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Pristinamycin in the treatment of MSSA bone and joint infection—authors' response

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Sir,

This is our response to Plant and Auckland[1] concerning our study.[2] Our study was primarily designed to describe pristinamycin use and tolerance during treatment of *Staphylococcus aureus* bone and joint infection (BJI). The retrospective design and the heterogeneity of antibiotic regimens and surgical strategies led us to consider outcome data with caution. However, and although the limited number of patients does not allow definite conclusions, it seemed important to mention that the three patients infected with an erythromycin- and clindamycin-resistant isolate experienced treatment failure. Previous series reporting an acceptable efficacy of pristinamycin in erythromycin-resistant *S. aureus* BJI did not provide resistance mechanisms of the implicated isolates. As proven by DNA microarray analysis, all of our three isolates harboured the *erm* gene, conferring the MLS_B resistance phenotype of which apparent *in vitro* pristinamycin susceptibility is due to the moderate bacteriostatic activity of the S_A compound. However, only the bactericidal synergy of the two compounds enables a 100-fold higher activity compared with each of the compounds alone.[3] Even if the clinical interest of the mechanism of antibiotic killing in BJI is still debated, bactericidal agents may be theoretically superior to bacteriostatic ones.[4] We fully agree with the authors regarding the need of randomized controlled trials addressing this question, which would be difficult to answer clinically. Pending such data, our clinical experience leads us to avoid pristinamycin use in patients bearing *S. aureus* isolates with the MLS_B resistance phenotype when other bactericidal agents with acceptable bone penetration are available.

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Transparency declarations

None to declare.

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