Psychosomatic Fever Mimicking Lupus Flare in a Young Male with Cutaneous SLE and Severe Hyponatremia: a Rare and Challenging Diagnostic Scenario

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ABSTRACT

Fever in systemic lupus erythematosus (SLE) may result from disease activity, infection, or non-inflammatory conditions, making the diagnostic process challenging. We report a case of a man in his twenties with a history of cutaneous lupus who presented with persistent fever, fatigue, vomiting, and active skin lesions. Laboratory findings revealed mild anemia, vitamin D deficiency, and positive anti-ribosomal P and anti-Ro60 antibodies without evidence of major organ involvement. The patient received immunosuppressive therapy and metabolic correction; however, the fever persisted. Psychiatric evaluation revealed a depressive episode, and following the initiation of maprotiline therapy, the fever completely resolved. This case highlights the importance of considering psychogenic fever in patients with SLE in order to avoid overtreatment and underscores the need for a multidisciplinary approach.

Keywords: SLE; psychogenic fever; depression; cutaneous lupus; anti-ribosomal P

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by multisystem involvement, including the skin, joints, kidneys, nervous system, and other organs [1]. Fever is a common presenting symptom and may reflect disease activity, infection, or drug-related effects; therefore, careful clinical evaluation is required [2]. Determining the underlying cause of fever is essential, as misinterpretation may lead to unnecessary immunosuppressive therapy and increase the risk of complications [3].

SLE is less common in males than in females and often presents unique diagnostic challenges [4]. In addition to organic manifestations, psychiatric disorders such as depression are frequently observed in patients with SLE and may significantly affect clinical symptoms and quality of life [5]. Psychogenic fever is defined as hyperthermia associated with psychological stress in the absence of an identifiable inflammatory process and has been reported in several psychosomatic conditions [6]. This report aims to highlight the importance of considering psychosomatic causes in cases of persistent fever in patients with SLE.

CASE PRESENTATION

A 24-year-old man with a history of cutaneous lupus presented with persistent fever for several days accompanied by fatigue, recurrent vomiting, and worsening skin lesions. The patient also reported headache and an unintended weight loss of approximately 3 kg over the past month. There were no complaints of cough, dysuria, or diarrhea. A family history of autoimmune disease was denied.

On admission, the patient appeared weak but was fully conscious. Vital signs revealed blood pressure ranging from 105–123/70–71 mmHg, pulse rate of 83–98 beats per minute, respiratory rate of 19–20 breaths per minute, oxygen saturation of 98% on room air, and fluctuating body temperature. Body weight was 51 kg with a body mass index of 19.7 kg/m². Physical examination showed no focal signs of infection, edema, or neurological deficits. Dermatological examination showed well-demarcated hyperpigmented plaques on the malar areas, with central atrophy and mild scaling, consistent with cutaneous lupus erythematosus (Figure 1).

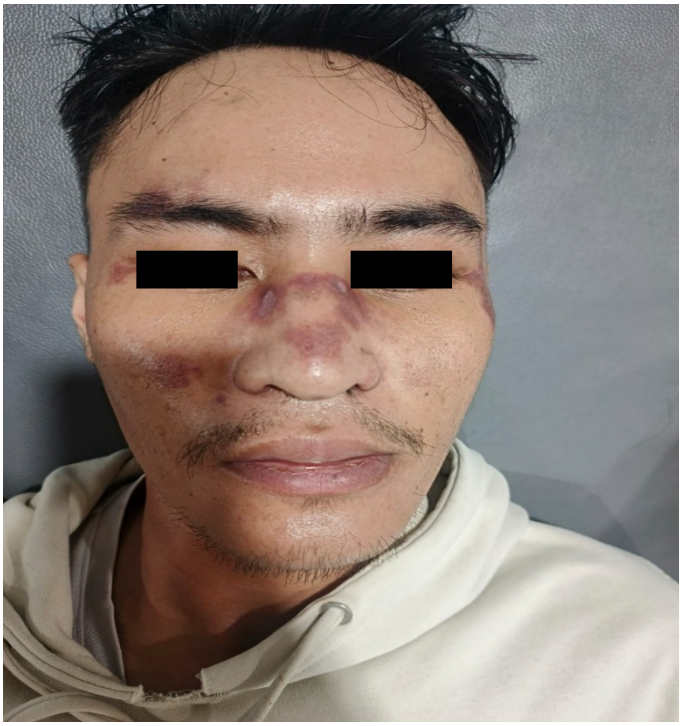

Figure 1. Clinical picture showed well-demarcated hyperpigmented plaques in malar areas with central atrophy and mild scaling.

