

Association between Platelet-to-Albumin Ratio and Breast Cancer: A Retrospective Study

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Abstract

Objective: This study explored the correlation between platelet-to-albumin ratio (PAR) and breast cancer. **Methods:** We enrolled Individuals with suspicious breast lesions. These lesions were found during routine health check-ups at the Health Management Center of Deyang People's Hospital from January 2024 to December 2025. Patients whose breast cancer was confirmed by pathology made up the case group. Participants with benign results served as controls. We adopted multivariable logistic regression to evaluate the relationship between PAR and breast cancer. **Results:** We enrolled 336 participants in total. Among them, 62 had pathologically confirmed breast cancer and were assigned to the case group. The remaining 274 had benign lesions and served as the control group. The case group was older than the control group. Cases also showed higher rates of smoking, alcohol consumption, and hypertension (all $P < 0.05$). On contrast, PAR levels were significantly lower in cancer patients than in controls ($P < 0.05$). We then fitted a multivariate logistic regression model. This model adjusted for age, smoking, alcohol consumption, BMI, diabetes, and hypertension. In that analysis, PAR was marginally related to breast cancer risk (OR = 0.781, 95% CI: 0.609 - 1.000, $P = 0.0499$). **Conclusion:** PAR may serve as a biomarker for breast cancer. Nevertheless, we view our findings as preliminary and we interpret them with caution. Further prospective studies are needed to determine its clinical usefulness.

Keywords

Platelet-to-Albumin Ratio, Breast Cancer, Association, Risk

1. Introduction

Breast cancer remains one of the major contributors to cancer-associated illness and deaths in women globally. Rising incidence has been observed around the

world [1]. According to 2020 global surveillance records, approximately 2.3 million women received a new breast cancer diagnosis, while 685,000 died from the disease [2]. Epidemiological forecasts indicate that annual incident cases will surpass 3 million by 2040. This corresponds to a more than 40% rise relative to 2020 levels, and predicted deaths will grow by over 50% over the same two decades. Countries scoring low on the Human Development Index (HDI) will witness the steepest upward trend. These shifts will impose a substantial burden on public health infrastructure within such areas [2].

In clinical practice, 64.9% of patients are diagnosed with breast cancer at an advanced stage. This situation largely leads to unsatisfactory survival outcomes [3]. In China, breast cancer incidence and mortality rates keep rising rapidly [4]. These trends highlight an urgent demand for effective prevention and early screening strategies. Such strategies should match the local epidemiological characteristics.

Chronic inflammation can promote the occurrence, progression and metastasis of tumors. This important association has been confirmed by numerous studies [5] [6]. Given its affordability and widespread availability, platelet testing has recently captured growing interest among researchers. In breast cancer, platelet-related parameters hold considerable promise as auxiliary tools for both diagnostic workup and prognostic evaluation. High platelet count is one of the common hematological abnormalities in breast cancer patients, and it plays a crucial role in regulating the tumor microenvironment. This marker can reveal inflammatory reactions induced by malignancies, while also illustrating the reciprocal interplay of systemic inflammation, thrombotic activity and tumor progression [7]. Researchers have proposed various biomarkers for assessing systemic inflammatory responses, including the neutrophil-to-lymphocyte ratio (NLR), the platelet-to-lymphocyte ratio (PLR), and the lymphocyte-to-monocyte ratio (LMR). These biomarkers, easily measured in blood, mirror the tumor-associated systemic inflammatory state. These biomarkers enable serial assessment of treatment response and provide predictive value for both therapeutic outcomes and prognosis across diverse tumor types [8]-[11].

Cumulative findings from existing work stress the value of cancer prophylaxis and early diagnostic screening. Current literature contains limited analyses exploring the correlation linking PAR to breast malignant tumors. This work aims to examine the relationship between PAR and breast cancer among adult women.

2. Materials and Methods

2.1. Research Design

We screened health examination data from Deyang People's Hospital between January 2024 and December 2025. We initially identified 878 patients who presented BI-RADS 4A or higher lesions on breast ultrasonography. We excluded subjects according to preset criteria. Finally, 336 participants were included in the analysis. The cohort contained 62 patients with newly diagnosed breast cancer and

274 control subjects. All controls had pathologically confirmed benign lesions. We collected information included social demographic features, body measurement indicators, as well as routine blood and biochemical test results.

Inclusion criteria: 1) female patients aged ≥ 18 years; 2) histopathologically confirmed breast cancer. The excluded subjects included 248 patients without any subsequent hospital visits, 221 patients who attended clinic visits received follow-up recommendations from breast specialists and did not undergo surgical treatment, 53 participants who underwent screening but could not be traced to obtain final diagnostic results, 19 patients diagnosed with other malignant tumors, and one patient with hematological disease. The Ethics Committee of Deyang People's Hospital approved this study (2025-04-081-K01). Since this work adopted a retrospective design, researchers waived the requirement for written informed consent. All patient information was anonymized to protect personal privacy.

