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LAPORAN KASUS

Staphylococcal Scalded Skin Syndrome in Healthy Infant

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Abstrak

Staphylococcal scalded skin syndrome (SSSS) menggambarkan spektrum kelainan kulit dengan gambaran kulit melepuh superfisial yang disebabkan oleh toksin eksfoliatif *Staphylococcus aureus* yang dapat berasal dari fokus infeksi dari konjungtivitis purulen, otitis media atau infeksi nasofaring. Kelainan diawali dengan demam, iritabel, dan makula eritema di kulit berwarna kemerahan, nyeri dan menyeluruh dan berkembang menjadi erupsi dan melepuh dalam 24 sampai 48 jam. Diagnosis harus dibedakan dari penyakit kulit lainnya seperti nekrolisis epidermal toksik, epidermolisis bulosa, eritema multiforme, impetigo akibat Streptokokus atau Listeriosis dan luka bakar, yang dapat mempunyai gejala yang sama. Prognosis SSSS pada anak biasanya baik dengan angka kematian kurang dari 5%. Kasus merupakan serang bayi laki-laki usia 3 bulan yang dirawat di ruang Anak RS M. Djamil dengan keluhan utama kulit dan mengelupas sejak 2 hari sebelum rawatan. Hasil kultur kulit, mata dan hidung adalah *Staphylococcus aureus* dan pasien diberikan terapi ampisilin dan gentamisin selama tujuh hari.

Kata kunci: *Staphylococcal scalded skin syndrome;bayi;infeksi*

Abstract

Staphylococcal scalded skin syndrome (SSSS) describes a spectrum of superficial blistering skin disorders caused by the exfoliative toxins of *Staphylococcus aureus* that 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, paint, orange-red, macular erythema with cutaneous tenderness, and the rash progress from scarlatiniform to a blistering eruption in 24 to 48 hours. A diagnosis must distinguish SSSS from other skin diseases, such as toxic epidermal necrolysis, epidermolysis bullosa, bullous erythema multiforme, Streptococcal impetigo or listeriosis and thermal or chemical burns, all of which can manifest with similar symptoms. The prognosis of SSSS in children who are appropriately treated is good, with a mortality of less than 5%. A case was a three months old boy hospitalized in Pediatric ward M. Djamil hospital with chief complain redness and peeling of the skin sinces 2 days before hospitalized. Culture of the skin, eyes and nose was *Staphylococcus aureus*, and patients was given ampicillin and gentamycin for seven days.

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Keywords: *Staphylococcal scalded skin syndrome;infant;infection*

INTRODUCTION

Staphylococcal scalded skin syndrome (SSSS) describes a spectrum of superficial blistering skin disorders caused by the exfoliative toxins (also known as epidermolytic toxins, epidermolysins and exfoliatins) of *Staphylococcus aureus*. Its severity varies from localized blisters to generalized exfoliation affecting the entire body surface.[1] SSSS commonly occurs in neonates and young children less than 6 years of age. It is due to lack of antitoxin antibodies and due to poor renal excretion of the toxins. Currently, the incidence of this disease is increasing in all ages. Its resistance to conventional antibiotic treatment is also a new reality.[2,3] The incidence is estimated to be 1 to 1.4 cases per million inhabitants per year. Internationally, the incidence is higher in developing countries.[4]

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. Within 24 to 48 hours, the rash progress from scarlatiniform to a blistering eruption.[5] A diagnosis must distinguish SSSS from other skin diseases, such as toxic epidermal necrolysis, epidermolysis bullosa, bullous erythema multiforme, Streptococcal impetigo or listeriosis and thermal or chemical burns, all of which can manifest with similar symptoms.[6]

The prognosis of SSSS in children who are appropriately treated is good, with a mortality of less than 5%. Nevertheless, some children will die despite antibiotic therapy. In contrast, mortality rates in adults are very high, usually due to underlying diseases, despite aggressive treatment.[7] We describe a case of an infant, 3 months of age with SSSS and discuss relevant pathology, clinical issue, and management

