DESQUAMATING SKIN CONDITIONS

Desquamation is the shedding of the outer layers of the skin. The shedding or peeling of the epidermis in scales. The word comes from the Latin *'desquamare'* , meaning "to scrape the scales off a fish".

SUNBURN!

Risk factors- Photosensitizes

Signs
Sufficient exposure to Ultraviolet Light
Immediate erythema
Occurs within minutes of exposure
Fades within 30 minutes
Delayed erythema
Reappears 3 to 6 hours after exposure
Peaks at 12 to 24 hours
Persists for days

Associated findings - Oedema
Vesicles
Desquamation often within 1 week

Management
Burn Management
Cool wet compresses
Consider topical anaesthetics
Avoid Benzocaine preparations (sensitizer)
Consider Aspirin (adults)
Consider Prednisolone for 4-6 days in severe cases
Adults: 20 mg PO bd for 4-6 days

Prevention- Sunscreen, Decreased sun exposure

Complications- 2 sunburns before age 18 predisposes to Melanoma

KAWASAKI SYNDROME *(BMJ)*

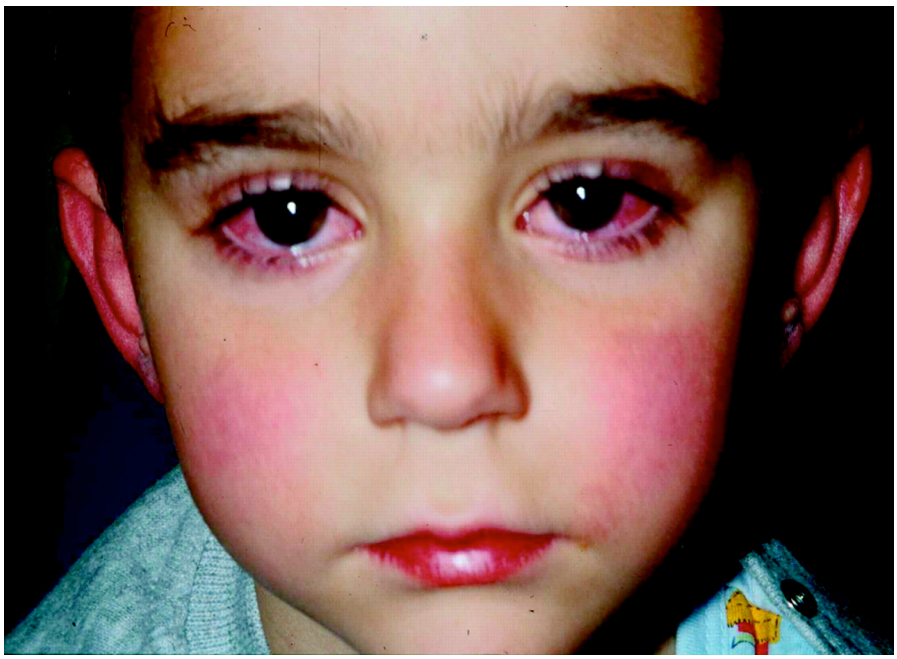
Diagnosis

Kawasaki disease is a systemic vasculitis predominantly affecting children under the age of 5 years. It has a number of classic clinical

features required for diagnosis.

In 1990 the American Heart Association committee on rheumatic fever, endocarditis, and Kawasaki disease[2] gave the case definition that has been generally accepted—i.e., a febrile illness of at least five days with at least four of the five following signs and no other reasonable cause for the findings:

1.Bilateral conjunctival injection – (there is no corneal ulceration but there may be a concomitant anterior uveitis on slit lamp examination)
2.Oral changes (erythema of lips or oropharynx, strawberry tongue due to prominent papillae, or fissuring of the lips)
3.Peripheral extremity changes (oedema, erythema, or generalised or periungal desquamation); erythema is seen in the first week whereas desquamation begins about 14–21 days after the onset of the illness
4.Rash – this starts in the first few days; it is often diffuse and polymorphic and lasts a week before fading. Vesicles are rarely seen; however, the rash can appear macular, maculopapular, urticarial, scarlettina or even morbilliform
5.Cervical lymphadenopathy is found in about 50% of cases; most often there is a painful solitary enlarged lymph gland, > 1.5 cm in diameter



