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Case Report

Staphylococcal Scalded Skin Syndrome in an Infant

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Abstract

Staphylococcal scalded skin syndrome is a bullous disease of the skin caused by toxins (exfoliatins) secreted by certain types of Staphylococcus aureus. These toxins may act as superantigens, stimulating the proliferation of T lymphocytes, with a massive release of cytokines and consequent epidermal damage. In neonates, the lesions are mostly found on the perineum or periumbilically, while the extremities are more commonly affected in older children. The disease begins with erythema and fever, followed by formation of large fluid filled bullae which quickly rupture on slightest pressure to leave extensive areas of denuded skin. We present a case of staphylococcal scalded skin syndrome in infant.

Key words: Staphylococcal, Scalded skin, Nikolsky sign, Infant

Introduction

Staphylococcal scalded skin syndrome (SSSS) is the term used for a collection of blistering skin diseases induced by the exfoliative toxins of Staphylococcus aureus. It primarily affects neonates and young children, although adults with underlying diseases are also susceptible[1,2]. SSSS originates from a focus of infection that may be a purulent conjunctivitis, otitis media, or occult nasopharyngeal infection. It usually begins with fever, irritability, and a generalized, faint, orange-red, macular erythema with cutaneous tenderness[3,4]. Within 24-48 hours, the rash progresses from a scarlatiniform to a blistering eruption. Characteristic tissue paper–like wrinkling of the epidermis is followed by the appearance of large, flaccid bullae in the axillae, in the groin, and around the body orifices. Subsequent generalized involvement occurs elsewhere on the body, but infection spares the mucous membranes. SSSS diagnosis is currently based mainly on clinical grounds, supported by the presence of staphylococcal aureus in nasal, conjunctival, pharyngeal, umbilical, or other swabs[5]. Here we reported a case of staphylococcal scalded skin syndrome in infant.

The Case

A 10 month old male child, presented with history of redness and peeling of the skin all over the body and fever for 1 day. There was pus discharge from bilateral ear lobules since the last two days. There was also history of purulent discharge from both eyes. The redness and peeling started around the mouth and spread to involve extensive area of the body. History of bilateral ear lobe was pricked by jeweler 15 day back. On examination, the patients was febrile (39^0C), PR: 130/min, RR: 30/min and SPO2: 98. Superficial skin peeling over the forehead, face, chest, axilla, buttock and upper limb was present. There were few bullae present on the abdomen. Nikolsky sign was positive.

On investigation, complete blood cell count was: Hb: 8.4gm%, TC: 6,500/cmm (LY32.3%, MO 10.2%, GR 57.5%), platelet count was 4.9 Lac/cmm. Urea and electrolytes were normal. Conjunctival swab and wound swab

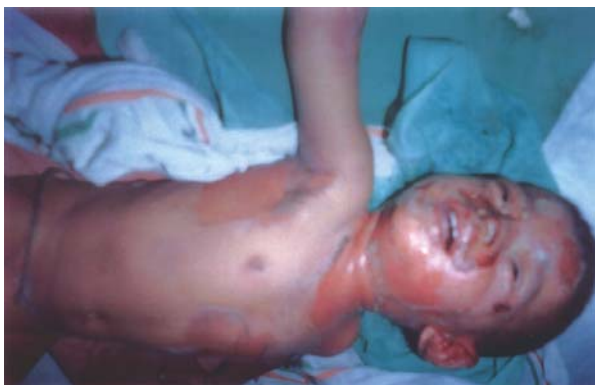

Fig 1: Showing generalized SSSS in a previously well infant shows the characteristic well-demarcated erythematous superficial exfoliation with areas of skin sparing.

revealed staphylococcus aureus growth whereas blood culture showed coagulase positive staphylococcus. Patient was put on injection cefotaxime and cloxacillin for 5 days and then shifted to oral antibiotic on syrup cefixime and syrup amoxycloxacillin for next 5 days. All skin lesions were healed within 10 days after course of antibiotics.

Discussion

Staphylococcal scalded skin syndrome is an acute epidermolysis caused by a staphylococcal toxin. The disorder is caused by toxigenic strains of *Staphylococcus aureus,* usually belonging to phage group 2 (types 3A, 3B, 3C, 55, or 71). Two exotoxins (ETs), epidermolytic toxin A (ET-A) and epidermolytic toxin B (ET-B), are responsible for the pathologic changes seen in SSSS. These toxins cause intraepidermal splitting through the granular layer by specific cleavage of desmoglein 1, a desmosomal cadherin protein that mediates cell-to-cell adhesion of keratinocytes in the granular layer[6]. It is primarily a disease of neonates and children, and clinical features vary from localized blisters to severe exfoliation affecting over 90% of the entire body surface. In localized forms of SSSS, also known as bullous impetigo, tropical bullous impetigo, measles pemphigoid, and bullous varicella, characteristic fragile, thin-roofed, flaccid bullae are formed, which rupture easily to release fluid that varies from a thin, cloudy, amber liquid to purulent, opaque, white or yellow pus[7]. In neonates, the lesions are found mostly on the perineum, periumbilical area, or both, while in older children, the lesions are found most often on the extremities[8]. In the generalized forms of SSSS, widespread involvement of the entire skin surface can occur but the mucous membranes are usually spared. The disease usually follows a localized infection of the upper respiratory tract, inner ear, conjunctiva, or umbilical stump, although rare cases of SSSS caused by staphylococci isolated from patients with pneumonia, septic arthritis, pyomyositis, and maternal breast abscesses have been reported. Patients with generalized SSSS initially develop fever, malaise, and lethargy, with poor feeding and irritability, followed by a generalized, tender erythematous rash, which generally begins on the head and neck and spreads to the rest of the body within a few days. The rash is more marked in flexural creases. Soon thereafter, large, fragile, thin roofed blisters appear, which rapidly rupture on the slightest pressure (with a positive Nikolsky sign), resulting in large sheets of epidermis sloughing off to leave extensive areas of raw, denuded, varnish-like skin[9].

An abortive form of SSSS, known as the scarlatiniform variant, shows the early erythrodermic and final desquamative stages seen in SSSS, but the bullous stage does not occur[10].

The definitive diagnosis depends on culture and biopsy results. Slide latex agglutination, double immunodiffusion, and enzyme-linked immunosorbent assay tests can identify the toxins responsible for SSSS. *S. aureus* may be cultured from the conjunctiva, nasopharynx, feces, or pyogenic foci on the skin. Blood cultures are almost always negative in children, but they may be positive in adults[11]. Mild forms of SSSS such as bullous impetigo should be managed with cleaning and drying of the lesions with compresses and administration of oral antibiotics that are effective against penicillin-resistant staphylococci as well as streptococci. Methicillin, nafcillin, dicloxacillin, cloxacillin, and flucloxacillin have all been used to treat *S. aureus*, and other proposed regimens include cloxacillin plus tobramycin, and erythromycin. Severe forms require more aggressive treatment with intravenous antistaphylococcal antibiotics and extra care of denuded skin to prevent secondary infection and fluid losses and to maintain body temperature, especially in neonates. Additional broad-spectrum antibiotics should be considered if secondary skin infection is suspected, and they should cover *Pseudomonas* species[12].

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