CASE REPORT

A three months old boy was hospitalized in Pediatric ward M. Djamil hospital with chief complain redness and peeling of the skin since 2 days before hospitalized. Alloanamnesis from his mother with present illness history: redness and purulent discharge on both of eyes since 3 days before hospitalized. Patient suffer from low grade fever since 3 days ago, intermittent, no shivering and no seizure. Redness around mouth, nose, behind ears and neck since 2 days ago, and spread into extensive area of the body, become blistering then peeling starts from the mouth since 1 day before hospitalized. Patient looked irritable and difficult to breastfed since 2 days before hospitalized. He breastfeds on demand since birth, every 2 to 3 hours for 15 to 20 minutes and

never got formula milk before. There was no vomit, no breathlessness, cold and cough. There was no infection and excoriation on the mother's breast and no history of applying traditional medicines on his skin. There was no history of skin infection and consuming any drugs before. Urination and defecation was still normal. Patient had been brought to midwife 2 days before hospitalization and got paracetamol and amoxicillin.

Patient never suffered from disease like this before and there was no family history of similar skin lesions or conjunctivitis. Patient was the third sibling of 3 children, sectio caesarean delivery due to premature rupture of membrane (18 hours), aterm with birth weight 3850 grams and birth length 52 cms, directly crying. Basic immunization was completed. History of growth and development was normal. Hygiene and sanitation was good. His mother was 37 years old, graduated from senior high school, a housewife. His father was 40 years old, graduated from senior high school, work as an entrepreneur. Patient has 2 older brother, the oldest was 11 years old and the second was 6 years old. Patient and her family live in their own home, a permanent house with good hygiene and sanitation.

Patient look moderately ill, alert, blood pressure 80/50 mmHg, heart rate 126 times per minute, respiratory rate 34 times per minute, body temperature 38,5 ^{o}C, body weight 6,5 kgs, body height 62 cms, weight for age was 0-(+2) SD (Z score), height for age was 0-(+2) SD (Z score), weight for height was 0-(+2) SD (Z score), well nourished, there was no anemic, no icteric, no edema and no cyanotic. Skin was warm, turgor was good. There was superficial skin exfoliation over the forehead, mouth, nose, ear, neck, axilla, buttock, upper and lower limb, exposing a red moist surface with yellowish squama around mouth, nose, eyes, ear, and neck, mucous was good, Nicolsky's sign was positive. There was no regional lymph enlargement.

Head was round and symmetric, head circumference was 40 cms (normocephal), major fontanela was not sunken, eyes were not sunken, conjunctiva was not anemic, sclera was not icteric, purulent secret with edema and hyperemic on both of eyes, there were no injection on conjunctivae, pupil isochors with diameter 2mm/2mm, light reflex was positive normal. Throat was not hyperemic, oral mucous was wet, not hyperemic, there was no oral trush. There was no neck rigidity. The chest was symmetric, there was no retraction, heart: regular rhythm, no murmur, lung: bronchovesicular, no rales, no wheezing. There was no distension at abdominal, liver and spleen was not palpable, genitalia: puberty state $A_1P_1G_1$. Extremities was warm with good perfusion, there was no edema.

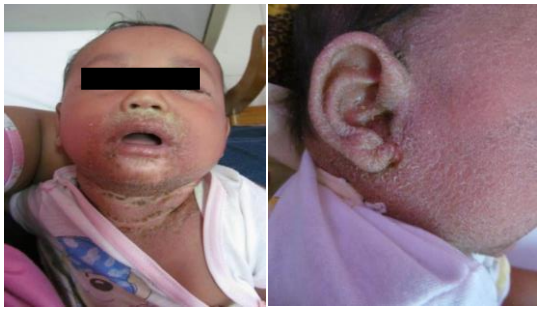

Figure 1. Baby F, at first admission. There was superficial skin exfoliation over the forehead, mouth, nose, ear, neck, axilla, buttock, upper and lower limb, exposing a red moist surface with yellowish squama around mouth, nose, eyes, ear, and neck.

Laboratory finding:
Haemoglobin: 11,5 gr/dl; leucocyte: 21.700/mm^3, differential count: 0/0/0/58/42/0, platelet: 370.000/mm^3. Random blood glucose was 98 mg/dl, sodium was 137 mmol/L, potassium was 3,7 mmol/L. Urinalisis: albumin (-), reduction (-), keton (-), leucocyte 1-2/LPB, erytocyte 1-2/LPB.

