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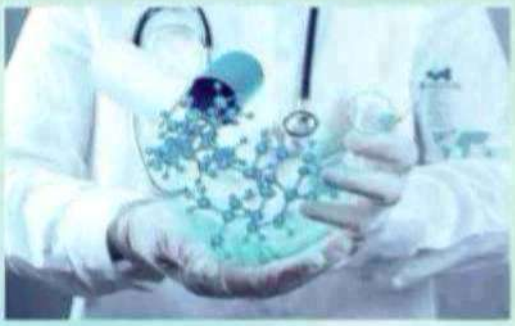

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ISSN 2181-712X.
EiSSN 2181-2187

3 (89) 2026

# ТИББИЁТДА ЯНГИ КУН
# НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
# NEW DAY IN MEDICINE

***Илмий-рефератив, маънавий-маърифий журнал***
***Научно-реферативный,***
***духовно-просветительский журнал***

УЧРЕДИТЕЛИ:

БУХАРСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ ИНСТИТУТ
ООО «ТИББИЁТДА ЯНГИ КУН»

Национальный медицинский исследовательский центр хирургии имени А.В. Вишневского является генеральным научно-практическим консультантом редакции

Журнал был включен в список журнальных изданий, рецензируемых Высшей Аттестационной Комиссией Республики Узбекистан (Протокол № 201/03 от 30.12.2013 г.)



**3 (89)**

**2026**
*март*

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***Received: 20.02.2026, Accepted: 06.03.2026, Published: 10.03.2026***

**УДК 616.72-002.77:612.017**

# ХАРАКТЕРИСТИКА ИММУННОЛОГИЧЕСКОГО СОСТОЯНИЯ БОЛЬНЫХ ГЕМАРТРОЗАМИ

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✓ ***Резюме***

***Гемартроз при внутрисуставных повреждениях сопровождается не только механическим разрушением тканей, но и комплексной иммунопатологической реакцией - как на системном, так и на местном уровне. Эти данные не только уточняют патогенез травматического воспаления, но и создают основу для дальнейшей разработки прогностических критериев и оценки потенциала иммунокоррекции как элемента персонализированной терапии.***

***Ключевые слова: гемартроз, клеточный и гуморальный иммунитет, изменения.***

# GEMATROZLI BEMORLARDA IMMUNOLOGIK HOLATNING TAVSIFI

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✓ ***Rezyume***

***Bo‘g‘im ichi sinishlari natijasida rivojlangan gemartroz nafaqat to'qimalarni mexanik ravishda shikastlanishi balki murakkab mahalliy hamda sistemali immunopatologik reaktsiya bilan kechadi. Ushbu ma'lumotlar nafaqat travmatik yallig'lanishning patogenezini aniqlaydi, balki bashoratlash mezonlarni yaratishda asos bo‘lib personallashtirilgan immunokoreksiyani o‘tqazishga zamin yaratadi.***

***Kalit so'zlar: gemartroz, hujayra va gumoral immunitet, o'zgarishlar.***

# IMMUNE STATUS IN PATIENTS WITH HEMARRHOSIS

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✓ ***Resume***

***Hemarthrosis in intra-articular injuries is accompanied not only by mechanical destruction of tissues, but also by a complex immunopathological reaction - both at the systemic and local levels. These data not only clarify the pathogenesis of traumatic inflammation, but also create a basis for further development of prognostic criteria and assessment of the potential of immunocorrection as an element of personalized therapy.***

***Key words: hemarthrosis, cellular and humoral immunity, changes.***

## Relevance

The formation of hemarthroses (HA) following traumatic joint injuries is accompanied by activation of systemic and local immune responses. The inflammatory cascade initiated by tissue damage involves both innate and adaptive mechanisms, encompassing a wide range of immunological components (2,4,6,8). The most sensitive to traumatic effects are parameters of cellular and humoral immunity, as well as the cytokine profile, which reflects the balance of proinflammatory and anti-inflammatory mediators. Intra-articular fractures of the extremities represent one of the most complex forms of traumatic injury, accompanied by disruption of the anatomical integrity of the articular surfaces, capsular-ligamentous apparatus, and synovial membrane (1,3,5,7).

