CLINICAL CONTRIBUTION Rhinology, 27, 125-128, 1989

Toxic shock syndrome after nasal surgery: Is prevention possible?

A case report and review of the literature

N. de Vries and S. van der Baan

Dept. of O.R.L., Free University Amsterdam, The Netherlands

SUMMARY

Toxic shock syndrome (TSS), is an acute illness with four major criteria: involvement of multiple organ systems, fever > 38.9° C, hypotension or shock and rash with subsequent desquamation. TSS was first reported by Todd et al. in 1978, and is a rare complication of staphylococcal infection. Although it at first was thought to be a childhood disease and an illness of menstruating women using intravaginal tampons, it has now been described as a complication of minor surgery, burns and minimal skin infections (Reingold et al., 1982; Jacobson et al., 1983). More than 2800 cases have been reported at the Centers for Disease Control (CDC) in Atlanta (Reingold, 1985). Jacobson and Kasworm (1986) estimate the incidence after nasal surgery to be 16.5 per 100.000, which in fact is higher than the incidence in women of menstrual age using intravaginal tampons. TSS usually occurs within 24-48 hours after surgery, often starting with nausea and vomiting. Although the syndrome can be lethal or can have troublesome sequelae, as prolonged weakness fatique and neuropsychological disturbances, complete recovery is often the case.

Case history

A female, 25 years of age, who had her last menstruation two weeks before surgery, underwent routine septoplasty under local anaesthesia. The total surgery time lasted 30 minutes. Postoperatively nasal splints and foamrubber tampons covered with Adaptic® were inserted. Ten hours after surgery the patient vomited, developed high fever, hypotension and a rash. Nasal packs and splints were removed, and the patient was transferred to the Intensive Care Unit. Here, the patient developed an acute respiratory distress syndrome, and a transient diabetes mellitus. She was treated with dopamine, antibiotics, and was intubated for six days. Three weeks postoperatively, she developed an extensive desquamation of the skin. Blood cultures were repeatedly negative, while cultures from the nose, nasal splints and nasal packs showed S. aureus. Production of entero-

toxin B was positive, while antibody titers to enterotoxin B were low positive, but were not raised postoperatively. Production of Toxic Shock Syndrome Toxine (TSSS-1) - also called Enterotoxin F, which most often causes TSS - was negative. Phage-typing showed phage-type 12. The patient eventually recovered completely.

Etiology and bacteriology

TSS is usually associated with the presence of and/or infection by S. aureus. S. aureus appears relatively innocuous because it elaborates low concentrations of haemolysin, lipase and nuclease. However, it produces large amount exotoxins (Schlievert et al., 1982). Because of the absence of local inflammatory response and negative blood cultures, it is suggested that TSS is caused by the production, absorption and action of toxins, rather than infection (Fisher et al., 1982; Davis et al., 1982; Bartlett et al., 1982). Staphylococcal pyrogenic exotoxin C, and staphylococcal enterotoxin F are similar and possibly identical (Tofte and Williams, 1982). Both are produced by phage-types 29 and 52, which are the most common S. aureus bacteriophages isolated from TSS patients (Bergdoll et al., 1981; Bergdoll et al., 1982; Schlievert et al., 1981; Altemier et al., 1982). Although usually caused by enterotoxin F, TSS can be caused - as in our patient - by enterotoxin B.

Risk factors

In discussing the prevention, or diminishing the risk of TSS after nasal surgery, four issues are important: 1. identification of persons at risk, 2. preoperative desinfection, 3. perioperative antibiotic prophylaxis and 4. postoperative splinting and packing.

1. Identification of persons at risk

The nasal rate of S. aureus is 20-80% (Breda et al., 1987). Twenty percent of S. aureus produces enterotoxin F (Jacobson and Kasworm, 1986). Eighty percent of the normal adult population has high antibody titers to enterotoxin F. In contrast, 95% of patients who developed TSS had a low, or no antibody titers to enterotoxin F (Bergdoll et al., 1982). This suggests that it may be possible to serologically identify persons who are vulnerable to the development of TSS (Fisher, 1986). However, because of the low incidence of TSS, routine screening for the above mentioned factors is not cost-effective. Recently, TSS caused by enterotoxin B is also reported.

2. Preoperative desinfection

Preoperative desinfection of the nasal mucosa by iodine is recommended by Wagner and Toback (1986). However, this procedure is to be discouraged because of the possible absorption across mucous membranes of iodine and its potent allergenic properties.

3. Perioperative antibiotic prophylaxis

Perioperative antibiotic prohylaxis would theoretically be of value. Still, TSS has been reported during perioperative systemic antistaphylococcal prophylaxis, and after the use of nasal packs coated with bacitracin ointment (Breda et al., 1987).

