

Brief Reports

Postoperative toxic shock syndrome in a man

D. PORTNOY, MD
E.J. HINCHEY, MD
O.W. MARCUS-JONES, FIMLS, ART
G.K. RICHARDS, MD, MRC PATH

Toxic shock syndrome (TSS) was first described by Todd and colleagues¹ in seven children aged between 8 and 17 years. It is characterized by fever, myalgia, hypotension or shock, nausea, vomiting and diarrhea, a polymorphous rash with subsequent desquamation, and laboratory evidence of the involvement of multiple organ systems.² TSS has most frequently occurred in young women during menstruation, especially in those who used tampons. We report a case of TSS developing in a man following surgery. *Staphylococcus aureus*, phage group II, type 55, was isolated postoperatively from an infected surgical wound. The shock-like state disappeared within 24 hours following fluid replacement and early intravenous administration of cloxacillin and gentamicin.

Case report

A 31-year-old man was well until 3 months before admission to hospital, when he noticed an enlargement of his left breast. A mobile, nontender mass 2 cm in diameter was palpable beneath the left nipple. There were no other physical abnormalities. A simple subcutaneous mastectomy was performed. Subsequent histologic examination of the tissue showed benign intraductal papillomatosis. The patient's recovery immediately after the operation was uneventful, but 24 hours later the temperature rose to 39.3°C, the blood pressure was 85/60 mm Hg (it had been 110/80 mm Hg at the time of admission) and his pulse rate was 160 beats/min. Both conjunctivae were injected,

but the buccal and pharyngeal mucosa was normal. A diffuse, confluent erythematous rash (Fig. 1) that blanched when the skin was pressed covered his face, chest and arms. The wound site did not appear to be infected. Drug-induced toxic erythema secondary to the preoperative medications or the anesthetic was suspected, and the patient was treated with diphenhydramine hydrochloride; however, his condition did not improve. Later that morning he had two episodes of diarrhea, vomited three times and complained of generalized myalgia. Within 3 hours of the onset of these symptoms his blood pressure fell further, to 60/40 mm Hg, and he became abusive and combative. TSS was suspected. Although the wound site appeared to be uninfected, needle aspiration yielded 0.5 ml of sanguineous fluid containing many neutrophils and both intra- and extracellular gram-positive cocci (Fig. 2).

The wound was incised but there was no further drainage. Culture yielded penicillin-resistant *S. aureus*, phage group II, type 55 (the phage-typing was done by the staphylococcal reference laboratory, ministère des Affaires so-

ciales, Ste.-Anne-de-Bellevue, PQ). Samples of skin from the patient's back, abdomen, chest and nose were cultured, and all yielded the same phage strain. Blood and urine cultures gave negative results, and stool specimens yielded no recognized pathogens. Swabs were taken from the nostrils, arms and hands of all the members of the surgical team and cultured for *S. aureus*. All but one — a nasal swab from which *S. aureus*, phage type 96 was grown — yielded negative culture results.

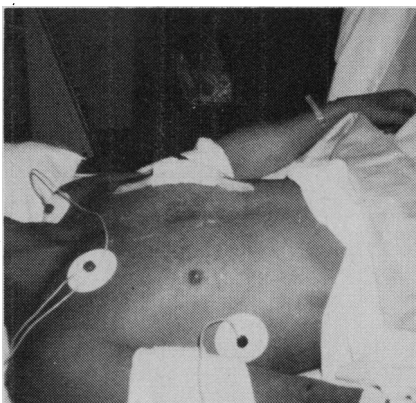


FIG. 1—Erythema following onset of toxic shock syndrome.

