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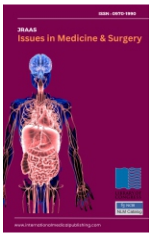

Research Article

Section: Pathology

Evaluation of P-53 & E-Cadherin Status in Breast Carcinoma & Its Correlation with Histological Grading

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HIGHLIGHTS

- p53 correlates with higher grades
- E-Cadherin loss indicates aggressiveness
- Combined markers improve diagnostic accuracy
- Ductal carcinoma shows p53 positivity
- Lobular carcinoma shows E-Cadherin

Key Words:
Breast carcinoma
p53
E-Cadherin
Histological grading
Immunohistochemistry
Nottingham grading system
Prognostic markers

ABSTRACT

Introduction: Breast carcinoma is the most frequently diagnosed malignancy among women worldwide and a leading cause of cancer-related mortality. Although histological grading remains important for prognostic assessment, variability in tumor behavior within similar grades necessitates the evaluation of additional molecular biomarkers. **Aim & Objectives:** To evaluate the expression of p53 and E-Cadherin in breast carcinoma and to correlate their expression with histological subtype and tumor grade. **Material & Methods:** This study included 100 histopathologically confirmed cases of breast carcinoma. Immunohistochemical analysis for p53 and E-Cadherin was performed on paraffin-embedded tissue sections. Histological grading was carried out using the Modified Bloom Richardson (Nottingham) grading system. The expression patterns of both markers were correlated with histological subtype and grade. **Results:** p53 positivity was observed in 58% of cases and showed a significant association with invasive ductal carcinoma and higher tumor grades. Altered E-Cadherin expression, including partial and complete loss, was noted in a considerable number of cases, with complete loss predominantly seen in invasive lobular carcinoma. Increasing loss of E-Cadherin expression was significantly associated with higher histological grades. Combined evaluation of p53 positivity and E-Cadherin loss demonstrated a stronger correlation with tumor grade and better diagnostic performance compared to individual markers. **Conclusion:** The combined assessment of p53 and E-Cadherin provides better insight into tumor aggressiveness and correlates significantly with histological grading. Integration of these molecular markers with routine histopathology may improve prognostic stratification in breast carcinoma.

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INTRODUCTION

Breast carcinoma remains one of the most significant public health challenges in contemporary oncology due to its high global incidence, biological heterogeneity, and substantial contribution to cancer-related mortality among women. It is the most frequently diagnosed nonskin malignancy in females worldwide and continues to be the leading cause of cancer-related death among women in many countries [1]. According to the GLOBOCAN 2020 estimates, breast cancer accounted for approximately 2.3 million new cases and nearly 685,000 deaths globally, reflecting both its growing prevalence and the persistent burden it imposes on healthcare systems [2]. Although advancements in screening programs, imaging techniques, molecular diagnostics, and targeted therapies have improved early detection and survival outcomes in high-income regions, significant disparities persist, particularly in low and middle-income countries where late-stage presentation remains common [1,2].

Breast cancer is not a single disease entity but rather a biologically heterogeneous group of malignancies characterized by diverse morphological, molecular, and clinical features. This heterogeneity explains the wide variation in disease progression, metastatic behavior, and therapeutic response. Morphologically, breast carcinoma encompasses several histological subtypes, including invasive ductal carcinoma, invasive lobular carcinoma, mucinous carcinoma, medullary carcinoma, and metaplastic carcinoma. Molecularly, it is classified into luminal A, luminal B, HER2-enriched, and triple-negative subtypes based on receptor expression and gene profiling. Clinically, these subtypes demonstrate differing patterns of growth, recurrence, and survival [3]. Such diversity underscores the limitations of relying solely on conventional histopathological parameters for prognostic assessment.

Prognostic evaluation in breast carcinoma traditionally incorporates tumor size, lymph node involvement, histological type, and histological grade, typically assessed using the Nottingham grading system, which evaluates tubule formation, nuclear pleomorphism, and mitotic activity. While these parameters remain fundamental, considerable variability in clinical outcomes is observed even among tumors of similar stage and grade, suggesting that additional molecular determinants influence tumor behavior [4].

