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THE SCOPE OF ANTI-INFLAMMATORY DRUGS IN MEDICAL PRACTICE AND THE RELEVANCE OF THEIR IMPROVEMENT

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Abstract. There are significant ramifications for public health, treatment, and prevention when it comes to the correlation and causal role of infectious pathogens in chronic inflammatory illnesses. Antibiotics and anti-inflammatory medications are among the many medications that must be administered in the pharmacological treatment of combination infection and inflammatory illnesses. However, this can cause adverse effects, and therefore, dual-action drugs need to be developed. Promising candidates seem to be anti-inflammatory medications that have demonstrated antibacterial qualities. Clinical trials involving individuals with cellulitis and uncomplicated UTIs were conducted to investigate NSAIDs, specifically aceclofenac, diclofenac, and ibuprofen. Patients with UTIs experienced symptom relief when ibuprofen, a medication studied in the greatest number of studies, was administered. Furthermore, ibuprofen showed strong in vitro antibacterial activity against *Bacillus cereus, Escherichia coli* and *Staphylococcus aureus*, including methicillin-resistant *S. aureus* (MRSA) (MIC 0.625–2.5 mg/L), and generated a high survival rate in mice infected with Pseudomonas aeruginosa. The majority of anti-inflammatory medications only have data demonstrating their antibacterial properties in vivo and in vitro. Among these, mice afflicted with *Enterococcus faecium, S. aureus* and *Clostridium difficile* showed a high survival rate when treated with auranofin. Additionally, it demonstrated a potent in vitro growth-inhibitory action against *Bacillus subtilis, C. difficile, E. faecalis, E. faecium, Streptococcus agalactiae, S. pneumoniae, S. aureus, S. epidermidis, E. faecalis* and *Mycobacterium TB* (MIC 0.0015–5 mg/L). Aspirin has demonstrated strong to moderate in vitro action against *E. coli, B. cereus, P. aeruginosa, Enterobacter aerogenes, Klebsiella pneumoniae* and *Salmonella choleraesuis* (MIC 1.2–5 mg/L) and a good survival rate in mice infected with *M. tuberculosis*. Additionally, mice's MRSA load was significantly reduced when celecoxib was applied topically. But in vitro, it only had mild effects against *Bacillus subitilis, S. aureus* and *S. epidermidis* (MIC 16–64 mg/L). According to these findings, some non-steroidal anti-inflammatory medicines (NSAIDs) show promise as dual-action medication candidates for the possible treatment of infectious and inflammatory conditions such tuberculosis, musculoskeletal infections, and urinary tract infections. However, before these NSAIDs are used in practice, more clinical trials must be carried out to determine their bactericidal efficacy.

Keywords. Bacterial infection, cellulitis, cystitis, inflammation, osteomyelitis, septic arthritis, dual-action medications, single-drug treatment.

Introduction. There may be a link between infectious pathogens and chronic inflammatory disorders, according to clinical and epidemiological research. For instance, urinary tract infections (UTIs), musculoskeletal infections (MSKIs), and tuberculosis (TB) can all cause persistent inflammation, which can result in serious tissue damage. Patients may need to be treated with both antibiotics and anti-inflammatory medications in order to combat these illnesses. However, because of drug-drug and drug-disease interactions, administering numerous drugs (a practice known as polypharmacy) may have negative health effects. For instance, there is an increased risk of over-anticoagulation when large dosages of amoxicillin/clavulanate are administered in conjunction with warfarin. Overmedication is a common problem in the modern

