



Journal Website:  
<https://theusajournals.com/index.php/ijmscr>

Copyright: Original  
content from this work  
may be used under the  
terms of the creative  
commons attributes  
4.0 licence.



## BIOCHEMICAL CHANGES IN THE BLOOD INFLAMMATORY DISEASES OF THE NOSE AND PARANASAL SINUS IN PATIENTS WITH MYOCARDITIS

Submission Date: April 16, 2023, Accepted Date: April 21, 2023,

Published Date: April 26, 2023

Crossref doi: <https://doi.org/10.37547/ijmscr/Volume03Issue04-16>

Djuraev J.A.

Tashkent Medical Academy, Uzbekistan

Khasanov U.S.

Tashkent Medical Academy, Uzbekistan

Khodjanov Sh.Kh.

Tashkent Medical Academy, Uzbekistan

Akhundjanov N.A.

Tashkent Medical Academy, Uzbekistan

Makhamadaminova Sh.A.

Tashkent Medical Academy, Uzbekistan

Jumanov D.A.

Tashkent Medical Academy, Uzbekistan

### ABSTRACT

In approaches to the treatment of infectious lesions of the myocardium, contradictions remain. Scientific studies evaluating the effectiveness of various drugs in infectious myocarditis that developed against the background of acute respiratory infections are few and contradictory. Metabolic agents for various pathologies of the myocardium, according to some authors, are undoubtedly necessary, but others consider their appointment unreasonable. Almost 80% of patients with various acute infectious diseases reveal certain changes in cardiovascular activity. Most of them represent a natural functional reaction of the body to the action of the pathogen and completely disappear as a result of the treatment of the underlying pathology.

### KEYWORDS

Myocarditis, sinusitis, acute heart failure.

## INTRODUCTION

Infectious diseases, despite the modern possibilities of treatment and prevention, remain the main pathology of the population [2]. Today, it is believed that from 1 to 5% of all patients with acute respiratory diseases, including influenza, have signs of infectious myocarditis [1,5]. It is very difficult to establish the true frequency, since latent and mild forms, which are most characteristic of childhood, range from 24 to 33%, are rarely diagnosed and end on their own in the absence of any special treatment, or transform into a chronic process with nonspecific symptoms [2,3, 7].

The immediate cause of death is acute heart failure, which progresses against the background of water and electrolyte disorders, intoxication, and the direct effect of the pathogen or its toxin on cardiomyocytes [6]. Any infectious disease may be accompanied by certain changes in the functioning of the cardiovascular system of varying severity and duration.

Dysfunction of the cardiovascular system can occur in about 80% of patients with various acute infectious diseases [4,9]. Most of them are a natural functional reaction of the body, they end on their own, without additional special treatment. But some patients develop rhythm disturbances and heart failure, often with a long protracted course, and sometimes with a risk of death.

It is now known that any of the known pathogens can cause myocardial damage, including myocarditis [5]. Often, viruses that can directly interact with cardiomyocytes become an etiological factor, which leads to cell apoptosis [6].

The diagnosis of myocarditis or cardiomyopathy is not in doubt, usually in severe forms of myocardial

damage. With mild or moderate severity, the observed clinical symptoms of complications are nonspecific and varied, depending not only on the etiology and severity of the manifestations of the underlying process, but also on the individual characteristics of the organism.

In approaches to the treatment of infectious lesions of the myocardium, contradictions remain. Scientific studies evaluating the effectiveness of various drugs in infectious myocarditis that developed against the background of acute respiratory infections are few and contradictory. Metabolic agents for various pathologies of the myocardium, according to some authors, are undoubtedly necessary, but others consider their appointment unreasonable [5,10].

Almost 80% of patients with various acute infectious diseases have certain changes in cardiovascular activity [8]. Most of them represent a natural functional reaction of the body to the action of the pathogen and completely disappear as a result of the treatment of the underlying pathology. However, in some cases, an independent pathological process develops in the heart, which can directly affect not only the course of infection, its duration and outcome, but also determine the quality and life expectancy [4,9].

The purpose of this study is to identify biochemical changes in the blood during inflammatory diseases of the nose and paranasal sinuses in patients with myocarditis.

Materials and research methods. The study included 186 patients with myocarditis who were hospitalized at the Republican Specialized Center of Cardiology. The patients were divided into two groups. The first group consisted of 80 patients with chronic inflammatory

diseases of the nose and paranasal sinuses. The second group consisted of 106 patients without pathology of the nose and paranasal sinuses. All patients were subjected to a comprehensive clinical and laboratory study, which included the collection of an anamnesis of the disease, laboratory tests, nasal endoscopy, X-ray examination and biochemical studies. The control group consisted of 20 healthy volunteers from the staff of the 2nd clinic of the Tashkent Medical Academy.

Research results. Group I patients complained of difficulty in nasal breathing (92.5%), nasal discharge (78.4%), impaired sense of smell (22.2%), subfebrile fever (36.4%), general weakness (42.5%). Headaches were also often noted (78.4%), more in the maxillary region. Patients of the II group had practically no complaints from the nose and paranasal sinuses. With comparative indicators of blood tests in the studied groups (Table 1), it was revealed:

Table 1

Parameters of the general blood test in patients with myocarditis

Indicators	I, M±m (n=80)	II group, M±m (n=106)	Control group, M±m (n=20)
Leukocytes (10 <sup>9</sup> /l)	7.54±0.60*	6.84 ± 0.52 __	6.15±0.39
ESR (mm/h)	21.05±3.40*	18.73 ± 3.05 * __	6.36±0.80
Lymphocytes (%)	22.64±1.96	26.45 ± 1.87 __ __	30.87±1.90
Monocytes (%)	4.61±0.56	4.25 ± 0.42 _	3.58±0.37
Eosinophils (%)	1.24±0.26	1.46 ± 0.30 _	2.47±0.32
Band (%)	4.23±0.77	3.28 ± 0.62 _	2.66±0.36
Segmented (%)	67.22±1.50	64.55 ± 1.62 _	60.75±1.86

\*- the difference is highly significant, p< 0.001.

