



Staphylococcal Scalded Skin Syndrome

Staphylococcal scalded skin syndrome (SSSS), also known as Ritter's disease, is a severe blistering skin condition caused by toxins produced by *Staphylococcus aureus* infection. This disease primarily affects neonates and young children but can occasionally occur in adults, particularly those with compromised immune systems. The condition is characterized by the rapid onset of widespread erythema, blistering, and subsequent desquamation (peeling) of the skin, resulting from the action of epidermolytic toxins.

Pathophysiology

SSSS is caused by *Staphylococcus aureus*, which produces two distinct exfoliative toxins, known as epidermolytic toxins A and B. These toxins are proteases that cleave desmoglein 1, a critical protein in the desmosomes that hold skin cells (keratinocytes) together in the outermost layer of the epidermis. This cleavage leads to the separation of the epidermal layer from the underlying dermis, resulting in the formation of large, fluid-filled blisters (bullae) and skin sloughing. Although the bacteria themselves do not directly invade the skin, the toxins they produce cause the characteristic skin damage.

Epidemiology and Risk Factors

SSSS is most commonly seen in children under the age of 5, particularly neonates and infants, due to their immature immune systems and underdeveloped renal function. The condition is also more prevalent in individuals with compromised immune systems, including those with chronic skin conditions (e.g., atopic dermatitis), renal failure, or those who are immunosuppressed. Adults with certain comorbidities, such as HIV or those undergoing chemotherapy, are also at risk. The disease is often triggered by an underlying *S. aureus* infection, which can occur in various body sites, including the nose, eyes, throat, or umbilical area.

Clinical Features

SSSS typically begins with localized signs of infection, including erythema (redness) and tenderness at the site of bacterial entry, such as the nose, throat, or umbilical area. These symptoms are accompanied by systemic manifestations, including fever and malaise. Within 1–2 days, a diffuse erythematous rash develops, often with a sandpaper-like texture, and spreads to the trunk, face, and flexural areas (e.g., groin, neck, armpits). The rash is most prominent in the creases and may be associated with scaling and mild edema.





As the disease progresses, the skin becomes more fragile, and large blisters (bullae) form, typically with thin, fragile roofs that rupture easily. The skin beneath these blisters is moist, red, and glistening. Desquamation (skin peeling) occurs within several days, leaving behind a red, raw surface. The lesions often heal within 7–10 days with minimal scarring, but the process is painful and distressing. Despite its typically benign course, SSSS can be fatal if not treated promptly, particularly in neonates or immunocompromised individuals.

Diagnosis

The diagnosis of SSSS is primarily clinical, based on the characteristic appearance of the skin lesions, such as the widespread erythema, blistering, and desquamation. However, a skin biopsy may be helpful in confirming the diagnosis, particularly if the presentation is atypical. Histopathological examination typically reveals separation of the epidermis from the dermis (epidermal splitting), with mild inflammation at the base of the epidermis. The blisters are typically thin-roofed and easily rupture, with a lack of significant infiltration by *S. aureus* at the lesion site.

In cases where the diagnosis is unclear, bacterial cultures from the nose, throat, eyes, or blood may be obtained to identify the causative strain of *S. aureus*. In some cases, polymerase chain reaction (PCR) testing can be used to detect the presence of the genes responsible for the production of exfoliative toxins A and B.

Treatment

Treatment for SSSS depends on the severity of the disease and whether systemic involvement is present. Management strategies are focused on eradicating the *S. aureus* infection, preventing complications, and supporting skin healing.

> Mild to Moderate Cases

In less severe cases of SSSS, treatment can often be managed on an outpatient basis. Oral antibiotics that target *S. aureus*, such as dicloxacillin or cephalosporins (e.g., cephalexin), are typically used. These antibiotics are effective in eradicating the bacteria responsible for toxin production.

> Severe Cases and Hospitalization

Patients with severe SSSS, particularly those with extensive skin involvement, fever, or signs of systemic infection, require hospitalization and intravenous (IV) antibiotics. First-line IV antibiotics include nafcillin or oxacillin, which are effective against *S. aureus*. In cases where methicillin-resistant *S. aureus* (MRSA) is suspected, vancomycin or clindamycin may be used. Fluid and electrolyte balance should be carefully monitored, particularly in infants, as fluid loss from blistering can lead to dehydration.

> Corticosteroids

Corticosteroids should not be used in the treatment of SSSS, as they may interfere with the

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body's natural healing processes and potentially worsen the condition by inhibiting immune responses. Their use has been associated with increased risk of secondary infection and delayed wound healing.

> Supportive Care

Supportive care includes maintaining adequate hydration, managing pain, and preventing secondary infections. Topical wound care may also be needed to protect the skin as it heals, and dressing changes should be performed using sterile techniques to minimize the risk of infection.

Emerging Treatments and Research

Recent research has focused on improving the management of *S. aureus* infections, including those leading to SSSS. Advances in antimicrobial therapy, such as the development of novel antibiotics targeting MRSA, provide promising prospects for treating this disease, especially in hospitalized patients with complicated or resistant infections. Additionally, vaccines against *S. aureus* toxins are being explored, although they are not yet widely available.

Prognosis and Prevention

With early diagnosis and appropriate treatment, the prognosis for SSSS is generally favorable, and most children recover fully within 7–10 days. However, mortality can occur in severe cases, particularly in neonates or individuals with significant comorbidities, such as renal failure or immunosuppression. Preventive strategies include promoting good hygiene practices, particularly in healthcare settings, and addressing any underlying infections promptly to prevent toxin production.

Conclusion

Staphylococcal scalded skin syndrome (SSSS) is a serious yet treatable condition primarily affecting infants and young children, characterized by the widespread formation of blisters and skin peeling caused by *S. aureus* toxins. Early recognition and appropriate antimicrobial treatment are crucial for preventing complications and ensuring recovery. With advances in antimicrobial therapy and supportive care, the management of SSSS has improved significantly. However, further research into novel treatments and preventive strategies is needed to reduce the burden of this disease.

References

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