

## Laboratory Picture Of The Cardiotoxic Effect Of Immunodepressants Used In The Pathogenetic Therapy Of Patients With Chronic Glomerulonephritis

Muminov Davron Kadirovich

Professor, Department of Internal Medicine, Nephrology and Hemodialysis, Tashkent State Medical University, Uzbekistan. Tashkent State Medical University, Uzbekistan

Jalilov Shukhrat Khamidovich

PhD student, Department of Internal Medicine Nephrology and Hemodialysis, Tashkent State Medical University. Nephrologist, Republican Specialized Scientific and Practical Medical Center of Nephrology and Kidney Transplantation, Uzbekistan

Received: 30 Mar 2026 | Received Revised Version: 14 Apr 2026 | Accepted: 28 Apr 2026 | Published: 22 May 2026

Volume 08 Issue 05 2026 | Crossref DOI: 10.37547/tajmspr/Volume08Issue05-20

### Abstract

*The article presents a picture of changes in the side effects of these drugs on the cardiovascular system in 79 patients with glomerulonephritis who were prescribed immunosuppressive therapy of various compositions, using biochemical laboratory analyses as an example of cardiospecific indicators. At the same time, the results of studies on the specifics of the negative effects of immunosuppressive agents on the myocardium in the study groups where hormones and cytostatics were recommended were analyzed. Based on the results of the study, conclusions were drawn and relevant recommendations were formulated.*

Keywords. Aspartate aminotransferase, glomerulonephritis, immunosuppressive therapy, cardiotoxic, creatine kinase, myoglobin, troponin.

© 2026: Muminov Davron Kadirovich & Jalilov Shukhrat Khamidovich. This work is licensed under a Creative Commons Attribution 4.0 International License (CC BY 4.0). The authors retain copyright and allow others to share, adapt, or redistribute the work with proper attribution.

**Cite This Article:** Muminov Davron Kadirovich, & Jalilov Shukhrat Khamidovich. (2026). Laboratory Picture Of The Cardiotoxic Effect Of Immunodepressants Used In The Pathogenetic Therapy Of Patients With Chronic Glomerulonephritis. The American Journal of Medical Sciences and Pharmaceutical Research, 8(05), 106–109. <https://doi.org/10.37547/tajmspr/Volume08Issue05-20>

### 1. Introduction

It is well known that cardiovascular diseases (CVD) are the primary cause of morbidity and mortality among people worldwide, accounting for approximately 70% on average [3, 4]. In recent years, not only traditional cardiovascular diseases but also cardiovascular consequences arising from other causes have become the primary factor in ensuring the share of the aforementioned indicator. That is, although targeted and immunosuppressive therapy (IST), which are widely used in the treatment of autoimmune and

oncological diseases, have ensured great success in the field and serve to significantly improve the survival and quality of life of patients, on the other hand, these drugs lead to an increase in the risk of cardiovascular events and complications of cardiovascular diseases developing on their basis [14]. This, in turn, indicates the need to significantly increase attention to this problem and develop cardioprotective treatment measures from those areas [7]. This is because conservative treatment measures conducted today in the fields of nephrology, rheumatology, and

oncology are primarily based on immunosuppressive therapy, consisting of such areas as hormone therapy, chemotherapy, targeted (molecular-based) therapy, and immunotherapy (biotherapy). Today, cytostatics (cytotoxic and cytostatic), monoclonal antibody preparations, and even glucocorticosteroids (GCS), which are recognized in some literature as having "no side effects" [1, 5], have negative consequences for the cardiovascular system [2, 11, 13]. From this perspective, in our research, we studied the clinical and functional features of changes in the cardiovascular system caused by IST in patients with chronic glomerulonephritis.

**Purpose of the research**

Evaluation of the cardiotoxic properties of immunosuppressive drugs of various compositions in patients with glomerulonephritis by analyzing the pattern of changes in cardiospecific biochemical laboratory parameters.

