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Toxic Shock Syndrome Associated with Suction-Assisted Lipectomy

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Abstract. Toxic shock syndrome (TSS) is a serious, potentially life-threatening condition resulting from an exotoxin of *Staphylococcus aureus*. Presenting symptoms include high fever, diarrhea, nausea, and vomiting progressing to hypotension, oliguria, conjuctival hyperemia, and an erythematous rash over the trunk, abdomen, and extremities. TSS has been associated both with and without the use of tampons during menstruation, postsurgical infections, and stab wounds, and can occur in postrhinoplasty patients with and without nasal packing. There has been one case reported in the literature of TSS associated with suction-assisted lipectomy (SAL). This article discusses the diagnosis and treatment of two cases in which the patients developed TSS after outpatient use of SAL. In the first case, an abdominoplasty and SAL were performed in a healthy young female. The second case involved the harvesting of abdominal fat via syringe suction and subsequent facial fat injection in a female with systemic lupus erythematosus and severe facial atrophy.

Key words: Toxic shock syndrome—*Staphylococcus aureus*—Suction-assisted lipectomy

In 1978 Todd et al. [13] first described a severe systemic illness characterized by hypotension, high fever, and a rash with desquamation. This illness, which was caused by a toxin produced by phage 1 *Staphylococcus aureus*, became known as toxic shock syndrome (TSS). In 1980 Schrock and associates [9] reported toxic shock in menstruating women and later that year the Centers for Disease Control (CDC) reported an association of toxic shock syndrome with the use of highly absorbent tampons [3]. In 1982, Thomas and coworkers [11] reported a case of toxic shock following nasal surgery. Since this time TSS has been increasingly recognized as an uncommon but serious complication following clean elective surgical procedures.

In a review of the literature, cases of TSS have been associated with various plastic surgical procedures including rhinoplasty with and without nasal packing, chemical face peel, abdominoplasty, and augmentation mammoplasty with breast prostheses [6, 7, 12]. Two cases of TSS associated with suction-assisted lipectomy are presented.

Case Reports

Case 1

A 36-year-old white female underwent outpatient abdominoplasty with suction-assisted lipectomy. Three days postoperatively the patient presented to the emergency room with a temperature of 104°F and swelling of her hands and feet, arthralgia, and generalized myalgia. Physical exam revealed blood pressure of 105/88, pulse of 132, and a petechial rash over her fingers and toes. There were no obvious signs of wound infection. The laboratory data revealed a WBC of 10,100 with 93% neutrophils. Liver enzymes were elevated; SGOT: 142, LDH: 271.

The patient was admitted to the hospital. Blood, urine, wound, and drain sites were cultured and the

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patient was then started on nafcillin and ceftriaxone. She responded to intravenous antibiotics and agressive fluid resuscitation. On the fifth postoperative day, patient's fever defervesced and white count was normal. On the ninth postoperative day, she was noted to have scaling of both palms and soles. Twelve days postoperatively she was discharged from the hospital in satisfactory condition.

Wound and drain cultures grew *Staphylococcus aureus*. Blood and urine cultures were negative at seven days.

Case 2

A 43-year-old black female with a history of systemic lupus erythematosus and facial atrophy underwent outpatient harvesting of abdominal fat using syringe suction. The fat was then injected into the cheeks. On the fourth postoperative day, the patient presented to the emergency room with complaints of fever, chills, and diarrhea over the previous two days. Physical examination showed a blood pressure of 105/50, pulse of 110, and a temperature of 103.6°F. She exhibited a generalized erythematous rash over her trunk, abdomen, and palms. On further examination, she was noted to have hyperemic conjunctiva and pharynx and 1 + edema of her lower extremities. Surgical incisions were clean with no evidence of erythema or exudate. Laboratory data revealed an elevated white count: WBC: 11,600; 62% polys and 24% bands and liver enzymes: Alk Phos: 160; SGOT: 460; SGPT: 348.

The patient was admitted to the hospital with sepsis. Blood, urine, and throat cultures were obtained. She was fluid resuscitated and started on vancomycin and gentamicin. Her hospital course was significant for renal insufficiency which responded to aggressive fluid resuscitation and bumetanide. During this time, the patient's antibiotics were changed to ciprofloxacin since she has multiple drug allergies to penicillin and cephalosporins. On the seventh postoperative day, her fever defervesced and her rash was noted to be "fading fast." White count and renal and hepatic function returned to normal and she was discharged home 13 days postoperatively. On followup exam one week later, she had scaling of her palms and trunk. Blood, urine, and throat cultures were negative at seven days.

Discussion

Suction-assisted lipectomy (SAL) is a surgical procedure used to remove excess subcutaneous fat through small incisions. It is also useful in treating contour defects by extracting fat from one area by syringe SAL and transplanting the fat in a semiliquid form to fill a depressed area. In 1987, the ASPRS reported that in the previous five years, 100,000 cases of SAL operations were performed. There had been 11 deaths and 9 cases of serious morbidity [1]. The most common complications that are potentially fatal are thromboplebitis, pulmonary embolus, hypovolemia, and fat emboli. While infection may be a common complication following this procedure, to date there has been only one report in the literature of TSS associated with SAL.

