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Evaluation of platelet count response following single-donor thrombopheresis transfusion

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Abstract

Blood transfusion involves transferring blood or its components from a donor to a recipient, with thrombopheresis or single-donor platelet (SDP) transfusion being one of its key forms. Although generally safe, platelet transfusion carries risks, including immune and non-immune refractoriness and mild reactions. This study aimed to evaluate platelet count responses following single-dose thrombopheresis transfusion. A retrospective analysis was conducted using data from 42 patients who received 115 thrombopheresis units at Mayapada Hospital Tangerang between January 2019 and September 2020. The mean platelet increment was $16.9\times10^3/\mu L$, and the mean corrected count increment (CCI) was $10.8\times10^3/\mu L$. Based on CCI evaluation, 74% of patients showed a successful transfusion response, while 26% experienced platelet refractoriness. A 0.9% transfusion reaction rate was observed, manifesting as a single case of urticaria. Statistical analysis showed no significant differences in response among different disease groups ($p > 0.05$). These findings indicate that single-dose thrombopheresis is generally effective in improving platelet counts and is associated with a very low incidence of adverse reactions. However, the occurrence of refractoriness underscores the importance of identifying contributing factors, particularly immunologic mechanisms, to enhance transfusion outcomes and patient safety.

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INTRODUCTION

Blood is a vital tissue responsible for oxygen transport, immune defense, and coagulation [1]. It consists of plasma, which suspends cellular components including erythrocytes, leukocytes, and platelets. Platelets, or thrombocytes, are cytoplasmic fragments of megakaryocytes formed in the bone marrow through hematopoiesis, with each megakaryocyte producing approximately 2,000–4,000 platelets [2]. These anucleate, 1–4 µm fragments are essential for hemostasis through vascular repair, adhesion, and aggregation [3,4]. Low platelet counts (thrombocytopenia) can lead to bleeding, while excessive counts increase thrombotic risk [5]. Platelets typically survive for 8–12 days and are clinically indicated in thrombocytopenia, leukemia, cancer, and other malignancies [6,7].

Platelet transfusion can be performed using random donor platelets (RDP) from whole blood or single donor platelets (SDP) obtained via apheresis. RDP requires multiple donors, carries a higher risk of infection transmission, and increases the likelihood of anti-Human Leucocyte Antigen (HLA)/Human Platelet Antigen (HPA) alloimmunization, which may cause transfusion refractoriness [8]. Apheresis, first described by John J. Abel in 1914, enables the collection of high-quality platelets from a single donor with minimal risk [9,10]. Thrombopheresis can yield platelet quantities equivalent to 4–6 conventional units (~300 mL) [11,12] and has shown comparable hemostatic efficacy to pooled platelet concentrates in patients with acute myeloid leukemia (AML)/myelodysplastic syndrome (MDS), while achieving higher post-transfusion increments, particularly in adults [13-15].

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Despite its advantages, platelet transfusion carries potential risks, including immune-mediated refractoriness—defined as a post-transfusion platelet increment <7,500/μL after at least two transfusions—caused by sepsis, splenomegaly, disseminated intravascular coagulation (DIC), trauma, or alloimmunization [16-20]. Other adverse events range from mild reactions to severe anaphylaxis, and leukoreduced apheresis platelets may reduce infection risk, HLA sensitization, and improve transfusion response when post-transfusion increments exceed 30,000/μL [21-23].

Inappropriate platelet administration can lead to significant clinical and economic consequences. While thrombopheresis is increasingly utilized, data on its post-transfusion platelet increment, refractoriness rates, and transfusion reactions remain limited in certain patient populations. This study aims to evaluate platelet count increments following single-dose thrombopheresis, identify cases of post-transfusion refractoriness, and compare responses across disease groups. The findings are expected to guide safe transfusion practices and support thrombopheresis as an effective strategy to optimize therapeutic outcomes while minimizing adverse events.

