Case Report: STAPHYLOCOCCAL SCALDED SKIN SYNDROME IN A NEONATE

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ABSTRACT

Exfoliative skin diseases are rare in neonates. These diseases might cause significant complications and mortality. SSSS is caused by staphylococcal exfoliative toxins A or B, which split the granular layer of the skin, induces proteolysis, and might exhibit super antigen activities, such as epidermolysis and lymphocyte mitogenicity. We describe a male neonate who developed SSSS on day 15 of life, with no clinical signs of neonatal sepsis. After cultures from the pharyngeal, conjunctivae, nasal, umbilical swab and bloodstream were obtained; intravenous cefotaxime and cloxacillin therapy were started. Infection control measures were implemented instantly and included isolation of the infected infant, personnel hand washing. Cultures from swab revealed Staphylococcus aureus, whereas the bloodstream culture revealed Pseudomonas spp. The lesions resolved completely within 2 days. No similar skin lesions were noticed in other infants in the neonatal care unit. We discuss recent advances in understanding the pathogenesis of neonatal SSSS, highlight the importance of early diagnosis and treatment, and stress the need for new adjunctive therapies for this disease.

Keywords: 4S, neonate, diagnosis, management, result

INTRODUCTION

Staphylococcal scalded skin syndrome (SSSS) is the clinical term used for a spectrum of blistering skin diseases induced by the exfoliative (epidermolytic) toxins (ET) usually group II Staphylococci (phage type 3A, 3B, 3C, 55 or 71) and occasionally group I type 52 (in Japan) (Currant 1980; Makhoul 2001). Ritter von Rittershain first described SSSS in 1878 (Darmstadt 2000). The severity of SSSS varies from a few blisters in localized SSSS to severe exfoliation affecting almost the whole body surface in generalized SSSS (Itani 1992). These toxins cause intraepidermal splitting through the granular layer by specific cleavage of desmoglein 1, a desmosomal cadherin protein that mediates cell-cell adhesion of keratinocytes in the granular layer (Ladhani 1999). Patients who do not have antibody to the toxin develop generalized disease due to hematogenous dissemination of the toxin. The rash is characterized by a painful erythroderma with a positive Nikolsky sign (Todd 2005). Exfoliative skin disease is rare in neonates. Its incidence is estimated to be 1 to 1.4 cases per million inhabitants per year. Internationally: higher in developing countries. The male to female ratio of SSSS disease was 5:1 (Currant 1980). Whereas 2 cases of staphylococcal scalded-skin syndrome (SSSS) in five years later in neonates at Dr Soetomo Hospital, Surabaya, Indonesia from January 2001-January 2006. The aim to this paper is to present a rare case of staphylococcal scalded-skin syndrome in a neonate.

CASE REPORT

Baby K, a fifteen days old boy came to the Emergency Department Dr Soetomo Hospital, referred from Tuban Hospital on December 19th 2005 with the main complaint of scalded skin since 3 days before hospitalized. 4 days before admission there was mild fever, malaise, appears irritable, and got poor feeding. Onset was abrupt; with erythematic, initially visible on his groin area, that involved the entire body within 2 days. Then the skin became wrinkled, or flat bulla containing clear fluid appeared in various locations, in his face, extremities, armpits, back and diaper area. Gentle pressure on the skin caused the upper epidermis to slide off, leaving a denude base (positive Nikolsky sign). Then the skin exfoliated in large sheets, exposing a red moist surface. The patient was extremely agitated and sensitive to touch. His mother had a cold and sore throat since about 3 days before him ill. His mother brought him to midwife and he got amoxicillin syrup,
but because no improvement his parent went to Tuban Hospital. Tuban Hospital not treated the patient yet but it was referred to Dr. Soetomo Hospital. The baby was the first child. Midwife delivered the baby spontaneously as a term neonate. The birth body weight was 3500 grams and cried loudly right after he was born. The amniotic fluid was meconial. There was no jaundice or cyanosis. The mother was 20 years old and she was healthy during pregnancy. There was no history of illness, drugs or herbal medications taken during the pregnancy. The history of skin disease in the family was also denied. He was breastfed since birth.

