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The Relationship Between Serum Uric Acid Levels and Platelet Count in Post-Chemotherapy Breast Cancer Patients

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ABSTRACT

Introduction: Breast cancer is a malignant disease that has become a global health problem with an increasing incidence rate. Serum uric acid levels are the end product of purine metabolism and may increase following chemotherapy due to cellular damage and increased purine degradation. Platelets are involved not only in hemostasis but also in inflammation and tumor progression, and their count may be affected by chemotherapy. This study aims to determine the relationship between serum uric acid levels and platelet count in post-chemotherapy breast cancer patients.
Method: This study is observational analytical research with a cross-sectional approach. Medical record data from 93 post-chemotherapy breast cancer patients who met the eligibility criteria were included using total sampling. Serum uric acid levels and platelet count were obtained from laboratory examinations documented in the medical records during the post-chemotherapy evaluation period. The relationship between serum uric acid levels and platelet count was analyzed using Spearman's correlation test.
Results: The median serum uric acid level was 4.2 (2–11) mg/dL, while the median platelet count was 290 (67–656) $10^3/\mu L$. Spearman's correlation analysis showed a very weak positive correlation between serum uric acid levels and platelet count ($r = 0.101$), which was not statistically significant (*P* value = 0.335).

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Conclusion: A very weak positive correlation was observed between serum uric acid levels and platelet count in post-chemotherapy breast cancer patients. However, the association was not statistically significant.

Keywords: Breast cancer, chemotherapy, inflammation, platelet, serum uric acid levels

Introduction

Breast cancer is a malignant tumor that originates in the lobules or ductal epithelium of the breast and is characterized by uncontrolled cell proliferation, the ability to invade surrounding tissues, and infiltrate other organs; its biological characteristics vary, which can affect treatment response and patient prognosis.[1,2] Breast cancer is most commonly found in women and remains the most prevalent form of cancer globally. In 2022, research from Global Cancer Statistics estimated 2.30 million new cases and a total of 666,103 deaths due to breast cancer.[1–3] Breast cancer imposes a burden not only from mortality and incidence rates, but also from a decline in life quality, loss of productivity, and increased economic costs of health care.[2,3]

Breast cancer remains the disease with the highest incidence among women and is the leading cause of death in Indonesia.[4,5] Some of the early-detection innovations developed are insufficient to ensure treatment success and prevent mortality, as patients seek healthcare facilities only in advanced stages of the disease.[5] Approximately 68–73% of breast cancer patients in Indonesia are in stages III–IV, indicating that early diagnosis remains low. Possible problems include the referral system and a shortage of oncology specialists.[4,6] This issue requires concrete action in the form of early monitoring, active public education, equitable distribution of health care services, and evaluation of management practices to reduce incidence and mortality rates in Indonesia.[4,5]

Multimodal therapy is a treatment approach for breast cancer still used today, including chemotherapy, surgery, targeted therapy, radiation therapy, immunotherapy, and hormone therapy, all of which are tailored to the stage and molecular characteristics of the tumor.[7] Chemotherapy is one of the most commonly used treatments because of its ability to reduce the risk of recurrence, inhibit the growth of cancer cells, and maintain patients' quality of life; as a result, it plays a crucial role in neoadjuvant, adjuvant, and palliative therapy.[8] Regular monitoring is necessary because chemotherapy has hematologic and metabolic side effects.[9]

Aggressive tumor cell growth in breast cancer patients increases the demand for nucleotides, thereby activating purine metabolism and promoting the growth and survival of

cancer cells.[10] This process leads to increased cell turnover, then increases the breakdown of purines, which in turn increases serum uric acid levels via an enzymatic pathway involving xanthine oxidase.[11] This process can also influence the formation of reactive oxygen species (ROS), increasing the risk of cellular damage, oxidative stress, and active cellular inflammation mediated by various factors.[10–12] Serum uric acid levels play an important role as an extracellular antioxidant; elevated uric acid levels can exert pro-oxidant effects, activate inflammatory pathways, alter immune responses, and promote cancer cell migration.[11,12]

