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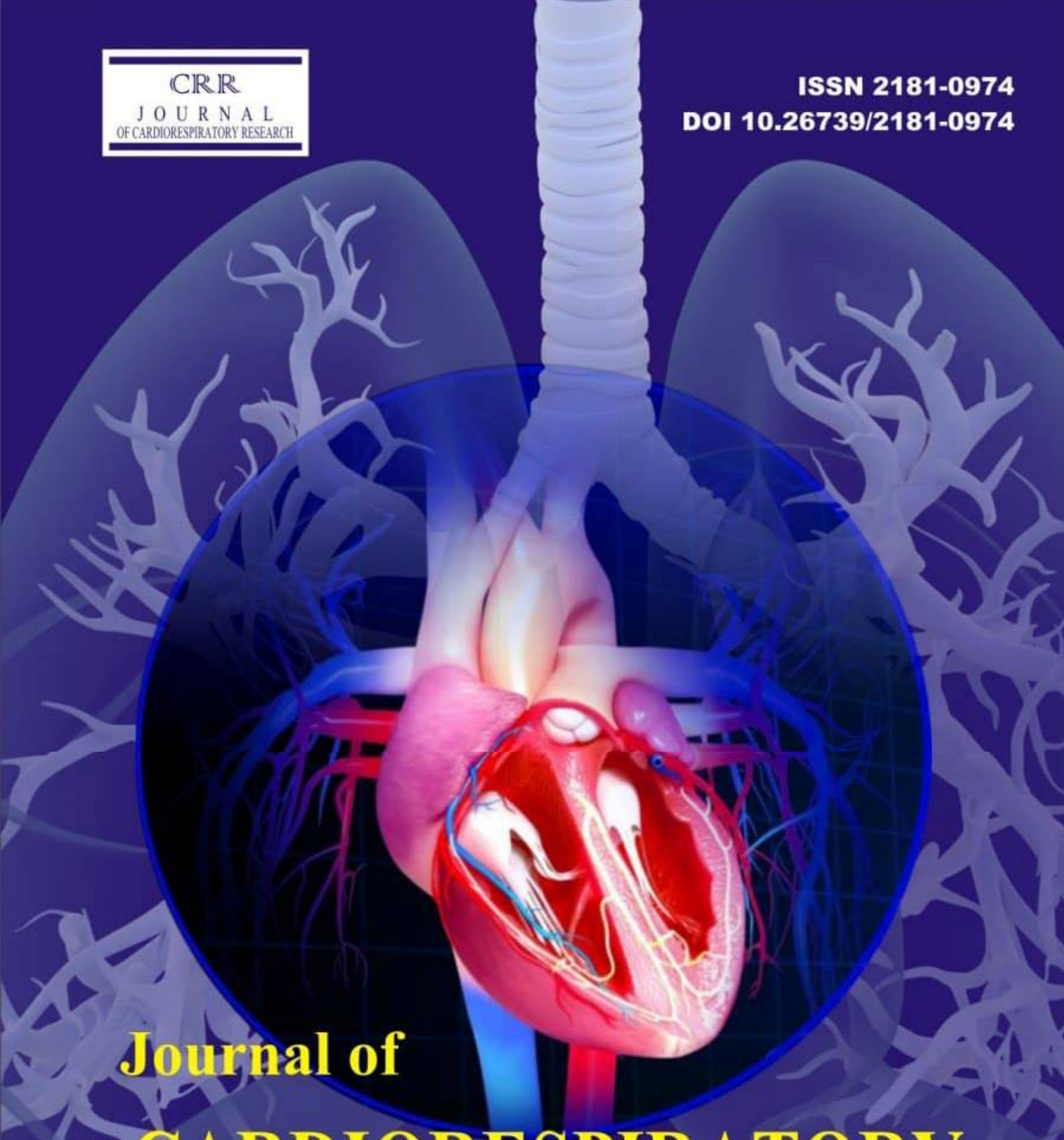

