

Platelet Analysis in Pediatric Patients with Acute Gastroenteritis at RSIA Sitti Khadijah 1 Makassar

Siti Muthiah Annisa Idris¹, Irna Diyana Kartika Kamaluddin², Febie Irsandy Syahrudin³, Irmayanti Haidir Bima², Andi Alamanda Irwan⁴

¹Medical Education Study Program, Faculty of Medicine, Universitas Muslim Indonesia

²Department of Clinical Pathology, Faculty of Medicine, Universitas Muslim Indonesia

³Radiology Section of Faculty of Medicine, Universitas Muslim Indonesia

⁴Department of Pharmacology Faculty of Medicine, Universitas Muslim Indonesia

Email: muthiaa21@gmail.com, irnadiyanakartika.kamaluddin@umi.ac.id, febie.irsandysy@umi.ac.id, irmayanti.irmayanti@umi.ac.id, andialamanda.irwan@umi.ac.id

Abstract. *Acute gastroenteritis (AGE) is an inflammation of the stomach and intestines with symptoms of diarrhea, nausea and vomiting, experienced for less than 14 days, with a fairly high prevalence worldwide. One of the laboratory tests carried out is routine blood, including platelet count. When inflammation occurs, platelet production will increase in response from the immune system. To analyze platelets in acute gastroenteritis (AGE) sufferers. The methods of the research is descriptive retrospective using medical record data of acute gastroenteritis (AGE) sufferers. Results showed that the age distribution of samples was grouped into three groups, namely ≤ 2 years, 3-5 years, and 6-10 years with the main complaints of diarrhea, vomiting, and diarrhea accompanied by vomiting. The platelet count of acute gastroenteritis (AGE) sufferers is more normal, but there is an increase in platelet count in the ≤ 2 years age group with complaints of diarrhea accompanied by vomiting. There is no relationship between platelet count and complaints of diarrhea, vomiting, and diarrhea accompanied by vomiting in acute gastroenteritis (AGE) sufferers.*

Keywords: Platelets, Children, AGE, Diarrhea, Vomiting

Received: April 9, 2026

Received in Revised: June 24,
2026

Accepted: August 25, 2026

INTRODUCTION

Gastroenteritis or acute diarrhea is diarrhea that occurs suddenly in babies and children who were previously healthy. Gastroenteritis is defined as inflammation of the mucous membrane of the digestive tract characterized by diarrhea or vomiting. Acute gastroenteritis (GEA) is inflammation of the stomach and intestines with symptoms of diarrhea, nausea and vomiting for less than 14 days (Bolon, 2019; Saputra et al., 2021; Rista & Jepisah, 2021). Gastroenteritis is a disease that has a fairly high prevalence throughout the world. This is proven by the epidemiology of gastroenteritis symptoms, namely diarrhea, which globally is recorded at around 1.7 billion cases every year. There were 525,000 cases of death due to diarrhea in children less than five years old during this incident. Diarrhea is the main cause of morbidity and mortality in children less than 5 years old (Waroka et al., 2022; Nardin, 2019).

The age group with the highest prevalence of diarrhea (based on diagnosis from health workers) is in the 1-4 year age group, namely 11.5% and in babies, namely 9%. The prevalence of diarrhea in women is 7.1% higher than in men, 6.5%. Viruses are the most common agents of GEA, accounting for >60% of pediatric cases, followed by bacteria and parasites (Waroka et al.,

2022; Nardin, 2019; Guarino et al., 2020). Platelets or platelets are fragments or small pieces of megakaryocyte cytoplasm. Platelets are the result of fragmentation of the cytoplasm of megakaryocytes formed in the bone marrow. The main regulator of platelet production is the hormone thrombopoietin (TPO), namely the growth factor hormone, which is the main regulator that regulates the number of platelets in circulation (Sherwood, 2020; Huether & McCance, 2019; Alivameita, 2019).

