LETTER TO THE EDITOR

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Detection of inducible clindamycin resistance ($MLSB_i$) among methicillin-resistant *Staphylococcus aureus* (MRSA) from Libya

Methicillin-resistant *Staphylococcus aureus* (MRSA) first emerged as nosocomial pathogens in the early 1960s are of great concern to public health and highly reported in human clinical samples (1). There are major international concerns about rising levels of MRSA and multi-drug resistant *S. aureus* owing to the difficulties of treating infections and the ease with which MRSA spreads within hospitals (2). Until recently, most infections of MRSA were acquired primarily in hospital settings, but now MRSA is responsible for both hospital and community-acquired infections (1). The objective of this study was to investigate MRSA collected isolates for MLSB phenotypes, in particular inducible clindamycin resistance ($MLSB_i$).

MRSA collected isolates of hospital-origin were further investigated at the Microbiology Department, Biotechnology Research Centre, Tripoli, Libya. One hundred and twenty-eight MRSA isolates were confirmed at species level as *S. aureus* by culturing onto mannitol salt agar (MSA) and API Staph test strips (bioMerieux). Confirmation as MRSA was by latex agglutination test for PBP2a and disc diffusion method against cefoxitin in accordance with British Society of Antimicrobials and Chemotherapy guidelines (BSAC) as described by Andrews (3). Isolates were also tested against erythromycin, clindamycin, and synercid for the characterization of MLSB phenotypic isolates. *D*-tests were performed on isolates exhibiting erythromycin resistance, to assay for the presence of inducible clindamycin resistance ($MLSB_i$ phenotype) as described by Fiebelkorn et al. (4).

Of the 128 tested MRSA isolates, 24.2% ($n=31$) were resistant to clindamycin, 63.2% ($n=81$) isolates were resistant to erythromycin, and 17.9% ($n=23$) were resistant to synercid. Twelve isolates (9.3%) exhibited $MLSB_c$ (constitutive) phenotype and *D*-tests identified six isolates (4.6%) with inducible resistance to clindamycin ($MLSB_i$ phenotype).

Clindamycin is an efficient and economic lincosamide drug used for the treatment of staphylococci infection (5). One macrolide resistance mechanism, modification of a drug binding site on the ribosome, results in resistance to macrolides, azalides, lincosamides, and group (B) streptogramins (MLSB). MLSB phenotypes can be either constitutive ($MLSB_c$) or inducible ($MLSB_i$). The inducible resistance to clindamycin ($MLSB_i$) in MRSA can severely compromise therapy and can result in failure of clindamycin treatment of MRSA infections when non-suitable therapy (e.g. erythromycin) is given (6). $MLSB_i$ strains can be successfully treated with clindamycin; however $MLSB_i$ can complicate therapy when $MLSB_i$ phenotype-switching into $MLSB_c$ occurs possibly due to mutation, in the absence of macrolide inducers (6). Clindamycin can still be used for MRSA infections in our hospitals. (7) However, susceptibility testing for the detection of inducible resistance to clindamycin should be routinely performed (7, 8).

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