4. *Postoperative splinting and packing*

The presence of nasal packs and splints in the nose render an ideal environment for bacterial growth. Breda et al. (1987) report that the usé of Merocel® nasal packing after nasal surgery is less likely to give growth of S. aureus than NuGauze®. Stucker and Ansel (1978) advise against the routine use of nasal tampons because of the discomfort for the patient and the risk of TSS. Jacobson and Kasworm (1986) report that the use of intranasal splints is possibly an ever higher risk factor factor than nasal tampons. Wagner and Toback (1986) report a case of TSS after septoplasty using splints without nasal tampons. In our opinion therefore, the use of splints should be kept as minimal as possible. Nasal packing however, is an important procedure for postoperative support and prevention of bleeding, and should not be abandoned for the low risk of TSS.

CONCLUSION

TSS after nasal surgery occurs in approximately 16.5 per 100.000 persons (Jacobson and Kasworm, 1986). The bacteriological factors involved are nasal carriership of S. aureus, production of enterotoxin F - or enterotoxin B - and the absence of, or low concentration antibodies against enterotoxin F (respectively enterotoxin B) in the patient. Since the first two factors are common, the latter factor may well be the most important in the development of the syndrome. The use of preoperative disinfection of the nasal mucosa with iodine is discouraged because its possible side effects. Although routine prophylactic systemic or local use of antistaphylococcal antibiotics would theoretically be of value, the exact role remains to be established. At this moment no watertight regimen against TSS is present. During treatment, next to the therapy of the shock itself, transfusion of blood products containing immunoglobulins combats the syndrome in patients who have no antibodies against enterotoxin F (Frame and Hackett, 1988).

REFERENCES

1. Altemier WA, Lewis SA, Schlievert PM et al. Staphylococcus aureus associated with toxic shock syndrome. Ann Intern Med 1982; 96:987-990.
2. Bartlett PC, Reingold AL, Graham DR et al. Toxic shock syndrome associated with surgical wound infections. J Am Med Ass 1982; 247:1448-1450.
3. Bergdoll MS, Crass BA, Reiser RF et al. A new staphylococcus enterotoxin, enterotoxin F, associated with toxic shock syndrome, Staphylococcus aureus isolates. Lancet 1981; 1:1017.
4. Bergdoll MS, Crass BA, Reiser RF et al. A enterotoxin-like protein in Staphylococcus aureus strains from patients with toxic shock syndrome. Ann Intern Med 1982; 96: 969-972.

5. Breda SD, Jacobs JB, Lebowitz AS et al. Toxic shock syndrome in nasal surgery: a physiochemical and microbiologic evaluation of Merocel® and NuGauze® nasal packing. Laryngoscope 1987; 97:1388-1390.
6. Davis JP, Vergeront JM, Chesney PJ. Possible host-defense mechanisms in toxic shock syndrome. Ann Intern Med 1982; 96:986-991.
7. Fisher GJ, Horowitz BZ, Nolan SM. The clinical spectrum of toxic shock syndrome. West J Med 1982; 135:175-179.
8. Fisher CJ Jr. Toxic shock syndrome. In: Schoemaker WC, Thompson WL, Holbrook PR, Eds. Textbook of critical care. Philadelphia: Saunders Company, 1984; 558-562.
9. Frame JD, Hackett M. Toxic shock syndrome after a minor surgical procedure. Lancet 1988; 1:1330.
10. Jacobson JA, Burke JP, Benowitz BA et al. Varicella zoster and staphylococcal toxic shock syndrome in a young man. J Am Med Ass 1983; 249:922-923.
11. Jacobson JA, Kasworm EM. Toxic shock syndrome after nasal surgery, Case reports and analysis of risk factors. Arch Otolaryngol Head Neck Surg 1986; 122:329-332.
12. Reingold A, Hargrett NT, Shands et al. Nonmenstrual toxic shock syndrome; A review of 130 cases. Ann Intern Med 1982; 96:871-874.
13. Reingold AL: Toxic shock in the United States of America: Epidemiology. Postgrad Med J 1985; 61(Suppl 1):23-24.
14. Schlievert PM, Shands KN, Dan BD et al. Identification and characterization of an exotoxin from Staphylococcus aureus associated with toxic shock syndrome. J Infect Dis 1981; 143:509-511.
15. Schlievert PM, Osterholm MT, Kelly JA et al. Toxin and enzyme characterization of Staphylococcus aureus isolates from patients with and without toxic shock syndrome. Ann Intern Med 1982; 96:937-940.
16. Stucker F, Ansel DG. A case against nasal packing. Laryngoscope 1978; 88:1314-1317.
17. Todd J, Fishaut M, Kapral F et al. Toxic shock syndrome associated with phase-group I- Staphylococci. Lancet 1978; 1116-1118.
18. Tofte RW, Williams DW. Clinical and laboratory manifestations of toxic shock syndrome. Ann Intern Med 1982; 96:843-846.
19. Wagner R, Toback JM. Toxic shock syndrome following septoplasty using plastic septal splints. Laryngoscope 1986; 96:609-610.

N. de Vries, M.D.
Dept. of O.R.L.
Free University Hospital
de Boelelaan 1117
1081 HV Amsterdam
The Netherlands