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Immunohistochemical expression of Ki-67 and p53 in triple-negative breast carcinoma and their association with clinicopathological parameters

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Abstract

Background: The current markers Ki-67 and p53 are well-known indicators of the aggressiveness of the tumour biology in a multitude of tumours characterized by less and different drivers and are poorly coordinated with the clinicopathologic presentation of Triple-Negative Breast Carcinoma (TNBC). In this study, the expression of Ki-67 and p53, and their relationship with other clinicopathological parameters was analysed. **Methods:** A total of 120 breast cancers were histologically confirmed triple-negative BCs from which 5 were excluded due to missing data. In this cross-sectional study, 120 breast cancers were included and 5 missing data excluded among histologically confirmed triple-negative BCs. The immunohistochemical expression of Ki-67 labelling index and p53 has been analysed and correlated with: age, tumour size, histological tumour grade, nodal status, stage at diagnosis, lymphovascular invasion, and histological tumour type. Independent predictors of nodal involvement were evaluated by a multivariable logistic regression. **Results:** High expression of ki 67 ($\geq 20\%$) was observed in 85.0% and abnormal expression of p53 in 49.2% of the tumours. High ki-67, tumour size ($p = 0.029$), histological grade ($p < 0.001$) and nodal involvement ($p < 0.001$) and stage ($p = 0.001$) were found to correlate significantly with each other. Grade ($p < 0.001$), nodal involvement ($p < 0.001$) and stage ($p = 0.025$) were significantly related to aberrant p53. Ki-67 levels increased in tumours with aberration in p53. Nodal invasion was independently predicted by both the Ki-67 (OR = 1.49 per 10% increase) and aberrant p53 (OR = 2.66). **Conclusions:** Ki-67 and p53 are shown to give complementary information regarding aggressive clinicopathological characteristics of triple negative breast carcinoma, and can contribute to pathological risk stratification, especially for nodal involvement.

Keywords: Triple-negative breast cancer, Ki-67, p53, Immunohistochemistry, Lymph node

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1. Introduction

The triple-negative breast carcinoma which lacks the receptor for oestrogen, the receptor for progesterone and which is not amplified by herceptin HER2 sub-type represents approximately one-seventh of all breast cancers and is the most aggressive of the breast cancer subtypes (Bianchini *et al.*, 2022; Denkert *et al.*, 2017). It lacks

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expression of the three receptors upon which the endocrine and anti-HER2 agents act, making them ineffective for treatment, and the main treatments that are used for these are targeted chemotherapy and the pathological evaluation of the resected tumour (Garrido-Castro *et al.*, 2019; Yin *et al.*, 2020). In this setting the immunohistochemical markers that report on the biology of the tumour retain a particular value, being inexpensive, widely available and applicable to routine paraffin sections.

Ki-67 is the most established of the proliferation markers. It is a nuclear protein expressed throughout the active phases of the cell cycle but absent from resting cells, so that the proportion of tumour nuclei that stain for it, the labelling index, measures the growth fraction of the tumour (Sun and Kaufman, 2018). A high index is associated with aggressive behaviour and adverse outcome across breast cancer, and in the triple-negative subtype, which is proliferative by nature, the index has been proposed both as a prognostic marker and as a means of stratifying the tumours that are otherwise grouped together (Nielsen *et al.*, 2021; Keam *et al.*, 2011; Wu *et al.*, 2019; Wang *et al.*, 2020).

The p53 protein, the product of the TP53 tumour-suppressor gene, is the second marker of interest. TP53 is the most frequently mutated gene in triple-negative breast cancer, altered in the majority of tumours, and the mutation typically stabilises the protein so that it accumulates to a concentration detectable by immunohistochemistry, the aberrant expression thus serving as a surrogate for the underlying mutation (Cancer Genome Atlas Network, 2012; Shah *et al.*, 2012; Yemelyanova *et al.*, 2011; Duffy *et al.*, 2018). Aberrant p53 has likewise been linked to high grade and to poor outcome, and because the two markers report on different aspects of tumour biology, proliferation and genomic integrity, they may carry complementary information (Kim *et al.*, 2016; Pei *et al.*, 2017).

