Richard Reithinger,*
Khoksar Aadil,* Jan Kolaczinski,*
Mohammad Mohsen,*
and Samad Hami*

*HealthNet International, Peshawar, Pakistan

References

1. World Health Organization. Cutaneous leishmaniasis, Afghanistan. Wkly Epidemiol Rec. 2002;77:246.
2. Reithinger R, Mohsen M, Aadil K, Sidiqi M, Erasmus P, Coleman PG. Anthroponotic cutaneous leishmaniasis, Kabul, Afghanistan. Emerg Infect Dis. 2003;9:727–9.
3. Reyburn H, Rowland M, Mohsen M, Khan B, Davies CR. The prolonged epidemic of anthroponotic cutaneous leishmaniasis in Kabul, Afghanistan: "bringing down the neighbourhood." Trans R Soc Trop Med Hyg. 2003;97:170–6.
4. Rasekh Z, Bauer HM, Manos MM, Iacopino V. Women's health and human rights in Afghanistan. JAMA. 1998;280:449–55.
5. Trouiller P, Olliaro P, Torreele E, Orbinski J, Laing R, Ford N. Drug development for neglected diseases: a deficient market and a public-health policy failure. Lancet. 2002;359:2188–94.
6. van Egmond K, Naeem AJ, Verstraelen H, Bosmans M, Claeys P, Temmerman M. Reproductive health in Afghanistan: results of a knowledge, attitudes and practices survey among Afghan women in Kabul. Disasters. 2004;28:269–82.

Address for correspondence: Richard Reithinger, 807 S Overlook Dr, Alexandria, VA 22305, USA; email: rreithinger@yahoo.co.uk

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% polynuclear 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. Cultures of blood, stool, and urine samples were negative for microbial agents. Puncture of the wound released 12 mL of pus; culture of the pus sample yielded an MRSA strain harboring a TSST-1 gene, detected by multiplex polymerase chain reaction as previously described (3).

By molecular typing, the strain belonged to the epidemic MRSA pulsed-field gel electrophoresis clone G10 and carried the staphylococcal chromosome cassette *mec* (SCC*mec*) type II. This clone belongs to the sequence type (ST) 5-SCC*mec* II clone, formerly named "New-York/Japan clone," which has been associated with neonatal TSS–like exanthematous disease in Japanese hospitals (4–6). This epidemic clone, which is widely disseminated in the United States, Japan, and Europe, has been found in 12% of Belgian hospitals during a national survey conducted in 2001 (6).

The treatment included aggressive intravenous fluid resuscitation, administration of dopamine, and antimicrobial agent therapy with teicoplamin 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 Mee-Marquet et al. (8) described the first case of neonatal TSS–like exanthematous 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 exanthematous 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 *S. aureus*, possibly MRSA. The possibility of MRSA must be considered when initiating antimicrobial agents to treat TSS.

Sophie Jamart,* Olivier Denis,*
Ariane Deplano,*
Georgios Tragas,*
Alexandra Vandergheynst,*
David De Bels,*
and Jacques Devriendt*

*Université Libre de Bruxelles, Brussels, Belgium

References

 1. Furukawa Y, Segawa Y, Masuda K, Takahashi M, Ootsuka A, Hirai K, et al. Clinical experience of 3 cases of toxic shock syndrome caused by methicillin cephem-resistant *Staphylococcus aureus* (MRSA). Kansenshogaku Zasshi. 1986; 60:1147–53.
 2. Meyer RD, Monday SR, Bohach GA, Schlievert PM. Prolonged course of toxic shock syndrome associated with methicillin-resistant *Staphylococcus aureus* enterotoxins G and I. Int J Infect Dis. 2001;5:163–6.
 3. Lina G, Piémont Y, Godail-Gamot F, Bes M, Peter MO, Gauduchon V, et al. Involvement of Panton-Valentine leukocidin-producing *Staphylococcus aureus* in primary skin infections and pneumonia. Clin Infect Dis. 1999;29:1128–32.
 4. Oliveira DC, Tomasz A, de Lencastre H. Secrets of success of a human pathogen: molecular evolution of pandemic clones of methicillin-resistant *Staphylococcus aureus*. Lancet Infect Dis. 2002;2:180–9.
 5. Kikuchi K, Takahashi N, Piao C, Totsuka K, Nishida H, Uchiyama T. Molecular epidemiology of methicillin-resistant *Staphylococcus aureus* strains causing neonatal toxic shock syndrome-like exanthematous disease in neonatal and perinatal wards. J Clin Microbiol. 2003;41:3001–6.
 6. Denis O, Deplano A, Nonhoff C, De Ryck R, de Mendonca R, Rottiers S, et al. National surveillance of methicillin-resistant *Staphylococcus aureus* (MRSA) in Belgian hospitals in 2001 indicates rapid diversification of epidemic clones. Antimicrob Agents Chemother. 2004;48: 3625–9.
 7. Schmitz FJ, MacKenzie CR, Geisel R, Wagner S, Idel H, Verhoef J, et al. Enterotoxin and toxic shock syndrome toxin-1 production of methicillin resistant and methicillin sensitive *Staphylococcus aureus* strains. Eur J Epidemiol. 1997;13: 699–708.
 8. van der Mee-Marquet N, Lina G, Quentin R, Yaouanc-Lapalle H, Fievre C, Takahashi N, et al. Staphylococcal exanthematous disease in a newborn due to a virulent methicillin-resistant *Staphylococcus aureus* strain containing the TSST-1 gene in Europe: an alert for neonatologists. J Clin Microbiol. 2003;41:4883–4.
 9. Waldvogel FA. *Staphylococcus aureus*. In: Mandell GL, Bennett JE, Dolin R, editors. Mandell, Douglas, and Bennett's principles and practice of infectious diseases. Philadelphia: Churchill Livingstone; 2000. p. 2069–92.
10. Sanford JP, Gilbert DN, Moellering RC Jr, Sande MA. The Sanford guide to antimicrobial therapy. 17th ed., Belgian/Luxemburg version. Hyde Park (VT): Antimicrobial Therapy, Inc.; 2003.
11. Issa NC, Thompson RL. Staphylococcal toxic shock syndrome. Postgrad Med. 2001; 110:55–62.

Address for correspondence: Sophie Jamart, Department of Intensive Care Medecine, Brugmann University Hospital, 4 Place Van Gehuchten, 1020 Brussels, Belgium; fax: 32-2-477-2631; email: sophie.jamart@chubrugmann.be

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 *Staphylococcus aureus*. Eichenwald et al. (5) showed that newborns whose noses are colonized with this bacterium disperse considerable amounts of airborne *S. aureus* and become highly contagious (i.e., superspreaders) after infection with a respiratory virus (e.g., adenovirus or echovirus). These babies caused explosive *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 *S. aureus* ("cloud adults") (6,7). Reports indicate that viral infections of the upper respiratory tract facilitate the transmission of other bacteria, including *Streptococcus pneumoniae*, *S. pyogenes*, *Haemophilus influenzae*, and *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