Patient was diagnosed with *Staphylococcal scalded skin syndrome* and conjunctivitis ocular dextra et sinistra and was given treatment breastfeed on demand, fluid infusion with KaEN 1B 105 cc/kgBW/24 hours, ampicillin 4x250 mg IV, gentamycin 2x16 mg IV,paracetamol 75 mg (T >38,5 °C), compress the skin with normal saline and planned to do culture of swab on conjunctivae, skin, nasal and throat, and blood culture.

Patient has been consulted to dermatology and venereology department with result *staphylococcal scalded skin syndrome*, and consultation to ophthalmology department was suspected conjunctivitis ocular dextra et sinistra. Patient was given ofloxacin eye drop and suggest to do culture of secret from the eyes.

Follow up 2nd day of hospitalization

Patient still fever, not high, intermittent, there was no cold, cough, and breathlessness. Patient still difficult to breastfed. Defecation and urination were normal. Patient still erythematic, desquamation around mouth and neck still present with yellowish squama, there was new lesion on the chest.

Patient look moderately ill, heart rate 110 times per minute, respiratory rate 36 times per minute, body temperature 37,8 ^{o}C. Skin was warm, turgor was good. There was superficial skin exfoliation over the forehead, neck, chest, axilla, buttock, upper and lower limb, exposing a red moist surface with yellowish squama around mouth, nose, eyes, behind ears, neck, and chest. Eyes: not hyperemic, secret was decreased, no edema. The heart and lung were normal. There was no distension at abdominal. Extremities was warm with good perfusion, there was no edema. Therapy were continued, and the patient got lanolin anhydrous ointments, applied 30 minutes before bathing.

3rd until 5th day of hospitalization

There was no fever, cold, cough, and breathlessness. Breastfeeding was good. Defecation and urination were normal. Desquamation around mouth was decrease, squama decrease, there was no new lesion on the skin. Patient look

moderately ill, heart rate 112 times per minute, respiratory rate 32 times per minute, body temperature 37 ^{o}C. Skin was warm, turgor was good. There was superficial skin exfoliation over the forehead, neck, chest, axilla, buttock, upper and lower limb, squama on mouth, neck and behid ears. The eyes: not hyperemic, no secret and edema. The heart and lung were normal. There was no distension at abdominal. Extremities was warm with good perfusion, there was no edema. Fluid infusion has been stopped, antibiotic therapy were continued.

Culture of the skin: *Staphylococcus aureus*, resistant to ampicillin and intermediate to gentamycin. Culture of swab from eyes: *Staphylococcus aureus*, sensitive to ampicillin and gentamycin. Culture of swab from nasopharyngeal: *Klebsiella spp*, sensitive to gentamycin and resistant to ampicillin. Culture of swab from nose: *Staphylococcus aureus*, sensitive to ampicillin and gentamycin. Blood culture was negative. Antibiotic was continued for seven days.

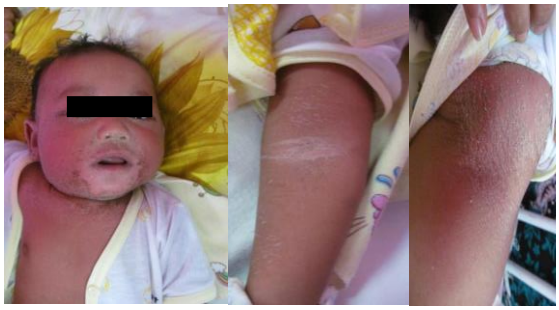

Figure 2. Baby F, at 5th of hospitalization. Skin exfoliation was decrease than before. There was no other new skin lession.

6th until 8th day of hospitalization

There was no fever, cold, cough, and breathlessness. Breastfed was good. Defecation and urination were normal. Desquamation on skin was decreased, there was no squama and no new lesion on the skin. Patient look moderately ill, heart rate 118 times per minute, respiratory rate 32 times per minute, body temperature 37,2 ^{o}C. Skin was warm, turgor was good. There was no new skin lession, there was skin exfoliation over the axilla, upper and lower limb, no squama. The heart and lung were normal. There was no distension at abdominal. Extremities was warm with good perfusion, there was no edema. Therapy were continued until seven days, and patient was discharge at eighth day. The parents and brother was suggested to do culture of swab from nose and throat, but they refused because of financial problem.

