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Association of Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet-to-Lymphocyte Ratio (PLR) With Molecular Subtypes of Breast Cancer

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ABSTRACT

Background & Objective: Breast cancer (BC) is the most common cancer and a leading cause of cancer-related deaths among Indian women. Inflammation and the tumor microenvironment have a significant influence on cancer development, progression, immune surveillance, and response to therapy. The neutrophil/lymphocyte ratio (NLR) and platelet/lymphocyte ratio (PLR) are simple, measurable markers of subclinical inflammation. This study aimed to evaluate the association of NLR and PLR with the molecular subtypes and clinicopathological parameters of BC.

Methods: A hospital-based study was conducted on 228 BC patients. Cases were classified into four molecular subtypes, which were further divided into Group A with a good prognosis (Luminal A and Luminal B) and Group B with a poor prognosis (HER2-enriched and TNBC). Cutoff values for NLR and PLR were calculated using the ROC curve.

Results: The cutoff values for NLR and PLR were 2.64 and 90, respectively. NLR was significantly higher (≥2.64) in TNBC (70.6%) and lower (<2.64) in Luminal A (79.2%). Elevated NLR and PLR were associated with clinical T stage (P=0.005 and P=0.028), high PI (P=0.006 and P=0.029), and advanced stage (P=0.002 and P=0.026). A high PLR (≥90) was observed in 60.3% of advanced BC cases, compared to 39.7% of early BC cases. NLR and PLR were negatively correlated with ER and PR (P<0.05).

Conclusion: A significant correlation was observed between high NLR and PLR and adverse prognostic clinico-pathological factors in breast cancer. NLR and PLR could serve as valuable, cost-effective markers for prognosis and prediction in breast cancer.

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Introduction

According to GLOBOCAN 2022, breast cancer has the highest incidence worldwide (23.8%) among women (1). In India, BC statistics are even more alarming (2). Despite increasing nationwide and state-wide awareness programs and improving treatment modalities, BC remains the leading cause of cancer, with an incidence rate of 26.6% in 2022, and the leading cause of cancer-related deaths in females, accounting for 13.7% of cancer deaths in 2022 (2,3).

The 5th WHO classification categorizes breast tumors into integrative clusters and subtypes based on genomic and transcriptomic data analysis (4). The various subtypes have different clinicopathological presentations, treatment modalities, and prognoses. Although this molecular classification is more accurate, standardized, and comprehensive, it is expensive and not easily accessible.

Surrogate molecular subtypes, including Luminal A, Luminal B, HER2-enriched, and triple-negative/basal-like, based on biomarkers—estrogen receptor (ER), progesterone receptor (PR), HER2, and proliferation index (Ki-67)—are routinely used (4).

Inflammation and the tumor microenvironment play key roles in various stages of cancer development, including initiation, promotion, and metastasis. They influence immune surveillance and responses to therapies (4). Cancer-promoting inflammatory effects may occur either directly through interactions between inflammatory cells (ICs) and tumor cells or indirectly via activation of resident stromal cells (4). Infiltrating leukocytes and activated stromal cells secrete a wide variety of growth factors and proteases (5,6). ICs also create an immunosuppressive microenvironment and evade the host immune response by recruiting

immunosuppressive regulatory T cells or suppressing CD8+ cytotoxic T cells (5,6).

NLR and PLR are simple, easily measurable markers of subclinical inflammation. Recent studies have shown these inflammatory markers to predict cancer recurrence and survival across various solid organ cancers (7-10). This study aimed to evaluate the association of NLR and PLR with molecular subtypes and clinicopathological parameters of breast cancer (BC).

Materials and Methods

A hospital-based, cross-sectional, prospective study was carried out over 18 months in the Department of Pathology at AIIMS Patna. The study received approval from the institutional ethics committee (letter no. AIIMS/Pat/IEC/PGTh/July 20/15), and informed consent was obtained from all participants.

All histopathologically confirmed BC cases were included, excluding patients undergoing protocol-specific breast cancer treatments, recurrent cases, those with active infection or bleeding, chronic inflammatory diseases, recent blood transfusions within the preceding 3 months, and inflammatory breast carcinoma.