Among the molecular alterations implicated in breast cancer progression, dysfunction of the tumor suppressor gene p53 and disruption of E-cadherin–mediated cell adhesion pathways have attracted substantial attention. The TP53 gene, located on

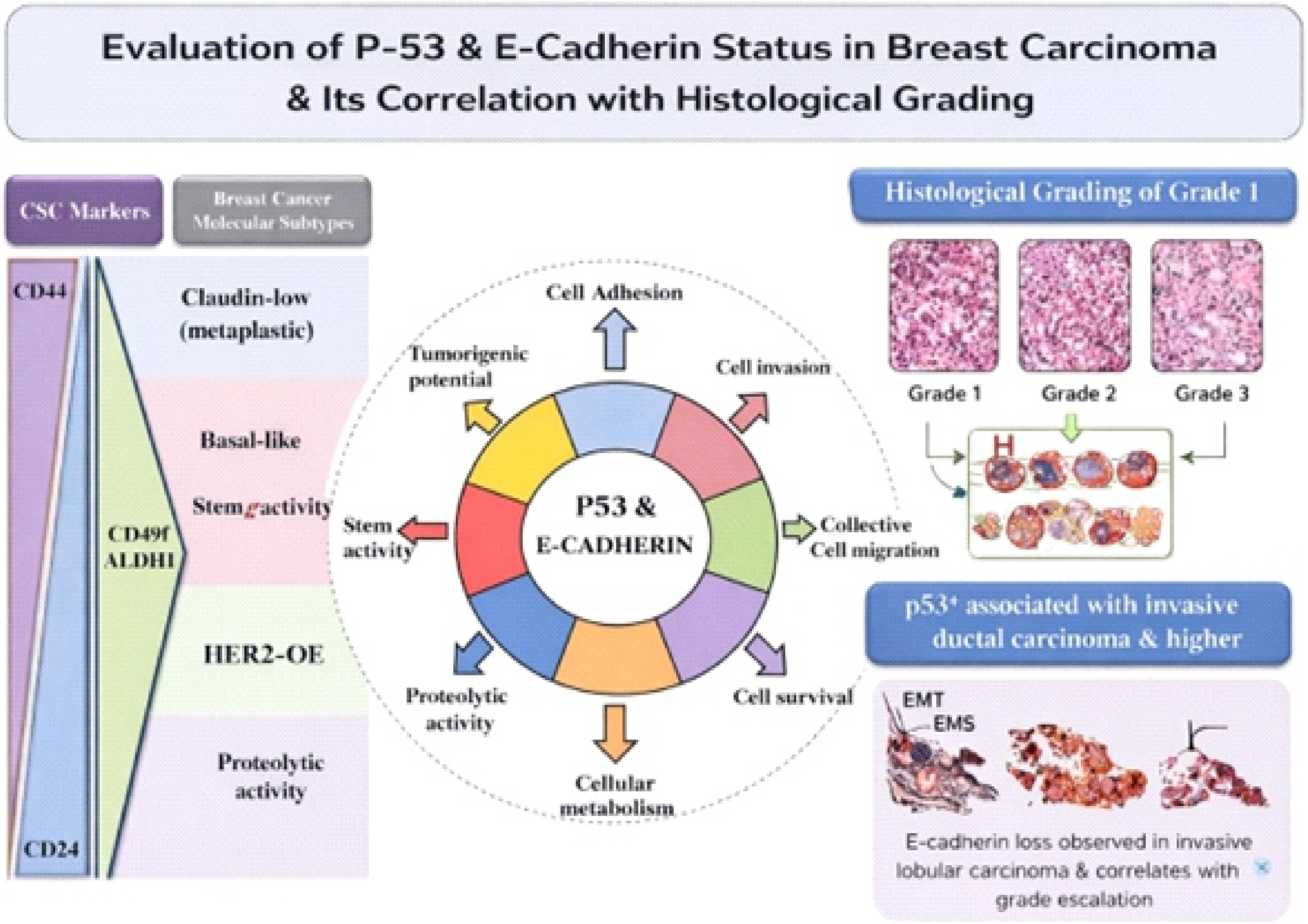

Figure 1: Schematic showing p53 and E-Cadherin roles in breast carcinoma and their correlation with tumor grade and progression. Adopted from [21].

chromosome 17p13.1, encodes a nuclear phosphoprotein that regulates cell cycle arrest, DNA repair, apoptosis, and genomic stability. Mutations in p53 are among the most frequent genetic abnormalities in breast carcinoma and are strongly associated with high-grade tumors, poor differentiation, aggressive clinical behavior, and unfavorable prognosis [5]. Particularly in HER2-enriched and triple-negative breast cancers, mutant p53 often acquires gain-of-function properties that promote tumor progression and therapeutic resistance [6].

