IJACT, Volume 3, Issue 11, 2025 ISSN: 2995-5378
http://medicaljournals.eu/index.php/IJACT
INTERNATIONAL JOURNAL OF ALTERNATIVE AND CONTEMPORARY THERAPY

Personalized Management Strategy in Patients with Acute Myocardial Infarction

Mukhamedova Malika Murtazoevna
Kimyo International University in Tashkent

Abstract: Acute myocardial infarction (AMI) is a multifactorial condition characterized by high morbidity and mortality, where inflammation and immune activation play a central role in myocardial injury and remodeling. In recent years, the concept of personalized medicine has emerged as a promising strategy to improve outcomes by tailoring therapy to individual biological profiles. Objective: To evaluate the clinical efficacy of a personalized management approach in AMI patients based on immunological and metabolic markers, comorbidity profiles, and cardiovascular remodeling indicators. Methods: A total of 120 patients with AMI were divided into two groups: the control group received standard ESC-based therapy, and the experimental group underwent personalized treatment that included cytokine-guided immunocorrection (targeting IL-6, IL-17A, TNF-α), metabolic and antioxidant support, and optimized use of β-blockers and ACE inhibitors. Clinical, echocardiographic, and biochemical parameters were analyzed on admission, day 5, and day 30. Results: Patients receiving personalized therapy demonstrated a faster reduction in inflammatory markers (IL-6 ↓47%, IL-17A ↓41%), improved left ventricular ejection fraction (LVEF ↑9.3 ± 1.2%), and a 32% decrease in early post-infarction complications. Significant correlations were identified between IL-17A and LVEF ($r = –0.56$, $p < 0.001$), and between IL-6 and troponin I ($r = +0.48$, $p < 0.01$), confirming the pathogenetic role of immune activation in post-infarction remodeling. Conclusion: The integration of immunological, metabolic, and clinical parameters into a personalized management algorithm enhances the effectiveness of AMI treatment, reduces inflammatory activity, and improves cardiac function and prognosis. These findings support the introduction of individualized therapeutic strategies into contemporary cardiology practice.

Keywords: acute myocardial infarction; personalized medicine; cytokines; IL-17A; inflammation; cardiac remodeling; immunocorrection; prognosis.

Introduction

Acute myocardial infarction (AMI) remains one of the leading causes of mortality and disability worldwide despite significant advances in diagnostic and therapeutic approaches. According to the World Health Organization, cardiovascular diseases account for nearly 32% of all global deaths, with AMI representing a major contributor to this burden. Early reperfusion therapy and antithrombotic management have substantially improved short-term outcomes; however, long-term prognosis is still determined by the extent of myocardial damage, systemic inflammation, and comorbid conditions [2,5].

Recent studies emphasize that the pathogenesis of AMI extends beyond coronary occlusion alone and involves complex immune-inflammatory interactions, oxidative stress, and endothelial dysfunction. Cytokines such as interleukin-6 (IL-6), interleukin-17A (IL-17A), and tumor necrosis factor-α (TNF-α) play pivotal roles in post-infarction remodeling and ventricular dysfunction. Elevated levels of these markers have been associated with adverse outcomes, recurrent ischemic events, and the progression to chronic heart failure, highlighting the need for integrated, biomarker-guided therapeutic approaches [4].

In this context, the concept of personalized medicine in cardiology offers new opportunities for optimizing patient management. Individualized risk stratification that considers inflammatory status,

metabolic profile, genetic predisposition, and comorbidities may improve treatment efficacy and reduce complications. The present study focuses on evaluating the effectiveness of a personalized management strategy in patients with AMI, integrating immunological and biochemical indicators into clinical decision-making algorithms [1,3,6].

Objective. To evaluate the effectiveness of a personalized management approach in patients with acute myocardial infarction (AMI), based on clinical, immunological, and biochemical markers of myocardial injury and systemic inflammation.