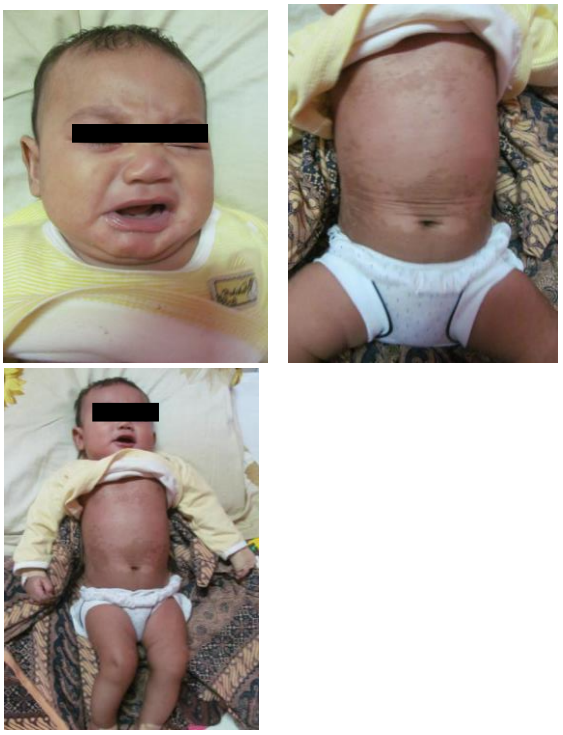

Figure 3. Baby F at discharge. There was no new skin lession, there was skin exfoliation over axilla, upper and lower limb.

DISCUSSION

Staphylococcal scalded skin syndrome is an acute epidermolysis caused by a staphylococcal toxin. SSSS usually presents with sore throat and purulent conjunctivitis as the source of infection. In neonates, the umbilical cord is often the source of infection. Within 48 hours the patient develops fever, malaise and extremely tender erythematous area of the face, neck, axilla and perineum then develop into large flaccid blisters and rapidly rupture, resulting in large areas of erosion surrounded by epidermal patches.[19,21] In this case, patient suffer from purulent discharge and low grade fever since 3 days before hospitalized accompanied with redness around mouth and spread into extensive area of the body. Peeling starts from the mouth since 1 day before hospitalized. Patient looked irritable and difficult to breastfed.

SSSS is primarily a disease of neonate and children, and clinical feature vary from localized blisters to severe exfoliation affecting over 90% of the entire body surface. In localized form, 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.[5] In generalized form, SSSS usually starts with a swift onset of painful, tender and red skin attenuated in flexural and periorificial areas. After 24 to 48 hours, flaccid blisters and erosions develop and large areas of the overlying epidermis loosen and peel like a scald which can be extended.[22]

Physical examination on admission revealed the skin lesion initially appeared as exfoliation most all over the body, exposing a red moist surface with perioral desquamation and also in neck. Perioral crusting was seen, there is no mucosal involvement, Nicolsky sign was positive. Nicolsky's sign is a clinical dermatologic sign in which blisters spread easily upon application of horizontal, tangential pressure to the skin. A positive Nicolsky's sign signifies a separation of epithelial cells either from one another or from the basement membrane.[18]

Several risk factors of SSSS have been suggested: poor renal clearance of the toxins, low antibody status, immunosuppression with immunosuppressive drugs and malignancy.[7,8] In this case, patient was well-nourished, got breastfed and never suffer from any illness or skin lesion before. Jeong Do et al (2010) found that most of patients with SSSS were preschool-aged children who were relatively healthy and they were not immunocompromised, and they had no chronic illness or renal disease.[8] Immunosuppression has been shown to increase the risk of developing generalized SSSS, while maternal antibodies have been shown to protect newborns. Anti-toxin antibodies were found in 80% of infant cord blood samples.

This finding reflect passive maternal antibodies, which, in turn, may protect newborn. The level of antibody fell to 30% in 3 and 24 months of age and then increase steadily to 50% in those older than 10 years and to 91% in those oleder than 40 years.[4,7]

Epidemiological studies looking at antitoxin antibodies suggest that, in the localized form, *S. aureus* enters the skin through a break in the skin barrier (such as grazes, atopic dermatitis, or chickenpox) and produces the toxin locally, but hematogenous spread is limited by the presence of antitoxin antibodies. In the generalized form, however, the toxin is usually produced at a distant site. This may be a colonization site (such as the nares, eye, umbilicus, groin or wound sites) or an infective site (such as pneumonia, osteomyelitis or endocarditis). Lack of protective antibodies allows the toxin to spread through the bloodstream to reach the mid-epidermis via dermal capillaries to produce generalized exfoliation.[7,8]

