

## CASE REPORT

# Breast abscess in a competitive high school swimmer: a case of toxic shock syndrome

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## Abstract

Toxic shock syndrome (TSS) is a severe, acute, toxin-mediated disease process characterized by fever, diffuse erythroderma, hypotension, multisystem organ dysfunction and desquamation of skin. TSS represents the most severe form of disease caused by exotoxin-producing strains of *Streptococcus pyogenes* and *Staphylococcus aureus*. Menstrual and non-menstrual TSS become significant causes of morbidity and mortality. As a result of public awareness and various campaigns, the majority of TSS cases tend to be non-menstrual related. The clinical course is fulminant and can result in abrupt decompensation and death. Management within the emergency department (ED) includes removal of the potential foreign body, fluid resuscitation, appropriate antibiotics, potential vasopressor support and possible surgical intervention. We present the unique case of a 16-year-old female competitive swimmer who presented to the ED twice, demonstrating the fulminant course of TSS. She initially presented with non-specific symptoms with an unremarkable evaluation. She returned within hours of discharge with an abrupt onset of diffuse macular erythroderma, placed on norepinephrine and was diagnosed with TSS secondary to a breast abscess.

## INTRODUCTION

The most common bacterium involved in the development of a breast abscess is *Staphylococcus aureus*. Toxic shock syndrome (TSS) secondary to mastitis is infrequently reported, with no reported cases to date involving the development of TSS from a breast abscess not due to mastitis. The overall incidence of TSS is 11 per 100 000 people in the United States [2, 5]. The mortality in pediatric patients approaches 5% for TSS secondary to *S. aureus* and up to 10% in TSS secondary to *Streptococcus pyogenes*. However, in adult patients, the overall mortality ranges from 30–80% depending on the initial clinical presentation and disease course [2, 3, 5].

## CASE REPORT

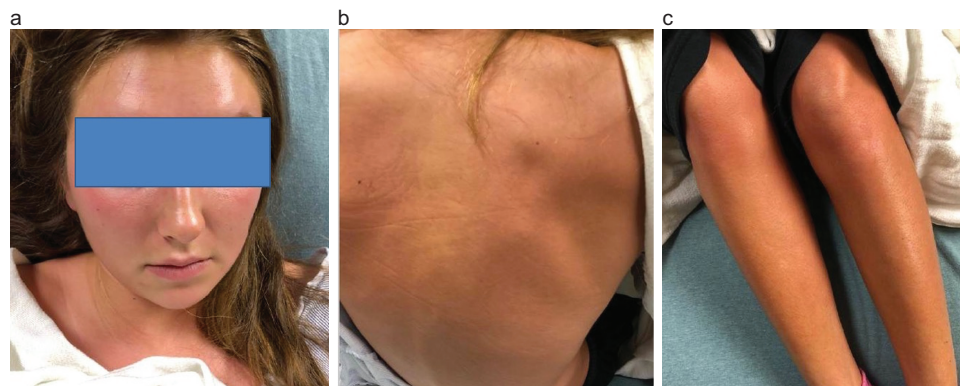
A 16-year-old female with a significant past medical history of asthma presented to our community emergency department (ED) with the chief complaint of abdominal pain, body aches, nausea, headache and shortness of breath of 3 days duration. The abdominal pain was localized to the right upper quadrant and described as achy in nature without radiation. Additionally, the patient reports she incidentally felt a lump within her right breast. The patient is an avid competitive swimmer. She is not sexually active. Her last menstrual period was ~3 weeks prior to arrival and denied tampon use since then.

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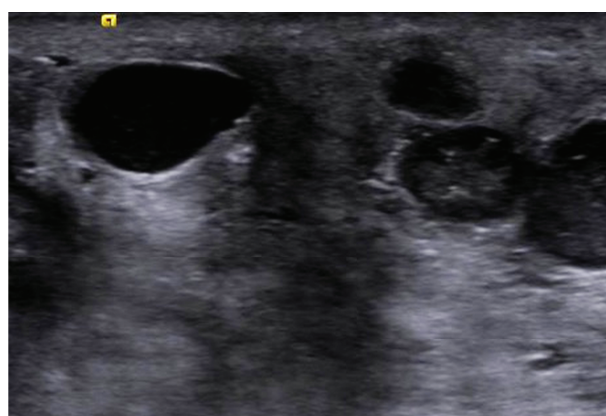


**Figure 1:** (a) Clinical photograph revealing blanchable diffuse macular erythroderma to the entire face/neck/upper chest. (b) Clinical photograph revealing blanchable diffuse macular erythroderma to the entire back. (c) Clinical photograph revealing blanchable diffuse macular erythroderma to the legs.