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ДОКЛИНИЧЕСКИЙ ПРОЦЕСС РАЗРАБОТКИ ЛЕКАРСТВ МЕХАНИЗМОВ РЕВМАТОИДНОГО АРТРИТА ДЛЯ ИЗУЧЕНИЯ МОРФОЛОГИЧЕСКИХ ИЗМЕНЕНИЙ СТРУКТУРЫ КЛЕТОК И СОСУДОВ СЕРДЦА У ЭКСПЕРИМЕНТАЛЬНЫХ ЖИВОТНЫХ

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АННОТАЦИЯ

Цель исследования: изучить доклинический процесс разработки лекарств механизмов ревматоидного артрита для изучения морфологических изменений структуры клеток и сосудов сердца у экспериментальных животных.

Материалы и методы: были применены для ревматоидного артрита полный адъювант Фрейнда у 100 белых рандомизированных крыс в возрасте от 18 до 24 месяцев, находящихся в стационарных условиях вивария Бухарского государственного медицинского института. Нами клинически были проведены ведение пациентов, где изучали гетерогенность в отношении эндогенных циркадианных ритмов, болезненных состояний, подтипов и продолжительности, а также паттерна аутоантител, цитокинов и инфильтрирующих иммунных клеток.

Результаты и обсуждение: при исследование доклинического процесса разработки лекарств механизмов ревматоидного артрита для изучения морфологических изменений структуры клеток и сосудов сердца у экспериментальных животных, важно отметить, что у животных естественным образом не развиваются аутоиммунные заболевания, такие как РА, что является неотъемлемым ограничением этих моделей артрита. Полученные результаты показали что, современные терапевтические подходы с использованием современных биологических препаратов очень успешны и эффективны у большинства пациентов с РА, включая пациентов с тяжелым прогрессированием заболевания.

Выводы: было определено наличие влияния моделированного ревматоидного артрита на сердце, который свидетельствует о том, что данная патология имеет достаточный уровень риска для организма;

Ключевые слова: ревматоидный артрит, сердечно-сосудистый риск, профилактика

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PRECLINICAL DRUG DEVELOPMENT PROCESS OF RHEUMATOID ARTHRITIS MECHANISMS TO STUDY MORPHOLOGICAL CHANGES IN THE CELL AND VASCULAR STRUCTURE OF THE HEART IN EXPERIMENTAL ANIMALS

ANNOTATION

Purpose of the study: to study the preclinical process of drug development of rheumatoid arthritis mechanisms to study morphological changes in the structure of cells and vessels of the heart in experimental animals

Materials and Methods. Complete Freund's adjuvant for rheumatoid arthritis was applied to 100 white randomized rats aged between 18 and 24 months in the inpatient vivarium of the Bukhara State Medical Institute.

Results and discussion: While investigating the preclinical drug development process of rheumatoid arthritis mechanisms to study morphological changes in the cell structure and vascular structure of the heart in experimental animals, it is important to note that animals do not naturally develop autoimmune diseases such as RA, which is an inherent limitation of these arthritis models. The results have shown that modern therapeutic approaches using modern biological agents are very successful and effective in most patients with RA, including those with severe disease progression.

Conclusions: The presence of the influence of simulated rheumatoid arthritis on the heart was determined which testifies to the fact that this pathology has a sufficient level of risk for the organism.

Keywords: rheumatoid arthritis, cardiovascular risk, prevention.

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EKPEREMENTAL HAYVONLARDA YURAK HUJAYRALARI VA QON TOMIRLARI TUZILISHIDAGI MORFOLOGIK O‘ZGARISHLARNI O‘RGANISH UCHUN KLINIKADAN OLDINGI JARAYONDA REVMATOID ARTRIT MEXANIZMLARNI ISHLAB CHIQISH

ANNOTASIYA

Tadqiqot maqsadi: ekperemental hayvonlarda yurak hujayralari va qon tomirlari tuzilishidagi morfologik o‘zgarishlarni o‘rganish uchun klinikadan oldingi jarayonda revmatoid artrit mexanizmlarni ishlab chiqish.

