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Article

The Effect of Single and Double Dose Platelet Apheresis on Platelet Count A Thrombopoietin Levels and Percentage of Immature Platelet Fraction of Donor Post Donation

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Abstract. Thrombapheresis is commonly used to obtain platelet concentrates; however, different collection doses may affect donor hematological responses. This study aimed to compare the effects of single-dose and double-dose thrombapheresis on platelet count, thrombopoietin (TPO) levels, and immature platelet fraction (IPF). A prospective comparative study with repeated measures was conducted in eight healthy donors (n = 8; 4 per group) at the Indonesian Red Cross Blood Donation Unit, Tangerang. Blood samples were collected before, immediately after, and on day 2 and day 4 post-donation. Platelet count and IPF were measured using an automated hematology analyzer, while TPO levels were assessed by ELISA. Data were analyzed using two-way ANOVA and Pearson correlation ($p < 0.05$). Platelet counts decreased immediately after thrombapheresis (23% in single-dose; 24% in double-dose) and gradually recovered. Significant effects of time and dose were observed ($p < 0.05$), but no difference between groups at day 4 ($p > 0.05$). TPO increased in both groups (50% vs 27%), and IPF changes were not significant ($p > 0.05$). A positive but non-significant correlation between TPO and IPF was found. Both procedures cause transient platelet reduction with gradual recovery. Findings should be interpreted cautiously due to the small sample size.

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1. Introduction

Blood transfusions, especially platelet transfusions, aim to prevent bleeding in patients with thrombocytopenia [1]. Blood transfusion is defined as the transfer of whole blood or blood components from healthy donors to recipients who require transfusion therapy [2]. According to World Health Organization (WHO) standards, blood needs will be met if at least 2% of Indonesia's population are regular donors [3]. As a country with a population of 284.438.800 [4]. Indonesia requires approximately 5.7 million bags of blood annually. The Indonesian Red Cross (PMI) Blood Donation Unit (UDD) is responsible for maintaining the national blood supply, including platelet components. Nevertheless, the increasing incidence of dengue, cancer, and aplastic anemia has led to a growing demand for platelet concentrates in hospitals [5–7].

Thrombocytopenia is a condition characterized by a platelet count below the normal range of 150.000–450.000/µL. This condition may occur due to various mechanisms, including decreased platelet production in the bone marrow, increased platelet destruction, increased platelet consumption, drug effects, infections, nutritional deficiencies, pregnancy, alcohol dependence, or splenomegaly [8].

In thrombocytopenia, thrombopoietin (TPO) levels increase to stimulate thrombopoiesis and platelet production [9-10]. This process results in the formation of young platelets that are larger and more active than mature platelets. The proportion of these young platelets can be measured using a hematology analyzer and is reported as the immature platelet fraction (IPF). IPF is a useful parameter to assess thrombopoiesis activity and to differentiate thrombocytopenia caused by increased platelet destruction from thrombocytopenia due to bone marrow suppression or damage [11–13].

Platelet transfusion has been shown to control bleeding and reduce mortality in patients with thrombocytopenia [14] Platelet transfusion is generally administered in the form of platelet concentrate (PC), which can be produced either from whole blood processing or collected directly from donors through the apheresis method) [12],[15]. Conventional platelet concentrates are derived from multiple donors, whereas apheresis platelet concentrates are obtained from a single donor. Exposure to platelets from multiple donors may increase the risk of leukocyte residue and HLA antigen exposure, which can lead to adverse transfusion reactions such as febrile non-hemolytic transfusion reactions (FNHTR), refractory thrombocytopenia, and transfusion-related acute lung injury (TRALI) [12],[15].

Thrombapheresis is a blood component collection technology that separates platelets from whole blood using a centrifugation-based machine while returning other blood components to the donor's circulation [16]. Currently, thrombapheresis products account for only about 22,5% of blood component production in Indonesia [17]. After thrombapheresis donation, donor platelet levels temporarily decrease and stimulate thrombopoiesis through increased thrombopoietin activity, which triggers megakaryopoiesis [17].

Thrombopoietin is a physiological regulator of platelet production, so research on thrombopoietin levels in thrombapheresis donors can provide an overview of megakaryopoiesis activity after donation to determine changes in thrombopoietin levels after donation and their relationship to platelet counts after donation. This is also related to platelet donor management based on Indonesian Minister of Health Regulation No. 91 of 2015, which requires apheresis donors to have a minimum platelet count of 150.000/mm^3 prior to donation and a minimum body weight of 55 kg for men and 60 kg for women [18].

The thrombapheresis procedure allows platelet collection in single-dose (yield 3×10^{11} platelets) or double-dose (yield 6×10^{11} platelets) products [19]. Double-dose thrombapheresis is considered more efficient because two therapeutic doses can be obtained from one donor and transfused to two recipients, which can help increase platelet availability in settings with limited apheresis donors [19-20]. However, the larger platelet collection in double-dose procedures may result in a greater decrease in platelet counts in donors.

