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ORIGINAL RESEARCH

The Effects of High Particulate Matter Levels on Platelet Recovery in Patients Receiving Prophylactic Platelet Transfusion

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Aim: Exposure to fine particulate matter, particularly PM2.5, has been associated with increased platelet activation and cardiovascular risks. However, its effect on platelet recovery after transfusion remains unclear.

Purpose: This study aims to assess the influence of PM2.5 exposure on platelet recovery in patients with hematologic malignancies receiving prophylactic platelet transfusions.

Patients and Methods: We conducted a cross-sectional study involving 66 patients with hematologic malignancies who developed chemotherapy-induced thrombocytopenia and received prophylactic platelet transfusions between January and December 2021. A total of 191 transfusion events were analyzed. Platelet increment and corrected count increment (CCI) were measured one hour post-transfusion. Transfusions were categorized based on mean PM2.5 levels one day prior to platelet collection: the control group (< 37.5 μg/m³) and the case group (≥ 37.5 μg/m³). Multivariate analyses were used to adjust for potential confounders.

Results: No significant differences were observed in platelet increment ($p = 0.128$) or CCI ($p = 0.828$) between the PM2.5 exposure groups. Correlation analyses showed no significant association between PM2.5 levels and platelet increment ($r = 0.0565$, $p = 0.437$) or CCI ($r = 0.0370$, $p = 0.614$). These findings suggest that exposure to elevated PM2.5 levels one day before donation does not significantly impair platelet recovery.

Conclusion: Short-term exposure to elevated PM2.5 levels does not significantly affect platelet recovery in patients receiving prophylactic platelet transfusions. These results provide important reassurance regarding the immediate effects of air pollution on transfusion outcomes, while highlighting the need for further research into potential long-term impacts.

Keywords: platelet recovery, particulate matter, prophylactic platelet transfusion, hematologic malignancies, corrected count increment

Introduction

Air pollution, particularly fine particulate matter (PM), has emerged as a significant public health concern worldwide. PM consists of particles suspended in the air, with PM2.5 (particles less than 2.5 micrometers in diameter) and PM10 (particles less than 10 micrometers in diameter) being particularly harmful due to their ability to penetrate deep into the respiratory tract and enter the bloodstream.[1,2] Numerous studies have linked high levels of PM exposure to various adverse health outcomes, including cardiovascular diseases,[3–5] respiratory conditions,[2,6] and increased mortality.[7,8] PM exposure has also been shown to activate inflammatory pathways,[9] potentially influencing blood coagulation and platelet function.[10,11]

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Platelets play a crucial role in hemostasis and thrombosis. They are responsible for stopping bleeding by forming clots and are essential for maintaining vascular integrity.[12] Platelet transfusion is a common therapeutic intervention used to prevent or treat bleeding in patients with hematologic malignancies undergoing chemotherapy-induced thrombocytopenia.[12] Despite the widespread use of platelet transfusions, the effects of environmental factors, such as air pollution, on platelet recovery and transfusion outcomes remain poorly understood.

Recent evidence suggests that air pollution, particularly high levels of PM2.5, may influence platelet function and von Willebrand factor.[5,10,11] Some studies have indicated that exposure to high levels of PM can enhance platelet activation,[10] increasing the risk of thromboembolic events.[5,13,14] Platelets exposed to inflammatory stimuli, such as PM, may undergo premature activation, which could reduce their functionality and lifespan when transfused into patients. Furthermore, particulate matter might induce oxidative stress and inflammatory responses,[9,11] leading to alterations in platelet aggregation and function.[13]

Prophylactic platelet transfusions are essential in managing chemotherapy-induced thrombocytopenia in patients with hematologic malignancies, such as acute myeloid leukemia (AML), acute lymphoblastic leukemia (ALL), and lymphoma.[12] These patients often experience a significant reduction in platelet count due to chemotherapy, increasing their risk of bleeding. Prophylactic transfusions aim to prevent hemorrhagic events by maintaining an adequate platelet count.[12] However, several factors can influence the effectiveness of platelet transfusions, as measured by platelet recovery post-transfusion.