**Material and Methods**

For the study, 102 patients with various forms of chronic glomerulonephritis were selected who received inpatient treatment in the departments of autoimmune diseases and inflammatory kidney diseases of the Republican Specialized Scientific and Practical Medical Center for

Nephrology and Kidney Transplantation and subsequently were under dispensary observation in this institution. Their average age was 31.64±5.26 years, and all of them had been receiving IST for at least more than 3 months. Of these, two study groups were formed, consisting of patients with glomerulonephritis who were prescribed hormones (n-53) and cytostatics (n-26), and a comparative analysis of changes in cardiac-specific biochemical laboratory parameters was conducted in all of them. At the same time, the concentration levels of troponin, creatinine kinase-MB, myoglobin, and aspartate aminotransferase enzymes in the patients' blood serum were determined. It should be noted that 23 patients receiving combined IST (GCS + cytostatic; n-23) were not included in this stage of the study. The obtained results were statistically analyzed and compared between the main study groups.

**Results and Discussion**

At the same time, two study groups were formed for patients receiving immunosuppressive drugs of various compositions. A comparative analysis of changes in cardiac-specific biochemical laboratory parameters was conducted in the study groups consisting of patients with glomerulonephritis who were prescribed hormone (n-53) and cytostatics (n-26).

**Table 1.**

**Characteristics of changes in cardiac-specific biochemical laboratory parameters in patients treated with immunosuppressive drugs of various compositions**

Indicators	Control group (n = 20)	Patients prescribed hormone therapy (n = 53)	Patients prescribed cytostatics (n = 26)	Intergroup statistical analysis
Troponin	20/0	51/2	19/7	$\chi^2=9.260$ (p=0,003)
Creatine kinase-MB (CK-MB) (0-5 ng/mL)	2,9±0,59	3,62±0,54	5,37±0,63**	^ p<0,05
Myoglobin (0-58 ng/mL)	33,16±5,67	38,6±6,28	59,1±7,87**	^ p<0,05
AST (Aspartate Aminotransferase)	23,5±4,06	41,6±5,91*	57,8±5,12***	^ p<0,05

Note: values are statistically significant compared with the control group. (\*- p<0,05, \*\*- p<0,01, \*\*\*- p<0,001); ^ - Differences are statistically significant compared with the values between the main study groups. (^ - p<0,05, ^^ - p<0,01, ^^ - p<0,001).

The degree of association between the appearance of troponin in serum and the prescribed immunosuppressive therapy regimen was calculated using the online statistical calculator at

Accordingly, in the group of patients prescribed hormone therapy, serum troponin was detected in 2 out of 53 patients, whereas in the group prescribed cytostatics it was detected in 7 out of 26 patients. When the results were statistically analyzed, the degree of association between the presence of serum troponin and the prescribed immunosuppressive therapy regimen showed a statistically significant value of  $\chi^2 = 9.260$  ( $p = 0.003$ ) (Table 1). In general, the significantly higher detection rate of troponin in the cytostatic group ( $n = 26$ ; 19/7) may be explained by the direct cardiotoxic effect of these agents; however, such an effect is not typical for glucocorticosteroids (GCS) [5, 6]. The rare occurrence of troponin in the hormone therapy group ( $n = 53$ ; 51/2) may be associated with severe arterial hypertension (AH) and myocardial ischemia.

In the hormone therapy group, creatine kinase-MB (CK-MB) was  $3.62 \pm 0.54$  ng/mL, reflecting a normal range, and its change compared with the control group was not statistically significant ( $p > 0.05$ ). In contrast, in the cytostatic therapy group, CK-MB was  $5.37 \pm 0.63$  ng/mL, which was significantly higher compared with the control group ( $p < 0.01$ ) and exceeded the normal reference values. When the results were compared between the main study groups, CK-MB levels were significantly higher in the cytostatic group compared with the hormone therapy group ( $p < 0.05$ ) (Table 1). The increase in this enzyme is explained by the direct cardiotoxic effect of cytostatics on the myocardium, whereas glucocorticosteroids (GCS) are not considered to have a direct negative effect on cardiomyocytes, as also reported in the literature [8, 9].