In a laboratory blood test, all patients showed leukocytosis and an increase in ESR, especially these changes were more pronounced in patients with chronic inflammatory diseases of the nose and paranasal sinuses. At the same time, in this group, the number of leukocytes was 7.54±0.60x10<sup>9</sup>/l, and the ESR was increased to 21.05±3.40 mm/hour.

Table 2

Indicators of biochemical blood tests in patients with myocarditis and in the control group

Indicators	I, M±m	II group, M±m	Control group, M±m
------------	--------	---------------	--------------------

	( n =80 )	( n =106)	( n =20)
Creatinine ( $\mu\text{mol/l}$ )	78.12 $\pm$ 3.10	82.22 $\pm$ 2.92	84.60 $\pm$ 2.82
Urea (mmol/l)	5.04 $\pm$ 0.23	5.85 $\pm$ 0.30	6.65 $\pm$ 0.45
ALT (mmol/ g.l )	0.70 $\pm$ 0.09	0.64 $\pm$ 0.07	0.42 $\pm$ 0.06
AST (mmol/ g.l )	1.13 $\pm$ 0.23*	0.94 $\pm$ 0.18	0.48 $\pm$ 0.06
LDH (mmol/ g.l )	7.97 $\pm$ 1.24*	7.20 $\pm$ 1.03*	4.90 $\pm$ 0.28
CPK (mmol/ gl )	10.68 $\pm$ 3.10*	8.76 $\pm$ 2.74*	2.86 $\pm$ 0.49
Protein (g/l)	73.40 $\pm$ 1.51	72.25 $\pm$ 1.52	70.18 $\pm$ 1.65

\*- the difference is highly significant,  $p < 0.001$ .

As shown in Table 2, the biochemical analysis of blood also shows more pronounced changes in patients of the first group than in patients of the second group.

An increase in AST (1.13 $\pm$ 0.23 mmol/ gl ) in patients with myocarditis is associated with cell destruction or increased plasma membrane permeability. There was also an increase in creatine phosphate kinase (CPK) in patients of both groups (10.68 $\pm$ 3.10 mmol/ gl and 8.76 $\pm$ 2.74 mmol/ gl , respectively), which indicates increasing myocardial damage.

When examining troponin levels I quantitative method in patients of the first group with chronic diseases of the nose and paranasal sinuses, its average concentration was significantly higher ( $p = 0.0001$ ) than in patients of the second group without pathology of the nose and paranasal sinuses (0.49  $\pm$  0.09 Ng / ml and 0.39 $\pm$ 0.06 Ng /ml, respectively ). Troponin test data I quantitative method are presented in table 3.

Table 3

Troponin level I quantitative method in patients with myocarditis

Groups	of patients n	Troponin level I , Ng / ml, M $\pm$ m
I group	80	0.49 $\pm$ 0.09
II group	106	0.39 $\pm$ 0.06
Control group	20	0.05 $\pm$ 0.02

\*- the difference is highly significant,  $p < 0.001$ .

## CONCLUSIONS

In connection with the foregoing, a laboratory study showed specific changes in the blood in the myocardium, as well as a comparative assessment of pronounced changes in the simultaneous course of inflammation in the myocardium and paranasal sinuses. Thus, our studies of patients with myocarditis revealed the fact that the clinical course of myocarditis is more pronounced in patients with chronic inflammatory diseases of the nose and paranasal sinuses due to the presence of an infection focus in the ENT organs.

## REFERENCES

1. Khasanov US, Juraev Zh.A., Toshpulatov Zh. Features of diseases of the nose and paranasal sinuses in patients with myocarditis. Young scientist. 2016(10):547-50.
2. Shamsiev D.F., Vohidov U.N., Karimov O.M. A modern view on the diagnosis and treatment of chronic inflammatory diseases of the nose and paranasal sinuses. Young scientist. 2018(5):84-8.
3. Buggey J., ElAmm CA Myocarditis and cardiomyopathy. Curr Opin Cardiol . 2018 May;33(3):341-346.
4. Ghogomu N., Kern R. Chronic rhinosinusitis : the rationale for current treatments. Expert Rev Clin Immunol . 2017 Mar;13(3):259-270.
5. Hekimian G., Combes A. Myocarditis . Rev Med International . 2017 Aug;38(8):531-538.
6. Mlejnek D., Krejčí J. Myocarditis and inflammatory cardiomyopathy. Vnitr Lek . Fall 2017;63(7-8):507-512.
7. Slovick A., Cornet M., Surda P., Tomazic PV Chronic rhinosinusitis : New understanding of specific and general Quality of life scores. Rhinology . 2016 Dec 1;54(4):289-291.
8. Van Linthout S., Tschöpe C. Inflammation - Cause or Consequence of Heart Failure or Both? Curr Heart Fail Rep . 2017 Aug;14(4):251-265.
9. Vokhidov UN State of reticular fibers in various forms of polypoid rhinosinusitis. European science review. 2015(7-8):39-40.
10. Wu J., Jain R., Douglas R. Effect of paranasal anatomical variants on outcomes in patients with limited and diffuse chronic rhinosinusitis . Auris Nasus Larynx . 2017 Aug;44(4):417-421.