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Association between vancomycin MIC with virulence genes expression and clonal complexes of methicillin-susceptible Staphylococcus aureus (MSSA) strains isolated from left-sided endocarditis

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Background: Higher vancomycin MICs have been associated with more complicated courses and higher mortality rates in *S. aureus* bacteremia and endocarditis, regardless of methicillin-susceptibility or the class of antibiotic used for treatment.

Material/methods: An analysis was performed on MSSA isolates from a previously published cohort (*Cervera C et al. CID. 2014; 58:1668*) demonstrating higher mortality rates and systemic emboli in MSSA left-sided endocarditis treated with beta-lactams where vancomycin MIC > 1 mg/L. The cohort included 53 isolates with vancomycin MIC < 1.5 mg/L and 40 with vancomycin MIC ≥ 1.5 mg/L. Mortality of MSSA endocarditis was three-fold higher among patients with high vancomycin MIC isolates. Isolates underwent spa typing to infer clonal complex (CC), biofilm studies and multiplex polymerase chain reaction for the presence of virulence genes.

Results: We found no differences in adhesins [fibronectin-binding proteins (fnbA, fnbB), clumping factors (clfA, clfB), collagen-binding antigen gene (cna), serine-aspartate repeat proteins for adhesion (sdrC, sdrD, sdrE), sialoprotein (bbp) and elastin-binding protein (ebps), and MHC class II analog proteins (MAP/EAP)], toxins [exfoliative toxins (eta, etb), enterotoxins (tst), staphylococcal enterotoxins (sea,seb), enterotoxins (sec, sed, see, seg, seh, sei, sej), Paton-Valentine leucocidin (PVL), and hemolysin (hlg)] or other putative virulence [fibrinogen-binding protein (efb), adhesion intracellular protein A (icaA), chemotaxis-inhibiting protein (chp), and serine endopeptidase (V8)] genes between MSSA isolates according to vancomycin MIC.Clonal complexes CC30, CC34 and CC45 represented nearly half of isolates and there was no association with vancomycin MIC. Agr subgroups I and III predominated, with no association with vancomycin MIC. Isolates with lower vancomycin MICs exhibited higher ability to form biofilm with and without the presence of vancomycin (2.48 vs 2.03 [p<0.001] and 2.87 vs 2.60 [p=0.022], respectively).

Conclusions: MSSA with higher vancomycin MICs exhibited less ability to form biofilms. No association was found between adhesins, toxins, agror other virulence gene expression and clonal complexes according to vancomycin MIC. Future studies should address whether a lower ability to form biofilms is associated with higher risk of embolism in left-sided endocarditis.