Molecular subtyping was performed using immunohistochemistry (IHC) for ER, PR, HER2, and Ki-67 on a representative tissue section, with an automated VENTANA BenchMark GX system. Patient information was collected from medical records.

Interpretation of IHC staining:

Expression of ER and PR was assessed using the Allred scoring system. HER2 status was determined by IHC, with a score of 3+ indicating a HER2-positive tumor. The proliferation index (PI) was calculated based on the highest nuclear expression of Ki-67 within the tumor area, counted out of 1000 tumor cells, and expressed as a percentage.

Molecular subtyping of breast cancer

Luminal A: ER+, PR +/-, HER2-, Ki-67 <14%; Luminal B: ER+, PR +/-, HER2- and Ki-67 >14% or HER2+ and Ki-67 <14%; HER2-enriched: ER-, PR-, HER2+, Ki-67 any; Triple-negative: ER-, PR-, HER2-, Ki-67 any.

Calculation of NLR and PLR

Complete blood count (CBC) tests, performed within 2 weeks prior to the biopsy, were conducted using the Sysmex XN-1000. If platelet plots appeared abnormal, the platelet count was estimated from a peripheral smear. For samples showing clumping or clotting, a repeat blood sample was requested. NLR was calculated by dividing the absolute neutrophil count by the absolute lymphocyte count, and PLR was obtained by dividing the absolute platelet count by the absolute lymphocyte count.

Statistical analysis

Descriptive analysis was conducted using Jamovi 2.3.18.0. The normality distribution of the continuous variable was evaluated through Q-Q plots and the Shapiro-Wilk test. Homogeneity was examined with Levene's test. Statistical differences between various molecular subtypes, NLR, and PLR were assessed using one-way ANOVA followed by a post hoc Games-Howell test. Cutoff values for NLR and PLR were identified using receiver operating characteristic (ROC) curves generated in Stata software. Categorical variables were analyzed through the chi-square test, while Pearson's correlation assessed relationships between continuous variables. The associations between the cutoff points for NLR, PLR, and clinicopathological variables were tested using either the chi-square or Fisher exact test. A P value <.05 was considered statistically significant.

Results

A total of 228 BC cases meeting the study criteria were evaluated. Most BC patients were female (98.7%), with a mean age of 50 years (range, 22-90 years), peaking in the fourth decade (32.9%). The majority presented with a unilateral breast lump (98.2%), predominantly of no special type (97.4%). Sixty-four percent of cases were grade 2, followed by grade 1 (21.1%) and grade 3 (14.9%). The mean tumor size was 5.22 ± 2.48 cm. At presentation, 78% of patients had axillary node metastasis, and metastasis was observed in 15.4% of cases. Most cases were diagnosed at stage III (57.5%), with the rest primarily at stage II.

The most common molecular subtype was Luminal B (37.7%), followed by HER2-enriched BC (29.4%), TNBC (22.4%), and Luminal A (10.5%).

We also found 23 cases of ER-/PR+ breast cancer. Of these cases, 43.5% showed overexpression of HER2 on IHC. There are conflicting reviews on their existence as a separate molecular subtype. These cases were excluded from this study.

Molecular subtypes and clinicopathological features:

Molecular subtypes were significantly associated with lymph node involvement (P < .01) and tumor stage (P = .007). TNBC exhibited the highest proportion of cases with lymph node metastasis and advanced stage. However, there was no association between molecular subtypes and age, sex, histological subtype, tumor size, or metastasis status in breast cancer.

Cutoff value of NLR and PLR

A one-way ANOVA test revealed significant differences in the mean NLR (P = .018) and PLR (P = .021) across the 4 molecular subtypes. The mean NLR values were 2.57, 2.65, 3.20, and 4.42, while the mean PLR values were 106.86, 99.69, 108.22, and 156.43 for the Luminal A, Luminal B, HER2-enriched, and TNBC groups, respectively.

Cases were further subdivided into 2 groups: (i) Group A, comprising molecular subtypes with a good prognosis—Luminal A and Luminal B; and (ii) Group B, consisting of subtypes with a poor prognosis—HER2-enriched and TNBC.