Platelets play an important role in the hemostasis system to stop bleeding from injured blood vessels. In addition, platelets have gained recognition for their inflammatory functions that modulate immune responses during infectious diseases. Platelets contain a variety of immunoreceptors that enable them to act as sentinels to recognize intravascular pathogens. Upon activation, platelets directly limit pathogen growth through immune cell activation (Alivameita, 2019; Portier & Campbell, 2021).

One of the laboratory tests carried out for acute diarrhea is a blood test. Counting platelets is one of the examinations needed to confirm the diagnosis, monitor the results of therapy, determine the prognosis and estimate the severity or absence of a disease. Platelets have gained recognition for their inflammatory functions that modulate immune responses during infectious diseases. When inflammation occurs, IL-6 will induce an increase in TPO production, so that the production of new platelets will also be increased. Pro-inflammatory cytokines such as IL-6 in the defense mechanism in gastroenteritis play a role in stimulating macrophages and neutrophils to produce reactive oxygen species to fight infection and also to produce inflammatory responses locally and systemically (Huether & McCance, 2019; Portier & Campbell, 2021; Syuhada et al., 2021; Zaki et al., 2020; Diah et al., 2018; Sugimoto & Eto, 2017).

METHODS

This study used a retrospective descriptive design by utilizing secondary data in the form of medical records of acute gastroenteritis (GEA) patients at RSIA Sitti Khadijah 1 Makassar in the period July 2022–October 2023. The research was carried out at RSIA Sitti Khadijah 1 Makassar, with implementation time planned for July 2023. The study population included all GEA patients at the hospital during the period July 2022–October 2023. The sample is part of population that meets the inclusion criteria and does not meet the exclusion criteria. Sampling used a purposive sampling technique, namely selecting samples based on characteristics determined in accordance with the research objectives. Inclusion criteria include medical records of pediatric patients with GEA who were hospitalized during the period July 2022–October 2023, aged ≤ 10 years, had data on main complaints in the form of diarrhea, nausea, or vomiting as well as laboratory test results, and had routine blood test results on the first or second day of treatment. The exclusion criteria were medical records of GEA patients accompanied by secondary infections. The research focuses on describing the platelet count of pediatric patients with GEA. Because it uses a descriptive design, GEA is the condition that is the basis for subject selection, while the platelet count is the variable that is described, so there is no need to divide the independent and dependent variables. Research data was obtained through reviewing patient medical records as a secondary data source, especially information on diagnosis, age, main complaint, presence of secondary infections, and results of platelet count examination.

Data Analysis

The data that has been collected from medical records will be subjected to bivariate analysis. The data will be processed using the chi square method to see whether or not there is a relationship between acute gastroenteritis (GEA) and the number of platelets in GEA sufferers.

Table 1. Operational Definitions and Measurement of Study Variables

No.	Variable	Operational Definition	Method of Measurement	Measuring Instrument	Measurement Results	Measurement Scale
1	Platelet Count	The number of platelets recorded in the routine blood test results of patients diagnosed with GEA at RSIA Sitti Khadijah 1 Makassar.	Observation	Medical records	Thrombocytosis: >450,000/ μ L Normal: 150,000–450,000/ μ L Thrombocytopenia: <150,000/ μ L	Ratio
2	Main Complaint	The primary complaint reported by patients diagnosed with GEA upon admission to RSIA Sitti Khadijah 1 Makassar.	Observation	Medical records	Diarrhea and vomiting Diarrhea Vomiting	Nominal