Laboratory evaluation revealed mild anemia with hemoglobin of 10.9 g/dL, leukocyte count of 6.4 $\times 10^3/\mu L$, platelet count of 202 $\times 10^3/\mu L$, and vitamin D deficiency with a level of 20 ng/mL. Immunological testing demonstrated positive ANA (1:100 dilution), strongly positive anti-ribosomal P antibodies (+++), and moderately positive anti-Ro60 antibodies, while anti-dsDNA and anti-Sm antibodies were negative (Table 1). These findings supported the presence of cutaneous lupus activity without renal or central nervous system involvement [7]. Disease activity assessment according to the 2019 EULAR/ACR classification criteria indicated mucocutaneous manifestations with systemic symptoms including fever [4].

Table 1. Laboratory parameter of the patient at admission.

Parameter	Result
Hemoglobin	**10.9**
Hematocrit	34
White Blood Cell	6.4
Platelet	202
Red Blood Cell	4.4
MCV	**78**
MCH	**24.9**
MCHC	**31.9**
Neutrophil	72.5
Lymphocyte	12
Monocyte	2.2
PT	13.6
APTT	31.8
INR	0.95
AST (SGOT)	49
ALT (SGPT)	88
Total Bilirubin	0.59
Albumin	3.5
Creatinine	0.7
Urea	40
Sodium	**124**
Potassium	3.8
Ionized Calcium	1.25
Osmolality	**260**
Ribosomal Protein P	**+++ (Systemic Lupus Erythematosus (SLE))**
SSA (Ro) antibody	**+ (Sjögren syndrome, SLE, Neonatal lupus)**

Initially, the fever was suspected to represent an SLE flare. However, several findings raised uncertainty, including the absence of major organ involvement, normal anti-dsDNA levels, lack of evidence of infection, a fluctuating fever pattern, and relatively stable general condition. Differential diagnoses considered included SLE flare, occult infection, drug-induced fever, metabolic disturbances, and psychogenic fever [8].

The patient was admitted to the ward and treated with intravenous methylprednisolone (62.5–125 mg/day), hydroxychloroquine 200 mg/day, mycophenolate sodium 180 mg twice daily, intravenous Ringer lactate, antipyretics (paracetamol), and other supportive therapies according to the management of cutaneous lupus [9]. Hyponatremia suspected to be secondary to dehydration was corrected with fluid therapy (Table 2). Despite metabolic improvement, the fever persisted.

Table 2. Electrolyte evaluation of the patient.

Parameter	Result	After Correction
Sodium	**124**	**134**
Potassium	3.8	3.7
Ionized Calcium	1.25	1.10

A psychiatric consultation was subsequently performed and revealed a diagnosis of depressive episode without psychotic features, with a Hospital Anxiety and Depression Scale–Depression (HADS-D) score of 12 and a Beck Depression Inventory (BDI) score of 21. Antidepressant therapy with maprotiline was initiated at 12.5 mg in the morning and 25 mg at night. Following initiation of antidepressant treatment, the fever resolved and the patient's clinical condition gradually improved (Figure 2).

During hospitalization, the patient's general condition improved with resolution of pain and no recurrence of fever. At follow-up evaluation, the patient remained afebrile with improvement in psychological symptoms and stable cutaneous lupus manifestations.