Renal function is also an important determinant in developing SSSS and may partly explain why young infant may be more susceptible. Poor renal clearance of the toxins by infant below 3 months of age and adult with impaired renal function is a major risk factor for developing SSSS. One-third of ETA would excrete within 3 hours compared to 1/15 of ETA within 3 hours in neonates and infants. As consequence, toxin levels reach a higher peak in newborn and decline slowly.[7,8]

Staphylococci may be transmitted by multiple routes, including contact with infected persons, contact with asymptomatic carriers, airborne spread, and contact with contaminated object. The disease usually follows a localized infection of the upper respiratory tract, inner ear, conjunctiva or umbilical stump. Of these, contact with a person with staphylococcal lesions appears to be particularly important in the spread of staphylococci.[4] In this case, the disease starts with the inflammation of the conjunctivae (conjunctivitis) which is a *Staphylococcus* commensal site like umbilicus and axilla. The history of previous skin infection or contact with skin-infected patients in which *S.aureus* infection was denied.

Most cases of SSSS are diagnosed on clinical grounds, supported by the presence of *S. aureus* in nasal, conjunctiva, pharyngeal, umbilical or other swabs. Detection of ET is required for diagnosis of SSSS.[19] Other laboratory investigations are required only if the clinical findings are equivocal or when outbreaks occur. Because the condition is the result of the exotoxins which may be produced by staphylococci at a distant site, the blister fluid in generalized SSSS tends to be sterile, although a positive culture can be found in 3% SSSS in children.[14] In this case, diagnosis based on clinical manifestation of redness and blistering of the skin that suddenly rupture resulting large erythematous area covering by yellowish squama with positive Nicolsky's sign, and supported with the

presence of *S. aureus* on skin, nasal, and conjunctiva.

In case where the diagnosis remains uncertain, the most useful investigation remains a skin biopsy, which is not practical in children. In SSSS, the biopsy would show mid-epidermal splitting at the level of the zona granulose, without cytolysis, cell necrosis or any inflammatory reaction. The biopsy is particularly useful in distinguishing SSSS from erythema multiforme/toxic epidermal necrolysis, where splitting occurs at the dermo-epidermal level.[1] In this case, we didn't perform skin biopsy since the clinical manifestation was clear and support with the presence of *S. aureus*.

The main differential diagnosis is made with toxic epidermal necrolysis (TEN), a severe variant of erythema multiforme usually related to drugs. The latter is characterized by dermal-epidermal separation, different from the separation in the granular layer of the epidermis seen in SSSS, as well as by an intense inflammatory infiltrate.[21] Unlike SSSS, the mucous membrane is almost always affected, causing extensive erosions in mouth, conjunctiva, trachea, bronchi, esophagus and genitalia.[7] Other differential diagnosis of exfoliative skin lesions includes bullous impetigo, epidermolysis bullosa, and herpetic lesions.[13] In patients with bullous impetigo, the toxin produces blisters locally at the site of infection, whereas in scalded skin syndrome, it circulates throughout the body, causing blisters at sites distant from the infection.[23]

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. Particular care must be given to pain management since the lesions are often very painful, and patients with severe peri-oral involvement may require short-term intravenous nutrition. Young infants require careful observation for dehydration, hypothermia and secondary infections.[1,24] In this case, although patient was irritable and poor feeding since 2 days before admission, there was no sign of dehydration, and electrolyte imbalance. Patient got fluid therapy intravenously for 2 days because of difficult to breastfed. The other complication was also not noted.

