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Sweet syndrome in the second trimester of pregnancy: a case report

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Case Report

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Abstract

Background

First reported by Robert Douglas Sweet, acute febrile neutrophilic dermatosis, or Sweet syndrome, is a rare inflammatory skin disease[1]. Its typical manifestations consist of tender erythematous skin plaques, fever and elevated neutrophil counts in peripheral blood. Gestational Sweet syndrome (PASS) is an infrequent obstetric-dermatological condition, and clinical onsets limited to the second trimester remain extremely rare[4,5]. Pregnant patients admitted for fever and cutaneous eruptions are usually suspected to suffer from infectious processes. This diagnostic bias frequently results in misclassification of PASS as cellulitis or sepsis, which brings about diagnostic delays and unnecessary antibiotic exposure for gravidas[6]. This paper describes a classic PASS episode emerging at 15 weeks of gestation in a patient without underlying chronic illnesses, highlighting diagnostic obstacles and positive clinical outcomes delivered through interdisciplinary collaboration between obstetric and dermatology teams.

Case presentation

A 40-year-old Han Chinese multiparous woman (G2P1) at 14^{+6} weeks of gestation presented with abrupt fever reaching up to 39.4 °C, together with chills, dizziness, general malaise and mild vomiting-predominant gastrointestinal complaints. Cutaneous assessment detected extensive tender erythematous plaques covered with tiny pustules over the face, trunk and extremities. No mucosal damages were observed in either oral or ocular sites. Intravenous cefuroxime given as initial empirical therapy failed to suppress febrile episodes. Blood tests showed white blood cell counts of 14.99×10^9/L with neutrophil proportion of 89.2 %, alongside significantly elevated high-sensitivity C-reactive protein(129.06 mg/L).Skin punch biopsy demonstrated epidermal hyperkeratosis, acanthosis, intra-epidermal neutrophil infiltration and microabscess formation. Dermal layers presented mixed inflammatory cell infiltration including lymphocytes, neutrophils and histiocytes. Immunohistochemical assays returned positive CD15 and MPO staining, while CD34 and CD117 tests came out negative. After formal dermatological consultation, Sweet syndrome was confirmed. Daily intravenous methylprednisolone 60 mg was initiated. The patient's fever subsided rapidly, and skin lesions gradually resolved within days. Treatment was then switched to oral methylprednisolone 28 mg daily following a pre-planned tapering protocol. Both maternal physical status and fetal condition stayed stable throughout hospital admission and subsequent follow-up. No recurrent fever or skin flare-ups were noted.

Conclusions

When pregnant women develop fever, painful erythematous plaques and peripheral neutrophilia that show no response to anti-infective treatment, clinicians ought to raise vigilance for Sweet syndrome. Early skin biopsy constitutes a vital tool to achieve definitive diagnosis and avoid prolonged unnecessary empirical antibiotic administration. Under joint monitoring by obstetricians and dermatologists, systemic

glucocorticoids with appropriate starting doses and gradual dose reduction strategies can generate favourable maternal-fetal clinical outcomes.

Background

Described in 1964 by Robert Douglas Sweet, acute febrile neutrophilic dermatosis (Sweet syndrome) belongs to an uncommon inflammatory dermatosis entity [1]. Clinically it presents with sudden-onset painful oedematous erythematous plaques, fever and peripheral neutrophilia. Histological features feature dense neutrophilic accumulation within dermal tissues, without true vasculitic changes. This disease can be divided into three major subgroups: classic idiopathic form, drug-induced type and malignancy-associated subtype [2].

PASS accounts for roughly 2% of all Sweet syndrome diagnoses and seldom appears in obstetric practice [3]. According to existing clinical records, most PASS episodes take place in the first trimester or postpartum phase. Second-trimester PASS cases are only sporadically documented across published literature [4, 5]. For pregnant women with fever and skin rashes, infectious etiologies such as cellulitis, pyelonephritis and chorioamnionitis occupy top priorities during initial diagnostic work-up. Under such clinical mindset, PASS patients may receive long-course empirical antibiotics with limited therapeutic effects [6]. Delayed correct identification will increase maternal inflammatory burden and expose fetuses to redundant pharmaceutical agents.

We report one classic Sweet syndrome case occurring at approximately 15 gestational weeks in a previously healthy patient. Initial clinical work-up inclined toward infectious-origin fever of unknown origin. The accurate diagnosis could only be established after broad-spectrum antibiotics yielded no improvement and skin biopsy results became available. Our case illustrates diagnostic challenges related to PASS, stresses the value of early skin biopsy, and validates the safety of systemic glucocorticoid intervention managed by multi-specialty teams. This case report has been prepared in full compliance with CARE reporting guidelines [7].

