Antibiotic susceptibility of clinical *Staphylococcus aureus* isolates and their biofilm formation capacity at varying concentrations of different antibiotics

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- Yousif Jahfer Khdhir, Sedat Çam & Rozhhalat Khudhur Jarjees

Abstract

- *Staphylococcus aureus* is a pathogen for both humans and animals, causing severe chronic infections due to its increased antibiotic resistance and biofilm formation capacity upon antibiotic exposure. This study evaluated the susceptibility of 40 clinical *S. aureus* strains against multiple antibiotics by VITEK 2 system and their in vitro biofilm-producing ability at 1/2 and 1/4 minimum inhibitory concentrations (MICs) of the selected four antibiotics. In the tested 18 antibiotics, the highest resistance was observed against benzylpenicillin (90%), cefoxitin (75%), oxacillin (67.5%), erythromycin (52.5%), clindamycin (50%), and tetracycline (47.5%). None of the strains were resistant to linezolid, teicoplanin, vancomycin, and tigecycline. Screening of drug resistance indicated that the majority (63.3%) of methicillin-resistant *S. aureus* (MRSA) strains were

multidrug + extensive drug-resistant. ~ 74% of biofilm-forming strains were MRSA. 65% of biofilm-producing MRSA strains were moderate to strong biofilm producers. Biofilm formation was generally enhanced by both 1/2 and 1/4 MICs of erythromycin, ciprofloxacin, tetracycline, and trimethoprim/sulfamethoxazole, ranging from 35 to 55% compared to the control groups without antibiotics. The highest increase in biofilm production by most strains was observed at 1/4 MICs of the respective four antibiotics (Bonferroni post hoc test; $P < 0.05$). Biofilm production in clinical *S. aureus* strains was differently influenced by strain-to-strain differences and the types and concentrations of antibiotics. Sub-MICs of antibiotics induced biofilm production for greater resistance to antibiotic stress. These data suggest that erythromycin, ciprofloxacin, tetracycline, and trimethoprim/sulfamethoxazole exposure at sub-inhibitory concentrations may contribute to the failure of clinical treatment regimens by enhancing *S. aureus* biofilm formation.