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Pustules in Stevens-Johnson Syndrome

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Patient: Male, 25-year-old
Final Diagnosis: Stevens-Johnson syndrome
Symptoms: Fever • rash
Clinical Procedure: Biopsy • blood test
Specialty: Allergology • Dermatology • General and Internal Medicine • Pathology

Objective: Rare disease

Background: Stevens-Johnson syndrome (SJS) is characterized by widespread, epidermal necrosis and mucosal involvement mediated by a delayed-type hypersensitivity reaction. Although rapidly progressive epidermal detachment is known to result in blister formation, pustular lesions are rare in SJS.

Case Report: A 25-year-old male patient with no significant medical history or regular medication presented to the emergency department with a fever and rash. The fever had developed 7 days before the current presentation and was followed 4 days later by lip swelling, conjunctival hyperemia, and sore throat, which caused difficulty with oral intake. Two days before presentation, a generalized rash and dysuria developed, prompting evaluation at our hospital. A clinical examination found multiple, 3-mm pustules surrounding erythema on the face, chest, and back. Scattered erosions were observed on less than 10% of body surface, including the distal extremities, lips, buccal mucosa, and genital area. The conjunctivae displayed marked pseudomembrane formation. Histopathological analysis found that the erosions contained necrotic keratinocytes in the epidermis, with mild vacuolar changes at the dermo-epidermal junction, while the pustules contained serous exudate with scattered neutrophils. Based on these findings, SJS was diagnosed. A drug-induced lymphocyte stimulation test was positive for loxoprofen. The patient responded well to prednisolone therapy.

Conclusions: Although no drug exposure or infection preceding the symptoms was identified at admission, SJS was diagnosed by exclusion on the basis of the clinical manifestations and diagnostic criteria. A drug-induced lymphocyte stimulation test may help identify the causative drug. Neutrophilic infiltration into blisters formed by progressive, epidermal necrosis may occasionally result in pustule-like eruptions in SJS.

Keywords: pathology • pustules • Stevens-Johnson syndrome

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/953161

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Introduction

Stevens-Johnson syndrome (SJS) is a severe mucocutaneous disorder characterized by widespread epidermal necrosis and mucosal involvement. It presents with fever, malaise, and rapidly spreading exudative erythematous lesions of varying sizes throughout the body, which makes it an important differential diagnosis of fever and rash. A delayed-type hypersensitivity reaction underlies the disease, with cytotoxic T cells and related immune mechanisms playing a central role in keratinocyte apoptosis. Furthermore, epidermal detachment in SJS involves less than 10% of the body surface area, whereas it involves more than 30% of the body surface area in toxic epidermal necrolysis (TEN). Although rapidly progressive epidermal detachment is known to cause blisters, pustular lesions are rare in SJS. Given that SJS and TEN are potentially fatal conditions having a 1-year mortality rate of 24% and 49%, respectively, prompt diagnosis and appropriate treatment are essential [1]. We herein report a case of SJS presenting with pustular lesions, which was diagnosed by exclusion on the basis of histopathological findings in a timely manner, allowing appropriate treatment to be administered.

Case Report

A 25-year-old male patient with no significant medical history or regular medication presented to the emergency department with a fever and rash. The fever had developed 7 days before the current presentation and was followed 4 days later by lip swelling, conjunctival hyperemia, and sore throat, which caused difficulty with oral intake. Tests by the local public health authority were negative for measles and rubella. Two days before presentation, a generalized rash and dysuria developed, prompting evaluation at our hospital. The patient denied upper respiratory and gastrointestinal symptoms. The skin findings revealed multiple, 3-mm pustules surrounded by erythema on the face, chest, and back, which in some areas coalesced into vesicles of up to 1 cm in diameter (**Figure 1A**). **Figure 1B** shows the patient's back 3 days after admission. Scattered erosions were observed over less than 10% of the body surface, including the distal extremities, lips (**Figure 1C**), buccal mucosa (**Figure 1D**), and genital area. Mild, corneal erosions were observed, and the conjunctivae displayed marked pseudomembrane formation (**Figure 1E**). There was no loss of visual acuity, visual field impairment, or ocular pain.