Cutoff values for NLR and PLR were determined based on these groups using receiver operating characteristic (ROC) curves generated with Stata statistical software. NLR was categorized as high (≥2.64) in 46.9% of cases and low (<2.64) in 53.1%. Similarly, high PLR (≥90) was observed in 53.1%, while low PLR (<90) was seen in 46.9% (Figs. 1 and 2).

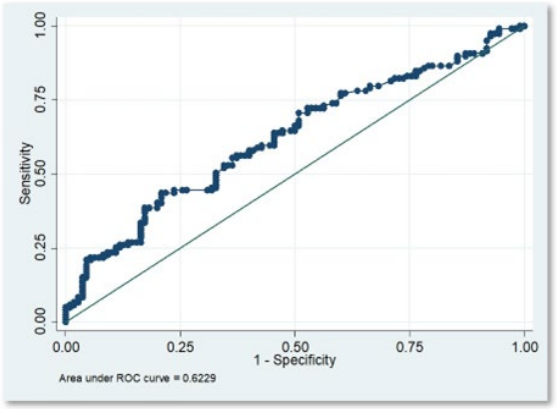

Fig. 1. ROC curve of NLR for Molecular subtypes in breast cancer. (Cutoff- 2.64).

AUC- 0.6229, Sensitivity- 56.30%, Specificity- 62.73%, Positive Likelihood Ratio- 1.5106, Negative Likelihood Ratio- 0.6966, Standard error- 0.0369, 95% CI (0.55058-0.69518); n=228.

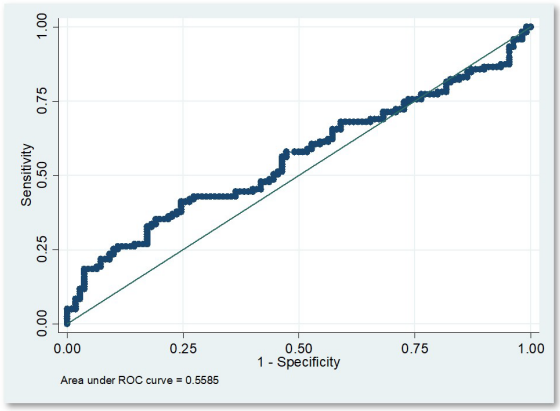

Fig 2. ROC curve of PLR for Molecular subtypes in breast cancer. (Cutoff- 90).

AUC- 0.5585, Sensitivity- 57.98%, Specificity- 52.73%, Positive Likelihood Ratio- 1.2266, Negative Likelihood Ratio- 0.7969, Standard error- 0.0382, 95% CI (0.48374-0.63330); n=228.

Association of NLR and PLR with molecular subtypes

The cutoff value of NLR was significantly different among the molecular subtypes (P < 0.001). The proportion of high NLR (≥2.64) increased from Luminal A (20.8%) to TNBC (70.6%). There was no statistically significant association of PLR among the molecular subtypes of breast cancer. However, the proportion of high PLR (≥90) was highest in TNBC (64.7%) (Table 1).

Association of NLR and PLR with clinicopathological parameters

NLR (P < 0.001) and PLR (P = 0.003) showed negative correlations with the Allred scores for ER and PR, respectively. PLR positively correlated with the Ki-67 level (P = 0.029). Clinical T stage (cT4) was significantly associated with high NLR (P = 0.005) and PLR (P = 0.028). Only elevated NLR (≥2.64) was significantly associated with cTNM stage (P = 0.022). There was no significant association with age group, sex, histological subtype, histological grade, lymph node metastasis, or metastasis (Tables 2 and 3).

Table 1. Chi square test revealed substantial significant association of NLR cut off value and molecular subtypes of BC. PLR cut off value showed no statistical association with molecular subtypes

Molecular subtypes	Low NLR (<2.64)	High NLR (≥2.64)	Low PLR (<90)	High PLR (≥90)	P value (χ^2 Test)
Luminal A	19 (79.2%)	5 (20.8%)	11 (48.5%)	13 (54.2%)	NLR <0.001 (Cramer V= 0.289)
Luminal B	50 (58.1%)	36 (41.9%)	47 (54.7%)	39 (45.3%)	
Her2 enriched	37 (55.2%)	30 (44.8%)	31 (46.3%)	36 (53.7%)	
TNBC	15 (29.4%)	36 (70.6%)	18 (35.3%)	33 (64.7%)	PLR 0.183

Table 2. Association of NLR and PLR with various clinico-pathological parameters of breast cancer (Chi square and Fisher's exact test).

