

TOXIC SHOCK SYNDROME (TSS)

REPORTING INFORMATION

- **Class B2:** Report by the end of the business week in which the case or suspected case presents and/or a positive laboratory result to the local public health department where the patient resides. If patient residence is unknown, report to the local public health department in which the reporting health care provider or laboratory is located.
- Reporting Form(s) and/or Mechanism: [Ohio Confidential Reportable Disease form](#) (HEA 3334, rev. 1/09), [Positive Laboratory Findings for Reportable Disease form](#) (HEA 3333, rev. 8/05), the local health department via the Ohio Disease Reporting System (ODRS) or telephone.
- [CDC Toxic Shock Syndrome Case Report](#) (form 52.3, rev. 3/85) is available for use to assist in local disease investigation and contact tracing activities. Information collected from the form should be entered into ODRS and not sent to ODH, unless otherwise requested.
- Additional reporting information, with specifics regarding the key fields for ODRS Reporting can be located in [Section 7](#).

AGENT

Toxin-producing *Staphylococcus aureus*. In most cases, toxic shock syndrome toxin-1 (TSST-1), an enterotoxin, has been identified.

CASE DEFINITION

Clinical Case Definition

An illness with the following clinical manifestations:

- Fever: temperature greater than or equal to 102.0°F (38.9°C)
- Rash: diffuse macular erythroderma
- Desquamation: 1-2 weeks after onset of rash
- Hypotension: systolic blood pressure less than or equal to 90 mm Hg for adults or less than fifth percentile by age for children aged less than 16 years
- Multisystem involvement (three or more of the following organ systems):
 - Gastrointestinal: vomiting or diarrhea at onset of illness
 - Muscular: severe myalgia or creatine phosphokinase level at least twice the upper limit of normal
 - Mucous membrane: vaginal, oropharyngeal, or conjunctival hyperemia
 - Renal: blood urea nitrogen or creatinine at least twice the upper limit of normal for laboratory or urinary sediment with pyuria (greater than or equal to 5 leukocytes per high-power field) in the absence of urinary tract infection
 - Hepatic: total bilirubin, alanine aminotransferase enzyme, or aspartate aminotransferase enzyme levels at least twice the upper limit of normal for laboratory
 - Hematologic: platelets less than 100,000/mm³
 - Central nervous system: disorientation or alterations in consciousness without focal neurologic signs when fever and hypotension are absent

Laboratory Criteria for Diagnosis

Negative results on the following tests, if obtained:

- Blood or cerebrospinal fluid cultures (blood culture may be positive for *Staphylococcus aureus*)

- Negative serologies for Rocky Mountain spotted fever, leptospirosis, or measles.

Case classification

Probable: A case which meets the laboratory criteria and in which four of the five clinical findings described above are present

Confirmed: A case which meets the laboratory criteria and in which all five of the clinical findings described above are present, including desquamation, unless the patient dies before desquamation occurs

Comment

See also Streptococcal Toxic Shock Syndrome (STSS).

SIGNS AND SYMPTOMS

Toxic shock syndrome (TSS) is characterized by sudden onset of fever, chills, vomiting, diarrhea, muscle aches and rash. It can rapidly progress to severe and intractable hypotension and multisystem dysfunction. Desquamation, particularly on the palms and soles can occur 1-2 weeks after onset of the illness

DIAGNOSIS

There is no definitive diagnostic test. Diagnosis is based on clinical presentation (see case definition).

EPIDEMIOLOGY**Source**

Epidemiologic studies have shown that colonization with TSST-1 producing strains of *S. aureus* is common. Most people, however, do not develop TSS.

Occurrence

Sporadic individual cases occur with no seasonal distribution. TSS is non-communicable. While cases most frequently occur in young menstruating women and are associated with the use of high absorbency tampons, cases have been reported in men, children and older women. In these latter cases, focal infection, as of skin or wounds, with *S. aureus* has been common, even though the infected site may appear grossly normal. Non-menstrual TSS has also been associated with use of diaphragms and contraceptive sponges, vaginal infections, childbirth, abortion and the postpartum state.

PUBLIC HEALTH MANAGEMENT**Case**Treatment

The focus of infection should be eradicated while supportive therapy is provided.

Contacts

No follow-up is needed for contacts.

Prevention and Control

Minimal use of high-absorbency tampons and patient education can prevent menstrual-related TSS. Treatment of first episodes of TSS with parenteral antibiotics and subsequent avoidance of tampons can prevent recurrences.

What is TSS?

TSS may be caused by toxin-producing *Staphylococcus aureus* or *Streptococcus pyogenes* (group A streptococci). Both organisms cause acute illness characterized by fever, generalized redness (rash), rapid-onset hypotension (low blood pressure), and symptoms of multisystem organ involvement that can include profuse watery diarrhea, vomiting, kidney and liver dysfunction and central nervous system abnormalities (disorientation or alterations in consciousness).

Both forms of TSS may occur without a readily identifiable focus of infection. TSS usually refers to *Staphylococcus aureus*-mediated illness commonly associated with tampon use in menstruating women. STSS refers to *Streptococcus pyogenes*-mediated toxic shock syndrome and is more closely associated with severe illness relating to what is commonly known as flesh-eating disease (please refer to STSS fact sheet).

Etiology of TSS

Staphylococcus aureus-mediated TSS usually is caused by strains producing toxic-shock syndrome toxin-1 (TSST-1) or possibly other related staphylococcal enterotoxins.

Diagnosis of TSS

Staphylococcus aureus-mediated TSS remains a clinical diagnosis. Blood culture results are positive for *Staphylococcus aureus* fewer than 5 percent of patients. Specimens for culture should be obtained from an identified site of infection because these sites usually will be positive and testing of isolated organisms can be performed. Because approximately one third of isolates of *Staphylococcus aureus* from non-menstrual cases produce toxins other than TSST-1, and TSST-1 producing organisms can be present as part of the normal flora of the anterior nares (nose) and vagina, production of TSST-1 by an isolate of *Staphylococcus aureus* is not helpful diagnostically.

***Staphylococcus aureus*-mediated TSS**

This syndrome first was recognized in 1978, occurring in children and adults both male and female; many early cases frequently were associated with tampon use in menstruating women, with a predilection for adolescents and young women with no circulating antibody to TSST-1. Although changes in tampon composition and use may have resulted in some decrease in the proportion of cases associated with menstruation, both menstrual and non-menstrual cases of TSS continue to occur.

What is the incubation period of *Staphylococcus aureus*-mediated TSS?

The incubation period for post-operative *Staphylococcus aureus*-mediated TSS can be as short as 12 hours. Menses-related cases can develop anytime during menses.

Transmission and Infection Control

Because person to person transmission of *Staphylococcus aureus*-mediated TSS is uncommon, only standard precautions are needed unless a draining wound is present.

Is there treatment for TSS?

The first priority is aggressive fluid replacement as well as management of respiratory or cardiac failure or irregular heart rhythm if present in addition to empiric antimicrobial therapy. Initial empiric antimicrobial therapy should include an antistaphylococcal

antimicrobial agent plus a protein synthesis-inhibiting antimicrobial drug such as clindamycin.