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Case Report

Staphylococcus Aureus Toxic Shock Syndrome With Urinary Tract Infection

Medha Gudavalli; M.D.[1], Jenny Thai; M.D.[1], Divya Reddy; M.D.[1], Caitlin MacMillen; D.O.[1], Michele Ritter; M.D.[2], Madison Kent; M.D.[3]

1 Department of Family Medicine, University of California San Diego, San Diego, California, United States of America
2 Department of Infectious Diseases, University of California San Diego, San Diego, California, United States of America
3 Department of Obstetrics and Gynecology, University of California San Diego, San Diego, California, United States of America

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Abstract

Objective: To describe a rare case of non-menstrual staphylococcal toxic shock syndrome (TSS) with Staphylococcus aureus urinary tract infection as the suspected primary source and to highlight the successful use of linezolid as adjunctive therapy when clindamycin resistance is present.

Case report: A 42-year-old woman presented with fever, vomiting, diarrhea, and severe vulvar pain. One month prior, she experienced vaginal pruritus with excoriations and fissuring that never fully resolved. On examination, she had conjunctival injection, tongue inflammation with non-vesicular lesions, and a diffuse erythematous macular rash. Laboratory evaluation revealed elevated transaminases, thrombocytopenia, and bandemia. Urinalysis demonstrated 100,000 CFU of Staphylococcus aureus. Gynecologic examination revealed vulvar and vaginal erythema with ulcerated cervical lesions. Vaginal culture also grew heavy Staphylococcus aureus. Given the constellation of fever, rash, mucosal involvement, hepatic dysfunction, and positive Staphylococcus aureus cultures, a diagnosis of toxic shock syndrome was made. The isolate was resistant to clindamycin, necessitating treatment with vancomycin, piperacillin-tazobactam, and linezolid. The patient improved and completed 14 days of antibiotic therapy with complete resolution of symptoms.

Conclusion: Staphylococcus aureus urinary tract infection is an exceedingly rare source of toxic shock syndrome. Clinicians should maintain a high index of suspicion for TSS in patients with any staphylococcal infection presenting with fever and multisystem involvement. Linezolid is an effective alternative to clindamycin for toxin suppression, particularly in cases involving clindamycin-resistant organisms. Prior mucosal barrier disruption may be an important risk factor for TSS development.

Keywords: Shock; Toxic; Staphylococcus Aureus; Urinary Tract Infections; Vaginitis; Linezolid; Drug Resistance, Bacterial; Superantigens; Anti-Bacterial Agents

Introduction

Toxic shock syndrome (TSS) was first described in 1978 by Todd et al (1). It was defined as a rare, but life-threatening, illness characterized by the rapid onset of fever, rash, and hypotension with multiorgan system involvement. The most common pathogens are *staphylococcus aureus* (*S. aureus*) and

Correspondence:
Dr. Medha Gudavalli
Email: mgudavali@health.ucsd.edu

streptococcus pyogenes (*S. pyogenes*) (2, 3).

Pathogenesis is mediated by toxins, superantigens, and host immune response. TSST-1 exotoxin isolated from *S. aureus* is produced by 40-60% of strains associated with nonmenstrual cases (4). Other implicated toxins include enterotoxins A, B (5), C, D, E, and H (6, 7). These toxins activate a large number of T cells, resulting in massive cytokine production (8). They also interact directly with glycoproteins on antigen presenting cells to create nonspecific T-cell activation, leading to IL-1, IL-2, TNF-alpha, TNF-beta, and IFN-gamma creation (9). Host immune response also plays a role since people with TSS have been shown to have an insufficient antibody response to TSST-1 and other enterotoxins (10, 11).

While classically associated with menstruation and tampon use, half of all TSS cases is nonmenstrual (12, 13) and are associated with comparable (14) if not higher, mortality rates (15). Skin and soft tissue infections are the most common sources of infection including, but not limited to, burns, post-surgical wounds, postpartum infections, upper respiratory tract infections, and osteomyelitis (2, 3, 14). Urinary tract infections (UTI) are rare primary sources since *S. aureus* is an atypical uropathogen. There have been two documented cases so far: a complication of continent urinary diversion (16) and a pediatric case report with toxic-shock-like syndrome with *staphylococcus epidermidis* cultured from urine (17). Another rare primary source of infection is relation to intrauterine contraceptives (IUC). Of these, the majority have been reported to be streptococcal organisms associated with insertion of Mirena IUC (18).