Clinico-pathological parameters	Low NLR (<2.64)	High NLR (≥2.64)	Low PLR (<90)	High PLR (≥90)	P value
Age groups (years)					
20-30	9 (69.2%)	4 (30.8%)	6 (46.2%)	7 (53.8%)	
30-40	28 (68.3%)	13 (31.7%)	17 (41.5%)	24 (58.5%)	NLR 0.77
40-50	36 (48%)	39 (52%)	36 (48.0%)	39 (52.0%)	
50-60	27 (51.9%)	25 (48.1%)	28 (53.8%)	24 (46.2%)	PLR 0.69
70-80	4 (28.6%)	10 (71.4%)	8 (57.1%)	6 (42.9%)	
>80	2 (100%)	0	1 (50.0%)	1 (50.0%)	
Histological subtype					
IBC, NST	119 (53.1%)	105 (46.9%)	105 (46.9%)	119 (53.1%)	
Special type of breast cancer	2 (66.7%)	1 (33.3%)	2 (66.7%)	1 (33.3%)	0.789
IBC, NST with special morphological pattern	0	1(100%)	0	1 (100%)	
Histological grade					
Grade 1	32 (66.7%)	16 (33.3%)	25 (52.1%)	23 (47.9%)	NLR 0.059
Grade 2	75 (51.4%)	71 (48.6%)	68 (46.6%)	78 (53.4%)	
Grade 3	14 (41.2%)	20 (58.8%)	14 (41.2%)	20 (58.8%)	PLR 0.329
cT stage					
T1	6 (60.0%)	4 (40.0%)	5 (50.0%)	5 (50.0%)	NLR 0.005
T2	37 (67.3%)	18 (32.7%)	30 (54.5%)	25 (45.5%)	
T3	26 (65.0%)	14 (35.0%)	25 (62.5%)	15 (37.5%)	PLR 0.028
T4	52 (42.3%)	71 (57.7%)	47 (38.2%)	76 (61.8%)	
Lymph node metastasis					
Negative	30 (58.8%)	21 (41.2%)	27 (52.9)	24 (47.1%)	NLR 0.350
Positive	91 (51.4%)	86 (48.6%)	80 (45.2%)	97 (54.8%)	PLR 0.329
Metastasis status					
No metastasis	95 (57.9%)	69 (42.1%)	84 (51.2%)	80 (48.8%)	NLR 0.052
Metastasis	13 (37.1%)	22 (62.9%)	11 (31.4%)	24 (68.6%)	
Data not available	13 (44.8%)	16 (55.2%)	12 (41.4%)	17 (58.6%)	PLR 0.084
Clinical TNM stage					
Stage I	5 (55.6%)	4 (44.4%)	5 (55.6%)	4 (44.4%)	
Stage II	37 (71.2%)	15 (28.8%)	27 (51.9%)	25 (48.1%)	NLR 0.022
Stage III	54 (51.4%)	51 (48.6%)	53 (50.5%)	52 (49.5%)	
Stage IV	12 (36.4%)	21 (63.6%)	10 (30.3%)	23 (69.7%)	PLR 0.254
Data not available	13 (44.8%)	16 (55.2%)	12 (41.4%)	17 (58.6%)	
Clinical stage					
Early-stage BC	66 (66.0%)	34 (34.0%)	57 (57.0%)	43 (43.0%)	NLR 0.002
Advance stage BC	42 (42.4%)	57 (57.6%)	38 (38.4%)	61 (61.6%)	
Data not available	13 (44.8%)	16 (55.2%)	12 (41.4%)	17 (58.6%)	PLR 0.026
ER status					
Negative	48 (42.5%)	65 (57.5%)	48 (42.5%)	65 (57.5%)	NLR 0.001
Positive	73 (63.5%)	42 (36.5%)	59 (51.3%)	56 (48.7%)	PLR 0.182
PR status					
Negative	54 (44.3%)	68 (55.7%)	53 (43.4%)	69 (56.6%)	NLR 0.004

