

Toxic shock syndrome (TSS), Staphylococcal

For streptococcal toxic shock syndrome, see Group A strep (GAS)

Disease plan

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Questions about this disease plan?

Contact the Utah Department of Health and Human Services Office of Communicable Diseases: 801-538-6191.

TSS, Staphylococcal critical clinical information

Clinical evidence
Signs/symptoms <ul style="list-style-type: none">• Sudden onset of high fever, vomiting, profuse watery diarrhea, and muscle pain, followed by multi-system organ involvement• Hypotension• Septic shock• Rash with peeling skin 1–2 weeks after onset of symptoms• Evidence of <i>Staphylococcus aureus</i> infection
Period of communicability <ul style="list-style-type: none">• Variable, roughly 30% of people are carriers of <i>S. aureus</i>• Transmission lasts as long as lesions drain or carrier state exists.
Incubation period <ul style="list-style-type: none">• Varies from hours to days depending on the source and route of infection.• Post operative TSS incubation period can be as short as 12 hours.
Mode of transmission <ul style="list-style-type: none">• TSS itself is not transmissible person to person.• Transmission is primarily through person-to-person contact, either with a person who has a purulent lesion or with an asymptomatic carrier.• Contaminated objects can also serve as sources of infection.• Airborne spread is rare but has been demonstrated in patients with associated viral respiratory disease.
Laboratory testing
Type of lab test <ul style="list-style-type: none">• Toxic shock syndrome is not a laboratory diagnosis, but a clinician diagnosis.• Culture, serologic, or molecular testing for <i>S. aureus</i>• Generally performed at hospital/commercial labs
Type of specimen—clinical <ul style="list-style-type: none">• Blood• Cerebrospinal fluid• Wound swabs• It may be useful to test tampons or other inserted vaginal devices. Would need to be performed by CDC or non-clinical lab.

Treatment recommendations
Type of treatment <ul style="list-style-type: none">• Local measures (decontamination, debridement)• Fluid resuscitation and circulatory support• Empiric antibiotic therapy• Give IV immune globulin if TSS is severe• Immediate removal of infectious source material (e.g tampons, sponges, infected tissues)• Methicillin-susceptible <i>S. aureus</i>: clindamycin as well as oxacillin or nafcillin• Methicillin-resistant <i>S. aureus</i>: clindamycin and vancomycin, or linezolid and vancomycin, or linezolid alone depending on the source of the infection.
Time period to treat <ul style="list-style-type: none">• The management of staphylococcal TSS calls for immediate correction of hypotension and shock with vigorous fluid replacement, attention to the site of <i>S. aureus</i> colonization or infection, and systemic antimicrobial therapy with an antistaphylococcal agent
Prophylaxis <ul style="list-style-type: none">• Generally not recommended
Contact management
Isolation of case <ul style="list-style-type: none">• None
Quarantine of contacts <ul style="list-style-type: none">• None
Infection control procedures
<ul style="list-style-type: none">• CDC does not provide infection control recommendations for TSS, staphylococcal infections because they are often not communicable• Standard contact precautions for <i>S. aureus</i>

Why is TSS, staphylococcal, important to public health?

Toxic shock syndrome (TSS) is a set of severe symptoms that involves many systems of the body. TSS occurs when certain bacterial infections release toxins into the bloodstream, causing an over-activation of the immune system, which damages organ systems and causes severe illness. *Staphylococcus aureus* infection is the main cause of toxic shock syndrome and is the focus of this disease plan. Invasive *Streptococcus pyogenes* infection can also trigger a similar condition (see Streptococcal toxic shock in the [Group A Strep disease plan](#)).

Toxic shock syndrome is widely known from a number of cases in the late 1970s and early 1980s related to the use of highly absorbent tampons. These tampons are no longer sold in the US, and the incidence of tampon-induced TSS has declined since it was first identified.¹ Today, TSS is often due to postoperative wound or soft tissue infections. Symptoms can quickly develop from a variety of *S. aureus* infections.

Staphylococcal TSS can be divided into 2 categories based on the causal association of infection: menstrual and nonmenstrual. Menstrual TSS usually occurs a few days after initiation to a few days after completion of menstruation and is associated with tampon usage in women colonized vaginally by superantigen-producing *S aureus*.

Nonmenstrual TSS occurs as a complication of *S aureus* infections after surgical procedures, burns, or influenza pneumonia.