Epidemiology andAetiology

Kawasaki disease is the most common cause of acquired heart disease in

children in the developed world. The exact cause has not yet been established but there is considerable support for it is to be due to an infectious agent causing disease among genetically vulnerable individuals. Kawasaki disease is most common in Japan where rates are 10 times those in the USA, and 30 times that in the UK and Australia; worldwide the annual reported incidence varies from 3.4–100/100 000. Japanese immigrants also have raised rates compared with the population of their new country, supporting the idea of a genetic influence; siblings of index cases in Japan having an increased incidence of 8–9%.

Children under the age of 5 years are predominantly affected, with a peak incidence at 9–11 months. There is a peak occurrence in winter and spring months.

The true incidence is unknown as there is probably underreporting of Kawasaki disease, particularly in its mildest or incomplete form as these patients may not come into contact with medical services—for example, in 1990 five cases in the UK were recognised only post mortem.

Elucidating the aetiology of the disease would direct treatment and provide a more rational basis for its management. Towards this aim there has been considerable focus on a bacterial superantigen toxin being the cause of Kawasaki disease over the past decade; this superantigen is believed to act in a similar fashion to the superantigen toxins of staphylococcal and streptococcal toxic shock syndromes.

There are laboratory based studies that lend support to this hypothesis. One study found that the peripheral blood macrophages/monocytes (which function as antigen presenting cells (APC)) of patients with Kawasaki disease are decreased following the administration of immunoglobulin (IVIG). The APC and the T cell are implicated in superantigen disease, as the superantigen binds across the APC to the variable region of the T cell non-specifically at the V ß2 region hence causing a massive upregulation of T cell activation; as IVIG has considerable benefit in treating Kawasaki disease, this would lend support to the idea of superantigen involvement in its aetiology.

In Kawasaki disease there is a large increase in circulating B cells and fewer T cells; the effect of IVIG in vitro on peripheral lymphocytes is to decrease the percentage of B cells, increase T cells, CD4, CD8 T and CD5 + cells in acute Kawasaki disease (there is a much lesser effect with aspirin alone).

Another in vitro study looking for the superantigen aetiology was more equivocal. In this study three colour flow cytometry was used to look at the T cell antigen receptor variable ß region families from T cells of Kawasaki disease patients. These were examined pre- and post-immunoglobulin treatment and at 40 days after treatment, being compared with matched paediatric patients and one of their own healthy parents. Of all the V ß families studied only VF ß 2 exhibited statistical differences between pre- and post-IVIG, but the authors of this work questioned the importance of this in the underlying pathoimmunology. There was no association between V ß 2 findings and T cell activation or adhesion markers, but with V ß 2 abnormality both CD4+ and CD8+ abnormalities were found. The authors also suggested that patients with Kawasaki disease and cardiac involvement had a more restricted cytotoxic T cell response.

No bacterial agent elaborating a Kawasaki superantigen toxin has been found.

Differntial Diagnosis

1.Streptococcal infection (including scarlet fever, toxic shock-like syndrome)
2.Staphylococcal infection (such as toxic shock syndrome or scalded skin syndrome)
3.Measles, rubella, roseola infantum, Epstein Barr virus, influenza A and B, adenovirus
4.*Mycoplasma pneumoniae*
5.Stevens-Johnson syndrome
6.Systemic idiopathic juvenile arthritis

Complications of Kawasaki disease

1.Irritability and aseptic meningitis
2.Gallbladder hydrops
3.Diarrhoea
4.Hepatitis
5.Otitis media
6.Pancreatitis
7.Myositis
8.Pericarditis and myocarditis
9.Coronary artery Aneurysm formation can lead to peripheral gangrene, cerebral infarction and cardiac arterial aneurysm (this may lead to thrombosis, myocardial infarction and dysrhythmia)

Recommended guideline for the management of Kawasaki disease in the UK

STEVENS JOHNSONS SYNDROME *(Emedicine)*

Pathophysiology: SJS is an immune-complex–mediated hypersensitivity disorder that may be caused by many drugs, viral infections, and malignancies. Cocaine recently has been added to the list of drugs capable of producing the syndrome. In up to half of cases, no specific etiology has been identified.