day and is frequently linked to self-medication. Self-medication is the practice of taking medication with out-of-date prescriptions or without a legitimate medical prescription [1-5]. Without proper scientific oversight, it entails people accepting personal responsibility for their medication based on accepted information and beliefs. In Brazil, this behavior is particularly common. Numerous negative consequences, most notably the rise of bacterial strains resistant to antibiotics, have been linked to this technique. One of the main topics of discussion in the modern era is antimicrobial resistance. Research has shown a worrying growing trend in widespread bacterial resistance, especially among antibiotics classified as "reserve antibiotics," highlighting its direct effects on mortality rates, hospitalization rates, and quality of life. Additionally, there is worry that the combination of antihypertensive medications with non-steroidal anti-inflammatory drugs (NSAIDs) may raise the risk of acute renal illness. multiple-action medicines, which are molecules that combine two distinct desirable pharmacological actions and feature multiple mechanistic effects due to their targeting of various effector systems, are widely needed to minimize the severe side effects induced by polypharmacy [6-12]. In addition to fewer side effects, using these medications can increase their effectiveness. For instance, compared to selective acetylcholinesterase inhibitors like donepezil and galantamine, rivastigmine, a dual inhibitor of acetylcholinesterase and butyrylcholinesterase, showed noticeably higher responses of cognitive and behavioral functions in dementia patients. Similarly, bupropion, an antidepressant drug used to treat depression, functions by dual suppression of norepinephrine and dopamine reuptake, according to preclinical and clinical research. In addition to being as effective as other antidepressants, bupropion therapy does not cause sleepiness, weight gain, or other frequent antidepressant adverse effects. Romosozumab is a dual-action osteoanabolic medication used to treat osteoporosis. It has a dual effect by promoting bone formation and decreasing bone resorption by binding to and inhibiting sclerostin, a natural inhibitor of bone formation. Because romosozumab exhibits both osteoanabolic and antiresorptive properties, it is best described as a dual agent. Romosozumab serves as an excellent example of the potential market for dual-action medications in the future [13-20]. The use of multi-target medications, which concurrently act on several routes, is another viable substitute for polypharmacy. For complicated illnesses like cancer, inflammation, diabetes, and problems of the central nervous system, this method provides safer and more efficient therapy alternatives. Imatinib, for instance, is a successful multi-target medication used to treat gastrointestinal stromal tumors and chronic myelogenous leukemia. This medication selectively targets platelet-derived growth factor receptors implicated in cancer signaling, tyrosine-protein kinase ABL1, and proto-oncogene c-Kit. Drug repurposing, deprescribing, and targeted drug delivery systems are other tactics. By facilitating the administration of medications to their precise target spot in the body, a targeted drug delivery system increases therapeutic effectiveness while lowering side effects. Because of their analgesic and anti-inflammatory qualities, non-steroidal anti-inflammatory medications (NSAIDs) are widely prescribed and used in a variety of healthcare settings to treat pain. However, despite their beneficial effects, this class of medications might present serious hazards to the patient because of potential interactions and side effects, particularly for polypharmaceutical individuals [21-28]. A major global public health concern is the possibility of upper gastrointestinal (GI) problems, particularly when using NSAIDs. Traditional NSAIDs that are sold over-the-counter have a wide range of risk characteristics. Combinations of two or more NSAIDs found in seemingly safe drugs used to treat colds and the flu are especially concerning. To assess the risk-benefit profile of NSAIDs, demographic data on medicine consumption—including the frequency of self-medication—must be gathered. Making educated decisions about health policies requires this information. Regular NSAID use has been linked to a two- to three-fold increase in the risk of cardiovascular events,

gastrointestinal tract problems, and mild to severe renal damage. A patient may take an aspirin for a headache, a cold remedy (such as paracetamol + antipyretics and nasal decongestants), or other combinations (like metamizole + muscle relaxants) on the same day without realizing it, possibly going over the recommended daily dosages for NSAIDs. The likelihood of suffering adverse effects from these drugs can be greatly increased by this everyday behavior [29-35]. This study aims to clarify the trends of antibiotic and NSAID use among Brazil's adult population. The 90-day period prior to the survey will be the focus of this study, which will include both prescription and self-medication use in this population. In light of this, we are able to consider the suggestion of health policies that seek to reduce instances of irrational medicine use and self-medication. Most significantly, the risk of adverse drug reactions brought on by co-administration of numerous drugs may be decreased by utilizing NSAIDs as a single-drug therapy. However, a thorough analysis of the findings of recent studies on these medications' antimicrobial effects is still lacking. The in vitro, in vivo, and clinical data regarding the antibacterial effectiveness of anti-inflammatory medications are thus compiled and critically examined in this study [36-41].

The main purpose of the presented manuscript is to provide a brief overview of the results of reputable scientific papers on the scope of the use of anti—inflammatory drugs in medical practice and the relevance of their improvement.

