

Molecular subtypes of breast cancer in Nigeria: clinicopathological insights from immunohistochemical profiling

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Abstract

Background: Breast cancer demonstrates substantial molecular heterogeneity that influences prognosis and therapeutic decisions. Immunohistochemistry (IHC) provides an accessible surrogate method for molecular classification in low-resource settings. This study evaluated the distribution and clinicopathological significance of IHC-defined molecular subtypes among breast cancer cases in a Nigerian population.

Methods: A retrospective cross-sectional study was conducted on 273 histologically confirmed breast cancers diagnosed between 2019 and 2025. Formalin-fixed, paraffin-embedded tissues were stained for estrogen receptor (ER), progesterone receptor (PR), human epidermal growth factor receptor 2 (HER2), and Ki-67. Tumors were categorized into Luminal A, Luminal B (HER2-), Luminal B (HER2+), HER2-enriched, and Triple-Negative Breast Cancer (TNBC). Statistical analysis was performed using SPSS version 27. Associations between molecular subtype and clinicopathological parameters were evaluated using Chi-square tests, with $p < 0.05$ considered significant.

Results: TNBC was the most prevalent subtype (33.7%), followed by Luminal A (20.5%), Luminal B (HER2+) (16.1%), HER2-enriched (16.1%), and Luminal B (HER2-) (13.6%). Infiltrating duct carcinoma (NOS) accounted for 85.3% of cases. A significant association was observed between age group and molecular subtype ($\chi^2 = 14.36$, $df = 8$, $p = 0.023$). No significant relationship was found between molecular subtype and histologic grade ($p = 0.149$). The Ki-67 index was high ($\geq 20\%$) in 41% of tumors.

Conclusion: The predominance of aggressive subtypes, particularly TNBC, highlights an urgent need to integrate routine molecular classification into breast cancer diagnostics in Nigeria. IHC-based profiling is a practical tool for improving risk stratification and guiding individualized therapy in resource-limited regions.

Keywords: Breast cancer, Molecular subtypes, Immunohistochemistry, Estrogen receptor, HER2, Triple-negative breast cancer, Ki-67, Nigeria

Background

Breast cancer remains the most frequently diagnosed malignancy among women worldwide and a leading cause of cancer-related mortality [1]. Its burden continues to rise in low- and middle-income countries [2,3], where late presentation, limited diagnostic capacity, and uneven access to targeted therapies contribute substantially to poor outcomes [4]. Although traditionally classified using histomorphology, breast cancer is now recognized as a biologically diverse disease composed of distinct molecular entities, each characterized by unique patterns of growth, behavior, and therapeutic response [5].

The introduction of gene-expression profiling revolutionized breast cancer taxonomy by identifying intrinsic subtypes, Luminal A, Luminal B, HER2-enriched, and basal-like, each associated with distinct prognostic profiles [6]. However, genomic technologies remain largely inaccessible in many

resource-constrained settings. Immunohistochemistry (IHC) has therefore emerged as a practical surrogate method for molecular classification [7]. Assessment of ER, PR, HER2, and Ki-67 permits reasonably accurate assignment of tumors to clinically meaningful surrogate molecular groups [8,9]

Despite increasing reliance on IHC for clinical decision-making, data on the molecular landscape of breast cancer in African populations remain limited. Several studies suggest a disproportionate burden of biologically aggressive tumors, particularly triple-negative breast cancer (TNBC), among women of African ancestry [10–12]. Studies from West, East, and Southern Africa consistently report higher frequencies of TNBC than in European or Asian cohorts [13–15]. Yet many of these regional reports are constrained by small sample sizes, outdated classification systems, or incomplete biomarker profiling. Consequently, there is a critical need for robust, contemporary, population-level data to better understand disease biology and guide treatment planning.

This study addresses this gap by evaluating the distribution of IHC-defined molecular subtypes and their clinicopathological correlates in a large cohort of Nigerian breast cancer patients. By characterizing the molecular profile of tumors within this population, the study aims to support context-specific strategies for diagnosis, prognostication, and individualized therapy.

Methods

Study design and setting

This study employed a retrospective cross-sectional design involving breast cancer cases diagnosed at the Anatomic Pathology Unit of SynLab Nigeria, Lagos. The study period spanned January 2019 to January 2025, during which all eligible cases received routine histopathologic evaluation and immunohistochemical profiling. SynLab is a high-volume diagnostic center serving a broad population that includes both referred tertiary hospital patients and individuals presenting directly for diagnostic evaluation. This setting provides a diverse and representative sample of breast cancer diagnoses in the region.