The overall case fatality rate is from 2% to 5%, which is lower than the reported fatality rates of streptococcal TSS. Mortality is increased significantly—by up to 50%—if adult respiratory distress syndrome or refractory hypotension is apparent. Moreover, nonmenstrual TSS seems to carry a worse prognosis than menstrual TSS, probably because of the delay in diagnosis and the more serious nature of the primary infection.²

Disease and epidemiology

Clinical description

TSS is a severe, toxin-mediated illness characterized by sudden onset of high fever, vomiting, profuse watery diarrhea, and muscle pain, followed by hypotension and multisystem organ involvement. The systems affected may include the gastrointestinal, muscular, mucocutaneous (including vagina, pharynx, and conjunctivae), renal, hepatic, respiratory, hematologic, and central nervous systems. Severe cases may result in shock and death. A “sunburn-like” rash is often present during the acute phase of the illness, with peeling skin—especially on the soles and palms—typically occurring 1–2 weeks later. The gastrointestinal symptoms and peeling skin are more commonly present with *S. aureus*-mediated TSS than GAS-mediated TSS. Both forms of TSS

may be associated with invasive infections and can be fatal. TSS is usually diagnosed clinically and may also occur without an identifiable focus of infection.

Causative agent

Toxic shock syndrome is a serious complication of infection with strains of *Staphylococcus aureus* and *Streptococcus pyogenes* (group A streptococci or GAS) that produce certain toxins (TSS toxin 1 for *S. aureus*, pyogenic exotoxin A for GAS).

Differential diagnosis

Since TSS can be confused with many infectious and non-infectious causes of fever with mucocutaneous manifestations, diseases such as Rocky Mountain spotted fever, leptospirosis, meningococemia, scarlet fever, Reye syndrome, staphylococcal scalded skin syndrome, measles, and viral exanthematous diseases should be ruled out. These disorders are ruled out by specific clinical differences, cultures, and serologic tests.

Laboratory identification

Staphylococcus aureus are easily cultured in most clinical labs. Bacteria are more readily cultured from suspected sites of infection. Blood cultures are rarely positive. It is important to note that tests that are positive for “toxic shock syndrome antibody, MAID” do **not** indicate the presence of toxic shock syndrome; rather they generally signify a previous immune response and may confer protection from developing clinical symptoms. Most antibody tests are for research use only. TSS is a clinical diagnosis so expect that *S. aureus* may not be isolated from all patients.

UPHL: The Utah Public Health Laboratory (UPHL) does not perform routine cultures for streptococcal or staphylococcal diseases.

Treatment

For menstruation-related TSS, tampons or other inserted vaginal devices (e.g. contraceptive sponges) should be removed immediately.

Treatment of TSS is largely supportive, but the underlying infection should be addressed through source removal and appropriate antibiotics.

In short, treatment could include:

- Local measures (decontamination, debridement)
- Fluid resuscitation and circulatory support
- Removal of devices or tampons
- Empiric antibiotic therapy—clindamycin in combination with a β -lactamase-resistant antistaphylococcal agent. Clindamycin should not be used alone, because strains of *S.*

aureus with clindamycin resistance have been reported. Because of the emergence of staphylococcal TSS caused by MRSA, empirical antibiotic therapy should be with vancomycin, daptomycin, or linezolid. Protein synthesis inhibitors such as clindamycin and linezolid have been shown to suppress toxic shock syndrome toxin production-1.

- Give IV immune globulin if TSS is severe. IVIG has been shown to reduce the morbidity and mortality of staphylococcal and streptococcal TSS, although most evidence was from streptococcal TSS.²⁻⁵

Case fatality

While still a serious illness, TSS due to *S. aureus* has lower mortality rates than Streptococcal TSS due to group A strep. Mortality due to non-menstrual TSS is around 3–5%. The mortality of menstrual TSS has declined from 5.5% in 1979–1980 to 1.8% from 1987–1996.⁶

The incidence of TSS is estimated to be around 0.8 to 3.4 per 100,000 in the U.S.⁶

Reservoir

Staphylococcus aureus is common human bacteria; humans are the primary reservoir. Thirty percent (30%) of people carry *S. aureus* in their nose, and 2% carry methicillin-resistant *S. aureus* (MRSA).⁷

Transmission

While TSS itself is not communicable from person to person, the organisms that cause TSS are transmissible. *S. aureus* bacteria is transmitted from person to person through direct contact with lesions or contaminated respiratory secretions, including droplets. Airborne spread is rare but has been demonstrated in patients with associated viral respiratory disease. Transmission can also take place via asymptomatic carrier. A major site of colonization is the human nasal vestibulum; 20–30% of the population is a nasal carrier of *S. aureus*.⁸ *S. aureus* transmission involving indirect contact through objects has occurred in schools (contaminated wrestling mats) and in daycare centers (through play food and shared toys).

