Volume 11 No. 2, June 2015

Case Report

Staphylococcal scalded skin syndrome from a trivial injury

Ka-Ka SIU 邵嘉嘉,* Eric Yat-Tung CHAN 陳日東, Daniel Kwok-Keung NG 吳國強
Department of Paediatrics, Kwong Wah Hospital, Hong Kong

Abstract
Staphylococcal scalded skin syndrome (SSSS) is a life-threatening toxin-mediated condition caused by staphylococcal aureus. It could be a complication after trivial injury. In this report we describe a 3-year-old girl who suffered from SSSS after minor laceration of right finger and lip.

Keywords: Staphylococcal scalded skin syndrome, Staphylococcal skin infection, Toxin-shock

Case

A 34-month-old girl had acute onset of widespread blisters with yellowish discharge over the neck, ear, trunk and thigh. There was no fever. She was seen by doctor in Mainland China and oral 1st generation cephalosporin was given. She had history of slipped and fell injury in a restaurant with minor laceration of right ring finger and lower lip (Figure 1), 3 days before the consultation.

Her condition further worsened and she attended our unit 2 days after the consultation. She had fever, 38.5 degree Celsius. Blood pressure was 113/70 mmHg. Heart rate was 90/minute. Capillary refill was instant. Bullae and blisters were noted over chest, back, dorsum of hand, neck, abdomen (Figure 2). Nikolsky sign was positive. Mucosa was intact. Area of skin involvement was 5%. She was transferred to intensive care unit on next day due to extensive bullae formation and skin peeling involving 9% of total body surface area. Investigations showed high white cell counts ($25.9x10^{x9}$/L) with neutrophilia ($15.6x10^{x9}$/L). C-reactive protein was elevated (30 g/dL). Skin biopsy showed blister, with level of separation in the stratum corneum. No subepidermal separation was noted. Gram stain shows few clumps of gram positive cocci. Overall picture favor staphylococcal scalded skin syndrome.

Initially, she was put on intravenous clindamycin for anti-toxin effect as well as vancomycin for coverage of methicillin-resistant staphylococcus infection. She had tachycardia with normal blood pressure, compensated metabolic acidosis on admission to paediatric intensive care unit (PICU). There was no fever. Bedside echocardiogram showed normal cardiac index (CI) and contractility. Stroke volume index and corrected flow time were low. Tachycardia was related to hypovolaemia. One dose of normal saline bolus was given. Parkland Formula was referred for fluid replacement in first 48 hours when there were active bullae formation and skin peeling. Besides intravenous fluid, nutritional support with high-energy milk formula on top of solid diet up to 110% of estimate energy requirement was given since day 2. Adequate fluid rehydration was reflected by satisfactory urine output and resolution of metabolic acidosis. However, she developed tachycardia on day 2 again. This time, the systemic vascular resistance index was low which suggested early distributive shock. Noradrenaline and intravenous immunoglobulin was started for the toxin-mediated shock. Meropenem was added for possible secondary skin infection. Systemic vascular resistance index (SVRI) and CI normalised; tachycardia resolved as noradrenaline infusion up to 0.18 mcg/kg/min. Skin healing started since day 3. Maintenance fluid was reduced since day 3 as blistering subsided and skin healing started. Chest X-ray showed air bronchogram over right lower zone, compatible with pneumonia. Other possible causes were line infection and persistent effect of staphylococcal toxin. Noradrenaline weaned off since day 4. Fever subsided since day 7. Skin swab yielded methicillin-sensitive staphylococcal aureus. Blood culture was sterile. Antibiotic was changed to intravenous cloxacillin, and later switched to oral amoxicillin and clavulanate potassium for total 14 days for coverage of concurrent pneumonia. She made an uneventful recovery with intact skin upon discharge on day 10.

*Author to whom correspondence should be addressed.
Email: skk053@ha.org.hk

Discussion

Staphylococcal scalded skin syndrome (SSSS) has an estimated incidence of 0.56 cases per year per million inhabitants from 1997 to 2007 in France.[1] The condition is caused by strains of Staphylococcal aureus that release serine protease exfoliate toxins (exfoliative toxin A and B) that cleave desmoglein 1 complex, in the superficial epidermis, resulting in destruction of cell-cell adhesion and creating blistering and denuding of the skin. Gentle pressure applied to the skin results in separation of the upper epidermis and wrinkling of the skin (Nikolsky's sign). SSSS in children begins with erythematous areas, which rapidly progress to generalised erythema. This is worse in flexures. Large bullae then form and rupture. This causes large area of epidermis to slough off resulting in scalded skin appearance.[2]

