Toxic Shock Syndrome After Anterior-Posterior Nasal Packing

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Abstract

Toxic shock syndrome (TSS) is a severe, potentially life-threatening multisystem disease. It is rather rare with the incidence estimated to be 16.5/100,000 of nasal surgery performed in the United States. A milder degree of TSS may be more common. Surgeons who perform sino-nasal surgery should be aware of this disease, although certain criteria for definite TSS are absent. Early recognition and prompt intervention is important to minimize the morbidity and mortality associated with this disease. This paper presents the first reported case of a milder degree of TSS after anterior-posterior nasal packing in Thailand. The criteria for diagnosis, the pathogenesis, and the management of TSS were reviewed.

Key word : Toxic Shock Syndrome, Nasal Packing, Epistaxis

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Staphylococcal toxic shock syndrome (TSS) was first described in 1978 by Todd et al (1). This severe, potentially fatal multisystem infectious disease is characterized by fever, rash, skin desquamation, hypotension, and multiorgan failure(2,3). In the head and neck region, TSS has been associated with sino-nasal surgery, pharyngitis, deep neck abscess, and otologic surgery(4,5). The incidence of TSS after nasal surgery is estimated to be 16.5 per 100,000 of nasal surgery performed in the United States(6). In Thailand, the incidence has never been assessed because TSS has never been reported. Although it is a rare syndrome, it has a high mortality rate of approximately 5-10 per cent(7). This paper presents the first reported case of TSS associated with anterior-

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posterior nasal packing in Thailand. The clinical presentations in this case were not typical and should be classified as a milder degree of TSS. Early recognition and prompt management of possible TSS is important to minimize the morbidity and mortality associated with this syndrome.

CASE REPORT

A 56-year-old man with a history of right posterior epistaxis was admitted for anterior-posterior nasal packing. His past history revealed he had had nasopharyngeal carcinoma that was treated with radiation and chemotherapy ten years previously with no recurrence of the tumor. He denied history of hypertension. The anterior-posterior nasal packing with nitrofurazone-impregnated gauze was performed under local anesthesia. The patient was started on oral amoxicillin-clavulanate and 40 per cent oxygen *via* facial mask. The anterior-posterior nasal packing was kept in place for seven days without any complication.

Two days after removal of the nasal packing, bleeding recurred and the second anterior-posterior nasal packing was repeated; and he developed diffuse erythematous maculopapular rash involving his face, chest wall, and upper extremities two days later. All of the vital signs were normal. The dermatologist suggested changing the antibiotic from oral amoxicillin-clavulanate to oral clindamycin, but the rash did not resolve.

Three days later, he developed fever, sore throat, and dyspnea. Physical examination revealed a temperature of 38.9°C, heart rate of 95 beats per minute, blood pressure of 180/100 mm Hg, and respiratory rate of 24 breaths per minute. Diffuse erythematous maculopapular rash was present over his face, chest wall, and upper extremities. The oral and pharyngeal mucosa appeared hyperemic without purulent discharge or a specific focus of infection. Expiratory rhonchi and wheeze were present in both lungs.

Laboratory data revealed white blood cells of 17,800 /mm^3 with a neutrophil of 81 per cent, and platelets of 448,000 /mm^3. Urinalysis, blood urea nitrogen, creatinine, electrolytes, and liver function tests were normal. Electrocardiogram showed an old inferior wall myocardial infarction and chest X-ray was unremarkable. Blood and urine cultures yielded negative results but the nasal discharge culture disclosed *S. aureus*.

The patient was started on bronchodilator *via* nebulizer and the antibiotic was changed from oral clindamycin to intravenous cloxacillin and amikacin as suggested by the internist. His condition had progressively worsened with a respiratory rate of 28-32 breaths per minute, heart rate of 120 beats per minute, blood pressure of 190/100 mm Hg, and oxygen saturation of 85 per cent. Chest X-ray showed pulmonary congestion and borderline cardiomegaly without pulmonary infiltration. His symptoms slightly improved despite endotracheal intubation. The anterior-posterior nasal packing was removed, and his rash and dyspnea disappeared within 48 hours. His vital signs returned to normal and his condition improved.

Five days later, he started to have another episode of epistaxis and the temporary anterior nasal packing with vaseline gauze and absorbable gelatin sponge was placed. Next morning, diffuse erythematous maculopapular rash reappeared on his face and chest wall but his general condition was still good. He was sent to the operating room for transnasal endoscopic ligation of the sphenopalatine artery using the authors' technique[8]. Postoperatively, he had no further bleeding and the rash disappeared within 24 hours after surgery. He was then discharged from the hospital without any complication. He had desquamation on the palmar side of the distal end of his fingers about 30 days after onset.