The significant clinical significance of intra-articular fractures of the extremities is due to their frequency, the high likelihood of complications, and the risk of permanent loss of joint function, including due to the development of hemarthrosis (7,10).

High morbidity rates, the frequency of complications, the inadequacy of early prognosis and risk stratification systems, and the significant socioeconomic burden associated with the treatment and rehabilitation of patients with intra-articular fractures necessitate a comprehensive study of the clinical and immunological mechanisms of hemarthrosis and its outcomes.

**Study Objective:** To analyze the quantitative and qualitative assessment of immune homeostasis disturbances that can influence the clinical course and recovery potential after injury.

## Material and Methods

This study was conducted as part of a prospective, comparative, clinical and immunological study. The study design utilized a stepwise approach to conceptual development and data interpretation, enabling the development of differentiated methods for predicting the outcome of comprehensive treatment for hemarthrosis in intra-articular fractures of the extremity bones.

The total sample size was 150 individuals: 60 patients with hemarthrosis associated with intra-articular fractures in the study group, 60 in the comparison group with isolated hemarthrosis without bone pathology, and 30 clinically healthy individuals in the control group. All patients were treated at the Samarkand branch of the Republican Specialized Scientific and Practical Medical Center for Traumatology and Orthopedics from 2021 to 2024. The average age of those examined in the control group was 45.3 ± 8.9 years, 44.8 ± 9.2 years in the comparison group, and 46.1 ± 8.7 years in the study group.

During the laboratory examination, an extensive assessment of immunological status was conducted.

All studies were conducted dynamically (before treatment, on days 7, 14, and 30 of complex treatment) in compliance with biosafety standards and double internal quality control.

## Results and discussion

Analysis of cellular immune system parameters revealed significant and consistent differences between the study groups, reflecting the degree of immunosuppression associated with traumatic injuries of varying severity.

Lymphocyte levels in the peripheral blood were reduced in patients in both clinical groups compared to the control group. In the comparison group, lymphocyte levels were 16% lower than normal ($p=0.014$), while in the main group, they were 29% lower than in the control group ($p=0.003$). Furthermore, the level in the main group was also significantly lower than in the comparison group (by 15%, $p=0.028$), indicating a direct relationship between the depth of lymphopenia and the severity of injury.

A decrease in the total number of T-lymphocytes (CD3+) was also noted. In the comparison group, their level was 20% lower than in the control group ($p=0.011$), while in the main group, the decrease reached 32% ($p <0.001$). The difference between the two groups of patients was approximately 15% ($p=0.036$), indicating progressive T-cell depletion with increasing traumatic exposure.

The T-helper subset (CD4+) also showed a clear downward trend. In patients with fractures, CD4+ levels were 30% lower than in controls ($p <0.001$) and 17% lower than in the comparison group ($p=0.021$). In the group with isolated injuries, this indicator decreased by approximately 15%

compared to controls (p=0.019), further confirming the sensitivity of this parameter to the degree of immune destabilization.

Of particular interest was the behavior of cytotoxic T lymphocytes (CD8+). In the study group, a 19% decrease was observed compared to the control group (p=0.017), while in the comparison group, no significant differences from the norm were observed (p> 0.05). The difference between the study and comparison groups was approximately 9% (p=0.042), which may indicate selective depletion of the cytotoxic component during more severe injuries.

Changes in the level of NK cells (CD16+/56+) were particularly noteworthy. In the study group, their number was 36% higher than in the control group (p=0.009) and approximately 23% higher than in the comparison group (p=0.038). These changes can be interpreted as compensatory activation of innate immunity in the face of depleted adaptive mechanisms.

The functional parameter of innate immunity (FI) was significantly lower in patients in both groups. In the comparison group, the decrease was approximately 7% compared to the control group (p=0.018), while in the study group it was almost 13% (p=0.002). The difference between the clinical groups was statistically significant (p=0.031), indicating a decrease in phagocytic activity depending on the severity of the injury.