Research Flow

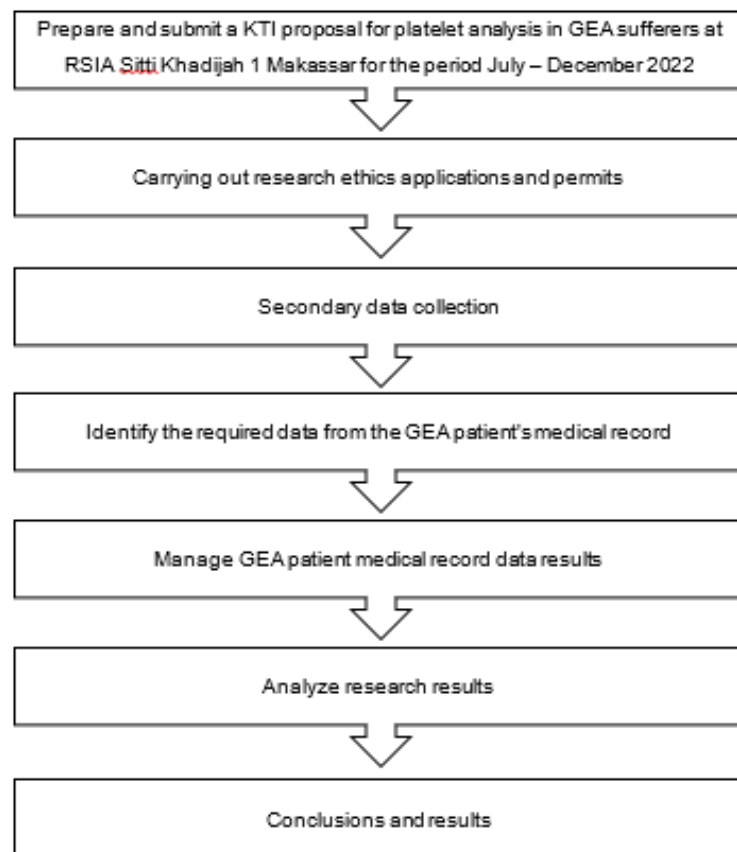


Figure 1. Research Procedure for Platelet Analysis in Patients with Acute Gastroenteritis (GEA)

RESULT AND DISCUSSION

The research analyzed platelet counts in patients with acute gastroenteritis (GEA) at RSIA Sitti Khadijah 1 Makassar. The independent variable examined in this study was acute gastroenteritis (GEA). The data were obtained from the medical records of patients who visited the hospital from July 2022 to October 2023. Based on the secondary data obtained, a total of 220 medical records were included in the study. The research results are presented in the following tables, accompanied by their respective explanations.

Age

Table 2. Age Distribution of Patients with Acute Gastroenteritis (GEA)

Age	Frequency (n)	Percentage (%)
< 2 years	175	79.5
3–5 years	32	14.5
6–10 years	13	5.9
Total	220	100.0

Based on Table 2, the highest proportion of patients with acute gastroenteritis (GEA) was in the age group of < 2 years, accounting for 79.5%. This was followed by the 3–5 years age group at 14.5% and the 6–10 years age group at 5.9%.

Platelet Count

Table 3. Distribution of Platelet Counts among Patients with Acute Gastroenteritis (GEA)

Platelet Count	Frequency (n)	Percentage (%)
Thrombocytosis	72	32.7
Normal	147	66.8
Thrombocytopenia	1	0.5
Total	220	100.0

Based on Table 3, 32.7% of patients with acute gastroenteritis (GEA) experienced thrombocytosis, 66.8% had normal platelet counts, and 0.5% experienced thrombocytopenia.

Table 4. Mean Platelet Count among Patients with Acute Gastroenteritis (GEA)

Platelet Count	Frequency	Minimum ($10^3/\mu\text{L}$)	Maximum ($10^3/\mu\text{L}$)	Mean ($10^3/\mu\text{L}$)
Thrombocytosis	72	456	828	572.67
Normal	147	184	449	344.78
Thrombocytopenia	1	119	119	119

Based on Table 4, the platelet count among patients with acute gastroenteritis (GEA) with thrombocytosis had a minimum value of $456 \times 10^3/\mu\text{L}$, a maximum value of $828 \times 10^3/\mu\text{L}$, and a mean of $572.67 \times 10^3/\mu\text{L}$. Patients with normal platelet counts had a minimum value of $184 \times 10^3/\mu\text{L}$, a maximum value of $449 \times 10^3/\mu\text{L}$, and a mean of $344.78 \times 10^3/\mu\text{L}$. Patients with thrombocytopenia had a minimum value of $119 \times 10^3/\mu\text{L}$, a maximum value of $119 \times 10^3/\mu\text{L}$, and a mean of $119 \times 10^3/\mu\text{L}$.

