

## To the Editor

The recent case report by Clarendon et al.[1] and the subsequent discussion within the Journal [2,3] suggests an additional cause underlying the patient's clinical condition. This patient presented with extremely aggressive shock with rapidly evolving multiple organ failure, attributed to septic shock from staphylococcal bacteremia and gastrointestinal failure. In addition to what might be termed conventional sepsis, the case report and subsequent discussions have ignored the potential contribution of staphylococcal toxin-mediated disease and "toxic shock" syndrome. Unfortunately, there is no information on the genotype and toxin gene profile of the causative organism, or indeed the antimicrobial sensitivity pattern. Staphylococcal superantigenic toxins stimulate the immune system with up to 1000-fold potency in comparison with conventional antigens, and the resulting cytokine storm can be catastrophic [4].

Although research case definitions for staphylococcal toxic shock syndrome typically include rash with subsequent exfoliation, exfoliative toxin production and desquamating rash are not universal features. It is important to consider the potential contribution of toxic shock to fulminant Gram-positive sepsis, because the clinical management can then be tailored through the addition of antimicrobials capable of modulating toxin production [5] (e.g., clindamycin and linezolid), and consideration can be given to administration of IV immunoglobulin, which contains antibodies to these superantigens (often absent or at low titer in susceptible patients). Concerns have been expressed regarding empiric use of vancomycin and other glycopeptides for *S aureus* bacteremia, given the potential for reduced sensitivity to these agents [6], although in this case meropenem was also administered. Staphylococcal toxins can be produced by methicillin-sensitive or methicillin-resistant *S aureus*, and even by coagulase-negative staphylococci, and vigilance is key. Toxic shock syndrome must be considered in cases in which Gram-positive organisms are isolated in the presence of virulent sepsis, and additional investigations to characterize the infecting organism are warranted. Aggressive antimicrobial therapy is critical and IV immunoglobulin should be considered in cases in which improvement does not rapidly follow surgical management.

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## REFERENCES

1. Clendenen SR, Robards CB, Wang RD, Greengrass RA. Continuous interscalene block associated with neck hematoma and postoperative sepsis. *Anesth Analg* 2010;110:1236 – 8
2. Naughton R, Mannion S. Staphylococcal infections, not just skin deep. *Anesth Analg* 2011;112:992–3
3. Clendenen SR, Robards C. In response. *Anesth Analg* 2011;112:993
4. Lappin E, Ferguson AJ. Gram-positive toxic shock syndromes. *Lancet Infect Dis* 2009;9:281–90
5. Stevens DL, Ma Y, Salmi DB, McIndoo E, Wallace RJ, Bryant AE. Impact of antibiotics on expression of virulence-associated exotoxin genes in methicillin-sensitive and methicillin-resistant *Staphylococcus aureus*. *J Infect Dis* 2007;195:201–11
6. Thwaites GE, Edgeworth JD, Gkrania-Klotsas E, Kirby A, Tilley R, Torok ME, Walker S, Wertheim HFL, Wilson P, Llewelyn MJ. Clinical management of *Staphylococcus aureus* bacteraemia. *Lancet Infect Dis* 2011;11:208 –22