E-cadherin, encoded by the CDH1 gene on chromosome 16q22.1, is a calcium-dependent transmembrane glycoprotein critical for epithelial cell-cell adhesion and maintenance of tissue architecture. Loss or reduction of E-cadherin expression disrupts intercellular cohesion, facilitates epithelial mesenchymal transition (EMT), and enhances tumor invasiveness and metastatic potential [7]. In invasive lobular carcinoma, CDH1 inactivation is considered a hallmark molecular event, whereas in invasive ductal carcinoma, altered or aberrant E-cadherin expression may contribute to collective invasion and disease aggressiveness [8].

The interplay between p53 dysfunction and E-cadherin loss is particularly relevant in the context of EMT, a biological process that enables epithelial cells to acquire mesenchymal characteristics, increased motility, and invasive capacity. EMT has been implicated in tumor dedifferentiation, metastatic dissemination, and resistance to therapy. Mutant p53 may promote EMT through transcriptional repression of adhesion molecules, including E-cadherin, thereby accelerating tumor progression [6,9]. Consequently, simultaneous evaluation of p53 and E-cadherin expression may provide deeper insight into tumor biology and prognostic stratification.

Given the biological significance of p53 and E-cadherin in regulating genomic stability, cellular differentiation, and metastatic potential, correlating their expression with histological grading may enhance prognostic precision beyond conventional morphological assessment. Integrating molecular markers with routine histopathology may help refine risk stratification, guide therapeutic decisions, and contribute to improved individualized management of breast carcinoma. Role of p53 tumor suppressor gene and E-cadherin cell adhesion molecule in breast carcinoma and their correlation with tumor grade and progression (**Figure 1).**

MATERIALS & METHODS

This cross-sectional study was conducted over a period of two years in the Department of Pathology, GSVM Medical College, Kanpur, in collaboration with the Department of Surgery and LLR & Associated Hospitals. A total of 100 histopathologically confirmed cases of breast carcinoma were included. All lumpectomy, trucut biopsy, simple mastectomy, and modified radical mastectomy specimens received during the study period were evaluated. Consecutive (non-probability) sampling was used, & cases meeting the inclusion and exclusion criteria were enrolled after obtaining written informed consent. Male breast malignancies, benign breast lesions, and patients receiving chemotherapy or radiotherapy were excluded.

Sample size was calculated using the formula $n = Z^2P(1–P)/d^2$, assuming a 95% confidence interval (Z=1.96), expected prevalence of p53 positivity at 50%, and 10% precision, yielding a minimum size of 96; this was rounded to 100 to account for inadequate samples or dropouts.

Specimens were grossed, processed, and stained with hematoxylin and eosin (H&E) as per standard protocols. Histological grading was performed using the Modified Bloom–Richardson (Nottingham) grading system, based on tubule formation, nuclear pleomorphism, and mitotic count. Immunohistochemistry (IHC) was performed on 4 µm paraffin-embedded sections for p53 and E-cadherin after antigen retrieval using citrate buffer (pH 6.0). A marker was considered positive when ≥10% of tumor cells showed staining.

Data including age, menstrual and lactation history, lymph node status, and radiological findings were collected. Statistical analysis was performed using SPSS/GraphPad Prism. Associations between biomarker expression and histological grade were assessed using Chi-square or Fisher's exact test. ROC curve analysis evaluated diagnostic performance. A p-value <0.05 was considered statistically significant.

The study adhered to the principles of the Declaration of Helsinki and was approved by the institutional ethics committee.

RESULT

A total of 100 histopathologically confirmed cases of breast carcinoma were evaluated for p53 and E-Cadherin expression and correlated with histological subtype and tumor grade.

p53 Expression

p53 positivity was observed in 58% (58/100) of cases, indicating frequent alteration of the p53 tumor suppressor pathway in breast carcinoma.