Study population

The study included 273 women of age ranged from 22 to 86 years, with histologically confirmed primary breast cancer. Each case underwent detailed morphologic examination followed by immunohistochemical analysis as part of diagnostic workup. All available patient records and tumor blocks meeting the inclusion criteria were reviewed consecutively to minimize selection bias.

Inclusion and exclusion criteria

Inclusion criteria

- Histologically confirmed malignant breast tumors.
- Adequate and well-preserved formalin-fixed, paraffin-embedded (FFPE) tissue blocks suitable for IHC staining.
- Complete pathology records, including demographic and clinical data.

Exclusion criteria

- Severely autolyzed, crushed, or poorly preserved FFPE blocks unsuitable for IHC evaluation.

- Specimens from patients previously treated with neoadjuvant chemotherapy, as therapy may alter biomarker expression.
- Tumors with missing or incomplete immunohistochemical data.

Histopathologic evaluation

Archived FFPE blocks were retrieved, sectioned at 4 µm thickness, and stained with hematoxylin and eosin (H&E). Tumors were re-evaluated by a consultant pathologist, blinded to initial reports, to ensure diagnostic accuracy and uniform application of the 2019 WHO Classification of Breast Tumors [16].

Tumor characteristics assessed included

Histologic type

Histologic grade using the Nottingham grading system (I–III) [17]

Presence of lymphovascular invasion (LVI)

Tumor margins and architectural patterns

Any discrepancies between the original report and the review were resolved by consensus.

Immunohistochemistry (IHC)

IHC staining was performed using SynLab's standardized automated platform.

The stains are highly specific with high purity, staining what was expected to stain and the type of stain is Indirect Staining: This is a two-step (or multi-step) method involving an unlabeled primary antibody binds to the target antigen, and then a labelled secondary antibody, which recognizes the primary antibody and significantly amplifying the signal and increasing sensitivity.

The following primary antibodies were used:

Estrogen receptor (ER), Progesterone receptor (PR), HER2/neu (c-erbB-2), Ki-67 proliferative index.

Positive and negative controls were included for each run to ensure assay reliability.

Interpretation criteria

ER and PR

A tumor was considered positive if $\geq 1\%$ of tumor nuclei demonstrated immunoreactivity.

HER2

Scored according to cri/CAP guidelines (2018) [18]:

0 or 1+ = negative

2+ = equivocal; reflex testing recommended

3+ = positive

Due to resource constraints, HER2 2+ cases without confirmatory FISH were categorized as negative, as is standard in many LMIC diagnostic settings.

Ki-67

Dichotomized as low (<20%) and high ($\geq 20\%$), consistent with contemporary recommendations for IHC-based surrogate

classification. The 20% Ki-67 cut-off was selected primarily because studies showed it best stratifies breast cancer patients into distinct prognostic groups (low vs. high risk of recurrence and death), with the high Ki-67 group showing significantly poorer outcomes. This cut-off also showed good concordance with molecular tests and effectively identified patients who might benefit from more aggressive therapies like chemotherapy [19,20].

Molecular subtype classification

Tumors were assigned to one of five IHC-defined molecular subtypes:

Luminal A: ER+ and/or PR+, HER2-, Ki-67 <20%

Luminal B (HER2-): ER+ and/or PR+, HER2-, Ki-67 ≥20%

Luminal B (HER2+): ER+ and/or PR+, HER2+

HER2-Enriched: ER-, PR-, HER2+

Triple-Negative Breast Cancer (TNBC): ER-, PR-, HER2-

This classification mirrors surrogate systems widely used in clinical practice and research settings where genomic profiling is unavailable.

Data extraction

Demographic and clinicopathologic data were extracted from SynLab's Laboratory Information System (LIS). Variables collected included:

- Age at diagnosis
- Histologic diagnosis
- Histologic grade
- IHC biomarker status (ER, PR, HER2, Ki-67)
- Derived molecular subtype

All data were anonymized to protect patient confidentiality.

Statistical analysis

Data analysis was performed using IBM SPSS Statistics version 27.

Descriptive statistics

Categorical variables were summarized using frequencies and percentages. Continuous variables (e.g., age) were expressed as means ± standard deviations where appropriate.

Inferential statistics

Associations between molecular subtypes and clinicopathological variables (age group, grade, histologic type, Ki-67) were tested using the Chi-square test (χ^2).