Main Complaint

Table 5. Distribution of Main Complaints among Patients with Acute Gastroenteritis (GEA)

Main Complaint	Frequency (n)	Percentage (%)
Diarrhea and vomiting	163	74.1
Diarrhea	38	17.3
Vomiting	19	8.6
Total	220	100.0

Based on Table 5, the most common complaint among patients with acute gastroenteritis (GEA) was diarrhea and vomiting, accounting for 74.1% of patients. Patients with GEA who experienced diarrhea only accounted for 17.3%, while those who experienced vomiting only accounted for 8.6%.

Analysis of Platelet Counts among Patients with Acute Gastroenteritis (GEA) Based on Main Complaints

Table 6. Analysis of Platelet Counts among Patients with Acute Gastroenteritis (GEA) Based on Main Complaints

Platelet Count	Diarrhea & Vomiting	Diarrhea	Vomiting	Total
Thrombocytosis	56	13	3	72
Normal	106	25	16	147
Thrombocytopenia	1	0	0	1
Total	163	38	19	220

p-value = 0.538

Based on Table 6, among patients with GEA who presented with diarrhea and vomiting, 56 patients experienced increased platelet counts (thrombocytosis), 106 patients had normal platelet counts, and 1 patient experienced decreased platelet counts (thrombocytopenia). Among patients with GEA who presented with diarrhea only, 13 patients experienced increased platelet counts (thrombocytosis), while 25 patients had normal platelet counts. Among patients with GEA who presented with vomiting only, 3 patients experienced increased platelet counts (thrombocytosis), while 16 patients had normal platelet counts.

Based on the results we found, there was no association between increased platelets and sufferers of acute gastroenteritis (GEA), and our research is not in line with research conducted by Maralihalli et al., which compared platelet counts with MPV and involved 30 sufferers of acute gastroenteritis in the age group <5 years who experienced a statistical increase in the number of platelets (thrombocytosis) compared to controls, namely children who did not suffer from acute gastroenteritis (GEA). In the distribution of platelet results data, we found patients with acute gastroenteritis (GEA) whose platelet count was normal at 66.8% and thrombocytopenia at 0.5%. This is because our sample is not homogeneous like the sample of Maralihalli et al., so the results found are not statistically significant (Machiwal et al., 2021). Reactive thrombocytosis is recognized as a frequent secondary finding in childhood infection and inflammation, although its occurrence and magnitude may vary according to the underlying disease, age, and clinical characteristics of the population studied (Schwarzenberg et al., 2021; Elber-Dorozko et al., 2023). Platelet responses during infection are also influenced by interactions between platelets, inflammatory mediators, endothelial cells, and immune cells, indicating that platelet count changes may not occur uniformly across different infectious conditions (Tokarz-Deptuła et al., 2021; Allaoui et al., 2021; O'Reilly et al., 2022). Consequently, differences in sample characteristics and disease conditions may contribute to variations in platelet findings between studies.

From the distribution of data on platelet results for patients with acute gastroenteritis (GEA) with complaints of diarrhea accompanied by vomiting, 56 children with complaints of diarrhea and vomiting showed increased platelet values (thrombocytosis). This is in accordance with the theory that when an inflammatory response occurs, a vascular response will occur, resulting in vasodilation and increased blood vessel permeability. Vasodilation increases blood supply, and increasing capillary permeability will make the endothelium facilitate the passage of many neutrophils and monocytes into the tissue as part of the inflammatory response and make plasma proteins exit into the tissue, so that many proteins and fluids leave the capillaries, which will result in an increase in the concentration of cells in the blood, one of which is platelets (Wahid & UA, 2016; Baratawidjaja & Rengganis, 2022). Platelets participate in inflammatory responses through interactions with damaged or activated endothelial cells and immune cells, while also

