Case Report

A Case of *Staphylococcus* Toxic Shock Syndrome Presenting with Multiple Pneumatocoeles in the Chest

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ABSTRACT

Staphylococcus toxic shock syndrome is a severe illness caused by infection with toxin producing *Staphylococcus aureus* and is associated with a poor outcome. We report a case of *Staphylococcus* TSS presenting with cough and expectoration along with multiple pneumatoceles visible on the chest radiograph that progressed to acute respiratory distress syndrome with eventual foci in brain. The patient was aggressively managed and recovered completely.

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Key words: Acute respiratory distress syndrome, *Staphylococcus aureus*, Pneumatoceles, Toxic shock syndrome, Pyelonephritis, Septic emboli.

INTRODUCTION

Staphylococcus aureus toxic shock syndrome (TSS) is a potentially fatal, multi-system disorder presenting with fever, hypotension, myalgia, vomiting, diarrhoea, mucosal hyperemia and an erythematous rash that desquamates subsequently during convalence. Most often, this is associated with infection of mucosal or sequestered sites by TSS toxin (TSST)-producing *S. aureus* strains usually belonging to bacteriophage group 1 (TSST-1). *Staphylococcus* enterotoxins and TSST-1 are superantigens that are potent activators of T-lymphocytes.[1] The term "superantigen" was adopted to describe the ability of these toxins to cause a remarkable expansion of T-lymphocytes displaying specific β chain variable regions of the T-cell antigen receptor.[2] Super antigens bypass normal antigen presentation and can stimulate over 20% of all T-cells, whereas a conventional antigen stimulates T-cells only in the order of 1 in 10000 T cells.

Two distinct categories of TSS are known — the menstrual (forming 99% of the reported cases) related to the use of intravaginal tampons and the non-menstrual, forming only a small fraction of cases. The case presented here was a non-menstrual form of TSS presenting with respiratory manifestations.

CASE REPORT

A 22-year-old female patient presented to the emergency department with complaints of cough with expectoration, rapid and laboured breathing, a reduced urinary output, a history of abortion 15 days back followed by high grade fever and pain in the left hip with restriction of movement for the past 10 days. Her vital signs were: blood pressure-100/70 mmHg, pulse rate-112 breaths per minute, oral temperature-102 °F, respiratory rate-30 per minute, SpO_2 -80% breathing room air. Examination of the patient revealed bilateral coarse crepitations, reduced air entry, tachypnoea, tenderness in the lower abdomen, pain and restriction of movement of the left hip joint. Arterial blood gas analysis showed mixed respiratory and metabolic acidosis with type-2 respiratory failure. Chest radiograph (postero-anterior view) revealed multiple pneumatoceles (Figure 1). Laboratory investigations were as follows: white cell count 5200/mm^3, haemoglobin 6g/L%, platelet count 1.2 lakhs/mm^3, blood urea 238.2mg% and serum creatinine 5.4mg%, SGOT=54U/L, SGPT=48U/L, total serum proteins 5.7g%, serum albumin 2.8g%, serum globulin 2.9 g%, A:G ratio 0.96:1, alkaline phosphatase 555U/L, calcium 7.3mg%, sodium 125mg%, potassium 3.8mg%. Urine examination revealed albumin 3+, pus cells 15-20/hpf and red blood cells 10-15/hpf. Blood, urine and vaginal fluid and synovial fluid from the involved hip joint specimens were obtained for culture for bacterial pathogens.