Many cases of TSS do not have a confirmed source of infection. Risk factors are thought to include colonization with toxin-producing strains even without an obvious infection focus, particularly after the disruption of skin or mucous membranes (3).

This case report describes a patient with nonmenstrua*l S. aureus* TSS suspected to have originated from *S. aureus* UTI vs. *S.aureus* vaginitis. It highlights the importance of maintaining clinical suspicion for TSS in patients with any staphylococcal infection presenting with systemic symptoms and multisystem involvement.

Case report

A 42-year-old woman presented to the ED with fever and severe vulvar burning. Of note, the patient has a past medical history notable for copper IUD (placed 9 years prior to presentation), well controlled vulvar vestibulitis with vaginal hormone therapy, and pelvic floor dysfunction.

One month prior to presentation, the patient experienced vaginal pruritus resulting in excoriation and fissuring. She was treated for yeast vaginitis at that time, and she recalled that her vulvar tissue never fully returned to baseline.

Two days prior to presenting to the ED she experienced vomiting and diarrhea. One day before arrival, she developed dysuria and progressively worsening and severe vulvar pain. Self-examination revealed discolored, inflamed vaginal tissue with white discharge. She was evaluated at her primary care office, and her external genital exam was notable for moderate amount of white vaginal discharge and fissuring without ulceration, most prominent at the posterior vaginal introitus. Vaginitis PCR was collected from the vaginal discharge and areas of fissuring were tested for HSV/VZV via PCR. She was empirically treated for candidal vulvovaginitis with fluconazole. The following day her urinalysis revealed 100,000 CFU of *S. aureus* and she took 1 dose of trimethoprim-sulfamethoxazole. Despite treatment with antibiotics, fluconazole, acetaminophen, and phenazopyridine, her symptoms progressed. She developed pruritus and erythema of her hands and feet with mild skin cracking, conjunctival injection, and oral pain with tongue inflammation, prompting ED evaluation.

Upon arrival, vitals were notable for mild tachycardia (heart rate 104 beats per minute), temperature of 37.3 degrees Celsius, blood pressure 131/87 mmHg, respiratory rate of 20 breaths per minute, and oxygen saturation of 100% on room air. Two hours later, she developed a fever of 38.5 degrees Celsius. Physical examination demonstrated mild scleral injection, an erythematous coarse tongue with numerous non-vesicular lesions on the posterior aspect, abdominal tenderness, costovertebral angle tenderness, and erythema of bilateral digits on both dorsal and palmar surfaces with mild skin cracking.

Laboratory evaluation revealed normal creatinine (0.71 mg/dL) but mildly elevated transaminases (ALT 70 U/L, AST 77 U/L), which were previously normal. White blood cell count was normal ($8.8 \times 10^3/mm^3$) but with notable bandemia (28%). Platelet count was decreased at $130 \times 10^3/mm^3$ (previously normal). Lactate was within normal limits (1.2 mmol/L). Inflammatory markers showed ESR 11 mm/hr and CRP 14.40 mg/dL. Broad-spectrum

antibiotics were initiated with vancomycin 1 g every 12 hours and piperacillin-tazobactam 4.5 g every 8 hours.

Gynecologic examination revealed two 2-3 mm hypopigmented white lesions on the left labia minora near the clitoral hood. Well-circumscribed erythema extended from the 4 o'clock to 8 o'clock position on the medial aspect of the labia minora at the introitus, with multiple small (1 mm) non-tender, non-ulcerated nodules within the erythematous area. Speculum examination demonstrated subcentimeter ulcerated lesions at the 12 and 1 o'clock positions on the ectocervix and upper vaginal wall near the cervix, with erythema and speckling of both vaginal fornices. Moderate yellowish vaginal discharge was present, and IUD strings were visible. A fine, diffuse, erythematous macular rash extending from the elbows and knees outward was noted, which had not been present the previous day.