DISCUSSION

TSS is a severe, potentially life-threatening multisystem disease with a mortality rate of approximately 5-10 per cent[7]. Although it was predominantly described as a disease of menstruating women, TSS is now recognized to occur after a variety of diseases or surgical procedures, including pharyngitis, deep neck abscesses, otologic, and sino-nasal surgeries[4,5]. To fulfill the definition of classic TSS, the patient must exhibit all of the four major criteria, as well as at least three of the minor criteria (Table 1)[2,3]. The onset of TSS after nasal surgery is usually within 24 hours[6], although delayed TSS can develop 25 days after endoscopic sinus surgery[9]. Early symptoms include fever, vomiting, diarrhea, and myalgia, followed by development of hypotension and, in severe cases, shock[2]. An erythematous, "sunburn-like" rash is present during the acute phase of the

Table 1. Toxic-shock syndrome case definition[2].

1. Fever (temperature ≥ 38.9°C (102°F).
2. Rash (diffuse macular erythroderma).
3. Desquamation, 1-2 weeks after onset of illness, particularly of palms and soles.
4. Hypotension* (systolic blood pressure ≤ 90 mm Hg. for adult or < 5 th percentile by age for children < 16 years of age, or orthostatic syncope).
5. Involvement of 3 or more of the following organ systems:
   A. Gastrointestinal (vomiting or diarrhea at onset of illness).
   B. Muscular (severe myalgia or creatinine phosphokinase level ≥ 2 x upper limits of normal for laboratory).
   C. Mucous membrane (vaginal, oropharyngeal, or conjunctival hyperemia).
   D. Renal (BUN or Cr ≥ 2 x upper limits of normal for laboratory or ≥ 5 white blood cells per high-power field – in the absence of a urinary tract infection).
   E. Hepatic (total bilirubin, SGOT, SGPT ≥ 2 x upper limits of normal for laboratory).
   F. Hematologic (platelets ≤ 100,000/mm^3).
   G. Central nervous system (disorientation or alterations in consciousness without focal neurologic signs when fever and hypotension are absent).
6. Negative results on the following tests, if obtained:
   A. Blood*, throat, or cerebrospinal fluid cultures.
   B. Serologic tests for Rocky Mountain spotted fever, leptospirosis, or measles.

* Orthostatic dizziness is now considered sufficient evidence of hypotension and the presence of *S. aureus* in blood cultures does not exclude a case from consideration[3].

illness; followed by desquamation of the skin, particularly of the palms and soles about ten days after onset[2].

In the study of Jacobson et al[10], about 35 per cent of the population were asymptomatic nasal carriers of *S. aureus*. Of these, 25 per cent (8.75% of the population) carried toxic shock syndrome toxin 1 (TSST-1) strain. Persons who carried toxin-producing strain of *S. aureus* were more likely to have high levels of antibody to TSST-1 (98.4%) than those who did not carry these organisms (91.8%). However, patients who carried a toxigenic *S. aureus* and lacked antibody to TSST-1 and had surgery at the colonized site did not always develop TSS. This indicates that additional factors and, perhaps, other toxins are likely to be involved in the pathogenesis of TSS.

The pathogenesis of TSS requires that a number of events must coincide[6,11]. First, an individual must either be colonized or infected with *S. aureus* that produced TSST-1. Next, there must be some alteration in the integrity of the mucous membrane at the site of colonization or infection. Finally, the patient must have susceptibility to infection by lacking antitoxin antibodies or other protective factors. Local factors, including a neutral pH and an aerobic environment, appear play important role in the pathogenesis of TSS as well[12]. The clinical manifestations occur due to the effect of the toxin[13]. It causes vasodilatation and rapid movement of serum proteins and fluid from the intravascular to the extravascular compartment. The multisystem involvement may simply be a reflection of the rapid onset of hypotension and decreased organ perfusion, or there may be a direct effect of a toxin on the parenchymal cells of different organs.

In Thailand, the incidence of asymptomatic nasal carriers of TSST-1 producing *S. aureus* who lack antibody to TSST-1 is not known. Sino-nasal surgery performed at King Chulalongkorn Memorial Hospital were about 500-1,000 operations per year and most of them had either kind of nasal packing, but a classic case of TSS has not been encountered within the last twenty years. We think that the incidence of classic TSS after sino-nasal surgery in Thailand should be less than the incidence estimated by Jacobson et al[6]. However, what about the milder degree of TSS?