Several studies have evaluated changes in platelet counts and thrombopoietin levels following thrombapheresis donation. However, the larger platelet collection in double-dose procedures may result in a greater decrease in platelet counts in donors [19-20]. Several studies have reported changes in platelet counts and thrombopoietin levels following thrombapheresis donation [17]. However, studies assessing the relationship between thrombopoietin levels, platelet count recovery, and immature platelet fraction after single-dose and double-dose thrombapheresis remain limited, particularly in Indonesia. Therefore, this study aims to evaluate the effect of single-dose and double-dose thrombapheresis on thrombopoietin levels, platelet count recovery, and immature platelet fraction in platelet donors.

2. Experimental Section

This analytic descriptive study evaluated the effects of single-dose and double-dose thrombocyte apheresis on platelet count, thrombopoietin (TPO) levels, and immature platelet fraction (IPF) in donors at the Unit Donor Darah (UDD) PMI Kota Tangerang, Indonesia, from July 2024 to July 2025.

The study population consisted of healthy thrombocyte donors aged 18–65 years who met the Indonesian Red Cross (PMI) standard operating procedures (SOP) for apheresis donation. Inclusion criteria included body weight ≥ 60 kg and hemoglobin ≥13 g/dL. Based on PMI eligibility standards, donors undergoing single-dose thrombapheresis had a minimum pre-donation platelet count of $\geq 200 \times 10^3/\mu L$, whereas donors eligible for double-dose thrombapheresis had a minimum platelet count of $\geq 250 \times 10^3/\mu L$. Exclusion criteria included the use of antiplatelet drugs such as aspirin or clopidogrel within 10 days before donation, difficult venous access, pregnancy or lactation, and other conditions not meeting PMI SOP requirements.

The minimum sample size was calculated based on previous thrombapheresis studies with $\alpha = 0.05$ and statistical power of 0.90, assuming a standard deviation of $42 \times 10^3/\mu L$ and a minimum detectable difference of $110 \times 10^3/\mu L$, resulting in four donors per group. Due to limited availability of eligible donors, convenience sampling was applied. Participants were assigned to single- or double-dose procedures based on eligibility criteria defined in the PMI SOP, therefore randomization was not performed.

Venous blood samples (2 mL, EDTA tube) were collected at four time points: before thrombapheresis (baseline), immediately after the procedure, on day 2, and on day 4 post-donation. Platelet counts were measured using an automated hematology analyzer (Sysmex XR), while immature platelet fraction (IPF) was measured using the Sysmex XN-1000 analyzer based on a flow cytometry principle.

For thrombopoietin measurement, blood samples were centrifuged at 1000 × g for 15 minutes at 8°C to obtain serum, which was then stored at −30°C until analysis. Serum thrombopoietin levels were determined using a sandwich enzyme-linked immunosorbent assay (ELISA) kit (Elabscience Human TPO ELISA Kit, E-EL-H1588) according to the manufacturer's instructions.

All thrombapheresis procedures were performed using centrifugation-based apheresis machines with acid citrate dextrose solution A (ACD-A) as anticoagulant. The target platelet yield was approximately 4×10^{11} platelets for single-dose procedures and 6×10^{11} platelets for double-dose procedures. The procedure duration ranged from 60 to 90 minutes.

Data analysis was performed using GraphPad Prism version 10. Normality was assessed using the Shapiro–Wilk test. Differences in platelet count, thrombopoietin levels, and IPF between groups and observation times were analyzed using two-way ANOVA followed by Bonferroni post-hoc tests. Pearson correlation analysis was used to assess the relationship between thrombopoietin levels and platelet count recovery. A p-value <0.05 was considered statistically significant. Ethical approval for this study was obtained from the Ethics Committee of the Faculty of Medicine, Universitas Indonesia. All participants provided written informed consent prior to participation.