When expected cell counts were <5, Fisher's exact test was applied. Statistical significance was set at $p < 0.05$, while Cramer's V was calculated for effect size to aid interpretation of significant relationships.

Ethical considerations

Ethical approval for this study was granted by the appropriate institutional review board. All data were handled in accordance with the principles of the Declaration of Helsinki. As a retrospective review of anonymized records, the requirement for informed consent was waived.

Results

Patient demographics

A total of 273 breast cancer cases met the inclusion criteria with a mean age of 51.8 ± 12.6 years (Table 1). More than half of the cohort (54.6%) fell within the 41–60 years age group, while 19.8% were below 40 years and 25.6% were above 60 years. Thus, the disease predominantly affected middle-aged women, but a substantial subset of younger patients was also represented [15,21].

Histologic characteristics

The vast majority of tumors were infiltrating ductal carcinoma (NOS), accounting for 85.3% ($n = 233$) of cases (Table 1). Less frequent diagnoses included lobular carcinoma (5.1%), high-grade ductal carcinoma in situ with microinvasion (2.2%), inflammatory carcinoma (1.8%), neuroendocrine carcinoma (1.5%), and other rare subtypes (4.0%). This distribution reinforces the well-

Table 1. Clinicopathologic features of breast cancer cases ($n = 273$).

Variable	Category	n	%
Age (years)	< 40	54	19.8
	41–60	149	54.6
	> 60	70	25.6
Histologic grade	I	32	11.7
	II	166	60.8
	III	75	27.5
ER status	Positive	166	60.8
	Negative	107	39.2
PR status	Positive	135	49.5
	Negative	138	50.5
HER2 status	Positive (3+)	54	19.8
	Negative (0–1+)	219	80.2
Ki-67 index	High (≥ 20 %)	112	41
	Low (< 20 %)	161	59

documented predominance of ductal carcinoma in global breast cancer epidemiology [22].

Histologic grading showed a predominance of grade II tumors, comprising 60.8% of cases, followed by grade III (27.5%) and grade I (11.7%). The predominance of intermediate-grade tumors suggests a moderately differentiated overall tumor profile in this cohort.

Hormone receptor and proliferative marker expression

Overall, 60.8% of tumors were ER-positive, while 39.2% lacked ER expression. PR expression demonstrated a balanced distribution, with 49.5% positivity and 50.5% negativity.

HER2 overexpression (3+) was identified in 19.8% of cases, while the majority (80.2%) were HER2-negative (0–1+ or 2+ without confirmatory testing) (Table 1).

The Ki-67 proliferation index was high ($\geq 20\%$) in 41% of tumors, reflecting a substantial subset of highly proliferative malignancies. The remaining 59% demonstrated a low Ki-67 index ($< 20\%$) [20]

(Table 1). Figure 1 shows representative immunohistochemical expression patterns of breast cancer biomarkers.

Molecular subtype distribution

Based on combined expression of ER, PR, HER2, and Ki-67, as shown in Table 2, the IHC-defined molecular subtypes were distributed as follows:

- Triple-Negative Breast Cancer (TNBC): 33.7% (n = 92)
- Luminal A: 20.5% (n = 56)
- Luminal B (HER2-): 13.6% (n = 37)
- Luminal B (HER2+): 16.1% (n = 44)
- HER2-Enriched: 16.1% (n = 44)

TNBC emerged as the single most common molecular subtype, accounting for one-third of all cases. Together, Luminal A and Luminal B (HER2-) tumors represented approximately 34% of the cohort, consistent with the predominance of hormone receptor-