Physical examination on admission revealed a weak and irritable baby boy. Anemia, icteric, dyspnoea, cyanosis were not found. The body weight was 3500 grams, regular heart rate of 140 times per minute, respiration rate of 40 times per minute, and axillary temperature of 37°C. There was no retraction of chest wall. The heart and lung were normal. The abdomen was flat, no meteoric, the liver was palpable 2x1x1 cm, but the spleen was not enlarged. The extremities were warm. There was no edema. Laboratory examination revealed at admission revealed hemoglobin was 12.4 g/dl, leukocyte count was 5.700/mm³, platelet count was 776.000/mm³, and hematocrit was 35 %. Random blood glucose was 112 mg/dl, Diff count was -/-2/49/49, Serum electrolytes were in normal limits with potassium level of 5.14 meq/l, sodium level of 136 meq/l and chloride level of 121 meq/l. Urine examination: within normal limit. Also Chest X-ray showed normal condition. Based on the data, the working diagnosis was “Staphylococcal Scalded Skin Syndrome “. He was admitted to Neonatal Intermediate ward at the Department of Child Health for further management. We consulted Dermatology and Venereal disease Department and they found epidermoliisis, erosion of whole body skin. They agreed that this patient suffered from Staphylococcal Scalded Skin Syndrome. And advised to give antibiotics intravenously and wounds compress. The initial treatments included fluid infusion with dextrose 10 % and 0.18 normal saline 400 cc/ 24 hours, injection of cefotaxime 175 mg two times daily, injection of cloxacillin 55 mg three times daily, compress with normal saline for denudes skin. We also planned to exam the C-reactive protein, blood culture, and the swabs of nasal, conjunctivae, pharyngeal, and umbilical for further investigation.

On the 3rd day of hospitalization, there was no fever. The temperature was 37 °C. The breast-feeding was good and the defecation and urination were normal. The patient still in generalized erythematic. The moist, red, and denuded areas of blisters and bulla dried rapidly, appearing with desquamation of large flakes, which not develop to scar formation visible on his abdomen and extremities. Laboratory examination showed hemoglobin was 12.8 g/dl, leukocyte count was 13.200/mm³, platelet count was 716.000/mm³, and hematocrit was 38.7%. The intravenous fluid was stopped and the other treatments were continued with orally antibiotic cloxacillin 55 mg three time’s daily and topical wound.

On the 7th day hospitalization, the erythematous areas that did not exfoliate showed coarse desquamation. Laboratory examination showed hemoglobin was 11.7 g/dl, leukocyte count was 5.500/mm³, platelet count was sufficient, and hematocrit was 32.1%. Blood culture result revealed pseudomonas spp. Result of the culture from pharyngeal, conjunctivae, and nasal, umbilical stump swab revealed Staphylococcus aureus. Result of urine culture revealed Entero bacter aerogenes. C-reactive protein was negative. The bacterial sensitivity test resulted resistance of Cefotaxime and Netylmicin and sensitive with Amoxicillin and Cloxacillin. According to the result of sensitivity test we continued the orally antibiotics with cloxaciln 55 mg three times daily. The skin biopsy was planned on 7th day hospitalization for the definitive diagnosis.

Follow up 14th day hospitalizations, there was no complaint. The baby was in good condition. The skin was clean. There was no a blister or bulla anymore. The desquamation almost completely disappeared. The result of skin biopsy revealed that there was sub corneal blister, papilla dermis contained lymphocyte infiltrate, and seemed infiltrate lymphohistiocytic perivascular superficial contain many PMN. The summary of biopsy revealed the sub corneal split within granular layer of the epidermis. The patient was discharged from the hospital in the good condition and no clinical complaints were found. We advised to visit Pediatric and Dermatology outpatient clinic.