As shown in **Table 1**, p53 expression demonstrated a significant association with histological subtype (p = 0.04). Among invasive ductal carcinoma (IDC) cases (n = 92), 56 were p53 positive, whereas only 2 of 8 invasive lobular carcinoma (ILC) cases showed positivity, suggesting greater involvement of p53 alterations in ductal tumors. Correlation with tumor grade (**Table 2**) showed increasing p53 positivity with higher grade, with Grade III tumors demonstrating the highest frequency. The association was borderline significant (p = 0.05), indicating a possible relationship between p53 overexpression and tumor aggressiveness. Representative immunohistochemical staining is shown in **Figure 2** (p53 Positive - High Power View) & **Figure 3** (p53 Negative Expression), demonstrating strong nuclear staining in positive cases and absence of staining in negative tumors. Illustrative staining patterns are shown in **Figure 4** (Complete Loss of E-Cadherin Expression) & **Figure 5** (E-Cadherin Expression in Invasive Ductal Carcinoma), highlighting the contrast between preserved membranous staining and complete loss.

Combined Marker Analysis

The combined marker pattern (p53 positivity with E-Cadherin loss) showed a statistically significant association with tumor grade (**Table 5**, p = 0.02). Grade III tumors demonstrated the highest frequency of this combined expression pattern, suggesting synergistic contribution of genomic instability and adhesion loss in tumor progression. Diagnostic performance analysis (**Table 6**) showed that the combined marker analysis had the highest discriminatory ability (AUC = 0.84) compared to p53 (AUC = 0.72) and E-Cadherin (AUC = 0.78). The ROC curve comparison is illustrated in **Figure 6** (ROC Curve Comparison of Markers).

Table 1. p53 expression in IDC and ILC

Subtype	p53 +	p53 -	Total	p-value
IDC	56	36	92	0.04
ILC	2	6	8	
Total	58	42	100	

Table 2. Association of p53 Expression with Histological Grade

Grade	p53 +	p53 -	Total	p-value
I	7	8	15	0.05
II	28	23	51	
III	23	11	34	
Total	58	42	100	

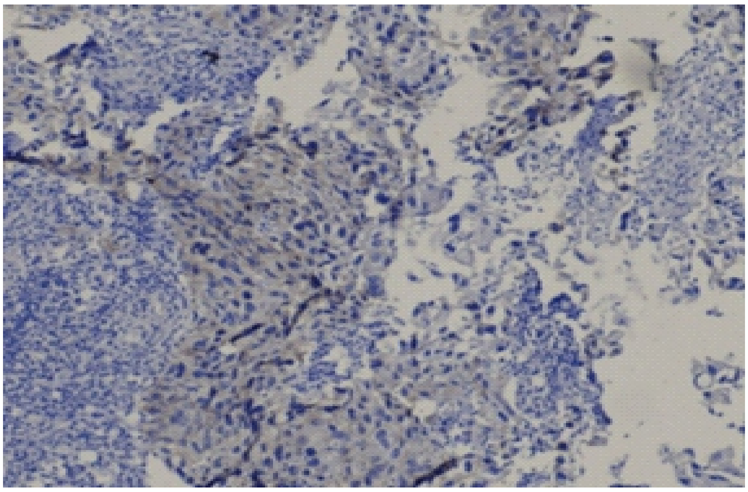

Figure 2: p53 Positive - High Power View

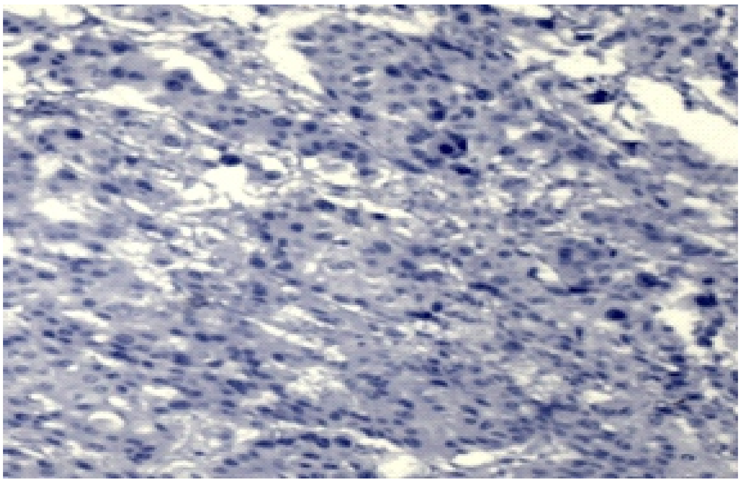

Figure 3: p53 Negative Expression

Table 3. Distribution of E-Cadherin Expression Across Age Groups

Age	Preserved	Partial Loss	Complete Loss	Total	p-value
<40	14	14	4	32	0.88
41-50	9	9	1	19	
51-60	11	10	1	22	
>60	12	13	2	27	
Total	46	46	8	100	