Analysis of humoral immune system parameters demonstrated a characteristic restructuring, accompanied by shifts both toward activation and toward depletion of individual components of the immune response. These changes were progressive and more pronounced in patients with HA associated with intra-articular fractures.

IgA levels were elevated in both clinical groups compared to the control group. In the comparison group, IgA levels increased by approximately 11% relative to the norm (p=0.027), while in the study group, they increased by more than 20% (p <0.001). The difference between the study and comparison groups was also statistically significant (p=0.031). This increase in IgA may reflect activation of mucosal and synovial immunity in response to persistent inflammation.

IgG levels also decreased with increasing injury severity. Although the changes in the comparison group were not statistically significant (p> 0.05), a 16% decrease was observed in the study group compared to the control group (p=0.008), as well as a significant difference compared to the non-fracture group (p=0.043). A decrease in IgG levels may indicate exhaustion of the specific humoral response and the consumption of previously synthesized antibodies.

CIC levels are of particular importance. In both clinical groups, their levels were significantly higher than in the control group: by approximately 23% (p=0.012) in the comparison group and by over 47% (p <0.001) in the study group. The difference between the groups also reached statistical significance (p=0.015). An increase in CIC levels reflects the active phase of the immune response, possibly with impaired clearance, which can contribute to the development of secondary inflammatory reactions in the synovial membrane. When assessing the complement system, a consistent decrease in the C3 and C4 components was noted. In the study group, the C3 level was 21% lower than in the control (p=0.004) and 14% lower than in the comparison group (p=0.039). For the C4 component, the decrease was 13% compared to the control in the comparison group (p=0.031) and 25% in the study group (p <0.001), with a significant difference between the groups (p=0.027). Such changes are characteristic of complement consumption during activation of the classical immune inflammatory pathway.

Thus, humoral immunity in HA undergoes bidirectional changes - a combination of activation (increase in IgA, circulating immune complexes) and depletion (IgM, IgG, lysozyme, complement), which is especially pronounced in intra-articular fractures.

IL-1β was found to be one of the most sensitive markers of inflammatory activation.

IL-6 demonstrated the most pronounced dynamics among all mediators analyzed. Its level in the comparison group exceeded control values by 67% (p < 0.001), while in the main group, it exceeded control values by 143% (p < 0.001). A significant difference was also recorded between the clinical groups (p = 0.019), underscoring the key role of IL-6 as a mediator of acute inflammation and a prognostic marker for the severity of tissue damage.

TNF-α also increased significantly in response to injury. In the comparison group, its concentration was 43% higher than in the control group (p=0.014), while in the study group, it was 93% higher

(p<0.001). The difference between the groups was approximately 35% (p=0.034), confirming an enhanced systemic cytokine response in the context of more extensive tissue damage.

IL-10, the main anti-inflammatory cytokine, also increased, indicating a compensatory regulatory response. In the comparison group, its level was 31% higher than normal (p=0.022), while in the study group, it was 61% higher (p<0.001). The difference between the groups was statistically significant (p=0.041), reflecting the body's enhanced attempt to limit the inflammatory cascade during more severe injuries. Thus, the cytokine profile in traumatic AA is characterized by a significant and dose-dependent shift in both proinflammatory (IL-1β, IL-6, TNF-α) and regulatory (IL-10) mediators. The data obtained confirm the systemic nature of the inflammatory response and emphasize the importance of these parameters as potential markers of injury severity and clinical prognosis.

An analysis of cellular and humoral immune system parameters revealed significant and consistent changes depending on the nature of the traumatic injury. Patients with AA, especially those with intra-articular fractures, exhibit progressive suppression of the adaptive immune response against a background of moderate activation of innate mechanisms.

Thus, patients with traumatic GA exhibit a complex disruption of immune homeostasis, characterized by dose-dependent suppression of the cellular and humoral components of the immune system. These changes are most pronounced in the presence of intra-articular fractures and likely have prognostic significance, determining the increased risk of complications and delayed recovery.