The reported associations of these two markers with the established clinicopathological features of the disease have, however, been inconsistent. While some studies find that a high Ki-67 index and aberrant p53 accompany high grade, nodal involvement and advanced stage, others find associations with only some of these features or with none, and the two markers are not always examined together in the same cohort with a formal test of their independence (Rana *et al.*, 2024). The value of the markers in routine practice depends on which of these associations are robust, and on whether the markers add to one another, questions best addressed by examining both in a single, uniformly assessed series.

The current study was thus aimed at evaluating the immunohistochemistry expression of Ki-67 and p53 in a series of triple-negative breast carcinomas and their clinicopathological correlation. It aimed to identify how often each parameter (Ki-67 index and aberrant p53 expression) is seen, to test the association of each parameter with age, tumour size, histological grade, nodal status, stage, lymphovascular invasion and histological type, to explore the relationship between the two parameters and to evaluate using logistic regression whether the markers are independent of each other with respect to their ability to predict nodal status.

2. Methodology

2.1. Study design and sample selection

This cross-sectional study examined 120 histologically confirmed triple-negative breast carcinomas resected at the institution. Reporting follows the recommendations for observational studies (Vandenbroucke *et al.*, 2007). Triple-negative status was confirmed by the absence of the oestrogen and progesterone receptors and of HER2 overexpression or amplification, assessed and interpreted according to the prevailing guidelines (Allison *et al.*, 2020; Wolff *et al.*, 2023). Cases that had received neoadjuvant systemic therapy before resection were excluded to avoid treatment-related alterations in tumour morphology and biomarker expression. Cases with insufficient viable tumour tissue for immunohistochemical evaluation were also excluded.

2.2. Clinicopathological assessment

From the pathohistology of tumour placement, clinicopathological data were included: Age, size of the tumour, the histologic type, the histologic grade, lymph-node status, pathological stage and the presence of lymphovascular invasion. Histological grading was done by using a Nottingham combined histological grading system (Elston and Ellis, 1991) and staging was done by an American Joint Committee on Cancer (AJCC) classification, eighth edition (Amin *et al.*, 2017). Tumour size was characterised as T1, T2 or T3; lymph-

node status as negative or positive; and tumour pathological stage as I, II or III. Histological type was classified as invasive ductal carcinoma of no special type (IDC-NST), metaplastic carcinoma or other histological types.

2.3. Immunohistochemistry and scoring

Representative paraffin sections were used for immunohistochemistry, and analyzed using the standard peroxidase technique with appropriate positive and negative controls, where appropriate, for Ki-67 and p53. Labeling was considered to be good when the percent of very clearly labelled cell nuclei was larger than 20 percent in the most active (hyperactive) area of the tumor and was called high (HI) Labelling Index (LI) ([Aziz et al., 2016](#)). A p53 nuclear stain of high level seen in a significant number of tumour cells was interpreted as an aberrant stain and a score of wild-type was obtained if this pattern was not present.