Clinico-pathological parameters	Low NLR (<2.64)	High NLR (≥2.64)	Low PLR (<90)	High PLR (≥90)	P value
Positive	67 (63.2%)	39 (36.8%)	54 (50.9%)	56 (49.1%)	PLR 0.258
Her2 status					
Negative	70 (52.6%)	63 (47.4%)	62 (46.6%)	71 (53.4%)	NLR 0.875 PLR 0.911
Positive	51 (53.7%)	44 (46.3%)	45 (47.4%)	50 (52.6%)	
PI (%)					
Low	21 (77.8%)	6 (22.2%)	14 (51.9%)	13 (48.1%)	NLR 0.006 PLR 0.585
High	100 (49.8%)	101 (50.2%)	93 (46.3%)	108 (53.7%)	

Table 3. Correlation of NLR and PLR with various continuous variables in the study.

Clinico-pathological parameters	NLR	PLR
Age (years)	r=0.092, P=0.165	r=0.074, P=0.268
Tumor size (cm)	r=0.106, P=0.115	r=0.104, P=0.122
Allred score (ER)	r= -0.233, P<0.001	r= -0.195, P=0.003
Allred score (PR)	r= -0.236, P< 0.001	r= -0.197, P=0.003
Ki67 level (PI%)	r=0.104, P=0.116	r= 0.144, P=0.029
Number of metastatic sites	r= 0.062, P=0.356	r=0.152, P=0.022

Discussion

Recent literature reviews have proposed the role of inflammatory markers as predictive markers of treatment outcomes and prognostic significance in BC patients (9,11–13).

Distribution of molecular subtypes

In our study, the most common molecular subtype was Luminal, followed by HER2-enriched and TNBC. Among Luminal types, 78% were Luminal B, while Luminal A accounted for only 12%. A review of the literature indicated that Luminal A is the most common breast cancer subtype, which contrasts with our findings (13–19). Additionally, our study showed a notably high rate of TNBC at 22.4%, higher than in other regions of the world (20,21). Thakur et al reported that Indian women have the highest prevalence of TNBC at 27.9% (22).

Association of molecular subtypes and clinicopathological features

Molecular subtypes demonstrated a significant association with histological grade, cT stage, cTNM staging, lymph node status, and PI. The multivariate analysis revealed that TNBC is associated with poorer prognostic factors. Luminal A was the most indolent in terms of clinical and pathological behavior. Luminal B and HER2-enriched BC showed similar clinical and pathological characteristics. Unlike our study, where the average age of TNBC patients was 51 years, Kumar et al's systematic review found that TNBC typically develops at a younger age (23). Park et al reported in their study that TNBC had a younger age of onset, larger tumor size, higher grade, and higher PI (24). Most previous studies agree with our analysis, highlighting TNBC as being associated with poor prognostic features and tumor characteristics. Nevertheless, a few studies noted HER2-enriched BC to be the most aggressive subtype (18,25).

Association of NLR and PLR with molecular subtypes

The cutoff value of NLR ranged from 2.0 to 4.0 across various studies, similar to our findings (12,14,18,26–29). However, the cutoff for PLR showed significant variation, from 104.4 to 190.9, and was notably higher in other studies compared with our study (14,15,18,26–29).

NLR was significantly higher in TNBC but lower in Luminal A. High NLR also correlated with negative hormone receptor status and a low Allred score. However, the cutoff value of PLR did not show significant differences across the 4 molecular subtypes. HER2 status was not associated with either NLR or PLR.

Huszno et al found in their study that patients with increased NLR tended to have poor ER status, while those with high PLR were more often diagnosed with TNBC. Additionally, poorer overall survival (OS) was associated with elevated levels of NLR and PLR (29).

Xu et al found that patients with lower NLR and PLR responded better to treatment (18). Montano et al also observed no variation across molecular subtypes of BC in relation to the NLR cutoff (30). Yersal et al studied the median NLR and PLR across different molecular subtypes but found no significant differences (31).

Association of NLR and PLR with clinicopathological parameters

We found that NLR (P < .001) and PLR (P = .003) were negatively correlated with the Allred score for ER and PR, respectively. PLR showed a positive

correlation with PI (P = .029). The clinical T stage (cT4) was significantly linked to higher NLR (P = 0.005) and PLR (P = 0.028). Only elevated NLR (≥2.64) was significantly associated with cTNM staging (P = .022). No significant relationships were observed with age, sex, histological subtype, histological grade, lymph node metastasis, or metastasis.