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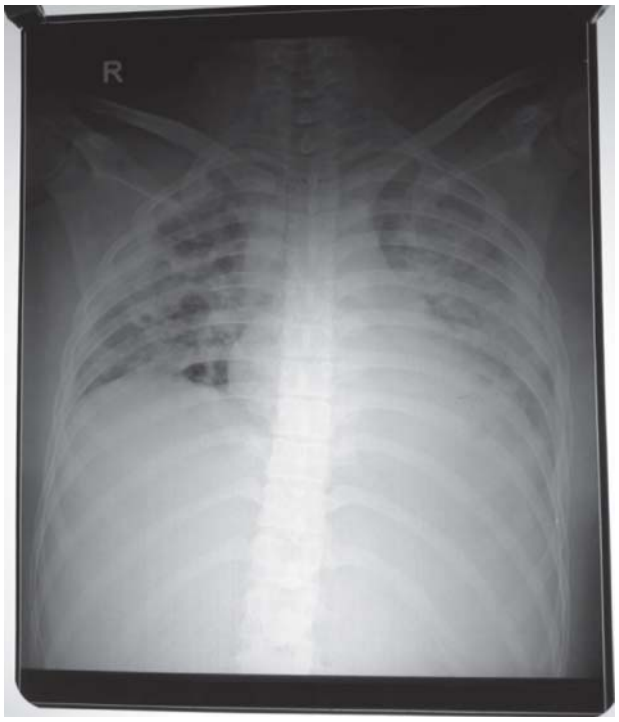

Figure 1. Chest radiograph (postero-anterior view) showing pneumatoceles before treatment.

The patient was admitted to the intensive care unit with a presumptive diagnosis of acute pyelonephritis associated with acute renal failure leading to ARDS. Emperically, broad-spectrum antibiotics those included ceftriaxone, piperacillin-tazobactam and clindamycin combination were started. An emergency ultrasound to evaluate the uterus and the adenexa was normal. There were no positive findings on a per vaginal examination. Haemodialysis was done. The patient had tachycardia and hypotension, requiring large volumes of fluid and vasopressor support to maintain her blood pressure.

The blood and urine cultures did not yield any pathogen. A direct microscopy of hip aspirate showed pus cells along with gram positive cocci. The culture of the hip aspirate grew *S. aureus*. Vaginal cultures on blood agar after incubation for 24 hours grew pin-head colonies of golden colour that were catalase positive suggesting *Staphyloccous* species. Positive slide coagulase and tube-coagulase tests, and mannitol positivity confirmed the presence of *S. aureus*. The organism was sensitive to ampicillin/sulbactam, doxycycline, linezolid, vancomycin and teicoplanin. Methicillin resistance was confirmed as it was resistant to oxacillin and cefoxitin. The antibiotics were accordingly changed to include linezolid and vancomycin. However, clindamycin was continued as the patient showed clinical improvement. Gradually, the condition of the patient improved, with decrease in respiratory distress and acidosis. Desquamating erythematous rashes appeared on the palms and toes (Figure 2). On the 20th day, the patient had an episode of seizure and a subsequent magnetic resonance imaging of the brain revealed septic emboli in the parietal and occipital regions (Figure 3).

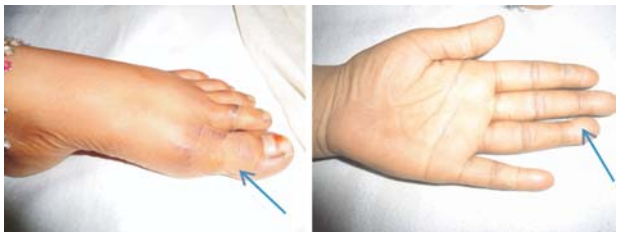

Figure 2. Photograph showing a desquamating rash developing during convalence in a case of *Staphylococcus* toxic shock syndrome.

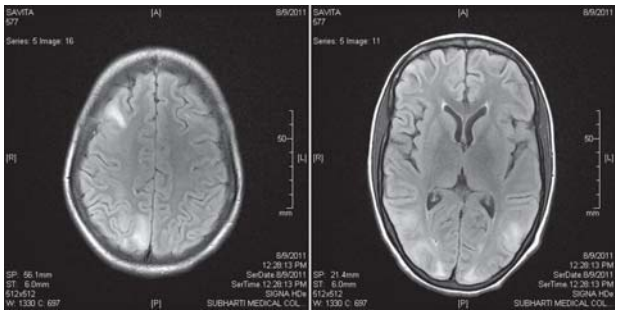

Figure 3. Magnetic resonance imaging of the brain showing septic emboli in the parietal and occipital regions.