releasing biologically active substances that influence leukocyte recruitment and inflammatory signaling (Tokarz-Deptuła et al., 2021; Zaid & Merhi, 2022). Platelet activation may therefore represent an important component of the interaction between vascular responses and innate immunity during infectious conditions (Carestia et al., 2023; Trivigno et al., 2023; Yan et al., 2023). The interaction between platelets and inflammatory cells can further contribute to the regulation of vascular integrity and immune responses during infection (Scherlinger et al., 2023; Nicolai et al., 2024). These mechanisms support the role of platelets as active participants in inflammatory processes rather than functioning exclusively in hemostasis (Rayes & Jenne, 2021; Hu et al., 2025).

Based on research conducted by Kucuk et al. regarding changes in MPV based on etiology and vesicular score in patients with acute gastroenteritis, involving 174 samples with an age range of 0–16 years, there was an increase in the number of platelets in GEA sufferers who experienced moderate and severe dehydration. As blood concentration increases, clotting factors will occur, where platelet aggregation will form to repair damaged blood vessels. Diarrhea and vomiting can cause dehydration due to loss of fluids and electrolytes in the body. Decreased fluid in the body causes a decrease in plasma volume and an increase in blood concentration (Sherwood, 2020; Küçük et al., 2015; Sugimoto & Eto, 2017). Changes in platelet responses during infectious conditions may also be related to the interaction between coagulation and inflammation, particularly when vascular integrity is disrupted (Mack et al., 2024; Koenen, 2023). Platelets can participate in immunothrombosis, in which coagulation and innate immune responses cooperate to limit pathogen dissemination and support vascular defense (Carestia et al., 2023; Nicolai et al., 2024). In pediatric infectious conditions, platelet responses may also reflect the inflammatory cascade and changes in the vascular environment (O'Reilly et al., 2022; Wang et al., 2023). These processes indicate that alterations in platelet counts may be influenced not only by the inflammatory response but also by changes in vascular and hemostatic conditions associated with fluid loss and infection.

Many platelet responses to pathogen invasion are hemostatic and represent an immune continuum, such as the expression of activated integrins $\alpha\text{IIb}\beta 3$ and P-selectin on the plasma membrane, formation of platelet aggregates and thrombosis, attachment to and damage of the endothelium, increased interaction with macrophages and neutrophils, promotion of NET formation, and release of cytokines. They interact with most cells of the (innate) immune system and release large amounts of cytokines to stimulate an immune response (Martinod & Deppermann, 2021; Worung et al., 2020). Platelets are increasingly recognized as important components of innate immune defense because they can recognize pathogens, interact with immune cells, and participate in pathogen containment through immunothrombosis (Antoniak & Mackman, 2021; Carestia et al., 2023). Their interaction with neutrophils is particularly important because platelet activation can promote NET formation, while NETs can subsequently provide a scaffold for platelet adhesion and activation (Colicchia et al., 2023; Chen & Boskovic, 2024). Platelet–neutrophil interactions therefore represent an important mechanism connecting inflammation, coagulation, and immune defense during infection (Maugeri & Manfredi, 2025). In addition, platelet activation can contribute to inflammatory and thrombotic responses when these mechanisms become excessive or dysregulated (Mack et al., 2024; Bo & Zhao, 2025). Thus, the immune and hemostatic functions of platelets form an interconnected continuum in which platelet activation can contribute to pathogen containment, immune-cell recruitment, and inflammatory regulation (Hu et al., 2025).

Research Limitations

In the sample in our study, the age distribution of the sample was not homogeneous, causing the results found to be statistically insignificant. In our study we used secondary data in the form of medical records and only looked at the history of complaints from patients with acute gastroenteritis (GEA) and the results of laboratory examinations so we did not know the severity of acute gastroenteritis (GEA). In our study we did not examine the history of immunization and breast milk consumption which could affect the immune system of acute gastroenteritis (GEA)

sufferers. In our study, the history of treatment that had been given before admission to the hospital was unknown

CONCLUSION

The highest number of platelets in patients with acute gastroenteritis (GEA) is a normal number. The main complaints most often experienced by sufferers of acute gastroenteritis (GEA) are diarrhea and vomiting. There is no relationship between platelet count and the main complaint of acute gastroenteritis (GEA) sufferers.