The patient had no significant medical history, regular medications, or known allergies. After the onset of the rash, he received acetaminophen, loxoprofen, and rebamipide. Laboratory tests revealed a C-reactive protein level of 28.4 mg/dL (reference range: $<$ 0.14 mg/dL), a normal white blood cell count, and unremarkable hematological, biochemical, coagulation, and infectious disease test findings (**Table 1**). Autoimmune tests for anti-desmoglein 1, anti-desmoglein 3, and anti-BP180 antibodies were negative. Urinalysis initially indicated pyuria. Blood and urine cultures returned negative, and chest radiography revealed no abnormalities.

A biopsy of a pustule on the back was performed 3 days after the start of prednisolone therapy because of an intervening holiday. Hematoxylin and eosin staining revealed more than 10 necrotic keratinocytes per × 200 field, neutrophilic and lymphocytic infiltration at the dermo-epidermal junction, and mild vacuolar changes, which led to the diagnosis of SJS (**Figure 2**). The TEN-specific severity illness score, known as SCORTEN, was zero, indicating a 3.2% mortality risk [2]. Histopathology of the pustular areas demonstrated serous exudate with scattered neutrophils, which differed from the features of typical pustules (**Figure 3**).

Oral prednisolone 60 mg/day was initiated on admission. The C-reactive protein level decreased rapidly, the fever subsided, and the patient gradually regained the ability to eat. The prednisolone dosage was tapered, and as there was no clinical deterioration at a dosage of 30 mg/day, the patient was discharged on day 19. Thereafter, the prednisolone dosage was further reduced, and when no exacerbation was observed at 20 mg/day, the therapy was discontinued on day 25. The dysuria and pyuria gradually improved with treatment, suggesting that these findings were attributable to SJS-related urethritis. A drug-induced lymphocyte stimulation test (DLST) was positive for loxoprofen. After the patient was informed of the test results, he stated that he recalled having taken loxoprofen prior to the onset of the rash.

Discussion

No standard diagnostic criteria for SJS exist, and the Japanese Dermatological Association's criteria (**Table 2**) and the Niigata criteria do not make allowances for the possibility of drug-related causation [3,4]. Although no drug exposure or infection preceding the symptoms was identified at admission, the presence of fever, widespread erosions, pustules, vesicles, and mucosal involvement corroborated the diagnosis of SJS and helped rule out the following alternative diagnoses. Erythema multiforme was considered unlikely because the patient lacked the characteristic acral-predominant, round, erythematous papules and multiple, dark-red target lesions. A drug reaction with eosinophilia and systemic symptoms (DRESS) was considered unlikely because of the absence of facial edema, lymphadenopathy, and hematological abnormalities, as well as the lack of exposure to drugs commonly implicated in DRESS, such as carbamazepine. Although the mucosal involvement was compatible with Behçet disease, the pustules in this case were not