Mortality/Morbidity:

In 3-15% of cases, patients with severe SJS die.

Lesions may continue to erupt in crops for as long as 2-3 weeks. Mucosal pseudomembrane formation may lead to mucosal scarring and loss of function of the involved organ system. Esophageal strictures may occur when extensive involvement of the esophagus exists. Mucosal shedding in the tracheobronchial tree may lead to respiratory failure.
Ocular sequelae may include corneal ulceration and anterior uveitis. Blindness may develop secondary to severe keratitis or panophthalmitis in 3-10% of patients. Vaginal stenosis and penile scarring have been reported. Renal complications are rare.

Prognosis

The SCORTEN score looks at a number of variables and uses them to prognosticate risk factors for death in both SJS and toxic epidermal necrolysis (TEN). The variables include the following:

Age >40 years
Malignancy
Heart rate >120
Initial percentage of epidermal detachment >10%
BUN >10 mmol/L
Serum glucose >14 mmol/L
Bicarbonate <20 mmol/L

Mortality rates are as follows:

SCORTEN 0-1 $\geq$ 3.2%

SCORTEN 2 ≥ 12.1%
SCORTEN 3 ≥ 35.3%
SCORTEN 4 ≥ 58.3%
SCORTEN 5 or more ≥ 90%

Race: A Caucasian predominance has been reported.

Sex: The male-to-female ratio is 2:1.

Age: Most patients are in the second to fourth decade of their lives; however, cases have been reported in children as young as 3 months.

History

Typically, the disease process begins with a nonspecific upper respiratory tract infection. This usually is part of a 1- to 14-day prodrome during which fever, sore throat, chills, headache, and malaise may be present. Vomiting and diarrhea are occasionally noted as part of the prodrome. Mucocutaneous lesions develop abruptly. Clusters of outbreaks last from 2-4 weeks. The lesions are typically nonpruritic.
A history of fever or localized worsening should suggest a superimposed infection; however, fever has been reported to occur in up to 85% of cases.
Involvement of oral and/or mucous membranes may be severe enough that patients may not be able to eat or drink.
Patients with genitourinary involvement may complain of dysuria or an inability to void.
A history of a previous outbreak of Stevens-Johnson syndrome (SJS) or of erythema multiforme may be elicited. Recurrences may occur if the responsible agent is not eliminated or if the patient is reexposed.
Typical symptoms are as follows:
Cough productive of a thick purulent sputum
Headache
Malaise
Arthralgia

Physical

The rash can begin as macules that develop into papules, vesicles, bullae, urticarial plaques, or confluent erythema.

The center of these lesions may be vesicular, purpuric, or necrotic.
The typical lesion has the appearance of a target. The target is considered pathognomonic.
Lesions may become bullous and later rupture, leaving denuded skin. The skin becomes susceptible to secondary infection.
Urticarial lesions typically are not pruritic.
Infection may be responsible for the scarring associated with morbidity.
Although lesions may occur anywhere, the palms, soles, dorsum of hands, and extensor surfaces are most commonly affected.
The rash may be confined to any one area of the body, most often the trunk.
Mucosal involvement may include erythema, edema, sloughing, blistering, ulceration, and necrosis.
Although some have suggested the possibility of SJS without skin lesions, most believe that mucosal lesions alone are not enough to establish the diagnosis.