REFERENCES

- Aliviameita AP. (2019). *Buku Ajar Mata Kuliah Hematologi*. Sidoarjo: UMSIDA Press
- Allaoui, A., Khawaja, A. A., Badad, O., Naciri, M., Lordkipanidzé, M., Guessous, F., & Zaid, Y. (2021). Platelet function in viral immunity and SARS-CoV-2 infection. *Seminars in Thrombosis and Hemostasis*, 47(4), 419–426. <https://doi.org/10.1055/s-0041-1726033>
- Antoniak, S., & Mackman, N. (2021). Platelets and viruses. *Blood*, 137(20), 2859–2866. <https://doi.org/10.1182/blood.2020005955>
- Baratawidjaja, K. G., & Rengganis, I. (2022). *Imunologi dasar* (12th ed.). Universitas Indonesia Publishing.
- Bo, Y., & Zhao, F. (2025). Platelets as central hubs of inflammation. *Frontiers in Immunology*, 16, 1683553. <https://doi.org/10.3389/fimmu.2025.1683553>
- Bolon, C. M. T. (2019, December). Hubungan Pola Asuh Ibu Dengan Status Gastroenteritis Pada Balita di Rumah Sakit Umum Imelda Pekerja Indonesia Medan Tahun 2015. In *SINTAKS (Seminar Nasional Teknologi Informasi Komputer dan Sains 2019)* (Vol. 1, No. 1, pp. 494-502).
- Carestia, A., Godin, L. C., & Jenne, C. N. (2023). Step up to the platelet: Role of platelets in inflammation and infection. *Thrombosis Research*, 231, 182–194. <https://doi.org/10.1016/j.thromres.2022.10.001>
- Chen, W. A., & Boskovic, D. S. (2024). Neutrophil extracellular DNA traps in response to infection or inflammation, and the roles of platelet interactions. *International Journal of Molecular Sciences*, 25(5), 3025. <https://doi.org/10.3390/ijms25053025>
- Colicchia, M., Perrella, G., Gant, P., & Rayes, J. (2023). Novel mechanisms of thrombo-inflammation during infection: Spotlight on neutrophil extracellular trap-mediated platelet activation. *Research and Practice in Thrombosis and Haemostasis*, 7(2), 100116. <https://doi.org/10.1016/j.rpth.2023.100116>
- Diah, H., Zahra, & Athena, A. (2018). Kejadian gastroenteritis dan faktor penyebabnya pada siswa SD di Kelurahan Beji Timur, Kota Depok. *Jurnal Ekologi Kesehatan*, 17(2), 96–104. <https://doi.org/10.22435/jek.17.2.377.96-104>
- Elber-Dorozko, S., Kerem, L., Wolf, D., Brodie, S., Berkun, Y., Brooks, R., & Breuer, O. (2023). Platelet count and risk of severe illness in hospitalised children with influenza-like illness. *Acta Paediatrica*, 112(10), 2191–2198. <https://doi.org/10.1111/apa.16875>
- Guarino, A., Aguilar, J., Berkley, J., Broekaert, I., Vazquez-Frias, R., Holtz, L., ... & Treepongkaruna, S. (2020). Acute gastroenteritis in children of the world: what needs to be done?. *Journal of pediatric gastroenterology and nutrition*, 70(5), 694–701. <https://doi.org/10.1097/MPG.0000000000002669>
- Hu, Y., Dai, S., Qiao, C., Ye, Y., Ren, J., Wang, K., Li, L., & Liu, Z. (2025). Platelets in infection: Intrinsic roles and functional outcomes. *Frontiers in Immunology*, 16, 1616783. <https://doi.org/10.3389/fimmu.2025.1616783>
- Huether SE, McCance KL. 2019. *Buku Ajar Patofisiologi*. Vol 1. 6th ed. Singapore: Elsevier Pte Ltd

