Staphylococcal scalded-skin syndrome The Cleveland Clinic experience[1]

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Staphylococcal scalded-skin syndrome (SSSS) or staphylococcal-induced toxic epidermal necrolysis (TEN) is a severe blistering disease of young children. The causative organism is *Staphylococcus aureus*. We describe the clinical characteristics of this disease in 6 children seen at The Cleveland Clinic Foundation between 1970 and 1979.

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Staphylococcal scalded-skin syndrome (SSSS) or staphylococcal-induced toxic epidermal necrolysis (TEN) is primarily a bullous disease of young children that is usually produced by group 2, coagulase-positive *Staphylococcus aureus*. Between 1970 and 1979, 6 children were hospitalized at The Cleveland Clinic Foundation with the diagnosis of SSSS. Our description is analogous to and patterned after the extensive review by Rasmussen[1] in 1975 from Buffalo Children's Hospital.

Results

In a retrospective review of the years 1970–1979, we identified 6 children who had been hospitalized at the Cleveland Clinic Foundation with the diagnosis of SSSS. All patients had scarlatiniform erythema with or without bullae, subsequent epidermal desquamation, and isolation of coagulase-positive *S. aureus*. We excluded all outpatients and any other children with a clinical presentation and/or diagnosis of erythema multiforme and/or Steven-Johnson syndrome.

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The clinical characteristics of all 6 children are summarized in the *Table*. The prodrome in all patients varied from one to six days with nonspecific symptoms. None of the children was seriously ill. All presented with scarlatiniform erythema on the head, trunk, and extremities; 3 had distinct, flaccid bullae. One child had tender skin. In all cases, the disease was generalized and desquamation developed. Although 3 children were febrile on admission, none had a fever 24 hours later. All patients had at least one positive culture for coagulase-positive *S. aureus*. One patient had phage typing. During hospitalization, each child received antibiotics, intravenously at first, then orally as primary therapy; 2 received penicillin for prodromal symptoms without apparent alteration of the disease process. Other therapy included antihistamines, compresses, lubrication, topical ophthalmologic antibiotics, and topical steroids. No patient received systemic steroids.

Table. Clinical characteristics

	Patient					
	1	2	3	4	5	6
Age	11 mo	2 mo	2 yr	3.5 yr	18 days	3.5 yr
Sex	F	M	F	M	M	M
Color	White	White	White	White	Black	White
Time of disease	July 1970	Nov 1971	Feb 1975	June 1975	Aug 1976	Mar 1979
First symptom	Puffy eyes	Rash	Rash	Head cold	Irritability	Rash
First site of skin disease	Left ear	Perioral area	Abdomen	Neck	Neck	Axilla
Scarlatiniform rash	Yes	Yes	Yes	Yes	Yes	Yes
Vesicles/bullae	Yes	No	No	Yes	Yes	No
Skin tenderness	No	No	Yes	No	No	No
Extent	Generalized	Generalized	Generalized	Generalized	Generalized	Generalized
Desquamation	Yes	Yes	Yes	Yes	Yes	Yes
Temperature on admission, rectal (F)	100	103.8	97.9	99.8	99.6	99
WBC, mm^3	11,600	3,700	12,300	14,400	12,700	7,000
Hemoglobin, g/dl	12.9	12.5	13.5	12.0	14.7	11.7
Skin culture	Negative	S.a.	Negative	Negative	S.a	ND
Throat culture	S.a.	S.a.	S.a.	S.a.	S.a.	S.epid.
Nose culture	ND	ND	ND	S.a.	S.a.	ND
Eye culture	S.a.	ND	ND	S.a.	ND	S. epid.
Blood culture	ND	Negative	Negative	Negative	Negative	Negative
Rectal culture	ND	ND	ND	ND	ND	S.a.
Phage typing	ND	ND	ND	ND	71 29/81	ND
Isolation	Yes	Yes	Yes	Yes	Yes	Yes
Antibiotics						
Intravenous	Oxacillin	Oxacillin	Oxacillin	Oxacillin	Oxacillin	Nafcillin
Oral	Dicloxacillin Cloxacillin	Dicloxacillin	Dicloxacillin	Erythromycin	Dicloxacillin	Dicloxacillin
Steroids (systemic)	No	No	No	No	No	No
Other medication						
Before hospitalization	Penicillin	Penicillin Antihistamines		Caladryl	Topical steroids	Antihistamines Topical steroids Aspirin
After hospitalization	Aspirin	None	Antihistamines	Chloral hydrate	Benadryl	Tylenol
Hospital stay, days	6	7	6	5	7	4
Resolution	Yes	Yes	Yes	Yes	Yes	Yes
Sequelae	None	None	None	None	None	None

ND = not done, S.a. = *Staphylococcus aureus*, S. epid. = *Staphylococcus epidermidis*.

All responded to therapy and were discharged after four to seven days (average stay, 5.8 days) without sequelae or recurrence.

Discussion

Staphylococcal-induced TEN, or SSSS, first described by Van Rittershain,[2] is a severe blistering disease that primarily affects children. The causative organism, *S. aureus*, produces an epidermolytic toxin[3] that is responsible for the bright red skin, superficial vesicles and/or bullae, and secondary desquamation. Erythema may be minimal, but usually begins on the face, especially around the mouth, becomes generalized in three to five days, and then fades promptly. Incomplete forms of the disease exist, and a localized disease may resemble impetigo. SSSS is usually an extensive, generalized disease, but has an excellent prognosis with minimal morbidity and negligible mortality, in contrast to the adult type (drug-induced TEN). The biopsy results of affected skin usually reveal a split in the upper epidermis (subgranular layer). Therapy involves the use of systemic penicillinase-resistant penicillin (e.g., oxacillin, dicloxacillin, nafcillin) followed invariably by complete recovery.

The 6 children hospitalized at The Cleveland Clinic Foundation with the diagnosis of SSSS during a ten-year period represent limited experience with this disease at this institution. Yet, this is not entirely unexpected since the Clinic is only one of several pediatric referral centers in northern Ohio. Nevertheless, our data compare with those previously reported.

All 6 children were younger than six years of age. The disease had no predilection based on the patient's gender. There was only a suggestion of seasonal clustering during the warmer months. As in other studies, most of the children were white (5 of 6).

As expected, all children had a prodrome which was nonspecific and viral-like in nature. No child became seriously ill, and none had any predisposing or associated illness. Bacteriologic cultures in all patients were positive for coagulase-positive *S. aureus*. Most cases of SSSS are produced by group 2 organisms, phage type 71.

Systemic antibiotics remain the therapy of choice for SSSS.[4] Therapy in all 6 children was prompt and involved the use of systemic antibiotics, administered intravenously at first, for a maximum duration of two weeks. No child received systemic corticosteroids, which have no established rationale in the therapy of SSSS and may delay resolution.[4,5] No child received topical corticosteroids during hospitalization, and all received proper skin care. All responded promptly to therapy without apparent sequelae from either the disease or therapy. Fortunately, fatalities from SSSS are uncommon and are usually the result of overwhelming sepsis and/or fluid and electrolyte imbalance.

References

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