The following signs may be noted on examination:
- Fever
- Orthostasis
- Tachycardia
- Hypotension
- Altered level of consciousness
- Epistaxis
- Conjunctivitis
- Corneal ulcerations
- Erosive vulvovaginitis or balanitis
- Seizures, coma

Causes

- Drugs and malignancies are most often implicated as the etiology in adults and the elderly.
- Pediatric cases are related more often to infections than to malignancy or a reaction to a drug.
- A medication such as sulfa, phenytoin, or penicillin had previously been prescribed to more than two thirds of all patients with SJS. Hallgren et al reported ciprofloxacin-induced SJS in young patients in Sweden and commented on several others. Metry et al reported SJS in 2 HIV patients treated with nevirapine and mentioned one other in the literature. That group speculated that the problem may extend to other non-nucleoside reverse transcriptase inhibitors. Indinavir has been mentioned.
- More than half of the patients with SJS report a recent upper respiratory

tract infection.
- The 4 etiologic categories are (1) infectious, (2) drug-induced, (3) malignancy-related, and (4) idiopathic.
- Viral diseases that have been reported include herpes simplex virus (HSV), AIDS, Coxsackie viral infections, influenza, hepatitis, mumps, mycoplasmal infection, lymphogranuloma venereum (LGV), rickettsial infections, and variola.

- Bacterial etiologies include group A beta streptococci, diphtheria, *Brucellosis,* mycobacteria, *Mycoplasma pneumoniae,* tularemia, and typhoid.
- Coccidioidomycosis, dermatophytosis, and histoplasmosis are the fungal possibilities.
- Malaria and trichomoniasis have been reported as protozoal causes.
- In children, Epstein-Barr virus and enteroviruses have been identified.

- Drug etiologies include penicillins and sulpha antibiotics. Anticonvulsants including phenytoin, carbamazepine, valproic acid, lamotrigine, and barbiturates have been implicated. Mockenhapupt et al stressed that most anticonvulsant-induced SJS occurs in the first 60 days of use. In late 2002, the US Food and Drug Administration (FDA) and the manufacturer Pharmacia noted that SJS had been reported in patients taking the cyclooxygenase-2 (COX-2) inhibitor valdecoxib.

- Various carcinomas and lymphomas have been associated.
SJS is idiopathic in 25-50% of cases.

Lab Studies

No laboratory studies (other than biopsy) exist that can aid the physician in establishing the diagnosis.
- A complete blood count (CBC) may reveal a normal white blood cell (WBC) count or a nonspecific leukocytosis. A severely elevated WBC count indicates the possibility of a superimposed bacterial infection.
- Determine renal function and evaluate urine for blood.
- Electrolytes and other chemistries may be needed to help manage related problems.
- Cultures of blood, urine, and wounds are indicated when an infection is clinically suspected.

Imaging Studies

Chest radiography may indicate the existence of a pneumonitis when

clinically suspected. Otherwise, routine plain films are not indicated.

Other Tests

Skin biopsy is the definitive diagnostic study but is not an emergency department (ED) procedure.
Skin biopsy demonstrates that the bullae are subepidermal.
Epidermal cell necrosis may be noted.
Perivascular areas are infiltrated with lymphocytes.

Emergency Department Care

Most patients present early and prior to obvious signs of hemodynamic compromise. The single most important role for the ED physician is to detect SJS early and initiate the appropriate ED and inpatient management.

Care in the ED must be directed to fluid replacement and electrolyte correction.
Skin lesions are treated as burns.
Patients with SJS should then be treated with special attention to airway and hemodynamic stability, fluid status, wound/burn care, and pain control.
Treatment of SJS is primarily supportive and symptomatic. Some have advocated cyclophosphamide, plasmapheresis, hemodialysis, and immunoglobulin, but none of those should be considered standard at this time.
Manage oral lesions with mouthwashes.
Topical anesthetics are useful in reducing pain and allowing the patient to take in fluids.
Areas of denuded skin must be covered with compresses of saline or Burow solution.
Underlying diseases and secondary infections must be identified and treated. Offending drugs must be stopped.
The use of systemic steroids is controversial. Some authors believe that they are contraindicated. Treatment with systemic steroids has been associated with an increased prevalence of complications.
Address tetanus prophylaxis.

Consultations: Consultants may help establish the diagnosis and direct inpatient care. A dermatologist is the most likely clinician to establish the diagnosis, with or without biopsy.

Severe cases may require the involvement of a burn specialist or plastic surgery specialist.
Internal medicine, critical care, or pediatrics consultants direct inpatient care.
Ophthalmology consultation is mandatory for those with ocular involvement.
Depending on organ system involvement, consultations with a gastroenterologist, pulmonologist, and nephrologist may be helpful.

