Pro Index | Journal of Science in Medicine and Life
Volume: 3 Issue: 10 Year: 2025
ISSN: 2992-9202 | http://journals.proindex.uz

Metabolic Changes in Acute Rheumatic Fever

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Key points: Acute rheumatic fever (ARF), metabolic changes.

Introduction

Rheumatic fever, or acute rheumatic fever, is an inflammatory process that occurs as a complication of a streptococcal infection. This disease of the joints, mucous membranes, nervous system, or heart can become chronic and lead to heart defects and heart failure. Acute rheumatic fever (ARF) is a systemic inflammatory disease of connective tissue that occurs after streptococcal infection and is characterized by damage to the heart, joints, and nervous system. Despite advances in antibiotic prophylaxis, ARF remains a significant problem in developing countries. In recent years, particular attention has been paid to the study of metabolic changes in ARF, as they reflect the relationship between inflammation, energy metabolism, and disease outcome [1,6].

Etiopathogenesis of acute rheumatic fever

ARF is caused by group A β-hemolytic streptococcus. The immune system forms cross-antibodies to streptococcal antigens and the body's own tissues (the phenomenon of molecular mimicry), which leads to autoimmune inflammation. Cytokines (IL-1, IL-6, TNF-α) released during macrophage activation cause a systemic inflammatory response that affects metabolic processes—increases catabolism, increases glucose levels, and changes the lipid profile [2,5].

Metabolic disturbances in systemic inflammation

Systemic inflammation in ARF is accompanied by alterations in energy metabolism. Gluconeogenesis increases, lipolysis intensifies, leading to hyperglycemia and hyperlipidemia.

Cytokines act on the liver, stimulating the synthesis of acute-phase proteins (CRP, fibrinogen), and also increase the consumption of amino acids for the synthesis of immune proteins [3]. Such changes are aimed at maintaining the energy supply of the immune response, but with prolonged inflammation they become pathological.

Changes in carbohydrate, lipid and protein metabolism in ARF

In ARF, signs of stress-induced hyperglycemia are observed, caused by the activation of cortisol and catecholamines. Triglyceride levels increase and high-density lipoprotein levels decrease, forming an atherogenic profile [4]. Protein metabolism is characterized by muscle protein catabolism and increased acute-phase reactant protein concentrations. Disturbances in protein metabolism exacerbate inflammation, as breakdown products activate macrophages and increase cytokine production.

The influence of cytokines and oxidative stress

Proinflammatory cytokines (TNF-α, IL-6) play a central role in altering metabolism. They stimulate the expression of glucose transport proteins, increasing energy consumption by inflamed tissues. Simultaneously, lipid peroxidation processes are activated, generating reactive oxygen species that

damage cell membranes and mitochondria [5]. Oxidative stress reduces the efficiency of oxidative phosphorylation, which leads to energy deficiency in myocardial and endothelial cells.

Modern diagnostic approaches and therapeutic corrections

Modern diagnostics of metabolic disorders in ARF include determination of glucose, insulin, lipid profile, oxidative stress markers, and cytokines. Metabolomic analysis methods are used to identify specific biomarkers of inflammation and energy imbalance [6].

Treatment of ARF should be comprehensive: in addition to anti-inflammatory therapy (NSAIDs, glucocorticosteroids), correction of metabolic disorders is recommended using antioxidants (vitamin E, coenzyme Q10), carnitine, as well as a balanced diet aimed at maintaining energy balance [7].

Conclusion

Metabolic changes in acute rheumatic fever reflect the systemic nature of inflammation and are closely linked to immune and vascular disorders. The identified shifts in carbohydrate, lipid, and protein metabolism have not only adaptive but also prognostic significance. A better understanding of metabolic mechanisms will allow us to optimize the treatment of ARF, improve outcomes, and reduce the incidence of cardiac complications.

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