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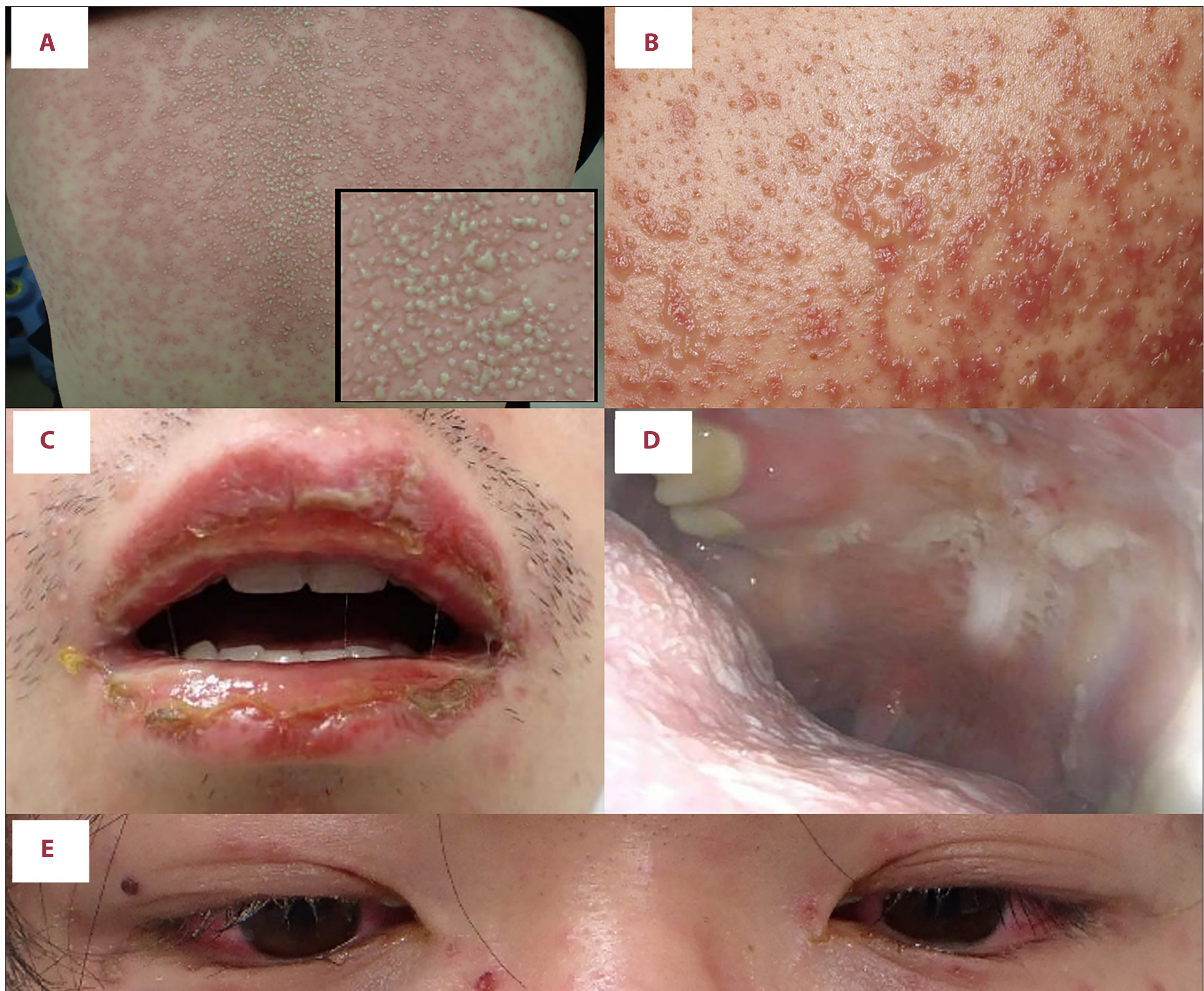

Figure 1. (**A**) Multiple pustules on the chest and back. (**B**) Blisters on the back photographed 3 days after admission. Some of the pustules had partially metamorphosed into flaccid blisters with erosion. (**C**) Scattered erosions on the lips. (**D**) Scattered erosions on the buccal mucosa. (**E**) Hyperemia and pseudomembrane formation on the bilateral conjunctivae. The patient gave his permission for the use of the photographs in this report.