Case presentation

A 40-year-old Chinese patient with advanced maternal age, G2P1, visited our medical centre on 13 August 2026 at 14^{+6} weeks gestation, complaining of abrupt fever and chills. Her body temperature was 37.7°C on admission. Associated manifestations covered dizziness, general fatigue and gastrointestinal discomfort: she vomited up to three times every day, with non-bilious and non-projectile gastric contents. She denied cough, sputum production, chest tightness, dyspnoea, abdominal pain, diarrhoea, vaginal bleeding or amniotic fluid leakage. She had no noteworthy past medical or obstetric history, and had never experienced febrile illness or skin lesions before this pregnancy.

Given persistent fever, she got admitted to the infectious disease department on 14 August 2026 for fever of unknown origin. Empirical intravenous cefuroxime 1.5 g was administered every 12 h.

Nevertheless, fever persisted and deteriorated, with peak temperature hitting 39.4°C. On 15 August 2026, she was transferred to the obstetric ward for further evaluation and therapeutic adjustment.

Physical examination after ward transfer identified widespread tender erythematous plaques across the face, trunk and limbs(Figure 2). Scattered pinpoint pustules could be seen on top of erythematous skin, accompanied by mild pruritus. No abnormalities were detected in oral mucosa or conjunctivae. Obstetric assessment confirmed viable intrauterine pregnancy matching 15-week gestational age, and fetal heart rhythm remained regular.

Baseline laboratory findings on admission were listed below: white blood cell count 14.99 × 10^9/L (reference range, 3.5–9.5×10^9/L); neutrophil percentage 89.20% (40–75%); lymphocyte percentage 5.50% (20–50%); monocyte count 0.78×10^9/L; eosinophil percentage 0%; haemoglobin 118 g/L; high-sensitivity C-reactive protein 129.06 mg/L (< 5 mg/L).

Punch biopsy specimens were collected from erythematous lesions on her right upper extremity. Under ×200 magnification of haematoxylin-eosin staining, epidermal hyperkeratosis, acanthosis and elongated rete ridges could be visualised. Massive neutrophil infiltration occurred within epidermis, and microabscesses formed inside the stratum corneum. Superficial dermis contained mixed inflammatory cell infiltrates including lymphocytes, neutrophils and histiocytes, whereas leukocytoclastic vasculitis was excluded. Immunohistochemical staining showed CD15(+) and MPO(+), proving myeloid lineage of infiltrating inflammatory cells. CD34 and CD117 negative results excluded vascular and mast-cell neoplastic lesions, while CD68 positivity marked histiocyte components(Figure 1).

Combining typical clinical features (painful pustule-covered erythematous plaques, sustained fever and peripheral neutrophilia) and histological evidence showing dense dermal neutrophilic infiltration without vasculitis, Sweet syndrome was diagnosed upon dermatology specialist consultation. The patient reached 15 gestational weeks at the time of final diagnosis.

After multidisciplinary group discussion and obtaining informed patient consent, systemic glucocorticoid therapy was initiated. From 15 August to 18 August 2026, intravenous methylprednisolone 40 mg was given once daily(Figure 3). However, fever control remained suboptimal, and body temperature fluctuated between 36.2°C and 39.5°C. The dose was raised to intravenous methylprednisolone 60 mg per day starting on 19 August 2026. Her temperature returned to normal and remained stable for more than 72 h. On 24 August 2026, intravenous dosage was reduced to 40 mg daily August 2026, intravenous methylprednisolone 40 mg was given once daily（Figure 4）.On 28 August 2026（Figure 5）, treatment shifted to oral methylprednisolone 28 mg taken in a single morning dose (seven 4-mg tablets). Body temperature variations in the patient during treatment（Figure 6）. A standardized tapering strategy was formulated: if no fever or new skin lesions appeared within three consecutive days, one tablet would be reduced every three days. Dose reduction continued from 24 mg downwards until reaching one-tablet daily maintenance dose, which would be sustained for 1-2 weeks before complete discontinuation. Serial obstetric monitoring including fetal heart rate examination and

ultrasound scans demonstrated normal fetal growth and amniotic fluid volume consistent with gestational age.

At discharge, skin lesions had largely regressed, leaving residual post-inflammatory hyperpigmentation without newly-emerging eruptions. The patient left hospital under stable physical condition. During outpatient follow-up, she strictly complied with tapering medication schedules. Up to manuscript preparation, she stayed afebrile, with continuous improvement of original skin lesions and zero disease recurrence. Joint follow-up managed by dermatology and obstetric departments is ongoing to monitor maternal inflammatory status as well as fetal wellbeing, and the pregnancy remains uncomplicated.

