8 [5]) to be catalase negative. Therefore—although the allure of the catalase hypothesis is strong, its directness of explanation is soothing, and it has longstanding prominence in the field—the clinical and laboratory evidence overwhelmingly indicates that catalase is neither necessary nor sufficient for virulence in CGD.

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Staphylococcal Toxic Shock Syndrome, Superantigenicity, and Hypersensitivity

To the Editor—With great interest, we read the article by Chandy et al [1] concerning staphylococcal toxic shock syndrome (TSS) and its association with superantigenicity and hypersensitivity. The authors describe a 16-year-old girl who presented with severely acidic hyperglycemia and concomitant Staphylococcus bacteremia, with accompanying TSS without rash or desquamation. Blood and urine cultures revealed methicillin-susceptible Staphylococcus aureus. The patient was treated with clindamycin for 5 days, cefazolin for 14 days, and immunoglobulin (1 mg/kg).

The authors state that 10% of S. aureus strains produce enterotoxin, but the prevalence of TSS is much lower than 10%. Another explanation for this clinical picture is septic shock associated with community-acquired S. aureus bloodstream infection. Furthermore, the enterotoxin production could be an innocent bystander. Also, the patient did not meet the criteria for TSS because there was no rash or desquamation, but she did meet the criteria for community-acquired S. aureus bloodstream infection [2]. Given the limited clinical evidence for efficacy of immunoglobulin and clindamycin for the treatment of staphylococcal TSS, it seems unlikely that there was a causal relationship between the administration of clindamycin and immunoglobulin and the hemodynamic improvement after administration.

Therefore, we concluded that this patient had a complicated S. aureus bloodstream infection that was acquired in the community [2, 3]. This infection has a high risk of hematogenous complications, and although there was no accompanying endocarditis, a recent review in this journal recommended at least 4 weeks of high-dose anti-staphylococcal penicillin for community-acquired S. aureus bloodstream infection [2, 3]. The associated urine culture also grew S. aureus, and the patient had type 1 diabetes. Both factors are also associated with hematogenous complications and, possibly, provide an extra argument in favor of at least 4 weeks of high-dose therapy [4, 5].

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References


Reply to Landman and Groeneveld

To the Editor—We appreciate the interest of Landman and Groeneveld [1] in our study. They note that the child described in our article may have had septic shock due to community-associated Staphylococcus aureus infection rather than staphylococcal toxic shock syndrome (TSS). We cannot state definitively that the child had staphylococcal TSS, because all of the diagnostic criteria for staphylococcal TSS other than erythroderma and desquamation—the 2 criteria that the patient lacked—are common to both TSS and septic shock. It is also noteworthy that the presence of septic shock does not preclude the patient from having TSS. Although the majority of patients with staphylococcal TSS do not have blood cultures positive for S. aureus, a significant