folliculocentric, and no folliculitis-like lesions were observed. Reactive infectious mucocutaneous eruption, which typically follow infections, such as those by *Mycoplasma pneumoniae*, was considered unlikely because of the absence of upper respiratory symptoms and clinical findings suggestive of a respiratory infection. Pustular psoriasis was considered unlikely because there was no personal or family history of psoriasis, no psoriatic findings on a physical examination or histopathological analysis, and rapid improvement of the lesions over a short period. Although an overlap with acute generalized exanthematous pustulosis (AGEP) was considered, the scattered, 3-mm pustules on the erosive lesions differed from the characteristic, pinhead-sized pustules found in large numbers in edematous erythema associated with AGEP. The pustules, erythema, and the distribution of these features were incompatible with AGEP. Moreover, there was no neutrophilia (neutrophils >7000/μL). The patient had a score of 2 on the EuroSCAR scoring system [5]. The onset, which involved a fever and mucosal symptoms, was acute, occurring within 10 days of exposure to loxoprofen, and the condition resolved within 15 days. However, there was no increase in neutrophils and polymorphonuculear leukocytes, and the histology was atypical of AGEP, making the latter diagnosis unlikely. Based on these considerations and the clinical findings, SJS was diagnosed even though the underlying cause was unknown. No alternative diagnosis adequately explained the pustular lesions, which were therefore considered to be a symptom of SJS by exclusion. While 90% of SJS cases are drug-induced, infections by pathogens, such as *Mycoplasma pneumoniae*, and chemical exposures have also been identified as potential causes [6,7]. Although the ALDEN algorithm is commonly used to identify causative drugs, information bias and other factors account

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Table 1. Laboratory data on admission.

	Reference range	Results		Reference range	Results
CBC			**Immunology**		
WBC (/μL)	3300-8600	6,200	Anti-desmoglein 1 antibody	–	–
Neutrophils (%)	38.5-80.5	68	Anti-desmoglein 3 antibody	–	–
Lymphocytes (%)	16.5-49.5	18	Anti-BP180 antibody	–	–
Monocytes (%)	2.0-10.0	12	EBV IgM	< × 10	× 10
Eosinophils (%)	0.0-8.5	2	EBV IgG	< × 10	× 40
Basophils (%)	0.0-2.5	0	CMV IgM (INDEX)	< 0.85	0.83
RBC (μL)	4.35-5.55	4.90×10^6	CMV IgG (AU/mL)	< 6.0	51.3
Hb (g/dL)	13.7-16.8	14.2	HBV	–	–
Hct (%)	40.7-50.1	43.2	HCV	–	–
PLT (/μL)	15.8-34.8	15.2×10^4	HIV	–	–
			Syphilis	–	–
Chemistry			Mycoplasma (CF)	< × 4	< × 4
TP (g/dL)	6.6-8.1	6.8	Covid antigen	–	–
Alb (g/dL)	4.1-5.1	3.8	Influenza antigen	–	–
Na (mmol/L)	138-145	140	Urine chlamydia PCR	–	–
K (mmol/L)	3.6-4.8	4.2	Urine gonorrhea PCR	–	–
Cl (mmol/L)	100-108	100			
Ca (mg/dL)	8.8-10.1	9.3	**Urinalysis**		
BUN (mg/dL)	8.0-20.0	10.5	Dipstick urine test		
Cr (mg/dL)	0.65-1.07	0.92	Protein	–	1+
T-bil (mg/dL)	0.4-1.5	0.9	Hematuria	–	-
AST (U/L)	13-30	23	Ketone bodies	–	3+
ALT (U/L)	10-42	56	WBC	–	3+
ALP (U/L)	38-113	60	Nitrite	–	–
CK (U/L)	59-248	109	Sediment		
LDH (U/L)	124-222	240	WBC (/HPF)	< 5	> 100
CRP (mg/dL)	< 0.14	28.35	Bacteriuria	–	–

Abbreviations: CBC, complete blood count; WBC, white blood cell; RBC, red blood cell; Hb, hemoglobin; Hct, hematocrit; PLT, platelet; TP, total protein; Alb, albumin; Na, sodium; K, potassium; Cl, chlorine; Ca, calcium; BUN, blood urea nitrogen; Cr, creatine; T-bil, total bilirubin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; CK, creatine kinase; LDH, lactate dehydrogenase; CRP, C-reactive protein; Glu, glucose; EBV, Epstein-Barr virus; CMV, cytomegalovirus; HBV, hepatitis B virus; HCV, hepatitis C virus; HIV, human immunodeficiency virus; CF, complement fixation; PCR, polymerase chain reaction; HPF, high power field.

