Staphylococcal Scalded Skin Syndrome

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Background

Staphylococcus aureus is one of the most common etiologies of bacterial skin infections and may present in a variety of ways. One of the most severe manifestations is staphylococcal scalded skin syndrome (SSSS), an exotoxin-mediated disease characterized by diffuse bullae formation [1]. The incidence is estimated to be about 0.09–0.56 cases per million inhabitants in the general population [2] and has been shown to have higher incidence in summer and fall seasons [3, 4]. Historically, it has also been called Ritter's disease and pemphigus neonatorum [5]. The toxins responsible are exfoliative toxins A and B (ETA, ETB), produced by coagulase-positive *S. aureus* [2]. ETA is more common (89% of cases) than ETB, although ETB is much more virulent [2].

Initial site of infection occurs most commonly in sites of colonization, such as the nasopharynx or conjunctiva. The exotoxin produced by *S. aureus* then enters into the bloodstream and circulates, spreading diffusely to the skin. The exfoliative toxins accumulate in the skin tissue and cause breakdown of desmoglein 1 (Dsg1), which is essential to keratinocyte cell-to-cell adhesion within the superficial layers of the epidermis [2, 6, 7]. This leads to the formation of diffuse bullae and subsequent desquamation. The blisters are typically sterile as the skin findings are toxin-mediated rather than secondary to direct infection. Symptoms will continue to progress until the exotoxin has either been bound by antibodies or cleared by the kidneys. This generally lasts 24–48 h, depending on the immune status and renal function of the infected patient [2].

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Although SSSS affects patients of all ages, it is most common in the pediatric population, especially in those under the age of 5 [2–4, 7, 8]. An immature renal system unable to clear the exfoliative toxin and a lack of protective antibodies against staphylococcal toxins [2–4] likely contribute to the increased risk of infection in the young. Newborns are particularly at risk and represent about 80% of SSSS cases. This is particularly true in areas where umbilical cord care with antiseptics is discouraged [9]. Older kids and adults typically have preformed antibodies against the staphylococcal exotoxins, making the development of SSSS less likely. However, these populations are at risk for SSSS if they are immunosuppressed due to medications or disease or in the setting of renal failure [2, 8, 9].

Classic Clinical Presentation

The time course of disease is summarized in Table 10.1. Once the triggering staphylococcal infection has been established, SSSS findings will develop in the following few days [11]. Skin findings often begin as faint but well-demarcated patches that quickly coalesce into a diffuse erythema that is significantly tender to palpation [1, 2, 12] (Fig. 10.1). Diffuse erythema may be scarlatiniform in appearance with sandpaper feel to palpation [10]. Flaccid bullae develop in the following 24–48 h with a positive Nikolsky sign (skin desquamation with gentle pressure). Rupture of the bullae reveals underlying moist, red skin with the classic "scalded" appearance (Fig. 10.2). This erythematous base then rapidly dries to create a shiny surface. Areas with creases, such as the axillae, groin, nose, and ear, are often the most noticeable areas with desquamation that mimics the appearance of tissue paper [9, 12]. While perioral lesions are common, mucous membranes are not affected (Fig. 10.3) [8]. In the newborn, the diaper area is often the first to develop these signs (Fig. 10.4). These areas will typically dry within the following 24 h. Lesions will fully heal within 7–10 days without scarring.

Table 10.1 A timeline of skin findings

Initial	Hours	24–48 h	Days 3–5	10 days
Abrupt, faint, erythematous, tender patches	Patches become well demarcated and coalesce into a confluent scarlatiniform erythema	Flaccid bullae form within the erythematous areas, beginning on the central face, axillae, groin, and neck and spreading to form diffuse, large sheets, *sparing mucous membranes*. Rupture of bullae reveals a moist, red surface that appears scalded	Within 24 h of exfoliation, areas dry with a thin, shiny crust. Fissuring occurs in perioral and periorbital skin	Skin heals without scarring

Data from Pollack [1], Handler and Schwartz [2], and Marina et al. [10]