How nonsteroidal anti-inflammatory medications work. Cyclooxygenase inhibition is linked to the analgesic and anti-inflammatory properties of nonsteroidal anti-inflammatory medications (NSAIDs). The majority of NSAID side effects are likewise caused by this mechanism. After cell stimulation, such as cell injury, phospholipase A2 releases arachidonic acid from cell membrane phospholipids, which is necessary for the synthesis of prostaglandins (PGs). After then, arachidonic acid proceeds through a sequence of enzyme processes known as the arachidonic acid cascade, in which cyclooxygenase (COX) and lipoxygenase (LOX) are the two primary routes. PG, prostacyclin, and thromboxanes (TX) are prostanoids produced by the COX route, whereas leukotrienes are produced by the LOX pathway (ketoprofen is one of the few NSAIDs that inhibit this mechanism). COX-1 and COX-2 are the two recognized types of COX [1-6]. Although the two isoforms are encoded by different genes, located in different cells, and exhibit different actions, they are roughly 60% homologous and share similar molecular weights and active locations. Through the production of PGs, the constitutive enzyme COX-1 protects the stomach mucosa and influences blood vessels. Damaged tissues, endothelial cells, macrophages, and fibroblasts all produce COX-2. It is the enzyme's inducible form and is crucial to inflammatory reactions. By acetylating the COX enzyme, NSAIDs function as competitive COX inhibitors. The acetyl group of NSAIDs forms a covalent bond with the hydroxyl group of serine (Ser-530 in human platelets) at the N-terminal end of the COX molecule during this process. This blockage cannot be lifted. By preventing arachidonic acid from reaching the COX catalytic center, serine acetylation results in nonspecific inhibition of the enzyme-substrate reaction. Therefore, NSAIDs only block the endoperoxide production complex reaction during its initial COX-mediated stage; they have no effect on the reaction involving peroxidase [7-13].

Nonsteroidal anti-inflammatory medication (NSAID) division. NSAIDs are a class of medications with a range of chemical compositions and potential applications, but they have at least three crucial characteristics: the same pharmacological characteristics, a similar fundamental mode of action, and comparable side effects. They are frequently separated into more manageable, consistent groupings because of the great diversity of medications in this category. The chemical structure of NSAIDs can be used to categorize them. The ability of NSAIDs to block the activity of certain COX isoenzymes, however, seems to be the most clinically significant classification [14-21].

The categories of NSAIDs are as follows:

nonselective COX-1 inhibitors (also known as traditional NSAIDs), which have a higher affinity for COX-1 than for COX-2;

selective COX-1 inhibitors, such as acetylsalicylic acid at a cardiac dosage of 75–150 mg.

Preferential COX-2 inhibitors, which have a higher affinity for COX-2 than for COX-1, include ibuprofen (which has the highest affinity for COX-2 in this group),

diclofenac, ketoprofen, and naproxen (which have an intermediate affinity for COX-2), acetylsalicylic acid in the classic dose, piroxicam, and indomethacin (which have the lowest affinity for COX-2 in this group).

Nimesulide, meloxicam, and aceclofenac are examples of selective COX-2 inhibitors (also known as coxibs) that have an affinity for COX-2 that is at least 200 times greater than that of COX-1. At the moment, celecoxib and etoricoxib are among them [22-28].

Nonsteroidal anti-inflammatory medication use. NSAIDs are still widely used and play a significant role in rheumatology and the larger pain management approach, despite significant advancements in pharmacotherapy and the introduction of numerous new medications. International therapeutic standards state that NSAIDs, also known as "first-line drugs," are the main class of medications used to treat seronegative spondyloarthropathies. Additionally, NSAIDs are used to treat rheumatoid arthritis, juvenile idiopathic arthritis, various autoimmune illnesses that cause arthritis, overload syndromes of the so-called soft tissue rheumatism, and, most commonly, osteoarthritis (OA) and spondyloarthritis. When selecting NSAIDs for osteoarthritis, it is important to consider their chondroprotective impact, or how they protect articular cartilage. In addition to their anti-inflammatory properties, some NSAIDs, such as coxibs, promote articular cartilage by promoting the production of hyaluronate and glycosaminoglycans (GAGs) as well as the extracellular matrix of cartilage [5-12]. Unfortunately, some findings suggest that ibuprofen and naproxen impede the formation of glycosaminoglycan in articular cartilage. NSAIDs are used to treat almost every kind of pain, including pain from cancer. On the so-called analgesic ladder, they are the first rung. They are prescribed as analgesics for a variety of conditions, including migraines, menstrual pain, neuralgia, root syndromes, discopathy, post-traumatic and muscle pain, pain following surgery or tooth extraction, and renal and hepatic colic. They can be used in conjunction with opioids for more severe pain. Unfortunately, dyspeptic symptoms, damage to the gastric and duodenal mucosa (erosions, ulcers, gastrointestinal bleeding, perforation), impaired renal function and renal papillary necrosis, liver damage, increased cardiovascular risk and symptoms of circulatory failure, hemolytic anemia, granulocytopenia or impaired platelet function, ototoxic effects, and hypersensitivity reactions (skin lesions, aspirin-induced asthma). The introduction of selective COX-2 inhibitors, or "coxibs," into medicine appeared to be a significant advancement. Compared to COX-1, these substances show an affinity for COX-2 that is more than two hundred times greater. Although selective COX-2 inhibitors have been demonstrated to have less gastrointestinal side effects in big clinical trials, this does not necessarily mean that total treatment problems will decrease [31-41].

