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Case Report

Staphylococcal Toxic Shock Syndrome in a Nine Years Old Boy

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Keywords

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Abstract

Toxic Shock Syndrome (TSS) is an acute, toxin-mediated illness which results in shock and multi-organ failure early in its clinical course. Causes include toxin-producing strains of *Staphylococcus aureus* and Invasive Group A Streptococcus (e.g. *Streptococcus pyogenes*). The diagnosis of Toxic Shock Syndrome in children is mainly clinical. The treatment must not be delayed in suspected cases of toxic shock syndrome till laboratory confirmation. We experienced a 9 years old boy presented with fever, rash with hypotension and diagnosed as toxic shock syndrome.

ABBREVIATIONS

CDC: Centers for Disease Control and Prevention; **CRP:** C-Reactive Protein; **ESR**: Erythrocyte Sedimentation Rate; **LFT:** Liver Function Tests; **TSS**: Toxic Shock Syndrome.

INTRODUCTION

Staphylococcal Toxic shock syndrome is a multi systemic illness with abrupt onset. Symptoms include fever, chills, myalgia, headache, malaise, sore throat, muscle tenderness, fatigue, vomiting, watery diarrhea, and abdominal discomfort [1]. It is not rare in children, except that it has not commonly been reported. Treatment in suspected cases of toxic shock syndrome should be immediate when suspected because Staphylococcal TSS is rapidly progressive with an estimated mortality between 20- 50% [2,3].

CASE PRESENTATION

We reported a case of TSS due to an MRSA strain. He was a 9 years old boy, admitted to the pediatric ward of Adan Hospital, Kuwait, with 2 days history of high grade fever, lethargy and persistent vomiting. He had a post traumatic left ankle sprain managed with splint . On physical examination, the patient was confused, tachypneic, pale with sweating. His temperature was 39.8°C, Heart rate 130/minute and blood pressure 80/50 mm Hg. He had bilateral conjunctivitis. There was a diffuse hyperemic rash all over his body with a big bulla 4x6 cm over his left heel [Figure 1]. Other systemic examination was unremarkable with no signs of meningeal irritation. Laboratory results included the following: Total leucocytic count 40.6 x 10^9/L with 94% polynuclear neutrophils, Hb 10.9 gm/dl, platelets 322 x 10^9/L, CRP 220 mg/L, ESR 39 mm/hr. His creatinine 75 umol/L, BUN 9.9 mmol/L, Sodium 131 mmol/L, bicarbonate 13 mEq/L, LFT was normal, creatine kinase 242 U/L. Urine routine showed WBCs 8-12/hpf & RBCs 10-15/hpf. Cultures of blood, stool, and urine samples were negative for microbial agents. Puncture of the bulla released 12 ml pus; culture of the pus sample yielded an MRSA strain.

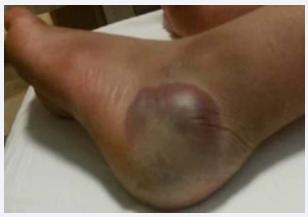

Figure 1 Left heel bulla.

The treatment included aggressive intravenous fluid resuscitation and administration of antimicrobial therapy with Cefotaxime and Vancomycin. The treatment outcome was favorable with resolved impaired renal function and the biological abnormalities. On the seventh day, the patient started to have extensive peeling on both patient's hands. Our patient met the criteria of TSS: He had fever, disturbed level of consciousness, rash, desquamation, hypotension, vomiting, mucus membrane involvement in the form of conjunctivitis and impaired renal function [Figure 2,3]. The diagnosis of staphylococcal TSS was confirmed by bacteriologic results.

DISCUSSION

Toxic shock syndrome (TSS) is an acute severe superantigen-mediated condition that is caused by toxin-producing strains of Staphylococcus aureus and Streptococcus pyogenes. The

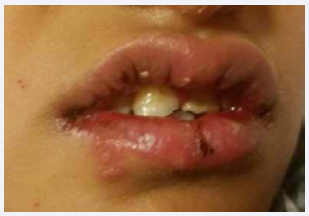

Figure 2 Mucus membrane involvement.

