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Case report

SSSS in Pediatric Patients: Is Early Recognition Always Straightforward?

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ABSTRACT

Staphylococcal Scalded Skin Syndrome (SSSS) is a rare bullous dermatosis caused by staphylococcal exfoliative toxins, predominantly affecting children under six years of age. A six-year-old boy developed intense erythema and facial edema while camping, following recent impetigo. The initial presentation included peri-orificial erythema, hyperemic lips, and conjunctivitis without fever. Within 48 hours, desquamation, bullae, and a positive Nikolsky sign developed. Treatment with clindamycin resulted in rapid clinical improvement. The camping setting initially suggested sunburn; however, the characteristic peri-orificial distribution and positive Nikolsky sign confirmed the diagnosis of SSSS. This case highlights the importance of considering SSSS in pediatric bullous eruptions following staphylococcal infections. Although rare, SSSS requires prompt recognition and appropriate antibiotic therapy. Clinicians should maintain high suspicion for SSSS in pediatric patients with acute bullous lesions, even when clinical settings suggest alternative diagnoses.

1. Introduction

Staphylococcal Scalded Skin Syndrome (SSSS) represents a serious skin disorder triggered by specific *Staphylococcus aureus* strains that release exfoliative toxins into the bloodstream (1). The overall incidence of SSSS in the general population is estimated to range between 0.09 and 0.56 cases per one million people (2). In the United States, the annual incidence is 7.67 (range 1.83–11.88) per one million children, with 45.1 cases per one million infants under two years of age (3). However, the highest incidence is observed in infants aged two to three years (4).

The disease predominantly affects pediatric populations and individuals with compromised immune systems. It is characterized by extensive superficial skin peeling and erythema that can resemble other critical dermatological emergencies such as Stevens–Johnson syndrome or toxic epidermal necrolysis (5). We present this case because recent epidemiological data suggest an increasing prevalence of SSSS, highlighting the need for greater clinical awareness to facilitate prompt recognition and management.

2. Case report

A six-year-old boy on vacation at a campsite with his parents came to our attention due to the onset of burning and pruritic erythema. For several days, he had been treated with topical antibiotic therapy for impetigo. He had no fever, had not taken any medications, and had no known allergies.

The child appeared distressed and complained of burning pain and intense itching while maintaining good general condition. The erythema was markedly intense, involving the face, neck, and trunk. The lips were hyperemic, the face appeared edematous, and purulent conjunctivitis was present. The remaining physical examination was normal. A rapid strep test was negative.

After the administration of an antihistamine with minimal benefit, and suspecting a bacterial etiology, oral amoxicillin–clavulanic acid (50 mg/kg/day) was started. However, it was discontinued after one day when the clinical picture became consistent with SSSS. Blood chemistry tests showed mild neutrophilic leukocytosis and negative inflammatory markers (CRP). The nasopharyngeal swab for viruses and common bacteria was negative.

Within the following 48 hours, the erythema began to desquamate (Fig. 1), and bullae and vesicles started to appear, which rapidly ruptured (Fig. 2). Peri-oral fissures worsened. Nikolsky's sign was positive. With a strong suspicion of bullous dermatitis caused by epidermolytic exotoxins (Staphylococcal Scalded Skin Syndrome - SSSS), intravenous clindamycin therapy was started at a dose of 40 mg/kg/day, divided into three doses, resulting in rapid improvement of the clinical picture. After four days, the therapy was switched to the oral route as the patient was returning to his home country. The skin swab for MRSA was negative.

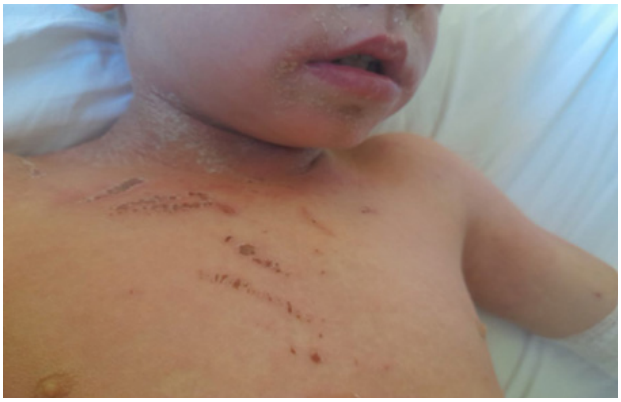

Fig. 1. *Generalized erythroderma with superficial desquamation of the neck and peri-labial area where honey-crusted lesions are visible. Ruptured bullae leave superficial erosions with brown crusts.*

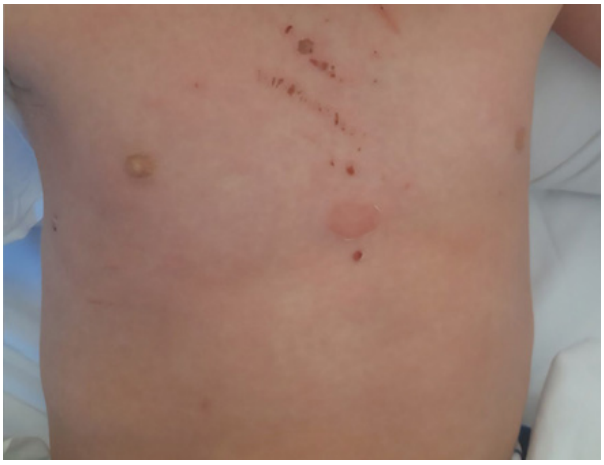

Fig. 2. *Ruptured bullous lesion over the sternum with exposed denuded skin.*

3. Discussion

SSSS is a bullous dermatosis caused by exfoliative exotoxins (ETA and ETB) produced by certain strains of *Staphylococcus aureus* (5). It predominantly affects neonates and children under six years of age (4–5), often occurring after impetigo, pharyngotonsillitis, or conjunctivitis. The onset is typically acute, with cutaneous pain as a characteristic feature.