A study of immunological parameters in the SF of patients with traumatic GA of the knee joint allowed for a more in-depth assessment of the local inflammatory mechanisms directly occurring within the joint cavity. The data obtained demonstrated a pronounced local immune response, which is significantly enhanced by the presence of an intra-articular fracture.

The leukocyte count in the aspirate of patients in the study group was significantly higher than in the comparison group—an increase of over 50% (p=0.004), reflecting intense cellular infiltration at the site of injury. This leukocyte accumulation confirms the activation of a local innate inflammatory response. Cytokine profile analysis revealed a significant increase in proinflammatory mediator levels in the study group. Thus, IL-1β concentrations in fracture patients were 60% higher than in the comparison group ($p < 0.001$), IL-6 increased by more than 60% ($p < 0.001$), and TNF-α increased by almost 63% ($p = 0.002$). These differences confirm the development of active local inflammation involving a cytokine cascade that promotes tissue damage, increased vascular permeability, and the progression of synovial hyperproduction.

IL-10 levels, reflecting the anti-inflammatory component of regulation, were also higher in the study group (by 34%, $p = 0.041$), indicating the body's attempts to limit inflammation at the local level. However, the increase in IL-10 occurred in parallel with an increase in the proinflammatory response, indicating A relative deficiency of regulatory activity.

Of particular importance for assessing pathogenesis is the level of MMP-9, which is sharply elevated in the puncture fluid of patients with fractures—almost 1.7 times higher than in patients with isolated trauma ($p < 0.001$). Of the humoral immunity components, a significant increase in IgG (by about 50%, $p = 0.009$) and circulating immune complexes (CIC) (an increase of 44%, $p = 0.007$) was noted in patients with fractures. These data may indicate activation of the B-cell response, increased antibody production, and the potential for immune damage to the joint membrane in a secondary autoimmune mechanism.

Against this background, a decrease in lysozyme concentration was detected in the study group (by almost 19%, $p = 0.031$), indicating a weakening of nonspecific antibacterial protection in the joint cavity and creating conditions for the development of complications such as secondary synovitis or infection. Thus, immunological parameters in the synovial fluid differ significantly in patients with different types of joint damage. Intra-articular fractures are characterized by a more pronounced activation of inflammatory and tissue-destructive mechanisms, including cytokine hypersecretion, increased production of metalloproteinases, and local B-cell hyperreactivity. These changes are key to the development of the pathological process and can be considered as links in the pathogenesis of GA complicated by structural damage.

Immunological analysis of aspirate from the synovial fluid revealed a consistent activation of inflammatory mediators and enzymatic components, similar to that observed in CL injuries, but with

lower absolute values. However, the trends and differences between the groups remained statistically significant.

The leukocyte count in the synovial fluid in patients with intra-articular fractures of the synovial fluid was more than 57% higher than in the comparison group (p=0.006), indicating pronounced infiltration of the joint cavity by inflammatory cells. Levels of key proinflammatory cytokines (IL-1β, IL-6, and TNF-α) were significantly elevated in patients in the study group. IL-1β exceeded the comparison group level by 57% ($p < 0.001$), IL-6 by 58% ($p < 0.001$), and TNF-α by 58% ($p = 0.003$). These differences confirm the activation of a local cytokine cascade in more severe joint injury.

As in GA HSS, IL-10 levels were elevated in patients with fractures by approximately 33% compared to the comparison group ($p = 0.037$), which can be considered a response to excessive proinflammatory stimulation. However, despite the activation of the anti-inflammatory mechanism, the balance is clearly skewed toward inflammation. A significant increase in MMP-9 levels was observed, reaching over 65% in patients with fractures compared to those with isolated injuries ($p < 0.001$). IgG concentrations in the puncture fluid of the study group were approximately 48% higher than in the comparison group ($p = 0.009$), confirming the activation of the humoral response and the probable involvement of antibodies in maintaining the inflammatory process. A significant increase in circulating immune complexes (CIC) was also observed (by 43%, $p = 0.013$), reflecting an enhanced autoimmune component.