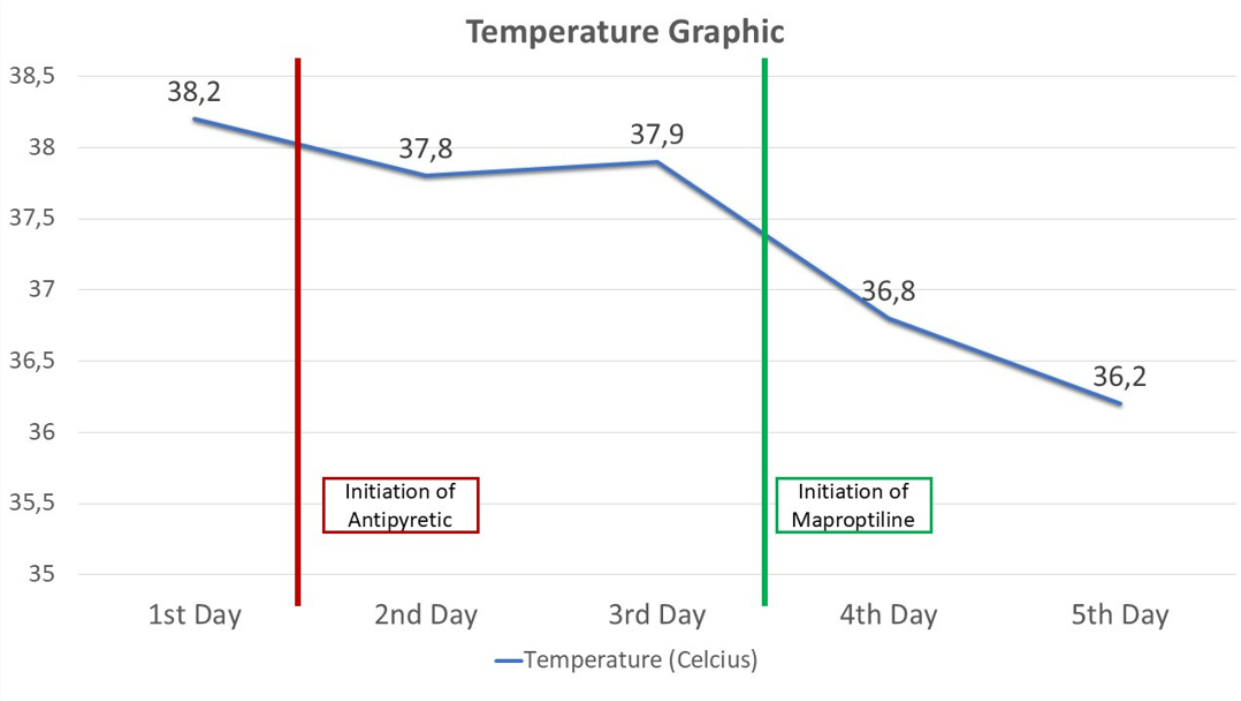

Figure 2. Trend of body temperature during hospitalization. The patient initially presented with persistent fever (38.2 °C on day 1). Antipyretic therapy was initiated on day 2 (red vertical line), resulting in only minimal reduction in temperature with persistent low-grade fever. Maprotiline therapy was initiated on day 4 (green vertical line). Following the initiation of maprotiline, the patient's body temperature progressively decreased and normalized, supporting the diagnosis of psychogenic fever.

DISCUSSION

Persistent fever in patients with SLE represents a common yet complex clinical challenge. Fever may be caused by disease activity, infection, medication reactions, or non-inflammatory conditions such as psychogenic fever [8]. Accurate identification of the underlying cause is essential, as inappropriate escalation of immunosuppressive therapy may increase the risk of adverse outcomes.

In this case, the absence of major organ involvement, stable laboratory parameters, negative anti-dsDNA antibodies, and lack of evidence of infection made active SLE flare less likely. Although the patient initially received immunosuppressive therapy, the persistent fever despite clinical stabilization prompted further evaluation [10,11].

Psychogenic fever is a stress-related hyperthermic condition mediated by autonomic nervous system dysregulation rather than inflammatory cytokine pathways [6]. Psychological stress can activate sympathetic pathways and increase body temperature through central thermoregulatory mechanisms. This condition has been reported in patients with psychiatric disorders, particularly depression and anxiety.

