Toxic Shock Syndrome Originating from the Foot

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The most familiar etiology of toxic shock syndrome (TSS) is that of menstruation and tampon use. Nonmenstrual TSS has been described in all types of wounds including postsurgical, respiratory infection, mucous membrane disruption, burns, and vesicular lesions caused by varicella and shingles. A case of TSS occurring in a diabetic male patient with foot blisters is presented. Early recognition by an infectious disease specialist and appropriate medical management led to complete recovery. There have been no reported cases of Staphylococcus aureus TSS originating in the foot to date. (The Journal of Foot & Ankle Surgery 40(6):411–413, 2001)

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Toxic shock syndrome (TSS) is often confused with septic shock, although the clinical manifestations are vastly different. The hallmark signs of TSS include desquamation of the palms and soles, diffuse erythroderma, conjunctival and pharyngeal hyperemia, muscle injury, rapidly accelerated renal failure, and gastrointestinal symptoms (1). The onset of TSS is abrupt, with clinical signs and symptoms of a fever over 104°F (40°C), chills, malaise, headache, sore throat, myalgias, muscle tenderness, fatigue, vomiting, diarrhea, abdominal pain, and orthostatic dizziness or syncope (2). Within a few hours of onset, a sunburn-like rash develops which may be diffuse or limited to the face, trunk, or extremities. Desquamation does not begin until at least the 5 day of the illness, or, in many cases, much later (3). Prolonged hypotension, interstitial edema, and vascular congestion may result in complications of persistent neuropsychological abnormalities, ischemic organ failure, mild renal failure, rash, and cyanotic arms and legs. Thus, the most important part of nonspecific treatment is aggressive fluid replacement with saline solutions and colloids.

In 1994, at least 42% of reported cases of TSS were nonmenstrual (2). Nonmenstrual TSS has been reported in postsurgical wounds, burns, vesicular lesions secondary to varicella and shingles, respiratory infections, and following mucous membrane disruption (4). The Center for Disease Control and Prevention has shown an incidence of TSS to be roughly one case per 100,000 people in the United States (3). While the overall mortality rate decreased from 10% in 1980 to 2.6% in 1983, it is interesting to note that between 1980 and 1986, the mortality rate for men was 17.1% (5).

We present a case of TSS that originated from a foot infection in a diabetic male. To our knowledge, this is the first reported case of this condition with *Staphylococcus aureus* as the inciting organism in the foot.

Case Report

A 31-year-old white male presented to the Emergency Department with a 2-day history of nausea and vomiting. He also reported fever, chills, lightheadedness, dizziness, and sinus drainage. His medical history was significant for type II diabetes controlled with glipizide. Upon admission, his oral temperature was 104.5°F, pulse 153/min, and respiration 28/min. Orthostatic changes were noted in his blood pressure with readings of 107/61 in the recumbent position and 67/43 in the supine attitude. Pulse oximetry was 98% on room air. There was a fine reticular rash all over his body. Serous blisters were noted on the medial aspect of the left hallux, dorsal, and lateral third digit of the right foot. There was an inflamed nail border and blisters on the dorsal surface on the right hallux. The blisters developed after wearing ill-fitting dress shoes 3 days previously.

The patient was admitted to the hospital for IV hydration with normal saline and hetastarch. Despite almost 20 liters of IV fluids, the patient required vasopressors for blood pressure support. Dopamine was initiated; however,

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the blood pressure remained low and phenylephrine was added. Over the first 4 hours, the patient developed respiratory failure and required intubation.

Initial labs revealed a white blood cell count of 8.2 thou/μl (nl = 4.5–10.0 thou/μl) with 51% segs (nl = 50–70%) and 38% stabs (nl = 2–6%), hematocrit 46.2% (nl = 40–54%), and hemoglobin 15.6 g/dl (nl = 13.5–18.0 g/dl). Platelet count was 126 thou/μl (nl = 140–440 thou/μl), partial thromboplastin time 53.5 s (nl = 19.0–32.0 s), prothrombin time 15.5 s (nl = 11.4–13.4 s), and the INR was 1.6. The disseminated intravascular coagulation laboratory panel was negative. Urinalysis was positive for ketones, WBC esterase, and nitrates. Aerobic and anaerobic cultures were taken from the right hallux and the blister was aspirated. Gram stain of the aspirated material was positive for gram-positive cocci suggestive of *Staphylococcus sp.* Throat and urine cultures were negative for pathogens.