**DISCUSSION**

A fifteen-days-old boy came to Emergency Department with main complaint of scalded skin since 3 days before hospitalized. His mother had a cold and sore throat since about 3 days before him ill. Physical examination on admission revealed the skin lesion initially appeared as redness patches and tender on his groin area. Then this patch develops fluid blister. The lesion spread to armpits and around of the mouth area. The mucus membrane was normal. Within 2 days almost of his body was affected. There were no lesions on his eyes, lips or genitalia. Some of the blisters had ruptured, there was desquamation of the skin, and large, fragile, the blisters easily ruptured on the slightest pressure. The
trication applied to the skin reveals separation of epidermis from the dermis (with a positive Nikolsky sign) (Todd 2005). Supportive examinations revealed leucocytosis and the result of the culture from pharyngeal, conjunctivae, nasal, umbilical stump swab revealed *Staphylococcus aureus*. The result of skin biopsy revealed that there was sub corneal blister, papilla dermis contained lymphocyte infiltrate, and seemed infiltrate lymphohistiocytic perivascular superficial contain many PMN. The summary of biopsy is it might be SSSS that have been treated. Based on the history, physical examination, culture and the skin biopsy, the diagnosis of “Staphylococcal Scalded Skin Syndrome (SSSS)” was established. Treated initially with cefotaxime and cloxacillin injections. He was better and according the result of bacterial sensitivity that there was resistance of Cefotaxime and Netylmicin and sensitive with Amoxicillin and Cloxacillin, we continued the orally antibiotics with cloxacillin 55 mg three times daily.

Most cases of SSSS are diagnosed on clinical grounds (Darmstadt 2000; Ladhani 1998; Ladhani 1999), supported by the presence of *S aureus* in nasal, conjunctiva, pharyngeal, umbilical, or other swabs (Currant 1980). Detection of ET is required for diagnosis of SSSS (Ladhani 1998; Ladhani 1999). The differential diagnosis of the described exfoliative skin lesions in neonates includes staphylococcal scalded-skin syndrome (SSSS), bullous impetigo (BI), drug-induced toxic epidermal necrolysis, epidermolysis bullosa, bullous mastocytosis, herpetic lesions, and neonatal pemphigus (Darmstadt 2000; Makhoul 2001). Children younger than 5 years of age, and particularly neonates, are most commonly affected by SSSS. This epidemiologic pattern is explained by the importance of mature renal function in the clearance of ETs. The combination of decreased ability to achieve renal clearance of toxin and lack of specific immunity to the toxins make neonates the highest risk group (Ladhani 1999; Resnick 2003; Todd 2005).

SSSS was first described in 1878 by Ritter von Rittershain (Darmstadt 2000). Staphylococcal scalded skin syndrome (SSSS) is the clinical term used for a spectrum of blistering skin diseases induced by the exfoliative (epidermolytic) toxins (ET) of *Staphylococcus aureus* (Currant 1980; Darmstadt 2000; Ladhani 1998). The disease results from the effect of one of the two-epidermolytic toxins ET-A and ET-B, secreted mainly from phage II *staphylococci* and strains 71,3A, 3B, 3C, and 55 (Currant 1980; Ladhani 1998; Makhoul 2001; Todd 2005). In the absence of specific antibodies against ET-A and ET-B, as in the case mainly in infants and children, these toxins spread homogenously and cause SSSS (Darmstadt 2000; Makhoul 2001).

*Staphylococci* are hardy aerobic bacteria that are present in the environment and as normal flora of humans and animals. They are resistant to heat and drying and may be recovered from the environment months after contamination. These organisms are gram-positive cocci that grow in characteristic grapelike clusters. Species are classified as *Staphylococcus aureus* if they are coagulase-positive or as one of many species of coagulase-negative *staphylococci* (e.g., *S epidermidis, S saprophyticus*). *S aureus* is the most common cause of pyogenic infection of the skin; it also may cause osteomyelitis, septic arthritis, wound infection, abscess, pneumonia, empyema, endocarditis, pericarditis, meningitis, and toxin mediated diseases, including food poisoning, staphylococcal scarlet fever, scalded skin syndrome, and toxic shock syndrome (TSS). Coagulase-negative *staphylococci* tend to be less pathogenic unless a foreign body (e.g., intravascular catheter) is present (Todd 2005).