Although previous studies have investigated platelet transfusion efficacy using single-donor apheresis, most have focused on hematology or oncology populations in tertiary centers outside Indonesia, with variable methodologies and post-transfusion evaluation times. For instance, Gurkan et al. [13] and Singh et al. [14] reported adequate platelet increments and hemostatic efficacy of single-donor apheresis in AML/MDS patients, while Prawita et al. [38] found a mean post-transfusion increment of 22.6×10^3/μL and a CCI of 13.6×10^3/μL in patients at Sanglah Hospital Bali. However, these studies often lacked comprehensive analysis of transfusion refractoriness and its potential clinical determinants, such as sepsis, Idiopathic trombocytopenic purpura (ITP), or other comorbid conditions frequently encountered in general hospital populations. Furthermore, limited data exist regarding the real-world outcomes of single-dose thrombopheresis transfusions in Indonesia, particularly outside major teaching hospitals. Therefore, this study aims to fill that gap by evaluating platelet count responses, corrected count increments, and refractoriness rates following single-dose thrombopheresis at Mayapada Hospital Tangerang. By comparing transfusion outcomes across disease groups and documenting the frequency of adverse reactions, this research provides updated, context-specific evidence on the effectiveness and safety of thrombopheresis in a broader clinical population, thereby complementing and extending previous findings [13,14,38,39,40].

MATERIALS & METHODS

This study employed a retrospective descriptive-analytic design using patient data obtained from Mayapada Hospital Tangerang between January 2019 and September 2020. The variables and operational definitions used in this study are summarized in Table 1, which outlines the process of patient selection, data retrieval, and variable classification for analysis. Patient screening and eligibility assessment were conducted prior to data extraction to ensure only complete and valid records were included for statistical evaluation.

Table 1. Research variables

No.	Variable	Definition of Operational Variable (DOV)	Source	Unit
1	Platelet values pre- and post-transfusion	Platelet values before and after platelet transfusion is administered.	Patient register	/μL
2	CCI	The correction value of post-transfusion platelet count minus pre-transfusion platelet count multiplied by the body surface area and divided by the yield of the plateletpheresis product.	Calculation data	/μL
3	Diagnosis	Data of accompanying diseases for the patient	Patient register	

All patients who received single-donor thrombopheresis transfusions within the study period were screened. Inclusion criteria were: (1) patients with complete medical records including

pre- and post-transfusion platelet counts within 1–24 hours, (2) documented diagnosis and demographic data, and (3) transfusion of a single unit (dose) of thrombopheresis. Exclusion criteria included: (1) incomplete or missing hematology data, (2) concurrent transfusion of other blood components (e.g., packed red cells or plasma), (3) repeated transfusions within 24 hours that could confound platelet increment measurement, and (4) documented clinical conditions that significantly alter platelet kinetics such as splenectomy, massive bleeding, or bone marrow suppression therapy within the observation window.

Patient data were retrieved from the hospital's Laboratory Information System (LIS) and medical records. Variables included gender, age, ABO blood type, diagnosis, pre- and post-transfusion platelet counts, and transfusion reactions. To minimize confounding, patients with sepsis, DIC, or medications known to affect platelet survival (e.g., heparin, aspirin, Nonsteroidal anti-inflammatory drugs (NSAIDs)) were analyzed separately in subgroup comparisons.

Data were processed using Statistical Product and Service Solution (SPSS) version 23.0. Descriptive statistics were used to summarize demographic and hematological parameters. Continuous variables (e.g., platelet counts, CCI) were presented as mean ± standard deviation. Differences between pre- and post-transfusion platelet counts were tested using the paired t-test for normally distributed data or the Wilcoxon signed-rank test for non-normal distributions. The association between transfusion response (successful vs. refractory) and categorical variables such as sex, diagnosis, and blood type was analyzed using the chi-square test or Fisher's exact test. One-way ANOVA (Analysis of Variance) was applied to compare mean CCI values across different disease groups, with a significance threshold set at $p < 0.05$.

This study follows a retrospective workflow beginning with the identification of all thrombopheresis recipients, followed by eligibility screening based on inclusion and exclusion criteria. Eligible patients' pre- and post-transfusion laboratory data were extracted and analyzed to calculate platelet increments and CCI. The results were then compared across diagnostic categories and demographic variables to evaluate transfusion effectiveness and refractoriness trends.