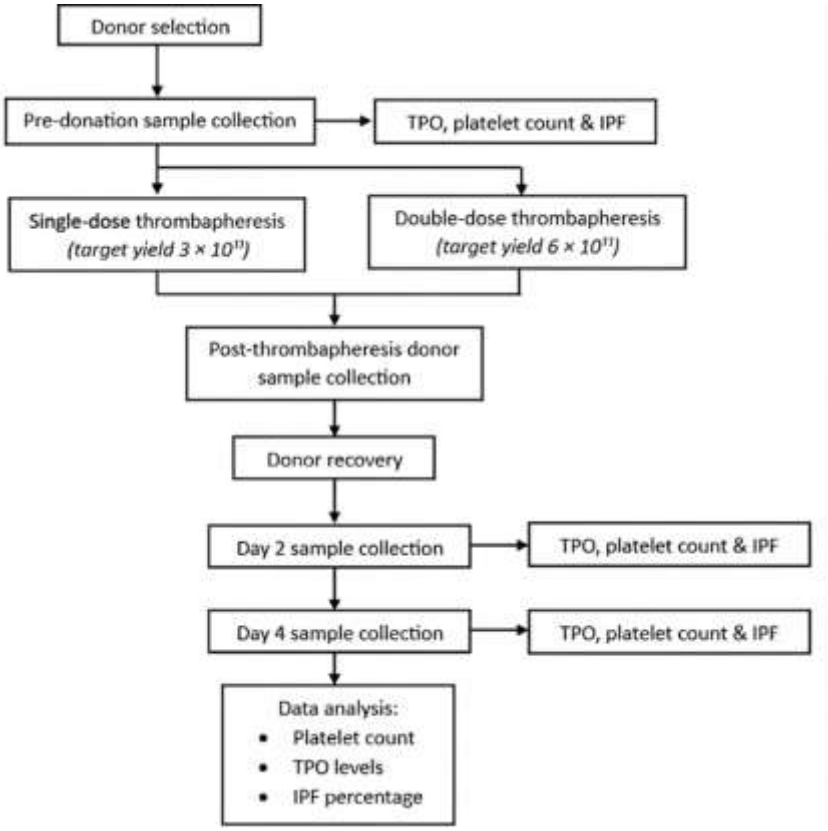


Figure 1. Flowchart of research

3. Results and Discussion

Table 1 presents the mean platelet counts and percentage changes compared with baseline at four observation times (pre-apheresis, post-apheresis, H+2, and H+4) in donors undergoing single-dose and double-dose thrombapheresis.

Table 1. Mean platelet count results and percentage changes in platelet count compared to baseline

Measurement time	Single doses		Multiple doses	
	(x 10^3 µL)	(%)	(x 10^3 µL)	(%)
Pre	272 ± 49.75	100	425.3 ± 34.63	100
Pasca	207.6 ± 26.32	77.08 ± 5.67	323.3 ± 22	76.14 ± 2.95
H+2	233.7 ± 22.85	87.39 ± 10.63	329.5 ± 20.79	77.68 ± 4.14
H+4	280.5 ± 55.91	103.64 ± 14.28	343.2 ± 18.76	81.43 ± 4.57

Immediately after thrombapheresis, platelet counts decreased in both groups. In the single-dose group, the mean platelet count decreased from 272 ± 49,75 ×10^3/µL at baseline to 207,6 ± 26,32

$\times 10^3/\mu L$ post-apheresis, representing a reduction of approximately 23%. Similarly, in the double-dose group, platelet counts decreased from 425,3 ± 34,63 $\times 10^3/\mu L$ to 323,3 ± 22 $\times 10^3/\mu L$, corresponding to a decrease of approximately 24%.

During the follow-up period, platelet counts gradually increased in both groups. In the single-dose group, platelet levels increased to 233.7 ± 22.85 $\times 10^3/\mu L$ at H+2 and reached 280.5 ± 55.91 $\times 10^3/\mu L$ at H+4, indicating recovery toward baseline values. In contrast, the double-dose group showed a slower recovery pattern, with platelet counts of 329.5 ± 20.79 $\times 10^3/\mu L$ at H+2 and 343.2 ± 18.76 $\times 10^3/\mu L$ at H+4, which remained below baseline levels.

Two-way ANOVA demonstrated significant effects of dose ($p < 0.05$) and time ($p < 0.05$) on platelet counts. However, the difference between groups at H+4 was not statistically significant ($p > 0.05$). These findings suggest that although platelet recovery appeared slower following double-dose thrombapheresis, statistical evidence of between-group differences at the final observation time was not observed.

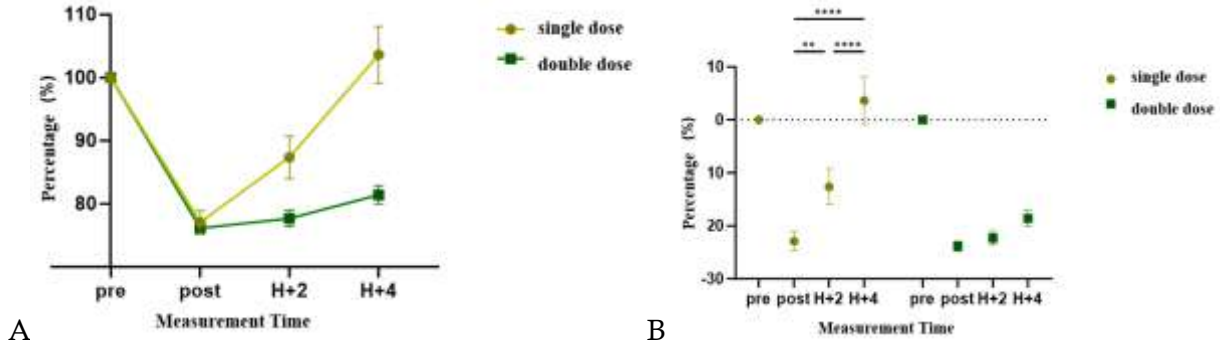


Figure 2. *(*A)Mean percentage of platelet count (Mean + SEM) per measurement time in both groups. (B)Average change in platelet count compared to pre-thrombophoresis condition.

The results of platelet count measurements after thrombapheresis showed a similar average decrease between the single-dose and double-dose groups, which was around 23%. At the H+2 measurement, a faster increase in thrombosis was found in the single dose compared to the double dose, with a difference of approximately 10%. At the H+4 measurement, the average platelet count had recovered to its pre-thrombapheresis condition, while in the double-dose group, the average platelet count had not recovered, which was still about 19% lower than before thrombophoresis.