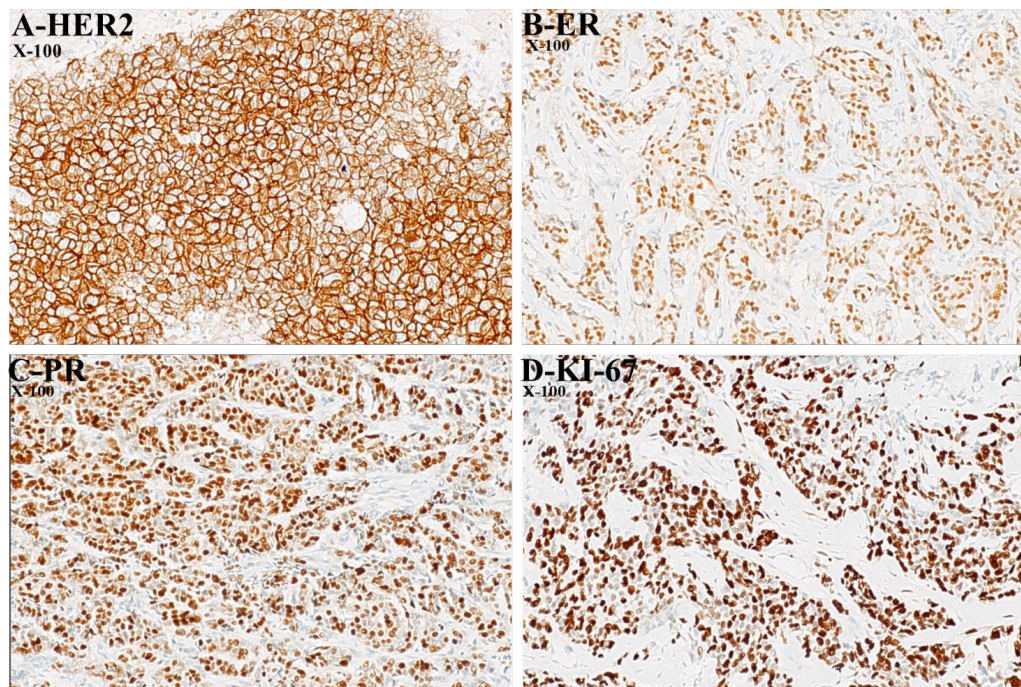


Figure 1. Representative immunohistochemical expression patterns of breast cancer biomarkers. (A) HER2 showing strong, complete membranous staining of tumour cells (score 3+), indicating HER2 overexpression ($\times 100$), (B) Estrogen receptor (ER) showing distinct brown nuclear positivity in a subset of tumour cells ($\times 100$), (C) Progesterone receptor (PR) demonstrating diffuse brown nuclear staining in tumour cells ($\times 100$) and (D) Ki-67 showing strong brown nuclear immunoreactivity in a high proportion of tumor cells, indicating a high proliferative index ($\times 100$).

Table 2. Distribution of molecular subtypes of breast carcinoma (n = 273).

Subtype	Definition	n	%
Luminal A	ER+/PR+, HER2-, Ki-67 < 20 %	87	31.9
Luminal B	ER+/PR+/-, HER2+/-, Ki-67 ≥ 20 %	67	24.5
HER2-enriched	ER-/PR-/HER2+	27	9.9
Triple-negative	ER-/PR-/HER2-	92	33.7

positive disease across many populations but highlighting a greater burden of aggressive subtypes in this setting.

Relationship between molecular subtypes and clinicopathological features

Age and molecular subtype

A statistically significant association was observed between age group and molecular subtype ($\chi^2 = 7.56$, $df = 3$, $p = 0.023$) (Table 3).

TNBC predominated among women under 40 years and those 41–60 years, whereas luminal tumors, especially Luminal A, were more common among patients above 60 years. This suggests a potential age-related shift in tumor biology, with younger women demonstrating more aggressive phenotypes (Table 3).

Histologic grade and molecular subtype

No statistically significant association was found between histologic grade and molecular subtype ($p = 0.149$). Although high-grade morphology was more frequently observed in TNBC and HER2-enriched tumors, the overlap across subtypes diluted statistical significance. This finding reinforces the well-known heterogeneity of grade within luminal and non-luminal tumors.

Histologic type and molecular subtype

Infiltrating ductal carcinoma exhibited the full range of molecular subtypes, with TNBC and luminal groups represented prominently. Lobular carcinoma cases predominantly fell within the luminal categories, reflecting their known steroid receptor-driven biology.

Conversely, inflammatory carcinoma showed a strong inclination toward TNBC, consistent with its aggressive clinical behavior.

While histologic type showed descriptive differences across subtypes, the association did not achieve statistical significance ($p > 0.05$), likely due to small numbers in rare categories.

Ki-67 and molecular subtype

High Ki-67 expression ($\geq 20\%$) was most frequently observed in HER2-enriched, Luminal B, and TNBC tumors, whereas Luminal A tumors were predominantly low-proliferative. This marked difference aligns with the biological framework that positions Luminal A tumors as more indolent, while the remaining subtypes are characterized by heightened mitotic activity.

Discussion

This study provides a comprehensive characterization of the molecular landscape of breast cancer in a Nigerian cohort using immunohistochemistry as a surrogate for intrinsic subtyping. Several important patterns emerge, with meaningful clinical and biological implications for breast cancer care in sub-Saharan Africa [13,15].