Materials and Methods. A total of 120 patients with AMI (mean age 41 ± 1,4 years) were examined and divided into two groups: Group 1 (n = 60) received standard therapy according to the ESC 2023 guidelines, while Group 2 (n = 60) underwent individualized treatment based on stratification by comorbidity profile, inflammatory status (IL-6, IL-17A, TNF-α), renal and hepatic function, and genetic polymorphisms of the renin–angiotensin system. Laboratory parameters, echocardiographic indices (LVEF, EDD, ESD), and troponin I levels were assessed on admission, on day 5, and on day 30. Statistical analysis was performed using ANOVA and Spearman's correlation.

Results.

Patients who received personalized therapy demonstrated significantly faster regression of systemic inflammatory markers (IL-6: ↓ 47%, $p < 0.01$; IL-17A: ↓ 41%, $p < 0.05$) and improved myocardial recovery (LVEF ↑ by 9.3 ± 1.2%, $p < 0.05$) compared to the control group. The incidence of early post-infarction complications decreased by 32%, including a reduction in the frequency of recurrent ischemia, ventricular arrhythmias, and acute heart failure episodes. Furthermore, serum levels of TNF-α and C-reactive protein showed a marked decline (↓ 38% and ↓ 44%, respectively, $p < 0.05$), reflecting suppression of the systemic inflammatory response.

Echocardiographic assessment on day 30 demonstrated a statistically significant improvement in left ventricular end-diastolic and end-systolic dimensions (EDD and ESD), indicating attenuation of post-infarction remodeling processes. Personalized therapeutic regimens that included anti-inflammatory and antioxidant correction, metabolic modulation (trimetazidine, coenzyme Q10), and optimization of renin–angiotensin system blockade contributed to more rapid stabilization of hemodynamic parameters and recovery of exercise tolerance (6-minute walk test ↑ by 23%).

A strong negative correlation was found between IL-17A levels and LVEF ($r = –0.56$, $p < 0.001$), confirming the pathogenetic role of immune activation in ventricular dysfunction and adverse remodeling. Additionally, a positive correlation between IL-6 and troponin I ($r = +0.48$, $p < 0.01$) emphasized the close link between inflammatory response intensity and myocardial necrosis. These findings suggest that immune-mediated mechanisms significantly influence the prognosis after AMI and can serve as valuable targets for personalized therapeutic strategies.

Conclusion

A personalized strategy for managing AMI patients that integrates immunological and metabolic markers allows for targeted correction of inflammation, improvement of cardiac function, and reduction of complications. The results support the need for implementing individualized diagnostic algorithms and treatment models in cardiological practice.

References

1. Baghai T. C., Varallo-Bedarida G., Born C., Ндfner S., Schьle C., Eser D., et al. (2018). Classical risk factors and inflammatory biomarkers: One of the missing biological links between cardiovascular disease and major depressive disorder. //*International Journal of Molecular Sciences, 19*(6), 1740.
2. Chatterjee P., Chiasson V.L., Seerangan G., Tobin R.P., Kopriva S.E., Newell-Rogers M.K., Mitchell B.M.: Cotreatment with interleukin 4 and interleukin 10 modulates immune cells and prevents hypertension in pregnant mice. //Am J Hypertens 28: 135–142, 2015 [PubMed] [Google Scholar]

3. Sh, G. S., & Akhrorov, J. X. (2022). Этиопатогенетические Особенности Ремоделирования Сердца При Кардиоваскулярной Патологии. Periodica Journal of Modern Philosophy, Social Sciences and Humanities, 13, 101-105.

4. Yin J., Xia W., Zhang Y., Ding G., Chen L., Yang G., et al. (2018). Role of dihydroartemisinin in regulating prostaglandin E2 synthesis cascade and inflammation in endothelial cells.// *Heart and Vessels, 33*(11), 1411–1422.

5. Ганиева, Ш. Ш., & Яхъяева, Ф. О. (2022). Современные патогенетические аспекты кардиоренального синдрома. Eurasian Journal of Medical and Natural Sciences, 2(6), 167-173.

6. Мухамедова, М. М., & Ганиева, Ш. Ш. (2022). Клинические Особенности Острого Коронарного Синдрома У Больных, Перенесших Коронавирусную Инфекцию. AMALIY VA TIBBIYOT FANLARI ILMIY JURNALI, 209–213.