Intravenous antibiotics are administered to decrease the Staphylococcal burden. Early administration of intravenous antibiotics, such as cloxacillin or a cephalosporin, results in a good recovery within seven to ten days. Intravenous penicillinase-resistant, anti-staphylococcal antibiotics, supportive skin care, as well as fluid and electrolyte management in the setting of disrupted skin barrier function, will ensure rapid recovery.[24] Patient got combination of ampicillin and aminogligocydes intravenously and this antibiotics was continued until 7 days, since the culture of swab from skin, eyes

and nose were *Staphylococcus aureus*, sensitive to ampicillin and gentamycin. For most skin and skin structure infections, empiric antibacterial therapy should be directed against the most likely pathogen. In most of the patients, antibiotics were initiated before the culture and sensitivity report and as the children responded to the same, antibiotics were continued.[25,26]

Penicillin remains the drug of choice if the isolate is sensitive to it. Antimicrobial combinations have been used to increase bactericidal activity or to prevent the development of antimicrobial resistance. The combination of beta lactams and aminoglycocides increases bacterial killing in vitro. Ampicillin, commonly known as a broad-spectrum penicillin, is a type of aminopenicillin, a semisynthetic group of β-lactams that were developed for effectiveness against both gram-negative and gram-positive organisms. The mechanisms of action of ampicillin are interference with cell wall synthesis by attachment to penicillin-binding proteins (PBPs), inhibition of cell wall peptidoglycan synthesis and inactivation of inhibitors to autolytic enzymes.[12]

Because the condition is toxin mediated, exfoliation usually continues 24 to 48 hours after starting appropriate antibiotic treatment, although new lesions are uncommon after this period. There have been several cases of methicillin-resistant S. aureus causing SSSS and this must be considered if the patient is not responding to empiric antibiotic treatment. In most cases, the skin lesions heal rapidly over the next 7 to 10 days and because of exfoliation is superficial, scarring is rare.[1,8] In this case, the new lesion still present on the second day, and exfoliation still found until patient was discharge.

The use of topical agents to treat superficial skin infections can limit exposure to systemic agents and deliver high drug concentrations to the site of infection. In this case, patient only got lanolin anhydrous and never got topical antibiotic. Antibiotic or antiseptic ointments should also not applied on large areas of the skin in generalized SSSS because of systemic toxicity by absorption of antibiotics, especially in neonates or infants.[21] It is also not effective, since the focus of infection is usually not known and often distant from the site of blistering.[17,21]

Skin care generally consists of bland emollients (petroleum jelly) and minimal handling of patients. Muporicin can be used at foci of infection. Gentle cleansing may help prevent secondary infection. Removal of dried skin with bathing is not necessary initially when the skin is too tender to touch but is used an adjunct later.[17] Severe periorbital lesion was managed with eye ointments containing antibiotics. Patient got lanolin anhydrous as emollients and used 30 minutes before bathing.

The prognosis of SSSS is usually good in children and is more guarded in infants. Complications include sepsis, secondary infections, electrolyte imbalance, fluid losses and hypothermia. The mortality rate in SSSS is less than 5%. In many cases,

patients respond rapidly to treatment, with complete recovery within 2 to 3 weeks. Because the cleavage plane in SSSS is superficial, lesions heal without scarring.[17] In this case, under treatment, erythema and exfoliation decrease within 7 days and was completely healed after 2 weeks of follow up, with the disappearance of the inflammatory syndrome and total body surface restored.

Any patient developing SSSS should be immediately isolated. Anyone else at risk of developing SSSS who may have been exposed to ET-producing *S. aureus* strains should be rapidly identified. Unfortunately, a rapid diagnostic kit for SSSS is not yet available. Microbiology investigation from nose and throat take longer time and couldn't determine ET-producing *S. aureus* strains. There was lack evidence about administration of prophylactic antistaphylococcal antibiotics.[14,22]

CONCLUSSION

SSSS is more commonly seen in neonates and young children less than 6 years of age. It is due to lack of antitoxin antibodies and due to poor renal excretion of the toxins. The clinical features of SSSS vary along a spectrum. The localized form often presents with a few localized fragile superficial blisters filled with colorless to purulent fluid. In generalized form patients often initially present with fever and erythema, with constitutional symptom such as malaise, poor feeding and irritability, followed by large superficial blisters that quickly rupture, particularly in areas of friction. The diagnosis of SSSS is mainly based on clinical appearance, and it is confirmed by the isolation of S. aureus from foci of infection, such as from the nose, conjunctivae, umbilicus, or nasopharynx, or the exfoliative toxin (ET) and/or the histopathological findings.[3,17] Nikolsky's sign is a clinical dermatologic sign in which blisters spread easily upon application of horizontal, tangential pressure to the skin. The treatment of SSSS should aim at eradicating S. aureus, which usually requires intravenous antibiotics. Penicillin remains the drug of choice if the isolate is sensitive to it.

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