A case series of toxic shock syndrome in a low-middle-income country burn service: creating awareness about the lesser known and potentially lethal complication

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Introduction: Toxic shock syndrome (TSS) is a bacterial exotoxin-mediated disease that can be a complication of thermal injury in paediatric patients. This disease is acute in onset, occurring mostly within the first 48 hours after thermal injury, and it progresses rapidly to shock and death if appropriate management is not instituted. This case series describes the clinical diagnosis, course of management and outcome of TSS in a single institution in KwaZulu-Natal to highlight the condition in our setting and create awareness.

Methods: The burn admission database was retrospectively searched for patients with a diagnosis of TSS between January and December 2022. Demographic, injury, laboratory and outcome data were collected.

Results: Four out of 106 paediatric admissions were managed for TSS. The average age was 23 months, with 3 out of 4 children being female. The mechanism was hot water scald in all cases, with percentage total surface area burns between 15% and 30%. All patients survived, with one admission to intensive care and one patient developed acute kidney injury, which resolved by the time of discharge.

Conclusion: Our series demonstrates the typical presentation and laboratory features described in the literature. TSS is a lesser-known complication of burn injuries in young children with a high mortality rate if the diagnosis is missed. Awareness of toxic shock as a diagnosis in a child whose condition deteriorates within the first 48 hours of injury, combined with a treatment protocol, can effectively reduce morbidity and mortality.

Keywords: paediatrics, toxic shock syndrome, burn injury

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Introduction

Toxic shock syndrome (TSS) is a bacterial exotoxin-mediated disease that can be a complication of thermal injury in paediatric patients.[1] This disease is acute in onset, occurring mostly within the first 48 hours after thermal injury, and it progresses rapidly to shock.[2] Young children under 4 years (48 months) of age have limited exposure to the exotoxins produced by *Staphylococcus aureus* and other organisms; therefore, no antibodies are formed against the toxin. This explains why the paediatric population is particularly at risk for developing TSS.[2,3] The first cases were reported in 1985. A systemic literature review revealed 59 toxic shock cases in children with burns in 25 publications from 1985 up to 2016, from 10 high-income countries around the world.[1] There are no reports from low-middle-income countries. This case series will describe the clinical diagnosis, course of management and outcome of four burn-injured patients who developed toxic shock in a single institution in KwaZulu-Natal, South Africa. We aim to highlight the condition in our low-middle-income setting and create awareness about this lesser-known condition which has serious morbidity and mortality if missed.

Setting

The Pietermaritzburg Burn Service (PBS) consists of Greys Hospital (six paediatric and six adult burn beds) and Harry Gwala Regional Hospital (24 burn beds) in KwaZulu-Natal, South Africa. Greys Hospital is the tertiary surgical service for Western KwaZulu-Natal, comprising 3 million inhabitants. Referrals are done through the Vula Medical Referral Application.[4] In 2021, 858 burn referrals were managed. Every referral is assessed by a specialist surgeon and the decision to treat at the referring centre or decision to transfer is made. Immediate transfer is typically for larger surface area burns or patients with severe sepsis. Data of admitted patients are routinely collected electronically at the time of admission and discharge, as well as surgery.

Methods

The admission burns database was retrospectively searched for patients with a diagnosis of TSS between January and December 2022. The diagnosis was made by the burn surgeon at the time based on the clinical picture. Data extracted included clinical and laboratory features typical in TSS, as well as age, gender, total surface area burn, injury mechanism, location and depth of injury. Outcome data collected included mortality, morbidities, particularly acute kidney injury and encephalopathy, intensive care admission, and length of stay.

Results

Of the 106 children admitted to Greys Hospital for burn management in 2022, four were managed for TSS. All these patients were under 4 years of age at the time of admission (average age of 23 months, range 7–46

Table I: Patient, burn characteristics and outcome

	Patient 1	Patient 2	Patient 3	Patient 4
Age (months)	15	7	46	24
Gender	F	F	M	F
TBSA %	15%	17%	30%	17%
Injury mechanism	Hot water	Hot water	Hot water	Hot water
Location of injury	RUL; RLL; LLL	HN; RUL; LUL; B	RUL; LUL; A; B; RLL; LLL	B; RUL; LUL
Depth of injury	15% SP	11%SP; 6% DD	29% SP, 1%DD	17% SP
Length of stay (days)	12	37	34	14
ICU admission	N	Y	N	N
Mortality	N	N	N	N
Morbidities	none	AKI	none	none

F – female, M – male, RUL – right upper limb, LUL – left upper limb, RLL – right lower limb, LLL – left lower limb, B – back, HN – head and neck, A – abdomen, SP – superficial partial, DD – deep dermal, I – indeterminate, ICU – intensive care unit, Y – yes, N – no, AKI – acute kidney injury

Table II: Clinical and laboratory diagnostic criteria for TSS

	Patient 1	Patient 2	Patient 3	Patient 4	Normal values
Clinical criteria					
Fever > 39 °C	Y	Y	N	Y	
Hypotension/shock	Y	Y	Y	Y	
Tachycardia	Y	Y	Y	Y	
Tachypnoea	Y	Y	Y	Y	
GIT manifestations	Y	Y	N	Y	
Irritability	N	Y	N	Y	
Lethargy	Y	Y	N	Y	
Rash	N	N	N	N	
Onset of symptoms (hr)	40	15	48	21	
Laboratory criteria					
Blood cultures	NG	NG	NR	NG	N/A
White cell count	6.3	5.26	5.9	4.44	6–18 x10^9/L
Platelets	438	157	449	475	180–440 x10^9/L
Haemoglobin	12.9	7.9	14.8	11.3	10.7–13.1 g/dL
Albumin	31	11	22	NR	32–47g/L