Figure 5.Near-complete resolution of skin lesions with residual post-inflammatory hyperpigmentation after 11 days of corticosteroid therapy.

Figure 6. Body temperature trend during hospitalization. (Take 00:00 on August 14, 2026 as the base time)

Discussion

Sweet syndrome, or acute febrile neutrophilic dermatosis, represents a rare auto-inflammatory dermatosis driven by over-activated innate immune responses [16]. Its core manifestations include abrupt-onset tender erythematous plaques, fever, elevated peripheral neutrophils and prominent dermal neutrophilic infiltration, without primary leukocytoclastic vasculitis. Four major subgroups have been recognised: classic idiopathic variant, malignancy-related form, drug-induced subtype, plus special histological patterns such as subcutaneous Sweet syndrome. PASS falls within classic idiopathic Sweet syndrome, contributing to approximately 2% of all Sweet syndrome diagnoses [3]. Clinical evidence regarding PASS mostly comes from scattered individual case reports rather than large-scale cohort studies, which limits the strength of clinical guidance [16].

The exact pathogenic pathways for Sweet syndrome have not been fully clarified. Current viewpoints categorise it as an innate-immunity-mediated auto-inflammatory disorder, instead of conventional autoimmune diseases mediated by autoantibodies. Triggering factors including infections, drug exposure, malignant tumours and gestation can activate keratinocytes and tissue-resident macrophages. These activated cells secrete multiple pro-inflammatory cytokines such as IL-1β, IL-6, TNF-α and G-CSF [8]. Such mediators promote neutrophil maturation in bone marrow and recruit neutrophils toward dermal tissues, eventually leading to epidermal oedema, erythema and skin tenderness. Recent research has revealed the roles of NETosis and downstream IL-36 pathway amplification in sustaining local skin inflammation, which may partly interpret steroid-insensitive clinical presentations [9]. During gestation, immune reconstruction events covering Th1-Th2 immune shift, elevated oestrogen-progesterone level and placenta-derived cytokine production create an immune microenvironment susceptible to neutrophilic dermatosis [8]. This mechanism matches our patient, for whom we detected no additional predisposing risk factors.

Diagnostic frameworks for Sweet syndrome were first put forward by Su and Liu in 1986 and later revised by von den Driesch in 1994, which are still widely applied in clinical practice and case-report publications [10, 14]. Diagnostic confirmation demands satisfaction of two major criteria plus at least two out of four minor criteria. Major criteria contain: (1) acute onset of painful or tender erythematous plaques or nodules; (2) histology showing heavy dermal neutrophilic infiltration without leukocytoclastic vasculitis. Minor criteria include body temperature higher than 38°C; preceding or relevant triggers such as infection, malignancy, inflammatory disorders or pregnancy; rapid clinical improvement after systemic glucocorticoid or potassium iodide treatment; laboratory abnormalities including elevated ESR, CRP, white blood cell or neutrophil percentage. Our patient fulfilled both major criteria and multiple minor criteria: peak fever up to 39.4°C, pregnancy as the trigger factor, markedly increased high-sensitivity CRP (129.06 mg/L), leukocytosis (14.99 × 10^9/L) and obvious neutrophilia (89.2%). The above evidence supports the diagnosis of classic Sweet syndrome. Given that some pregnant patients may not satisfy all minor criteria, skin biopsy acts as the decisive tool to obtain definite diagnosis.

For febrile pregnant patients accompanied by skin rashes, infectious diseases always rank high among differential diagnoses. Our patient initially received intravenous cefuroxime targeting presumed bacterial infection, which aligns with diagnostic-delay phenomena described in previous PASS literature [6]. Subcutaneous-pattern Sweet syndrome, whose inflammatory lesions mainly involve subcutaneous fat tissue, is highly prone to being misdiagnosed as cellulitis. Such misjudgement may bring long-term empirical antibiotic therapy and delayed biopsy collection [6, 11]. Other differential diagnoses cover pyoderma gangrenosum, erythema nodosum, ANCA-associated vasculitis, drug eruption and adult-onset Still's disease. If appropriate anti-infective interventions cannot control fever while skin lesions keep progressing, clinicians should suspect non-infectious inflammatory dermatosis and arrange timely skin biopsy. In our case, biopsy results displayed epidermal hyperkeratosis, microabscess formation and dense dermal neutrophilic infiltration validated via CD15 and MPO immunohistochemistry; vasculitic changes were absent, consolidating our final diagnosis.