2.2. Definition of the Platelet-to-Albumin Ratio

The PAR is calculated as platelet count divided by the serum albumin concentration.

2.3. Covariates

We referred to previous studies [12], age, smoking status, alcohol intake, body mass index (BMI), diabetes, and hypertension as potential confounders in our multivariable models. BMI was derived from measured height and weight, with the formula: $BMI = \text{weight (kg)} / \text{height (m)}^2$.

2.4. Statistical Analysis

All statistical analyses were performed using Python (version 3.12.7). Normally distributed variables with equal variances were shown as mean $\pm$ standard deviation (SD). Variables that did not follow a normal distribution were reported as median (interquartile range, IQR). Group comparisons for normally distributed continuous variables were conducted using Student's t-test; otherwise, the Mann-Whitney U test was applied. Categorical variables were compared using the chi-square test, with Fisher's exact test employed when the expected frequency in any 2×2 cell was <5 . Multivariable analysis was performed using binary logistic regression to identify independent factors. A $P < 0.05$ was considered statistically significant.

3. Results

3.1. Baseline Characteristics of the Participants

A total of 336 participants were enrolled, including 62 patients with newly diagnosed breast cancer and 274 controls. At baseline, breast cancer patients were significantly older, with higher smoking and alcohol consumption rates and a higher prevalence of hypertension compared with controls (all $P < 0.05$), whereas no significant differences were found in BMI or diabetes ($P > 0.05$). Importantly, the

PAR was significantly lower in the case group than in the control group ($P < 0.05$) (**Table 1**).

Table 1. Baseline characteristics of the participants.

Variables	Controls (n = 274)	Breast cancer (n = 62)	P
Age	48.90 ± 10.54	51.76 ± 10.07	0.034
BMI	23.14 ± 2.83	23.18 ± 2.49	0.750
Smoking			0.034
yes	6 (2.2%)	5 (8.1%)	
no	268 (97.8%)	57 (91.9%)	
Drinking			<0.001
yes	2 (0.7%)	7 (11.3%)	
no	272 (99.3%)	55 (88.7%)	
hypertension			0.030
yes	34 (12.4%)	15 (24.2%)	
no	240 (87.6%)	47 (75.8%)	
diabetes			0.401
yes	7 (2.6%)	3 (4.8%)	
no	267 (97.4%)	59 (95.2%)	
PAR	4.85 (3.98, 5.76)	4.53 (3.47, 5.29)	0.015

3.2. The Relationship between PAR and Breast Cancer

Table 2. Multivariable logistic regression analysis of PAR and breast cancer.

Model	Adjustments	OR (95% CI)	P
Model 1	Unadjusted	0.778 (0.621 - 0.974)	0.0288
Model2	Adjusted for age	0.806 (0.639 - 1.015)	0.0670
Model3	Adjusted for age, smoking, alcohol, BMI, diabetes, and hypertension	0.781 (0.609 - 1.000)	0.0499

Logistic regression was used to evaluate the association between PAR and breast cancer (**Table 2**). In the unadjusted model, each 1-unit increase in PAR was associated with a 22.2% reduction in breast cancer risk (OR = 0.778, 95% CI: 0.621 - 0.974, $P = 0.029$). After adjustment for age, this association became non-significant ($P = 0.067$). In Model 3, we adjusted for smoking status, alcohol intake, BMI, diabetes and hypertension. The estimated effect size remained similar (OR = 0.781). However, the 95% confidence interval (0.609 - 1.000) crossed the null value. The P value was 0.0499, which was right at the widely adopted threshold for statistical significance. The findings only offered weak and borderline evidence for this association. We added the quadratic term of PAR to Model 3 to test nonlinearity. The outcome showed no statistical significance ($P = 0.610$). This suggested that

PAR had no nonlinear correlation with breast cancer risk.

3.3. Subgroup Analysis by Age

To assess potential effect modification by age, we stratified the study population into two age groups (≤ 60 years and > 60 years). The younger subgroup comprised 286 participants (50 cases), and the older subgroup comprised 49 participants (12 cases). Both subgroup analyses were adjusted for the same set of covariates as in the primary model. The test for interaction yielded a P value of 0.925, indicating no significant heterogeneity in the PAR-breast cancer association across age strata. Although the stratified estimate reached nominal statistical significance in the younger subgroup (OR = 0.758, 95% CI: 0.581 - 0.990, $P = 0.042$), this finding was not supported by the interaction test (Table 3). No significant association was detected in the older subgroup ($P = 0.353$). Collectively, these data do not support age as a modifier of the observed association.

