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 colleagues[1] 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 sociales, 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.

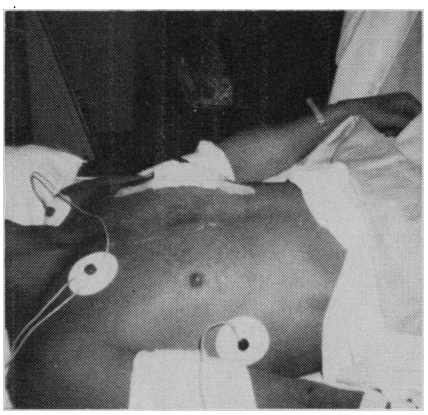

FIG. 1—Erythema following onset of toxic shock syndrome.

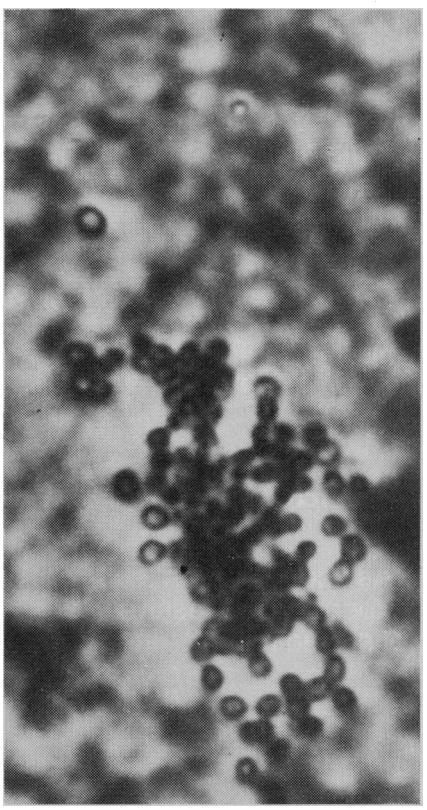

FIG. 2—Pus from wound contains characteristic staphylococcal clusters and neutrophil debris (oil immersion, phase contrast, dichroic green filter ×1000).

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

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 μmol/l) (normal 0 to 2.1 mg/dl [3.42 to 17.10 μmol/l]) and amylase 350 units/dl (normal 60 to 185 units/dl). The leukocyte count was 11.5 × 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 × 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 × 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 colleagues[1] 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 TSS[4] 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

1. TODD J, FISHAUT M, KAPRAL F, WELCH T: Toxic-shock syndrome associated with phage-group-I staphylococci. *Lancet* 1978; 2: 1116–1118
2. Follow up on toxic shock syndrome. *Morb Mortal Wkly Rep* 1980; 29: 441–445
3. KAPRAL FA, MILLER MM: Product of Staphylococcus aureus responsible for the scalded-skin syndrome. *Infect Immun* 1971; 4: 541–545
4. SCHLIEVERT PM, SHANDS KN, DAN BB, SCHMID GP, NISHIMURA RD: Identification and characterization of an exotoxin from *Staphylococcus aureus* associated with toxic-shock syndrome. *J Infect Dis* 1981; 143: 509–516
5. SHANDS KN, DAN BB, SCHMID GP: Toxic shock syndrome: the emerging picture. *Ann Intern Med* 1981; 94: 264–266
6. BERGDOLL MS, CRASS BA, REISER RF, ROBBINS RN, DAVIS JP: A new staphylococcal enterotoxin, enterotoxin F, associated with toxic-shock syndrome *Staphylococcus aureus* isolates. *Lancet* 1981; 1: 1017–1021
7. BENNETT JV: Toxins and toxic-shock syndrome. *J Infect Dis* 1981; 143: 631–632
8. Follow-up on toxic shock syndrome. *Morb Mortal Wkly Rep* 1980; 29: 297–299