

A CASE OF SYSTEMIC LUPUS ERYTHEMATOUS PRESENTING AS THROMBOCYTOPENIA

Thanh Nguyen- Kim Le^{1*}, Son Le Hoang¹

¹Department of Applied Biochemistry, Faculty of Biotechnology, Ho Chi Minh City International University - Vietnam National University, Ho Chi Minh City, Vietnam.

Article Received: 16 January 2025 | Article Revised: 05 February 2025 | Article Accepted: 27 February 2025

*Corresponding Author: Thanh Nguyen- Kim Le

Department of Applied Biochemistry, Faculty of Biotechnology, Ho Chi Minh City International University - Vietnam National University, Ho Chi Minh City, Vietnam. DOI: <https://doi.org/10.5281/zenodo.14967031>

How to cite this Article: Thanh Nguyen- Kim Le, Son Le Hoang (2025). A CASE OF SYSTEMIC LUPUS ERYTHEMATOUS PRESENTING AS THROMBOCYTOPENIA. World Journal of Pharmaceutical Science and Research, 4(1), 937-941. <https://doi.org/10.5281/zenodo.14967031>



Copyright © 2025 Thanh Nguyen- Kim Le | World Journal of Pharmaceutical Science and Research.

This work is licensed under creative Commons Attribution-NonCommercial 4.0 International license (CC BY-NC 4.0)

ABSTRACT

This report demonstrated a rare prevalence of thrombocytopenia associated with systemic lupus erythematosus, indicative of extremely low concentrations of platelets. A 28-year-old female patient presented with a 15-year history of thrombocytopenic purpura associated with lupus for over 11 years. Unfortunately, the patient was unable to tolerate the higher doses of medications previously prescribed, owing to the severity of side effects caused by long-term medical treatment, typically water retention and high blood pressure, remarkably recorded with narrow blood pressure. Ultimately, integrative therapy combining medical treatment with patient therapeutic education and herbal supplements would be hopefully effective in bringing thrombocytopenia under control.

KEYWORDS: Autoimmune, chronic thrombocytopenia, hypertension, proteinuria, systemic lupus erythematosus.

INTRODUCTION

Systemic lupus erythematosus (SLE), commonly referred to as lupus, is a multisystemic autoimmune disease characterized by chronic inflammation causing damage to various organs including kidneys, skin, blood, joints, heart, lungs, and nervous system.^[1] Patients with lupus might experience periods of severe symptoms (flare-ups), followed by periods of remissions (symptoms are mild or no signs of lupus).^[2] The symptoms and frequency of lupus vary considerably from person to person and from time to time, commonly attributed to various factors such as age, phenotypes, and inflammatory response profiles.^[3] Typical symptoms of lupus include fatigue, fever, rash, and arthritis.

Effective management of SLE necessitates regular clinical assessment and biochemical assays, providing updated information on disease severity and adverse reactions associated with medications. Pharmacotherapy generally aims to improve the quality of life by alleviating symptoms, reducing and limiting progressive organ damage, and managing

control of triggering factors. This study reported a case of a 28-year-old female who improved clinically after long-term medication treatment. However, a challenge emerged as the patient presented with biochemical abnormalities in thrombocytes and blood pressure.

CASE PRESENTATION

A 28-year-old female patient presented with a history of thrombocytopenic purpura diagnosed in 2010, characterized by the specific symptoms of subcutaneous hemorrhages, indicative of typically low platelet (PLT) counts (under 20,000 platelets/mm³). Other symptoms included fever, fatigue, and weight loss (BMI 16.3). The patient was then treated with corticosteroids at varying doses from 16 - 48 mg per day based on platelet index.

In 2014, the patient presented with typical symptoms of SLE such as rheumatoid inflammation (fingers, knee, shoulders, toes, and ankles), and malar rash. On biochemical assay, positive ANA, an extremely high value of anti-ds-DNA at 150 U/mL, and a significant reduction in C3 and C4 complement were recorded. The medications were then continuously prescribed with the addition of hydroxychloroquine 200 mg per day. In 2021, lupus nephritis was diagnosed, indicating a kidney complication of SLE, characterized by abnormality of proteinuria (4,572- 5,530 mg/24 h) and hypertension (**Table 1**). Along with previous medications, the patient was treated onwards with mycophenolate mofetil 500 mg (2,000 mg/day) and telmisartan (40 – 80 mg/day). Additionally, calcium (500 mg) and omeprazole (40 mg) were given, aiming to reduce adverse reactions throughout the treatment.