Many neonates are colonized with *S aureus* within the first postnatal week. Thereafter, up to 50% of healthy individuals carry at least one strain of *S aureus* in the anterior nares at any given time. The organisms may be transmitted from the nose to the skin, where colonization seems to be more transient. Persistent umbilical perianal and vaginal carriage has been described. *S aureus* generally is transmitted by direct contact, primarily on the hands. Autoinfection is common. Hand washing by caretakers between contacts with patients decreases the spread of staphylococci from patient to patient (Todd 2005).

Many strains of *S aureus* release exotoxins. Exfoliatin A and B are two serologically distinct proteins that produce skin separation by splitting the desmosome and altering the intracellular matrix in the stratum granulosum, resulting in localized (e.g., bullous impetigo) or generalized (e.g., scalded skin syndrome, staphylococcal scarlet fever) rashes (Todd 2005). The toxins act by separating cells from the stratum granulosum and stratum spinosum via disruption of the desmosomes within the epidermis (Peters 1998). Histological studies on the epidermal effects of ETs led to speculations that the initial appearance followed by disappearance of intercellular vesicles may be due to the release of proteolytic lysosomal enzymes by nearby cell (Ladhani 1999).

The nucleotide sequences SSSS is predominantly a disease of infancy and early childhood, with only a few adult cases reported (Ladhani 1998). In the neonate, the usual onset is between days 3 and 16 of age (Resnick
2003); Factors responsible for the age distribution include renal immaturity leading to decreased toxin clearance in neonates (Behrman 1991) and lack of immunity to the toxin (Prince 1992). 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 (Prince 1992). 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. Antibody levels are relatively low in premature infants (Currant 1980; Pollack 1996) compared to those in full-term babies, and this could have been an additional pathogenic factor for development of SSSS.

In this case, the 15-days-old neonate suffered from staphylococcus scalded skin syndrome preceded by redness patches and tender on his groin area. Within 2 days almost of his body was affected. The mucous membrane was normal. The patient betting better with treatment of antibiotics and topical toilet wound. Further evaluation revealed the staphylococcal scalded skin syndrome that was later confirmed as the culture of pharyngeal, conjunctivae, nasal, umbilical stump swab revealed caused by Staphylococcus aureus. It was a relatively common pathogen in neonate with renal immaturity leading to decreased toxin clearance and the lack of immunity to the toxin, which were sensitive to antibiotics treatment given then. The clinical course of disease in this case was appropriate with Staphylococcus Scalded Skin Syndrome caused by S. aureus. But the history of previous skin infection or contact with skin-infected patients in which S. aureus infection was denied. Unfortunately, because of technical problem, identification of exfoliative toxins A (ET-A) and B (ET-B) was not performed in our case. When the baby suffered from blisters some disease must be considered (Todd 2005). The differential diagnosis of the described exfoliative skin lesions in neonates includes staphylococcal scalded skin syndrome (SSSS), bullous impetigo (BI), drug-induced toxic epidermal necrolysis, epidermolysis bullosa, bullous mastocytosis, herpetiform lesions, and neonatal pemphigus (Makhoul 2001; Todd 2005).

SSSS (Ritter’s disease) and BI have many clinical features in common, and the lesions of BI are actually considered to represent a localized form of SSSS. 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. The surrounding sin remains normal and there is no systemic symptom or sign. 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 (Currant 1980; Todd 2005). Characteristically many of the bullae have ruptured, leaving dried-up lesions scattered in contiguous areas. The diaper area is affected most frequently. Staphylococcus aureus is the organism responsible for this infection, with exfoliative toxin released locally by this bacteria-causing production of the bulla (Todd 2005). However, compared with BI, the skin lesions of SSSS are larger, CPSA is less frequently isolated, and less inflammatory infiltrate in the skin lesions is noticed (Peters 1998; Todd 2005). Characteristically, SSSS consist of diffuse erosions, with epidermal separation in the sub corneal layer through the granular layer. The rash is characterized by a painful erythroderma with a positive Nikolsky sign (Todd 2005).

