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Editorial

Antibacterial activities of Meloxicam, Ibuprofen and Diclofenac: an Additional Approach to their Immunological Therapy

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Non-steroidal anti-inflammatory drugs (NSAIDs) are anti-inflammatory, antipyretic, and analgesic drugs that are frequently utilized in the treatment of musculoskeletal disorders, mild to moderate pain, and fever [1]. Such immunological activities are not the sole method by which NSAIDs can prevent diseases. They also have direct potential inhibitory effects on several types of organisms. Antibacterial activities of some NSAIDs have been observed against different pathogenic bacteria.

In aspect of meloxicam activity, the biological activities of Pseudomonas aeruginosa (PAO1), such as biofilm formation, motility, matrix synthesis, and expression of quorum-sensing genes, are inhibited by meloxicam [2]. Another strain of P. aeruginosa showed resistance to meloxicam even though all other strains of Gram-negative and Gram-negative bacteria were sensitive to it [3]. Production of staphyloxanthin, one of the virulence factors produced by Staphylococcus aureus, and its encoded genes, was inhibited by 47-59 µg/mL of meloxicam or diclofenac [4]. Antibacterial activities against Gram-positive and Gram-negative bacteria can be achieved by synthesizing synthetic ligand complexes from meloxicam with glycine [5]. Complex type A in that study had the lowest MIC on Listeria spp. and Escherichia coli (10.8 µg/mL), making it the most antibacterial agent.

In addition to meloxicam, ibuprofen, and diclofenac have antibacterial effects on several clinical bacterial isolates. Diclofenac could have more antibacterial effects on bacteria than ibuprofen. Of four NSAIDs, diclofenac had strong antibacterial action on the methicillin-resistant S. aureus (MRSA) strain and other ten clinical isolates of bacteria, followed by ibuprofen, which only failed to affect Bacillus cereus [6]. The study of the antibacterial action of different pharmaceutical drugs on 89 strains of Gram-negative bacteria showed that 5 of 12 NSAIDs have strong antibacterial activities on the bacterial species at MIC >3200 mg/L, including diclofenac (100%), acetylsalicylic acid (100%), and salicylamide (76%) [7]. Other members showed either lower activities, such as ibuprofen (35%) and naproxen (29%), or no activities, as with indomethacin, mefenamic acid, and meloxicam. E. coli strains isolated from the UTI were significantly inhibited by low concentrations of diclofenac (MIC90, 256 µg/mL) compared to high concentrations of ibuprofen (MIC90, 1024 µg/mL) [8]. Increasing the concentration of diclofenac may be required to inhibit resistant strains of bacteria, such as multidrug-resistant Proteus mirabilis [9]. However, the antibacterial action of diclofenac cannot hide the inhibitory effects of ibuprofen on different bacteria, particularly on Gram-positive bacteria, compared to Gram-negative bacteria [10]. Additionally, the antibacterial activity of NSAIDs can be affected by the pH value of the media, and any elevation in the pH above 7 can block these activities [10].

Multidisciplinary therapeutic uses of NSAIDs for treatment of immunological and antimicrobial diseases promote them to be the best choice in cases with serious infectious diseases. They could be an alternative to the antibiotics with low antibacterial action.

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