RESULTS AND DISCUSSION

Characteristics of Research Subjects

Between January 2019 and September 2020, a total of 42 patients underwent single-donor thrombopheresis transfusion at Mayapada Hospital Tangerang. The demographic characteristics of the research subjects are presented in Table 2, showing the distribution of patients by age and gender. The mean ± SD (Standard Deviation) age of subjects was 57.3 ± 14.9 years, ranging from 18 to 79 years. The gender distribution was nearly equal (male = 20 [47.6%]; female = 22 [52.4%]). Older patients (> 50 years) represented 71.4% of the total, reflecting the higher transfusion demand among those with hematologic or infectious diseases at advanced age. The predominance of elderly patients is consistent with previous reports showing an increased need for platelet transfusion with advancing age due to comorbidities and degenerative conditions [13, 23].

Table 2. Distribution of patients by age and gender.

Variable	Patient Distribution	Total	
		Frequency	%
Age (years)	<40	10	23.8
	41-50	2	4.8
	51-60	11	26.2
	61-70	10	23.8
	>70	9	21.4
Gender	Male	20	47.6
	Female	22	52.4

Blood Type and Clinical Diagnosis

The distribution of blood types and clinical diagnoses among the study population is summarized in Table 3. Blood group O was most common (47.6%), followed by A (26.2%), B (16.7%), and AB (9.5%). This trend aligns with the general Indonesian population distribution [24].

Table 3. Distribution of patients by ABO blood type and underlying diagnosis.

Variable	Patient Distribution	Frequency	%
Blood Type	A	11	26.2
	B	7	16.7
	O	20	47.6
	AB	4	9.5
Diagnosis	Sepsis	13	31
	Idiopathic trombocytopenic purpura	10	23.8
	Acute Lymphoid Leukemia	1	2.4
	Acute Myeloid Leukemia	6	14.3
	Myelodysplastic syndrome	4	9.5
	Disseminated Intravascular Coagulation	2	4.8
	Others	6	14.3

Regarding clinical indications, sepsis accounted for 31% of cases, followed by immune thrombocytopenic purpura (ITP) (23.8%), acute myeloid leukemia (AML) (14.3%), myelodysplastic syndrome (MDS) (9.5%), disseminated intravascular coagulation (DIC) (4.8%), and other conditions (16.6%). These data underscore that infectious and hematologic pathologies remain the leading causes of platelet transfusion requirements in this setting. The overall distribution of blood groups among recipients is also visualized in Figure 1, illustrating that type O predominates and type AB is the least frequent—consistent with the national ABO distribution profile.

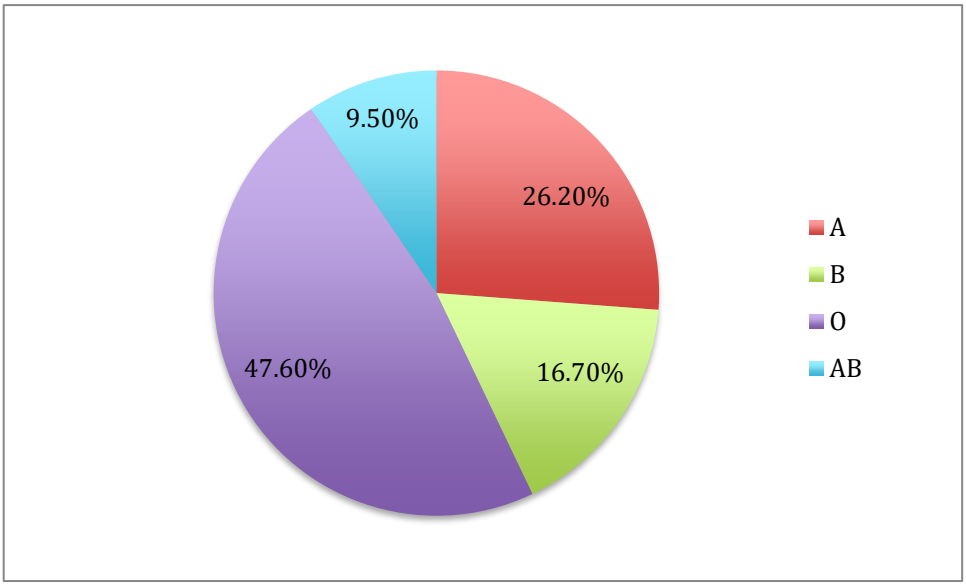

Figure 1. Distribution of research subjects based on blood type

Platelet Count Response

The change in platelet count before and after thrombopheresis transfusion is summarized in Table 4, while Figure 2 provides a graphical representation of mean pre- and post-transfusion platelet values.