There are several factors influencing platelet recovery after prophylactic transfusion, such as blood group compatibility between donor and recipient, with ABO-incompatible transfusions potentially leading to lower platelet increments.[15] Another key factor is the duration of platelet storage.[16] Platelets stored for extended periods are more likely to undergo structural and functional degradation, reducing their efficacy upon transfusion.[16–18] Other factors, including the type of platelet product (single donor platelets or pooled platelet concentrates), the use of irradiated products, and the recipient's clinical condition, such as fever or infection, may also impact the transfusion outcome.[19,20] These factors are crucial considerations in evaluating platelet recovery and corrected count increment (CCI).[21]

In studies assessing platelet transfusion efficacy, several potential influencing factors including age, sex, body weight, the underlying hematologic malignancy, and previous transfusion history may all influence platelet recovery.[15–20] For instance, patients with larger body surface areas may require higher platelet doses for optimal recovery, and the presence of alloimmunization due to prior transfusions can reduce the effectiveness of subsequent transfusions. Understanding how these variables interact with environmental factors, such as PM exposure in platelet donors, is essential in interpreting the results of studies evaluating platelet recovery post-transfusion.

Despite the known impact of air pollution on cardiovascular events and platelet activation, there is limited research exploring the direct effects of PM2.5 on platelet recovery post-transfusion in patients with hematologic malignancies. Current evidence lacks a comprehensive evaluation of how ambient air pollution might influence platelet increment and CCI following prophylactic transfusions. This study aims to address the gap by investigating whether platelet donor's exposure to high levels of PM2.5 is associated with reduced platelet recovery in patients receiving prophylactic platelet transfusions. Specifically, the study seeks to determine whether high particulate matter levels negatively affect platelet increment and CCI in hematologic malignancy patients undergoing chemotherapy-induced thrombocytopenia.

Materials and Methods

Study Design and Participants

This cross-sectional study was conducted at Maharaj Nakorn Chiang Mai Hospital, located in Chiang Mai, Thailand, between January and December 2021. This study was reviewed and approved by the Institutional Research Ethics Committee No.4 of the Faculty of Medicine, Chiang Mai University (study code: MED-2563-07766), with approval granted on January 6th, 2021. All procedures adhered to the ethical principles outlined in the Declaration of Helsinki.

The study included adult patients diagnosed with hematologic malignancies, such as AML, ALL, and lymphoma, who developed chemotherapy-induced thrombocytopenia requiring prophylactic platelet transfusion. Patients were eligible for

inclusion if they were 18 years or older, had received chemotherapy resulting in thrombocytopenia (defined as platelet counts < 20,000/μL), and required at least one platelet transfusion during the study period.

Patients with platelet refractoriness were excluded. Platelet refractoriness was defined as a corrected count increment (CCI) of less than 7,500/μL measured 1 hour post-transfusion, based on the AABB guidelines.[21] This threshold indicates inadequate platelet recovery after at least two ABO-compatible platelet transfusions and reflects both immune- and non-immune-mediated causes.[21] Patients receiving therapeutic transfusions for active bleeding were also excluded.

Informed consent was obtained from all participants prior to enrollment. Clinical and demographic data were collected from electronic medical records, including patient age, sex, body weight, height, underlying hematologic malignancy, blood group, and clinical status at the time of transfusion. Details of each platelet transfusion, including the type of platelet products, volume, platelet number, ABO compatibility between donor and recipient, and whether irradiated products were used, were recorded. The duration of platelet storage (days) prior to transfusion was also documented.

Platelet Transfusion

Platelet products were collected, processed, and stored at the Blood Bank Section of Maharaj Nakorn Chiang Mai Hospital. Single donor platelet (SDP) from ABO-compatible donors were the preferred choice for most transfusion requests. However, leukoreduced pooled platelet concentrates (LPPC) were used in case where ABO-compatible SDP was not available. For patients with severe thrombocytopenia (< 10,000/μL) and without access to ABO-compatible platelets, ABO-incompatible platelet transfusions were permitted with the approval of the attending physician. All platelet transfusion followed the guidelines set by the Thai National Red Cross. The type of platelet product (either SDP or LPPC), the volume transfused, and any post-collection modifications, such as platelet irradiation, were determined by the attending physicians based on the patients' clinical condition.