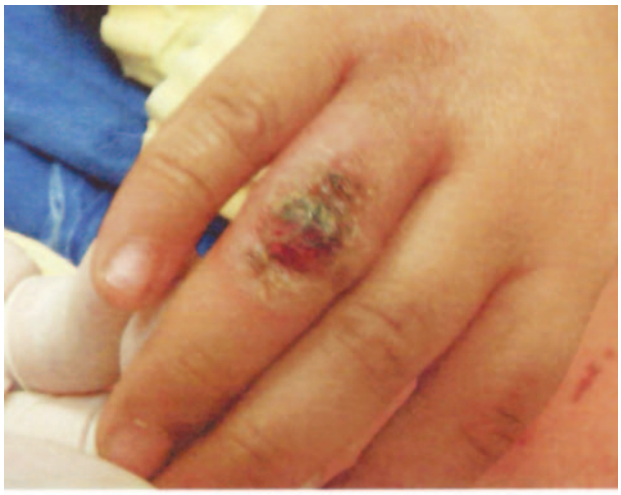

Figure 1. Staphylococcal scalded skin syndrome as a result of minor laceration of finger.

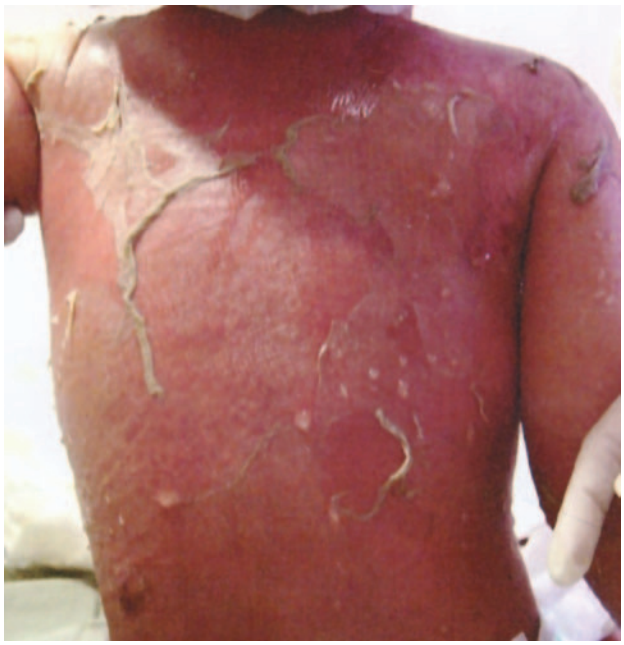

Figure 2. Extensive skin involvement with bullae formation over the trunk.

SSSS may initially resemble other blistering disorders, including toxic epidermal necrolysis (TENS), staphylococcal toxic-shock syndrome, Steven Johnson syndrome (SJS), enterovirus infection. Clinical feature and skin biopsy will differentiate among different causes. Unlike TENS and SJS, mucous membranes are not involved in SSSS but may appear hyperemic. Skin biopsy in SSSS shows superficial intraepidermal cleavage under the stratum corneum; while biopsy specimens of TEN reveal a subepidermal cleavage plane and epidermal necrosis.

Treatment of SSSS includes supportive measures with appropriate fluid and nutrition, antibiotics, skin care and pain control.

For fluid regime, fluid resuscitation in first 48 hours of admission in SSSS is crucial. As there are reports of fluid overload with excessive fluid administration,[2] probably due to inappropriate vasopressin secretion occurs in patients with burns,[3] judicious use of intravenous fluids with close monitoring of fluid status by clinical parameters (heart rate, blood pressure, urine output, blood gas and cardiac indices) is recommended. Only isotonic solutions should be used because of the risk of hyponatraemia.[2] The cause of shock could be assessed by non-invasive monitoring of cardiac indices, which may interplay in SSSS. For tachycardia due to septic shock, one would expect a low SVRI and high CI. In contrast, patients with hypovolaemic shock had a high SVRI and reduced CI.

For nutritional support, enteral nutrition should be started within 48 hours of PICU admission, in order to limit protein catabolism, elevated glucose levels and support the hypermetabolic phase which may continue with infection. Enteral feeding can be used in SSSS patient, if they are not in shock and there is no significant mucosal damage (e.g. SJS, TEN). Parental nutrition is indicated if one cannot tolerate enteral feeding or inadequate calories provided by enteral feed. A high energy and protein diet is recommended. At the start,

the aim is to at least achieve basal energy expenditure (BEE)(Table 1). Then gradually step up to meet the daily energy requirement which would be BEE x Stress factor (1.5 for sepsis patient). Daily protein requirement would be recommended daily allowance (RDA) x Stress factor. Once clinically improved and beyond the critical phase (day 7-10 and beyond), the goal will be resumption of growth. The RDA can be referred at this stage (Table 2).

