

RISK OF DEVELOPMENT AND MECHANISMS OF HYPERTENSION FORMATION IN RHEUMATOID ARTHRITIS.

Sultanbayev Laziz Rustamovich

Chirchik branch of Tashkent State

Medical University

Uzbekistan, Tashkent region, Chirchik city

sultanbaev2012@gmail.com

Tukhtaeva Nigora Khasanovna

DSc, Associate Professor of the department

“Propaedeutics of Internal Diseases No. 2”,

Tashkent State Medical University,

Tashkent, Uzbekistan

nigora.to'xtayeva@tma.uz

<https://orcid.org/0009-0006-8181-041X>

Abstract: The paper examines the relationship between rheumatoid arthritis (RA) and the development of hypertension (HTN) as a comorbid condition. The author analyzes the mechanisms by which systemic inflammation influences the development of endothelial dysfunction and vascular remodeling through pro-inflammatory cytokines (TNF- α , IL-6). Particular attention is paid to the role of drug therapy (NSAIDs and glucocorticoids) and traditional risk factors in increasing blood pressure in this patient group. The study substantiates the need for comprehensive monitoring and the integration of cardiac control into management protocols for RA patients to reduce the risk of fatal cardiovascular complications.

Keywords: Rheumatoid arthritis, hypertension, systemic inflammation, pro-inflammatory cytokines, endothelial dysfunction, cardiovascular risk, comorbidity, non-steroidal anti-inflammatory drugs.

Introduction

Rheumatoid arthritis (RA) is a chronic systemic inflammatory disease that affects not only the joints but also various organs and systems of the body [1,2,7]. One of the significant comorbid conditions in patients with RA is arterial hypertension (AH), which substantially increases the risk of cardiovascular complications [3,4,6].

Chronic inflammation in RA plays a key role in the development of endothelial dysfunction, contributing to elevated blood pressure. Proinflammatory cytokines such as tumor necrosis factor- α (TNF- α) and interleukin-6 (IL-6) are involved in the disruption of vascular tone and vascular wall remodeling. In addition, long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs) and glucocorticosteroids, commonly used in RA treatment, may contribute to increased blood pressure [4,5].

Patients with RA have a higher risk of developing hypertension compared to the general population, which is associated both with the activity of the inflammatory process and with traditional risk factors such as obesity, physical inactivity, and metabolic disorders. The presence of hypertension in these patients significantly increases the likelihood of developing coronary heart disease, stroke, and other cardiovascular diseases [4,6].

Objective of the study – to investigate the pathways of development of arterial hypertension in rheumatoid arthritis.

Materials and methods. A total of 78 patients with rheumatoid arthritis were examined, of whom 34 (44%) were diagnosed with arterial hypertension using 24-hour ambulatory blood pressure monitoring (ABPM). The mean age of the patients was 42.7 ± 8.6 years; among them, 22 (65%) were men and 12 (35%) were women.

Results. All 34 patients underwent, in addition to ABPM, blood tests including a coagulation panel and lipid profile. According to ABPM data, systolic blood pressure (SBP) ranged from 137 to 178 mmHg, while diastolic blood pressure (DBP) ranged from 84 to 112 mmHg. In the lipid profile analysis, particular attention was given to low-density lipoprotein cholesterol (LDL-C), which averaged 2.7 mmol/l (range: 1.8-3.2) in this group of patients. Coagulation studies revealed that in 27 patients, fibrinogen levels were elevated, activated partial thromboplastin time (APTT) was shortened, and prothrombin index (PTI) was increased, indicating a higher risk of thrombosis. Other parameters (International Normalized Ratio, thrombin time, D-dimer) were within normal limits. In the remaining 7 patients, all coagulation parameters were within normal ranges.

Conclusion. Based on the results of 24-hour ambulatory blood pressure monitoring (ABPM) and laboratory studies, rheumatoid arthritis is associated with an increased risk of developing arterial hypertension. This allows RA to be considered an independent cardiovascular risk factor. These findings necessitate a comprehensive approach to patient management, including regular blood pressure monitoring, correction of risk factors, and optimization of anti-inflammatory therapy.

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