Materiallar va usullar. Revmatoid artrit uchun to‘liq Freund 100 ta oq randomizasiyalangan kalamushlar Buxoro davlat tibbiyot institutining vivarium stasionar sharoitida 18 oydan 24 oygacha tekshirilgan.

Natijalar va munorazalar: Eksperemental hayvonlarda yurak hujayralari va qon tomrlari tuzilishidagi morfologik o‘zgarishlarini o‘rganish uchun revmatoid artrit mexanizmlarini dori darmonlarni ishlab chiqishning klinikadan oldingi jarayonini o‘rganishdan test usulida autoimmun kasalliklar rivojlanmaydi. Revmatoid artrit kabi artrit modellarning ajralmas cheklovi hisoblanib, natijalar shuni ko‘rsatdiki zamonaviy biologik preparatlardan foydalangan holda zamonaviy terapevtik yondashuvlar RA bilan og‘rigan bemorlarning ko‘pchiligida, shu jumladan og‘ir rivojlangan bemorlarda juda muvaffaqiyatli va samarali hisoblanadi.

Xulosa. Modulyasiyalangan revmatoid artritning yurakka ta'siri mavjudligi aniqlandi va bu ushbu patologiyaning organizm uchun yetarli darajada xavf mavjudligini ko‘rsatishga imkon yaratdi.

Kalit so‘zlar: Revmatoid artrit, yurak-qon tomir xavfi, oldini olish.

Introduction. Rheumatoid arthritis (RA) is a chronic, inflammatory, and systemic autoimmune disease affecting the connective tissue and primarily the joints. Untreated, RA eventually leads to progressive degeneration of cartilage and bone [1, 7]. The etiology of the pathogenesis of RA is unknown, suggesting that its clinical manifestations are heterogeneous and associated with autoantibodies directed against modified native epitopes. Although many RA models already exist for preclinical studies, many current arthritis model systems have limited predictive value because they are either based on animals of phylogenetically distant origin or suffer from overly simplistic in vitro culturing conditions. These limitations pose serious problems for preclinical studies and, therefore, for clinical applications. Here we summarize the most commonly used in vitro RA models and discuss their experimental feasibility and physiological proximity to human RA pathophysiology in order to highlight new avenues of RA research involving humans to expand our knowledge of human pathophysiology and develop effective targeted therapies. Here we summarize the most commonly used in vitro RA models and discuss their experimental feasibility and physiological proximity to human RA pathophysiology to highlight new avenues of research into RA with human involvement to expand our knowledge of human pathophysiology and develop effective targeted therapies [3,9,10].

Research objective: to study the preclinical process of drug development of rheumatoid arthritis mechanisms to study morphological changes in the structure of cells and vessels of the heart in experimental animals.

Materials and Methods: We investigated during 2020-2022 60 white randomized rats aged 18 to 24 months in the in-patient vivarium of the Bukhara State Medical Institute. 60 white randomized rats aged from 18 to 24 months in inpatient conditions of Bukhara State Medical Institute vivarium. Complete Freund's adjuvant was used to simulate rheumatoid arthritis. All animal experiments were performed in compliance with the international principles of the European Convention for the Protection of Vertebrate Animals used for experimental and other scientific purposes, as well as in accordance with the "Rules for work with experimental animals". All laboratory animals were divided into 4 groups:

Group 1- animals with experimentally induced rheumatoid arthritis who did not receive treatment;

Group 2 - animals with experimentally induced rheumatoid arthritis treated with GCS (glucocorticoid model) for 4 weeks;

Group 3 - animals with experimentally induced rheumatoid arthritis treated with cytostatics for 4 weeks;

Group 4 - intact animals that will be kept under standard vivarium conditions. The subject of the study was histological material obtained from different parts of the heart of the experimental animals.