Susceptibility

Toxic shock syndrome is a toxin-mediated illness where *Staphylococcus aureus* produces “superantigens” which are capable of stimulating T-cells to produce a massive, nonspecific immune response. This over activation of the immune system causes the clinical symptoms of TSS. Some patients fail to develop antibodies and may relapse after an episode of TSS.

Incubation period

The incubation period for *S. aureus*-mediated TSS varies from hours to days, depending on the source and the route of infection. For post-operative *S. aureus*-mediated TSS, the incubation period can be as short as 12 hours.⁹

Period of communicability

TSS itself is not communicable from person to person, but the bacteria that cause TSS are transmissible. With *S. aureus*, the infectious period lasts as long as lesions drain or the carrier state exists. Autoinfection of *S. aureus* is very common. Autoinfection refers to an infection caused by an organism already present in the body that is transferred from one part of the body to another.

Epidemiology

TSS became widely recognized in 1980 when an association between TSS and the use of tampons was established. However, cases occur in both males and females. Persons considered at higher risk for *S. aureus*-mediated TSS include:

- Women who use tampons or other inserted vaginal devices (such as diaphragms or contraceptive sponges);
- Women who have infections after pregnancy or abortion;
- Persons who have undergone nasal surgery; and
- Persons with post-operative staphylococcal wound infections.⁹

The proportion of cases associated with menstruation has decreased significantly since certain types of tampons were removed from the market. Currently, approximately half of reported TSS cases are not related to menstruation.¹⁰ Non-menstrual TSS has been observed in a variety of clinical situations, including surgical and postpartum wound infections, mastitis, septorhinoplasty, sinusitis, osteomyelitis, arthritis, burns, lesions (especially of the extremities, perianal area, and axillae), respiratory infections following influenza, and enterocolitis.

Public health control measures

Public health responsibility

- Investigate all suspect cases of disease; fill out and submit the appropriate disease investigation form.
- Provide education to the general public, clinicians, and first responders regarding disease transmission and prevention.
- Identify clusters or outbreaks of this disease.

Prevention

Women who have had staphylococcal TSS should refrain from using tampons and menstrual cups, cervical caps, contraceptive sponges, intrauterine devices, diaphragms, and pessaries. Advising all women to change tampons frequently or use napkins instead and to avoid hyper-absorbent tampons is key to reducing the risk of disease.

For other TSS caused by Streptococcal or Staphylococcal organisms, good hygienic practices, such as handwashing and respiratory etiquette, are helpful.

Prophylaxis

Chemoprophylaxis is generally not recommended for TSS specifically. Topical antiseptics might be considered in persons with recurring cutaneous *S. aureus* infections or in cases of ongoing transmission within a close cohort of people.¹¹

Vaccine

None

Isolation and quarantine requirements

Isolation: None

Hospital: Standard body substance and contact precautions

Quarantine: None

Case investigation

Reporting

All cases of TSS should be reported within 3 days of identification. Report any illness to public health authorities that meets the following criteria:

- Report any person whose healthcare record contains a diagnosis of non-streptococcal toxic shock syndrome.
- Report any person whose death certificate lists non-streptococcal toxic shock syndrome as a cause of death or a significant condition contributing to death.
- Report any person for whom 4 of the 5 clinical criteria, and all of the laboratory criteria listed in table 1, are met.

Toxic shock syndrome is a disease of clinical recognition, therefore the burden of reporting this disease falls to clinicians rather than to laboratories.

Note: the following is copied directly from [CSTE Position Statement 10-ID-14](#).

Report any illness to public health authorities that meets 4 of the 5 clinical criteria listed in table 1 **and** all of the laboratory criteria listed in table 1.

Clinical evidence

- fever: temperature higher than or equal to 102.0°F (higher than or equal to 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 younger than 16 years
- multisystem involvement (3 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 (higher 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 evidence

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

Table 1: Criteria to determine whether a case should be reported to public health

Criterion	Reporting	
<i>Clinical evidence</i>		
Fever (temperature $\geq 38.9^{\circ}\text{C}$ [102.0°F])		O*
Diffuse macular erythrodermal rash		O*
Peeling skin 1-2 weeks after onset of illness, particularly on the palms and soles†		O*
Hypotension (systolic blood pressure ≤ 90 mm Hg for adults or less than fifth percentile by age for children younger than age 16)		O*
Multisystem involvement, 3 of the following organ systems involved: <ul style="list-style-type: none"> - gastrointestinal: vomiting or diarrhea at onset of illness - muscular: severe muscle pain 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 (≥ 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 $< 100,000/\text{mm}^3$ - central nervous system: disorientation or alterations in consciousness without focal neurologic signs when fever and hypotension are absent 		O*
Healthcare record contains a diagnosis of non-streptococcal toxic shock syndrome	S	
Death certificate lists non-streptococcal toxic shock syndrome as a cause of death or a significant condition contributing to death	S	
<i>Laboratory evidence</i>		
Negative blood culture for organisms other than <i>Staphylococcus aureus</i> (if obtained)		N
Negative cerebrospinal fluid culture (if obtained)		N
Negative serologies for Rocky Mountain spotted fever (if obtained)		N
Negative serologies for leptospirosis (if obtained)		N
Negative serologies for measles (if obtained)		N

Notes:

S = This criterion alone is sufficient to report a case.

