A Case Of Toxic Shock Syndrome

P Kalikiri, J Kandala, R Singh Sachan

Citation

P Kalikiri, J Kandala, R Singh Sachan. *A Case Of Toxic Shock Syndrome*. The Internet Journal of Infectious Diseases. 2005 Volume 5 Number 1.

Abstract

Toxic Shock Syndrome (TSS) can be classified as menstrual and nonmenstrual based on the etiology. Menstrual and nonmenstrual cases account for approximately equal percentage of the reported TSS cases (1,2). Common nonmenstrual causes of TSS include surgical and postpartum wound infections, mastitis, sinusitis, osteomyelitis, arthritis, burns, cutaneous and subcutaneous lesions (especially of the extremities, perianal area, and axillae), and respiratory infections following influenza. Staphylococcus aureus, a highly virulent and ubiquitous aerobic gram-positive coccus is commonly implicated in menstrual cases of TSS. TSS toxin-1 (TSST-1), an exotoxin isolated from S. aureus is the principal agent involved in the pathogenesis of menstrual TSS (3,4) in addition to other enterotoxins such as enterotoxins A, C, D, E, and H, which have been implicated only in a smaller number of menstrual TSS cases. The incidence of menstrual TSS cases has declined from 9 out of 100,000 women in 1980 to 1 out of 100,000 women since 1986 (1). The case-fatality rate for menstrual TSS has also declined from 5.5 percent in 1979-1980 to 2.8 and 1.8 percent in 1981-1986 and 1987-1996, respectively (5). The decrease in the incidence of menstrual cases of TSS is partially explained by the withdrawal of highly absorbent tampons and polyacrylate rayon-containing products from the market; however, tampon use remains a risk factor for TSS (6). It is believed that women who use tampons with higher absorbencies, use tampons continuously for more days of their menstrual cycle, and keep a single tampon in place for a longer period of time are more likely to develop TSS.

CASE REPORT

A 37 year old female with no significant past medical history and who was menstruating since Dec 10, 2004 presented to our hospital on Dec 16, 2004 with fever, body aches and redness of the eye of 3 days duration; rash of 2 days duration; swelling of hands; nausea, vomiting and diarrhea of 1 day duration. The fever was high grade, intermittent and associated with rigors and sweating. The body aches were generalized, continuous, 7/10 in severity, no aggravating factors and relieved by Tylenol. Redness of the eye was insidious in onset, progressively increased in severity, not associated with any discharge and no visual disturbance. The rash first started on the hands and forearm, progressed to involve the whole body and was non prurutic. The swelling of hands was insidious in onset and progressively worsening in nature. She had three episodes of loose watery diarrhea, yellowish in color; non-bloody and non foul smelling. She threw up twice; the vomitus was non-bloody and nonbilious.

She denied history of recent travel, insect bite, joint pain and swelling, contact with sick persons, loss of appetite, loss of weight, chest pain, orthopnea, PND, palpitations, pedal edema, dizziness, syncope, visual disturbances, ear problems, seizures, weakness, bladder and bowel disturbances.

Her menstrual history is positive for heavy and prolonged periods of 7 days duration. She uses tampons and changes them everyday.

No previous hospitalizations or surgeries in the past. No known drug allergies and no other allergies. She was taking Tylenol for fever and body aches. She denied drug abuse, smoking and alcohol abuse. She works in a home care center, divorced and living with two children. She is sexually active with one male partner and admits to having unsafe sex. She had a HIV testing 2 years ago that was negative. She had a PPD testing 9 years ago that was negative. No significant family medical history.

PHYSICAL EXAM

Vitals: BP- 86/62 (Orthostatic drop of 18mm Hg), Temperature- 102.3, Pulse- 110 (regular), Respiratory rate-18.

On examination the patient was conscious, alert and oriented and appeared to be in acute distress. Skin examination showed diffuse, red, macular rash (resembling sunburn) all over the body. Examination of the eyes showed conjunctival injection. Oral cavity revealed hyperemia of oropharyngeal mucosa. Examination of extremities showed swelling of hands with no desquamation at the time of admission. Examination of the genitalia revealed vaginal hyperemia, peri-vaginal rash and bloody discharge. Examination of ear, nose, neck, breasts, lungs, heart, abdomen, rectum, lymph nodes, peripheral pulses and nervous system was normal.