The use of chemotherapy carries a risk of hematologic toxicity, such as decreased platelet count (thrombocytopenia), due to suppression of megakaryocyte proliferation and differentiation in the bone marrow.[13,14] In addition, thrombocytopenia may lead to a risk of bleeding, the need for transfusions, and affect the patient's quality of life.[13] A persistent decrease in platelet count can affect the success of treatment because it often requires delaying the next chemotherapy cycle or reducing the relative dose intensity of chemotherapy.[15]

The correlation between serum uric acid levels and platelet count in breast cancer patients following chemotherapy remains an active area of research, as both variables are influenced by oxidative stress, inflammatory processes, and changes in the body's metabolism.[16–18] Serum uric acid levels are the end product of purine metabolism that can activate inflammatory pathways and generate ROS, potentially affecting platelet function and activation.[16,17] Meanwhile, platelets do not merely serve a hemostatic function; they also play a role in tumor progression, angiogenesis, and metastasis within the tumor microenvironment.[9,18]

Previous studies have investigated the role of serum uric acid levels in cancer metabolism and prognosis, including its potential involvement in oxidative stress and inflammatory processes.[16] Other studies have examined the role of platelets in breast cancer progression and metastasis,[18] as well as chemotherapy-induced thrombocytopenia and its correlation with treatment-related factors such as chemotherapy regimens and cycles.[19] In Indonesia, Nabilah et al. reported hematologic changes among breast cancer patients following chemotherapy, including post-chemotherapy thrombocytopenia.[20] However, these studies have primarily examined serum uric acid levels, platelet-related changes, or chemotherapy-induced hematologic toxicity separately. Evidence specifically addressing the relationship between serum uric acid levels and platelet count in breast cancer patients following chemotherapy remains limited, particularly in the Indonesian population. Therefore, this study aimed to determine the relationship between serum uric acid levels and platelet count in post-chemotherapy breast cancer patients, addressing the existing gap regarding the potential

correlation between metabolic and hematologic changes in this population.

Methods

The study design used in this research was observational, analytic, and correlational, with a cross-sectional approach. Serum uric acid levels in post-chemotherapy breast cancer patients were correlated with platelet counts. Breast cancer patients were included based on diagnosis by the oncologic surgeon documented in their medical records; in some patients, the breast cancer was known to have metastasized based on the findings of the oncologic surgeon documented in their medical records. Non-metastatic breast cancer patients were defined as breast cancer patients with no evidence or indication of metastasis. These patients had chemotherapy treatment and were in the post-chemotherapy evaluation period.

The patient data were collected from January 2025 to June 2026 at Anutapura Regional General Hospital in Palu. A total of 190 breast cancer patient records were identified during the study period. Patients were included if they had a confirmed diagnosis of breast cancer, had received chemotherapy, and had complete data on serum uric acid levels and platelet counts during the defined post-chemotherapy evaluation period. Patients with incomplete medical records or missing data on the primary study variables were excluded.

In this study, "post-chemotherapy" was defined as the period following completion of a specific chemotherapy cycle. Serum uric acid levels and platelet counts were obtained from laboratory examinations documented in the patients' medical records following completion of the respective chemotherapy cycle. The timing of laboratory assessment was based on the date recorded in the medical record in relation to the corresponding chemotherapy administration.

A total sampling technique was used, whereby all medical records that met the eligibility criteria during the study period were included. After applying the eligibility criteria, 93 patients were included in the final analysis. Serum uric acid levels and platelet counts were then correlated using Spearman's correlation test, with $\alpha < 0.05$. This study was approved by the Research Ethics Committee of the Faculty of Medicine at Tadulako University under letter number 7947/UN28.10/KL/2026.

Result

This study included 93 post-chemotherapy breast cancer patients, and their baseline characteristics are shown in **Table 1**. By gender, most patients were female, with 1 male patient (1.1%). This indicates that breast cancer can still occur in the male population. Based on metastasis status, most patients were non-metastatic, with 36 (38.7%) patients metastatic. Based on age, the study population ranged from adults to the elderly. Serum uric acid levels and platelet counts were analyzed as variables to assess their relationship in this study.