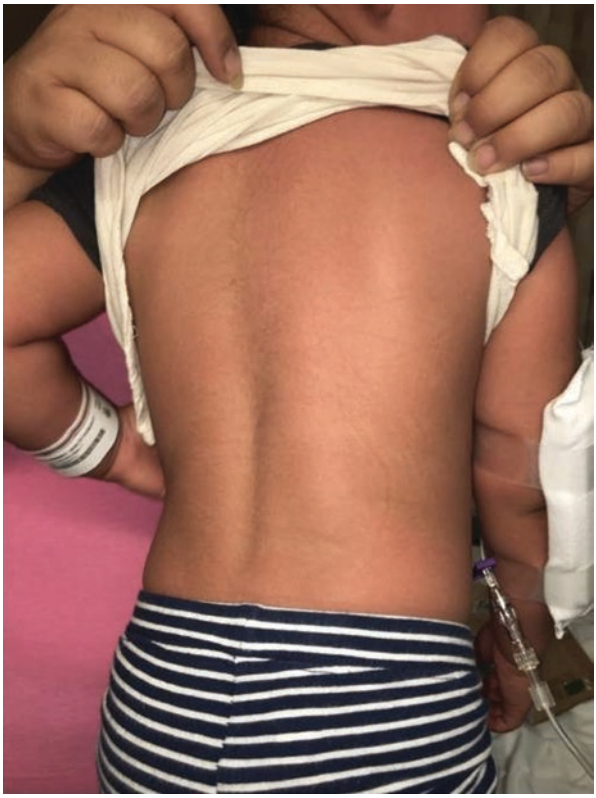

Fig. 10.1 A school-aged female with signs of early SSSS secondary to bullous impetigo. The skin developed diffuse erythema that was tender to palpation. (Patient consent obtained. Photo credit: Elizabeth Placzek, MD.)

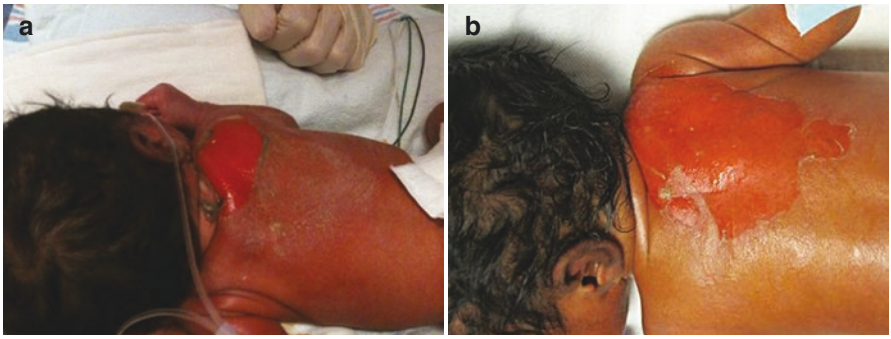

Fig. 10.2 (**a**) Sloughing of skin reveals moist, erythematous underlying skin that appears scalded. (**b**) Hours later. (From Arora et al. [13])

Atypical Presentation

The clinical presentation of SSSS may vary on a spectrum from mild to severe, depending on the strain of *S. aureus*, the amount of toxin, and the location of its release [10]. Mild cases, such as those seen in a report by Hubiche et al. [15], may

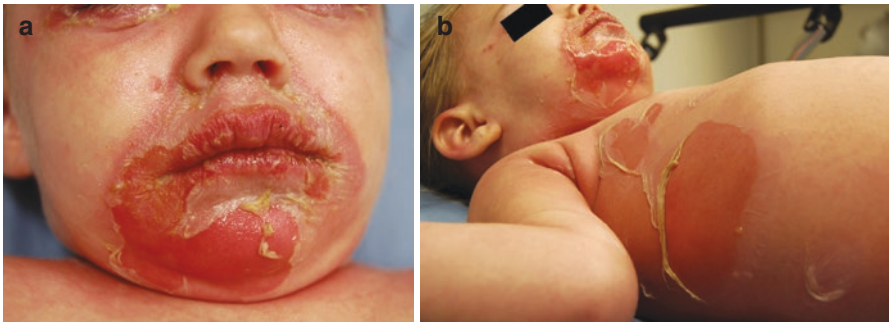

Fig. 10.3 (**a**) A school-age child with perioral SSSS skin lesions. (**b**) Significant truncal desquamation. (From Conway et al. [14])