- Koenen, R. R. (2023). Platelets: From simple fragments to inflammation regulators. *European Heart Journal*, 44(8), 633–635. <https://doi.org/10.1093/eurheartj/ehac705>
- Küçük, Ö., Uğraş, M., Biçer, S., et al. (2015). Mean platelet volume value changes in acute noninfectious and infectious diarrhea. *Yeditepe Medical Journal*, 9(33), 875–883.
- Küçük, Ö., Uğraş, M., Biçer, S., Giray, T., Çöl, D., Erdağ, G. Ç., ... & Vitrinel, A. (2015). Mean platelet volume value changes in acute noninfectious and infectious diarrhea. *Yeditepe Medical Journal*, 9(33), 875–883.
- Machiwal, D., Parmar, B. S., Kumar, S., Meena, H. M., & Deora, B. S. (2021). Evaluating homogeneity of monsoon rainfall in Saraswati River basin of Gujarat, India. *Journal of Earth System Science*, 130(3), 181.
- Mack, A., Vanden Hoek, T., & Du, X. (2024). Thromboinflammation and the role of platelets. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 44(6), 1175–1180. <https://doi.org/10.1161/ATVBAHA.124.320149>
- Martinod, K., & Deppermann, C. (2021). Immunothrombosis and thromboinflammation in host defense and disease. *Platelets*, 32(3), 314–324. <https://doi.org/10.1080/09537104.2020.1817360>
- Martinod, K., & Deppermann, C. (2021). Immunothrombosis and thromboinflammation in host defense and disease. *Platelets*, 32(3), 314–324. <https://doi.org/10.1080/09537104.2020.1817360>
- Maugeri, N., & Manfredi, A. A. (2025). Platelets as drivers of immunothrombosis in rheumatic diseases. *Nature Reviews Rheumatology*, 21(8), 478–493. <https://doi.org/10.1038/s41584-025-01276-z>
- Nardin Elias, D. P. (2019). Etiology and complications of acute gastroenteritis in hospitalized children. *Romanian Journal of Pediatrics*, 68(3), 172. <https://doi.org/10.37897/RJP.2019.3.4>
- Nicolai, L., Pekayvaz, K., & Massberg, S. (2024). Platelets: Orchestrators of immunity in host defense and beyond. *Immunity*, 57(5), 957–972. <https://doi.org/10.1016/j.immuni.2024.04.008>
- O'Reilly, D., Murphy, C. A., Drew, R., El-Khuffash, A., Maguire, P. B., Ni Ainle, F., & McCallion, N. (2022). Platelets in pediatric and neonatal sepsis: Novel mediators of the inflammatory cascade. *Pediatric Research*, 91(2), 359–367. <https://doi.org/10.1038/s41390-021-01715-z>
- Portier, I., & Campbell, R. A. (2021). Role of platelets in detection and regulation of infection. *Arteriosclerosis, thrombosis, and vascular biology*, 41(1), 70–78. <https://doi.org/10.1161/ATVBAHA.120.314645>
- Rayes, J., & Jenne, C. N. (2021). Platelets: Bridging thrombosis and inflammation. *Platelets*, 32(3), 293–294. <https://doi.org/10.1080/09537104.2021.1889310>
- Rista, N., & Jepisah, D. (2021). Tinjauan Pelaksanaan Pengkodean Penyakit Gastroenteritis Pasien Rawat Inap di Rumah Sakit PMC Pekanbaru Tahun 2020. *Jurnal Rekam Medis (Medical Record Journal)*, 1(2), 97–105. <https://doi.org/10.25311/jrm.Vol1.Iss2.358>
- Saputra, W. A., Mariadi, I. K., & Somayana, G. (2021). Karakteristik penyakit gastroenteritis akut pada pasien di rsup sanglah denpasar tahun 2018. *Jurnal Medika Udayana*, 10(7), 91–97.
- Scherlinger, M., Richez, C., Tsokos, G. C., Boilard, E., & Blanco, P. (2023). The role of platelets in immune-mediated inflammatory diseases. *Nature Reviews Immunology*, 23(8), 495–510. <https://doi.org/10.1038/s41577-023-00834-4>
- Sherwood L. 2020. *Fisiologi Manusia Dari Sel Ke Sistem*. 9th ed. Jakarta: EGC