After thrombophoresis, both in the single and double doses, there was a decrease in platelet count of about 23% from the baseline value. A study by Chopra et al. (2021) showed an average decrease of 35.55% in single-dose thrombapheresis (SDP) and 37.76% in double-dose thrombapheresis (DDP) [21]. This decrease is temporary and generally does not cause serious clinical effects. Despite a significant decrease in platelet count after DDP, no serious side effects were found, and hematological parameters remained within safe limits. The efficiency of collection and reduced exposure of allogeneic donors are important advantages of the DDP procedure, especially in the context of platelet inventory management [20].

In the single-dose group, platelet counts showed a consistent upward trend and reached baseline values on day four (D+4). Donor platelet counts recovered within 48 hours after a single-dose thrombapheresis [22]. The body exhibits an effective compensatory mechanism to restore platelet hemostasis after moderate loss. Platelet recovery can begin as early as the second day and reach baseline within 7 to 14 days depending on the donor's condition [23].

Conversely, in the double-dose group, platelet counts had not returned to baseline values even by the fourth day post-donation. Greater platelet depletion requires a longer recovery time and stricter clinical monitoring. The importance of donor selection and post-donation evaluation to ensure safety in the DDP procedure [21]. DDP is an effective and relatively safe method, but requires special

attention to the effects of citrate and evaluation of hematological parameters [20]. Therefore, monitoring up to day 7 or 10 post-donation is necessary to ensure full recovery. Although logistically more efficient, double-dose thrombapheresis demands greater attention to donor safety and recovery.

The results of platelet count measurements after thrombapheresis showed a similar average decrease between the single-dose and double-dose groups, which was around 23%. At the H+2 measurement, a faster increase in thrombosis was found in the single dose compared to the double dose, with a difference of approximately 10%. At the H+4 measurement, the average platelet count had recovered to its pre-thrombapheresis condition, while in the double-dose group, the average platelet count had not recovered, which was still about 19% lower than before thrombophoresis.

The decrease in platelet counts immediately after thrombapheresis is consistent with the mechanism of platelet removal during the apheresis procedure. Previous studies have reported similar reductions in platelet counts following plateletpheresis donation due to the direct collection of circulating platelets. The gradual recovery observed during follow-up likely reflects compensatory platelet production stimulated by thrombopoietin-mediated megakaryopoiesis.

However, given the limited sample size (n = 4 per group), the statistical power of the study is limited and smaller differences between groups may not have been detected.

Table 2. Average TPO concentration measurements and percentage changes in TPO concentration

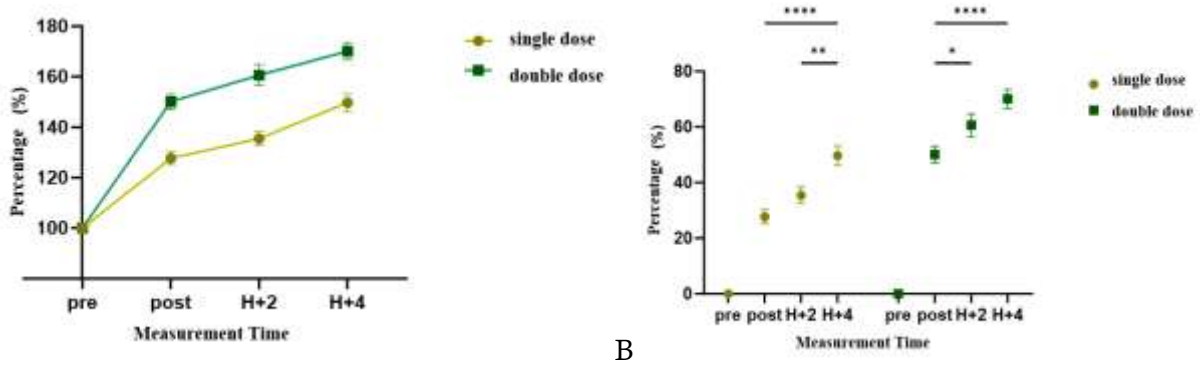
Measurement time	Single doses		Multiple doses	
	(x 10^3 µL)	(%)	(x10^3 µL)	(%)
Pre	66.9 ± 7.78	100	86.8 ± 34.63	100
Pasca	85.1 ± 7.91	127.70 ± 7.97	130 ± 10.17	150.09 ± 9.45
H+2	90.3 ± 9.27	135.44 ± 8.93	139.1 ± 12.35	160.65 ± 12.98
H+4	99.6 ± 8.36	149.69 ± 11.22	147.4 ± 12.52	170.11 ± 10.80

The results of TPO level measurements showed a faster increase in levels after thrombopheresis in the double-dose group compared to the single-dose group. A faster increase in TPO levels was observed in the double-dose group, approximately 22% after thrombopheresis, approximately 26% at H+2, and approximately 20% at H+4.

Serum thrombopoietin (TPO) levels increased following thrombapheresis in both groups. The increase appeared more pronounced in the double-dose group, with an approximate 50% increase relative to baseline, whereas the single-dose group demonstrated an increase of approximately 27%. Although the overall trend suggests increased TPO production following platelet depletion, substantial inter-individual variability was observed, as indicated by relatively large standard deviations. This variability may reflect individual differences in physiological regulation of thrombopoiesis among donors.