Platelet Recovery Assessment

Platelet recovery post-transfusion was assessed by measuring the platelet increment and the CCI one hour after each transfusion. The platelet increment was calculated as the difference between the post-transfusion and pre-transfusion platelet counts. The CCI was calculated in accordance with the AABB guidelines using the following formula:[21]

$$\text{CCI} = [\text{platelet increment} \times \text{body surface area } (m^2)]/ (\text{number of platelets transfused} \times 10^{11}).$$

A CCI of less than 7,500/μL was considered suboptimal, indicating poor platelet recovery. Patients with suboptimal CCI values were excluded for the final analysis.

Ambient Air Pollution Data

The mean daily ambient PM2.5 data were obtained from the Research Institute for Health Sciences (RIHES) at Chiang Mai University, in collaboration with the Pollution Control Department (PCD) under the Ministry of Natural Resources and Environment, Thailand. The data were collected from six fixed air quality monitoring stations, strategically located to represent central Chiang Mai and its surrounding areas. These stations are equipped with small photodiode sensor detectors (PMS7003, Beijing Plantower Co., Ltd., Beijing, China), which are calibrated and maintained according to PCD standards. Hourly concentrations of PM2.5, PM10, carbon monoxide (CO), nitrogen dioxide (NO2), and ozone (O3) were measured using light-scattering techniques and adjusted for relative humidity prior to reporting. Daily meteorological data, including air temperature, humidity, and wind velocity, were also obtained from the Northern Meteorological Center in Chiang Mai, Thailand.

As part of this collaboration, precautionary warnings are issued to residents during periods of severe air quality deterioration, encouraging protective measures, such as wearing masks, using air purifiers, or reducing outdoor activities.

To ensure reliability, data from the Central Air Quality Monitoring Station at Sri Phum, located 1 km from the Blood Bank Center, were used as a reference. Measurements from this central station showed a strong correlation ($r > 0.9$) with data from five other monitoring stations in the region, covering an area of 152 km^2. For this study, the mean PM2.5 and

PM10 levels from the day prior to each platelet collection were calculated to assess the impact of air pollution exposure on platelet recovery in transfusion recipients.

The PM2.5 cut-off levels used in this study were based on the interim target standards set by the PCD of Thailand, which align with the World Health Organization (WHO) Interim Target-3 (IT-3) for 24-hour PM2.5 exposure.[22,23] According to these guidelines, the threshold for acceptable air quality is a PM2.5 level of 37.5 μg/m³. In this study, PM2.5 levels below 37.5 μg/m³ were categorized as the control group, while levels equal to or exceeding 37.5 μg/m³ were classified as the high pollution group. These thresholds reflect current national efforts to address air quality concerns and are particularly relevant in northern Thailand, including Chiang Mai, where seasonal variations can result in elevated PM2.5 concentrations due to urban emissions and biomass burning.

Statistical Analysis

The transfusions were categorized into two groups based on the ambient PM2.5 levels on the day of transfusion: a control group (PM2.5 < 37.5 μg/m³) and a case group (PM2.5 ≥ 37.5 μg/m³).

Based on the finding from the "TRAP study",[24] the mean platelet increment was reported as 14,700 ± 5,200 (SD). We anticipated a 15% reduction in platelet recovery during periods of high pollution compared to normal conditions. With an alpha error of 5% and a power of 80%, we calculate the required sample size using the STATA command:

"sampsi 14700 12495, sd(5200) p(0.8) ratio(2)"

The estimated total sample size was 198 transfusion sessions, comprising 66 sessions in the case group (exposed to high pollution) and 132 sessions in the control group.

Continuous variables, such as age, body weight, height, platelet increment, and CCI, were summarized as mean ± standard deviation (SD). Categorical variables, such as sex, ABO compatibility, and use of irradiated products, were presented as frequencies and percentages.

Comparisons between the case and control groups were performed using Student's *t*-test for continuous variables and Fisher's exact test for categorical variables.

To evaluate the impact of PM levels on platelet recovery, multivariate linear regression models were constructed, adjusting for potential confounders, including age, sex, body weight, ABO compatibility, platelet storage duration, and clinical condition (fever or infection). The correlation between PM levels and platelet increments or CCI was analyzed using Pearson's correlation coefficient.

A p-value of <0.05 was considered statistically significant. All analyses were performed using STATA version 16.