Predominance of triple-negative breast cancer (TNBC)

The most striking finding is the high proportion of TNBC (33.7%), which stands out as the predominant molecular subtype in this cohort. This aligns with other African and African-diaspora data showing disproportionate representation of basal-like tumors among women of African ancestry [14,23]. However, the proportion identified here is substantial and reinforces concerns about the aggressive phenotype that characterizes breast cancer in many African populations. Biologically, TNBC tumors tend to exhibit basal-like gene expression, enriched for [24,25].

TP53 mutations

Proliferation pathway upregulation, defects in DNA repair pathways (including BRCA-associated mechanisms) and enhanced immune infiltration in some subsets.

These tumors typically present with rapid growth, early metastasis, and limited therapeutic options in settings with constrained access to immunotherapy or PARP inhibitors [26].

Clinically, the predominance of TNBC has sobering implications. Because TNBC lacks ER, PR, and HER2 targets, treatment largely depends on cytotoxic chemotherapy, an option that may not be readily accessible or well tolerated in many low-resource environments. Furthermore, supportive care for chemotherapy toxicities is not uniformly available, which can worsen outcomes. The high frequency of TNBC in this cohort, therefore, underscores an urgent need for earlier diagnosis, integration of genetic counseling, and enhanced chemotherapy support services.

Distribution of luminal tumors

Luminal A tumors accounted for 20.5% of cases, and Luminal B tumors (HER2- and HER2+) together accounted for approximately 29.7%. These proportions differ significantly from Western populations, where Luminal A tumors often comprise nearly half of all cases [22].

Table 3. Evaluation of age group and IHC molecular subtypes.

Age group (years)	IHC classification of molecular subtypes					P-value
	HER2- enriched	Luminal A	Luminal B (HER2-)	Luminal B (HER2+)	Triple negative	
20-24	0	1	0	0	0	0.023*
25-29	0	0	0	3	5	
30-34	5	4	4	1	5	
35-39	2	4	8	7	9	
40-44	6	18	4	6	12	
45-49	8	7	3	10	18	$\chi^2 = 7.56$, df = 3,
50-54	7	4	5	4	11	
55-59	10	4	2	6	9	
60 and above	6	14	11	7	23	

Several factors may explain this lower proportion of luminal disease:

- Younger age structure of African populations, with higher premenopausal incidence [21].
- Genetic predispositions influencing hormone receptor expression.
- Delayed presentation, which may lead to down-regulation of ER and PR in advanced tumors.
- Tumor dedifferentiation associated with chronic inflammation or microenvironmental factors.
- Luminal tumors typically respond well to endocrine therapy, which is relatively accessible and affordable [19]. Ensuring consistent access to tamoxifen, aromatase inhibitors, and ovarian suppression strategies may therefore produce substantial survival gains.

HER2-enriched and Luminal B (HER2+) subtypes

HER2 overexpression was identified in 19.8% of tumors, with HER2-enriched and Luminal B (HER2+) subtypes each contributing 16.1% to the total sample. This frequency is comparable to global estimates [27], suggesting that HER2-driven biology may be relatively stable across populations.

However, the clinical consequences differ greatly based on treatment availability. HER2-targeted therapies, trastuzumab, pertuzumab, and newer antibody, drug conjugates remain limited in many parts of Africa due to high cost. Consequently, women with HER2-positive tumors may not derive the survival benefits seen in high-resource countries. This situation highlights the importance of advocating for access programs, biosimilar trastuzumab formulations, and national insurance coverage [28].

Age–subtype relationship

A significant association was observed between age group and molecular subtype, with TNBC disproportionately affecting younger and middle-aged women. This age pattern has profound implications: Younger women often present with higher-grade, higher-proliferation tumors.

They may carry hereditary predispositions, including BRCA1 mutations known to drive basal-like/TNBC phenotypes [29,30]. Fertility preservation, psychosocial support, and survivorship planning become more complex. This pattern emphasizes the need for genetic risk assessment, particularly for young women presenting with TNBC.

Histologic grade and molecular subtype

Despite descriptive differences, histologic grade was not significantly associated with molecular subtype in this study. While TNBC and HER2-enriched tumors often trend toward higher grades, luminal tumors can also demonstrate grade heterogeneity, especially in resource-limited settings where delays in diagnosis may permit tumor progression.

This lack of association reinforces the view that grade alone is insufficient to determine tumor biology, and that molecular classification provides indispensable complementary prognostic information [22].

Ki-67 proliferation index and tumor behavior

High Ki-67 expression ($\geq 20\%$) was most common among HER2-enriched, Luminal B, and TNBC tumors. This is consistent with the aggressive clinical behavior associated with these subtypes [8].