The increase in thrombopoietin (TPO) levels following thrombophoresis in both groups indicates a physiological compensatory response to the decrease in platelet count, with a greater and more rapid increase observed in the double-dose group. This is consistent with the regulatory mechanism of thrombopoiesis, in which a decrease in platelet mass reduces TPO clearance via the c-Mpl receptor, thereby increasing circulating TPO levels and stimulating megakaryocyte production. The higher degree of increase in the double-dose group reflects a dose-dependent effect resulting from greater platelet depletion. Furthermore, the rapid response (observed from day 2 to day 4) indicates good hematopoietic adaptability in healthy donors. However, the presence of considerable inter-individual variation suggests that the TPO response is also influenced by individual physiological factors, such

as megakaryocyte reserves and hematopoietic regulation, and thus must be considered in donor safety evaluations and donation intervals. [24–26].



B

Figure 3. (A) Mean percentage change in TPO levels (Mean+SEM) per measurement time in both groups. (B) Average percentage change in TPO levels compared to pre-thrombophereesis conditions.

The data obtained from measuring TPO levels at Pre, Post, H+2, and H+4 thromboferesis were statistically tested using Two-Way ANOVA. The test showed a significant effect of TPO levels in terms of dose ($p < 0.05$), in terms of measurement time ($p < 0.05$), and a significant combined effect of dose and measurement time on TPO levels ($p < 0.05$). Further Post Hoc analysis showed that there were significant differences in TPO levels between Pre and Post measurement times ($p < 0.05$), Pre and H+2 ($p < 0.05$), Pre with H+4 ($p < 0.05$), Post with H+4 ($p < 0.05$), and H+2 with H+4 ($p < 0.05$) in the single-dose group. In the double-dose group, there were significant differences in TPO levels between Pre and Post measurements ($p < 0.05$), Pre with H+2 ($p < 0.05$), Pre with H+4 ($p < 0.05$), Post with H+2 ($p < 0.05$), Post with H+4 ($p < 0.05$), and H+2 with H+4 ($p < 0.05$). Trombopoietin (TPO) levels increased after the thrombopheresis procedure, both at single and double doses. This increase is a physiological response to the decrease in the number of platelets in circulation, which reduces TPO consumption by platelets, thereby increasing its concentration in plasma. Increased TPO levels will stimulate the proliferation and maturation of megakaryocytes in the bone marrow as a form of compensation for platelet loss.

Donor TPO levels increased significantly on days 3 to 5 after thrombopheresis, in line with a 30% decrease in platelet count after donation [17]. In addition, this recovery response appeared to be slower in female donors, indicating individual physiological variations in compensatory mechanisms. Post-apheresis increase in TPO was negatively correlated with stem cell factor (SCF) levels, reflecting early hematopoietic activation [9]. TPO levels peaked on the first day after donation, especially after double-dose procedures, due to greater platelet loss.

Interestingly, studied the difference in platelet counts after TC transfusion and apheresis platelets in patients with thrombocytopenia [27]. In that study, apheresis platelet transfusion resulted in a higher increase in platelets compared to platelet concentrates, with an average increase of 49.286/μL vs 20.143/μL. This indicates that manipulating platelet counts through apheresis may also potentially stimulate a stronger physiological TPO regulatory response, albeit in the recipient population rather than donors. These findings support the hypothesis that a greater platelet loss or requirement triggers more intensive TPO regulation.

Thus, monitoring TPO levels is not only relevant in the context of post-thrombapheresis donors, but also has potential as an indicator of recovery and transfusion therapy effectiveness in

thrombocytopenic patients. This reinforces the importance of TPO as an integral part of the platelet homeostasis mechanism.

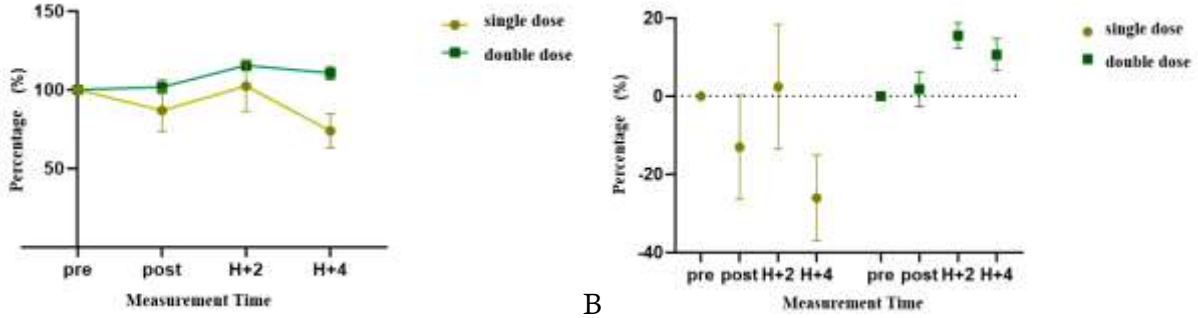
Tabel 3. Average IPF level measurement results

Measurement time	Single doses (%)	Multiple doses (%)
Pre	3.26 ± 3.21	1.02 ± 0.12
Pasca	1.69 ± 1.20	1.05 ± 0.24
H+2	2.13 ± 1.82	1.18 ± 0.19
H+4	1.47 ± 0.96	1.14 ± 0.25