Table 1: Summary of biochemical indices from June 2021 to December 2024.

| Period | Platelets (/mm ³) ($\rho = 0.000$) | Blood pressure (mmHg) | Proteinuria 24h (mg/L) ($\rho = 0.000$) | ANA ($\rho = 0.002$) | Anti ds-DNA (U/mL) ($\rho = 0.168$) | C3 (mg/dL) ($\rho = 0.491$) | C4 (mg/dL) ($\rho = 0.117$) |
|----------|---|-----------------------|--|----------------------------|--|----------------------------------|----------------------------------|
| II- 2021 | 208.40 ± 17.31 ^a | 125/93 | 5064.20 ± 421.27 ^a | 2.56 ± 0.48 ^a | 16.20 ± 8.90 ^a | 88.00 ± 12.88 ^a | 14.26 ± 2.70 ^a |
| I- 2022 | 196.00 ± 11.83 ^a | 141/103 | 989.33 ± 730.74 ^b | 1.92 ± 0.11 ^{ab} | 12.30 ± 5.29 ^a | 105.17 ± 16.19 ^a | 15.83 ± 1.65 ^{ab} |
| II-2022 | 206.00 ± 32.77 ^a | 131/95 | 1580.60 ± 737.83 ^b | 1.17 ± 0.23 ^{abc} | 17.00 ± 4.77 ^a | 109.50 ± 13.03 ^a | 17.18 ± 1.27 ^{ab} |
| I- 2023 | 156.33 ± 31.97 ^b | 128/80 | 636.42 ± 196.53 ^b | 1.30 ± 0.96 ^{abc} | 18.57 ± 3.62 ^a | 94.33 ± 10.73 ^a | 16.52 ± 2.72 ^{ab} |
| II- 2023 | 137.25 ± 17.33 ^b | 144/103 | 826.85 ± 557.33 ^b | 1.61 ± 0.95 ^{abc} | 16.51 ± 6.32 ^a | 93.63 ± 9.86 ^a | 14.98 ± 3.96 ^b |
| I-2024 | 131.17 ± 13.12 ^b | 132/105 | 453.27 ± 170.91 ^b | 0.87 ± 0.75 ^{bc} | 18.13 ± 2.83 ^a | 101.67 ± 21.05 ^a | 16.53 ± 2.81 ^b |
| II-2024 | 88.50 ± 11.91 ^c | 148/112 | 455.08 ± 142.52 ^b | 2.09 ± 0.25 ^c | 21.22 ± 7.92 ^a | 103.00 ± 29.79 ^a | 13.55 ± 3.79 ^b |

*"I" and "II" indicate the first and second half period of the year, respectively.
^{a, b, c}: Different letters within each column indicate the statistically significant differences.*

The biochemical assay conducted in 2024 showed that most of the SLE-related parameters were improved, including ANA, anti-ds-DNA, C3, C4 complement, and 24h-proteinuria. However, the PLT index was abnormally recorded starting to decline lower than 100,000/mm³ with the re-occurrence of subcutaneous hemorrhages (**Fig. 1 & 2**).



Fig. 1: Images of the subcutaneous hemorrhages: (L1), (L2)- left arm; (R1), (R2)- right arm.



Fig. 2: The variations in blood pressure, platelet concentrations, proteinuria 24h, and anti-ds-DNA indices of 12 months in 2024.

DISCUSSION

SLE, an autoimmune disorder, occurs when the body's own immune system mistakenly attacks the body.^[4] This event leads to the production of antibodies (B cells) attacking healthy organs, tissues, and cells, resulting in inflammation and damage to a variety of organs, typically skin, kidneys, and joints. There is no cure for lupus, but pharmacotherapy is the most common option for ameliorating symptoms. Based on the types of response, medications prescribed to treat lupus generally fall into 4 categories including immunosuppressants, antimalarials, corticosteroids, and monoclonal antibodies. However, these drugs are taken over a period of time or even a lifetime which can cause serious side effects.