Erythema, intensely hyperemic, initially develops in skin folds and the peri-orificial region. Subsequently, flaccid bullae and widespread desquamation appear, accompanied by a positive Nikolsky sign (gentle pressure causes detachment of the upper epidermal layers). Facial edema is frequent, and radial peri-oral fissures are typical. Fever, lethargy, irritability, and reduced oral intake may also occur.

The diagnosis is essentially clinical, as laboratory tests are not definitive. Culture swabs from the suspected source of infection may help confirm the presence of *S. aureus* and guide targeted antibiotic therapy based on antibiogram results. Differential diagnoses include Stevens–Johnson syndrome (usually with mucosal involvement), toxic epidermal necrolysis (often following drug exposure), and scarlatiniform rash (typically with pharyngitis and a positive strep test). Treatment is primarily antibiotic (anti-staphylococcal) and supportive when necessary (6).

This case exemplifies a classic presentation of SSSS in a pediatric patient. The six-year-old patient presented with the characteristic triad of burning erythema, facial edema, and peri-oral involvement, following a preceding impetigo infection—a well-recognized trigger for SSSS development. The early phase demonstrated pathognomonic signs: intense erythema with peri-orificial distribution, hyperemic lips, and facial edema consistent with exotoxin-mediated epidermal damage. The absence of fever and maintenance of good general condition align with the typical presentation of SSSS, distinguishing it from more systemic conditions.

The diagnostic approach illustrates both the challenges and typical laboratory patterns associated with SSSS. The negative rapid strep test appropriately ruled out Group A Streptococcal (GAS) involvement, while mild neutrophilic leukocytosis with negative inflammatory markers (CRP) reflects the localized nature of toxin production rather than systemic bacterial invasion. The evolution to desquamation, bullae formation, and positive Nikolsky sign within 48 hours provided definitive clinical confirmation. This temporal evolution is characteristic of SSSS, where exfoliative toxins (ETA and ETB) cause loss of cell-to-cell adhesion in the granular layer of the epidermis, resulting in superficial blistering and subsequent desquamation (7).

The clinical presentation required careful consideration of several conditions within the differential diagnosis. Initially, given the camping setting and intense erythema, sunburn or solar erythema could have been considered; however, the characteristic peri-orificial distribution, facial edema, and associated pain pattern were inconsistent with typical sun exposure injury. Stevens–Johnson syndrome was appropriately excluded given the absence of mucosal involvement. Toxic epidermal necrolysis was less likely given the patient's age and absence of drug exposure history. Negative strep testing helped differentiate from scarlatiniform eruptions, which typically present with pharyngitis and positive GAS results.

The initial empirical therapy with amoxicillin–clavulanate represented a reasonable broad-spectrum approach for a suspected bacterial etiology. However, the subsequent switch to clindamycin demonstrated appropriate clinical reasoning, as clindamycin provides superior anti-staphylococcal coverage and has the additional benefit of inhibiting bacterial protein synthesis, thereby reducing toxin production (6, 8, 9). The rapid clinical improvement following clindamycin initiation supports the diagnosis and highlights the importance of prompt, appropriate antibiotic therapy in SSSS management. The negative MRSA culture confirmed that this case involved methicillin-sensitive *Staphylococcus aureus*, making clindamycin an optimal therapeutic choice (6-10). In Italy, the MRSA rate stood at 26.6% in 2023, with clindamycin resistance accounting for 34.7% (11).

This case underscores several important clinical principles in SSSS management. Early recognition based on characteristic clinical features—particularly the positive Nikolsky sign and typical distribution pattern—enables prompt therapeutic intervention (12). The excellent response to targeted antibiotic therapy demonstrates the generally favorable prognosis of SSSS when appropriately managed, particularly in immunocompetent pediatric patients. The case also highlights the importance of considering SSSS in children presenting with bullous eruptions following superficial staphylococcal infections, emphasizing close monitoring of even minor skin infections in the pediatric population.

4. Conclusion

Although Staphylococcal Scalded Skin Syndrome remains a relatively uncommon condition, this case highlights the critical importance of including SSSS in the differential diagnosis of pediatric patients presenting with acute erythematous and bullous skin lesions. Early recognition of characteristic clinical features, particularly following antecedent staphylococcal infections, enables prompt initiation of appropriate therapy and prevents potential complications associated with delayed diagnosis. Clinicians should maintain a high index of suspicion for SSSS in the appropriate clinical context, as timely intervention significantly impacts patient outcomes and prevents progression to more severe manifestations of this toxin-mediated dermatologic emergency

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