- Sherwood, L. (2020). *Fisiologi manusia dari sel ke sistem* (9th ed.). EGC.
- Sugimoto, N., & Eto, K. (2017). Platelet production from induced pluripotent stem cells. *Journal of Thrombosis and Haemostasis*, 15(9), 1717–1727. <https://doi.org/10.1111/jth.13736>
- Sugimoto, N., & Eto, K. (2017). Platelet production from induced pluripotent stem cells. *Journal of Thrombosis and Haemostasis*, 15(9), 1717–1727. <https://doi.org/10.1111/jth.13736>
- Syuhada, S., Izzuddin, A., & Yudhistira, H. (2021). Perbandingan trombosit dengan antikoagulan K2EDTA. *Jurnal Ilmiah Kesehatan Sandi Husada*, 10(1), 170–176. <https://doi.org/10.35816/jiskh.v10i1.575>
- Tokarz-Deptuła, B., Palma, J., Baraniecki, Ł., Stosik, M., Kołacz, R., & Deptuła, W. (2021). What function do platelets play in inflammation and bacterial and viral infections? *Frontiers in Immunology*, 12, 770436. <https://doi.org/10.3389/fimmu.2021.770436>
- Trivigno, S. M. G., Guidetti, G. F., Barbieri, S. S., et al. (2023). Blood platelets in infection: The multiple roles of the platelet signalling machinery. *International Journal of Molecular Sciences*, 24(8), 7462. <https://doi.org/10.3390/ijms24087462>
- Wahid, S., & UA, M. (2016). *Imunologi: Lebih mudah dipahami*. Brilian Internasional.
- Wang, L., Liu, A., Wang, Y., Huang, Y., Hou, L., Zhang, A., Wang, S., & Hu, Q. (2024). Clinical analysis of 386 cases of secondary thrombocytosis in children. *Journal of Clinical Pediatrics*.
- Waroka E, Fadillah Q, Edlin. 2022. Gambaran Diagnostik dan Penatalaksanaan Gastroenteritis Dehidrasi Ringan-Sedang Pasien Anak Rawat Inap di Rumah Sakit Royal Prima Tahun 2021. *Jurnal Pendidikan dan Konseling*. 4(5):5652-5658.
- Worung, I. M., Mahartini, N. N., & Herawati, S. (2020). Hitung trombosit metode otomatis dikonfirmasi dengan hapusan darah tepi (HDT) tanpa pewarnaan dan dengan pewarnaan Giemsa di RSUP Sanglah, Bali, Indonesia. *Intisari Sains Medis*, 11(3), 1387–1391. <https://doi.org/10.15562/ism.v11i3.799>
- Yan, C., Wu, H., Fang, X., He, J., & Zhu, F. (2023). Platelet, a key regulator of innate and adaptive immunity. *Frontiers in Medicine*, 10, 1074878. <https://doi.org/10.3389/fmed.2023.1074878>
- Zaid, Y., & Merhi, Y. (2022). Implication of platelets in immuno-thrombosis and thrombo-inflammation. *Frontiers in Cardiovascular Medicine*, 9, 863846. <https://doi.org/10.3389/fcvm.2022.863846>
- Zaki, M. E. S., Alsayed, M. A. L., & Shrief, R. (2020). Study of the diagnostic value of interleukin-6 and interleukin-8 in children with acute gastroenteritis. *Germs*, 10(1), 27–33. <https://doi.org/10.18683/germs.2020.1182>