Ki-67, despite variability in scoring across laboratories, remains a useful marker of proliferative activity and is central to distinguishing Luminal A from Luminal B tumors. In this cohort, the strong alignment between high Ki-67 and aggressive subtypes supports its continued use and the need for standardized scoring protocols [20].

Histologic type and subtype distribution

The predominance of infiltrating ductal carcinoma (85.3%) mirrors global patterns. Lobular carcinoma cases were mostly luminal, reflecting their hormone receptor-rich biology, while inflammatory carcinoma clustered toward TNBC, consistent with its aggressive natural history.

Although the association between histologic type and subtype did not reach statistical significance, the descriptive patterns observed align with established tumor biology and provide additional context for clinical interpretation.

Clinical and public health implications

This molecular profile paints a clear picture: breast cancer in this population is characterized by a higher-than-expected burden of aggressive subtypes, which contributes to poor outcomes seen across sub-Saharan Africa [28,31].

Implications include

- Urgent need for earlier detection, particularly among younger women.
- Expansion of IHC-based molecular testing across the region to support risk stratification.
- Improved access to chemotherapy and endocrine therapy, which remain relatively cost-effective.
- Advocacy for affordable HER2-targeted therapies, including biosimilars.
- Genetic counseling and BRCA testing, especially for young TNBC patients.
- This study adds valuable evidence toward developing more nuanced, resource-appropriate breast cancer treatment guidelines in Nigeria and comparable settings.

Comparison to existing evidence and explanation of differences

The findings align with reports from East, West, and Southern Africa but contrast sharply with European and North American cohorts where luminal tumors dominate. Reasons for these differences include:

Genetic ancestry and the influence of genomic architecture, Variations in reproductive patterns and hormonal exposures, Delays in detection leading to more advanced, dedifferentiated tumors.

Limited access to screening and diagnostic services.

These factors collectively shape the unique molecular epidemiology observed in the region.

Conclusion

This study provides a detailed and contemporary characterization of breast cancer molecular subtypes in a Nigerian cohort using immunohistochemistry as a practical surrogate for genomic profiling. The molecular landscape observed is dominated by Triple-Negative Breast Cancer, accompanied by relatively fewer Luminal A tumors than typically reported in Western populations. These findings underscore a significant burden of biologically aggressive disease [24], particularly among younger women, and highlight the need for targeted strategies to improve outcomes.

Routine incorporation of IHC-based molecular classification into diagnostic workflows can empower clinicians to make more individualized treatment decisions, even in resource-limited environments. The high prevalence of TNBC and HER2-enriched tumors further emphasizes the importance of expanding access to effective systemic treatments, strengthening chemotherapy delivery systems, and advocating for affordable HER2-targeted therapies. This work therefore contributes valuable evidence supporting regional efforts toward precision oncology and provides a foundation for future studies, particularly those integrating genetic profiling and longitudinal outcome data.

Strengths and Limitations

Strengths

This study leverages a relatively large sample size ($n = 273$), providing robust insights into the molecular profile of breast cancer in a Nigerian population.

The use of a standardized IHC panel (ER, PR, HER2, Ki-67) ensures reliable subtype classification, consistent with global surrogate definitions.

Uniform re-evaluation of histology using the 2019 WHO classification enhances diagnostic accuracy and comparability.

The study reflects a real-world diagnostic environment, offering pragmatic data relevant to clinical decision-making in low-resource settings.

Limitations

As a retrospective study, it is subject to limitations related to variability in documentation, incomplete clinical information, and selection bias.

HER2 2+ cases lacking confirmatory FISH testing may have led to minor misclassification of HER2 status, a constraint imposed by resource availability.

Absence of genomic profiling means intrinsic molecular subtypes could not be directly compared with their genetic counterparts.

Clinical outcome data (e.g., survival, response to therapy) were not available, limiting the ability to correlate molecular subtypes with prognosis.

Despite these limitations, the study offers valuable insights into the regional biology of breast cancer and highlights priority areas for strengthening diagnostic and therapeutic services.

Declarations

Ethics approval and consent to participate

Ethical approval for this study was granted by the appropriate

institutional ethics committee. All patient data were anonymized prior to analysis. Given the retrospective nature of the study, the requirement for informed consent was waived.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request and with appropriate institutional approval.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

The corresponding author conceptualized the study, participated in data curation and analysis, and led manuscript development. All authors contributed to critical review and approved the final version of the manuscript.

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