Tabel 4. Mean percentage change in IPF relative to baseline in the single-dose and multiple-dose groups

Measurement time	Single doses (%)	Multiple doses (%)
Pre	100	100
Pasca	86.96 ± 42.21	101.82 ± 14.01
H+2	102.41 ± 50.28	115.53 ± 10.29
H+4	73.99 ± 34.79	110.65 ± 12.98

The results of IPF level measurements after thromboferesis showed a decrease of approximately 13% in the single-dose group, but remained relatively stable in the double-dose group. Subsequent measurements of the single-dose group at H+2 showed an increase back to approximately the IPF level before thrombophoresis, and a decline again at H+4 with a decrease of approximately 26% compared to the IPF level before thrombophoresis. In the double-dose group, the IPF level tended to increase by approximately 15% at H+2 and 10% at H+4 compared to the initial IPF level before thrombophoresis.



A B

Figure 4. (A) Mean percentage change in IPF levels (Mean+SEM) per measurement time in both groups. (B) Average percentage change in IPF compared to pre-thrombophereesis conditions.

The immature platelet fraction (IPF), which reflects newly produced platelets released from the bone marrow, showed fluctuations during the observation period in both groups. However, statistical analysis did not demonstrate significant changes in IPF over time ($p > 0.05$). Immature Platelet Fraction (IPF) reflects the proportion of immature platelets in the blood circulation and has been recognized as a hematological parameter that reflects real-time thrombopoiesis activity [28]. In this

study, changes in IPF were evaluated in two groups of platelet donors: single-dose and double-dose, to observe the regenerative response to platelet mass loss due to the thrombapheresis procedure.

In the single-dose group, there was an initial decrease in IPF immediately after donation, which then returned to baseline values on day 2 (D+2). However, a renewed decrease on day 4 (D+4) indicated short-term compensatory fluctuations. This pattern suggests that although the thrombopoiesis mechanism successfully restored platelet production rapidly, homeostasis was not yet fully stable within 96 hours.

Conversely, in the double-dose group, IPF showed an increasing trend from H+2 to H+4. This increase is strongly suspected to be a response to greater platelet loss, which triggers increased secretion of thrombopoietin (TPO), the main hormone that stimulates megakaryocyte proliferation and differentiation as well as the release of young platelets [29]. The linear relationship between increased TPO and IPF has been confirmed in various studies as an indicator that IPF can serve as a valid regenerative biomarker in the context of increased thrombopoiesis demand [30].

These findings reinforce the idea that IPF, especially when paired with TPO levels, can be used as a complementary parameter to monitor thrombopoiesis activity after interventions such as thrombopheresis. This has practical implications in blood donor clinics, particularly in assessing the speed and stability of hematological recovery and in anticipating the long-term effects of repeated donations. A Pearson correlation analysis was performed to examine the relationship between platelet count, TPO levels, and IPF levels in the single-dose and double-dose groups, and a strong relationship was found between IPF levels and TPO levels in the double-dose group, but the relationship was not statistically significant ($p > 0.05$).

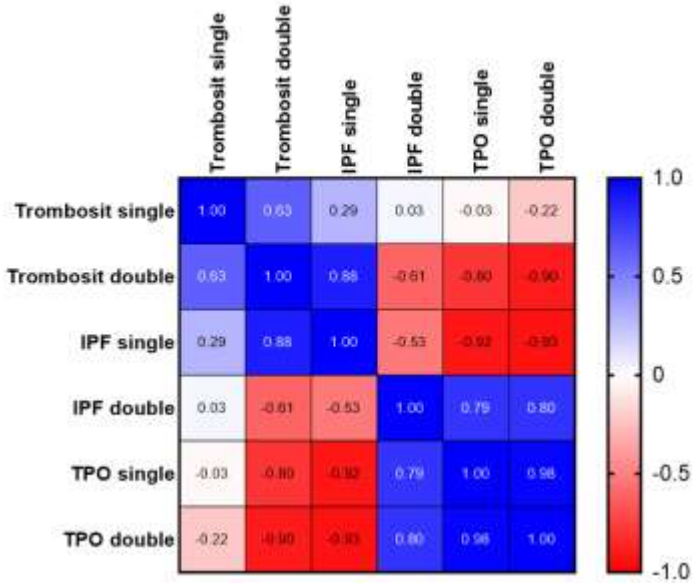


Figure 5. The relationship between platelet count, IPF percentage, and TPO levels in single-dose and double-dose conditions

Based on the image above, the bluer the color, the stronger the relationship between the two parameters. Meanwhile, the redder the color, the less related they are. From this data, there appears to be relationship between double-dose TPO and double-dose IPF, but the relationship is not significantly meaningful ($p > 0.05$).

The results of Pearson's correlation analysis between thrombopoietin (TPO) levels and Immature Platelet Fraction (IPF) in the double-dose platelet donor group show a numerically strong positive correlation. However, this relationship did not reach statistical significance ($p > 0.05$), indicating that mathematically there is insufficient evidence to conclude that there is a definite linear association in

this study population. This phenomenon reflects statistical limitations that may be caused by biological variability between individuals and a relatively small sample size, which reduces the power of statistical tests and increases the potential for type II errors [28].