For choice of antibiotics, cloxacillin plus clindamycin for its anti-toxin effect is recommended as empirical treatment for SSSS.[4] Switching to vancomycin should be considered for community-acquired methicillin-resistant staphylococcal aureus if no improvement seen. Alternative choice for MRSA skin infection includes linezolid, which has a bacteriostatic activity against *S. aureus*. It has parental and oral formulations, with good oral bioavailability. A 5-day course of intravenous

Table 1. Estimate basal energy expenditure for weight and sex

Age 1 week to 10 months		Age 11 to 36 months			Age 3 to 16 years		
Weight (kg)	MR (kcal/day)	Weight (kg)	MR (kcal/day)		Weight (kg)	MR (kcal/day)	
	Male or Female		Male	Female		Male	Female
3.5	202	9.0	528	509	15	859	799
4.0	228	9.5	547	528	20	953	898
4.5	252	10.0	566	547	25	1046	996
5.0	278	10.5	586	566	30	1139	1092
5.5	305	11.0	605	586	35	1231	1190
6.0	331	11.5	624	605	40	1325	1289
6.5	358	12.0	643	624	45	1418	1387
7.0	384	12.5	662	646	50	1512	1486
7.5	410	13.0	682	665	55	1606	1584
8.0	437	13.5	701	684	60	1699	1680
8.5	463	14.0	720	703	65	1793	1776
9.0	490	14.5	739	722	70	1886	1874
9.5	514	15.0	758	741	75	1980	1973
10.0	540	15.5	778	760			
10.5	566	16.0	797	782			
11.0	593	16.5	816	802			

Table 2. Recommended dietary allowances for infants and children (once passed the critical phase of illness)

	Age (years)	Weight (kg)	Height (cm)	Cals (kcal/kg)	Prot (gm/kg)	Fluid (ml/kg)
Infants	0.0-0.5	6	60	108	2.2	140-160
	0.5-1.0	9	71	98	1.5	125-145
Children	1-3	13	90	102	1.23	115-125
	4-6	20	112	90	1.2	90-110
	7-10	28	132	70	1.0	70-85
Males	11-14	45	157	55	1.0	70-85
	15-18	66	176	45	0.8	50-60
Females	11-14	46	157	47	1.0	70-85
	15-18	55	163	40	0.8	50-60

immunoglobulin (0.4 g/kg/day) to neutralise the exotoxins, has been reported to be effective in treatment of children.[2,5]

Dressing over the denuded skin is necessary to prevent secondary infection and fasten recovery. Soft silicone primary wound dressing covered by saline-soaked gauze can be used at initial stage.[6] Hydrocolloid dressing may be applied as skin heals.[2] Excessive skin itchiness during skin healing can be controlled with topical emollient. There is no data support the use of topical antibiotics.

Pain control with acetaminophen and opiates, e.g. fentanyl, can be given as needed. Warming is recommended with forced-air warming blankets to keep core temperature at 37-38 degree Celsius in patients with extensive skin involvement.[2] Physiotherapy should be performed to encourage joint mobility.[2,7]

Conclusion

Staphylococcal scalded skin syndrome can arise from trivial injury. A high index of suspicion is needed in making diagnosis and initiate appropriate management. Treatment included antibiotics, exotoxin neutralising agents, careful fluid and electrolytes management, nutrition, wound care, prevention of secondary infection through skin wound, analgesia, temperature control and physiotherapy. Skin healing occurs within 2 weeks without scarring.

References

1. Lamand V, Dauwalder O, Tristan A, Casalegno JS, Meugnier H, Bes M, et al. Epidemiological data of staphylococcal scalded skin syndrome in France from 1997 to 2007 and microbiological characteristics of Staphylococcus aureus associated strains. Clin Microbiol Infect 2012;18(12):E514-21.
2. Blyth M, Estela C, Young AE. Severe staphylococcal scalded-skin syndrome in children. Burns 2008;34(1):98-103.
3. Shirani KZ, Vaughan GM, Robertson GL, Pruitt BA Jr, McManus WF, Stallings RJ, et. al. Inappropriate vasopressin secretion (SIADH) in burned patients. J Trauma 1983;23(3): 217-24.
4. Braunstein I, Wanat KA, Abuabara K, McGowan KL, Yan AC, Treat JR. Antibiotic sensitivity and resistance patterns in pediatric staphylococcal scalded skin syndrome. Pediatr Dermatol 2014;(3):305-8.
5. Takei S, Arora YK, Walker SM. Intravenous immunoglobulin contains specific antibodies inhibitory to activation of T cells by staphylococcal toxin superantigens. J Clin Invest 1993; 91(2):602-7.
6. Greenwood JE, Dunn KW, Davenport PJ. Experience with severe extensive blistering skin disease in a paediatric burn unit. Burns 2000;26(1):82-7.
7. Handler MZ, Schwartz RA.Staphylococcal scalded skin syndrome: diagnosis and management in children and adults. J Eur Acad Dermatol Venereol 2014;28(11):1418-23.