Existing narrative reviews summarise PASS clinical experiences and indicate most episodes arise in the first trimester or postpartum period, while second-trimester cases remain uncommon [11]. Many previously-reported second-trimester PASS cases co-existed with underlying comorbidities. One documented dialysis-dependent pregnant patient suffering from diabetic nephropathy developed PASS together with life-threatening myopericarditis and pericardial /tamponade requiring pericardiocentesis [4]. Another subcutaneous-variant PASS case simulated cellulitis; the patient underwent long-time antibiotic administration before biopsy confirmed the real diagnosis [6]. A 2025 published case reported a 24-year-old primigravida at 17 weeks gestation presenting with special dirt-adherent-like facial lesions without prominent fever or obvious leukocytosis [5]. PASS combined with inflammatory bowel disease may respond poorly to glucocorticoids and requires anti-TNF-α biological agents [13]. Severe postpartum necrotising Sweet syndrome with extensive skin necrosis and shock has also been reported in the literature [15]. Distinct from those above-mentioned cases, our patient was 40-year-old with advanced maternal age, free of pre-existing systemic diseases. Her disease manifested at 14^{+6} weeks gestation as

purely cutaneous-limited classic Sweet syndrome, characterised by generalised painful pustule-containing erythematous plaques, epidermal microabscess and prominent systemic inflammatory markers. This case enriches clinical evidence for uncomplicated second-trimester PASS without underlying comorbidities.

Systemic glucocorticoids remain first-line therapeutic options for Sweet syndrome. For pregnant populations, prednisone and prednisolone are generally preferred. Placental 11β-HSD2 enzyme can metabolise 80-90% of these agents, limiting fetal drug exposure [12]. Methylprednisolone adopted for acute-phase management in our case possesses comparable placental metabolic features. Our clinical observation suggested that 40 mg daily initial methylprednisolone failed to suppress inflammatory activity, and dose escalation to 60 mg per day achieved rapid fever resolution. Subsequent conversion to oral preparation plus gradual tapering was well-tolerated and prevented disease rebound. Glucocorticoid use during pregnancy carries potential maternal risks such as gestational diabetes, hypertension, premature rupture of membranes and preterm labour. Nevertheless, these hazards should be weighed against risks induced by uncontrolled maternal systemic inflammation, which may trigger fetal growth restriction and adverse pregnancy outcomes. In our clinical scenario, close maternal-fetal surveillance throughout treatment did not detect obvious adverse events. For mild localised PASS cases, expectant observation or high-potency topical glucocorticoids can be considered. Drugs including dapsone, colchicine and potassium iodide are not favoured in pregnancy due to insufficient safety data. For steroid-refractory PASS, anti-TNF-α biologics have been mentioned in sporadic reports, yet multi-disciplinary risk-benefit evaluation is mandatory before application [13].

With timely diagnosis and appropriate intervention, most PASS patients achieve favourable maternal-neonatal prognosis. Still, severe complications including myopericarditis, pericardial tamponade and necrotising skin lesions may induce serious maternal harm [4, 15]. Most patients witness skin lesion regression after delivery, though postpartum recurrence may occur in a subset of individuals. Patients with PASS history have higher relapse risk in subsequent pregnancies, and such information ought to be delivered during clinical counselling. Our patient is still under dual-specialty follow-up, and her pregnancy proceeds without complications.

Several limitations of the present case report should be noted. First, this study is merely a single-case description. Long-term outcome data for the mother and neonate are not yet available since follow-up is still ongoing. Second, similar to other published PASS-related literature, clinical evidence in this report is derived exclusively from case reports and small-size case series. Large-sample controlled studies cannot be performed owing to the rarity of this disorder [16]. Nevertheless, the typical clinical manifestations together with the rapid therapeutic response to glucocorticoids strongly support the diagnosis of Sweet syndrome in this case, rather than vasculitis or connective-tissue autoimmune disease.

Key learning points

1. Sweet syndrome should be included in the differential diagnosis of persistent fever with painful skin lesions in the second trimester of pregnancy, especially when empiric antibiotic therapy shows no effect.
2. Early skin punch biopsy is the key examination to confirm the diagnosis and avoid prolonged unnecessary antibiotic exposure in pregnant patients.
3. Multidisciplinary collaboration between obstetricians and dermatologists enables timely diagnosis, appropriate glucocorticoid dose titration and safe maternal-fetal monitoring for PASS.

Conclusions

Clinicians should keep Sweet syndrome within differential diagnosis for pregnant patients with acute fever, painful erythematous plaques and peripheral neutrophilia refractory to standard antimicrobial therapy. Prompt skin biopsy facilitates definite diagnosis and avoids prolonged unnecessary antibiotic usage. Under joint obstetric-dermatology supervision, adequate-dose systemic glucocorticoids with gradual tapering strategies can realise satisfactory disease control with acceptable maternal-fetal safety. Enhanced awareness toward PASS among obstetricians and emergency-department physicians helps shorten diagnostic delay and optimise maternal-neonatal prognoses.