Table 3. Subgroup analysis of the association between PAR and breast cancer by age, with interaction testing.

Age subgroup	OR (95% CI)	P	P for interaction
≤ 60 years	0.758 (0.581 - 0.990)	0.042	0.925
> 60 years	0.721 (0.361 - 1.438)	0.353	

4. Discussion

In this study, PAR showed a borderline inverse association with breast cancer, with the fully adjusted model yielding an OR of 0.781 (95% CI: 0.609 - 1.000, $P = 0.0499$). While the confidence interval bordered the null value, the P value reached the conventional threshold for significance. We performed subgroup analyses stratified by age. No significant effect modification was detected ($P = 0.925$). This indicated that the marginal association we found did not vary substantially across different age groups.

Platelets are anucleate blood cells. Researchers have acknowledged their core roles in hemostasis and thrombosis. To carry out these functions, platelets adhere to damaged vascular endothelium and subsequently aggregate. In addition to their hemostatic role, growing evidence suggests platelets participate in many pathological processes, include inflammation, atherosclerosis, tumor progression, and metastasis [13]. Platelets are viewed as promising liquid biopsy biomarkers for cancer screening in a study. Furthermore, their value in identifying hematological malignancies and solid tumors have been verified in several previous studies [14]-[16]. A critical mechanism underlying platelet-tumor crosstalk is tumor cell-induced platelet aggregation (TCIPA), a process developed by bioactive mediators released from both cancer cells and platelets, such as ADP, thromboxane A_2 , serine proteases, heparanases, and matrix metalloproteinases (MMPs) [17]. TCIPA enables cancer cells to escape immune surveillance. It enhances the attachment of tumor cells to vascular endothelium, and stimulates the secretion of growth fac-

tors, thereby promoting tumor proliferation [17]. Additionally, platelet-derived microvesicles (PMVs) have been implicated in regulating hemostasis, inflammation, and angiogenesis, indicating a close link between platelets and malignancy [18].

Serum albumin is the most abundant protein in human plasma. It is commonly used to evaluate nutritional status by clinicians, and reflects systemic inflammation [19]. Lower albumin concentrations are associated with higher mortality and greater risks of tumor recurrence. These findings suggest that albumin might provide valuable prognostic value [20] [21]. The PAR is a new composite marker. Several studies demonstrate PAR can reflect inflammatory status more accurately than other platelet-related indicators. A possible explanation is that PAR is less susceptible to transient physiological fluctuations [22] [23]. Consistent with this study, published works have confirmed that PAR is associated with the severity and clinical outcomes of various inflammatory diseases and malignant tumors [24] [25].

In univariate analysis, breast cancer patients showed a significantly lower PAR level compared with controls ($P < 0.05$). After adjusted for potential confounders in multivariate models, PAR presented a borderline inverse correlation with breast cancer risk ($P = 0.0499$). This result is not consistent with a prior study that found a null association based on a U.S. population. This might be related to the varied baseline characteristics of the subjects and the inconsistency of the covariate adjustment strategies. Variations can also be explained by platelets' multifaceted biological activities. These cells modulate tumor growth bidirectionally via complex crosstalk between immune cells and stromal compartments [12].

Substantial evidence has proven that age exerts a prominent impact on breast cancer risk [26]. To explore whether age could alter the correlation between PAR and breast cancer, we conducted subgroup analyses stratified by age. The negative association reached significance among participants aged ≤ 60 years ($P < 0.05$). We found no statistically significant relationship in the elderly subjects aged ≥ 60 years yet ($P > 0.05$). No significant association was observed in the subgroup older than 60 years ($P > 0.05$). The P value for interaction was 0.925. This indicates that the association exhibits no significant disparities among different age subgroups. Thus, this subgroup-specific significant finding is likely due to random chance and should be interpreted cautiously.

This study has several limitations. First, this is a single-center retrospective study, which may introduce inherent selection bias. Second, several critical factors, including menopausal status, family history, reproductive profile and hormone exposure, were incomplete recorded in our dataset. Therefore, we could not adjust for these unmeasured confounders, which may have interfered with the true association between PAR and breast cancer risk. In addition, the small sample size may reduce statistical precision. This limitation was evidenced by the wide confidence interval approaching the null value in the fully adjusted model. Therefore, although our findings suggest a potential protective correlation between PAR

and breast cancer, these results are exploratory rather than conclusive.

5. Conclusion

This study revealed a borderline negative relationship between PAR and breast cancer risk. This result suggests that systemic inflammation, nutritional status, and metabolic dysfunction might contribute to breast cancer development. PAR is readily available in routine practice and low cost. For risk grouping and preliminary cancer screening, this marker may function as a supplementary assessment tool. However, its practical clinical value remains to be confirmed. Prospective cohort studies with more covariate adjustment are still required.

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Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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