Patients with a milder degree of TSS have been reported by many authors[5,11,13-15]. In our case, the patient had two episodes of probable TSS. He developed diffuse maculopapular rash involving the face, chest wall, and upper extremities two days after the second placement of anterior-posterior nasal packing, followed by fever, sore throat, high blood pressure, and respiratory distress from bronchospasm and pulmonary congestion. He also

had desquamation on the palmar side of the distal end of his fingers about 30 days after onset. The rash and other symptoms were still present even though the antibiotic was changed. There was no history of hypotension, and no laboratory confirmation of possible coagulation, hepatic, or renal abnormalities. So, he can not be diagnosed as a classic case of TSS by using CDC (Centers for Disease Control) criteria. However, the reason that his condition recovered rapidly after removal of the nasal packing and also the recurrent nature of the rash after the nasal packing was repeated which, again, recovered rapidly after removal of the nasal packing is suggestive of a milder degree of TSS. Before any specific serologic marker is available to identify all patients with TSS, the diagnosis in subjects without all features of classic TSS will depend on exclusion. The differential diagnosis of TSS includes bacteremia with shock, meningococcemia, *S. aureus* bacteremia, scarlet fever, toxic epidermal necrolysis (scalded skin syndrome), acute rheumatic fever, leptospirosis, Rocky Mountain spotted fever, rubeola, Kawasaki syndrome, erythema multiforme, and Stevens-Johnson syndrome(16). Accurate diagnosis of a milder degree of TSS can be made by maintaining a high index of suspicion for almost any illness occurring during nasal packing. We believe early recognition and prompt intervention of possible TSS is important to minimize the morbidity and mortality associated with this disease. Surgeons who perform sino-nasal surgery should keep TSS in mind, even though certain criteria for definite TSS are absent.

Management of TSS varies widely depending on the severity of the disease and its complications. This includes elimination of the focus of the infection by removal of the contaminated foreign body or drainage of the offending abscess, supportive care by aggressive rehydration, and appropriate antibiotic administration. Early administration of antistaphylococcal antibiotic therapy is essential. It reduces the risk of recurrence of TSS but does not affect the toxin already elaborated (17). Prognosis is affected by the duration of the shock, the secondary organ dysfunction, the speed of recognition, and the appropriateness of medical intervention. Prophylactic antistaphylococcal antibiotics have not been shown to alter nasal carriage of *S. aureus* and appear to be ineffective in preventing TSS(10,18).

SUMMARY

To the best of our knowledge, TSS after sino-nasal surgery has never been reported in Thailand. Rhinologic surgeons who perform sino-nasal surgery should consider the diagnosis of this disease, even though certain criteria for definite TSS are absent. In case of suspicion and definite diagnosis can not be made, it would seem prudent to begin treatment by removal of the whole nasal packing. Early recognition and prompt intervention is important to minimize the morbidity and mortality associated with this disease.

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กลุ่มอาการท็อกสิกช็อกที่เกิดจากการประจุผ้าในส่วนหน้าและส่วนหลังของโพรงจมูก

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กลุ่มอาการท็อกสิกช็อกเป็นโรคที่ทำให้เกิดความผิดปกติของระบบหลายระบบของร่างกาย อาการของโรครุนแรงและอาจถึงแก่ชีวิตได้ ประมาณว่าในประเทศสหรัฐอเมริกาพบอุบัติการณ์ของโรคนี้ 16.5 ราย ต่อการผ่าตัดจมูก 100,000 ราย โรคนี้จึงพบค่อนข้างน้อย อย่างไรก็ตามโรคนี้ชนิดที่มีอาการรุนแรงน้อยกว่าอาจพบได้มากกว่า แพทย์ที่ทำผ่าตัดจมูกและโพรงอากาศข้างจมูกควรจะนึกถึงโรคนี้ด้วยถึงแม้ว่าอาการและอาการแสดงต่างๆจะมีไม่ครบทุกอาการ ซึ่งการดูแลรักษาโดยเร็วตั้งแต่เริ่มแรกจะช่วยลดภาวะข้างเคียง ตลอดจนอัตราตายได้ รายงานฉบับนี้เป็นรายงานแรกในประเทศไทยที่รายงานผู้ป่วยที่เป็นโรคนี้ชนิดที่ไม่รุนแรงที่เกิดจากการประจุผ้าในส่วนหน้าและส่วนหลังของโพรงจมูก และได้กล่าวถึงหลักเกณฑ์ในการวินิจฉัย พยาธิกำเนิด และแนวทางในการรักษาโรคไว้

คำสำคัญ : กลุ่มอาการท็อกสิกช็อก, การประจุผ้าในโพรงจมูก, เลือดกำเดาไหล

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