Case Report

Staphylococcal Scalded Skin Syndrome – A Rare Cause of Neonatal Morbidity

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ABSTRACT

Staphylococcus scalded skin syndrome (SSSS) is a rare skin syndrome which is caused by exfoliative toxins of *Staphylococcus aureus*. We present a case of a 1 day old infant with exfoliative wound over right foot extending from below knees to ankle. The diagnosis of SSSS was reached on basis of clinical features and holoprosencephaly. Baby responded well to antibiotic and surgical treatment. Early diagnosis and treatment and following strict aseptic measures in neonatal intensive care unit are important in the management of such a case. This case is important from maternal point of view as the mother was very depressed with the baby's condition and was not content with the antenatal care.

Key words: Exotoxin, methicillin-resistant *Staphylococcus aureus*, Nikolsky's sign

INTRODUCTION

Mortality in children with *Staphylococcus* scalded skin syndrome (SSSS) is approximately 4%.[1] *Staphylococcus aureus* is a bacterium commonly found harmlessly colonizing human skin and mucosa without causing any morbidity.[1] Colonization begins soon after birth. Sometimes, it breaks through the skin causing infection. In neonates, the risk of SSSS is much higher due to inadequate immunity and immature renal clearance of exotoxin in them.[2] The severity of SSSS varies from a few blisters to severe exfoliation affecting the entire body.[3] The epidermolytic toxins (ETs) released by *S. aureus*, particularly splitting and ETB, lyse desmoglein-1, present on desmosomes located in the strata granulosum of the epidermis, causing a loss of cell splitting adhesion between the keratinocytes, and leading to intraepidermal splitting.[3] Aseptic and antiseptic precautions taken during delivery and in neonatal intensive care unit (NICU) can prevent such cases in infants.

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CASE REPORT

A full term primigravida was delivered through cesarean section. The baby had skin peeling over right lower limb, blisters on vulva, blisters under umbilicus, and on toes. Baby was taken to NICU. Lesions were noted to increase in size. She was irritable with slight abdominal distention. The lesions over the baby's body were bad that caused depression in mother and anxiousness among the relatives, discontent with the antenatal and intranatal care of the mother and concern

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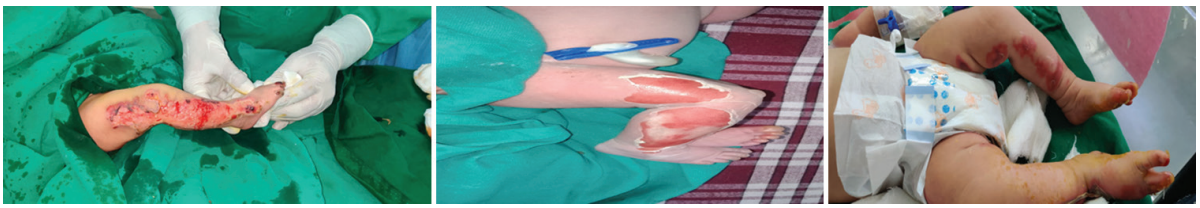
regarding the management of the baby's condition and cost of treatment.

The baby cried immediately after birth. The mother had no history of consumption of any drug, except for iron, and folic acid tablets. There was no history in the family of any skin diseases. The baby developed the above symptoms immediately after birth. The mother was also restless and anxious about the prognosis of the baby. On examination, the baby was toxic looking and irritable. Baby weight was 3.9 kg. SSSS was suspected based on clinical features. On admission, blood culture was sent. C-reactive protein was sent which came negative. Immunofluorescence and genetic studies, to detect exfoliative toxins (Ets) and ET genes, could not be performed as patient was non affording. On the basis of blood culture and clinical picture, diagnosis of SSSS was made. The infant was started on Inj meropenem 80 mg IV, inj vancomycin 40 mg IV, inj amikacin 60 mg IV, and inj calcium gluconate on admission. Blood culture showed growth of *methicillin-resistant S. aureus* (MRSA). Antibiotics were upgraded according to the sensitivity pattern. Parental linezolid was started.

On day 8 of life, baby was having black necrotic tissue on right limb and was posted in OT for debridement of necrotic tissue over right lower limb. It was uneventful. Pus culture and Histopathology examination of necrotic tissue was taken during OT and was sent to lab.

Before Debridement

After Debridement



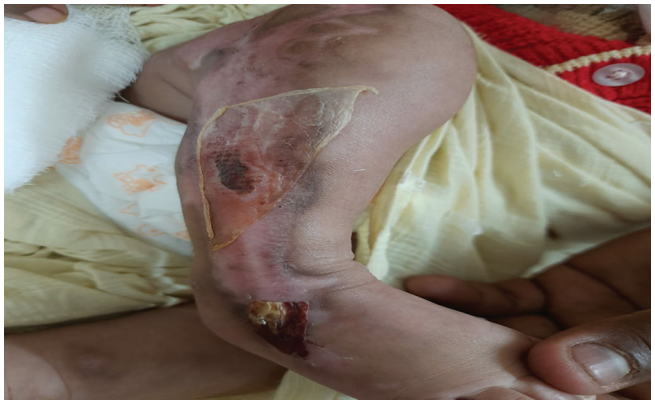
Pus culture came positive for MRSA and sensitive for gentamycin and moxifloxacin. Baby was started on inj. linezolid 20 mg TDS and inj gentamycin 40 mg OD.

HPE findings showed fragmented tissue bits showing focal epidermal lining of stratified squamous lining epithelium which was extensively necrosed and covered with inflammatory exudate. Subcorneal bullae filled with acute inflammatory infiltrate were noted.

