

Case Reports

A case of toxic shock syndrome in an eight year old girl

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Introduction

Toxic shock syndrome (TSS) is an acute febrile illness with mucocutaneous manifestations and multi-system involvement, often associated with focal staphylococcal infection¹. Many cases occur in menstruating women who are 15-25 years of age and use tampons or other vaginal devices in the presence of vaginal colonisation or infection with toxin-producing strains of *staphylococcus aureus*². TSS, however, also occurs in children, non-menstruating women and men associated with wound infection, nasal packing, sinusitis, tracheitis, pneumonia, empyema, abscesses, burns, osteomyelitis and primary bacteraemia³. Several cases have been reported in children^{1,4}.

Staphylococcus aureus produces several super-antigenic exotoxins such as toxic shock syndrome toxin-1 (TSST-1) and staphylococcal enterotoxins⁵. Overactivation of T cells by these exotoxins and resultant overproduction of cytokines are the primary causes of TSS⁶.

Group A streptococcus can cause a similar TSS-like illness termed streptococcal TSS⁷. Kawasaki disease closely resembles TSS clinically. However, diffuse myalgia, vomiting, abdominal pain, diarrhoea, azotaemia, hypotension and shock are rare in Kawasaki disease⁸.

The diagnostic criteria of staphylococcal toxic shock syndrome are shown in Table 1. All 3 major criteria together with any 3 minor criteria establish the diagnosis.

There is no specific laboratory test. Selective tests reveal involvement of multiple organ systems including hepatic, renal, muscular, gastrointestinal, cardiopulmonary and central nervous systems⁸.

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Table 1

Diagnostic criteria of staphylococcal toxic shock syndrome

Major criteria (All required)

Acute fever >38.8°C
Hypotension (orthostatic or shock)
Rash (erythroderma with late desquamation)

Minor criteria (Any 3)

Mucous membrane inflammation
Vomiting, diarrhoea
Liver abnormalities
Renal abnormalities
Muscle abnormalities
CNS abnormalities
Low platelets

Bacterial cultures of associated focus (e.g. vagina, abscess) before administration of antibiotics usually yield *S. aureus*, although this is not a required element of the definition⁸.

Case report

An 8 year old girl was admitted to Lady Ridgeway Hospital in April 2000 with a history of high fever with chills and rigors and headache of 5 days duration and a watery diarrhoea of one day duration following an abscess in left buttock.

On admission, she was febrile (38.9°C), toxic and restless but conscious. Her lips were red and cracked. There was a diffuse erythematous macular rash on anterior aspect of shoulder. An abscess was found in left buttock, 5 cm infero-lateral to anus. Her pulse rate was 150/min and blood pressure 70/40 mm Hg. Her respiratory rate was 24/min and there were no added sounds. The abdomen was soft; there was a tender hepatomegaly.

Her white cell count (WBC) was $17 \times 10^9/L$ with a neutrophil leucocytosis. Her haemoglobin (Hb) was 10.8 g/dl. The platelet count was $205 \times 10^9/L$. Peripheral blood picture showed normochromic normocytic red cells with predominant neutrophils showing toxic granulation. The blood urea was 9.3mmol/L, serum sodium 123mmol/L, ESR 60 mm, random blood sugar 3mmol/L, SGPT 139 IU/L, SCOT 124 IU/L. The prothrombin time was normal.

She was resuscitated with normal saline, given initially as an IV bolus of 20 ml/kg over 15 minutes and then at the rate of 5 ml/kg/hr. After 2 hours her pulse rate was 100/min and BP 100/70 mm Hg. She was treated with IV penicillin, cloxacillin and hydrocortisone. Her vital parameters were checked hourly and a fluid balance chart maintained. She was haemodynamically stable for next 48 hours. However, fever persisted and she continued to appear toxic. Incision and drainage of abscess was done and pus was sent for culture and ABST on 6th day of illness.

2 days later her condition deteriorated. The pulse rate was 160/min and BP 90/70 mm Hg. A 2D ECHO revealed myocarditis with left ventricular dysfunction and an ejection fraction (EF) of 34%. She was treated with captopril 6.25 mg bd for 8 weeks. Cardiac function gradually improved over next 3 days. A 2D ECHO done after 3 days showed remarkable improvement of cardiac function with an EF of 61%. However, high spiking fever persisted and was intermittent. Desquamation of skin occurred on 11th day of illness, initially from trunk and later from hands and feet. Blood transfusion was given to counter anaemia on day 11.

Subsequent investigations confirmed improvement in her condition. WBC was $12.2 \times 10^9/L$, Hb 12.8 g/dl, platelet count $240 \times 10^9/L$, SGPT 22 IU/L and SGOT 26 IU/L. Blood culture was sterile but pus culture revealed a mixed growth of *S. aureus* and coliforms. As both organisms were sensitive to co-amoxyclav, this was added to the regime.

On the 13th day of illness fever settled. Urine output gradually declined from 12th day of illness with rising blood urea. On 14th day, her blood urea was 64.9 mmol/L with a serum sodium of 133 mmol/L and a serum potassium of 2.1 mmol/L. Peritoneal dialysis was started on 15th day and continued till 19th day of illness. During initial phase of dialysis, 3 mmol of potassium chloride was added to each litre of Perisolution till serum potassium reached 3mmol/L. Her serum creatinine was 4.45 mg/dl. Dialysis was discontinued when the urine output was 300 ml. At

this stage an ultrasound scan (USS) of abdomen showed enlarged kidneys with increased cortical echogenicity and loss of corticomedullary demarcation with a radiological diagnosis of acute cortical necrosis.

After one month, renal function returned to normal. Her blood urea was 6.3mmol/L, serum creatinine 1.1 mg/dl and creatinine clearance 104 ml/min. Subsequent USS of abdomen showed normal sized kidney with loss of corticomedullary demarcation. Third 2D ECHO, done after one month, showed an EF of 66%.

Antibiotics were given for 14 days. Treatment was supplemented with vitamins and high calorie diet. She was discharged after 2 months without any residual effects and followed up in the clinic and remains well.

Discussion

All 3 major criteria were present in this 8 year old girl viz. high fever, hypotension and rash. In addition, several minor criteria were present viz. mucous membrane inflammation, diarrhoea, liver abnormalities and renal abnormalities. She also had myocardial dysfunction which is not listed among the minor criteria. Finally, *S. aureus* was cultured from the pus. Thus the diagnosis of TSS is established. This is the first case of TSS reported in children in Sri Lanka.

As TSS is a medical emergency, a high index of suspicion is essential for early recognition of the disease. Parenteral administration of anti-staphylococcal antibiotics together with drainage of focus of infection are the mainstay of treatment⁸. Fluid replacement should be aggressive to prevent or treat hypotension, renal failure and cardiovascular collapse. Inotropic agents may be needed to treat shock⁸. There is recent evidence that steroids diminish toxicity of strains of staphylococcus associated with TSS resulting in more rapid clinical improvement⁹.

Whenever a child presents with an acute febrile illness accompanied by hypotension, the possibility of TSS should be considered and investigations carried out to detect multiorgan dysfunction.

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