Table 4. Pre- and post-transfusion platelet counts and corrected count increment (CCI).

Parameter	Minimal value (/µL)	Maximal value (/µL)	Rate value (/µL)
Pretransfusion Platelet Count	1,000	121,000	35,643 ± 28,410
Posttransfusion Platelet Count	1,000	176,000	52,348 ± 35,217
Increased Platelet Count	-40,000	126,000	16,904 ± 22,563
CCI	-30,170	71,927	10,890 ± 14,204

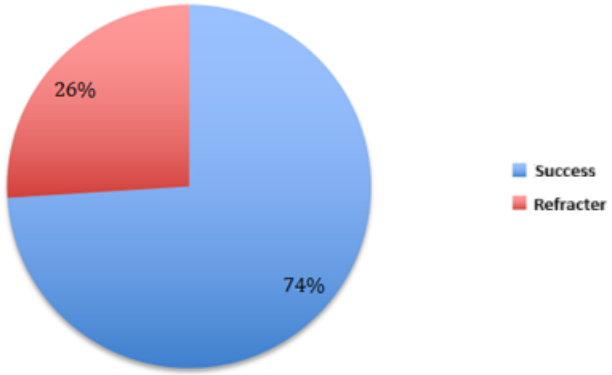

Figure 2. The increase in platelet count 24 hours after thrombopheresis administration.

The mean platelet count increased significantly after thrombopheresis ($p < 0.001$, paired t-test), confirming the clinical effectiveness of single-donor platelet transfusion. Based on CCI results, 31 patients (74%) demonstrated a successful transfusion response, while 11 patients (26%) were classified as refractory. While CCI is commonly used to evaluate platelet transfusion efficacy, its calculation in equation (1) may be limited in settings where smaller platelet doses are administered.

$$\text{CCI} = \frac{(\text{posttransfusion} - \text{pretransfusion platelet count})(10^9/\text{L}) \times \text{body surface area}(\text{m}^2)}{\text{number of platelets transfused}(10^{11})} \quad (1)$$

Analysis of Refractory Cases

The comparison of transfusion success between male and female patients is shown in Table 5. Although a slightly higher success rate was observed among females (52%) than males (48%), this difference was not statistically significant ($p > 0.05$).

Table 5. Transfusion success and refractoriness by gender.

Variable	Patient Distribution	Quantity			
		Success	%	Refractory	%
Gender	Male	15	48	5	45
	Female	16	52	6	54
Total		31	100	11	100

Refractoriness occurred predominantly in sepsis (n = 6) and ITP (n = 3) cases. The mean CCI of refractory patients was $3.4 \pm 2.1 \times 10^3/\mu L$, significantly lower than in responders ($14.8 \pm 9.2 \times 10^3/\mu L$, $p < 0.001$). The causes of refractoriness can be divided into immunologic and non-immunologic factors. Immunologic causes involve alloimmunization against HLA or HPA antigens [20, 43, 49], while non-immunologic factors include sepsis, DIC, fever, splenomegaly, and drug-related platelet destruction [16, 17, 44]. In this study, non-immunologic causes—particularly infection—were the major contributors. Compared with international standards, the AABB recommends that ≥ 75% of thrombopheresis transfusions yield a $\geq 7.5 \times 10^3/\mu L$ increment at 1 hour [6], and European guidelines set a $\geq 20 \times 10^3/\mu L$ threshold. The observed 74% success rate in this study approaches AABB (American Associated of Blood Banking) expectations and aligns with

previous reports by Jaime-Pérez et al. (67%) [39] and Slichter et al. (72%) [40], demonstrating comparable efficacy.

