Abstract

The aim of this thesis is to describe the mechanism of the resistance to glycopeptide antibiotics in a genus *Staphylococcus*, especially in a species *Staphylococcus aureus* which is common cause of nosocomial infections resulting frequently in expensive and long-term treatment. This pathogen is dangerous due to its ability to acquire resistance to most antibiotics used in a clinical practice. The resistance of these microorganisms can develop very easily due to inappropriate treatment (administration, drug concentration, duration), which, if not detected, could ultimately results in treatment failure and the death of the patient. The vancomycin resistance of *S. aureus* could be divide into groups according to their values of vancomycin MIC: VSSA, VISA, hVISA and VRSA. Vancomycin intermediate resistance is associated with mutation, e.g., which affect cell wall synthesis. In contrast, VRSA is associated with the transfer of the mobile genetic element with the *vanA* or *vanB* operon from genus *Enterococcus*. This transmission is due to co-infection with both pathogens. Glycopeptide resistance has also been shown to be very common in coagulase-negative staphylococci (CNS), such as *S. capitis*, which cause infection in preterm infants. Glycopeptide resistance in CNS and intermediate resistance of *S. aureus* is associated with characteristic phenotypic features such as a thicker cell wall and irregular division. This work also focuses on the prevalence of VISA, hVISA and VRSA strains.

Key words: *Staphylococcus aureus*, vancomycin, VISA, hVISA, sVISA, VRSA, coagulation-negative staphylococci