SCALDED SKIN SYNDROME

Pathophysiology: A toxin produced by phage group 2 *Staphylococcus aureus* causes SSSS. An infection commonly occurs at a site such as the oral or nasal cavities, throat, or umbilicus. Two epidermolytic toxins (A and B) are then produced and act at a remote site leading to a red rash and separation of the epidermis beneath the granular cell layer. This is due to binding of the toxins to desmoglein 1 in desmosomes, causing it to break down. Bullae form, and diffuse sheetlike desquamation occurs. Involvement may occasionally be localized or patchy rather than diffuse. Children are more at risk because of lack of immunity and immature renal clearance capability.

Healing typically occurs within 1-2 weeks.

Frequency: In the US: SSSS is most common in children and neonates. It is rare in adults. **Internationally:** Overall incidence is higher in developing countries.

Mortality/Morbidity: Mortality rate from SSSS in children is very low (1-5%), unless associated sepsis or an underlying serious medical condition exists. Mortality rate in adults is higher (as high as 20-30%). Significant morbidity can result from hematologic or local spread of infection.

Sex: No gender predilection is documented in children. In adults, the male-to-female ratio is approximately 2:1.

Age: SSSS primarily is a disease of children.

The disease can occur individually or as outbreaks in nurseries.
Most children (62%) are younger than 2 years, and almost all (98%) are younger than 6 years.
SSSS is very rare in adults, with fewer than 50 cases formally reported in the literature. Adults with SSSS are most often immunocompromised or have renal failure.

History

Staphylococcal scalded skin syndrome (SSSS) presents as a red rash followed by diffuse epidermal exfoliation.
A prodromal localized *S aureus* infection of the skin, throat, nose, mouth, umbilicus, or GI tract occurs. Such an infection often is not apparent before the SSSS rash appears.
General malaise
Fever
Irritability
Skin tenderness

Physical

Fever, although patients may be afebrile
Tenderness to palpation
Warmth to palpation
Diffuse erythematous rash - Image 1Sandpaperlike, Accentuated in flexor creases (see Image 2)
Bullae - Flaccid, Ill-defined
Nikolsky sign (gentle stroking of the skin causes the skin to separate at the epidermis)
Exfoliation of skin, which may be patchy or sheetlike in nature
Facial edema
Perioral crusting
Most patients do not appear severely ill.
Dehydration may be present and significant.

Causes

Infection by group 2 phage *S aureus* (several types) leads to release of exotoxin.
Exotoxin is a protein and is classified as either type A or B. Most are type

A.
Exotoxin causes separation of the epidermis beneath the granular cell layer.
Cases of SSSS have been reported among infants who have breastfed from mothers with *S aureus* breast abscess.
A case has been reported of neonatal SSSS secondary to maternal-fetal transmission at birth.
Outbreaks have been reported in neonatal and newborn nurseries.
Reports are increasing implicating community-acquired methicillin *Staphylococcus aureus* (CA-MRSA) as a cause of SSSS.

Lab Studies:

White blood count (WBC) may be elevated; however, often WBC is normal.
Erythrocyte sedimentation rate (ESR) frequently is elevated.
A polymerase chain reaction (PCR) serum test for the toxin is available.
Cultures of bullae are negative.
Blood culture
Usually negative in children
Usually positive in adults
A Gram stain and/or culture from the remote infection site may confirm staphylococcal infection.

Procedures: Frozen section of the peeled skin confirms the site of cleavage as superficial. Toxic epidermal necrolysis (TEN) shows deeper cleavage below the epidermis.

Emergency Department Care:

The major focus of ED care should be to identify staphylococcal scalded skin syndrome (SSSS) and to stabilize the patient's condition.
Patients need fluid rehydration, topical wound care similar to the care for thermal burns, and parenteral antibiotics to cover *S aureus*.
Consideration must be given for the sharply increasing rates of community-acquired *S aureus* infection (CA-MRSA).
Fluid rehydration is initiated with Lactated Ringer solution at 20 cc/kg initial bolus. Repeat the initial bolus as clinically indicated followed by maintenance therapy with consideration for fluid losses from exfoliation of skin being similar to a burn patient.
Topical wound care should begin with saline, followed by topical antibiotic ointment.