2.4. Statistical analysis

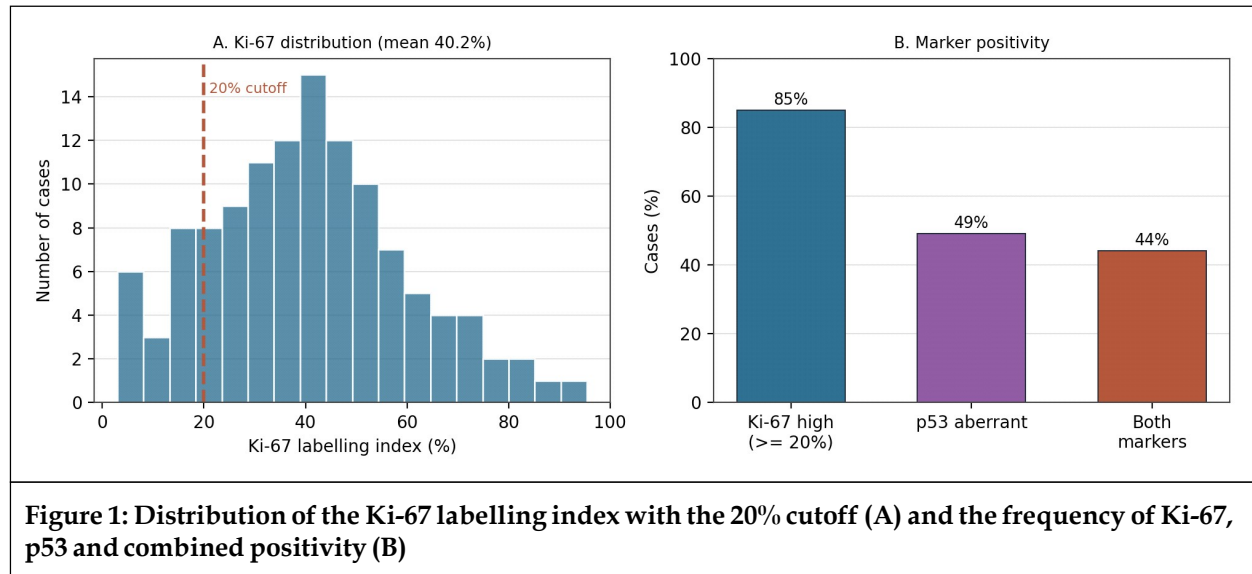
The frequency of high Ki-67 index and aberrant p53 was calculated and the relationship of each parameter with clinicopathological parameters was examined using chi-square test or Fisher exact test if the expected cell numbers were small. Both the association of the two markers and the mean Ki-67 index of the p53-aberrant and wild-type tumours were analyzed by the χ^2 test, and the mean Ki-67 index was compared among histological grades with the Kruskal-Wallis test. The Ki-67 index, p53 status, grade, tumour size and lymphovascular invasion were determined to be independent predictors of nodal involvement by multiple logistic regression. A difference with a probability of < 0.05 was considered significant.

3. Results

3.1. Clinicopathological profile and marker expression

The study included 120 patients with a mean age of 48.1 years; 64 (53.3%) were younger than 50 years and 56 (46.7%) were aged ≥ 50 years. Most of the tumors were T2 (55.0%), histological grade II (60.0%) and no special type of invasive ductal cancer (85.0%). Lymph nodes involvement was noted in 47 cases and in 44 cases with the presence of lymphovascular invasion (39.2% and 36.7% respectively). The majority of cases were in stage III ($n = 11.7\%$), stage II ($n = 39.2\%$) and stage I ($n = 49.2\%$). The mean Ki-67 labelling index was 40.2%, and a high expression index (Hi-C kit; $\geq 20\%$) seen in 102 tumours (85.0%). In 59 cases there was aberrant expression of p53 (49.2%). The clinicopathological profile and distribution of biomarkers is provided in an exhaustive fashion in Table 1 and Figure 1.

Parameter	n (%)	Parameter	n (%)
Age < 50 years	64 (53.3)	Age ≥ 50 years	56 (46.7)
Tumour size T1	38 (31.7)	Tumour size T2	66 (55.0)
Tumour size T3	16 (13.3)	Histological grade I	23 (19.2)
Histological grade II	72 (60.0)	Histological grade III	25 (20.8)
Lymph node positive	47 (39.2)	Lymphovascular invasion	44 (36.7)
Stage I	59 (49.2)	Stage II	47 (39.2)
Stage III	14 (11.7)	IDC-NST	102 (85.0)
Metaplastic carcinoma	10 (8.3)	Other histology	8 (6.7)
Ki-67 high ($\geq 20\%$)	102 (85.0)	p53 aberrant expression	59 (49.2)