N = All "N" criteria in the same column are required to report a case.

O* = Four of the five clinical criteria are required to meet the criteria for reporting the illness.

† This criterion is not necessary if the patient died before sufficient time elapsed for desquamation to occur.

Case definition

TSS, staphylococcal, last updated by CSTE in 2011

Clinical case definition

An illness with the following clinical manifestations:

- fever: temperature higher than or equal to 102.0°F (higher than or equal to 38.9°C)
- rash: diffuse macular erythroderma
- desquamation (peeling skin): 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 younger than age 16
- multisystem involvement (3 or more of the following organ systems):
 - gastrointestinal: vomiting or diarrhea at onset of illness
 - muscular: severe muscle pain 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 evidence

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 4 of the 5 clinical findings described above are present

Confirmed: a case which meets the laboratory criteria AND in which all 5 of the clinical findings described above are present, including desquamation (peeling skin), unless the patient dies before desquamation occurs

Table 2. Criteria for classifying a case of non-streptococcal toxic shock syndrome.

Criterion	Case definition	
	Confirmed	Probable
<i>Clinical evidence</i>		
Fever (temperature $\geq 38.9^{\circ}\text{C}$ [102.0°F])	N	O*
Diffuse macular erythrodermal rash	N	O*
Desquamation 1-2 weeks after onset of rash†	N	O*
Hypotension (systolic blood pressure ≤ 90 mm Hg for adults or less than fifth percentile by age for children younger than age 16)	N	O*
Multisystem involvement, signified by 3 or more of the following findings: <ul style="list-style-type: none"> - gastrointestinal: vomiting or diarrhea at onset of illness - muscular: severe muscle pain 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 (≥ 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 $< 100,000/\text{mm}^3$ - central nervous system: disorientation or alterations in consciousness without focal neurologic signs when fever and hypotension are absent 	N	O*
<i>Laboratory evidence</i>		
Negative blood culture for organisms other than <i>Staphylococcus aureus</i> (if obtained)	N	N
Negative cerebrospinal fluid culture (if obtained)	N	N
Negative serologies for Rocky Mountain spotted fever (if obtained)	N	N
Negative serologies for leptospirosis (if obtained)	N	N
Negative serologies for measles (if obtained)	N	N

Notes:

N = All “N” criteria in the same column are required to report or confirm a case.

O* = 4 of the 5 clinical criteria are required to meet the criteria for reporting and for the illness to be classified as a probable case.

† This criterion is not necessary if the patient died before sufficient time elapsed for desquamation to occur.

Case investigation process

- Determine if disease meets the criteria of TSS, staphylococcal.
- Complete investigation and appropriate investigation tabs within EpiTrax.
- Identify clusters or outbreaks of this disease promptly and initiate appropriate preventative measures.
- Determine source and manner of spread if applicable.

Outbreaks

An outbreak is defined as 2 or more epidemiologically linked cases in unrelated people occurring at a hospital, school, or childcare facility in a 30-day period. Outbreaks may warrant additional investigation and should be reported to public health.

Identifying case contacts

Generally not recommended unless under unusual circumstances, such as an outbreak.

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Additional resources

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Version control

Updated September 2024: Added “Critical clinician information”, “Why is toxic shock syndrome important to public health?”, “Version control”, and “UT-NEDSS/EpiTrax minimum/required fields by tab” sections. All remaining sections were updated including references. Added CSTE tables.

Updated July 2017: “Critical clinician information”, “Why is toxic shock syndrome important to public health”, “Version control”, “Acknowledgements” and “UT-NEDSS minimum/required fields by tab” sections added. “Reservoir”, “Causative agent”, and “Case fatality” sections were updated. Information added to “Differential diagnosis”, “Treatment”, “Epidemiology”, “Reporting”, and “Case classification” sections.

UT-NEDSS/EpiTrax minimum/required fields by tab

Demographic

- First name
- Last name
- Date of birth
- Birth gender
- Ethnicity
- Race
- State

Clinical

- Date diagnosed
- Date of death
- Died
- Disease
- Onset date

Laboratory

- Collection date

Epidemiological

- Imported from

Reporting

- Date first reported to public health

Administrative

- Outbreak name
- State case status