SLE can be complicated by cytopenias, of which thrombocytopenia ($20 - 50 \times 10^3/\text{mm}^3$) rarely occurs in lupus patients, accounting for approximately 3 – 10%..^[5] In this case, the platelets index recorded considerable variations over time, starting at the lowest concentrations ($< 20,000/\text{mm}^3$) in 2010. This index was clinically improved under the corticosteroid treatment ($> 150,000/\text{mm}^3$) in 2023. However, the platelets index declined in 2024, with the lowest value of $75,000/\text{mm}^3$ in 8/2024. Unfortunately, the patient was unable to tolerate the higher doses of medications previously prescribed, owing to the severity of side effects caused by long-term medical treatment, typically water retention and high blood pressure. Notably, from 5/2024 to 9/2024, the patient exhibited a clinically significant narrow pulse pressure (**Figure 2**), characterized by a systolic-diastolic blood pressure differential of less than 30 mmHg.^[6] During this period, the patient was restricted from strenuous physical exertion to minimize the risk of precipitating hemorrhagic shock. Regular blood pressure monitoring was then implemented to assess potential hemodynamic instability and to proactively determine the necessity for blood transfer.^[7]

Therapeutic patient education (TPE) was then targeted as a vital therapy along with current medications. TPE involves facilitating patients with the necessary knowledge and skills related to their health conditions including self-management strategies, mental health, and appropriate nutrition, as well as enhancing medication adherence.^[8] On the other hand, herbal supplements were also recommended to patients, acting as tonic agents having the capability of

restoring and maintaining the function of organs adversely affected by medications.^[9] There is thus a significant need for continued monitoring and regular assessment of this case, and establishing appropriately personalized treatment approaches, aiming to effectively manage serious thrombocytopenia associated with SLE.

CONCLUSION

This report emphasized the rare prevalence of thrombocytopenia associated with SLE, indicative of extremely low concentrations of platelets. Regular laboratory tests and clinical assessments are necessary to address this complication. Ultimately, integrative therapy combining medical treatment with patient therapeutic education and herbal supplements would be hopefully effective in bringing thrombocytopenia under control.

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board of the International University and the Department of Nephrology and Endocrinology, Gia Dinh Hospital (Ho Chi Minh City).

ACKNOWLEDGMENTS

The authors would like to thank the patient (code: 701310-21094102) for her approval and support from Gia Dinh Hospital and Ho Chi Minh International University - Vietnam National University for granting the study authorizations.

Conflicts of Interest

The authors declared to have no conflict of interest.

Funding

This study received no funding.

REFERENCES

1. Ameer MA, Chaudhry H, Mushtaq J, Khan OS, Babar M, Hashim T, et al. An Overview of Systemic Lupus Erythematosus (SLE) Pathogenesis, Classification, and Management. *Cureus*, 2022 Oct 15.
2. Adamichou C, Bertias G. Flares in systemic lupus erythematosus: diagnosis, risk factors and preventive strategies. *MJR*, 2017 Jan 1; 28(1): 4–12.
3. Rodríguez RD, Alarcón-Riquelme ME. Exploring the contribution of genetics on the clinical manifestations of systemic lupus erythematosus. *Best Practice & Research Clinical Rheumatology*, 2024 Dec; 38(4): 101971.
4. Corzo P, Agustí Claramunt A, Garcia-Duitama I, Carrión-Barberá I, Marsico S, Duran Jordà X, et al. SLE inflammatory musculoskeletal abnormalities, confirmed by MRI, show a specific profile with a worse health-related quality of life. *Lupus*, 2025 Jan; 34(1): 10–7.
5. Galanopoulos N, Christoforidou A, Bezirgiannidou Z. Lupus thrombocytopenia: pathogenesis and therapeutic implications. *MJR*, 2017 Jan 1; 28(1): 20–6.
6. Warren J, Moazzez A, Chong V, Putnam B, Neville A, Singer G, et al. Narrowed pulse pressure predicts massive transfusion and emergent operative intervention following penetrating trauma. *The American Journal of Surgery*, 2019 Dec; 218(6): 1185–8.
7. Bonanno FG. Management of Hemorrhagic Shock: Physiology Approach, Timing and Strategies. *JCM*, 2022 Dec 29; 12(1): 260.

8. Kankaya H, Karadakovan A. Effects of web-based education and counselling for patients with systemic lupus erythematosus: self-efficacy, fatigue and assessment of care. *Lupus*, 2020 Jul; 29(8): 884–91.
9. Chou CT. Alternative therapies: what role do they have in the management of lupus? *Lupus*, 2010 Oct; 19(12): 1425–9.