Results

Patient Characteristics

During the screening process, 12 patients were excluded due to known platelet refractoriness (CCI < 7,500/μL), leaving a total of 66 eligible patients for the final analysis. This included 32 male (48.5%) and 34 female (51.5%) patients. The mean age of the participants was 39.3 ± 14.5 years, with a mean body weight of 56.3 ± 10.1 kg, and a mean height of 160.6 ± 9.4 cm. Among the patients, 28 (42.4%) were diagnosed with AML, 18 (27.3%) with ALL, and 15 (22.7%) with lymphoma. Among the lymphoma patients, 7 (46.7%) were diagnosed with diffuse large B-cell lymphoma (DLBCL), 3 (20.0%) were peripheral T-cell lymphoma (PTCL), and the remaining 5 (33.3%) had various subtypes, including marginal zone lymphoma, follicular lymphoma, anaplastic large cell lymphoma, extranodal NK/T-cell lymphoma, and mantle cell lymphoma. Seven patients (10.6%) had bone marrow involvement, and 15 (22.7%) were undergoing stem cell transplantation.

Regarding chemotherapy regimens, 7 patients (10.6%) and 20 patients (30.3%) were undergoing induction and consolidation treatment for AML, respectively. Six patients (9.1%) received the Thai version of the pediatric adapted regimen, and 10 patients (15.2%) were treated with the HyperCVAD regimen for ALL. Eight patients (12.1%) received salvage chemotherapy for relapsed lymphoma, and 15 patients (22.7%) received conditioning regimens during stem cell transplantation. Clinical characteristics of the patients are presented in Table 1.

Table 1 Baseline Characteristics of Participants

Characteristics of each participant	Total = 66 patients (n, %)
Age (years): mean ± SD	39.3 ± 14.5
Male gender	32 (48.5)
Body weight (kg): mean ± SD	56.3 ±10.1
Height (cm): mean ± SD	160.6 ± 9.4
Diagnosis	
Acute myeloid leukemia (AML)	28 (42.4)
Acute lymphoblastic leukemia (ALL)	18 (27.3)
Lymphoma	15 (22.7)
Others	5 (7.6)
Lymphoma subtype	
Diffuse large B-cell lymphoma (DLBCL)	7 (46.7)
Peripheral T-cell lymphoma (PTCL)	3 (20.0)
Others	5 (33.3)
Chemotherapy	
AML induction (7+3 regimen)*	7 (10.6)
AML consolidation	20 (30.3)
Thai pediatric adapted regimen for ALL	6 (9.1)
HyperCVAD regimen**	10 (15.2)
Salvage chemotherapy for lymphoma	8 (12.1)
Conditioning regimen for stem cell transplantation	15 (22.7)
Blood group	
A	13 (19.7)
B	18 (27.3)
AB	2 (3.0)
O	33 (50.0)

Notes: * 7+3 regimen: A standard chemotherapy protocol for acute myeloid leukemia (AML), consisting of 7 days of cytarabine and 3 days of an anthracycline (eg, idarubicin). **HyperCVAD: A chemotherapy regimen comprising cyclophosphamide, vincristine, doxorubicin, and dexamethasone.
Abbreviation: SD, Standard deviation.

Platelet Products

A total of 191 platelet transfusions were administered to the patients, ranging from 1 to 9 sessions per patient, with a median of 5 transfusions (interquartile range [IQR]: 4–8 sessions). Of these, 148 transfusions (77.5%) occurred on days when PM2.5 levels were below 37.5 μg/m³, and 43 transfusions (22.5%) occurred on days when PM2.5 levels were ≥ 37.5 μg/m³.

Overall, 140 transfusion (73.3%) involved SDP, and 51 (26.7%) involved LPPC. A total of 58 platelet units (30.4%) were irradiated before use. No significant differences were observed in the proportions of SDP and LPPC used, the volume of platelet transfused, or the proportion of irradiated products between the cases and control groups. The storage duration of platelet products was also similar between the case group (3.25 ± 1.27 days) and the control group (3.31 ± 1.29 days) (p = 0.805). There was no significant association storage duration and platelet increment (p = 0.125).

Approximately two-third of the platelet donors were male. The mean age of donors for transfusion performed during high PM2.5 concentrations was 35.9 ± 7.8 years, which was significantly younger than the mean age of donors for transfusion performed during normal conditions (39.9 ± 9.1 years, p = 0.046). Additionally, there was a higher proportion of donors with blood groups A and B during high PM2.5 period compared to normal conditions (p = 0.001). The baseline characteristics of the platelet products are summarized in Table 2.