Cultures from the exfoliated sites as well as nose, throat, and other potential sites of the original focus of infection should be performed.
A chest radiograph should be considered to rule out pneumonia as the original focus of infection.
Steroids are not indicated at this time.

Consultations: Paediatrics +/- infectious disease and burn specialists

TOXIC SHOCK SYNDROME

Pathophysiology: TSS is a toxin-mediated disease. Endotoxin toxic shock syndrome toxin-1 (TSST-1) is the major toxin produced by strains of *S aureus* that are responsible for causing TSS. *Streptococcus pyogenes* exotoxin A (SPEA) and *S pyogenes* exotoxin B (SPEB) are the major toxins produced by group A beta-hemolytic streptococci. The toxins activate production of superantigens, such as tumor necrosis factor, interleukin-1, M protein, and gamma-interferon.

Almost every organ system can be involved, including the cardiovascular, renal, skin, mucosa, GI, musculoskeletal, hepatic, hematologic, and central nervous

Mortality/Morbidity: Mortality varies with the bacteria involved.

With staphylococcal TSS, the mortality rate is less than 3%.
With streptococcal TSS, the mortality rate is 30-70%.
Recurrences have been reported in 30-40% of cases.

Race: No race predilection exists in TSS.

Sex: With staphylococcal TSS, the incidence is higher in women than in men. With streptococcal TSS, either sex can be affected.

Age: TSS predominantly occurs in young, healthy individuals. With staphylococcal TSS, patients primarily are aged 15-35 years. With streptococcal TSS, patients primarily are aged 20-50 years.

History

Symptoms are similar for streptococcal TSS and staphylococcal TSS. The major difference is that a source of infection usually is identified with

streptococcal TSS. Symptoms may include the following:

- Prodromal period of 2-3 days
- Fever and/or chills
- Nausea and/or vomiting
- Profuse watery diarrhea with abdominal pain
- Lightheadedness and/or syncope
- Myalgias and/or arthralgias
- Pharyngitis and/or headache
- Confusion (more common with staphylococcal TSS than with streptococcal TSS)
- Pain at site of infection (most common symptom of streptococcal TSS)

Physical

At physical examination, findings may include the following:

- Fever higher than 102°F
- Hypotension - Systolic BP less than 90 mm Hg or orthostatic decrease in systolic BP of 15 mm Hg
- Skin findings
- Diffuse rash, occasionally patchy and erythematous, with desquamation occurring approximately 1-2 weeks later
- Rash initially appearing on trunk, spreading to arms and legs, and involving palms and soles
- Signs of multiorgan involvement
- Physical findings associated with ventricular arrhythmias, renal failure, or hepatic failure
- DIC
- ARDS
- Necrotizing fasciitis and/or myositis
- Altered consciousness (CNS involvement)
- Mucosal inflammation (e.g., vaginitis, conjunctivitis, pharyngitis)

The Centers for Disease Control and Prevention (CDC) criteria for the diagnosis of staphylococcal TSS are as follows:

-Fever, hypotension, and rash (as defined above)
- Involvement of 3 or more organ systems
- Absence of serologic evidence of Rocky Mountain spotted fever, leptospirosis, measles, hepatitis B, antinuclear antibody, positive Venereal Disease Research Laboratory (VDRL) test results, and antibodies at Monospot testing

The CDC criteria for streptococcal TSS are as follows:

- Isolation of group A streptococcus from a normally sterile site (e.g., blood, cerebrospinal fluid [CSF], surgical wounds) or a nonsterile site (e.g., throat)
- Hypotension (as defined above)
- Involvement of 2 or more organ systems

Causes

An absence of protective immunity is postulated to be a major risk factor for acquisition and recurrence of TSS.

TSS is caused by coagulase-positive staphylococci (*S aureus*) and group A beta-hemolytic streptococci (*S pyogenes*).