In this case, the patient was weak and become poor feeding since four days before admission; there was no sign of dehydration. The sign of shock and respiratory distress was not noted. The baby suffered from exfoliative skin lesion in almost entire the body. Gentle pressure on the skin caused the upper epidermis to slide off, leaving a denude base (positive Nikolsky sign). Then the skin exfoliated in large sheets, exposing a red moist surface. The patient was extremely agitated and sensitive to touch. The clinical features of SSSS vary from localized blisters to severe exfoliation affecting over 90% of the entire body surface (Currant 1980; Darmstadt 2000; Makhoul 2001; Osamu 2005). In neonates, the lesions are found mostly on the perineum, periumbilical area, or both, while in older children, the lesions are found most often the extremities (Ladhani 1998; Makhoul 2001). In the generalized forms of SSSS, widespread involvement on the entire skin surface can occur (Gemmel 1995).

Staphylococci may be transmitted by multiple routes, including contact with infected persons, contact with asymptomatic carriers, airborne spread, and contact with contaminated object (Ladhani 1998). The disease usually follows a localized infection of the upper respiratory tract, inner ear, conjunctiva, or umbilical stump (Darmstadt 2000; Makhoul 2001; Osamu 2005). Of these, contact with a person with a staphylococcal lesion appears to be particularly important in the spread of staphylococci (Ladhani 1998). Person with open draining lesions disseminate organism into their environment and to others via direct contact (Ladhani 1998; Osamu 2005).

In this case, contact with his mother was suspected the cause of the disease. The development of staphylococcal disease is related to resistance of the host to infection and to virulence of the organism (Ladhani 1998; Ladhani 1999; Osamu 2005). The intact skin and mucous membranes serve as barriers to invasion by
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**Staphylococci** (Osamu 2005). A whole range of host and organism factors govern the development and severity of SSSS. Initially, anatomical differences in the skin were believed to be the reason why neonates and young children are more susceptible to SSSS than adults (Ladhani 1999). SSSS is diagnosed on clinical grounds or by a culture of *S. aureus*, ideally with demonstration by the isolate of toxin production or the toxic gene.

In this case, the result of nasal, pharyngeal, conjunctival and umbilical swabs revealed *Staphylococcus aureus*. But the blood culture revealed *Pseudomonas spp.*, it might have happened because the secondary infection. The blood count of the patient revealed leucocytosis. Leucocytosis may occur in infections or inflammatory conditions (Louhead 1992). A leucocytosis is common in the erythroderma and it may be found even when all effort has been made to exclude infection of the skin or other organs as the cause (Jernigan 1996). C reactive protein (CRP) of this patient was negative after 5 days treatment. C reactive protein reacts with C carbohydrate protein (CRP) of this patient was negative after 5 days treatment. In our case the negative result of CRP indicated that an infection was subsided.

A biopsy of the blister is one of the most definitive diagnostic tests in SSSS. The characteristic histological changes in SSSS are the formation of cleavage plane high in epidermis, in the granular cell layer, and separation of the epidermal layer by edema fluid producing typical bullae (Prince 1992; Resnick 2003). The cleavage space may contain free-floating or partially attached acantholytic cells, but the remainder of the epidermis appears unremarkable and the dermis contains no inflammatory cells, except in cases of localized disease (bullous impetigo) (Prince 1992). Electron microscopy has demonstrated an increase of intracellular edema followed by necrosis of cells, suggesting that there is toxic damage after a short increase in metabolic activity (Resnick 2003). In our case, we did the skin biopsy on 7th day hospitalization, and the result of skin biopsy revealed that there was sub corneal blister, papilla dermis contained lymphocyte infiltrate, and seemed infiltrate lymphohistiocytic perivascular superficial contain many PMN. The summary of biopsy is it might be SSSS that have been treated.