Transfusion Reactions

Adverse reactions following thrombopheresis are summarized in Table 6. Among 115 transfused units, only one case (0.9%) of mild urticaria occurred, with no reports of transfusion-related acute lung injury (TRALI), transfusion-associated circulatory overload (TACO), or anaphylaxis.

Table 6. Observed transfusion reactions following single-donor thrombopheresis.

Transfusion Reaction	*n*	(%)
No transfusion reaction	114	99.1
Urticaria	1	0.9

The 0.9% reaction rate is markedly lower than the 4–5% frequency reported in prior studies [46], confirming that single-donor apheresis is generally safe. Urticaria was managed effectively with antihistamines, and no further complications occurred.

Interpretation and Clinical Implications

Overall, thrombopheresis transfusion produced a mean platelet increment of 16.9 ± 22.6 × 10^3/µL and mean CCI of 10.8 ± 14.2 × 10^3/µL, with significant pre- to post-transfusion improvement ($p < 0.001$). The refractoriness rate of 26% underscores the influence of both immunologic and clinical factors—especially infection—on transfusion response. These results confirm that single-donor thrombopheresis is effective and safe, comparable with international data [13, 14, 38–40]. Implementing post-transfusion platelet monitoring (within 1 hour) and routine CCI calculation can serve as key quality indicators for transfusion efficacy. Furthermore, incorporating HLA/HPA screening in patients with repeated transfusions could reduce refractoriness and improve outcomes in Indonesian clinical settings.

CONCLUSION

This study demonstrated that a single dose of thrombopheresis effectively increased platelet counts, with a transfusion success rate of 74% and a mean platelet increment of 16.9×10^3/µL. Platelet refractoriness occurred in 26% of cases, while transfusion reactions were rare (0.9%, limited to mild urticaria). No statistically significant differences in transfusion outcomes were observed among various disease groups, suggesting that thrombopheresis provides consistent efficacy across clinical indications. Routine post-transfusion complete blood counts and CCI evaluation within one hour are recommended to ensure quality control and monitor transfusion efficacy in clinical practice.

For future research, larger multicenter prospective studies are needed to explore the immunologic mechanisms underlying platelet refractoriness, particularly involving HLA and HPA antibody screening. Investigating genetic compatibility factors, donor–recipient antigen matching, and cytokine response profiles could provide deeper insights into transfusion success predictors. Additionally, integrating machine learning or predictive models to identify patients at high risk of refractoriness may help optimize transfusion strategies and support evidence-based implementation of personalized thrombopheresis protocols in Indonesian clinical settings.

AUTHOR CONTRIBUTIONS

Fajar Nugroho contributed to the conception and design of the study, data collection, statistical analysis, and drafting of the manuscript. Atmadi contributed to data interpretation, critical revision of the manuscript for important intellectual content, and overall supervision of the research

process. Cornelia Victoria Anghel contributed to the study design, data validation, statistical analysis, and critical revision of the manuscript for intellectual content. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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ETHICAL APPROVAL

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Ethical approval for the use of patient data was obtained from the Ethics Committee of Mayapada Hospital Tangerang (Ethical Approval No.: 002/PKS/PT-SRAJ/IX/2020). As this research involved a retrospective review of anonymized medical records, the requirement for informed consent was waived by the committee. All patient data were handled confidentially, and no identifiable personal information was disclosed or published.

REFERENCES

[1] Ritchie NK. Hematologi Dasar Tentang Transfusi Darah. Jakarta (ID): Paramedis Teknologi Transfusi Darah; 2011.

[2] Sundaryono A, Asli A. Penggunaan Batang Tanaman Betadin (Jatropha multifida Linn) untuk Meningkatkan Jumlah Trombosit pada Mus musculus. Media Medika Indonesiana. 2011.

[3] Grondahl G. An Introduction in Veterinary Hematology. 4th ed. Stockholm (SE): Boule Medical AB; 2005. p. 26–7.

[4] Hoffrand A, Pettit J. Kapita Selekta Hematologi. 4th ed. Jakarta (ID): EGC; 2005.

[5] Hulin L. Pathophysiology of Blood and Hematologic System. 1995. p. 69–70.