Patient perspective

This 40-year-old second-gravida patient experienced tremendous psychological distress after sudden fever and widespread skin eruptions. She recalled feeling confused and frightened: clinicians initially suspected infectious aetiology, yet her symptoms kept worsening despite antibiotic therapy. She appreciated the multi-specialty collaborative model from obstetric and dermatology teams. After thorough risk-benefit explanation for both herself and fetus, she fully understood and agreed to skin biopsy as well as glucocorticoid treatment. She expressed considerable anxiety regarding potential steroid-related fetal hazards, and repeated clinical communication plus serial surveillance alleviated such worries. After treatment intervention, her physical status improved rapidly: fever disappeared and skin lesions faded progressively. She felt relieved that her pregnancy stayed stable. She hoped that publication of this real-world case could help obstetric caregivers and pregnant populations recognise this rare dermatosis at an early stage, so as to avoid redundant long-course antibiotics and obtain timely therapeutic management.and receive timely treatment.

Abbreviations

AFND
Acute febrile neutrophilic dermatosis
CRP
C-reactive protein
G-CSF
Granulocyte colony-stimulating factor

HE
Hematoxylinand eosin
IHC
Immunohistochemistry
IL
Interleukin
PASS
Pregnancy-associated Sweet syndrome
Th1
T helper type 1
Th2
T helper type 2
TNF-α
Tumor necrosis factor-alpha.

Declarations

Ethics approval and consent to participate:

This is a retrospective single-case report based on routine clinical care. According to the policy of the Medical Ethics Committee of Xiaogan Central Hospital, formal ethical approval was waived for this de-identified retrospective case report. All procedures were performed in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from the patient for participation in this report.

Consent for publication:

Written informed consent was obtained from the patient for the publication of this case report, including clinical photographs, histopathological images, temperature curve and relevant clinical data. All identifying personal information has been fully anonymized to protect patient privacy.

Competing interests:

The authors declare that they have no competing interests.

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Author Contribution

Jiao Hong collected clinical data, organized clinical materials, performed literature review and drafted the manuscript. Jia Hu conceived this case report, critically revised the manuscript, and gave final approval of the version to be published. All authors have read and approved the final submitted manuscript.

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Availability of data and materials:

All data supporting the findings are included within this article. Raw clinical records are stored in the electronic medical record system of Xiaogan Central Hospital and are not publicly available to preserve patient confidentiality.

