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EFFECT OF NON-STEROID ANTI-INFLAMMATORY DRUGS IN THE TREATMENT OF RHEUMATOID ARTHRITIS

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Relevance: Non-steroidal anti-inflammatory drugs are among the most popular and widespread drugs. NSAIDs are the main means of symptomatic therapy for rheumatic diseases, used to improve the quality of life of patients. With such a wide prevalence of NSAIDs, drugs can have quite a few side effects.

Keywords: rheumatic diseases, polyarthritis, remission, autoimmune inflammation, multifactorial pathogenesis.

Introduction. Rheumatoid arthritis (RA) is a common autoimmune disease, affecting up to 0.5–1.0% of the world's adult population. The main manifestation of RA is chronic destructive polyarthritis, accompanied by severe pain, progressive dysfunction of the musculoskeletal system and a significant deterioration in the quality of life. The systemic inflammatory response characteristic of this disease leads to the development of visceral pathology, increasing the risk of death [1–3]. Modern treatment of RA, in accordance with the generally accepted "Treat to Target" strategy, is aimed at the fastest and most complete reduction in disease activity and induction of remission. Pathogenetic therapy of RA is based on the use of pharmacological agents that suppress autoimmune inflammation: synthetic basic anti-inflammatory drugs - sDMARDs (methotrexate, leflunomide, sulfasalazine, etc.), genetically engineered biological drugs (GEBPs), as well as targeted sDMARDs (Janus kinase inhibitors). This

strategy has achieved significant success: in the last decade, the number of patients with severe, complicated forms of RA has significantly decreased, and there is a trend towards a decrease in mortality from this disease [1–3]. However, despite the use of modern pathogenetic drugs, many patients with RA, even against the background of low inflammatory activity, retain moderate or severe pain, increased fatigue, poor health and other symptoms that worsen the quality of life. This is due to the complex, multifactorial pathogenesis of this disease, which includes, in addition to inflammatory tissue damage caused by autoimmune aggression, degenerative processes (fibrosis and neoangiogenesis), secondary osteoarthritis (OA) and enthesopathies, biomechanical disorders, damage to the somatosensory system (mono- and polyneuritis), neuroplastic changes (central sensitization), as well as mental and psychological problems (depression, anxiety and catastrophizing) [4–6]. Chronic pain in RA determines the need to use symptomatic pain medications. The most rational seems to be the use of nonsteroidal anti-inflammatory drugs (NSAIDs), the analgesic effect of which is associated with the suppression of the activation of peripheral pain receptors caused by inflammatory mediators and proinflammatory cytokines [1]. In real clinical practice, NSAIDs are the most popular class of analgesics used in the complex treatment of RA, which is confirmed by a number of epidemiological studies. A. Steffen et al. [7] analyzed pharmacotherapy in 54,896 RA patients included in the German national registry: 64% of patients received NSAIDs during the first year of treatment and approximately 40% during the third year. In a cohort of 3225 RA patients observed by Australian scientists, 43.6% of patients regularly used NSAIDs, and 32.6% used opioids; while the majority (75,1%) of them took sDMARDs and biologically active drugs (55.9%) [8].

Similar data were presented by R. Kimsey et al. [9], who studied the range of medications prescribed to 16,680 patients with newly diagnosed RA. It was found that 42.2% of patients received NSAIDs, and 35.3% of patients received opioids. The effectiveness of NSAIDs for pain control in RA has been assessed in numerous randomized controlled trials (RCTs) [10]. A number of studies have demonstrated the ability of NSAIDs to improve the results of complex treatment of RA using biological medications [11]. However, many aspects of the use of NSAIDs in this disease have not been sufficiently studied. First of all, this concerns assessing the effect of NSAIDs on the activity of RA, as well as determining the risk of class-specific complications from the cardiovascular system (CVS), gastrointestinal tract (GIT) and kidneys characteristic of this class of analgesics.

Purpose of the work: To analyze the features of side effects of NSAID drugs in patients with rheumatoid arthritis.

Materials and methods of research: The objects of the study were patients with rheumatoid arthritis (55 people) who took part in the observation. Methods of mathematical statistics were used to process the results.

Study Findings: NSAIDs have a general spectrum of clinical toxicity, although the incidence of specific side effects varies depending on the compound. The danger of individual NSAIDs is related to their pharmacological characteristics, such as bioavailability and half-life, as well as their ability to inhibit COX-1 and COX-2. The survey revealed patients (28%) who took NSAID drugs suffered from diseases of the gastrointestinal tract. Among them, 2/3 were diagnosed with chronic gastritis, and the rest had gastric or duodenal ulcers. Attention is paid to renal, hepatic and cardiovascular side effects, which are especially important in patients with rheumatic diseases due to the age of the patients and the use of drugs. Sodium retention occurs in 20% of patients receiving NSAIDs. Decreased sodium excretion in patients receiving NSAIDs may lead to weight gain and peripheral edema. In 4% of patients receiving NSAIDs, hyporeninemic hypoaldosteronism developed, which is manifested by type IV renal tubular acidosis and hyperkalemia. The degree of hyperkalemia is usually mild; however, patients with renal impairment or patients who may be prone to hyperkalemia for other reasons

(particularly patients with diabetes mellitus and patients taking angiotensin-converting enzyme inhibitors or potassium-sparing diuretics) may be at greater risk. NSAIDs may cause changes in blood pressure, with an average increase in mean arterial pressure of 5 to 10 mmHg.

Conclusion: In summary, NSAID use may increase the risk of initiation of antihypertensive therapy in older patients, with the magnitude of the increased risk being proportional to the dose of NSAID. Correction of risk factors reduces diseases caused by complications during treatment.

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