In serum, myoglobin levels in the hormone therapy group were  $38.6 \pm 6.28$  ng/mL, reflecting values within the normal range and showing an insignificant change compared with the control group ( $p > 0.05$ ). In contrast, in the cytostatic therapy group, myoglobin was  $59.1 \pm 7.87$  ng/mL, which was significantly higher than in the control group ( $p < 0.01$ ) and exceeded the reference range. When the results of the main study groups were compared, myoglobin levels, similarly to CK-MB, were significantly higher in the cytostatic group compared with the hormone therapy group ( $p < 0.05$ ) (Table 1). This increase is attributed to the direct cardiotoxic effect of cytostatics on the myocardium, whereas glucocorticosteroids do not have a direct negative effect on cardiomyocytes [12, 16]. However, although

statistical analysis did not show significant differences, a noticeable increase in myoglobin in the hormone therapy group may be explained by steroid-induced myopathy [15].

Changes in aspartate aminotransferase (AST), a marker of hepatocyte and cardiomyocyte lysis, in serum demonstrated the following pattern across the study groups. In the hormone therapy group, AST was  $41.6 \pm 5.91$  mmol/L, showing a statistically significant increase compared with the control group ( $p < 0.05$ ). In the cytostatic therapy group, AST was  $57.8 \pm 5.12$  mmol/L, which was also significantly elevated compared with the control group ( $p < 0.001$ ). When the results of the main study groups were compared, AST, similar to other cardiospecific enzymes, showed a statistically significant increase in the cytostatic group compared with the hormone therapy group ( $p < 0.05$ ) (Table 1).

Under the influence of GCS therapy, AST levels were observed to be almost twofold higher compared with healthy individuals. The literature also reports that, in the context of hormone therapy, activation of hepatocyte metabolism leads to increased synthesis of this enzyme, while the mild cytolytic effect of GCS may also contribute to a certain elevation of AST. However, this finding does not usually require clinical concern, since attention is generally warranted only when AST increases by at least 2–3 times together with ALT elevation and in the presence of jaundice.

In contrast, cytostatics - particularly agents such as cyclophosphamide and tacrolimus—are known to possess not only cardiotoxic but also significant hepatotoxic effects. Therefore, in our study, AST levels in this group were significantly higher both compared with the control group and the hormone therapy group. This indicates that strict monitoring of this enzyme is required during immunosuppressive therapy [5, 10].

## Conclusion

Thus, in modern clinical medicine, drugs widely used in immunosuppressive therapy (IST) inevitably exert adverse effects on the cardiovascular system, similar to their toxic effects on other organs and tissues, including hepatotoxic, nephrotoxic, and neurotoxic actions. In our study, we also observed a certain increase in all investigated cardiospecific laboratory parameters in patients receiving these agents compared with healthy individuals.

However, the significance of our findings lies in the fact that troponin was detected significantly more frequently in the cytostatic group compared with the hormone therapy group. In addition, creatine kinase-MB, myoglobin, and AST levels

were not only higher compared with healthy individuals, but also significantly elevated and exceeded normal values when compared with patients receiving glucocorticosteroid therapy.

From this perspective, it is necessary to strictly monitor cardiospecific laboratory parameters in patients with chronic glomerulonephritis receiving cytostatics as part of IST, as well as to implement timely detection and appropriate corrective measures. This approach will help prevent the development of cardiovascular events and subsequent cardiovascular complications associated with IST in patients with glomerulonephritis.