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Figures

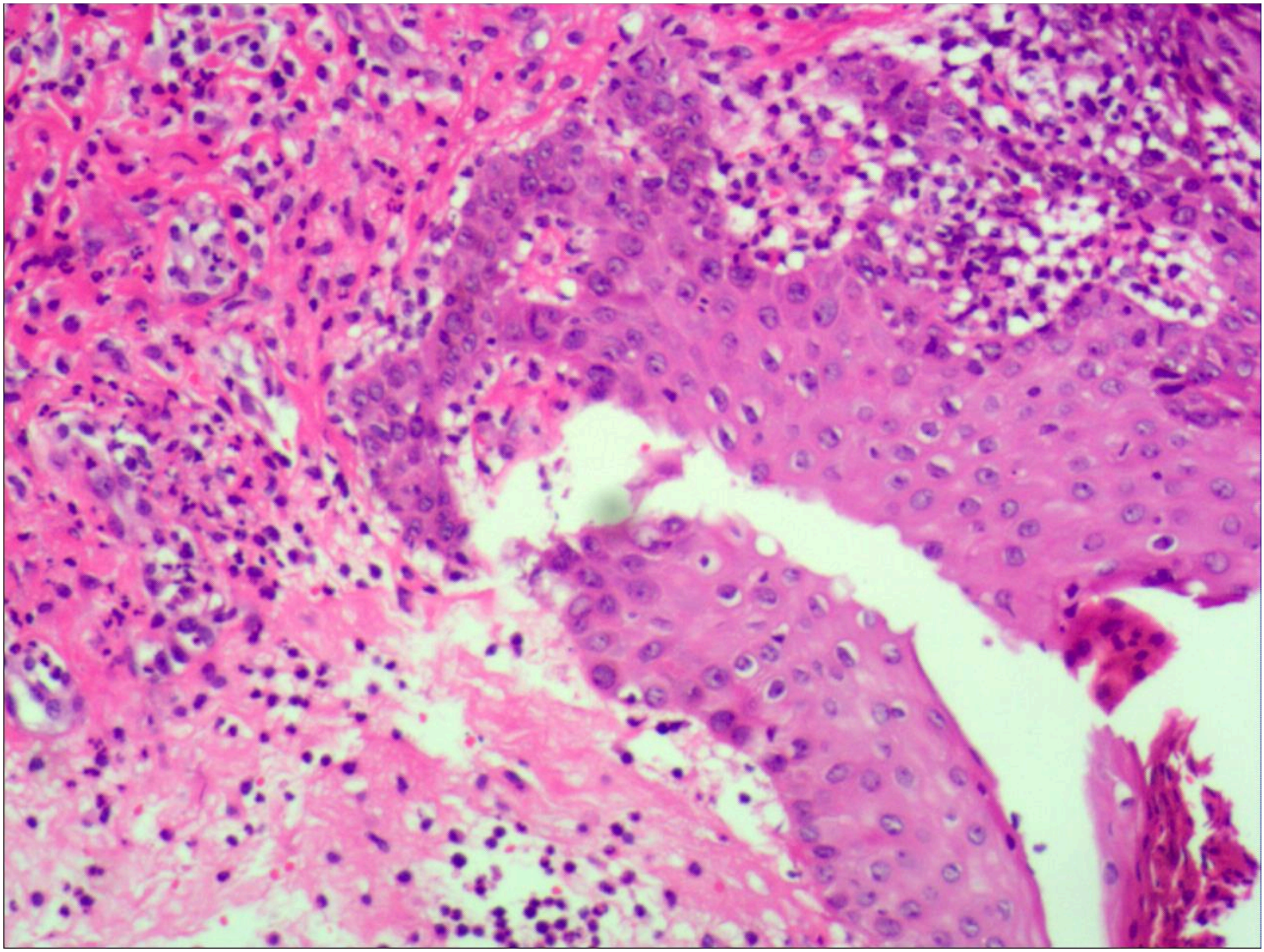


Figure 1

Skin histopathology of the right upper extremity lesion (hematoxylin and eosin staining, ×200).