Cardiovascular complications associated with NSAID use. NSAID use in cardiac patients is addressed in several different papers, which are mentioned below. The European Society of Cardiology (ESC) guidelines for the treatment of non-ST-elevation myocardial infarction (NSTEMI) indicate that NSAIDs, such as ibuprofen and naproxen, can inhibit the irreversible blockade of this enzyme by acetylsalicylic acid through combining with COX-1. Selective COX-2 blocking is also associated with a risk of prothrombotic effects. For this reason, the use of these drugs in combination therapy with acetylsalicylic acid should be avoided (Recommendation Class III, Level of Evidence C). Aceclofenac, celecoxib, diclofenac and ketoprofen should be

preferred as these drugs do not exhibit this effect and can be combined with acetylsalicylic acid. On the other hand, NSAIDs are not advised as analgesics for anginal discomfort in the ESC guidelines for ST-elevation myocardial infarction (STEMI) [17-23]. These recommendations emphasize that the use of NSAIDs other than acetylsalicylic acid, such as selective COX-2 inhibitors, should be stopped in cases of STEMI since they raise the risk of death, re-ischemia, heart rupture, and other sequelae. Additionally, because NSAIDs quadruple the risk of acute heart failure, decrease renal function, and even raise the chance of gout exacerbation, they should be administered especially carefully in heart failure patients. In conclusion, regardless of treatment method, NSAIDs (including nonselective NSAIDs and COX-2 inhibitors) should not be used three to six months following an acute coronary syndrome since high doses of these medications have been linked to an elevated risk of cardiovascular disease. Additionally, extra care must be taken when using NSAIDs for cardiac arrhythmias. Nearly 2% of people worldwide suffer with atrial fibrillation (AF), the most often diagnosed arrhythmia. Nearly 30% of adults over 80 experience persistent AF, and the frequency rises with age. These individuals are treated with anticoagulants, including a new class of medications known as novel oral anticoagulants, or NOACs, to prevent stroke. These include the direct thrombin inhibitor dabigatran and the factor X inhibitors rivaroxaban and apixaban. Because naproxen interacts with certain medications, it is not advised to use it while taking them [28-39].

Gastrointestinal issues when taking NSAIDs. Gastrointestinal side effects account for the great majority of NSAID-related side effects. Reduced PGE2 effect, decreased submucosal flow, mucus, and bicarbonate production, as well as increased Helicobacter pylori cytotoxicity and decreased gastric juice volume that lowers pH and inhibits angiogenesis and cell proliferation, all of which jeopardize healing processes, are all consequences of COX-1 inhibition. Additionally, the activation of myeloperoxidase and free radicals causes direct damage to the gastrointestinal mucosa. Although the upper gastrointestinal tract is far more commonly affected than the lower, the entire gastrointestinal tract is impacted. Dyspepsia (epigastric pain, postprandial fullness, or early satiety), loss of appetite, belching, various abdominal pains, nausea and vomiting, increased symptoms of gastro-oesophageal reflux disease, and bowel movement disorders (diarrhea, constipation, and flatulence) are the most frequently reported gastrointestinal symptoms. Weight loss and indications of gastrointestinal bleeding (vomiting blood or substances that resemble coffee grounds and/or blood in stools or tarry stools) are less frequent symptoms [11-19]. Old age (> 70), renal and hepatic disease, a history of peptic ulcer disease, smoking, alcoholism, dialysis, Helicobacter pylori infection, COX isoenzyme blocking with predominance of COX-1, high NSAID dosage, use of more than one NSAID, use of H2 blockers (which do not protect against NSAID-related complications), and concurrent use of other medications that harm the gastrointestinal mucosa (corticosteroids, bisphosphonates, anticoagulants, and mucolytics). Combining NSAIDs with low dosages of acetylsalicylic acid is crucial since it is linked to a markedly increased risk of gastrointestinal issues, particularly in heart patients. Effective prevention of harmful medication interactions is made possible by understanding how different NSAIDs affect hepatic metabolism. Patients using statins (atorvastatin), antibiotics (such clarithromycin or azithromycin), or medications that are heavily metabolized in the liver by cytochrome CYP3A4 (like paracetamol) must choose their NSAIDs carefully. Furthermore, by blocking CYP2C9, omeprazole and metronidazole may worsen the effects and raise the risk of side effects when using NSAIDs that are metabolized by this cytochrome, such as celecoxib, diclofenac, ibuprofen, meloxicam, or mefenamic acid [37-41].