The patient eventually recovered completely with complete resolution of the pneumatoceles on chest radiograph (Figure 4) and was discharged from the hospital after 40 days of in-patient treatment.

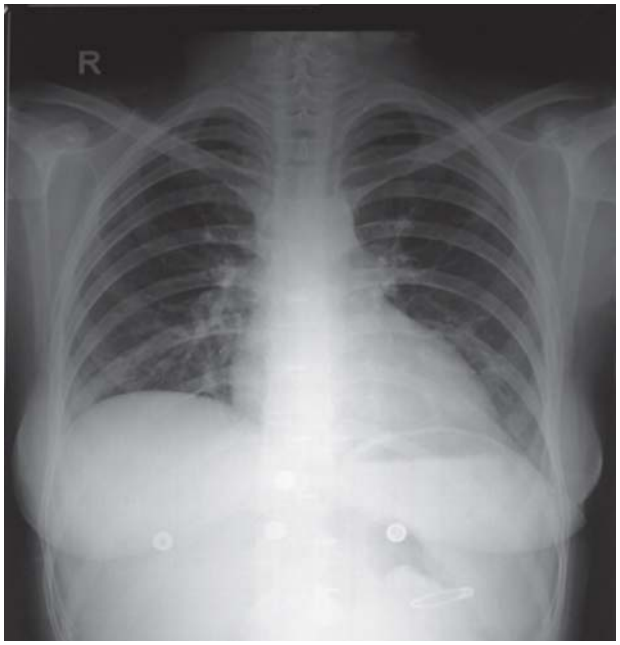

Figure 4. Chest radiograph (PA view) complete resolution of pneumatoceles after treatment.

DISCUSSION

The term TSS was first coined in 1978 by Todd *et al*,[3] who reported the symptom complex in a group of seven children aged 8 to 17 years with an acute febrile illness. During the next few years, the number of cases increased, and TSS typically became associated with young menstruating women, though both genders were affected. It is now known that three conditions are required to develop TSS: (1) the patient is colonised or infected with *S. aureus*, (2) the bacteria produce toxic shock syndrome toxin-1 (TSST-1) or similar toxins, and (3) the toxins have a route of entry into the circulatory system.[4] TSS is caused by a strain of *S. aureus* that produces the toxins TSST-1 and enterotoxins A.[5] The TSST-1 suppresses neutrophil chemotaxis, induces T-suppressor function, and blocks the reticulo-endothelial system.[6] The toxins act together as super-antigens that stimulate the release of various cytokines, prostaglandins, and leukotrienes, that produce the signs and symptoms of the syndrome.[7] The TSST-1 produces an antibody response *in vivo* that is believed to be protective.

Pneumatoceles are thin-walled, air-filled cysts that develop within the lung parenchyma. These may be single, emphysematous lesions but are more often multiple, thin-walled, air-filled, cyst like cavities. Pneumatocele formation is much more common in children, but rare in adults. The typical signs and symptoms of TSS are a high fever (38.9 °C), headache, vomiting, diarrhoea, myalgias, and an erythematous rash characterised as a sunburn. Other signs and symptoms may include meningismus,[8] pharyngitis, conjunctivitis, vaginitis, oedema, arthralgias, irritability, fatigue, and abdominal pain.[9] Shock, ARDS, disseminated intravascular coagulation, and renal failure are the serious and often fatal terminal events.

In conclusion, most often, pneumatocele occur as a sequel to acute pneumonia caused by *S. aureus* but are also associated with *S. pneumonia, Haemophilus influenza, Klebsiella pneumoniae, S. marcescens, Escherichia coli,* Group A streptococci, *Mycobacterium tuberculosis, Pseudomonas aeruginosa* and adenovirus.[12] Pneumatoceles have an incidence of about 60 percent.[11] A TSS as a differential diagnosis in patients presenting with dyspnoea, tachypnoea, fever, rash, hypotension and a toxic clinical picture along with a radiograph showing pneumatoceles made for the primary source of infection and organism involved as aggressive management may lead to complete recovery.

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