LABS

CBC was remarkable for WBC of 17 x 103 U/L, platelets of 118 x 103 U/L, segments-97.2%, lymphocytes-1.8%, bands-37% and toxic granulations. Basal metabolic panel was remarkable for Na of 131.4 mEq/L, BUN of 16, creatinine of 1.3, calcium of 6.8 and magnesium of 1.3 mEq/L. PT/PTT/INR were normal. Urine pregnancy test was negative. Urinalysis was remarkable for protein of 30 mg/dl, trace ketones, moderate leukocyte esterase, WBC of 5-10, many bacteria and small bilirubin. Rubella and measles IgG were positive and immune. Blood, Urine and CSF cultures showed no growth. Genital swab showed no growth. CSF cell count with differential, gram stain, protein and glucose were normal. ESR was normal, CRP was elevated (17 mg/dl) and ANA was negative. Hepatic panel was remarkable for GGT of 70 IU/L, ALT of 164 IU/L, Albumin of 2.5 g/dl and Total protein of 4.8 g/dl.

HOSPITAL COURSE AND OUTCOME

Our differential diagnosis was TSS, Rocky Mountain spotted fever, Meningococcemia, Bacterial endocarditis, Kawasaki's disease and Drug allergy. A probable diagnosis of TSS was made based on CDC case definition (7) showed in Table I. The patient had five of the six clinical findings showed in Table I. The patient received supportive therapy with IV fluid replacement, acetaminophen and analgesics. We started the patient on clindamycin because it suppresses protein and hence toxin synthesis by S. aureus, thus believed to be more more efficacious than cell wall active agents such as beta lactams (₈₇₉). She became afebrile (>24 hours) and her blood pressures were stable at the end of second day of treatment. Her rash and swelling of hands disappeared completely without any desquamation on day three of treatment. Since the patient wanted to go home as soon as possible to take care of her kids, we discharged her on day 4 with oral cephalexin for 10 days. The patient was seen in the clinic two weeks after hospital discharge. She had completely recovered at that time; however she had

desquamation of the skin of both hands, which she first noticed 6 days after hospital discharge. **Figure 1**

Table 1

Case Definition of Taxic Shock Syndrome from the CDC* Form Taxic Shock (102.0%) Dystolic Bloop resurce 500 mm/dy orthostatio drop in diastolic 300 mm/dy orthostatio 300 mm

CORRESPONDENCE TO

Pramood C Kalikiri, M.D., Ms Department Of Medicine 82-68 164 Street N705, Jamaica, New York 11432, USA. Email: Pramood.Kalikiri@Mssm.Edu,

Drkalikiri@Yahoo.Com,Drkalikiri@Gmail.Com.

References

1. Reduced incidence of menstrual toxic-shock syndrome: United States 1980-1990. Morb Mortal Wkly Rep 1990; 39:421.

2. Gaventa, S, Reingold, AL, Hightower, AW, et al. Active surveillance for toxic shock syndrome in the United States, 1986. Rev Infect Dis 1989; 11 Suppl 1:S28.

3. Bergdoll, MS, Crass, BA, Reiser, RF, et al. A new staphylococcal enterotoxin, enterotoxin F, associated with toxic-shock-syndrome Staphylococcus aureus isolates. Lancet 1981; 1:1017.

4. Schlievert, PM, Shands, KN, Dan, BB, et al. Identification and characterization of an exotoxin from Staphylococcus aureus associated with toxic-shock syndrome. J Infect Dis 1981; 143:509.

5. Hajjeh, RA, Reingold, A, Weil, A, et al. Toxic shock syndrome in the United States: surveillance update, 1979 1996. Emerg Infect Dis 1999; 5:807.

6. Broome, CV. Epidemiology of toxic shock syndrome in the United States: overview. Rev Infect Dis 1989; 11 Suppl 1:S14.

7. Case definitions for infectious conditions under public health surveillance. Morb Mortal Wkly Rep 1997; 46:39. 8. Parsonnet, J, Modern, PA, Giacobbe, KD. Effect of subinhibitory concentrations of antibiotics on production of TSST-1 [abstract 29]. Program and Abstracts of the 32nd Meeting of the IDSA, Orlando. 1994:6A.

9. Schlievert, PM, Kelly, JA. Clindamycin-induced suppression of toxic-shock syndrome--associated exotoxin production. J Infect Dis 1984; 149:471.

Author Information

Pramood C. Kalikiri, M.D., M.S. Mount Sinai School Of Medicine

Jagdesh Kandala, M.D. Mount Sinai School Of Medicine

Reena Sachan Gajraj Singh Sachan, M.B.B.S. Mount Sinai School Of Medicine