Discussion. The most commonly used medications in the world are nonsteroidal anti-inflammatory medicines (NSAIDs), which exhibit high efficacy in analgesic and anti-inflammatory therapy. In addition to their effectiveness, NSAIDs' ubiquitous availability—

especially since some can be obtained over-the-counter—has contributed to their widespread use. Sadly, there are a lot of negative side effects, sometimes severe ones, associated with this class of medications. Because NSAIDs are so widely used, practitioners need up-to-date information on this class of medications in order to make the best therapeutic choices. Every patient needs customized treatment because, when it comes to NSAIDs, the selection of a particular medication is crucial in connection to the kind and severity of pain as well as any possible side effects. One of the major issues related to medicine overuse in Brazil is self-medication without a doctor's or dentist's prescription. Microbial agent resistance and gastrointestinal issues are caused by the improper use of antibiotics and non-steroidal anti-inflammatory medications (NSAIDs). This study aimed to clarify the use trends of NSAIDs and antibiotics in Brazil's adult population [1-7]. In 2023, 400 Brazilian residents with access to the connection completed the questionnaire. Because NSAIDs have so many common indications, the general populace utilizes them extensively. Due to the numerous potential side effects on various organ systems, patient education regarding the use of NSAIDs is a crucial aspect of care that doctors must focus on. Physicians, nurses, and pharmacists must pay particular attention to a patient's medical history and educate them on risks and dosage because these side effects are far more common in individuals with certain comorbidities. Whether it is a short-term or long-term regimen, therapy will be started by the treating physician. In addition to checking for any drug-drug interactions, the pharmacist must confirm the dosage and administration. Additionally, pharmacists should instruct patients on how to minimize side effects and use their NSAIDs as effectively as possible. This is especially important when the patient uses NSAIDs as an over-the-counter medication. In order for the physician to make an informed decision when prescribing NSAID therapy, nurses must also take a thorough drug history that includes OTC NSAID use. In order to modify the patient's regimen as necessary, medical professionals, pharmacists, and clinicians must be aware of the warning signs and symptoms of NSAID toxicity or side effects [13-22]. The "golden mean" of NSAIDs and the knowledge of interactions between certain NSAIDs and the cardioprotective acetylsalicylic acid have been taken into consideration as efforts have been made over the years to develop an algorithm for the selection of NSAIDs based on the risk of developing complications associated with drugs of this group in individual cases. Another attempt to summarize this problem is this paper. There is still the fundamental question of extending the current state of knowledge to include commonly used medications like diclofenac, ketoprofen, meloxicam, and etoricoxib. This is mainly due to the publication of the large PRECISION study, which is limited to only three molecules. This edition offers an algorithm for choosing NSAIDs based on personal gastroenterological and cardiovascular risk in addition to the PRECISION research's findings and a study conducted by an expert group on other NSAIDs available in Poland [27-33]. The results revealed that, regardless of whether these drugs were recommended by physicians or dentists, about 89.5% of the participants had used NSAIDs and 32.2% had used antibiotics. It was shown that symptoms associated with gastrointestinal issues accounted for a significant percentage of the unpleasant effects that the participants reported. The group under study had a high rate of NSAID use, which is consistent with the increased risk of adverse effects from these medications, especially in the gastrointestinal system. Regarding antibiotics, it was found that the population's non-prescription use of these drugs was deemed high, accounting for one-third of all volunteers who took them [34-41].

Conclusion. The exacerbation of NSAID-related adverse responses, especially in the gastrointestinal system, is influenced by the high incidence of NSAID usage among the population under study. When it comes to antibiotics, the issue of using them without a doctor's or dentist's prescription not only exacerbates the global issue of bacterial resistance due to the

indiscriminate use of this class of medication, but it also contributes to the decline of the health system.

To guarantee that every patient receives the right dosage for their particular disease and comorbidities—one that is both high enough for efficacy and as low as feasible to lower the occurrence of side effects—the medical staff should collaborate and communicate. NSAID therapy can provide the greatest possible benefit with the fewest possible drawbacks through cooperative interprofessional collaboration.

NSAIDs are among the most widely used medications in the world. NSAIDs are widely used because of their strong analgesic and anti-inflammatory properties as well as their accessibility, even without a prescription. Unfortunately, the mechanism of action of NSAIDs and the existence of comorbidities, as well as the concurrent use of other medications or dietary supplements, are linked to a rather significant risk of various adverse effects. As a result, selecting the right medication for each patient appears to be crucial. This group of drugs offers a variety of options with slightly different mechanisms of action, allowing one to choose a prescription based on the likelihood of complications.

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