Staphylococcus Scalded Skin Syndrome is now clearly distinguished from other diseases causing generalized epidermal necrosis, such as toxic epidermal necrolysis (Dancer 1988; Jernigan 1996). TEN also known as Lyell’s syndrome is probably the most important differential diagnosis of SSSS, since their treatments are very different. TEN is an uncommon but life-threatening cutaneous disorder that occurs as frequently in children as in adult (Ladhani 1998; Resnick 2003). TEN occurs 1 to 3 weeks after drug ingestion, although reexposure to the offending drug may result in a more rapid and very severe presentation, with involvement of multiorgan systems including the liver, kidneys, gut and lungs (Peters 1998; Resnick 2003). The clinical features are characterized by an initial pro duct of pyrexia, malaise, anorexia, pharyngitis and a tender, morbiliform rash followed by epidermal exfoliation in sheet. As in SSSS, fragile blisters are also seen in TEN and the Nikolsky sign is positive. However, unlike SSSS, eroding mucosal lesions of the mouth, conjunctiva, trachea, bronchi, esophagus, anus and vagina are almost always present in TEN (Resnick 2003). In SSSS, histological evaluation of skin reveals separation of skin layers at the granular layer within the epidermis as opposed to separation between the dermis and epidermis that is seen in toxic epidermal necrolysis (TEN) due to causes other than *S. aureus* (Anjali 1999). In our case, although there was history of drug medication by midwife, the presentation of the disease was different and there was no mucosal involvement in this patient. It was confirmed by the result of skin biopsy.

Management of SSSS is primarily supportive, with careful monitoring of electrolyte levels because of the potential fluid shifts across the denuded skin. Because the unprotected skin is susceptible to infection, antiseptic measures should be undertaken. Intravenous antibiotics are administered to decrease the staphylococcal burden. Steroids are not helpful in SSSS or TEN associated with *S. aureus* toxin and, in fact predispose to additional infection (Anjali 1999). Actually, SSSS is easily managed with antibiotics to eradicate the offending toxin-producing *S. aureus* strain and appropriate nursing care to prevent and/or treat any secondary symptoms (Current 1980; Makhoul 2001; Osamu 2005; Resnick 2003).

Antimicrobial therapy should be guided by the susceptibility profile of the organism. Beta-lactamase-producing strains of methicillin-susceptible *S. aureus* (MSSA) preferably are treated with semi-synthetic penicillin (e.g., intravenous nafcillin, oxacillin, and oral dicloxacillin in patients not allergic to penicillin. First-generation cephalosporin (e.g., oral cephalaxin, intravenous cefazolin is an alternative. Vancomycin should only be used for the treatment of MSSA in patients allergic to penicillin because of overuse and development of resistant organisms, and because clearance of bacteremia may be slow (Bamberger 2005).

Vancomycin is preferred for treatment in severe MRSA infections and is used only intravenously because the oral formulation is not readily absorbed from the gastrointestinal tract. Vancomycin-intermediate
susceptible and vancomycin-resistant strains of *S. aureus* have been reported. Even in patients with vancomycin-susceptible MRSA, there have been reports of treatment failure with vancomycin, which is thought to be because of heterogeneous subpopulations with varying susceptibility to vancomycin (Bamberger 2005). Localized impetigo may be treated topically with mupirocin (Bactroban). Because of the increasing concern of community-acquired MRSA, purulent lesions that require systemic therapy should be cultured so that antimicrobial susceptibility testing can be performed, and initial empiric treatment should consider the local prevalence of community-acquired MRSA (Osamu 2005).

In this case, we used Cefotaxime and Cloxacillin intravenously because the severe form of SSSS and we suspected secondary infection. Severe from require more aggressive treatment with intravenous anti staphylococcal antibiotics and extra care of denuded skin to prevent secondary infections and fluid losses to maintain body temperature, especially in neonates (Darmstadt 2000; Ladhani 1999). Additional broad-spectrum antibiotics should be considered if secondary skin infection is suspected, and they should cover *Pseudomonas sp* (Darmstadt 2000; Prince 1992). We give the patient cefotaxime as a broad-spectrum antibiotic. The Dermatology and Venereal disease Department advised to give normal saline for compress moist lesion to topical treatment. After 7th hospitalization, according with the bacteria sensitivity test revealed that sensitive to MSSA antibiotics, and CRP test was negative we continued with antibiotic orally cloxacillin 55 mg three times daily.

