

Neonatal staphylococcal scalded skin syndrome: an outbreak in a Neonatal Intensive Care Unit in Portugal

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Abstract

Staphylococcal scalded skin syndrome (SSSS) is a blistering skin condition caused by exfoliative toxin-producing strains of *S. aureus*. It usually occurs in children under 5 years old but is rare in neonates, especially in very low birth weight, premature infants. Although infrequently, clinical outbreaks have been reported. We describe an outbreak of SSSS that occurred among 3 premature infants admitted to a level III Neonatal Intensive Care Unit (NICU).

As soon as it was realized that there was an outbreak, the Infection Control Commission (ICC) was informed and measures to contain the outbreak were immediately applied.

Keywords

Staphylococcal scalded skin syndrome, infection outbreak, premature infants.

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Introduction

Staphylococcal scalded skin syndrome (SSSS) includes a spectrum of blistering skin diseases, which range from mild localized blistering lesions to extensive areas. It is characterized by a positive Nikolsky sign, leaving extensive areas of denuded skin [1] and in newborns it is also known as Ritter von Ritterschein disease [2]. The location of the lesions varies according to the age of the child. In neonates, the lesions are mostly found on the perineum or periumbilical area, or both, while in older children the extremities are usually more commonly affected [3].

SSSS is caused by exfoliative toxins, such as ETA and ETB, which are produced by a small percentage of *S. aureus*. The mechanism by which the aforementioned toxins cause exfoliation had until recently been uncertain. However, it has been theorized that they function as proteases targeting the protein desmoglein-1 (DG-1), thus compromising keratinocyte cell-to-cell attachment in the superficial epidermis [4-6].

In most cases, treatment with B-lactamase penicillins such as flucloxacillin is usually effective. However, there have been reports of an increase in community-acquired methicillin resistant *S. aureus* (CA-MRSA) causing SSSS [7-9]. The prognosis usually is good if diagnosis and treatment are made quickly.

Case reports

We describe the cases of three premature babies admitted to our Neonatal Intensive Care Unit (NICU), who, within a week, developed skin lesions compatible with SSSS.

The index case was a 27-week gestational premature boy that was delivered via an emergency C-section due to placental abruption, weighing 1,100 g and Apgar 7/9. On day 25 of life, an erythematous lesion with macerated skin was noted around the infant's nose and mouth (Fig. 1). The infant was receiving supplemental oxygen via nasal continuous positive airway pressure (nCPAP) and fortified enteral feeds via orogastric tube, fixed with adhesive around the mouth. On the following two days, the lesions rapidly progressed to the thorax, ear lobes, hands, feet, buttocks and scrotum.

On the same day, another premature twin baby boy (gestational age of 28 weeks, born due to premature rupture of membranes [PROM], weighing 1,240 g and Apgar 8/7), who also was

under nCPAP treatment, appeared with similar skin lesions around the nose and mouth on day 14 of life (Fig. 2), rapidly progressing to the upper left limb and thorax.



Figure 1. Case index, desquamation around the infant's nose and mouth.



Figure 2. Case two, desquamation around the infant's nose and mouth.

After cultures from the lesions and bloodstream were obtained, intravenous gentamicin and vancomycin therapy was started on both neonates and they were placed under isolation measures. After completing 3 days of antibiotic therapy, a third case emerged with the same lesions (**Fig. 3**), on day 18 of life (the twin of the second child, a 28-week gestational age girl, weighing 1,180 g and Apgar 8/9). All the cases were in the same room. A nosocomial outbreak of a staphylococcal infection was recognised and the ICC was contacted.

All 3 cases changed antibiotics to flucloxacillin and clindamycin for 10 days according to the antibiogram obtained from the results of the lesion cultures (methicillin-sensitive *S. aureus*, susceptible to flucloxacillin and clindamycin) and all did topic treatment with L-Mesitran®. All bloodstream cultures were negative.

All lesions resolved completely within the next few days, and the infants' subsequent course was uneventful.

The two initial cases were managed as individual episodes but, after a third case was identified, an official outbreak was recognised. The ICC was contacted as soon as a nosocomial outbreak was identified and infection control measures were implemented immediately according to General Health Department guidelines. These measures included isolation for all patients hospitalized in the NICU, placement of infected patients in a cohort, reinforcement of asepsis measures in the handling of patients and a colonization study for all patients, including those in the paediatric unit, since the nursing team was shared with this unit. For 72 hours, new patients were not hospitalized and cleaning and disinfection of the facilities and



Figure 3. Case three, desquamation on the infant's left upper limb, thorax and around the mouth.

equipment were carried out. Since no more cases occurred, an epidemiologic investigation was not conducted.

Discussion

In epidemiology, an outbreak is an increase, often sudden, in the number of cases of a disease, above what is normally expected in that population in a limited geographic area. SSSS can occur individually or as outbreaks in nurseries. Outbreaks are usually due to asymptomatic carriers who spread the disease to susceptible individuals [10].

As far as a differential diagnosis is concerned, SSSS differs from bullous impetigo in the sense that, despite both being blistering skin diseases caused by staphylococcal exfoliative toxins, these toxins are localized and restricted to the area of infection in bullous impetigo, thus allowing a positive bacterial culture from the blister contents. However, in SSSS the toxins spread hematogenously from a localized source, which leads to possible epidermal damage in distant sites and sterile cultures of the bullous material [11]. Blood culture is usually negative in children [12].

SSSS usually occurs in children under 5 years old, but is rare in neonates especially in very low birth weight premature infants [13]. Even though SSSS is usually easily diagnosed on clinical grounds, and readily treated with conventional antibiotics, it still presents with high mortality rates and outbreaks may be difficult to control. Secondary complications, which are particularly common in neonates, can often be lethal. These secondary complications include fluid loss and dehydration, cellulitis, pneumonia, sepsis, osteomyelitis, septic arthritis and necrotizing fasciitis [14, 15].

The first treatment option with vancomycin and gentamicin was justified because all cases were very preterm infants admitted to the NICU and MRSA could have been the causative agent [16, 17]. As soon as the result of the sensitivity was obtained, the antibiotic therapy was adjusted.

It is crucial to have a high index of suspicion, quick diagnosis, implementation of infection control measures and early institution of treatment to prevent the expansion of SSSS in the infant, avoiding complications, decreasing mortality, and preventing the spread of disease to other infants [12, 13].

In our cases, the above-mentioned infection control measures were successful in preventing

nosocomial spread to the other NICU infants, and no systemic signs of sepsis were evident in any of the three cases.

Declaration of interest

The Authors declare that there is no conflict of interest.

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