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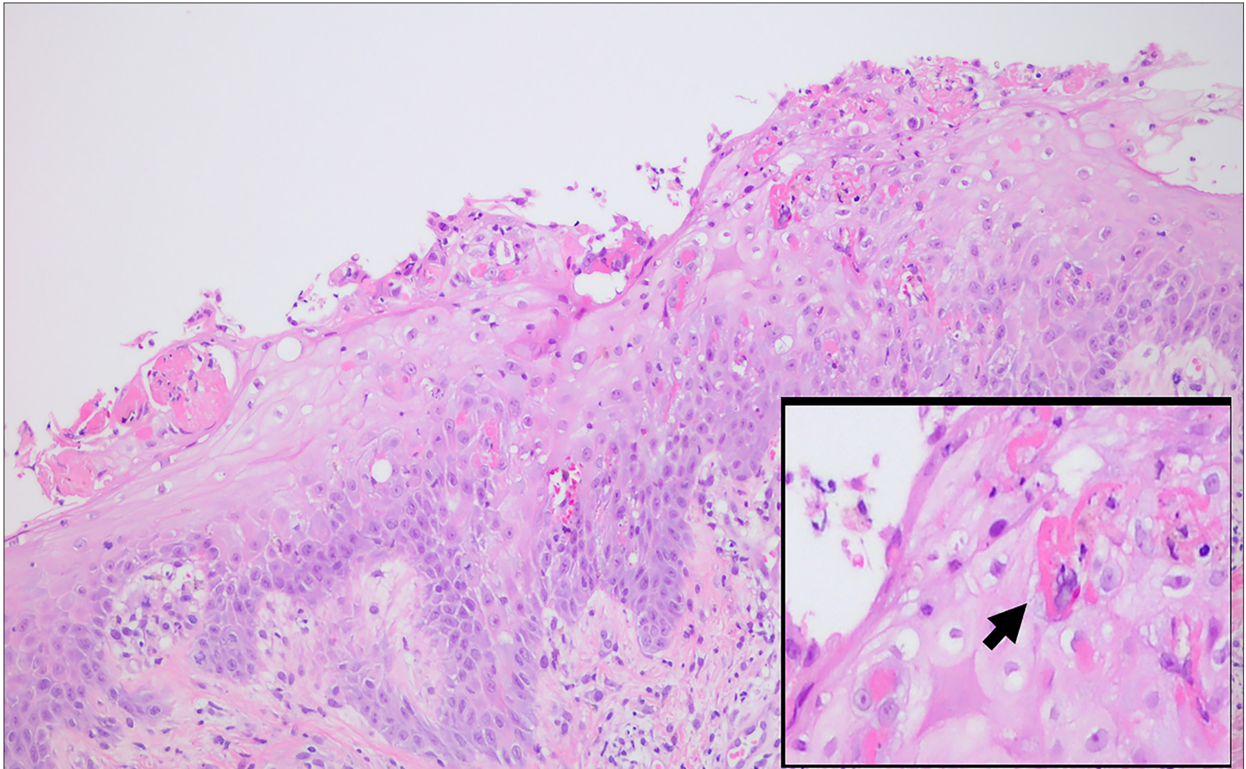

Figure 2. More than 10 necrotic keratinocytes per field were identified in the epidermis (black arrows). Hematoxylin and eosin stain, original magnification × 200.