Baby was stable on room air and was on direct Breast feed after the debridement. Daily dressing of baby was done and on day 20 of life, collagen dressing of baby was done by plastic surgeon skin grafting was considered to be the first option but the relatives were not affording so collagen dressing was done as the next best option. Daily dressing of the baby and antibiotics cover was the key factors in recovery of baby. As antibiotics were the most important line of treatment, Inj piptaz was given for 5 days. Syrup folvite and calcium were given as supplements. Baby was shifted to mother and was stable and we counseled the patient so she could bond with the baby. She was also explained about how to maintain asepsis by washing of hands before handling the baby, daily changing of clothes, and careful handling of the baby.

The mother was having depression due to the serious condition of the baby and was worried about the prognosis, cost of treatment and was having doubts regarding her antenatal care. Mother and relatives were counseled daily regarding the ongoing treatment of the baby and prognosis to relieve their anxiety and gain confidence. They were shown the pictures of the baby daily and explained about the improving condition of the baby and finally she was willing to breastfeed the baby.

A 2nd time collagen dressing was done after 7 days of first collagen dressing. It was uneventful. Baby was discharged on day 29 of life after careful examination of wound and general condition of the baby on antibiotics and supplementary medications.



This picture shows the wound after second collagen dressing on day 15 of life.

DISCUSSION

SSSS in neonate is a serious and occasionally fatal condition.[4] The ETs produced by S. aureus are considered to be the pathogenic agent in SSSS. There are two exfoliative toxins that are identified, exotoxin A and exotoxin B. Most strains of *S. aureus* isolated from patient suffering from SSSS belong to phage Group II (about 80%).[5] These toxins have exquisite specificity in causing loss of desmosome-mediated cell adhesion within the superficial epidermis only. When the toxins are released into the bloodstream, the lack of protective antitoxin antibody in neonates allows the toxins to reach the epidermis where they act locally to produce the characteristic skin lesions.[4] These human exfoliative toxin antibodies which have neutralizing properties decrease from 0 to 3 months. SSSS clinical presentation includes fever, facial edema, conjunctivitis, perioral crusting with mucous membranes being spared, dehydration, and Nikolsky's sign being positive.[6] When the intact superficial epidermis is dislodged by a shearing force, it indicates that there is a plane of cleavage at the dermal-epidermal junction and this is referred to as Nikolsky's sign. The diagnosis is usually made on clinical grounds (characteristic appearance of the rash with fever) and the presence of S. aureus in the nasal, conjunctival, pharyngeal, wound swabs, or positive blood culture. Antibiotics are the mainstay of SSSS treatment. Consideration needs to be given to pain management, temperature regulation, fluid management (rehydration), nutrition, and skin care. Corticosteroids are contraindicated. We added important rehydration, antipain, and skin care.

The treatment was effective, and the neonate is healing after 2 weeks. The percentage of carriers of antibody to ET-A decreases from 88% immediately after birth to a minimum of 30% from 4 months to 2 years and then rises again. Thus, lack of transplacental ET-A antibodies due to no immunity of the mother as well as decreasing antibody titers may contribute to SSSS.[7] The differential diagnosis of SSSS includes drug-induced toxic epidermal necrolysis, epidermolysis bullosa, bullous mastocytosis, herpetic lesions, and neonatal pemphigus. SSSS can occasionally lead to serious complications such as pneumonia, septic arthritis, hypothermia, dehydration, and secondary infections.[8] SSSS has an incidence between 0.09 and 0.56 cases/million.[8] Phage typing the *S. aureus* is found to be useful, as almost 80% of the strains of *S. aureus* causing SSSS belong to phage Group II.[8] Other sparingly used diagnostic tools are techniques measuring the titers of the ETs and isolating their gene sequences. As most strains of *S. aureus* causing SSSS are methicillin-sensitive, penicillinase-resistant beta-lactam agents such as cloxacillin, dicloxacillin, oxacillin, flucloxacillin, and nafcillin are the first-line antibiotics.[8] If the patient is not responding to these agents, MRSA should be suspected, for which vancomycin and linezolid are the drugs of choice. Topical therapy should constitute either fusidic acid and/or mupirocin. Exposed, damaged areas can be treated with emollients which soothe and moisturize the skin. With the rise of MRSA strains and increase in the mortality rate of SSSS, some newer therapies have been investigated by researchers. Infusion of fresh frozen plasma obtained from adults into children with SSSS has been found to be successful in a pediatric age group.[9] Although theoretically developing desmoglein-1 antitoxins/ analogues to antagonize the ETs causing SSSS should show superior results, no clinical or experimental studies exploring this therapy are available. Vaccines targeting *S. aureus* have failed in the Phase III of clinical trials and are still in developmental phase[10] such as this, emphasize the importance of dermatological conditions in NICU. The significance of skin failure as a distinct entity comparable to any other organ system failures leading to high mortality in a NICU setting.

CONCLUSION

SSSS responds well to specific antibiotic therapy, it remains an emergency and a potential fatal condition in neonates. Hence, early diagnosis, prompt treatment, and following aseptic measures in NICU are the mainstay for its successful management. Isolating neonates is mandatory to prevent the outbreak of SSSS

in the unit. Future policies to prevent similar situations may include continuous monitoring and surveillance of infection rates, effective nursery design and staffing, emphasis on hand washing to prevent cross infection, educational programs, and feedback to nursing staff to improve infection control.

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