On her first visit to the ED around 7:00 am her vitals were unremarkable and within normal limits. On the physical exam the patient appeared well and was in no acute distress. Her abdominal exam revealed minimal tenderness in the right upper quadrant with no peritoneal signs. Examination of the right breast revealed no fluctuance, erythema or obvious lump, however, was mildly tender to palpation inferior to the right areola. Her remaining physical exam was unremarkable, as were chest radiography, abdominal ultrasound and laboratory evaluation. The patient's tachycardia improved with intravenous hydration and her myalgias improved with intravenous ketorolac. She was subsequently discharged with recommended follow-up with her OB/Gyn and to return to the ED if symptoms worsen. The patient and her parents presented to her OB/Gyn around 3:00 pm regarding worsening of the lump in her right breast along with development of a diffuse rash and appearing flushed. Her temperature was 101.4°F. She now appeared ill, presence of chills, with full body erythema. Examination of the right breast revealed no external evidence for infectious process, however, there was a deep 2.5-cm exquisitely tender nodule located inferior and lateral at the areolar border of the right breast. She was then referred back to the ED.

Vitals on arrival for her second ED visit at 5:00 pm: 98.6°F (acetaminophen given prior to arrival), heart rate of 102 beats/min, blood pressure of 92/47 mmHg, respiratory rate of 17 breaths/min and pulse oximetry of 99% on room air. On the physical exam she now had a blanchable diffuse full-body macular erythroderma, (Fig. 1a–c). Examination of the right breast revealed the known deep 2.5-cm exquisitely tender nodule, however, now with the presence of overlying erythema consistent with cellulitis. Her abdominal exam was identical to prior. Her remaining physical exam including a complete neurological exam was unremarkable. A repeat laboratory evaluation was notable for

- White blood cell count of 17 (4.5–13 bil/L)
- Alkaline phosphatase of 136 (37–134 U/L)
- Aspartate aminotransferase of 122 (10–37 U/L)
- Alanine aminotransferase of 151 (5–20 U/L)
- Total bilirubin of 1.4 (0.3–1.2 mg/dL)
- Lactic acid of 1.3 (0.5–2.2 mmol/L)
- Creatinine kinase of 50 (30–150 U/L)
- Influenza A/B, respiratory syncytial virus and infectious mononucleosis by nucleic acid amplification were negative



**Figure 2:** Right breast ultrasound revealing several cystic areas with the largest up to 2.0 × 1.0 × 1.3 cm. The cystic areas demonstrate internal debris and posterior acoustic enhancement.

Ultrasound of the right breast, Fig. 2, revealed several cystic areas, with the largest up to 2.0 × 1.0 × 1.3 cm. These areas demonstrated internal debris and posterior acoustic enhancement. Given the breast abscess as the likely source of TSS, a family discussion was had regarding a performing a pelvic exam and a decision was made to defer. The patient was empirically started on intravenous vancomycin (40 mg/kg/day divided 4 times a day) and clindamycin (40 mg/kg/day divided 3 times a day). The patient's erythroderma continued to rapidly get worse and despite 30 cc/kg of fluid resuscitation, her blood pressure continued to decline to 82/46 mmHg and she was subsequently started on norepinephrine at 1 mcg/kg/min and transferred to a tertiary center pediatric ICU. A subsequent pelvic exam was performed for completeness that was negative for acute process or foreign body. The patient was taken to the operating room with pediatric general surgery for an incision and drainage of her right breast abscess. Multiple abscess cavities were noted with purulent material being evacuated along with debridement involving the skin/subcutaneous/breast tissue. Culture of wound later showed *Staphylococcal aureus* methicillin-resistant before *Staphylococcal aureus* (MRSA), with both vancomycin and clindamycin susceptibilities. By Day 2, the patient was off norepinephrine with an improving rash. The patient continued to rapidly improve and was discharged on oral clindamycin on Day

4 with a planned antibiotic duration of 10 days. At her 1-month general surgery follow-up the patient continues to do well and has returned to competitive swimming.