Several previous studies support a rational biological relationship between TPO and IPF, as TPO is known to play a direct role in stimulating megakaryocyte differentiation and the production of young platelets into the circulation [31]. However, statistical correlations between biological variables are often influenced by individual heterogeneity, comorbidities, and the timing of post-intervention sampling, which can vary between subjects. On the other hand, alternative parameters such as absolute immature platelet number (AIPN) have been reported to have a more stable correlation with megakaryopoiesis activity in some clinical contexts, although they have not completely replaced the role of IPF [31]. The statistical insignificance in this study does not necessarily negate the clinical or biological value of the TPO–IPF relationship, but emphasizes the need to replicate the results in studies with larger sample sizes and longitudinal designs.

This study provides preliminary insight into the hematological responses of platelet donors following single-dose and double-dose thrombapheresis. However, several limitations should be acknowledged. The small sample size, single-center design, and convenience sampling method may limit the generalizability of the findings. In addition, potential confounding factors such as donor hydration status, nutritional condition, and previous donation interval were not controlled. Future studies with larger and more diverse donor populations are therefore necessary to validate these findings.

4. Conclusion

Single-dose thrombapheresis resulted in an approximate 23% decrease in platelet counts immediately after donation, with values tending to return toward baseline by day 4. It was also associated with an increase in thrombopoietin (TPO) levels of approximately 27% compared to pre-donation levels, while the immature platelet fraction (IPF) showed a decrease of around 13%, although this change was not statistically significant. In contrast, double-dose thrombapheresis led to a slightly greater reduction in platelet counts (approximately 24%) and a slower recovery pattern, with platelet levels not yet returning to baseline by day 4. The increase in TPO levels was more pronounced in the double-dose group (approximately 50%), accompanied by a modest increase in IPF (1.82%), which was also not statistically significant. Overall, although a trend toward a stronger physiological response was observed in the double-dose group, no statistically significant differences between groups were identified at the end of the observation period. Therefore, these findings should be interpreted cautiously.

This study has several limitations, including a small sample size (n = 8), which limits statistical power and increases the risk of Type II error, as well as a single-center design that may restrict generalizability. The use of convenience sampling without randomization may introduce selection bias, and differences in baseline platelet counts between groups may act as potential confounding factors. In addition, variables such as donor hydration status, nutritional condition, interval since previous donation, and apheresis machine variability were not controlled and may have influenced the results. Further studies with larger sample sizes, controlled study designs, and standardized donor conditions are needed to confirm these findings and better understand the hematological responses to different thrombapheresis doses.

References

[1] Agarwal, A., Khan, A. I., & Anwer, F. (2024). Platelet transfusion. In *StatPearls [Internet]*. StatPearls Publishing.

[2] Allard, S. (2009). Blood transfusion. *Medicine*, *37*(3), 172-176.

[3] Carson, J. L., Stanworth, S. J., Guyatt, G., Valentine, S., Dennis, J., Bakhtary, S., ... & Pagano, M. B. (2023). Red blood cell transfusion: 2023 AABB international guidelines. *Jama*, *330*(19), 1892-1902.
[4] Statistik, B. P. (2025). Statistik Indonesia 2025: Statistical yearbook of Indonesia 2025 (Vol. 53). *Badan Pusat Statistik*.
[5] Aji Muhawarman. (2025). Kasus Kanker Diprediksi Meningkat 70 Persen pada 2050, Kemenkes Perkuat Deteksi Dini
[6] Aji Muhawarman. (2025). Nyamuk lebih mematikan daripada hewan buas. Kementerian Kesehatan Republik Indonesia
[7] Lee, E. J., & Lee, A. I. (2016). Thrombocytopenia. *Primary Care: Clinics in Office Practice*, *43*(4), 543-557.
[8] Ramaiah, L., Erkens, T., Sirivelu, M., & Vitsky, A. (2025). Hematopoietic system. In *Haschek and Rousseaux's handbook of Toxicologic pathology* (pp. 337-436). Academic Press.
[9] Kaushansky, K. (2024). Thrombopoietin, the primary regulator of platelet production: from mythos to logos, a thirty-year journey. *Biomolecules*, *14*(4), 489.
[10] Gauer, R. L., & Braun, M. M. (2012). Thrombocytopenia. *American family physician*, *85*(6), 612-622.
[11] Jeon, K., Kim, M., Lee, J., Lee, J. S., Kim, H. S., Kang, H. J., & Lee, Y. K. (2020). Immature platelet fraction: a useful marker for identifying the cause of thrombocytopenia and predicting platelet recovery. *Medicine*, *99*(7), e19096.
[12] Asghar, M. B., Akhtar, F., Mahmood, A., Rafique, N., Rana, N. A., & Khalid, U. B. (2023). Diagnostic accuracy of immature platelet fraction (IPF) to differentiate between thrombocytopenia due to peripheral destruction versus bone marrow failure. *Age (years)*, *27*(19.1), 30-24.
[13] Ali, I., Graham, C., & Dempsey-Hibbert, N. C. (2019). Immature platelet fraction as a useful marker in the etiological determination of thrombocytopenia. *Experimental Hematology*, *78*, 56-61.
[14] Kaufman, R. M., Djulbegovic, B., Gernsheimer, T., Kleinman, S., Tinmouth, A. T., Capocelli, K. E., ... & Tobian, A. A. (2015). Platelet transfusion: a clinical practice guideline from the AABB. *Annals of internal medicine*, *162*(3), 205-213.
[15] Metcalf, R. A., Nahirniak, S., Guyatt, G., Bathla, A., White, S. K., Al-Riyami, A. Z., ... & Stanworth, S. J. (2025). Platelet transfusion: 2025 AABB and ICTMG international clinical practice guidelines. *JAMA*, *334*(7).
[16] Das SS, Sen S, Zaman RU, Biswas RN. (2021). Plateletpheresis in the Era of Automation: Optimizing Donor Safety and Product Quality Using Modern Apheresis Instruments. Indian Journal of Hematology and Blood Transfusion ;37:134–9. https://doi.org/10.1007/s12288-020-01337-1.
[17] Hans, R., Sharma, R. R., & Marwaha, N. (2019). Effect of plateletpheresis on postdonation serum thrombopoietin levels and its correlation with platelet counts in healthy voluntary donors. *Asian Journal of Transfusion Science*, *13*(1), 10-16.
[18] Indonesia, M. K. R. (2015). Standar Pelayanan Transfusi Darah. *Pelayanan Transfusi Darah*.
[19] Chopra, S., Kaur, P., Bedi, R. K., & Kaur, G. (2023). Effect of double dose plateletpheresis on target yield and donor platelet recovery. *Hematology, Transfusion and Cell Therapy*, *45*(1), 16-24.
[20] Makroo, R. N., Fadadu, D., Agrawal, S., & Chowdhry, M. (2018). Double dose plateletpheresis: a savior to shrinking donor pool and platelet inventory management. *Indian Journal of Hematology and Blood Transfusion*, *34*(4), 691-696.
[21] Thokala, R. P., Radhakrishnan, K., Anandan, A., & Panicker, V. K. (2016). Recovery of platelet count among apheresis platelet donors. *Journal of clinical and diagnostic research: JCDR*, *10*(12), EC01.