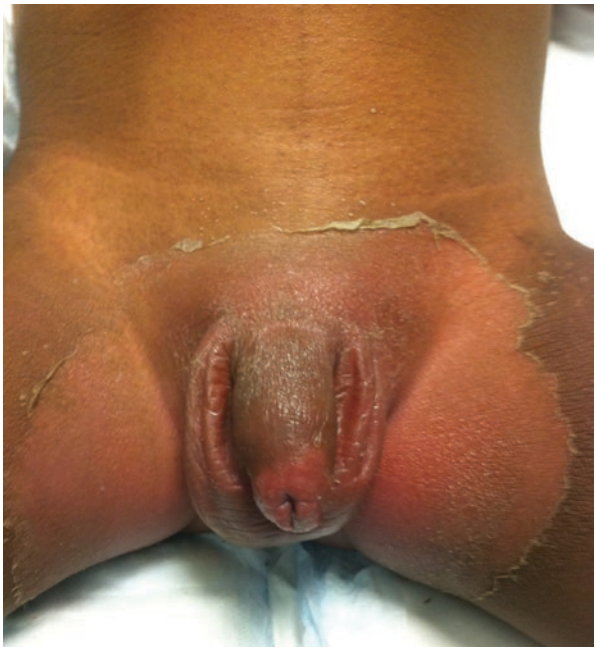

Fig. 10.4 An infant with inguinal desquamation due to SSSS. (Photo credit: Jennifer Ballinger, MD)

present with a mild, diffuse exanthem, followed by focal desquamation in the major skinfolds, with minimal or absent bullae formation. Additionally, some cases of SSSS may complicate other infections or conditions with cutaneous findings such as burns, graft versus host disease, and varicella (Fig. 10.5) [16–19].

Associated Systemic Symptoms

Pediatric patients often present with a short, non-specific prodrome of fever, irritability, and poor feeding, prior to onset of cutaneous manifestations [2]. Evidence of

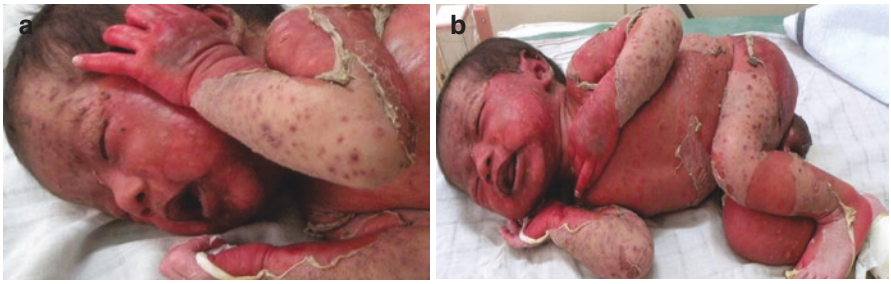

Fig. 10.5 (**a**) A neonate with varicella infection complicated by SSSS. (**b**) Irritable neonate with whole body involvement/desquamation. (From Singh et al. [16])

acute pharyngitis, conjunctivitis, or a mild upper respiratory infection have also been known to precede SSSS [11, 17]. It is suspected that upper respiratory tract infections, in particular, may alter the epithelium leading to proliferation of *S. aureus* in pediatric patients who are carriers of exfoliative toxin strains [17].

In adults, the source of infection is more varied and includes cellulitis or abscesses, septic arthritis, osteomyelitis, and endocarditis or as a complication of an invasive procedure or parenteral infection. Infection in adults is typically more severe at presentation and is commonly associated with bacteremia, shock, and a higher mortality rate. This is a stark contrast to the pediatric population, in which hypotension or signs of shock are rare on presentation and the prognosis is overall favorable with treatment [11, 20, 21].