Table 4. Distribution of E-Cadherin Expression According to Histological Subtype

Subtype	Preserved	Partial Loss	Complete Loss	Total	p-value
IDC	42	46	4	92	<0.001
ILC	0	2	6	8	
Total	**42**	**48**	**10**	**100**	

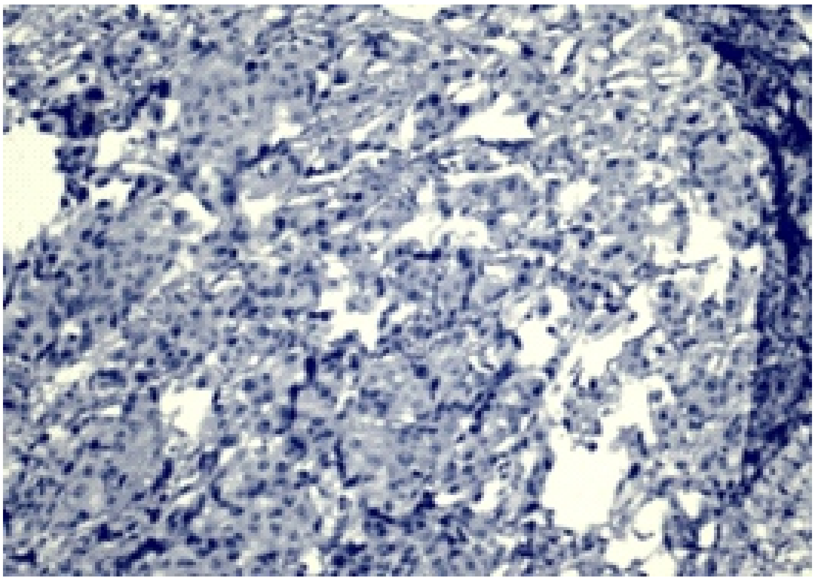

Figure 4: Complete Loss of E-cadherin Expression

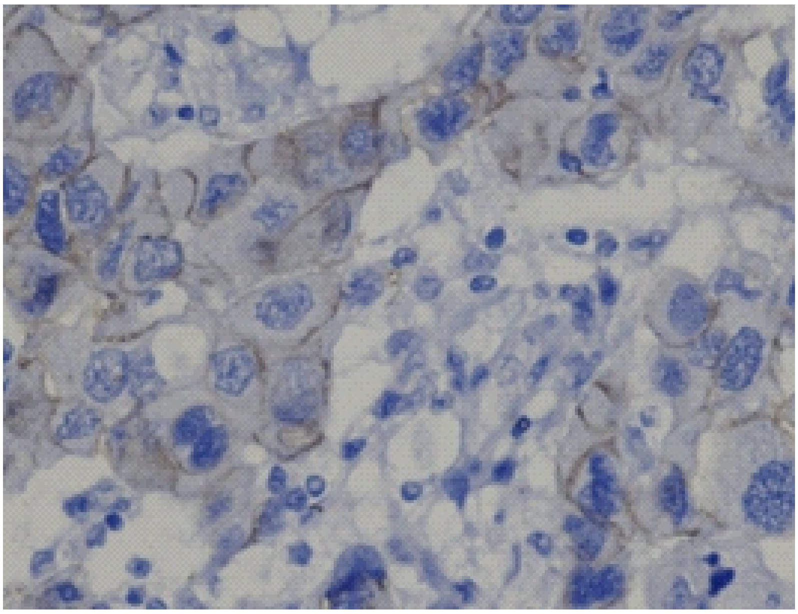

Figure 5: E-Cadherin Expression in Invasive Ductal Carcinoma

Table 5. Distribution of Combined Marker Expression (p53 Positivity with E-Cadherin Loss) According to Tumor Grade

Grade	p53+/Loss of E -Cadherin	Others	Total	p-value
I	3	12	15	**0.02**
II	17	34	51	
III	18	16	34	
Total	**38**	**62**	**100**	

Table 6. ROC Curve Comparison of p53, E-Cadherin, and Combined Markers

Marker	AUC	SE	95% CI	p-value
p53	0.72	0.05	0.63–0.81	0.001
E-Cadherin	0.78	0.04	0.69–0.86	<0.001
Combined	0.84	0.03	0.76–0.91	<0.001