3.2. Association of Ki-67 with clinicopathological parameters

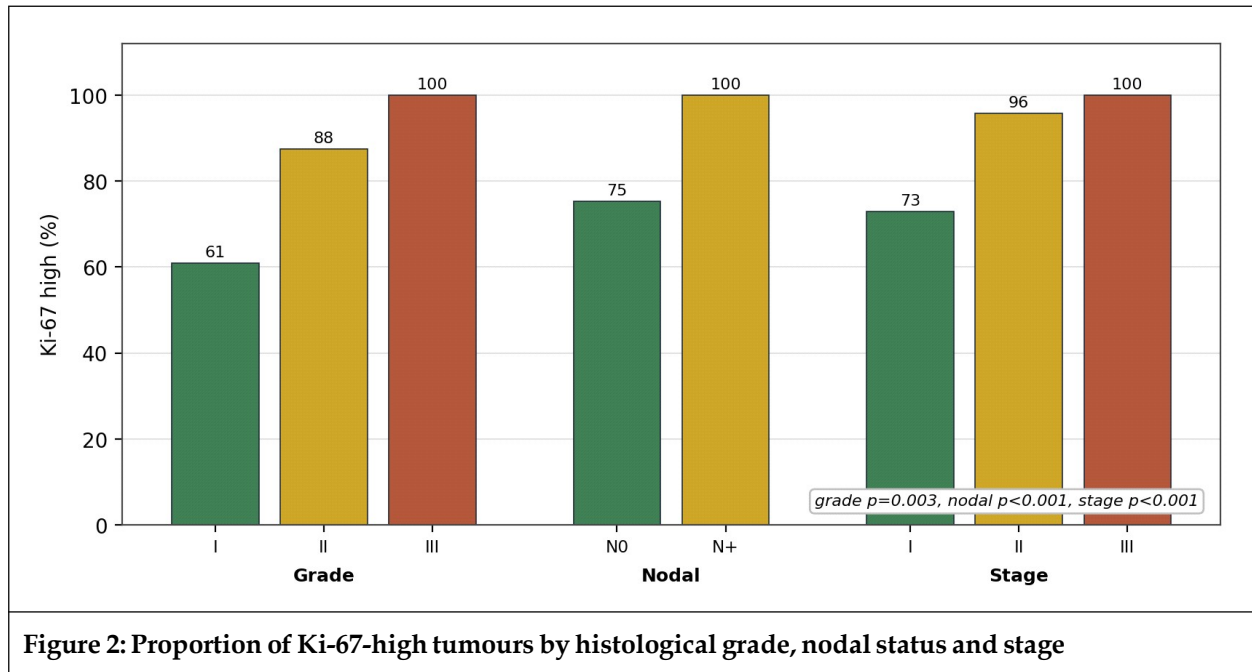
Strong correlation between the expression of Ki-67 with clinicopathological parameters. Slightly more than half of the cases showed high Ki-67 level, and this parameter showed a statistically significant association with several parameters of aggressive tumour behaviour. The percentage of Ki-67-high tumours gradually increased with tumour grading from 74% to 88% and 100% respectively in T2 and T3 tumours ($p = 0.029$). Significant relationship to histological grade was also observed, as 61% of grade I cases showed increased expression of Ki-67, 88% of grade II and 100% of grade III cases ($p < 0.001$). The number of tumours with high Ki-67 expression was significantly different among node-negative (75%) and node-positive (100%) tumours ($p < 0.001$). Similarly, pathological stage (73% to 96% to 100%, $p = 0.001$) was also significant in terms of increase in the frequency. No significant relationships were seen with age ($p = 0.573$), lymphovascular invasion ($p = 0.958$) or histological type ($p = 0.380$) (Table 2 and Figure 2).

Table 2: Association of a high Ki-67 index with clinicopathological parameters (positivity rate shown per category)

Parameter	Ki-67 high by category	p
Age (<50/>= 50)	88%/82%	0.573
Tumour size (T1/T2/T3)	74%/88%/100%	0.029
Histological grade (I/II/III)	61%/88%/100%	<0.001
Lymph node (neg/pos)	75%/100%	<0.001
Lymphovascular invasion	84%/86%	0.958
Stage (I/II/III)	73%/96%/100%	0.001
Histological type	86%/70%/88%	0.380

3.3. Association of p53 with clinicopathological parameters

Histological grade, lymph-node involvement and pathological stage were significantly associated with aberrant p53 expression. The frequency was significantly higher with the increasing histological grades (17% grade I tumours, 50% grade II and 76% grade III tumours, $p < 0.001$). This was also true of Tumour Infiltrating Lymphocytes (TILs) which had a significantly higher prevalence in node positive than node negative tumours ($p < 0.001$) (70% versus 36%). Increased frequency of progression was noted as a function of the stage of the



disease, with 37% in stage I disease, 57% in stage II and 71 % in stage III disease ($p = 0.025$). In contrast, there was no significant association between p53 and age ($p = 0.723$), tumour size ($p = 0.542$), lymphovascular invasion ($p = 0.143$) or histological type ($p = 0.997$). This was confirmed by comparing the mean labelling index of Ki-67 expressed by Ki-67 with histological grade, which also showed being more intense in tumours with a higher grade (Table 3 and Figure 3).

