Case Report

Staphylococcal Scalded Skin Syndrome in a newborn – A case report

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Abstract

Staphylococcal scalded skin syndrome (SSSS), also known as Ritter disease, and staphylococcal epidermal necrolysis, encompasses a spectrum of superficial blistering skin disorders caused by the exfoliative toxins of some strains of *Staphylococcus aureus*. Here, we have presented a case of ten-day-old newborn who was admitted with erythematous lesions over whole body surface area, of two days duration. A culture of the blood sample collected on the day of admission yielded a pure growth of *Staphylococcus aureus*, which was sensitive to oxacillin, cefazolin, cefatoxime, vancomycin, netilmycin, ticarcillin, and ciprofloxacin. In view of the extension of the lesions to other areas of the body the child was put on injection vancomycin and topical mupirocin in addition to ceftriaxone started earlier. A clinical diagnosis of Staphlococcal Scalded Skin Syndrome (SSSS) was made.

Key words

Staphylococcal scalded skin syndrome, SSSS, Ritter disease, *Staphylococcus aureus*.

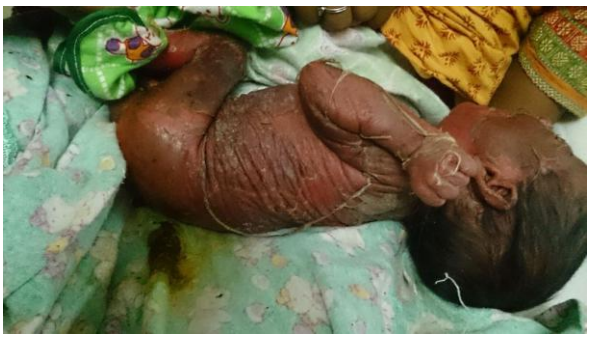
Introduction

Staphylococcal scalded skin syndrome (SSSS), also known as Ritter disease, and staphylococcal epidermal necrolysis, encompasses a spectrum of superficial blistering skin disorders caused by the exfoliative toxins of some strains of *Staphylococcus aureus* [1]. Staphylococcal Scalded Skin Syndrome (SSSS) is an extensive exfoliative dermatitis that occurs primarily in newborns and previously healthy children. The diagnosis of SSSS is mainly based on clinical appearance, and it is confirmed by the isolation of S. Aureus or the exfoliative toxin (ET) and/or the histopathological findings. However, the typical SSSS presents for several days, and the generalized SSSS is a rare condition that usually occurs in neonates. Thus, a structured management approach to this disease that involves a high index of suspicion is recommended [2]. Here, we have reported a case of SSSS in a newborn because of its rare occurrence.

Case report

A ten-day-old newborn was admitted with erythematous lesions over whole body surface area, of two days duration. The child was a full-term, normal baby. The child developed an erythematous rash on the 8th day of birth. The lesion started over the face and spread to other parts of the body within the next day. There was no history of administration of any drugs after birth. On examination, at admission, the child was toxic, febrile with erythematous lesions around the mouth, nose, and over the neck. The oral mucosa was normal. The lesions spread to the hands, gluteal region, thighs, and legs over the next day (**Photo – 1**). A culture of the blood sample collected on the day of admission yielded a pure growth of *Staphylococcus aureus*, which was sensitive to oxacillin, cefazolin, cefatoxime, vancomycin, netilmycin, ticarcillin, and ciprofloxacin. Swabs were collected from the cutaneous lesions on the day of admission did not yield any growth. In view of the extension of the lesions to other areas of the body the child was put on injection vancomycin and topical mupirocin in addition to ceftriaxone started earlier. A clinical diagnosis of Staphlococcal Scalded Skin Syndrome (SSSS) was made. The lesions healed over the next two days and no new lesions were observed. The child became afebrile and was feeding well.

Photo – 1: Staphylococcal Scalded Skin Syndrome.



Discussion

Exfoliative skin diseases of neonates are relatively rare [3] although cases of fetal congenital SSSS are reported [4]. SSSS is a toxin mediated staphylococcal infection. Staphylococcal ETA and ETB are secreted mainly from Phase II staphylococci. The split in the granular layer is attributable to binding of ETA or ETB to desmoglein within desmosomes and to keratohyalin granules and the granular layers [5]. These toxins induce proteolysis by trypsin like serine proteases [6].

In the neonate, the usual onset is between days 3 and 16 of age [7]; Factors responsible for the age distribution include renal immaturity leading to decreased toxin clearance in neonates [8] and lack of immunity to the toxin [9]. The percentage of carriers of antibody to ET-A decreases from 88% immediately after birth to a minimum of 30% at 4 months to 2 years and then rises again [9]. Thus, lack of trans-placental ET-A antibodies due to no immunity of the mother as well as decreasing antibody titers may have contributed to SSSS.

In infants and young children, potentially fatal complications include hypothermia, dehydration,

and secondary infections. Three forms of the Staphylococcal skin disease have been described in neonates, namely, SSSS, Bullous Impetigo (BI), and Staphylococcal Scarlet fever. SSSS and BI have many features in common. However, compared with BI the skin lesions of SSSS are larger, *Staphylococcus aureus* is less frequently isolated, and less inflammatory infiltrate in the skin lesions is noticed.

The diagnosis of SSSS is usually based mainly on the clinical manifestations, and the diagnosis is supported by the presence of S. aureus in the nasal, conjunctival, pharyngeal or wound swabs. Several risk factors of SSSS have been suggested: poor renal clearance of the toxins, low antibody status, immunosuppression with immunosuppressive drugs and malignancy [10].

The differential diagnosis of SSSS includes drug-induced toxic epidermal necrolysis, epidermolysis bullosa, bullous mastocytosis, herpetic lesions, and neonatal pemphigus. Clinically the SSSS mimics toxic epidermal necrolysis (Lyell's syndrome). The typical features of SSSS are involvement of periorificial face, de-epithelialization of friction zones, and absence of mucosal involvement. However, in toxic epidermal necrolysis (Lyell's syndrome) there is a severe involvement of visible mucosa and also the respiratory, gastrointestinal, and urinary tract mucosae [11].

Staphylococcal Scalded Skin Syndrome can occasionally lead to serious complications like pneumonia, septic arthritis, hypothermia, dehydration, and secondary infections.

In the case reported, the characteristic skin lesions and failure to isolate *Staphylococcus aureus* from the lesions, but from blood culture staphylococcus aureus was isolated, absence of any mucosal lesions, and no history of administration of drugs was suggestive of SSSS. With prompt treatment the progression of the lesions was arrested and no new lesions occurred. In the case described the child responded to injection vancomycin with no new lesions and became apyrexic within 48 hours.

Conclusion

Staphylococcal scalded skin syndrome (SSSS) manifests acute exfoliation of skin following a staphylococcal infection. The diagnosis of staphylococcal scalded skin syndrome was based on clinical grounds, supported by the presence of *Staphylococcus aureus* in nasal, conjunctivae, pharyngeal, umbilical swabs and the result of skin biopsy. Early diagnosis and appropriate treatment can prevent the mortality associated with SSSS complications.

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