NLR was significantly higher in cases with PI >14% (P = 0.006) and advanced stage (P = 0.002). Huszno et al showed that elevated NLR was associated with poor ER status, while high PLR was frequent in tumors with poor differentiation (29).

Multivariate analysis by Montano et al observed high NLR in younger patients and those with premenopausal status (P = 0.001), but no other clinicopathological features of BC were associated with NLR (15).

Graziano et al found a significant association between high NLR and age ≤50 years, as well as high PLR observed in advanced cancer stages (14).

Most previous research has found a relationship between NLR or PLR and age, as well as clinical stage. There are a few studies in which there was no correlation between NLR, PLR, and clinicopathological features in breast cancer (31).

In the study by Acikgoz et al, patients with low PLR had a higher rate of pCR (P = 0.001) compared with those with high PLR (27). Kim et al divided breast cancer patients into low and high groups based on cutoff values of NLR and PLR using the ROC curve and found that 44.3% of the low-ratio group responded better to NACT, versus only 15.3% in the high-ratio group (32). Savioli et al observed that elevated NLR and PLR were adverse prognostic variables (13).

Not only in BC, numerous published studies have demonstrated the usefulness of these inflammatory indicators as prognostic markers for recurrence and survival in colorectal cancer, hepatocellular carcinoma, lung cancer, gallbladder cancer, and renal cell carcinoma (7,8,10). Although the relationship between NLR, PLR, and adverse clinicopathological factors is not fully understood, studies have shown that NLR and PLR can serve as subtle indicators of inflammation (11). Neutrophils have been shown to suppress immune function, inhibit T-cell response, and promote tumor progression (11).

Additionally, platelets promote tumor cell proliferation either by directly interacting with tumor cells or indirectly through the release of angiogenic and mitogenic proteins. T-lymphocytes are crucial for immunological surveillance (5). This might explain the association of high NLR and PLR with poor clinicopathological behavior of BC in the present study.

Limitations

Due to the unavailability of cytogenetic analysis, fluorescence in situ hybridization (FISH) for HER2 IHC score 2+ was not performed, and these cases had to be excluded from the study. NLR and PLR may serve as more useful markers for managing BC if their association is established with prognostic outcomes, such as 5-year survival, 10-year survival, overall survival, or disease-free survival.

Conclusion

In our study, NLR varied significantly among molecular subtypes of breast cancer, with high NLR observed mostly in TNBC, while lower NLR was more common in Luminal A and Luminal B. Additionally, we found a strong association between high NLR and PLR with poor prognostic clinicopathological factors, particularly advanced clinical stage and high Ki-67 levels. Our results, along with previous research, highlight a significant correlation between inflammatory markers (NLR and PLR) and the clinicopathological features and molecular subtypes of breast cancer. These markers are easy to measure through routine tests. Few studies have explored the link between molecular subtypes and inflammatory markers in breast cancer, especially in India. In conclusion, we suggest that more extensive future research, particularly on prognosis, could establish NLR and PLR as valuable prognostic and predictive markers in breast cancer.

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Authors' Contributors

Pummi Kumari and Ruchi Sinha: Study concept and design, data interpretation, manuscript preparation and editing; Alok Ranjan: Statistical analysis; Surabhi Ajit, Tarun Kumar and Jagjit Kumar Pandey: Manuscript review.

Data Availability

The data supporting the results of this study are available upon request from the corresponding author.

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This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Ethics Approval

The study was approved by the Institutional Ethics Committee of All India Institute of Medical Sciences Patna (letter no. AIIMS/Pat/IEC/PGTh/July 20/15, dated September 20, 2021).

Consent to Participate

Written informed consent was obtained from all participants before enrollment in the study and before blood sample collection. Participants who were unable or unwilling to provide consent were excluded from the study. This is consistent with the Methods section, which states that eligible participants provided written informed consent and that inability or unwillingness to consent was an exclusion criterion.

Conflict of Interest

The authors declared no conflict of interest.

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