Given her fever, oromucosal symptoms, rash, elevated liver enzymes, thrombocytopenia, and positive *S. aureus* urine culture, a diagnosis of TSS was made. The initial treatment of vancomycin and piperacillin-tazobactam was continued with the addition of clindamycin. Acyclovir was added empirically to cover possible HSV infection based on the physical exam findings. When urine culture susceptibilities revealed clindamycin resistance, clindamycin was replaced with linezolid while continuing vancomycin and piperacillin-tazobactam.

On hospital day 2, the patient developed swelling and pruritus of her neck, torso, hands, lips, and face, raising concern for vancomycin infusion reaction. Following Infectious Disease consultation, she was transitioned to cefazolin and linezolid and then discharged home on a 14-day course of linezolid.

Microbiologic studies revealed a urine culture with growth of *S. aureus* resistant to clindamycin, erythromycin, and penicillin G. Vaginal culture grew heavy *S. aureus* with the same resistance pattern. The IUC was removed during hospitalization and also cultured positive for *S. aureus*. It is unclear if this represented secondary infection vs contamination from vaginal flora during transvaginal removal, rather than a primary source of infection. Vaginitis PCR was negative for bacterial vaginosis, Candida species, *Candida glabrata*, and *Trichomonas vaginalis*. Chlamydia and gonorrhea testing was negative. HSV-1/2 and VZV nucleic acid detection from both initial and repeat samples of ulcerated lesions were negative. Blood cultures showed no growth. Serologic testing revealed positive EBV IgG (negative IgM), negative HSV-1 and HSV-2 antibodies, and negative *Mycoplasma pneumoniae* IgG.

Following discharge, she developed uptrending liver function tests, and linezolid was changed to amoxicillin-clavulanate. She completed 14 total days of antibiotic therapy. Her vaginal lesions resolved completely, and a test-of-cure urine culture was negative. Her liver enzymes returned to normal, and her skin peeling ceased after 2 weeks. 1 month after discharge she continued to have fatigue, brain fog, and altered taste.

Discussion

This case of TSS was unique as the source of infection was highly unusual: *S. aureus* UTI, *S. aureus* cervicitis/vaginitis. We suspect the vaginal pruritus preceding this infection may have created mucosal disruption, allowing for colonized toxin-producing MSSA to become pathogenic. It is also hypothesized that the urinary tract was the primary source, with secondary vaginal involvement due to anatomic proximity and local inflammation. The recovery of *S. aureus* from the IUD likely represents secondary infection vs contamination during transvaginal removal rather than indicating the IUD as a primary source of infection. The patient had a long-standing IUD (9 years) without prior complications. While IUD-associated TSS has been reported, most cases involve streptococcal TSS occurring shortly after insertion (19, 20).

In the treatment of TSS, current recommendations include a beta-lactam or vancomycin (in areas with high methicillin-resistant *S. aureus* prevalence) combined with a protein synthesis inhibitor such as clindamycin to reduce toxin production (2). This patient's *S. aureus* was resistant to clindamycin, necessitating the use of linezolid. Recent evidence suggests linezolid is non-inferior to clindamycin for invasive streptococcal infections (21, 22) and may have an increasing presence given increasing clindamycin resistance.

The development of hepatotoxicity during linezolid therapy is a recognized adverse effect and was mitigated by transitioning to amoxicillin-clavulanate. A mechanistic drawback of amoxicillin-clavulanate monotherapy is the lack of toxin suppression. However, with the patient’s rising liver enzymes, the risk of hepatotoxicity outweighed the benefit of toxin suppression in the post-acute phase. The total duration of 14 days of antimicrobial therapy

is consistent with recommendations for severe staphylococcal infections with source control.

Conclusion

This case highlights the importance of maintaining a high index of suspicion for TSS in patients with *S. aureus* infections presenting fever and multisystem involvement, even when the source is an uncommon one such as UTI. Prompt recognition and appropriate antimicrobial therapy including toxin-suppressing agents are essential for quicker recovery and improved clinical outcomes. Linezolid is an effective alternative to clindamycin, particularly in cases involving clindamycin-resistant organisms. The highly unusual source of infection for this case of TSS emphasizes the importance of early broad infectious work up to identify all potential sources and expeditious sensitivity testing.

Conflict of Interests

Authors declare no conflict of interests.

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