[6] American Association of Blood Banking (AABB). Blood Component Preparation and Processing. In: AABB Technical Manual. 17th ed. New York (US): AABB Publishing; 2011. p. 198.

[7] Dewi S. Peningkatan jumlah trombosit setelah pemberian transfusi trombosit apheresis pada anak dengan penyakit keganasan disertai trombositopenia refrakter. Simdos Unud. 2016 [cited 2020 Nov 20]. Available from: https://simdos.unud.ac.id/uploads/file_penelitian_1_dir/f7d49d142e7994f4c3eb089d62093989.pdf

[8] Cable R, Carlson B, Kolins J, et al. Practice Guidelines for Blood Transfusion. 2nd ed. 2007.

[9] Banta H, Behney CJ, Langenbrunner JC, et al. Health Technology Case Study 23: The Safety, Efficacy, and Cost Effectiveness of Therapeutic Apheresis. Washington (US): U.S. Government Printing Office; 1983.

[10] New Zealand Blood Service (NZBS). Did you know? 10 historical facts about blood transfusion. NZ Blood Service [Internet]. 2015 [cited 2019 Sep 30]. Available from: https://www.nzblood.co.nz/news/2015/did-you-know-10-historical-facts-about-blood-transfusion/

[11] Kementerian Kesehatan Republik Indonesia. Peraturan Menteri Kesehatan Republik Indonesia Nomor 91 Tahun 2015 tentang Standar Pelayanan Darah. Jakarta (ID): Kementerian Kesehatan RI; 2015.

[12] Barbosa M, Silva K, Coelho D. Risk factors associated with the occurrence of adverse events in plateletpheresis donation. Rev Bras Hematol Hemoter. 2014;36(3):191–5.

[13] Gurkan E, Patah PA, Saliba RM, et al. Efficacy of prophylactic transfusions using single donor apheresis platelets versus pooled platelet concentrates in AML/MDS patients receiving allogeneic transplants. Bone Marrow Transplant. 2007;39(10):601–5.
[14] Singh RP, Marwaha N, Malhotra P, et al. Therapeutic efficacy of different types of platelet concentrates in thrombocytopenic patients. Indian J Hematol Blood Transfus. 2008;24(1):16–22.
[15] Miller Y, Bachowski G, Benjamin R. Practice Guidelines for Blood Transfusion: A Compilation from Recent Peer-Reviewed Literature. Washington (US): American Red Cross; 2007.
[16] Liumbruno G, Bennardello F, Lattanzio A, et al. Recommendations for the transfusion of plasma and platelets. Blood Transfus. 2009;7(2):132–50.
[17] Purba J, Mulatsih S, Nurani N, et al. Faktor risiko refrakter trombosit pada anak. Sari Pediatri. 2013;15:190–4.
[18] Slichter SJ. Evidence-based platelet transfusion guidelines. Hematology Am Soc Hematol Educ Program. 2007;1:172–8.
[19] Nydam T, Kashuk JL. Refractory postinjury thrombocytopenia is associated with multiple organ failure and adverse outcomes. J Trauma Acute Care Surg. 2011;70:401–7.
[20] Kurz M, Greinix H, Hocker P, et al. Specificities of anti-platelet antibodies in multitransfused patients with haematologic disorders. Br J Haematol. 1996;95(3):564–9.
[21] Chaffin DJ. Transfusion reactions: scope of the problem. BBGuy.org [Internet]. 2012 [cited 2020 Nov 20]. Available from: https://www.bbguy.org
[22] Kiswari R. Hematologi dan Transfusi. Semarang (ID): Erlangga; 2014.
[23] Puspita P. Platelets count increment after single donor apheresis in Hasan Sadikin Hospital. Vox Sang [Internet]. 2011 [cited 2020 Nov 20]. Available from: https://scholar.google.co.id/scholar?hl=id&as_sdt=0%2C5&q=platelet+count+increment+puspita
[24] Kementerian Kesehatan Republik Indonesia. Data dan Informasi: Profil Kesehatan Indonesia 2017. Jakarta (ID): Kemenkes RI; 2017.
[25] Klug W, Cummings M. Concepts of Genetics. 4th ed. New York (US): Macmillan; 1994.
[26] Chester M, Olsson M. The ABO blood group gene: a locus of considerable genetic diversity. Transfus Med Rev. 2001;15(3):177–200.
[27] Nester T, Aubuchon J. Hemotherapy decisions and their outcomes. In: AABB Technical Manual. 17th ed. Bethesda (US): AABB; 2011.
[28] Suparto S, Febyan F. Sepsis dan tata laksana berdasar guideline terbaru. Repository Ukrida [Internet]. 2018 [cited 2020 Dec 2]. Available from: http://repository.ukrida.ac.id/bitstream/123456789/403/1/peer%20sepsis.pdf
[29] Bakta M, Suastika K. Gawat darurat di bidang penyakit dalam. Repo Stikes Perintis [Internet]. 2020 [cited 2020 Dec 2]. Available from: http://repo.stikesperintis.ac.id/id/eprint/1079
[30] Cines DB, Bussel J, Liebman H, et al. Guidelines for the investigation and management of idiopathic thrombocytopenic purpura in adults, children, and in pregnancy. Blood. 2003;113(26):531–42.
[31] Shim YJ, Kim UH, Suh JK, et al. Natural course of childhood chronic immune thrombocytopenia using the revised terminology and definitions of the international working group. Blood Res. 2014;49:187–91.
[32] Dickinson H. The causes of childhood leukaemia. BMJ. 2005;330:1279–80.
[33] Grigoropoulos NF, Petter R, Scott M, et al. Leukaemia update. Part 1: diagnosis and management. BMJ. 2013;346:f1660.
[34] Terwilliger T, Hay MA. Acute lymphoblastic leukemia: a comprehensive review and 2017 update. Blood Cancer J. 2017;7:e577.