Table 3: Association of aberrant p53 expression with clinicopathological parameters (positivity rate shown per category)

Parameter	p53 aberrant by category	p
Age (<50/> = 50)	47%/52%	0.723
Tumour size (T1/T2/T3)	42%/52%/56%	0.542
Histological grade (I/II/III)	17%/50%/76%	<0.001
Lymph node (neg/ pos)	36%/70%	<0.001
Lymphovascular invasion	43%/59%	0.143
Stage (I/II/III)	37%/57%/71%	0.025
Histological type	49%/50%/50%	0.997

3.4. Relationship between the markers and prediction of nodal involvement

Strong correlation was noted between expression of Ki-67 and p53. The aberrant p53 expressing tumours, compared with the non-aberrant p53, had a higher mean Ki-K67 labelling index (47.9% versus 32.7%). The double positivity of both Parameters was observed in 44.2% of the patient population. There was enrichment of the nodes with positivity for both tumour markers (62% vs. 21% of the remaining tumours). The Ki-67 labelling index was independently associated with nodal involvement with an OR of 1.49 (95% CI 1.12-1.99; $p = 0.006$) for every 10% point increase in the index. The aberrant expression of p53 was also associated with nodal involvement independently (OR = 2.66 (95% CI 1.10-6.41); $p = 0.030$). However, in the multivariable model, histological grade III, tumour size >2 cm, and lymphovascular invasion did not show significant relationship (Table 4 and Figure 4).

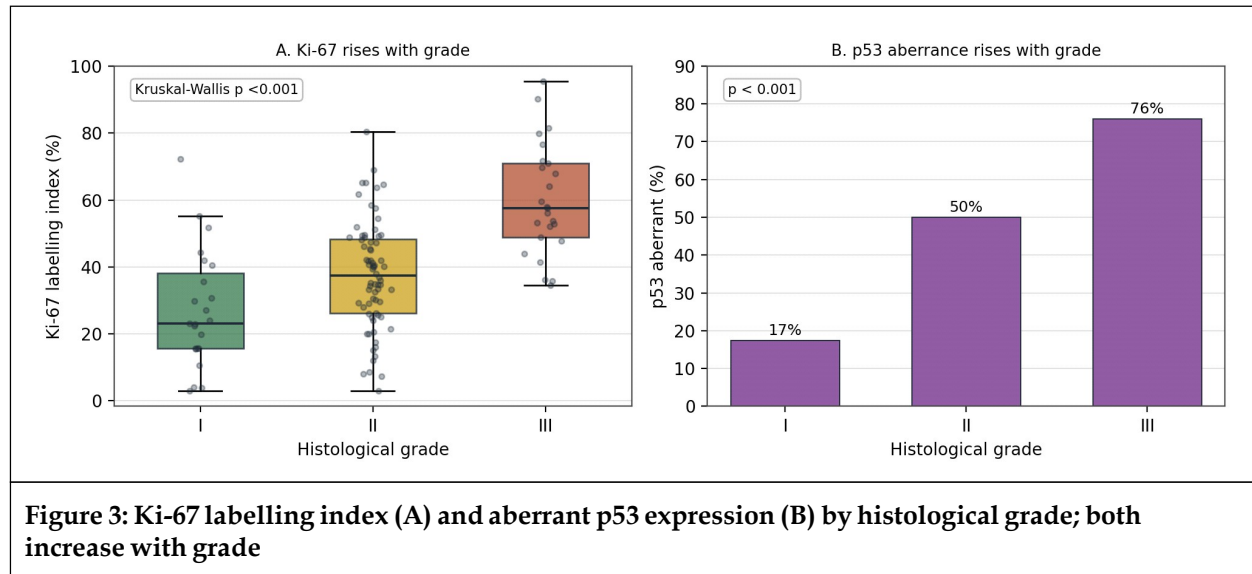


Figure 3: Ki-67 labelling index (A) and aberrant p53 expression (B) by histological grade; both increase with grade