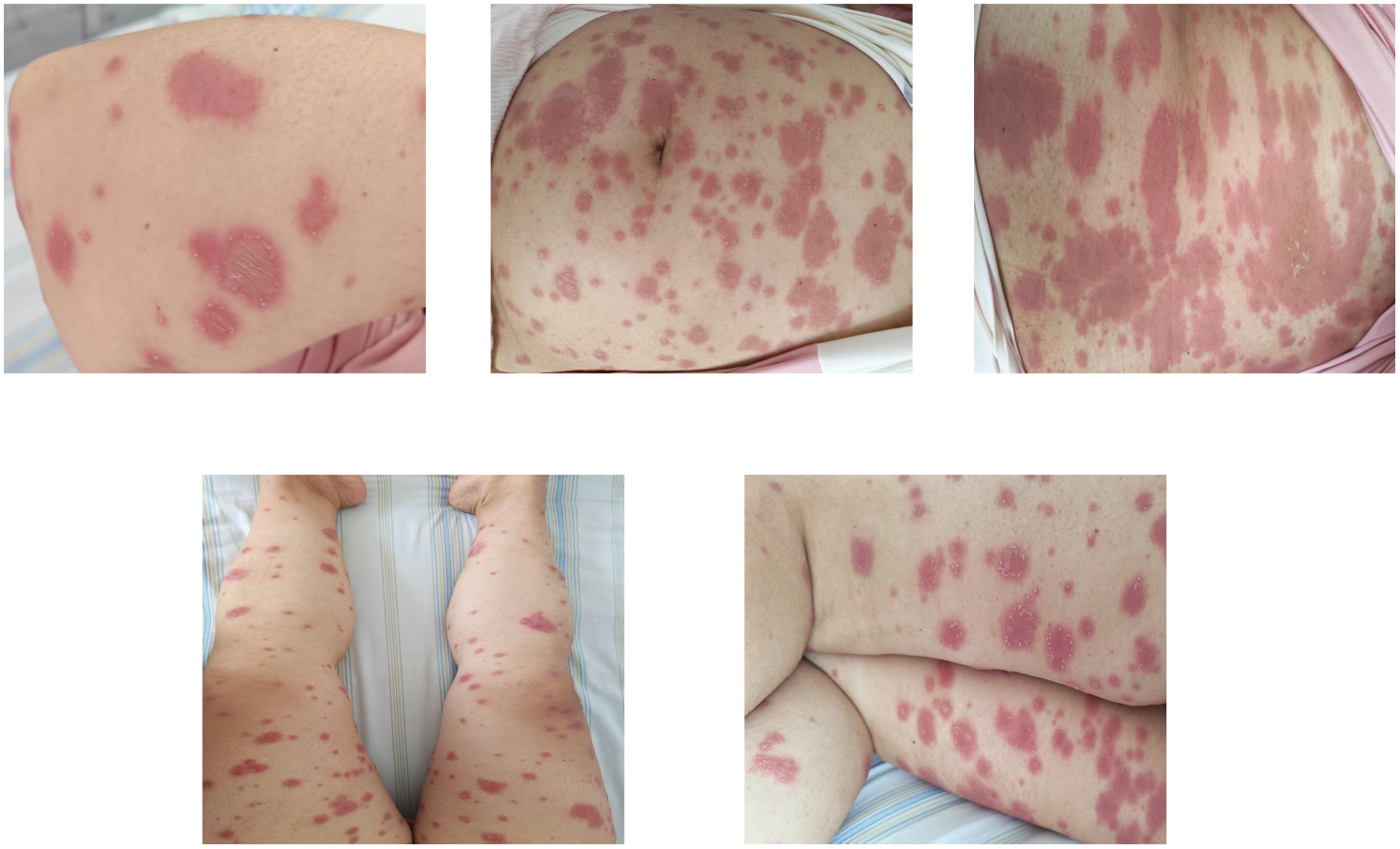


Figure 2

Widespread painful erythematous plaques with scattered pinpoint pustules on the face, trunk, and extremities before systemic corticosteroid therapy

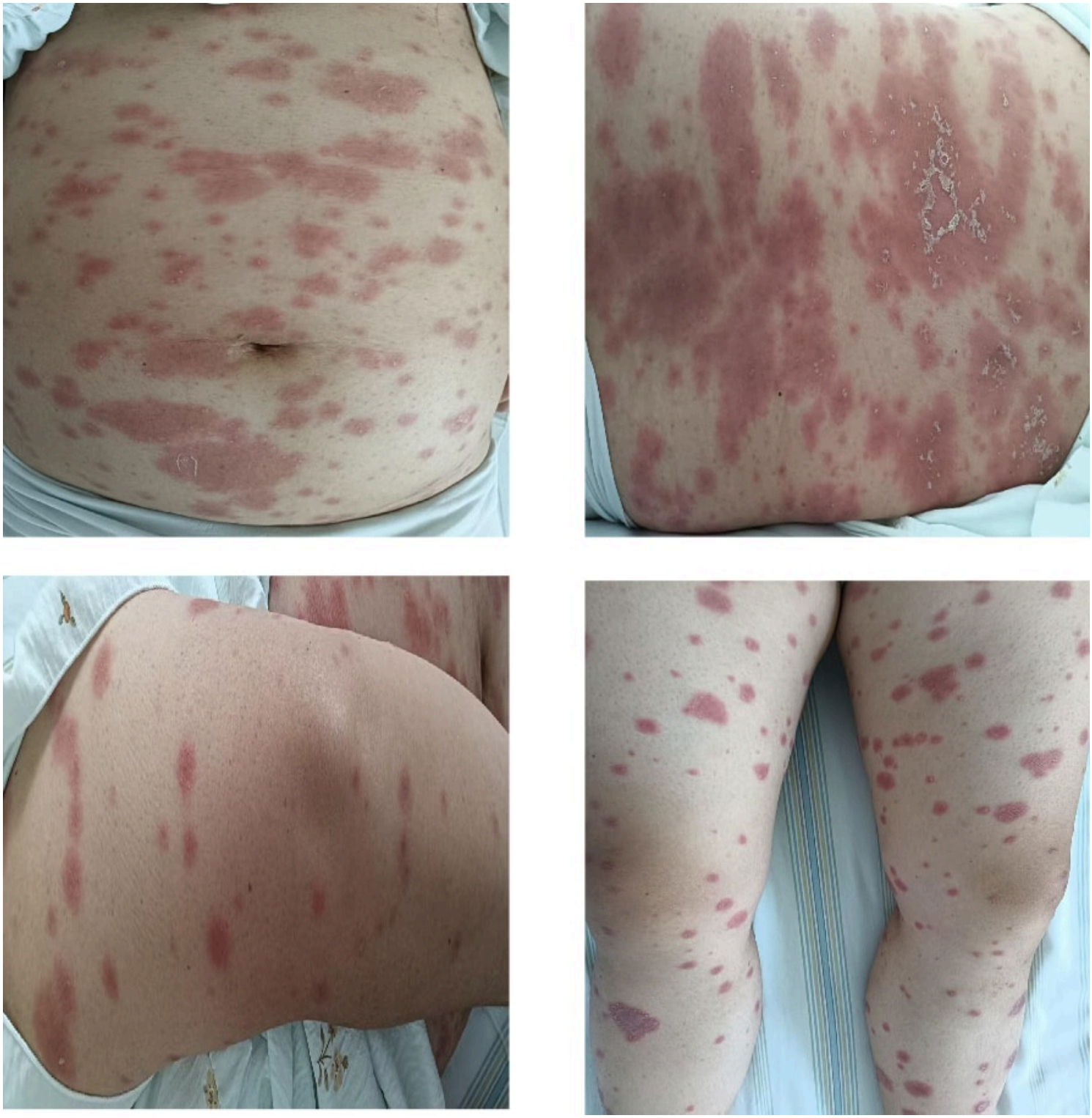


Figure 3

Partial resolution of erythematous plaques after 5 days of intravenous methylprednisolone therapy.



