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References

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Methicillin-resistant Staphylococcus aureus Toxic Shock Syndrome

To the Editor: Toxic shock syndrome (TSS), which can be life threatening, is defined by clinical and laboratory evidence of fever, rash, desquamation, hypotension, and multiple organ failure caused by Staphylococcus aureus toxins. TSS caused by methicillin-resistant S. aureus (MRSA) strains has been found extensively in Japan (1), rarely in the United States (2), and, thus far, not in Europe.

We report a case of TSS due to an MRSA strain that produced a TSS toxin 1 (TSST-1). A 54-year-old woman was admitted to the emergency ward of Brugmann University Hospital, Brussels, with a 2-day history of myalgia, diarrhea, and vomiting. She had undergone surgery for a palate neoplasia 2 months earlier, and again 2 weeks earlier, in another hospital. After the second operation, she had been treated for a local scar infection with amoxicillin–clavulanic acid for 1 week.

On physical examination, the patient was conscious, tachypneic, pale, and sweating. Her temperature was 38.2°C and her blood pressure was 70/50 mm Hg. Abdominal examination findings were normal. The cutaneous operative wound was red and swollen. Laboratory results included the following: leukocyte count 19,830/mm³ with 97% polymorphonuclear neutrophils, platelets 90,000/mm³, creatinine 2.1 mg/dL, bicarbonate 13 mEq/L, cyclic AMP receptor protein 43.7 ng/mL, creatine kinase 514 U/L, and myoglobulin 38,800 µg/L.

The treatment included aggressive intravenous fluid resuscitation, administration of dopamine, and antimicrobial agent therapy with teicoplanin and clindamycin. The treatment outcome was favorable. On the second day, a diffuse cutaneous macular rash appeared. The acute renal failure and the biological abnormalities resolved. On the fifth day, the patient was transferred back to the hospital where she had undergone surgery; extensive peeling then developed on both of the patient’s hands.

Our patient met the criteria of TSS: she had fever, rash, desquamation, hypotension, vomiting, diarrhea, myalgias, elevated creatine kinase, acute renal failure, and thrombocytopenia. The diagnosis of staphylococcal TSS was confirmed by bacteriologic results.

Although TSST-1 production by MRSA strains has been described in Europe (7), this case is the first of TSS due to TSST-1–producing MRSA in Europe. Recently Nathalie van der Meer-Marquet et al. (8) described the first case of neonatal TSS–like exanthematosus disease due to a MRSA strain containing the TSST-1 gene in Europe. They emphasized the risk of emergence of neonatal toxic shock syndrome–like exanthematosus disease outside Japan.

We would also like to emphasize the rising risk of TSS due to virulent MRSA strains outside Japan and particularly in Europe. The usual recommendations for the treatment of staphylococcal TSS do not consider this possibility and consist of a β-lactamase–resistant anti-staphylococcal agent and clindamycin in some cases (to decrease the synthesis of TSST-1) (9–11).

We immediately treated our patient with teicoplanin and clindamycin because we suspected a nosocomial
infection with \( \textit{S. aureus} \), possibly MRSA. The possibility of MRSA must be considered when initiating antimicrobial agents to treat TSS.

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**Are SARS Superspreaders Cloud Adults?**

**To the Editor:** The primary mode of transmission of severe acute respiratory syndrome (SARS) appears to be through exposure to respiratory droplets and direct contact with patients and their contaminated environment. However, in summarizing their experiences during the SARS outbreaks in Toronto and Taiwan, McDonald et al. (1) note that certain persons were very efficient at transmitting SARS coronavirus (SARS-CoV), and that in certain settings these so-called “superspreaders” played a crucial role in the epidemic. Airborne transmission by aerosols may have occurred in many of these cases. The same observation has been made by others (2–4), but the causes of these superspreading events and the reasons for the variable communicability of SARS-CoV are still unclear. Possible explanations include specific host characteristics (e.g., altered immune status, underlying diseases), higher level of virus shedding, or environmental factors (1–3).

We hypothesize that superspreading events might be caused by coinfection with other respiratory viruses. Such a mechanism has been identified in the transmission of \( \textit{Staphylococcus aureus} \). Eichenwald et al. (5) showed that newborns whose noses are colonized with this bacterium disperse considerable amounts of airborne \( \textit{S. aureus} \) and become highly contagious (i.e., superspreaders) after infection with a respiratory virus (e.g., adenovirus or echovirus). These babies caused explosive \( \textit{S. aureus} \) outbreaks in nurseries. Because they are literally surrounded by clouds of bacteria, they were called “cloud babies” (5). We have shown that the same mechanism also occurs in certain adult nasal carriers of \( \textit{S. aureus} \) (“cloud adults”) (6,7). Reports indicate that viral infections of the upper respiratory tract facilitate the transmission of other bacteria, including \( \textit{Streptococcus pneumoniae} \), \( \textit{Haemophilus influenzae} \), and \( \textit{Neisseria meningitidis} \) (8). Moreover, superspreading events have also been reported in outbreaks of viral diseases such as Ebola hemorrhagic fever and rubella (3).

Some observations suggest that coinfection with other respiratory viruses might cause superspreading events with airborne transmission of SARS-CoV. First, other viral pathogens, including human metapneumovirus, have been detected together with SARS-CoV in some patients with SARS (4). Second, few patients with SARS are superspreaders, and upper respiratory symptoms such as rhinorrhea and sore throat are a relatively uncommon manifestation of SARS (with prevalences of 14% and 16%, respectively) (4). Thus, some