptake and tumor biology discussed by Finkelman et al. (2023).[11] While some datasets show a slightly older peak in high-income settings, our distribution fits reports from mixed or tertiary-care cohorts where symptomatic presentation skews younger than population-screening cohorts, echoing the context described by Finkelman et al. (2023).[11]

In this study, high-grade (Grade III) tumors predominated (42.86%), with Grade II at 40.00% and Grade I at 17.14%. A high share of poorly differentiated tumors mirrors findings from tertiary centers serving symptomatic populations, similar to observations by Abubakr et al. (2024),[12] who reported a notable prevalence of Grade III cancers, and aligns with molecular-pathology perspectives that associate aggressive biology with higher grade in referral cohorts as discussed by Tekin et al. (2024).[13] Compared with series enriched by screening, where Grade I proportions are often higher, our grade distribution suggests later presentation and/or intrinsically more proliferative disease—again consistent with the referral-bias explanations in Abubakr et al. (2024).[12]

In this study, a high Ki-67 index (>30%) was most common (60.00%), followed by intermediate (15–30%) at 25.71% and low (<15%) at 14.29%. This predominance of high proliferation parallels reports linking Ki-67 enrichment to aggressive clinicopathological features and chemotherapy sensitivity, as emphasized by Rais et al (2024).[14] Cohorts examined by Madani et al,[10] (2016) also associated higher Ki-67 with adverse prognostic factors, and narrative syntheses in Finkelman et al,[11] (2023) outline similar ranges in tertiary settings. Our distribution therefore falls within the pattern of hospital-based series rather than screening-led cohorts, where low-to-intermediate Ki-67 fractions can be larger.

In this study, Ki-67 rose stepwise with grade: among Grade I cancers, 58.33% were low Ki-67 and only 8.33% were high; Grade II shifted toward higher proliferation (53.57% high); and Grade III was overwhelmingly high Ki-67 (86.67%), with no low Ki-67 cases. The association was statistically significant ($p < 0.001$, chi-square). This strong monotonic relationship mirrors the pattern documented by Abubakr et al,[12] (2024) and the

prognostic linkage summarized by Madani et al,[10] (2016) and it fits the biological rationale articulated by Finkelman et al,[11] (2023) that histologic de-differentiation travels with heightened mitotic and proliferative activity. The clinical implication—greater likelihood of chemosensitivity but poorer baseline prognosis in high-grade, high-Ki-67 tumors—is concordant with treatment-response data discussed by Rais et al (2024).[14]

In this study, mean Ki-67 increased progressively from 12.45% in Grade I to 32.86% in Grade II and 54.73% in Grade III, with a significant overall difference ($p < 0.001$, ANOVA). This gradient aligns with multi-modality and biomarker-integrated work showing that proliferative markers scale with adverse histology. For instance, imaging-pathology efforts to predict or mirror Ki-67—such as ultrasound-based models in Xing et al (2024),[15] diffusion/perfusion MRI composites in Zhang J et al (2024),[16] and peritumoral MRI signatures in Zhao et al,[17] (2024)—all build on the premise that higher-grade biology carries higher proliferation. Broader omics-correlates of aggressive phenotypes summarized by Wang et al,[18] (2024) also fit our stepwise increase, reinforcing Ki-67 as a central readout of tumor kinetics across grades.

CONCLUSION

In this study, a strong and statistically significant positive correlation was observed between histological tumor grade and Ki-67 proliferation index in invasive breast carcinoma. Higher tumor grades were associated with markedly higher Ki-67 expression, both in categorical and mean value analyses, indicating more aggressive tumor biology. These findings highlight the value of Ki-67 as an important prognostic marker that can complement histological grading in patient risk stratification and treatment planning, especially in tertiary-care settings where high-grade, rapidly proliferating tumors are prevalent.

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