Nonmenstrual toxic shock syndrome – a case report

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Summary

We present a young lady who satisfied the criteria for the diagnosis of toxic-shock syndrome (TSS). The differential diagnoses of TSS in the local setting are outlined. The pertinent clinical features of TSS and its increasing association with nonresponstruating females are highlighted.

Key words: Toxic-shock syndrome, nonmenstruating women, toxic-shock syndrome Toxin I.

Introduction

Toxic-shock syndrome (TSS) is a distinct multisystemic clinical entity of acute onset. Its pathogenesis is not fully understood. Early workers described TSS mainly in menstruating women.¹It is now being reported with increasing frequency in nonmenstruating females² as illustrated by our patient described below.

Case report

A previously healthy, 21-year old Malay lady presented with acute onset of fever, vomitting, diarrhoea and rash for two days.

Her complaints started two nights prior to admission when she experienced abrupt onset of generalised itching and fever with chills and rigors. The following morning, she noticed a generalised erythematous rash all over her body including the face, trunk and limbs. She was not on any medications that could account for the rash. Her general condition deteriorated over the next two days with more than twenty episodes of vomitting and diarrhoea. Her stools were watery with no mucous or blood.

Her menses had been irregular with each cycle at 2 to 3 months interval. Her last menstrual period was two months prior to admission. She was not menstruating at the time of her present illness.

On examination, she was acutely ill and disorientated. Her temperature was 39°C. She was hypotensive with a blood pressure of 80/60 mm of Hg. and had a small volume pulse at the rate of 120/ minute. The respiratory rate was 28/minute.

Her skin exhibited a marked erythematous mascular rash that blanched with pressure and had a sunburn appearance. The rash involved the face, trunk and limbs including the palms and soles. There were areas of desquamation over the forehead and perioral region. The conjuctivae were markedly suffused.

The physical examination was otherwise unremarkable. In particular, she was not pale or jaundiced. There was no organomegaly and had no neurological deficits.

Routine blood counts and biochemical evaluations done on admission are shown in Table I.

Hb. (g/dl)		12.9		
WBC (x10 ⁹ /1)		8.5		
Diff. count:	P :	93		
	L :	5		
	E :	2		
Platelets (X $10^{9}/1$)		150		
Blood urea		27.9	micromoles/L	(2.8 - 7.8 mmol/l)
Serum sodium		134	"	(135 – 152 ")
Sr. potassium		4.4	"	(3.6 – 5.4 ")
Sr. creatinine		282	"	(88 - 176")
Sr. creatine ph	osphokinase		4000	units/L $(24 - 170 \text{ U/L})$
Sr. Bilirubin		48	micromol/1	(3 - 18.8 micromol/l)
Sr. Alanine transaminase		280	R.F. unit/l	(4 - 30 R.F. unit/l)

Table INormal values in brackets where applicable

P: Polymorphs; L: Lymphocytes; E: Eosinophils

The septic workout including blood, stool, urine and throat swab cultures were sterile. The serological tests for typhoid, typhus and leptospirosis too were negative. The peripheral blood films were repeatedly negative for malarial parasites. Her chest x-ray was normal. The antinuclear factor and rheumatoid factors were negative.

She responded well to intravenous rehydration and a course of Cloxacillin. Her skin showed marked desquamation during the convalescent phase but no staining of the skin was noted. She made full recovery a week after admission. Her renal profile, liver function tests and creatine phosphokinase levels too returned to normal levels at the same time. She remains well six months later.

Discussion

Leptospirosis was the provisional diagnosis entertained in our patient at the time of admission. The spirochetal illness would have adequately accounted for her fever, suffused eyes, elevated creatine phosphokinase levels³ as well as the impaired renal profile and liver function tests. However, the patient had no calf muscle tenderness and the serological tests for leptospirosis too were negative. Further, the rash in leptospirosis is usually maculopapular, petechial and purpuric - i.e. unlike what our patient had.⁴

The other common infectious illness in the tropics including measles, scrub typhus and malaria too were excluded by the clinical course of the illness as well as by specific laboratory testing.

Thus the diagnosis of toxic shock syndrome (TSS) was made in our patient by excluding other common illnesses. Her subsiquent course in the ward and the response to treatment given too, pointed towards the same disorder.

The term, TSS, was first used in 1978 by Todd and co workers to describe an infectious syndrome in a group of seven children¹. It is a distinct clinical entity characterised by hypotension, fever, hyperemia of mucous membrane, erythroderma with subsequent desquamation and multisystemic involvement⁵.

In 1980, TSS was frequently observed in young women, with the onset mainly during menstruation. The hyperabsorable tampons which were popular at that time was thought to be responsible for the increasing number of patients with TSS reported between 1980 and 1982: 96% were women and 92% of these patients were menstruating at the time of onset of the illness.^{1,4}

Subsequently TSS began to be reported in nonmenstruating women. Currently it is estimated that about 50% of TSS occur in nonmenstruating females.²

There are no pathognomonic features of TSS. The Centre for Disease Control (CDC), Atlanta, has devised a set of diagnostic criteria one can use to diagnose TSS (Table II)⁶

Fever	:	Temperature – 38.9°C
Rash	:	Diffuse mascular erythroderma
Desquamation	:	One to two weeks after onset of illness, particularly of the palms and soles.
Hypotension	:	Systolic blood pressure < 90 mm hg for adults or below fifth percentile by age for children younger than 16 years; alternatively, orthostatic drop in diastolic blood pressure > 15 mm hg, or orthostatic syncope.
Three or more o	f the	Following Systems Involved:

Table II						
CDC	Case	Definition	for	Toxic	Shock	Syndrome

Thee or more of the Following Systems inv

G.I	:	vomitting or diarrhoea at onset of illness
Muscular	:	severe myalgia or creatine kinase level greater than twice upper limit of normal
Mucous membrane:		vaginal, oropharyngeal, or conjuctival hyperemia
Renal	:	BUN or creatinine greater than twice upper limit or normal or > 5 WBCs per
		high-power field in urinary sediment in absence of infection.
Hepatic	:	total bilirubin, AST, or ALT greater than twice upper limit of normal.
Hematologic	:	platelets < 100,000
Central nervous system	:	disorientation or alterations in consciousness without focal neurologic signs when fever and hypotensions are absent.

Negative Result on the Following Tests:

Blood, throat, or cerebrospinal fluid culture (blood may be positive for staphylococcus aureus)

Serologic tests for Rocky Mountain spotted fever, leptospirosis, or rubeola

Our patient satisfied the first four criteria as well as all the others except for thromocytopaenia. In our setting, serology for Rocky Mountain spotted fever is not applicable; a related rickettsial disease of scrub typhus was excluded.

TSS is generally thought to be due to a toxin (toxic shock syndrome toxin I or TSST-1) elaborated by phage group I Staphylococcus aureus. The source of the staphylococcal infection in our patient still eludes us. However, this in not surprising as it has been shown that blood cultures are only exceptionally positive in this condition.^{1,4,6} Thus an assay for TSST-I would have been helpful. This could not be done in our patient as the test was not available. Incidentally, a TSS-like syndrome has also been described in association with the erythrogenic toxin A of group A beta-haemolytic streptococci.⁷

Treatment of TSS is generally supportive.^{1,4,6} Intensive care monitoring and aggressive crystalloid fluid therapy are the mainstay of treatment. Anti-staphylococcal antibiotics like Cloxacillin may help to reduce the frequency of recurrence. Some workers feel corticosteroids may be indicated in the severe form of TSS⁴

TSS should be included in the differential diagnosis of a febrile and hypotensive patient with the typical rash in whom the usual septic workout was not helpful.

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