[22] Rajput, S., Makroo, R. N., Chowdhry, M., & Thakur, U. K. (2021). Changes in hematological parameters post plateletpheresis: Single center study from North India. *Transfusion and Apheresis Science*, *60*(6), 103283.

[23] Patel, K. B., Inaniya, K., & Padharia, D. P. (2023). Recovery period for attaining baseline haematological parameters after plateletpheresis donation-a cohort study. *J Clin Diagn Res*, *17*(4).

[24] Jiang, H., Jin, Y., Shang, Y., Yuan, G., Liu, D., Li, J., ... & Zhou, F. (2021). Therapeutic plateletpheresis in patients with thrombocytosis: gender, hemoglobin before apheresis significantly affect collection efficiency. *Frontiers in Medicine*, *8*, 762419.

[25] Elsayed, A., Elsayed, B., Elmarasi, M., Elsabagh, A. A., Elsayed, E., Elmakaty, I., & Yassin, M. (2024). Thrombopoietin receptor agonists in post-hematopoietic cell transplantation complicated by prolonged thrombocytopenia: a comprehensive review. *ImmunoTargets and Therapy*, 461-486.

[26] Hans, R., Sharma, R. R., & Marwaha, N. (2019). Effect of plateletpheresis on postdonation serum thrombopoietin levels and its correlation with platelet counts in healthy voluntary donors. *Asian Journal of Transfusion Science*, *13*(1), 10-16.

[27] Rosyidah, R. A., Anjani, N., Hartini, W. M., & Mardiyaningsih, A. (2023). Perbedaan Jumlah Trombosit Pasca Transfusi Thrombocyte Concentrate Dan Thrombocyte Apheresis Pada Pasien Trombositopenia. *Jurnal Kesehatan*, *11*(1), 169-182.

[28] Kariyawasan, C. C., Botenne, C. S., Balasuriya, B. L. T., Ruhunuhewa, U. S., Dissanayake, D. M. C., & Ranatunga, S. A. C. D. (2024). Diagnostic Utility of Immature Platelet Fraction (IPF) in Differentiating Thrombocytopenia Due to Increased Thrombopoietic Activity. *Achievements and Challenges of Medicine and Medical Science Vol. 9*, 60-73.

[29] Ding, S., Wang, M., Fang, S., Xu, H., Fan, H., Tian, Y., ... & Sun, X. (2025). Corrigendum: D-dencichine regulates thrombopoiesis by promoting megakaryocyte adhesion, migration and proplatelet formation. *Frontiers in Pharmacology*, *15*, 1504076.

[30] Bajaj, H., Rajpal, T., Sharma, M., Singh, P., Hemal, A., & Kumar, V. (2024). Role of immature platelet fraction in etiological diagnosis of thrombocytopenia. *Journal of Laboratory Physicians*, *16*(4), 496-500.

[31] Zhu, M., Sharma, P., Mete, M., Olcal, A., Ibrahim, I., Chen, W., & Bat, T. (2025). Absolute Immature Platelet Count: An Accessible Biomarker to Distinguish Aplastic Anemia and Immune Thrombocytopenia. *International Journal of Laboratory Hematology*, *47*(2), 357-361.