

Role of NSAIDs in Modulation in Resistance of Multi-Drug Resistant *Staphylococcus aureus*

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Staphylococcus aureus is gram positive bacteria and one of the most common cause of nosocomial infections, septicemia and surgical wound infections. More than 90% of *S. aureus* strains contain plasmid that encodes beta-lactamase enzyme that in turn degrades many but not all penicillin. Some strains of *S. aureus* are resistant to beta-lactamase-resistant penicillin such as methicillin by virtue of its changes in penicillin binding proteins in their cell membranes. These strains are commonly known as methicillin resistant *S. aureus* (MRSA). MRSA cause both health care-acquired and community acquired infections in addition to human health impacts. Due to excessive use of antibiotics, multidrug resistance is exhibited by *S. aureus* thus becoming MRSA (methicillin-resistant *S. aureus*) strains to complicate animal and public health. The only use of antibiotic is no longer enough for treating such infections due to extensive resistance exhibited by the bacteria. So, the new approaches other than antibiotic sources are required to cover such infections. NSAIDs are reported as non-antibiotics drugs, having reported anti-bacterial activity alone and in combination with antibiotics. These non-antibiotic drugs may act through mechanisms that are different from those exhibited by antibiotics thus enhancing antibiotic activity or reversing antibiotic resistance. The mostly used NSAIDs such as ibuprofen, aspirin, diclofenac and celecoxib etc. to treat fever, pain and inflammation will synergize the effect of antibiotics like chloramphenicol and cefuroxime, as reported in previous studies.

The research has noted interaction between ibuprofen/aspirin with cefuroxime against methicillin-sensitive *S. aureus* (MSSA) and the MRSA reference strain, whereas for MRSA clinical strains additive effects were observed for both NSAIDs and cefuroxime combinations. The combination of chloramphenicol with ibuprofen/aspirin was synergistic against all the tested MRSA strains and displayed an additive effect against MSSA. Celecoxib increases the sensitivity of bacteria to ampicillin, kanamycin, chloramphenicol, and ciprofloxacin by accumulating the drugs inside the cell, thus reversing resistance in bacteria. Accumulating evidence suggests that the cyclooxygenase-2 (COX-2)-specific inhibitor celecoxib would not only inhibit COX-2 but also help in the reversal of drug resistance. It is fact that NSAIDs alone don't have much power to fight with MRSA but with some specific antibiotic involve different mechanisms that altered bacterial growth, but some are still unclear. It has been found that diclofenac inhibits the DNA synthesis. This also works in a manner that under the effect of diclofenac sodium, increases the uptake of ethidium bromide by *S. aureus* cells so compromise cellular integrity. Changes in bacterial transcriptome has been observed under subinhibitory concentrations of diclofenac. Celecoxib decreases the levels of all inflammatory cytokines tested including tumor necrosis factor- α , interleukin-6, interleukin-1 beta, and monocyte chemo attractant protein-1 in wounds caused by MRSA infection. It is the need of the hour to replace antibiotics with NSAIDs like non-antibiotic chemicals where double benefit in terms of reduction in inflammation and antibacterial synergism may be found.

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