The patient was started on broad-spectrum antibiotic therapy of cefazolin and clindamycin for gram-positive sepsis until culture results were obtained. On hospital day 3, wound culture results were positive for *S. aureus* with B toxin. Repeated blood cultures remained negative throughout hospitalization.

The patient's condition improved by the 7th hospital day. Blood pressure and heart rate normalized and vasopressors were discontinued. His body temperature fluctuated between 100°F and 102°F and remained elevated until hospital day 10. He was extubated on hospital day 12. Desquamation of the palms and soles appeared on hospital day 13. The patient was discharged home on day 15. He failed to follow-up as an outpatient.

The patient's clinical presentation of high fever, diffuse macular erythroderma, an orthostatic drop in blood pressure, and sustained hypotension led to the initial suspicion of TSS. Confirmation was obtained with the positive culture and the desquamation process.

Discussion

Toxic shock syndrome may occur when a *S. aureus* strain produces the exotoxin, toxic shock syndrome toxin-1 (TSST-1), in conjunction with *Staphylococcus* enterotoxin A (SEA), *Staphylococcus* enterotoxin B (SEB), or *Staphylococcus* enterotoxin C (SEC) (2). *S. aureus* isolates from nonmenstrual cases were found to produce TSST-1 in 50–60% of cases with SEA being the most commonly reported in up to 54% of cases (6, 7).

In the present case the less common SEB strain was isolated. SEB is associated with approximately 20% of cases of TSS. SEB and TSST-1 are detected using reverse passive latex agglutination (RPLA) (8). In a study by Lehn et al., 73 of 183 *S. aureus* isolates tested produced toxins. SEA was found in 37 samples (22%), SEB in 14 (7.7%), and SEC in 10 (5.5%). TSST-1 was identified in 25 samples (13.7%) (9). Lee et al. reported that SEB production was slightly more prevalent in the presence of TSS (53%) and suggested that, in the absence of TSST-1, if may be an important cause of nonmenstrual TSS as identified in our patient (8).

Staphylococcus infections are rather common and often resolve without formal treatment; however, TSS is a very rapid and debilitating disease. The severe hypotension is life-threatening, but with prompt, appropriate treatment, the risk of long-term sequela is decreased. If TSS is suspected, it is important to initiate IV hydration, parenteral antibiotics, and multisystem monitoring.

TABLE 1 Diagnostic criteria for toxic shock syndrome

Criteria	Clinical Presentation
Clinical symptoms	
Fever	≥102°F (38.9°C)
Rash	Diffuse macular erythroderma
Desquamation	Affects palms, soles, fingers, and toes 1–2 weeks after onset of illness
Hypotension	In adults (≥16 years) systolic blood pressure ≤90 mm Hg; in children: systolic blood pressure less than 5th percentile by age. Orthostatic drop in diastolic blood pressure of ≥15 mm Hg from lying or sitting. Orthostatic syncope of dizziness
Organ Systems Involved (3 or more)	
Gastrointestinal	Vomiting and diarrhea at onset
Muscular	Severe myalgia or creatinine phosphokinase level >twice normal high
Mucous membrane	Vaginal, oropharyngeal, or conjunctival hyperemia
Renal	BUN or serum creatinine >twice the upper limit of normal or ≥5 WBCs per high power field in absence of UTI
Hepatic	Total bilirubin, SGOT, or SGPT >twice normal upper limit
Hematologic	Platelets < 100, 000/mm^3

Source: Centers for Disease Control. Case definitions for public health surveillance. MMWR 39(No. RR-13):38–39, 1990.

Timely diagnosis of TSS is facilitated by consultation with internal medicine physicians and infectious disease specialists. The diagnostic criteria listed in Table 1 should be used if TSS is suspected (1). Our patient's condition was very critical; however, early recognition and prompt, appropriate treatment by the various medical services led to complete recovery with no apparent morbidity. Even though the patient has not had clinical follow-up by us, he has presented to other services and no adverse sequelae have been identified.

This case demonstrates the existence of TSS outside the realm of a gynecological disorder and should alert physicians to consider the syndrome in the differential diagnosis in the presence of a wound and fever.

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