Community-acquired streptococcal toxic shock syndrome

Sindrome streptococcica da shock tossico acquisita in comunità

Antonio Sanchez-Porto¹, Manuel Casanova-Roman¹, Javier Casas-Ciria¹, Pedro Diaz de Sousa²

¹Department of Microbiology; ²Department of Internal Medicine, Hospital of La Línea, Cádiz, Spain

To the Editor

Group A streptococcus (Streptococcus pyogenes) may cause a variety of illnesses ranging from very common, usually clinically mild conditions such as pharyngitis and impetigo to less common severe infections including septicemia [1].

With the resurgence of serious forms of group A streptococcal infection noted in many parts of the world, cases of streptococcal toxic shock syndrome (STSS) have been reported. The generally accepted case definition for STSS specifies isolation of the organism, substantial hypotension and two or more of the following features: renal or hepatic failure, coagulopathy, adult respiratory distress syndrome, generalized rash and soft tissue necrosis [1]. Despite optimal treatment the mortality of STSS ranges from 30 to 70% [2-6]. Recently, published data from 11 European countries gave an incidence of STSS of 13% in streptococcal infection from any source [6]. In this letter, we report a case of STSS.

An 82-year-old woman was admitted to our hospital due to a 12-hour history of fever and altered mental status. She was allergic to penicillin and she had diabetes mellitus. On admission her body temperature was 39.1°C. Her pulse rate, blood pressure and respiratory rate were normal. She was confused. On physical examination, she had a small ulcer in her left foot.

Other physical examination findings were normal. The white cell count was 17,800/mm³ with a left shift and C-reactive protein was 13.8 mg/dl. Glucose was 143 mg/dl and liver dysfunction was revealed as a total bilirubin 2.2 mg/dl, aspartate aminotransferase 126 IU/L and alanine aminotransferase 50 IU/L. The prothrombin time was 52% and the PTT was twice the normal level.

On the first hospital day, antibiotic treatment with ciprofloxacin and clindamycin was started. On the third hospital day, she developed refractory hypotension and respiratory failure. Despite maximal supportive therapy, she died. Streptococcus pyogenes was isolated from blood culture.

The organism was typed as M1, T1 which was sensitive to penicillin, cephalosporin and clindamycin and was found to be producing streptococcal pyrogenic exotoxin (SPE) B and C in vitro.

In most cases of STSS the patients are previously healthy, and the site of infection is usually skin or soft tissue, although some are puerperal or, rarely, pharyngeal.

A number of predisposing factors for non-necrotizing cellulitis broadly includes conditions involving alterations in integrity of skin; alterations in vascularity of skin and alteration of host defences (eg, diabetes mellitus) [7]. Our patient had an ulcer in her left foot and she was also diabetic.

Most (60%) patients with STSS have a positive blood culture [8]. Presence or absence of bacteremia does not affect mortality. The diagnosis of STSS is confirmed when Streptococcus pyogenes are cultured from normally sterile body fluids in a patient with shock and multi-organ failure [9].
Although Group A streptococci are extremely sensitive to penicillin, penicillin can fail in the presence of a large microbiological burden. Moreover, penicillin does not halt the production of exotoxin [10]. For several reasons, clindamycin may be effective as part of the antibiotic therapy. First, clindamycin has been demonstrated to reduce exotoxin production and superantigen production by pathogenic strains of Group A streptococci [11]. Second, clindamycin can overcome the so-called Eagle effect, which is a manifestation of decreased bacterial replication, which can occur when local concentrations of bacteria are high [10].

In the management of STSS, massive fluid resuscitation is needed. Vasopressors, inotropes, drotrecogin- (activated) and stress-dose corticosteroids should be given in patients who meet the appropriate criteria. Another therapy to consider for the treatment of STSS is IV Ig (IVIG). IVIG may provide additional immunity against M proteins and further protection by neutralizing the effects of bacterial superantigens [12].

The pathogenesis of STSS is not fully understood. However, SPEs seem to play a pivotal role in the pathophysiologic process of the disease [2, 13]. SPEs belong to a family of bacterial proteins known as superantigens, which stimulate strong proliferation of T lymphocytes with concomitant production of massive quantities of inflammatory cytokines. The cytokines then mediate shock and tissue injury [14]. This may account for the pathogenesis of STSS characterized by rapid onset of hypotension and multiorgan failure. However, the differences in the susceptibility and immunity are also involved in the severity of the infection [15].

There are multiple associations between streptococcal superantigens and invasive diseases. Recent work suggests that soluble M1 proteins may also be superantigenic, preferentially activating T cells with V2 and V4 T-cell receptors. M proteins also activate T cells via Toll-like receptor 2 [16]. In the Danish data that contributed to a European work, either SPE A or SPE C was present in all cases of STSS [6].

In conclusion, we should keep in mind that Streptococcus pyogenes is one of the infectious agents causing a life-threatening disease with rapid progression. STSS is thought to be caused by massive cytokine release stimulated by bacterial exotoxin acting as a superantigen.

REFERENCES