Risk factors include the following:
- Use of superabsorbent tampons
- Postoperative wound infection
- Postpartum toxic shock
- Nasal packing
- Common bacterial infections
- Viral infection with influenza A or varicella
- Diabetes mellitus
- Infection with HIV
- Chronic cardiac and/or pulmonary disease
- An association of TSS with prior use of nonsteroidal anti-inflammatory drugs has been suggested, but a causal relationship has not been established.

Lab Studies

The CBC may reveal leukocytosis (77% of cases) with bandemia, mild anemia with abnormal cells on smears, and/or thrombocytopenia.
Electrolyte levels may indicate hyponatremia, hypokalemia, hypocalcemia out of proportion to hypoalbuminemia, hypophosphatemia, and hypomagnesemia.
Liver function test results (at least 2 times normal levels) may reveal hyperbilirubinemia (76% of cases), an elevated aspartate aminotransferase (SGOT) level (75% of cases), and an elevated alanine aminotransferase (SGPT) level (50% of cases).
Coagulation studies may reveal an elevated activated partial thromboplastin time (aPTT) (46% of cases) and fibrin split products.

Fibrinogen levels and prothrombin times (PTs) usually are normal.
Azotemia and/or acute tubular necrosis may be present.
Urinalysis may reveal sterile pyuria, myoglobinuria, and red cell casts.
Creatine kinase levels may indicate rhabdomyolysis (63% of cases).
ABG findings may indicate metabolic acidosis secondary to hypotension and/or hypoxia.
Culture all potentially infected sites (including blood). More than 50% of patients with streptococcal TSS have a positive blood culture result.
Recent studies have suggested that observing the expansion of TSS-1 reactive V beta2-positive T-cell receptors in peripheral blood mononuclear cells can make early and definitive diagnosis.

Imaging Studies

A CXR may show evidence of ARDS or pulmonary oedema.
Radiographs of the infected site may show soft-tissue swelling.
An echocardiogram may show wall-motion abnormality suggestive of toxic cardiomyopathy.
A CT scan should be obtained if the diagnosis is in question. Findings should be normal in TSS.

Other Tests

The ECG may show the following:

- Ventricular arrhythmias
- Bundle branch blocks
- First-degree heart block
- ST-T–wave changes, with ischemia

Serologic tests should be performed to assess Rocky Mountain spotted fever, leptospirosis, measles, hepatitis B surface antigen, and antinuclear antibody. The VDRL test and the Monospot antibody test also should be performed.
The rapid streptococcal test can be performed in 10-15 minutes. The test has a sensitivity of 87-95%.

Procedures: Lumbar puncture should be performed if the diagnosis is in question. Findings should be normal in TSS.

Swan-Ganz catheterization should be performed to enable monitoring of fluid resuscitation, pulmonary capillary wedge pressure, systemic vascular resistance, and cardiac output.

Emergency Department Care

Aggressive fluid resuscitation should continue in the ED. Administer oxygen therapy. Continuously monitor heart rate, respiratory rate, and BP.

The use of intravenous immunoglobulin G (IVIG) has been shown to be effective in neutralizing the TSS toxin and therefore aids in recovery.

Fluid resuscitation:
Crystalloids may be administered. As much as 10-20 L/d often is necessary.
Some authors suggest that colloids may decrease the risk of pulmonary oedema.

Oxygen therapy:
Administer supplemental oxygen therapy to maximize tissue oxygenation and to correct hypoxia and/or acidosis.
Assisted ventilation may be required if acute respiratory distress syndrome develops.
Hyperbaric oxygen therapy has been used in necrotizing soft-tissue infections, but the benefit of this intervention has not been proven.

Anibiotics: Naficillin (penicillin G-resistant staphylococcal infections)
Clindamycin (invasive group A streptococcal infections)
Erythromycin

Cardiac monitoring should be performed, with treatment of high-grade arrhythmias.
A Foley catheter should be placed to monitor urine output (assess adequacy of resuscitation).

Tampons and packing materials, if present, should be removed.
For patients with menstruation-related TSS, irrigation of vagina with isotonic sodium chloride solution or povidone-iodine solution has been advocated.

Consultations: Prompt consultation with a surgeon may be necessary for drainage, débridement, fasciotomy, or amputation of a clearly infected site.