The prognosis of SSSS in children who are appropriately treated is good, with mortality less than 5% (Darmstadt 2000; Makhoul 2001; Ladhani 1999). A high index of suspicion, prompt diagnosis, implementation of infection control measures, and early institution of treatment all are indispensible steps for halting the expansion of SSSS in the infant, avoiding complications and mortality, and preventing the spread of disease to other infants (Darmstadt 2000; Makhoul 2001; Todd 2005).

The disease might be associated with significant complications and mortality (Todd 2005). Possible Complications are severe bloodstream infection (sepsis); fluid regulation problems causing dehydration or electrolyte imbalance, poor temperature control (in young infants) and can spread to deeper skin infection (cellulites), pneumonia, osteomyelitis (Ladhani 1998; Makhoul 2001; Resnick 2003). Mortality rate from SSSS in children is very low (1-5%), unless associated sepsis or an underlying serious medical condition exists. Mortality rate in adults is higher (as high as 20-30%). Significant morbidity can result from hematological or local spread of infection. According to a recent review, the higher incidence of generalized SSSS in children may be due to less-efficient renal clearance of the toxin and to immunological immaturity (low anti-ET antibody titers) (Ladhani 1999; Resnick 2003; Todd 2005).

While the mortality rate from SSSS in neonates and children is low, it is important to make a correct diagnosis. Differential diagnosis of generalized SSSS in neonates is few include drug or virus mediated TEN (Toxic Epidermal Nectrolysis), burns, epidermolysis bullosa, erysipelas (Peters 1998; Resnick 2003). Chemical burns from petrol, paraffin, boric acid, ethylene; acryl nitrite fumigant spray must also be excluded (Resnick 2003). Erysipelas is rare in children; the onset is sudden with high fever, often with convulsions. Any part of the body may be affected, and the skin becomes livid and edematous and is very tender. Vesicles or bullae are multiple, tense and not readily ruptured. Histologically, the epidermis is normal in the early stages. Electron microscope showed dermal infiltrate that does not occur in SSSS (Peters 1998).

In the second-degree burns, there are vesicles and blisters on an erythematous base. The outline is irregular or follows the line of clothing. The area of affected skin does not change. Prodromal and mucous membrane change are absent (Peters 1998). Chemical burns may also cause difficulties when there is no history available, but Nikolsky’s sign is negative, the areas affected will have an irregular edge, and there will be signs of dermal damage (Peters 1998). The initial generalized erythematous rash seen in SSSS may be confused with streptococcal scarlet fever, but the very tender skin, erythematous maculopapular rash which has texture of sandpaper, the forehead and cheeks are flushed (Ladhani 1998), exfoliation, lack of a strawberry tongue, and possible perioral and periorbital crusting seen in SSSS should confirm the diagnosis (Resnick 2003). Possible complication in SSSS were severe bloodstream infection (sepsis), fluid regulation problems causing dehydration or electrolyte imbalance, poor temperature control (in young infant), spread to deeper skin infection (cellulitis) (Peters 1998).

In this case, although the patient was weak and poor feeding since four days before admission, there was no sign of dehydration. The other complication was not noted. Fortunately, there was no spreading of the disease to other babies. Any patient developing SSSS should be immediately isolated. SSSS can provoke serious outbreaks of the disease in the Neonatal Intensive Care (Todd 2005). Simple measures such as
hand-washing, minimal handling, cleaning objects such as stethoscopes and thermometers before use, and avoiding unnecessary invasive catheterization all contribute to prevention of cross-infection (Darmstadt 2000; Pollack 1996). Clinicians should be aware of possible outbreak, even if patients prevent with infection after hospital discharge (Currant 1980). Until now, the outbreak of the disease still did not happen in our Neonatal Intermediate ward.

CONCLUSION

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. We suggest the patient suffered from generalized forms of SSSS with secondary infection of _Pseudomonas sp_. Staphylococcal scalded skin syndrome (SSSS) is the clinical term used for a spectrum blisterng skin disease induced by the exfoliative (epidemolytic) toxin (ET) of _Staphylococcus aureus_. Treatment of antibiotics and topical wound therapy provided good improvements in this case. The prognosis in this case was excellent. A full recovery is expected.

REFERENCES