The results of the study, showed that animal models are an integral part of the preclinical drug development process and are used to study the pathophysiological mechanisms of RA. Although they are extremely useful for testing new approaches to intervention in many cases, concerns have been raised about the low success rates of clinical development of investigational drugs. It is important to note that animals do not naturally develop autoimmune diseases such as RA, which is an inherent limitation of these arthritis models.(Table 1). Instead, animal models can be used to study some specific pathophysiological aspects of human disease, such as the destructive pathways involved in articular cartilage and bone erosion. To this end, arthritis can be chemically induced in these animals with soluble agents (e.g., the type II collagen-induced arthritis model) or develop spontaneously after genetic manipulation (e.g., the transgenic human TNF model).Table 1). Although most of these models demonstrate hallmarks of human rheumatoid arthritis, such as inflammatory cell infiltrate, synovial hyperplasia, pannus formation, cartilage destruction, and bone erosions, they also demonstrate specific limitations, such as development of self-limiting arthritis, development of only arthritis. in susceptible rodent strains and pathophysiology that does not replicate endogenous tolerance disorder and exclude systemic components of the disease [2,6,11]. Mutations used in genetically engineered models of arthritis have not been identified in human rheumatoid arthritis [3,6]. When comparing the transcriptional programs of mice and humans, overlapping but markedly different gene expression patterns were observed. Consequently, therapeutic approaches, such as the use of biologics highly specific to human target proteins, cannot be proven using nonhumanized rodent models. Finally, mice and humans differ in their locomotion, longevity, evolutionary pressures, ecological niches, circadian rhythms, weight load, and ratio of leukocyte populations in the blood. Thus, none of the animal models is able to fully reproduce the pathogenesis of RA in humans, which explains the problems observed in clinical interpretation. Current treatment guidelines recommend early and rigorous treatment to achieve low disease activity or remission as soon as possible. Rheumatoid arthritis is currently treated with a wide range of therapeutic agents, ranging from steroidal/nonsteroidal anti-inflammatory drugs (NSAIDs), glucocorticoids (GCs) and disease-modifying antirheumatic drugs such as methotrexate, biological and biologic TNF inhibitors or IL-6 inhibitors), as well as targeted synthetic inhibitors that target specific immune cells, cytokines or proinflammatory pathways [1,8]. Modern

therapeutic approaches using modern biological drugs have been proven to be very successful and effective in the majority of patients with RA, including those with severe disease progression. Despite significant progress in the treatment of RA, there remains an acute unmet medical need, as not all patients achieve sustained clinical remission (less than half of patients with RA) and about 25% still suffer from moderate or even high disease activity [2,4]. Identifying patients with RA (I) refractory to available treatments among patients with RA who are under-treated or not adhering to treatment, (II) identifying objective biomarkers of disease states (e.g., early RA versus established RA) and/or (III) "refractory" states and finally (IV) for treatment response states is still the greatest unmet need in RA. The lack of therapeutic efficacy in patients with true refractory conditions may be due to the nature of the universal approach of standardized therapeutic regimens. Thus, the clinical management of patients often ignores their heterogeneity with respect to endogenous circadian rhythms, disease states, subtypes and duration, as well as the pattern of autoantibodies, cytokines and infiltrating immune cells. The identification of objective biomarkers to determine disease subtypes and response to treatment will be necessary to provide a "precision" individualized treatment strategy for each individual patient, expanding our repertoire in the fight against this potentially devastating disease.

Conclusions: Thus, preclinical models are needed to improve our understanding of pathological mechanisms and to develop and test new therapeutic approaches to meet this unmet medical need. This includes exploration of human-specific alternatives to identify objective biomarkers to determine disease subtypes and response to treatment, as well as novel targets to control immune cell function involved in RA pathogenesis.

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