Common Mimics and Differential Diagnosis

There are a number of rashes comparable to that of SSSS and are summarized in Table 10.2. Along the same spectrum as SSSS, is bullous impetigo, which is a milder, localized form. It most commonly affects neonates and presents with superficial bullae on the trunk and extremities (Fig. 10.6). The marked differences are the localized site, the absence of Nikolsky sign, and the presence of *S. aureus* within the bullous lesions. In few cases, bullous impetigo may progress to SSSS. For that reason, these patients should be followed closely, as the mortality of SSSS is significantly higher than the benign course of bullous impetigo [22, 23].

Other blistering conditions, such as Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), or pemphigus vulgaris, may resemble SSSS. The differentiating features of these conditions are summarized in Table 10.3.

It is important to differentiate SSSS from TEN as the treatments are markedly different. While corticosteroids may be utilized in the management of TEN, their use is contraindicated in cases of SSSS due to the potential for further immunosuppression and subsequent worsening of symptoms [11, 24]. History and physical are essential in making the diagnosis. A history of a recent new drug exposure may be suggestive of TEN [24]. There is also significant mucosal involvement in TEN, which is absent in SSSS. If there is a mixed clinical picture, frozen section histology

Table 10.2 Differential diagnosis of SSSS

Differential diagnosis
Toxic epidermal necrolysis
Erythema multiforme
Stevens-Johnsons syndrome
Pemphigus vulgaris
Pemphigus foliaceus
Toxic shock syndrome
Scarlet fever
Kawasaki disease
Coxsackie infection
Drug reaction with eosinophilia and systemic symptoms (DRESS)
Burn
Child abuse

Data from Patel and Finlay [11]

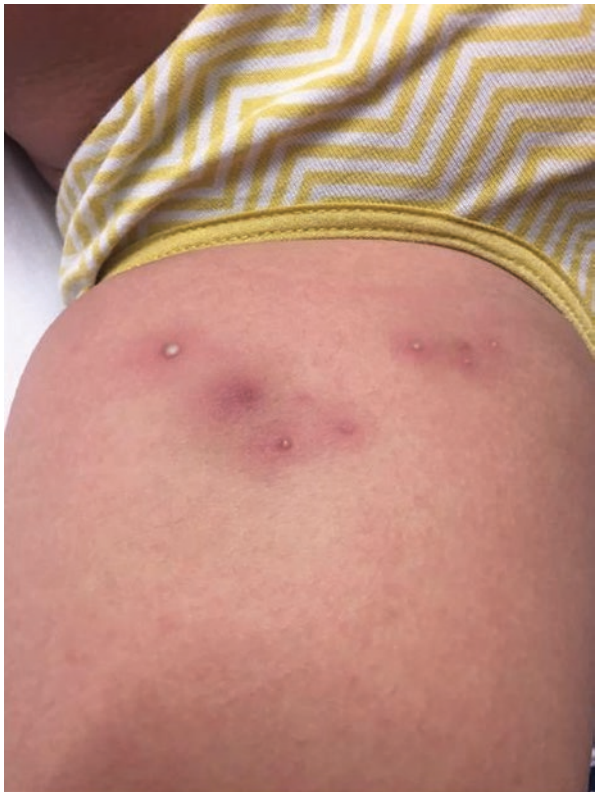

Fig. 10.6 A school-aged female with bullous impetigo. (Patient consent obtained. Photo credit: Elizabeth Placzek, MD)

of the roof of an acute blister or a sample of sloughed skin is often all that is needed to quickly differentiate the two in an acute setting [24, 25]. TEN will show subepidermal (deeper) cleavage with evidence of necrosis, while SSSS shows a superficial epidermal break without necrosis [25, 26].

Pemphigus vulgaris is an autoimmune condition that is similar in mechanism to SSSS, as it targets desmogleins. However, the antibodies are directed against