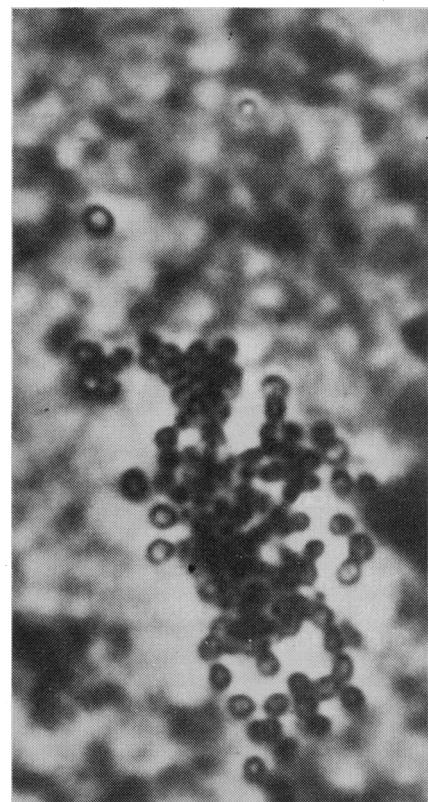


FIG. 2—Pus from wound contains characteristic staphylococcal clusters and neutrophil debris (oil immersion, phase contrast, dichroic green filter $\times 1000$).

From the departments of microbiology and surgery, Montreal General Hospital

Reprint requests to: Dr. D. Portnoy, Department of microbiology and infectious disease, Montreal General Hospital, 1650 Cedar Ave., Montreal, PQ H3G 1A4

Ativan*

(lorazepam)

An Uncomplicated Benzodiazepine

COMPOSITION: Ativan 1 mg.—Each white, oblong, scored tablet contains: Lorazepam 1 mg. (DIN 348325) Ativan 2 mg.—Each white, ovoid, scored tablet contains: Lorazepam 2 mg. (DIN 348333)

INDICATIONS: Ativan is useful for the short-term relief of manifestations of excessive anxiety in patients with anxiety neurosis.

CONTRAINDICATIONS: Ativan is contraindicated in patients with known hypersensitivity to benzodiazepines and in patients with myasthenia gravis or acute narrow angle glaucoma.

DOSAGE: The dosage of ATIVAN must be individualized and carefully titrated in order to avoid excessive sedation or mental and motor impairment. As with other anxiolytic sedatives, it is not recommended to prescribe or administer ATIVAN for periods in excess of six weeks, without follow-up and establishing the need for more prolonged administration in individual patients.

Usual Adult Dosage: The recommended initial adult daily dosage is 2 mg in divided doses of 0.5 mg, 0.5 mg and 1.0 mg, or of 1 mg and 1 mg. The daily dosage should be carefully increased or decreased by 0.5 mg depending upon tolerance and response. The usual daily dosage is 2 to 3 mg. However, the optimal dosage may range from 1 to 4 mg daily in individual patients. Usually, a daily dosage of 6 mg should not be exceeded.

Elderly and Debilitated Patients: The initial daily dose in these patients should not exceed 0.5 mg and should be very carefully and gradually adjusted, depending upon tolerance and response.

PRECAUTIONS: Use in the Elderly: Elderly and debilitated patients, or those with organic brain syndrome, have been found to be prone to CNS depression after even low doses of benzodiazepines. Therefore, medication should be initiated in these patients with very low initial doses, and increments should be made gradually, depending on the response of the patient, in order to avoid oversedation or neurological impairment.

Dependence Liability: Ativan should not be administered to individuals prone to drug abuse.

Caution should be observed in patients who are considered to have potential for psychological dependence. It is suggested that the drug should be withdrawn gradually if it has been used in high dosage.

Use in Mental and Emotional Disorders: Ativan is not recommended for the treatment of psychotic or depressed patients. Since excitement and other paradoxical reactions can result from the use of these drugs in psychotic patients, they should not be used in ambulatory patients suspected of having psychotic tendencies.

ADVERSE EFFECTS: The side effect most frequently reported was drowsiness. Other reported side effects were dizziness, weakness, fatigue and lethargy, disorientation, ataxia, anterograde amnesia, nausea, change in appetite, change in weight, depression, blurred vision and diplopia, psychomotor agitation, sleep disturbance, vomiting, sexual disturbance, headache, skin rashes, gastrointestinal, ear, nose and throat, musculoskeletal and respiratory disturbances.

