

**STAPHYLOCOCCAL SCALDED SKIN SYNDROME
MIMICKING TOXIC EPIDERMAL NECROLYSIS IN A
HEALTHY ADULT**Tomoko Oishi, Yuka Hanami, Yasunobu Kato, Mikio Otsuka,
Toshiyuki Yamamoto*Department of Dermatology, Fukushima Medical University, Hikarigaoka 1,
Fukushima 960-1295, Japan*

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Corresponding author: Prof. Toshiyuki Yamamoto

toyamade@fmu.ac.jp

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Abstract

Introduction: Staphylococcal scalded skin syndrome (SSSS) presents generalized form bullous impetigo caused by *Staphylococcus aureus* (*S. aureus*) infection, typically seen in infants and children. SSSS may occur also in adults; however, the majority of adult cases are those with immunosuppression. Atypical clinical features of impetigo in adults sometimes make it difficult to diagnose correctly.

Case Report: A 74-year-old healthy woman was hospitalized, complaining of extensive desquamative erythema and a number of erosions. She was administered oral antiviral drugs under suspicion of herpes zoster prior to 10 days. Initial diagnosis on the admission was toxic epidermal necrolysis (TEN) due to antiviral tablets; however, steroid pulse therapy resulted in no effect. Bacterial culture yielded coagulase-positive methicillin-resistant *S. aureus*, producing exfoliative toxin B. A biopsy specimen showed subcorneal splitting of the epidermis. The diffuse erosions gradually improved over 10 days by the treatment with intravenous antibiotics.

Conclusions: The differentiation between streptococcal scalded skin syndrome (SSSS) and TEN is sometimes difficult. It is important to remind SSSS when we suspect TEN, even in healthy adults.

Key words: SSSS; TEN; MRSA; adult**Cite this article:**

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Introduction

Streptococcal scalded skin syndrome (SSSS) presents generalized form bullous impetigo caused by *Staphylococcus aureus* (*S. aureus*) infection, typically seen in infants and children. SSSS may occur also in adults; however, the majority of adult cases are those with immunosuppression, overwhelming sepsis, kidney failure, or under immunosuppressive conditions. We herein report a case of SSSS due to methicillin-resistant *Staphylococcus aureus* (MRSA) in a healthy adult, which mimicked the clinical feature of toxic epidermal necrolysis (TEN).

Case Report

A 74-year-old woman initially visited a practical clinic, complaining of several bullae on the left hip. She was administered oral antiviral drugs under a suspicion of herpes zoster. However, erythema and erosions appeared and spread rapidly all over the body. She visited an emergency room of another hospital 10 days later. Then, she was prescribed oral prednisolone (30mg daily) under a diagnosis of drug eruption, and referred to our

hospital on the next day. On physical examination, there were broad erosions around her left hip and lower abdomen with a background of systematic erythema including her face (Fig. 1). Bulbar conjunctival hyperemia was seen. Nikolsky sign was positive on her back skin. She had no special past health histories and there were no significant laboratory findings expect for a slight degree of hypoproteinemia (TP 6.2g/dl, Alb 3.2g/dl) and inflammatory reaction (CRP 2.51mg/dl). Under suspicion of TEN due to antivirus tablets, we started a steroid pulse therapy with gamma globulin, which however resulted in no effects. Histological examination revealed splitting at the level of the granular cell layer (Fig. 2), without inflammation in the dermis. Bacterial cultures from skin erosion yielded coagulase-positive MRSA, producing exfoliative toxin B. Neither toxic shock syndrome toxin-1 (TSST-1) nor enterotoxin was detected. The diagnosis of SSSS was made, and antibiotics (teicoplanin (Targocid®); 200 mg/day for 5 days followed 400mg/day for 4 days) were administered. Because she developed drug eruption, antibiotics were changes to vancomycin (1g/day for 2 days). All eroded surface epithelized in 2 weeks.



Figure 1. Extensive desquamative erythema, erosions, and scales on the trunk

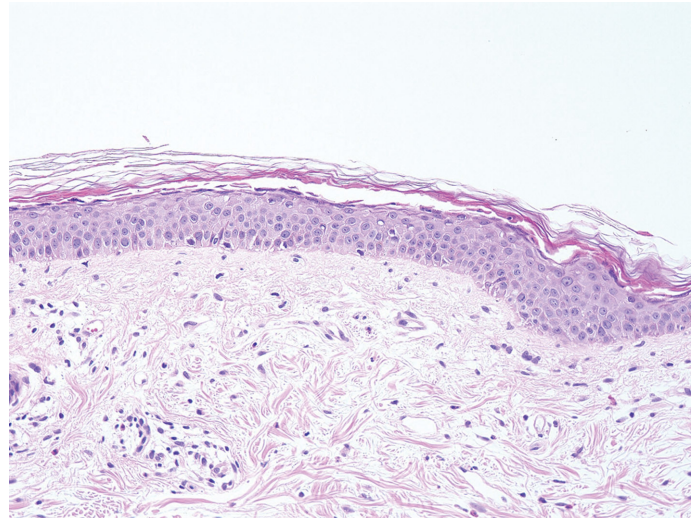


Figure 2. Histopathology showing cleavage in the upper epidermis. (H&E stain, X200)

Discussion

SSSS is an extensive exfoliative dermatitis caused by *S. aureus* infection. The blisters in SSSS is caused by exfoliative toxin (ET) released by *S. aureus*, occasionally by MRSA [1,2]. *S. aureus* infection results in a loss of keratinocyte cell-cell adhesion through desmoglein-1, leading to blister formation [3]. SSSS usually occurs in children, and is rarely seen in healthy adults [4-6]. SSSS in adults frequently occur in association with kidney failure, malignancy, and immunosuppression [7]. Although our case was an elderly female, she did not either present a prior condition for SSSS such as burn, wounds, or had diabetes, renal failure, or other immunosuppressive conditions. Our case presented diffuse erosive erythema with Nikolsky sign, following intake of antiviral drugs under a misdiagnosis of herpes zoster. Therefore we initially suspected TEN because the patient was healthy adult and had no significant past health history. However, a steroid therapy resulted in no effect and histology examination also denied TEN. Differentiation between TEN and SSSS is sometimes difficult. Nikolsky sign is not specific for TEN. Examination by histological examination by immediate cryosections, Tzanck test, and blister roof histology may be useful as rapid tools. In SSSS, blister roof histology shows that the epidermal cleavage is within the stratum granulosum [8].

Conclusion

It is important to remind SSSS in cases suspecting TEN even in healthy adults, because the treatment for both diseases is different and SSSS is still associated with mortality.

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