## DISCUSSION

Patients with the highest risk for *Staphylococcal* TSS are female patients with pre-existing *Staphylococcal* vaginal colonization who leave contraceptive sponges, diaphragms or tampons within the vagina [6]. Other risk factors include patients with burns, soft tissue injuries, retained nasal packing, post-abortion, post-surgical, post intrauterine device placement and abscess formation [2, 5]. For *Streptococcal* TSS, it can be seen in the setting of pharyngitis or associated with soft tissue trauma/focal infection [2, 5].

The symptoms associated with *Staphylococcal* TSS result from toxin production, in comparison to the site of infection seen with *Streptococcal* TSS. It is for this reason that patients with *Staphylococcal* TSS may be misdiagnosed at first with influenza or another viral syndrome. A patient may present to the ED with a low-grade fever, headache, sore throat, myalgias, vomiting and diarrhea a week prior to the onset of this potentially devastating disease process. At this stage, it mimics a viral syndrome like viral gastroenteritis. It is within the next 24–48 hours that a sunburn like rash develops. If left unrecognized, the patient then begins to decompensate.

Clinical criteria for the diagnosis of both menstrual and non-menstrual related *Staphylococcal* TSS includes an abrupt onset of a flu-like illness, in combination with a temperature of  $>102^{\circ}\text{F}$ , presence of diffuse macular erythroderma or morbilliform type rash (develops within 24–48 hours), systolic blood pressure  $<90$  mmHg (or presence of syncope or orthostatic hypotension), multisystem dysfunction and eventual desquamation to the palms and soles that begins anywhere from 1–2 weeks after onset of illness [2, 3].

To meet criteria for the diagnosis of *Staphylococcal* TSS, multi-system dysfunction involves at least three systems, including

- Gastrointestinal (vomiting, diarrhea)
- Muscular (myalgias, increased creatinine phosphokinase  $2\times$  normal)
- Mucous membranes (conjunctival, oropharyngeal or vaginal hyperemia)
- Renal (elevated blood/urea/nitrogen or creatinine, presence of pyuria, with no evidence for a urinary tract infection)
- Hepatic (transaminase or bilirubin at least  $2\times$  the upper limit of normal)
- Hematologic (platelets  $<100\,000$ , leukocytosis, thrombocytopenia, anemia)
- Central nervous system (altered mental status, disorientation, with no focal neurologic deficits) [1, 2]

A patient with *Streptococcal* TSS presents with similar criteria, however, the clinical manifestations and organ system involvement are slightly different. *Streptococcal* TSS presents with additional symptoms at the site of infection [2, 5]. A patient may present with severe pain at the site of the soft-tissue infection with associated necrotizing fasciitis, myositis or gangrene [2, 5]. Respiratory involvement can also include acute respiratory distress syndrome (55% of patients) [6].

Because TSS secondary to both *S. aureus* and *S. pyogenes* may be difficult to distinguish, the recommendation is to start broad-spectrum antibiotics against both pathogens. Once the

organism is identified, the antibiotic regimen is then adjusted. Additionally, source control by removing the foreign body and/or surgical intervention with potential debridement or drainage is imperative. Clindamycin is a commonly recommended antibiotic as it is a suppressor of bacterial toxin synthesis and possesses activity against *Streptococci* and some activity against *Staphylococci* [4]. An appropriate antibiotic regimen includes a penicillinase-resistant penicillin or a cephalosporin (piperacillin-tazobactam or cefepime), in addition to vancomycin and clindamycin [2, 5]. There are a few case reports utilizing adjunctive intravenous immunoglobulin (IVIG) in the setting of severe pediatric sepsis and septic shock when conventional therapy is not working. IVIG has been shown to contain antibodies against the toxins. At high enough concentrations, toxicity can be neutralized [7, 8]. Some observational studies have shown a decrease in mortality; however, further studies are needed on this [7, 8].

We present the unique case of a 16-year-old competitive swimmer that presented to the ED with a viral-like prodrome for only 3 days, and was found to have a completely unremarkable initial ED evaluation. However, within hours of her presentation she developed a diffuse macular erythroderma and septic shock requiring inotropic support and was diagnosed with TSS. She was subsequently taken to the operating room for an incision/drainage of her right breast abscess and was discharged on Day 4. To our knowledge, this is the first reported case report to document the fulminant course of non-menstrual related *Staphylococcal* TSS from a breast abscess not due to mastitis.

## CONFLICT OF INTEREST STATEMENT

None declared.

## FUNDING

No financial support was received for this study.

## ETHICAL APPROVAL

No approval is required.

## CONSENT

Informed patient consent was obtained.

## GUARANTOR

G.T. is the guarantor of this study.

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