Depression is relatively common in patients with SLE and may be related to chronic disease burden, immune dysregulation, and psychosocial stress [5,12]. In this case, psychiatric evaluation revealed moderate depressive symptoms. Remarkably, the patient's fever resolved completely following antidepressant therapy, strongly supporting the diagnosis of psychogenic fever.

This case highlights the importance of considering psychosomatic causes in patients with persistent fever when infectious and inflammatory causes have been excluded. Multidisciplinary collaboration between rheumatologists, internists, and psychiatrists is crucial to ensure accurate diagnosis and optimal patient management.

CONCLUSION

Persistent fever in patients with SLE does not always indicate disease activity. After excluding infection and systemic flare, psychogenic fever should be considered, particularly in patients with depressive symptoms. Early psychiatric evaluation may prevent misdiagnosis and unnecessary escalation of immunosuppressive therapy. A multidisciplinary approach is essential to achieve optimal clinical outcomes.

Informed Consent Statement

Informed consent was obtained from all subjects involved in the study. Written informed consent has been obtained from the patient(s) to publish this paper.

Conflicts of Interest[1]

The authors declare no conflict of interest

Author Contributions

For research articles with several authors, a short paragraph specifying their individual contributions must be provided. The following statements should be used "Conceptualization, Y.S.P. and B.P.P.R.; methodology, Y.S.P. software, J.V.R. validation, A.J.S, R.A.A..; formal analysis, B.P.P.R; investigation, Y.S.P..; resources, Y.S.P.; data curation, A.J.S.; writing—original draft preparation, Y.S.P..; writing—review and editing, R.A.A., A.J.S..; visualization, J.V.R.; supervision, A.J.S, R.A.A.; project administration, B.P.P.R.; funding acquisition, Y.S.P. All authors have read and agreed to the published version of the manuscript.

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Data Availability Statement

Data supporting the reported results are available in the medical record archives of Moewardi General Hospital. Due to privacy restrictions, the data is not publicly available.

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References

1. Siegel CH, Sammaritano LR. Systemic lupus erythematosus: a review. JAMA. 2024;331(17):1480–91.

2. Doria A, Zen M, Canova M, Bettio S, Bassi N, Nalotto L, et al. SLE diagnosis and treatment: when early is early. Autoimmun Rev. 2013;12(1):55–60.

3. Olivieri G, Ceccarelli F, Perricone C, Ciccacci C, Pirone C, Natalucci F, et al. Fever in systemic lupus erythematosus: associated clinical features and genetic factors. Clin Exp Rheumatol. 2022;40(11):2141–6.

4. Aringer M, Costenbader K, Daikh D, Brinks R, Mosca M, Ramsey-Goldman R, et al. European League Against Rheumatism/American College of Rheumatology classification criteria for systemic lupus erythematosus. Ann Rheum Dis. 2019;78(9):1151–9.

5. Hanly JG. Neuropsychiatric lupus. Rheum Dis Clin N Am. 2014;40(1):15–33.

6. Oka T. Psychogenic fever: a psychosomatic disease caused by stress. Temperature. 2015;2(3):368–78.

7. Okon LG, Werth VP. Cutaneous lupus erythematosus: diagnosis and treatment. JAMA Dermatol. 2013;149(3):284–92.

8. Tsokos GC. Systemic lupus erythematosus. N Engl J Med. 2011;365(22):2110–21.

9. Kuhn A, Landmann A, Bonsmann G. Cutaneous lupus erythematosus: update of therapeutic options part I. J Am Acad Dermatol. 2016;74(5):943–59.

10. King N. Advances in the diagnosis and management of systemic lupus erythematosus: a review of recent clinical guidelines. Int J Clin Rheumatol. 2024;19(9):313–5.

11. Lazar S, Kahlenberg JM. Systemic lupus erythematosus: new diagnostic and therapeutic approaches. Annu Rev Med. 2023;74:339–52.

12. Bertsias GK, Ioannidis JPA, Aringer M, Bollen E, Bombardieri S, Bruce IN, et al. EULAR recommendations for the management of systemic lupus erythematosus with neuropsychiatric manifestations. Ann Rheum Dis. 2010;69(12):2074–82.