Table 1. Characteristics of the sample of post-chemotherapy breast cancer patients (N=93)

Sample Characteristics	n (%)
Gender	
Male	1 (1.1%)
Female	92 (98.9%)
Metastasis Status	
No Metastasis	57 (61.3%)
Metastasis	36 (38.7%)
	Median (min-max)
Age (year)	51 (31-82)
Uric Acid (mg/dL)	4.27 (2-11)
Platelets (10^3/µL)	290 (67-656)

Table 2 reveals the correlation between serum uric acid and platelet count in post-chemotherapy breast cancer patients. Spearman's correlation analysis showed a very weak positive correlation between serum uric acid levels and platelet counts (r = 0.101). However, this correlation was not statistically significant (*P* = 0.335).

Table 2. The relationship between uric acid and platelet count in post-chemotherapy breast cancer patients (N= 93)

Serum Uric Acid Levels (mg/dL) Median (Min-Max)	Platelets (10^3/µL) Median (Min-Max)	Correlation Coefficient	*P* value
4.27 (2-11)	290 (67-656)	0.101	0.335

Spearman correlation test

Discussion

Among the 93 post-chemotherapy breast cancer patients included in this study, the median serum uric acid level was 4.27 mg/dL (range: 2–11 mg/dL), while the median platelet count was 290 × 10^3/µL (range: 67–656 × 10^3/µL). Spearman's rank correlation analysis showed a very weak positive correlation between serum uric acid levels and platelet count (r = 0.101). However, this correlation was not statistically significant (*P* = 0.335). The correlation coefficient, which was close to zero, indicates that the observed relationship was very weak. Therefore, the findings do not provide sufficient statistical evidence to conclude that serum

uric acid levels are significantly associated with platelet count in post-chemotherapy breast cancer patients.

Serum uric acid levels and platelet count in post-chemotherapy breast cancer patients may be affected by cytotoxic-induced cellular damage, but through distinct mechanisms. Chemotherapy-induced thrombocytopenia (CIT) has been reported in approximately 21% of cases, with its incidence varying according to cancer type and treatment regimen.[19] In contrast, chemotherapy-related increases in serum uric acid levels may occur following extensive cellular destruction, in which nucleic acids released from damaged cells are metabolized through the purine degradation pathway and ultimately converted to serum uric acid via xanthine oxidase.[21] This metabolic response is particularly recognized in tumor lysis syndrome (TLS), which may occur following cytotoxic therapy in patients with substantial tumor cell destruction.[22]

However, these two processes do not necessarily occur in parallel. Platelet count is primarily determined by bone marrow function and thrombopoiesis, whereas serum uric acid levels are more directly related to purine metabolism and renal excretion of uric acid. Therefore, chemotherapy-induced changes in serum uric acid levels may occur independently of changes in platelet count. This difference in underlying mechanisms may partly explain the very weak and non-significant correlation observed between serum uric acid levels and platelet count in the present study ($r = 0.101$; $P = 0.335$).

Metastatic status may have influenced the observed relationship between serum uric acid levels and platelet counts. Patients with metastatic disease may differ from those without documented metastasis in terms of tumor burden and biological activity, which may affect systemic inflammation, tumor cell turnover, and platelet production.[18,23] In this study, 36 patients (38.7%) had metastatic disease, whereas 57 patients (61.3%) were classified as non-metastatic. However, the correlation analysis was performed on the entire study population and was not stratified by metastatic status. Therefore, it cannot be determined whether metastatic status modified the strength or direction of the observed correlation. This is an important consideration when interpreting the very weak positive correlation ($r = 0.101$) that was not statistically significant ($P = 0.335$) observed in this study. Further studies with stratified analyses and adjustment for metastatic status are warranted to clarify its potential influence on the relationship between serum uric acid levels and platelet counts.