Figure 4

Marked improvement of skin lesions after 8 days of corticosteroid treatment.



Figure 5

Near-complete resolution of skin lesions with residual post-inflammatory hyperpigmentation after 11 days of corticosteroid therapy.

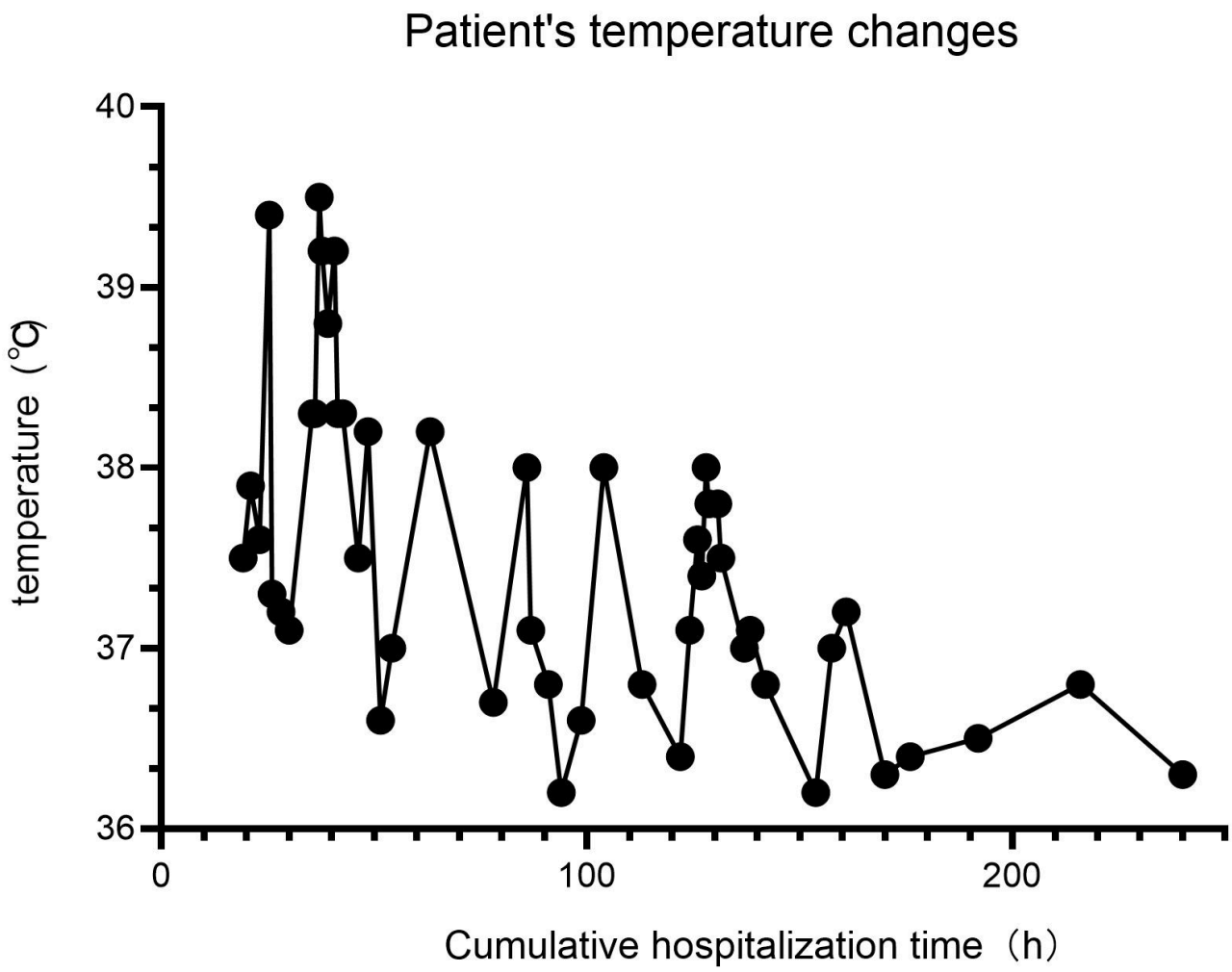


Figure 6

Body temperature trend during hospitalization. Take 00:00 on August 14, 2026 as the base time