TSS was first reported by Todd et al. in 1978 [13] and later became known as a disease affecting young women, most of whom were menstruating and using tampons. Recently, TSS has been reported following clean elective surgical procedures.

TSS is a serious, potentially life-threatening condition resulting from an exotoxin produced by phage 1 *Staphylococcus aureus*. The toxin, toxic shock toxin-1 (TSST-1), was isolated in 1981 by Schlievert and coworkers [8] and has been found in 90% of menstrual-related cases of TSS [2, 8] and 62% of nonmenstrual-related cases [5]. Furthermore, individuals with low anti-TSST-1 antibodies have been found to be at a higher risk for developing TSS [2]. TSS can occur in various clinical settings, in both sexes, and in all ages and racial groups.

There is no definitive diagnostic test for TSS so diagnosis is based on signs and symptoms and laboratory data. The four major clinical signs include high fever ($T > 102$°F), a diffuse macular rash, desquamation one or two weeks after the onset of illness (particularly of the palms and soles), and hypotension. Other clinical manifestations that reflect multiple organ involvement include nausea, vomiting, myalgia, arthralgia, and hyperemic mucous membranes. Multisystem dysfunction is also reflected in laboratory data, i.e., elevation of liver enzymes, creatinine, urea nitrogen levels. Table 1 summarizes the Centers for Disease Control (CDC) criteria for diagnosis of TSS.

The mean incubation period in postoperative patients is two days. A striking feature of postoperative TSS is that signs of local wound infection are rarely present. Blood, urine, throat, and CSF cultures are characteristically negative.

When establishing the diagnosis of TSS, other diseases with multiple system involvement should include viral syndromes, Kawasaki disease, staphylococcal scalded skin syndrome, streptococcal scarlet fever, Rocky Mountain spotted fever, and leptospirosis.

Management

The management of patients with TSS depends on the severity of the illness. If possible, any source of infection is removed to prevent formation of additional toxin. If present, tampons or nasal packings are removed, abcesses drained, and infected wounds debrided. Aggressive fluid resuscitation with crystalloid is the mainstay of therapy. In extreme cases,

Table 1. Centers for Disease Control (CDC) criteria for the diagnosis of toxic shock syndrome[a]

(1) Fever (>102°F)
(2) Rash (diffuse macular erythroderma)
(3) Desquamation (1–2 weeks after onset, especially of palms and soles)
(4) Hypotension
(5) Involvement of three or more organ systems:
Gastrointestinal (vomiting, diarrhea at onset)
Muscular (myalgia, elevated CPK)
Mucous membrane (conjunctiva, oropharynx)
Renal (BUN or Cr > 2 times normal)
Hepatic (bilirubin, SGOT, SGPT > 2 times normal)
Hematologic (platelets < 100,000)
CNS (disorientation)
(6) Negative results on the following studies, if obtained:
Blood, throat, or CSF cultures
Serologic tests for Rocky Mountain spotted fever
Leptospirosis, measles

[a] From Morbidity Mortality Weekly Rev **29**:441—445 1980

pharmacologic support with vasopressors may be needed.

In the critically ill patient, invasive monitoring with Swan–Ganz catheters and arterial lines may be indicated. Ventilatory support is occasionally needed if respiratory distress develops. *Staphylococcus aureus* strains associated with TSS are almost always resistant to penicillin and ampicillin. Appropriate first-choice antibiotics include B-lactamase-resistant penicillins (nafcillin, oxacillin), clindamycin, first-generation cephalosporins, or vancomycin. Antibiotics may not help the patient in the acute phase of illness, but antibiotic treatment is advised to prevent the significant risk of bacteremia. Antibiotic treatment has also been found to lower the rate of recurrence following infection [4].

Sequelae

A desquamation of skin, particularly of the palms and soles, occurs 5–12 days after the onset of acute illness in all patients with TSS. Delayed loss of hair and nails, renal insufficiency, renal failure, and gangrene have been reported and are presumed to be secondary to prolonged episodes of hypotension. Neuropsychiatric sequelae including difficulty concentrating, cognitive dysfunction, headache, and abnormalities on EEG have been described. Unilateral vocal cord paralysis, carpal tunnel syndrome, and diffuse myopathy have also been reported.

Summary

TSS is an acute febrile illness characterized by hypotension, an erythematous rash followed by desquamation, and multiorgan involvement. Treatment consists primarily of fluid resuscitation and supportive measures. Antistaphylococcal agents are indicated to treat primary wound infection and to prevent recurrent disease. The role of prophylactic perioperative use of antibiotics is unclear. However, based on the uncommon occurrence of TSS, we feel that use of prophylactic antibiotics in clean elective cases is unwarranted.

We have recently experienced TSS as a complication of SAL and recommend that TSS should be considered in postoperative patients presenting with high fevers ($T > 102$°F) and rash in the first few days following SAL. The success and effectiveness of treatment depends on early recognition of the disease with prompt initiation of fluid therapy and supportive care.

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