Table 10.3 Key differentiating features of top differential diagnoses of SSSS

Disease	Clinical features	Nikolsky sign	Mucous membrane involvement	Biopsy findings	Additional characteristics
SSSS	Diffuse erythema and bullae formation with underlying scalded skin	Positive	Absent	Intraepidermal cleavage along stratum granulosum. No inflammatory or necrotic cells	
Bullous impetigo	Localized cluster of flaccid bullae containing pus that may rupture and crust	Negative	Absent	Intraepidermal cleavage along stratum granulosum, dermal inflammatory infiltrates	
Toxic epidermal necrolysis (TEN)	Rapidly progressive rash with blistering and desquamation	Positive	Present	Subepidermal cleavage, dermal inflammatory infiltrates, and necrotic keratinocytes	Often result of a severe adverse drug reaction. More common in adult population
Pemphigus Vulgaris	Painful mucosal or mucosal and skin blistering	Positive	Present	Intraepidermal cleavage along stratum granulosum, dermal inflammatory infiltrates, and positive direct immunofluorescence	More common in adult population

desmoglein 3 (Dsg3), rather than Dsg1, which is present in deeper layers of the skin and is prominent in mucous membranes. Skin biopsy shows intraepithelial cleavage in the suprabasal layers and eosinophilic inflammatory infiltrates, which are absent in SSSS [8]. Pemphigus foliaceus, on the other hand, is clinically difficult to distinguish from SSSS, as it is also common in children and affects only Dsg1, making mucosal involvement absent. Often it is only differentiated by biopsy based on the presence of dermal inflammatory infiltrates and antibodies seen on direct immunofluorescence [5, 8, 9, 11, 26].

Diagnosis is based on characteristic skin findings, histopathology on skin biopsy, and identification of the inciting staphylococcal infection [27]. This may be done by taking cultures of the nose, throat, skin, and umbilicus, as these are common commensal sites of staphylococcal growth that have been known to cause SSSS [9, 12]. In the sicker patient, there may be a more obvious source of infection, such as pneumonia, septic arthritis, abscess, or endocarditis (Fig. 10.7) [29]. Bullae of SSSS are known to be sterile. Regardless, lesional swabs should be

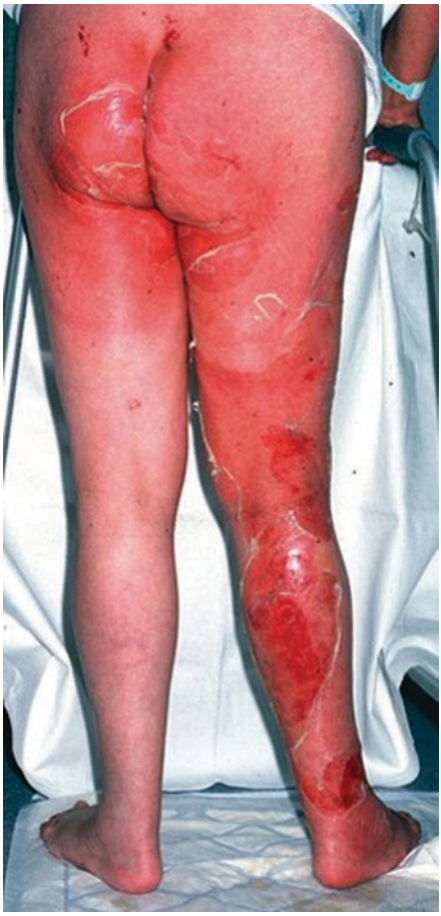

Fig. 10.7 A 63-year-old woman with SSSS affecting the buttocks and right lower extremity secondary to septic arthritis of the right knee. (From Sladden et al. [28])

obtained, as it may assist in identifying the presence of a secondary infection [29]. Blood cultures should also be obtained, though in most pediatric cases, they will remain negative [3, 29]. Blood cultures are much more likely to be positive in the adult population as there is generally a more severe infection and larger toxin burden due to immunosuppression [10, 29]. However, regardless of the patient population, a positive culture result is not required to make the diagnosis. It is also important to note that initial white blood cell counts and inflammatory markers, such as C-reactive protein (CRP), are often normal and should not provide false reassurance [30].

Though the diagnosis is typically clinical, definitive diagnosis may be made by skin biopsy, which reveals superficial intraepidermal cleavage within the stratum granulosum by acantholysis, with the roof of the bullae formed by the most superficial layer of the epidermis, the stratum corneum (Fig. 10.8). Remaining tissue will appear normal, without presence of inflammatory cells or signs of necrosis [2, 3, 12, 25, 31]. Frozen section of a blister roof may serve as rapid diagnostic tool; however, full-thickness biopsies should be obtained as well to confirm findings [24, 25, 27].