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The day of the patient's collapse the serum levels were as follows: urea nitrogen 28.0 mg/dl (urea 10.9 mmol/l) (normal 9.0 to 21.0 mg/dl [3.2 to 7.5 mmol/l]), bilirubin 2.5 mg/dl (42.7 μ mol/l) (normal 0 to 2.1 mg/dl [3.42 to 17.10 μ mol/l]) and amylase 350 units/dl (normal 60 to 185 units/dl). The leukocyte count was $11.5 \times 10^9/l$ (84% juvenile forms, 15% mature neutrophils and 1% monocytes), the hemoglobin level 17.2 g/dl, the hematocrit 45.2%, the erythrocyte sedimentation rate 6 mm/h and the platelet count $63.0 \times 10^9/l$. All these results had been recorded as normal at the time of admission. A throat specimen cultured for group A streptococcus gave negative results, as did antistreptolysin O and Streptozyme tests (Denver Laboratories, Toronto) performed on serum obtained in both the acute and convalescent stages of the illness.

Four hours after the onset of the fever and the associated symptoms the patient began receiving cloxacillin, 2 g intravenously every 4 hours, and gentamicin, 120 mg intravenously every 8 hours. Large volumes of intravenous fluids were required to maintain an adequate blood pressure, but he remained oliguric for several hours.

The patient's response to treatment was dramatic. Within 24 hours he was afebrile and the diarrhea had ceased. At 48 hours the rash had faded and a fine desquamation was noted, mainly on his back and chest. This disappeared rapidly, and no other physical abnormality was demonstrated. He was discharged from hospital on the sixth postoperative day and completed a 2-week course of oral cloxacillin therapy at home. All the laboratory values had returned to normal before discharge, although thrombocytopenia was still present (platelet count $64.0 \times 10^9/l$). However, the platelet count had returned to normal at the time of follow-up, 3 weeks after discharge.

Discussion

In 1978 Todd and colleagues¹ associated TSS with a new exfoliative exotoxin produced by certain strains of phage group I staphylococci. This new exotoxin was different from exfoliatin, another staphylococcal toxin, associated with the scalded skin syndrome.³ It has since been demonstrated that the exotoxin described by Todd and colleagues is not phage-group-specific; indeed, we demonstrated both toxins in the phage group II *S. aureus* we isolated from our patient. The new toxin has been shown to be produced by only 29 (71%) of 41 strains of *S. aureus* causing TSS⁴ and is produced just as often in

strains not causing this condition.⁵ This toxin is therefore unlikely to cause TSS, although it might be a contributing factor. The toxin associated with TSS has recently been identified.^{4,6,7}

More than 900 cases of TSS have been reported to the Centers for Disease Control in Atlanta, Georgia, most of which occurred in women during menstruation. Only 2.5% of the reported cases occurred in men.⁵ A significant association has been noted between the use of tampons and TSS. *S. aureus* was isolated from most of these patients, and the isolates were resistant to penicillin. The *S. aureus* isolated from our patient was resistant to both penicillin and ampicillin but sensitive to other routinely tested antibiotics. Treatment with penicillinase-resistant antibiotics has not been shown to modify the course of the disease, but it does appear to prevent a recurrence.⁸ TSS developed in our patient while he was in hospital; therefore, treatment with antibiotics and fluid replacement was instituted early in the course of his illness, which resulted in complete resolution of the fever and toxic state within 24 hours. It has been postulated that in patients with TSS, toxin produced by the staphylococci enters the bloodstream from the site of infection and is distributed throughout the body. The organism itself is apparently not highly invasive, because only a few patients have had positive results of blood cultures.⁵ Whether the early intravenous administration of antibiotics and fluids was responsible for our patient's dramatic improvement remains speculative; however, early antibiotic therapy may have rapidly eradicated the staphylococci before they could produce a significant amount of toxin.

References

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