Severe MRSA Enterocolitis Caused by a Strain Harboring Enterotoxins D, G, and I

Technical Appendix

Methods

At LSPQ, confirmation of \textit{S. aureus} identification was performed by PCR amplification of the \textit{nuc} gene (1) and confirmation of methicillin resistance by PCR amplification of the \textit{mecA} gene (2). The detection of the \textit{lukS-PV} and \textit{lukF-PV} genes coding for PVL toxin and the toxic shock syndrome toxin 1 (TSST-1) were done by PCR (3,4). Molecular characterization was performed using \textit{spa} typing (5). The obtained genotype was associated with its corresponding MRSA epidemic type according to the standard protocol of the National Microbiology Laboratory (NML) (6). The strain was sent to the NML for further toxin characterization. The detection of \textit{Staphylococcus} enterotoxin A, B, C, D, E, G, H, and I, as well as exfoliative toxin A and B were done by PCR (4,7).

The minimum inhibitory concentrations (MICs) were determined by broth microdilution according to CLSI guidelines (8–10). The following antibiotics were tested at concentrations varying from 0.06 to 64 mg/l (except for rifampin: 0.016–16 mg/l): daptomycin, doxycycline, fusidic acid, levofloxacin, linezolid, rifampin, trimethoprim-sulfamethoxazole and vancomycin. Susceptibility to clindamycin and to erythromycin were determined by disk diffusion and inducible resistance to clindamycin was detected by D-test according to CLSI guidelines (8). High-level mupirocin resistance was determined by disk diffusion according to CLSI guidelines (8). MICs were interpreted using breakpoints established by CLSI, except for fusidic acid where EUCAST breakpoints were used (11).
References


