Case Report

Staphylococcal scalded skin syndrome in a neonate presenting in the first 24 h of life

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Abstract

Staphylococcal scalded skin syndrome (SSSS) is an immense desquamating erythematous skin infection with features such as blistering and epidermal peeling. SSSS is rare in neonates presenting within 24 h of life in both term and preterm. We report a rare case presenting within 24 h, a 36-h-old neonate presenting with erythematous lesions over the genital area since 12 h of life, which progressed to involve the upper and lower limbs. The mucosal areas were spared. The culture from the lesion site isolated *Staphylococcus*, confirming SSSS. The baby improved on antibiotics administration with no fresh lesions and was discharged with a favorable outcome. A high index of suspicion, early and prompt initiation of treatment, and strict infection control measures are the indispensable steps in the management of SSSS in a neonate.

KEY WORDS: SSSS, infection control, NICU outbreak

Introduction

Staphylococcal scalded skin syndrome (SSSS) is an immense desquamating erythematous skin infection with features such as blistering and epidermal peeling. The coagulase-positive *Staphylococcus aureus* produces two epidermolytic or exfoliative toxins (ETs), ETA and ETB, which induce this clinical condition. Intraepidermal splitting via the granular layer by the particular cleavage of desmoglein 1, a desmosomal cadherin protein that mediates cell–cell adhesion of keratinocytes in the granular layer, occurs owing to the effects pronounced by these toxins.[1] It is usually seen in children aged < 5 years and affecting neonates in the late neonatal period (i.e., 3–15 days of life).[2] SSSS is rare in neonates presenting within 24 h of life in both term and preterm. Till date, only four cases have been reported in both term and preterm neonates, according to the available literature. We present a case of a term neonate with an onset of SSSS within 24 h of life who survived and was finally discharged home.

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A 36-h-old full-term, vaginally delivered, male neonate with no significant antenatal history was admitted to our neonatal intensive care unit (NICU) with erythematous lesions over the genital region of 16-h duration [Figure 1].

On examination, the baby was febrile and lethargic with diffuse, erythematous, bullous lesions around the genital area. Nikolsky's sign was positive. The mucosal areas were spared. A septic workup, cerebrospinal fluid (CSF) analysis, blood culture, and swab from skin lesion were sent for Gram staining and culture. The first-line antibiotics, intravenous (IV) ampicillin and gentamicin, were started awaiting reports. The skin was covered with petroleum jelly and occlusive dressings. An appropriate fluid therapy was initiated. The baby was kept in isolation, and strict aseptic measures were taken while handling the baby to avoid cross infection to other neonates. After admission, the lesions progressed to involve the lower and upper limbs along with the face and ears within the next 24 h. A clinical diagnosis of SSSS was made.

The blood culture showed the growth of *S. aureus* sensitive to cefotaxime and vancomycin. The skin culture grew *S. aureus* with Gram stain positive for Gram-positive cocci, but CSF analysis showed normal findings.

Figure 1: Neonate with SSSS.

After isolating the organism, the diagnosis of SSSS was confirmed. The lesions started to heal within 3 days of IV medication with no new lesions observed, and the baby was afebrile with adequate intake of breast feed. The baby was discharged after completing 14 days of IV antibiotics.

Discussion

Von Rittershain first described the clinical characteristics of SSSS in a newborn in 1870.[3] SSSS is referred to as a spectrum of superficial blistering skin disorder caused by the ETs of *S. aureus* with clinical conditions ranging from mild to severe forms spreading throughout the body surface. There are three serological forms of staphylococcal ETs.

They are ETA, ETB, and ETD, all of which cleave human desmoglein 1. Only ETA and ETB have been firmly linked to human SSSS.[4] Its incidence in neonates and infants is attributed to the lack of protective antibodies to ETA and ETB and immature renal function, which impairs the ability to excrete the toxin.[2]

The diagnosis of SSSS is based on clinical, histological, and microbiological findings[5]:

a) a clinical pattern of tenderness, erythema, desquamation, or bullae formation;
b) histopathological evidence of intraepidermal cleavage through the stratum granulosum;
c) isolation of ETA- and/or ETB-producing *S. aureus;*
d) the absence of pemphigus foliaceous by direct and indirect immunofluorescence.

The potential complications include fluid loss, dehydration, cellulitis, pneumonia, sepsis, osteomyelitis, septic arthritis, and necrotizing fasciitis.[7]

Differential diagnosis include toxic epidermal necrolysis, epidermolysis bullosa, bullous mastocytosis, neonatal pemphigus, gas gangrene, necrotizing fasciitis, Stevens–Johnson syndrome.

In the above-mentioned case , the child presented characteristic skin lesions with organisms isolated from blood and skin cultures. The child was admitted in our NICU and was isolated so as to prevent any NICU outbreaks. Strict infection control measures were implemented such as the isolation of the infected neonate, handwashing, and cohort of at-risk neonates. Skin biopsy was not performed, as the clinical features were compatible with the diagnosis. Skin culture and nasal swabs from the NICU staffs were not sent. With prompt antibiotic and supportive treatments, the baby improved with no secondary infections and was discharged.

Conclusion

SSSS in a neonate can be life-threatening and may lead to NICU outbreaks. A high index of suspicion, early and prompt initiation of treatment, and strict infection control measures are the indispensible steps in the management of SSSS in a neonate. This will prevent further complications and mortality.

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