Table 4: Multiple logistic regression for lymph node involvement

Predictor	Odds ratio	95% CI	p
Ki-67 (per 10%)	1.49	1.12 to 1.99	0.006
p53 aberrant	2.66	1.10 to 6.41	0.030
Grade III	0.68	0.21 to 2.18	0.515
Tumour size > 2 cm	1.30	0.49 to 3.43	0.602
LVI	1.47	0.63 to 3.46	0.375

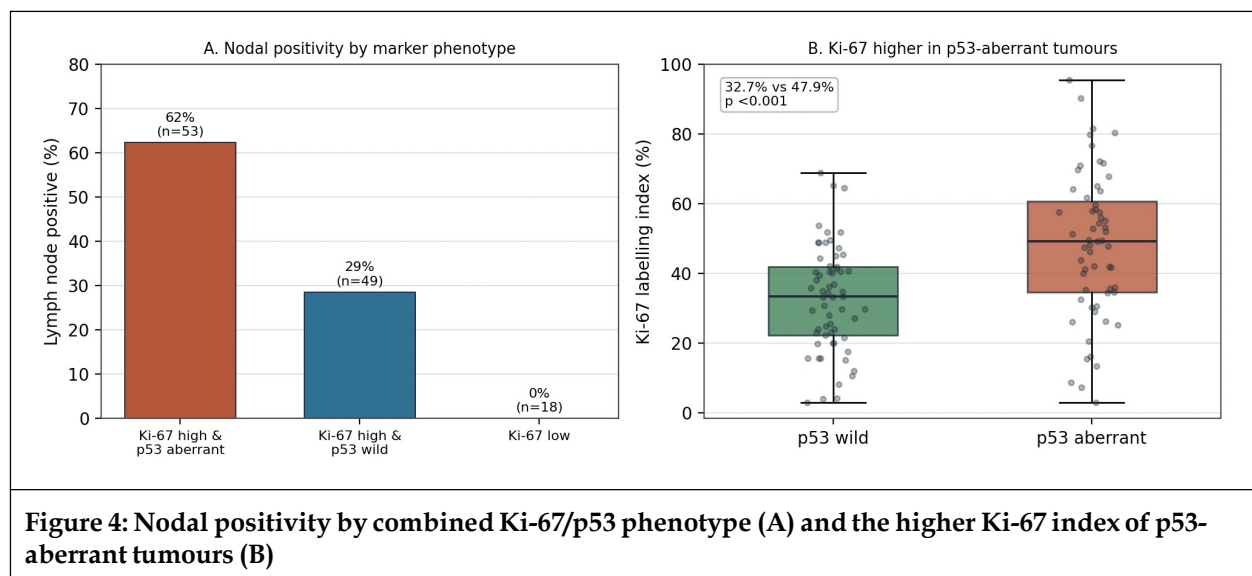


Figure 4: Nodal positivity by combined Ki-67/p53 phenotype (A) and the higher Ki-67 index of p53-aberrant tumours (B)

4. Discussion

The present study showed Ki-67 and p53 expression in triple-negative forms of breast carcinoma and investigated the association of these antigens with the known clinicopathological parameters. There were three main findings. The Ki-67 was often expressed, and was found to be significantly linked to the size of the tumour, the histological grade, involvement of lymph nodes and the pathological stage of the disease. Second, the expression

of p53 was linked to higher grade and nodal involvement, as well as to the stage. Third, Ki-67 and p53 did not give redundant information: The percentage of Ki-67 was greater in p53-aberrant tumours, and in the multivariable analysis, both Ki-67 and p53 independently predicted nodal involvement. These results indicate that there is an association between higher rate of proliferation, abnormal expression of p53 and more aggressive clinicopathological features in TNBC.

High Ki-67 expression was observed in 85.0% of the tumours, consistent with the highly proliferative nature of triple-negative breast carcinoma. Aberrant p53 expression was present in 49.2% of cases. Both observations are biologically plausible in the context of TNBC, in which high proliferative activity and TP53 alterations are frequently reported. Nevertheless, immunohistochemical p53 expression should be interpreted as a surrogate phenotype rather than as direct evidence of TP53 mutation, particularly because different molecular alterations can produce distinct staining patterns. The high prevalence of Ki-67 expression also indicates that, within TNBC, the magnitude of the labelling index may provide more biological information than simple positive-versus-negative classification.