Platelet Transfusion Outcomes

ABO-compatible platelet transfusions were conducted in 179 sessions (93.7%), while ABO-incompatible transfusion occurred in 2 sessions (4.7%) during periods of high ambient PM2.5 and 10 sessions (6.8%) during periods of normal PM2.5. No significant differences were found between the proportions of ABO-compatible and incompatible transfusions (p > 0.999).

Table 2 Baseline Characteristics of Platelet Products and Donors

Characteristics	Total n (%)	Platelet transfusion session		p - value
		PM2.5 < 37.5 μg/m³	PM2.5 ≥ 37.5 μg/m³	
All Transfusion (n, %)	191	148 (77.5%)	43 (22.5%)	
Platelet products				
Single donor platelets (SDP)	140 (73.3)	109 (73.6)	31 (72.1)	0.839
Leukoreduced pooled platelet concentrates (LPPC)	51 (26.7)	39 (26.4)	12 (27.9)	
Volume of transfused platelet (mL): mean ± SD	265 ± 54	264 ± 54	271 ± 54	0.443
Irradiated products	58 (30.4)	48 (32.4)	10 (23.3)	0.249
Storage duration (days): mean ± SD	3.30 ± 1.28	3.31 ± 1.29	3.25 ± 1.27	0.805
Platelet storage duration				
1–2 Days	55 (28.8)	42 (28.38)	13 (30.23)	0.813
≥ 3 Days	136 (71.2)	106 (71.62)	30 (69.77)	
Donors				
Age (years): mean ± SD	39.1 ± 8.9	39.9 ± 9.1	35.9 ± 7.8	0.046
Male gender	130 (68.1)	100 (67.6)	30 (69.8)	0.925
Body weight (kg): mean ± SD	78.3 ± 13.6	77.2 ± 12.6	82.2 ± 16.3	0.075
Blood group				
• A	27 (14.1)	14 (9.5)	13 (30.2)	0.001
• B	59 (30.9)	43 (29.0)	16 (37.3)	
• AB	4 (2.1)	3 (2.0)	1 (2.3)	
• O	101 (52.9)	88 (59.5)	13 (30.2)	

Abbreviations: PM2.5, Particulate matter with a diameter less than 2.5 micrometers; SDP, Single Donor Platelets; LPPC, Leukoreduced Pooled Platelet Concentrates; SD, Standard Deviation.

Table 3 Platelet Transfusion Outcomes Based on Donors' Short-Term Exposure to PM2.5

Characteristics	Total (n, %)	Platelet transfusion session		p - value
		PM2.5 < 37.5 μg/m³ (Control)	PM2.5 ≥ 37.5 μg/m³ (Case)	
All Transfusion (n, %)	191	148 (77.5%)	43 (22.5%)	
ABO Compatibility				> 0.999
• ABO-Compatible	179 (93.7)	138 (93.2)	41 (95.3)	
• ABO-Incompatible	12 (6.3)	10 (6.8)	2 (4.7)	
Platelet increment (/μL) (mean ± SD)	27,827 ± 12,540	27,081 ± 12,677	30,395 ± 11,840	0.128
• < 20,000	54 (28.3)	47 (31.8)	7 (16.3)	0.055
• ≥ 20,000	137 (71.7)	101 (68.2)	36 (83.7)	
Corrected count increment (CCI)	18,353 ± 8,385	18,281 ± 8,718	18,597 ± 7,229	0.828

Abbreviations: PM2.5, Particulate matter with a diameter less than 2.5 micrometers; SD, Standard Deviation.

The efficacy of platelet transfusions was assessed using platelet increment and CCI. The overall mean platelet increment was 27,827 ± 12,540 cells/μL. There was no statistically significant difference in platelet increment between the case group (30,395 ± 11,840 cells/μL) and the control group (27,081 ± 12,677 cells/μL) (p = 0.128). Similarly, there was no significant difference in the proportion of patients with platelet increments greater than 20,000 cells/μL between the two groups. The mean CCI was also comparable between the case group (18,597 ± 7,229 cells/μL) and the control group (18,281 ± 8,718 cells/μL) (p = 0.828). The platelet transfusion outcomes are summarized in Table 3.