### References

1. Vasyuk Yu.A. et al. Consensus statement of Russian experts on the prevention, diagnosis, and treatment of cardiovascular toxicity of anticancer therapy // *Russian Journal of Cardiology*. – 2021. – Vol. 26, No. 9. – P. 4703. doi:10.15829/1560-4071-2021-4703.
2. Degtyaryova E.A. et al. Rare immune-mediated adverse events: a literature review // *FARMATEKA*. – 2020. – No. 7. – P. 8–14. <https://dx.doi.org/10.18565/pharmateka.2020.7.8-14>
3. Ivanov D.O. et al. Diseases of the cardiovascular system as a cause of mortality in the Russian Federation: ways to solve the problem // *Medicine and Healthcare Organization*. – 2019. – Vol. 4, No. 2. – P. 4–12.
4. Kaprina A.D. et al. The state of oncological care for the population of Russia in 2022. – Moscow: P.A. Herzen Moscow Oncology Research Institute – Branch of the National Medical Research Center of Radiology of the Ministry of Health of Russia. 2022. 239 p.
5. Kozyavin N.A. et al. Cardiovascular complications of modern anticancer drug therapy: chemotherapy, targeted therapy and immunotherapy (textbook). – St. Petersburg: N.N. Petrov National Medical Research Center of Oncology, 2023. – 82 p. ISBN 978-5-6048250-2-0
6. Chazova I.E. et al. Eurasian clinical guidelines for the diagnosis, prevention, and treatment of cardiovascular complications in anticancer therapy // *Eurasian Journal of Cardiology*. – 2022. – Vol. 1, No. 37. – P. 6–79. doi:10.38109/2225-1685-2022-1-6-79.
7. Sharafeev A.Z. et al. Cardio-oncology – a new direction in cardiology // *Practical Medicine*. – 2020. – Vol. 18, No. 1. – P. 38–42.
8. Avila V.S. et al. Carvedilol for prevention of chemotherapy-related cardiotoxicity: The CECCY trial // *Journal of the American College of Cardiology*. – 2018. – Vol. 71(20). – P. 2281–2290. doi:10.1016/j.jacc.2018.02.049.
9. Chen D.Y. et al. Cardiovascular toxicity of immune checkpoint inhibitors in cancer patients: A review when cardiology meets immuno-oncology // *Journal of the Formosan Medical Association*. – 2020. – Vol. 119(10). – P. 1461–1475. doi:10.1016/j.jfma.2019.07.025.
10. Ghadri J.R. et al. International expert consensus document on Takotsubo syndrome (Part I): Clinical characteristics, diagnostic criteria, and pathophysiology // *European Heart Journal*. – 2018. – Vol. 39(22). – P. 2032–2046. doi:10.1093/eurheartj/ehy076.
11. Lyon A.R. et al. 2022 ESC Guidelines on cardio-oncology developed in collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO), and the International Cardio-Oncology Society (IC-OS): Developed by the ESC Cardio-Oncology Task Force // *European Heart Journal – Cardiovascular Imaging*. – 2022. – Vol. 23(41). – P. 4229–4361. doi:10.1093/eurheartj/ehac244.
12. Lyon A.R. et al. Baseline cardiovascular risk assessment in cancer patients scheduled to receive cardiotoxic cancer therapies: a position statement and new risk assessment tools // *European Journal of Heart Failure*. – 2020. – Vol. 22(11). – P. 1945–1960. doi:10.1002/ejhf.1920.
13. Mahmood S.S. et al. Myocarditis in patients treated with immune checkpoint inhibitors // *Journal of the American College of Cardiology*. – 2018. – Vol. 71(16). – P. 1755–1764. doi:10.1016/j.jacc.2018.02.037.
14. Moslehi J.J. et al. Increased reporting of fatal immune checkpoint inhibitor-associated myocarditis // *The Lancet*. – 2018. – Vol. 391(10124). – P. 933. doi:10.1016/S0140-6736(18)30533-6.
15. Patel R.P. et al. Cardiotoxicity of immune checkpoint inhibitors // *Current Oncology Reports*. – 2021. – Vol. 23(7). – P. 79. doi:10.1007/s11912-021-01070-6.
16. Zamorano J.L. et al. ESC position paper on cancer treatments and cardiovascular toxicity // *Russian Journal of Cardiology*. – 2017. – Vol. 3(143). – P. 105–139.