The relationship between both markers and histological grade was the one most common and reproducible, and is the one most consistently supported by the literature. The mean Ki-67 index increased from 27% in grade I tumours to 60% in grade III and alongside it had the same trend of increasing aberrant p53, from 22% in grade I to 76% in grade III, thus following the same path of the dedifferentiation that grade expresses. This is largely because grade already uses the Mitotic index, an absolute measurement of proliferation and because the absence of the function of p53 allows for the genomic instability associated with the high grade morphology and because it provides a degree of biological coherence to the markers as indicators of the aggressive behavior.

The correlation between Ki-67 and p53 indicates that high proliferation rates are often associated with abnormal p53 expression; however, there was no statistical overlap between the two markers. There was significant independent association with both lymph-node involvement after adjusting for histological grade, tumour size and lymphovascular invasion. The logistic regression analysis revealed that there was a significant association between 10-percentage-point increases in the Ki-67 percentage with nodal involvement (OR 1.49, 95% CI 1.24–1.77) as well as aberrant expression of p53 (OR 2.70, 95% CI 1.76–4.10). The higher frequency of nodal positivity among tumours expressing both markers further suggests that combined assessment may identify a subgroup with a particularly aggressive clinicopathological phenotype. However, these cross-sectional associations should not be interpreted as evidence that either biomarker directly causes nodal dissemination.

From a practical perspective, Ki-67 and p53 are attractive biomarkers because they can be evaluated using routinely available immunohistochemical techniques. Their associations with nodal involvement and other aggressive pathological characteristics suggest potential value as complementary markers for biological risk characterization in TNBC. However, the present findings do not establish that either marker should replace conventional histological grading, pathological staging, or direct assessment of lymph-node status. Rather, combined Ki-67 and p53 evaluation may provide additional information regarding tumour phenotype. Prospective studies with clinical follow-up and independent validation are required to determine whether this combined profile contributes prognostic or predictive information beyond established clinicopathological factors ([Li et al., 2024](#)).

5. Strengths and limitations

This study has several strengths, including the simultaneous assessment of Ki-67 and p53 within a uniformly defined TNBC cohort and the evaluation of their relationships with multiple established clinicopathological parameters. Multivariable analysis further allowed their associations with nodal involvement to be examined after consideration of conventional pathological factors. However, a few constraints should be recognized. Firstly, the retrospective cross-sectional method does not allow for the analysis of temporal relationships or information about recurrence, survival or treatment response. Second, the study was carried out in one centre with a modest number of participants, so risk of selection bias and limited statistical power for less prevalent subgroups in the clinicopathological analysis. Thirdly, there is potential for variation in the interpretation of immunohistochemistry associated with the fixation, staining of Ki-67, the selection of hotspot, and interpretation of Ki-67. Fourth, the status of TP53 mutation is not a perfect correlate of the function of the p53 protein with

regard to immunohistochemistry, especially if exceptional expression patterns of the p53 protein are taken into consideration. Lastly, using the same cut-off for Ki-67 of 20% could affect the categorical associations. Bigger multicentre trials including molecular assessment of TP53 and follow up clinical response are needed.

6. Conclusion

Triple-negative breast carcinoma cells frequently exhibited high Ki-67 levels and abnormal levels of expression of the p53 gene, and the former were correlated with several clinicopathological adverse factors. Tumour size, histological grade, involvement of lymph nodes and pathological stage were significantly associated with high Ki-67 expression whereas aberrant p53 expression was associated with histological grade, involvement of lymph nodes and pathological stage. There was a correlation between cellular proliferation (measured by the ki-67 antigen levels) and the atypical expression of the p53 protein in tumours. Most significantly, Ki-67 and p53 were still independently related to the development of lymph-node involvement in multivariate analysis, indicating that they are complementary markers of the aggressive characteristics of the tumour. Both their analysis and the traditional histopathological analysis might then make a contribution to the biological characterization of TNBC. The results of this study need to be interpreted in the light of limitations inherent in such a single center, retrospective, cross sectional study. Further prospective multicentre studies that include clinical outcomes and molecular characterisation are required to resolve the issue of whether there is independent prognosis or prediction from combined Ki-67 and p53 assessment.

Conflicts of interest

The authors declare that no conflict of interest exists in relation to this work.

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