Y – yes, N – no, GIT – gastroinstestinal tract, hr – hour, NG – no growth, NR – not recorded

months). Three out of four children were female. The mechanism was hot water scald in all cases, with percentage total surface area burns between 15% and 30%. All patients survived, with one admission to intensive care and one patient developed acute kidney injury, which resolved by the time of discharge. The length of stay was 1.2 days per % total body surface area (TBSA) burn, with a range of 0.8 to 2.2 days per % burn. The demographic, injury and outcomes are presented in Table I. TSS was diagnosed within 15 to 48 hours post-injury with the predominant feature of shock and fever. The presence of other features of TSS was variable and is presented in Table II together with the laboratory criteria.

Discussion

TSS is most commonly associated with exotoxins produced by *Staphylococcus aureus,* with TSST-1 (Toxic Shock Syndrome Toxin 1) being the most recognised of these toxins.[3] There are, however, other organisms that produce exotoxins that can cause TSS, like *Streptococcus pyogenes* (Group A Strep), *Pseudomonas* and *Klebsiella*.[1,2,5] Because this is a toxin-mediated disease, bacteraemia is often not present and blood cultures are frequently negative. TSS is diagnosed clinically with typical symptoms, including pyrexia, hypotension, tachycardia, tachypnoea, gastrointestinal disturbances (vomiting and diarrhoea), lethargy, irritability and rash. Importantly, the time of onset of the symptoms from the time of burn is typically within 48 hours. An early diagnosis is important in reducing mortality from TSS; thus, these typical symptoms in a paediatric burn within 48 hours of injury need urgent management.[1] Some laboratory criteria have also been added, but these are retrospective to diagnosing TSS because the initial diagnosis is made clinically. Long-term sequelae from TSS are mostly due to the decreased perfusion associated with shock resulting in acute kidney injury (AKI) and, in some cases, encephalopathies.[1]

Our series demonstrates the typical presentation and laboratory features described in the literature, except for a rash which was not found in any of our patients. All the children were under 2 years old, with

presentation within 2 days, but the burn injury was larger than the less than 10% surface area burn classically described.[5] This is easy to explain. In high-income settings, patients undergo early excision and blood products are commonly used to manage surgical bleeding.[6] Therefore, children with larger burns are likely to receive passive immunity from exposure to blood products at the time of injury, whereas those with smaller burns do not. However, in our setting, access to early excision is not the standard of care and explains the higher surface area injury complicating with TSS in our series.

Our outcomes were favourable compared to reported mortality between 11% and 50%, with only one acute kidney injury and one intensive care admission, and all patients surviving with a reasonable length of stay in our series. One could argue that none of the patients in this series had TSS, especially patient 3. It is a notoriously difficult diagnosis and criteria are controversial. An experienced burn surgeon made each diagnosis in our series. We did not record hyponatraemia or lymphopaenia as part of the assessment. This can increase the likelihood of a diagnosis of TSS and will be used in future.

The incidence of toxic shock in paediatric patients admitted to Greys Hospital over the one-year period was 3.8%. This may seem low, but four patients in one institution in one year is of concern in view of 59 cases in the literature over 20 years. It is unknown what the incidence in our referral area is due to the lack of data at district-level hospitals where patients may develop toxic shock and demise before referral to the burn service. The good outcomes reflect the high index of suspicion in our institution for the diagnosis and an existing treatment protocol. The treatment protocol includes inserting intravenous lines and a urinary catheter if not already present, initiating fluids to treat shock, and administering a beta-lactam antibiotic together with Clindamycin as a protein synthesis inhibitor. The rationale for using fresh frozen plasma (FFP) in treating TSS is to confer passive immunity, although it is not guaranteed.[5] Access to FFP in our setting is challenging and hence not included in our protocol.

The question arises whether we can prevent TSS in paediatric burn injuries. Previously, prophylactic antibiotics were routinely used to prevent wound infection in burns in general, but this has shown no benefit and current international guidelines do not promote the use of prophylactic antibiotics.[7] Although there is limited literature on TSS in burns, Mulgrew et al. specifically did a retrospective comparison of the presence of TSS before and after an institutional protocol change stopping the administration of prophylactic antibiotics.[8] This showed no difference in the incidence of TSS in the two groups. Although this paper is flawed by its retrospective nature and historical control group, it is somewhat reassuring that there was not a major increase in the incidence of TSS. The recommendation remains against the use of prophylactic antibiotics. There are currently no strategies published on the prevention of TSS.

Conclusion

TSS is a lesser-known complication of burn injuries in young children with a high mortality rate if the diagnosis is missed. Awareness of toxic shock as a diagnosis in a child whose condition deteriorates within the first 48 hours of injury, combined with a treatment protocol, can effectively reduce morbidity and mortality. This is the first series of toxic shock described in low-income countries. It is difficult to know whether this reflects a lack of awareness or a lack of reporting. We aimed to highlight the condition in our setting and create awareness about the lesser-known condition to prevent a missed diagnosis with the accompanying serious morbidity and mortality.

Conflict of interest
The authors declare no conflict of interest.

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Ethical approval
This database has class approval granted by the Biomedical Research and Ethics Committee of the University of KwaZulu-Natal (BCA106-14).

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