Association Between PM2.5 and Platelet Recovery

Correlation analyses revealed no significant association between PM2.5 levels and platelet increment (r = 0.0565, p = 0.437) or CCI (r = 0.0370, p = 0.614). The correlation plot of platelet increments and CCI following prophylactic platelet transfusion, stratified by different PM2.5 levels are shown in Figures 1 and 2.

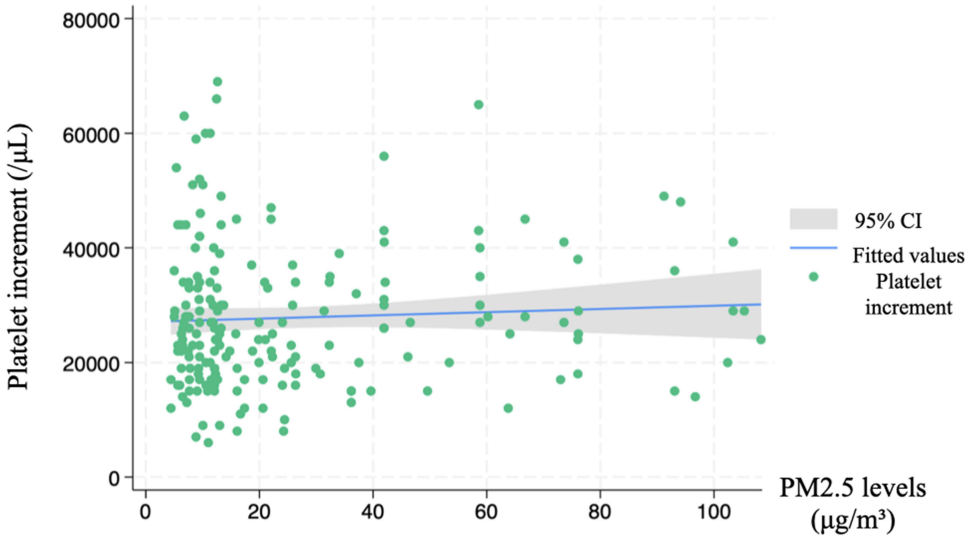

Figure 1 Scatter plot of platelet increment after prophylactic platelet transfusion in patients with chemotherapy-induced thrombocytopenia in relation to ambient PM2.5 levels.

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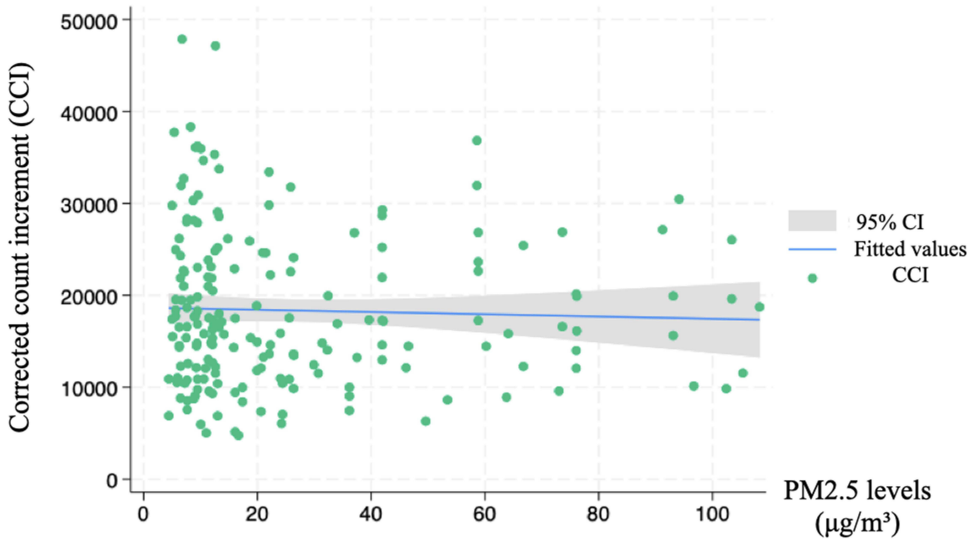

Figure 2 Scatter plot of corrected count increment (CCI) after prophylactic platelet transfusion in patients with chemotherapy-induced thrombocytopenia in relation to ambient PM2.5 levels.