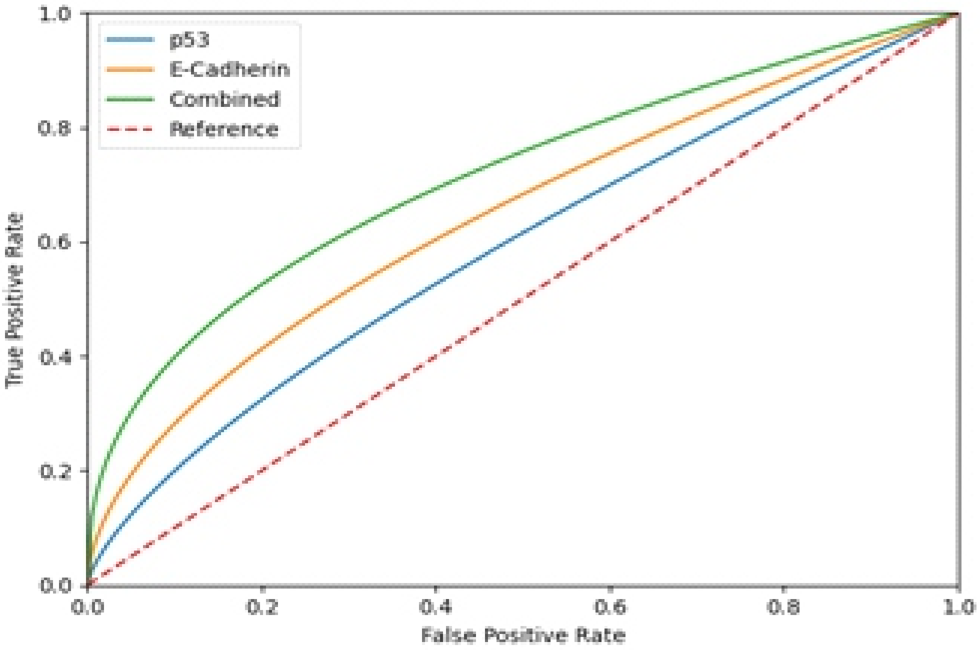

Figure 6. ROC Curve Comparison of p53, E-Cadherin, and Combined Markers

DISCUSSION

Breast carcinoma is a biologically heterogeneous malignancy in which molecular alterations significantly influence tumor differentiation, progression, and clinical outcome. The present study evaluated the expression of p53 and E-Cadherin in 100 histopathologically confirmed cases of breast carcinoma and analyzed their correlation with histological subtype and tumor grade. The findings provide meaningful insight into the relationship between tumor suppressor dysfunction, cell adhesion disruption, and tumor aggressiveness.

In the present study, p53 positivity was observed in 58% of cases. This frequency is consistent with previously published data reporting p53 overexpression in approximately 30–60% of breast carcinomas [10,11]. Nuclear accumulation of p53 protein on immunohistochemistry frequently reflects TP53 gene mutation, leading to loss of tumor suppressor function and genomic instability [12]. The relatively high proportion of p53 positivity in our study supports the established role of p53 alterations in breast carcinogenesis.

A statistically significant association was observed between p53 expression and histological subtype (p = 0.04), with higher positivity in invasive ductal carcinoma (IDC) compared to invasive lobular carcinoma (ILC). Previous molecular studies have demonstrated that TP53 mutations are more frequently encountered in ductal carcinomas, whereas ILC is more commonly associated with CDH1 gene inactivation and lower TP53 mutation rates [13,14]. The lower p53 positivity in ILC cases in our study therefore reflects known biological differences between these two major histological subtypes.

Correlation of p53 expression with histological grade revealed increasing positivity with higher grade tumors, with Grade III tumors demonstrating the highest frequency. Similar findings have been reported in earlier studies showing strong association between p53 mutation, high histologic grade, increased proliferative activity, and adverse prognosis [11,15]. Mutant p53 proteins may also acquire gain-of-function properties that promote tumor progression, invasion, and therapeutic resistance [19]. Thus, the increasing frequency of p53 positivity in higher grades observed in the present study reinforces its association with aggressive tumor behaviour.

E-Cadherin expression analysis demonstrated altered expression in a substantial proportion of tumors. A highly significant association was observed between E-Cadherin expression and histological subtype ($p < 0.001$), with complete loss predominantly seen in invasive lobular carcinoma. This finding is in accordance with established literature identifying CDH1 gene inactivation as a hallmark molecular event in ILC [14,16]. Loss of E-Cadherin disrupts epithelial cell adhesion and results in the characteristic discohesive growth pattern observed in lobular carcinoma.