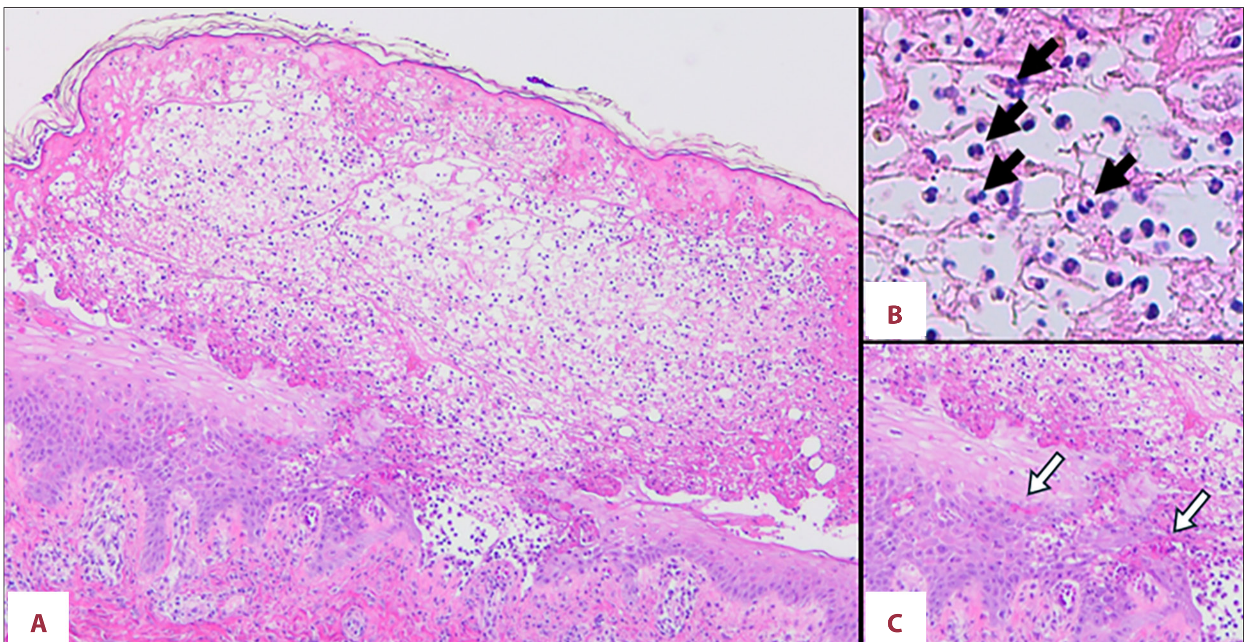

Figure 3. (**A**) The epidermis demonstrated necrosis with marked edema extending from the epidermis into the upper dermis resulting in blister formation (Hematoxylin and eosin stain, × 200). (**B**) Neutrophilic infiltration (black arrows) was present within the blister cavity (× 400). (**C**) Necrotic keratinocytes (white arrows) were also observed in the epidermis (× 400).

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Table 2. Japanese Dermatological Association's diagnostic criteria for Stevens-Johnson syndrome.

Major criteria	
1	Extensive, severe mucosal lesions, such as hemorrhagic erosions with crusting, are present at mucocutaneous junctions, including the eyes, lips, and genital area
2	Generalized erythema of the skin is accompanied by erosions and blisters resulting from necrotic epidermal injury and is followed by crusting and membranous desquamation during recovery. The total area of epidermal detachment involves less than 10% of the body surface area; however, areas in which the epidermis is expected to detach easily with minimal mechanical stress are included in this estimate
3	Fever is present
4	Histopathological examination reveals necrotic changes of the epidermis*
5	Erythema multiforme [EM] major can be excluded**
Minor criteria	
1	The erythema is generalized and predominantly involves the face, neck, and trunk. Non-elevated, flat, atypical target lesions with a dark red center having a tendency to coalesce are observed
2	Mucosal lesions are present at the mucocutaneous junctions. Ocular involvement manifests as bilateral, acute conjunctivitis accompanied by pseudomembrane formation and/or epithelial defects of the ocular surface
3	Systemic symptoms include an objectively severe, clinical condition and subjectively reported fatigue. Oral pain and sore throat cause varying degrees of difficulty with oral intake
4	Autoimmune bullous diseases can be excluded
Diagnosis	SJS is diagnosed when all five major criteria are met after considering the minor findings and assessing the entire clinical course
Supplements	
1	Differentiation from severe erythema multiforme should be made comprehensively by considering the five major criteria (1-5) together with the severity of systemic symptoms and fatigue, response to treatment, and the extent of necrotic epidermal changes on histopathological examination
2	* Pathologically, full-thickness, epidermal necrosis may be present; however, confirmation of at least 10 necrotic keratinocytes per × 200 high-power field is recommended
3	** EM major refers to erythema multiforme accompanied by relatively mild, mucosal involvement. The skin lesions are predominantly distributed on the extremities, and although fever is often present as a systemic symptom, the overall severity of the disease is limited. EM major is distinct from SJS
4	Stevens-Johnson syndrome can present with mucosal involvement alone