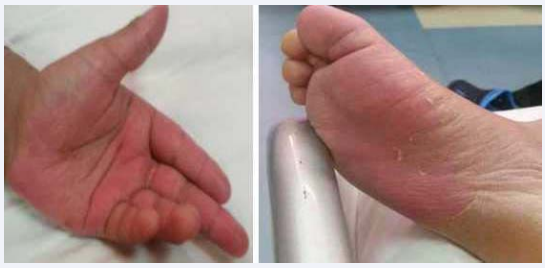

Figure 3 Skin desquamation after 7 days.

toxins act as superantigens, resulting in massive T cell activation and an uncontrolled release of proinflammatory mediators. The result of these on the vascular endothelium leads to capillary leak and 'shock syndrome', which manifests clinically as fever, rash, hypotension, and organ dysfunction [4]. No children around 4 months of age have antibodies to TSS toxin-1, yet by the time children are 13 years of, 80% have antibodies.

TSS was first described by Todd et al in 7 children presented with high grade fever, erythroderma and shock in 1978 [5]. The incidence of Staphylococcal TSS in children under the age of 15 years is less than 0.05 cases per 100,000 population as reported by the CDC in 1999 [5]. It was found that Infectious complications of casting and splinting range from mild, local skin infections, such as cellulitis or abscess, to life-threatening conditions such as necrotizing fasciitis, gangrene, or TSS [6]. The diagnosis of TSS is based on the clinical presentation.

Clinical criteria for staphylococcal toxic shock syndrome (issued by the Centers for Disease Control and Prevention)

Fever
T >38.9°C (102.0°F)
Hypotension
Systolic blood pressure ≤90 mmHg for adults or less than fifth percentile by age for children <16 years of age; orthostatic drop in diastolic blood pressure ≥15 mmHg
Orthostatic syncope or dizziness
Rash
Diffuse macular erythroderma
Desquamation
1 to 2 weeks after onset of illness, particularly involving palms and soles
Multisystem involvement (3 or more of the following organ systems)
Gastrointestinal: Vomiting or diarrhea at onset of illness
Muscular: Severe myalgia or creatine phosphokinase elevation >2 times the normal upper limit
Mucous membranes: Vaginal, oropharyngeal, or conjunctival hyperemia
Renal: Blood urea nitrogen or serum creatinine >2 times the normal upper limit, or pyuria (>5 white blood count/hpf)
Hepatic: Bilirubin or transaminases >2 times the normal upper limit
Hematologic: Platelets <100,000/microL
Central nervous system: Disorientation or alterations in consciousness without focal neurologic signs in the absence of fever and hypotension
Negative results on the following tests, if obtained
Blood, throat, or cerebrospinal fluid cultures for another pathogen (blood cultures may be positive for *Staphylococcus aureus*)
Serologic tests for Rocky Mountain spotted fever, leptospirosis, or measles

*Criteria for a confirmed case include: a patient with fever >38.9°C, hypotension, diffuse erythroderm and skin desquamation (unless the patient dies before desquamation can occur), with involvement of at least three organ systems. A probable case is a patient who is missing one of the characteristics of the confirmed case definition.

In a review of staphylococcal TSS, the frequencies of laboratory results were as follows: Elevated serum creatinine levels - 69%, Positive blood cultures - 60% of patients, Thrombocytopenia - 59%, Hypocalcemia - 58%, Azotemia - 57%, Hyperbilirubinemia - 54%, Elevated levels of hepatic enzymes - 50%, Leukocytosis - 48%, Abnormal urinary sediment - 46%, Elevated creatine kinase levels - 41%, Immature leukocytes - 36%. Hyponatraemia is another useful indicator [7].

The first line of treatment for TSS is supportive while hypotension is present. Extensive fluid replacement aims to maintain perfusion because of intractable hypotension and diffuse capillary leak. Although the blood pressure may be improved with fluids alone, vasopressor drugs (eg, dopamine and/or norepinephrine) may also be required. Any identified infectious focus must be drained. Antistaphylococcal antibiotic therapy is very beneficial in eradicating organisms and also to prevent recurrence [8].

Intravenous immune globulin (IVIG) therapy in staphylococcal TSS is a logical therapy if the individual is susceptible to TSS due to diminished antibody production to toxin [8]. The role of steroids in TSS is controversial with limited evidence that they may reduce the toxicity of strains of staphylococci associated with TSS [9].

The majority of patients with staphylococcal TSS recover uneventfully [1]. Death may occurs within the first few days due to refractory cardiac arrhythmias, cardiomyopathy, irreversible respiratory failure, and, rarely, bleeding due to coagulation defects [10,11].

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