Increasing loss of E-Cadherin expression was also significantly associated with higher tumor grade ($p = 0.049$). High-grade tumors demonstrated a greater proportion of partial or complete loss, indicating progressive disruption of intercellular adhesion with tumor dedifferentiation. Previous studies have shown that reduced E-Cadherin expression correlates with epithelial-to-mesenchymal transition (EMT), increased invasiveness, and poor clinical outcomes [17,18]. Loss of adhesion facilitates tumor cell migration and metastatic dissemination, contributing to tumor aggressiveness.

The combined marker analysis revealed a statistically significant association between p53 positivity with E-Cadherin loss and higher tumor grade ($p = 0.02$). Grade III tumors exhibited the highest frequency of this combined expression pattern. Biologically, this observation is plausible, as mutant p53 has been shown to promote EMT and suppress E-Cadherin expression through transcriptional and non-transcriptional mechanisms [19]. The coexistence of p53 dysfunction and E-Cadherin loss may therefore accelerate tumor progression by simultaneously promoting genomic instability and enhancing invasive potential.

ROC curve analysis further demonstrated that combined marker evaluation provided superior discriminatory ability (AUC = 0.84) compared to individual markers. Integrative biomarker approaches have been increasingly advocated to improve prognostic stratification beyond conventional histological grading [20]. Although the Nottingham grading system remains a fundamental prognostic tool in breast carcinoma [15], incorporation of molecular markers such as p53 and E-Cadherin may enhance predictive accuracy.

Overall, the present study confirms that p53 overexpression and E-Cadherin loss are associated with aggressive tumor features and higher histological grade. The findings support the concept that breast carcinoma progression involves both disruption of tumor suppressor pathways and impairment of cell adhesion mechanisms. Combined evaluation of p53 and E-Cadherin enhances diagnostic discrimination and may improve risk stratification in routine histopathological

CONCLUSION

The present study evaluated the expression of p53 & E-Cadherin in breast carcinoma and correlated these biomarkers with histological grading. p53 overexpression was observed in 58% of cases and demonstrated a significant association with invasive ductal carcinoma and higher tumor grades, suggesting its role in tumor progression and aggressive biological behavior. Increasing p53 positivity in higher-grade tumors reinforces the importance of TP53 pathway alterations in breast cancer dedifferentiation and genomic instability.

E-Cadherin expression analysis revealed altered expression in a substantial proportion of tumors, with complete loss predominantly seen in invasive lobular carcinoma. A significant correlation was observed between E-Cadherin loss and higher histological grade, indicating disruption of intercellular adhesion as tumors become less differentiated and more invasive.

Importantly, combined evaluation of p53 positivity with E-Cadherin loss showed a stronger association with high-grade tumors and demonstrated superior diagnostic performance compared to individual markers. This suggests that integrating molecular markers reflecting tumor suppressor dysfunction and adhesion loss enhances prognostic discrimination beyond conventional histopathological grading alone.

In conclusion, assessment of p53 and E-Cadherin status provides valuable adjunctive information in breast carcinoma. Their combined evaluation may improve risk stratification and contribute to a more comprehensive understanding of tumor biology, potentially aiding in individualized patient management and prognostic assessment.

LIMITATIONS & FUTURE PERSPECTIVES

The study's limitations include a single-centre setting, a relatively small sample size, and a short study duration, which may limit the broader applicability of the results. Future studies should incorporate multicentre designs with larger populations to enhance validity, assess long-term outcomes, and investigate advanced diagnostic and management approaches. Such efforts will improve overall patient care and help minimize complications.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical healthcare applications. It emphasizes the importance of translating scientific observations into meaningful improve-ments in patient care, diagnosis, and treatment outcomes. By highlighting real-world relevance, the study contributes to evidence-based medical practice and supports informed clinical decision-making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

p53: Tumor protein 53
E-Cadherin: Epithelial cadherin
IHC: Immunohistochemistry

MBR: Modified Bloom–Richardson
IDC: Invasive ductal carcinoma
ILC: Invasive lobular carcinoma

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AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines.

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CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

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All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

DATA AVAILABILITY

All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a re-asonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in thewriting or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

AUTHOR'S NOTE

This article serves as an important educational tool for the scientific community, offering insights that may inspire future research diretions. However, they should not be relied upon independently when making treatment decisions or developing public health policies.

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