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for 15% of cases remaining undiagnosed at the initial evaluation [8]. As a drug eruption, SJS can be fatal but is clinically diagnosable on the basis of its characteristic features even in the absence of an identifiable trigger.

In the present case, a DLST was positive for loxoprofen (stimulation index: 2.27; reference range < 1.8). Given the frequent involvement of loxoprofen in SJS, the DLST was performed to determine if the drug was the cause. DLST using the radioactive uptake method has a sensitivity of 62% and specificity of 91%, with the positivity rate declining from 1 week after onset [9,10]. In this case, the DLST was performed after prednisolone therapy was discontinued. Although the test was conducted 2 weeks after disease onset, it yielded a positive result. In the absence of any other plausible etiology, loxoprofen was considered the most likely cause.

One potential form of information bias is the exclusion of a drug as the causative agent because it had been previously used without adverse effects, as in the present case. According to the ALDEN algorithm, the typical latency period for SJS ranges from 5 to 28 days. However, in practice, the latency period varies among individuals, with some cases developing as early as 1 to 2 days after exposure [11,12] while other cases occur after the long-term use of medications that had previously been well tolerated [13,14]. In the latter instance, previous

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exposure may prime a drug-specific T-cell response, and subsequent re-exposure may precipitate disease onset. Host-related and environmental factors may further lower the threshold for immune activation [15]. Even when the timing of exposure is uncertain, a DLST may be worth performing in patients with suspected SJS who have a history of exposure to drugs commonly associated with the condition.

A search of the PubMed, Embase, Cochrane, and Ichushi databases identified only 3 reports of SJS presenting with pustular lesions [16-18], all of which described cases related to reactive infectious mucocutaneous eruption or a subcorneal, pustular pattern resembling AGEP. However, none of the reports addressed the underlying mechanism of pustule formation. In the present case, histopathological analysis found that the neutrophilic accumulation was insufficient for true pustules despite the macroscopically pustular appearance of the lesions. Instead, the findings were characterized by marked edema beneath a necrotic epidermis with neutrophilic infiltration. Because the biopsy was performed 3 days after the start of prednisolone therapy, the histopathology of the condition, particularly of the pustular contents, may have undergone some changes. Nevertheless, it is noteworthy that despite the clinically evident pustular appearance, neutrophilic infiltration was not prominent. Given the irregular, non-follicular morphology of the lesions, a primary pustular disorder was considered unlikely. Rather, the findings suggested a secondary, pustular process resulting from neutrophilic infiltration into blisters formed by progressive, epidermal necrosis during the early, highly inflammatory phase of the disease. Thus, clinicians should be aware that SJS can occasionally present with pustule-like eruptions.

Conclusions

Although our patient did not have a history of drug exposure or infection, SJS was diagnosed on the basis of the clinical presentation. SJS should always be included in the differential diagnosis, particularly in patients presenting with a fever and multiple erosions at mucocutaneous junctions and the skin, and it should be carefully distinguished from various infections and drug eruptions. Histopathological analysis of a skin biopsy specimen is essential for definitive diagnosis and for distinguishing SJS from other conditions. While pustules are atypical of SJS, blister formation can be followed by secondary changes producing a pustule-like appearance.

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Institution Where Work Was Done

Tokyo Metropolitan Tama Medical Center, Tokyo, Japan.

Consent

Appropriate written consent has been obtained from the patient.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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