Subsequent increases in serum uric acid levels can act as damage-associated molecular patterns (DAMPs) that activate the NLRP3 inflammasome and trigger sterile inflammation through the release of proinflammatory cytokines, such as IL-1β, IL-18, IL-6, and TNF-α.[9]

This inflammatory response is reflected in various systemic inflammatory markers, including the neutrophil-to-lymphocyte ratio (NLR), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), platelet-to-lymphocyte ratio (PLR), and platelet count (PLT).[9,23]

One of the parameters that can be influenced by inflammation in breast cancer research is platelets. In addition to serving as markers of inflammation, platelets also function as markers of tumor activity because they contribute to angiogenesis, proliferation, invasion, and metastasis of cancer cells through various growth factors, such as vascular endothelial growth factor (VEGF), platelet-derived growth factor (PDGF), and transforming growth factor-β (TGF-β). In patients who have undergone chemotherapy, platelet counts are affected by chemotherapy's cytotoxic effects on the bone marrow, which can suppress platelet production (thrombopoiesis). Therefore, changes in platelet counts in breast cancer patients following chemotherapy result from the interaction between an inflammatory response triggered by elevated serum uric acid levels, tumor activity, and the effects of chemotherapy on bone marrow function.[9,13,24]

Serum uric acid levels play a dual role in the body's redox balance; it can act as an antioxidant at normal levels and as a prooxidant at high levels. This can indirectly affect changes in platelet counts, which are sensitive to oxidative stress.[16] The relationship between serum uric acid levels and platelets is indirect; it does not stem from a direct biochemical cause-and-effect relationship, but rather from drug toxicity and the resulting cellular damage. Other confounding factors may also influence the correlation, such as the chemotherapy cycle, treatment regimen, and kidney function. This explains why the correlation test results in this study were very weak, with a p-value of 0.335 ($r = 0.101$).[21]

In accordance with theory, the results of this study are consistent with the understanding that independent physiological pathways regulate serum uric acid levels and platelet counts; purine metabolism, which produces serum uric acid, is regulated by xanthine oxidase, which is separate from the platelet production pathway.[25] On the other hand, platelet formation is activated by megakaryocytes under the influence of the hormone thrombopoietin and the bone marrow microenvironment through independent molecular mechanisms.[9]

This study found a very weak positive correlation between serum uric acid levels and platelet count, although the correlation was not statistically significant. This finding should be interpreted in the context of previous research on hematologic changes following chemotherapy, including those reported by Nabilah et al. Regarding breast cancer cases in Indonesia, where the incidence of post-chemotherapy thrombocytopenia was relatively low, approximately 9% of the total 82 patients, when compared to other hematological disorders.[20]

Previous research conducted by Zhou et al. also showed that post-chemotherapy thrombocytopenia is influenced by the degree of bone marrow suppression resulting from the chemotherapy cycle and regimen, without any correlation with serum uric acid levels.[19]

Serum uric acid levels and platelet counts remain relevant laboratory parameters in breast cancer patients, although this study found a very weak positive correlation that was not statistically significant. Serum uric acid levels cannot be used as the sole parameter for changes in platelet count; therefore, platelet counts must continue to be monitored through routine blood tests, while accounting for the chemotherapy regimen and cycle as confounding factors affecting platelet count changes.[20] This study may also open up opportunities to explore other parameters related to changes in platelet count.

The limitations of this study include the cross-sectional design, which can only demonstrate correlation between serum uric acid levels and platelet count at a single point in time; therefore, a cause-and-effect relationship cannot be established. Additionally, this study did not account for confounding variables, such as chemotherapy cycle, type, and regimen; surgical status; kidney function; and variations in the timing of post-chemotherapy sample collection.[21]

Conclusion

There was no statistically significant correlation between serum uric acid levels and platelet counts in post-chemotherapy breast cancer patients. Although a very weak positive correlation was observed, the correlation was not statistically significant. Routine periodic blood tests can continue to be performed to monitor platelet counts as an indicator of tumor activity and inflammation in breast cancer. Further research is needed to investigate the relationship between uric acid and other inflammatory markers, as well as other parameters that reflect the prognosis of breast cancer.

Conflicts of Interest

The authors declare that they have no conflicts of interest regarding the publication of this article.

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