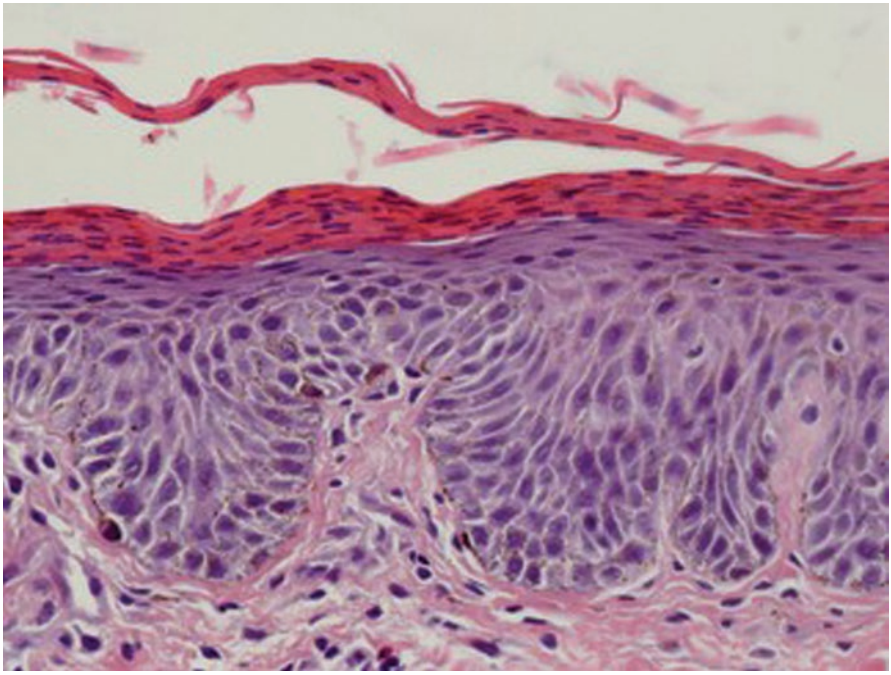

Fig. 10.8 Histology of SSSS showing superficial cleavage of the stratum granulosum. The thin layer of the stratum corneum forms the roof of the bulla (hematoxylin and eosin, original magnification ×400). (From Handler and Schwartz [2])

Recent development of more specialized testing, such as polymerase chain reaction (PCR), enzyme-linked immunosorbent assays (ELISA), and radioimmunoassays, now allows for the identification of the exfoliative toxin production in patients. However, these tests may take days to result, limiting their utility in the emergent setting. Additionally, they require sampling from the focus of infection, which is often unknown [5, 29].

Key Physical Exam Findings

Diffuse, scarlatiniform erythema with sandpaper feel to palpation and significant tenderness to palpation. Subsequent rapid development of flaccid bullae with a positive Nikolsky sign, that is most prominent in areas of skinfolds. Sloughing of the bullae reveals underlying eroded skin that is moist and brightly erythematous with a classic "scalded" appearance. There may be perioral involvement, but mucous membranes will remain unaffected [1, 2, 8–10, 12].

Management

Admission is recommended for all SSSS patients, especially in generalized cases [10]. As this condition leads to the loss of normal skin barrier, management should mimic that of a burn. Cases of severe and diffuse skin desquamation involving over 50% of body surface area should be transferred to a burn facility. Management is directed toward treating the causative staphylococcal infection and supportive care.

Parenteral anti-staphylococcal antibiotics should be administered promptly. First-line treatment is with a penicillinase-resistant synthetic penicillin, such as nafcillin or oxacillin [4]. Clindamycin may be beneficial due to its ability to inhibit bacterial toxin production [32]. However, studies of SSSS-causing bacteria have shown significant resistance rates against clindamycin, so it should not be used as monotherapy [4, 32]. Exfoliation may continue for 24–48 h despite antibiotics, until the toxin in circulation has been cleared. In an unstable, decompensating patient or if there is inadequate treatment response, vancomycin or linezolid should be given for methicillin-resistant *S. aureus* coverage [9, 29].