Discussion

Previous studies have highlighted the harmful effects of air pollution, particularly fine particulate matter (PM2.5), on cardiovascular health and coagulation processes.[5,13] PM2.5 has been shown to promote platelet activation and increase the risk of thromboembolic events through mechanisms such as oxidative stress and systemic inflammation.[9,11,13] However, our results did not show a significant impact of PM2.5 on platelet recovery following transfusion, suggesting that in the context of prophylactic transfusion, PM2.5 exposure may not induce the same acute effects observed in cardiovascular outcomes.

The lack of significant findings could be attributed to several factors. First, platelet transfusions in patients with hematologic malignancies are a controlled and acute intervention, where platelets may not be exposed to the long-term or chronic effects of pollution typically associated with PM2.5-induced inflammation. Second, while PM2.5 exposure has been linked to platelet activation, its impact on the functional integrity of transfused platelets and their ability to recover post-transfusion may differ from that of endogenously circulating platelets.

Our findings contrast with studies that have demonstrated the detrimental effects of environmental pollutants on blood components and coagulation. For example, exposure to high levels of air pollution has been associated with increased risks of thrombosis and adverse cardiovascular events, likely due to inflammatory responses and endothelial dysfunction. While these studies suggest that environmental pollutants can impair platelet function, our study did not find any significant impact of PM2.5 on platelet recovery in transfusion recipients, indicating that transfused platelets may be less vulnerable to the acute inflammatory effects of PM2.5.

This discrepancy may also be due to the relatively short time frame between PM2.5 exposure and transfusion in our study. Platelets, once transfused, may have already undergone quality control processes that mitigate the effects of environmental stressors. Additionally, the conditions under which platelets are collected, processed, and stored may protect them from the direct impact of air pollutants.

Although our findings did not show a significant effect of PM2.5 on platelet recovery, they underscore the importance of considering environmental factors in transfusion medicine. Patients with hematologic malignancies often rely on frequent platelet transfusions to manage thrombocytopenia, and identifying any potential environmental risks to transfusion outcomes remains a critical area of research. While PM2.5 exposure did not significantly affect transfusion efficacy in our study, it remains crucial for clinicians to monitor the potential long-term impacts of air pollution, especially in regions with poor air quality.

Further, while our study focused on short-term outcomes, chronic exposure to elevated PM levels may have cumulative effects that warrant further investigation. Prolonged exposure to air pollution could potentially affect both the donors' platelet function and the recipients' clinical outcomes, particularly in individuals who require repeated transfusions over an extended period.

Several limitations should be considered in interpreting the results of this study. First, this was a cross-sectional study, and the short duration of PM exposure assessment may not fully capture the potential long-term effects of air pollution on platelet recovery. Second, our sample size, while sufficient for detecting moderate effects, may not have been large enough to detect smaller but clinically relevant associations. Third, the study did not account for the blood donor's actual exposure to PM2.5 prior to donation. While we analyzed PM2.5 levels on the day prior to platelet collection, assuming that all donors were exposed to ambient pollution, cumulative or preceding exposure among donors could influence platelet functionality, as prolonged exposure to high PM2.5 levels has been linked to systemic inflammation and platelet activation. Incorporating donor-specific exposure histories could provide a more comprehensive understanding of how environmental factors impact platelet quality and transfusion outcomes. Finally, we did not assess other potential environmental pollutants, such as ozone or nitrogen dioxide, which may have synergistic effects with PM2.5.

Future research should explore the long-term effects of chronic PM2.5 exposure on both platelet donors and transfusion recipients. Longitudinal studies with larger sample sizes could help clarify whether cumulative exposure to air pollution influences platelet recovery and transfusion outcomes. Additionally, mechanistic studies examining how PM2.5 interacts with stored platelet products and affects their functionality post-transfusion would provide further insight into the environmental factors influencing transfusion medicine.

Conclusion

This study indicates that short-term exposure to elevated PM2.5 levels is not significantly associated with platelet recovery in patients receiving prophylactic platelet transfusions. While these findings suggest limited immediate impact, further research is warranted to explore the potential long-term effects of environmental pollutants on transfusion outcomes, particularly in regions with persistently high air pollution levels.

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Author Contributions

All authors made a significant contribution to the work reported, whether in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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