[35] Liebman H. Thrombocytopenia in cancer patients. Thromb Res. 2014;133:S63–9.
[36] Novotny VMJ. Prevention and management of platelet transfusion refractoriness. Vox Sang. 1999;76(1):1–13.
[37] Shastry S, Chaudhary R. Clinical factors influencing corrected count increment. Transfus Apher Sci. 2012;47(3):327–30.
[38] Prawita A, Mulyantari N, Herawati S. The description of corrected count increment on one hour and 24 hours after platelet apheresis transfusion in Sanglah General Hospital Denpasar. Bali Med J. 2019;8:1391–8.
[39] Jaime-Pérez JC, Fernández LT, Cantú-Rodríguez OG, et al. Platelet survival in hematology patients assessed by the corrected count increment and other formulas. Am J Clin Pathol. 2018;150(3):267–72.
[40] Slichter SJ, Davis K, Enright H, et al. Factors affecting posttransfusion platelet increments, platelet refractoriness, and platelet transfusion intervals in thrombocytopenic patients. Blood. 2005;105(10):4106–14.
[41] Holbro A, Infanti L, Sigle J, et al. Platelet transfusion: basic aspects. Swiss Med Wkly. 2013;143:w13885.
[42] Neunert C, Lim W, Crowther M, et al. Evidence-based practice guideline for immune thrombocytopenia. Blood. 2011;117(16):4190–207.
[43] Petz LD, Garratty G, Calhoun L, et al. Selecting donors of platelets for refractory patients on the basis of HLA antibody specificity. Transfusion. 2000;40(12):1446–56.
[44] Alcorta I, Pereira A, Ordinas A. Clinical and laboratory factors associated with platelet transfusion refractoriness: a case-control study. Br J Haematol. 1996;93(1):220–4.
[45] World Health Organization (WHO). Platelet Transfusion in Clinical Practice: Professional Guidance Document. Geneva (CH): WHO; 2012.
[46] Savage WJ, Savage JH, Tobian AA, et al. Allergic agonists in apheresis platelet products are associated with allergic transfusion reactions. Transfusion. 2012;52(3):575–81.
[47] Chandrashekar L, Rajappa M, Sundar I, et al. Platelet activation in chronic urticaria and its correlation with disease severity. Platelets. 2014;25(3):162–5.