Supportive measures include intravenous fluids, analgesia, and nutritional support [2]. Warming methods should be utilized to preserve normal body temperature. The blisters should be left intact, if possible, to avoid further insensible losses and to minimize chance of superimposed infection on the naive skin [12]. Pressure-relieving mattresses may assist in reducing sloughing of the skin. Areas of denuded skin should be kept moist with emollients [8]. Fortunately, due to the superficiality of the condition, residual scarring is rare (Fig. 10.9) [29].

Appropriate analgesia should be provided, as the skin lesions are often painful. Acetaminophen is preferred over nonsteroidal anti-inflammatory drugs to avoid the

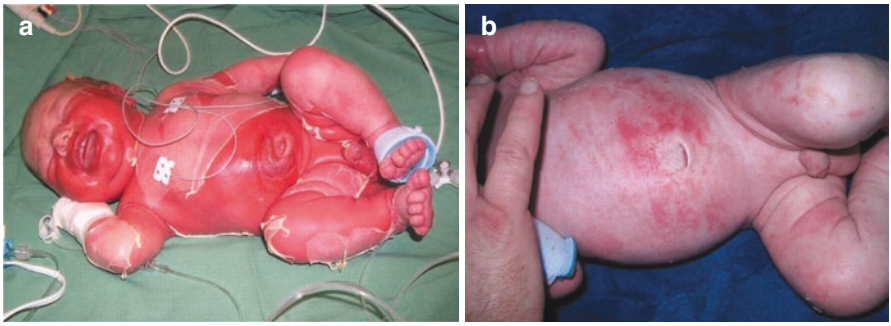

Fig. 10.9 A neonate with diffuse SSSS on day 1 of illness (**a**) versus day 7 of illness (**b**). (From Baartmans [33])

potential for renal injury and subsequent worsening of exfoliative toxin clearance [30, 34]. Patients with diffuse injury requiring regular dressing changes may require procedural sedation and/or opioids for analgesia [30].

Corticosteroids may cause worsening of SSSS and are not indicated. Treatment with fresh frozen plasma (FFP) and intravenous immunoglobulin (IVIG) have demonstrated some benefit and may be considered in refractory cases [35]. FFP is presumed to contain antibodies to the exotoxin, which could assist in curbing the exfoliative process. If no response to FFP is observed, IVIG may be considered to further attempt to neutralize circulating exotoxins [30]. Plasma exchange has also been successfully used in an adult patient who continued to have progression of skin lesions despite IVIG and ultimately improved after plasma exchange therapy [35].

Complications

The most common complications include electrolyte abnormalities, hypothermia, hypovolemia/dehydration, and secondary infection leading to cellulitis, pneumonia, and sepsis. Early diagnosis and initiation of treatment are critical to preventing serious complication and mortality [4, 7]. Mortality in children is about 4%, when substantial skin involvement is present and secondary infection develops [2, 12]. Mortality in adults is much higher, up to 40–60%, likely due the severity of staphylococcal infection as well as associated chronic underlying medical conditions particularly renal insufficiency/failure, immunosuppression, HIV/AIDS infection, or malignancy [29].

Episodes of SSSS are unlikely to recur as long as the patient develops an appropriate humoral response to the exposed exfoliative toxin. Rare cases of recurrent SSSS have been reported, mostly in premature infants with immature immune systems and therefore unable to develop an antibody response [36].

Bottom Line: Staphylococcal Scalded Skin Clinical Pearls

- SSSS is an uncommon but potentially fatal disorder mostly seen in infants and children under the age of 5 years.
- Adults have a higher mortality rates (40–60%); comorbid conditions and severe staphylococcal infections contribute.
- Caused by exfoliative toxin-producing *S. aureus* that leads to destruction of cell-to-cell connections in the most superficial layers of the epidermis.
- Starts with tender, diffuse, scarlatiniform erythema with overlying flaccid bullae that are Nikolsky sign positive.
- Rupture of bullae show moist, bright red eroded skin underneath that appears scalded.
- Absence of mucosal membrane involvement.
- Diagnosis is suspected clinically and confirmed by biopsy.
- Early treatment with penicillinase-resistant synthetic penicillin with or without clindamycin is crucial.

Resources

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