Lysozyme levels were reduced by approximately 21% in patients in the study group compared to the comparison group (p=0.027), indicating suppression of local nonspecific defense factors, similar to that observed in the KS.

Thus, intra-articular fractures of the AJ are accompanied by a pronounced local inflammatory immune response with active cytokine secretion, increased proteolytic activity, and disruption of local humoral homeostasis. These changes, in their mechanism and direction, replicate the patterns identified in the KS and indicate a typical pathogenetic cascade initiated by the destruction of joint structures.

The accompanying increase in IL-10 levels can be interpreted as a response of the immune regulatory system aimed at limiting the excessive inflammatory cascade. However, despite the enhancement of anti-inflammatory mechanisms, the balance in both locations was maintained, with a pronounced predominance of the proinflammatory component, especially in patients with fractures. The identified changes in humoral and enzymatic parameters have additional pathogenetic significance: increased IgG and circulating immune complexes (CIC), increased MMP-9 concentrations, and decreased lysozyme. These changes indicate B-cell activation, degradation of articular matrix components, and decreased nonspecific antimicrobial defense. The increase in MMP-9 is particularly important for explaining the mechanisms of synovial and cartilage destruction, as well as the development of post-traumatic degenerative changes.

A comparison of data between joints revealed that although absolute levels of cytokines and other parameters are somewhat higher in patients with joint injury, the direction and nature of the changes are largely the same. This allows us to consider the identified immune changes as a universal local pathogenetic mechanism of arterial hypertension, which is amplified by structural destruction of intra-articular tissues.

A comprehensive analysis of systemic and local immune parameters in patients with GA revealed coordinated and pathogenetically interconnected shifts reflecting the depth of the inflammatory response to joint injury. A peripheral blood immunogram reflected the development of a systemic post-traumatic immunological imbalance, while SF data demonstrated pronounced local activation of inflammatory and tissue-destructive mechanisms.

The systemic immune response was characterized by a significant decrease in the absolute number of T and B lymphocytes, humoral depletion (decreased IgM and IgG), increased circulating immune complexes (CIC) levels, and a shift toward an innate response (increased NK cells). These changes are particularly pronounced in patients with intra-articular fractures and indicate the development of secondary immunodeficiency with compensatory activation of non-specific immune systems. At the local level, the same trends were detected in the synovial fluid, but in a more pronounced form: a significant increase in IL-1β, IL-6, TNF-α, IgG, circulating immune complexes (CIC), and MMP-9, with a simultaneous decrease in protective factors such as lysozyme. Similar dynamics of IL-10 in

serum and puncture samples confirm the body's attempts to limit inflammation, but these efforts are clearly insufficient in the face of an active proinflammatory cascade.

A comparative analysis showed that local immune changes intensify and deepen the systemic response, creating a vicious cycle: joint inflammation maintains circulating cytokine activity, which in turn contributes to systemic immunosuppression and dysregulation.

Thus, the identified changes suggest that HA in intra-articular injuries is accompanied not only by mechanical tissue destruction but also by a complex immunopathological reaction, both systemically and locally. These data not only clarify the pathogenesis of traumatic inflammation but also provide a basis for the further development of prognostic criteria and assessment of the potential of immunomodulation as an element of personalized therapy.

**Conclusions**

1. A comprehensive analysis of clinical and immunological parameters in patients with varying degrees of traumatic HA revealed systemic patterns reflecting the severity of injury and the body's inflammatory response.

2. An immunological study revealed that even with relatively isolated joint trauma, there is a significant decrease in the absolute number of T and B lymphocytes, as well as serum IgM and IgG levels, which may indicate the development of post-traumatic immunodeficiency.

3. The study of SF allowed for the objectification of the local immune response. High concentrations of proinflammatory cytokines (IL-1β, IL-6, TNF-α), MMP-9, IgG, and circulating immune complexes (CIC) were recorded, along with a reduction in nonspecific protective factors (lysozyme). Local cytokine overexpression is an important mechanism for joint damage in AA. The increase in MMP-9 is particularly significant, as this enzyme is involved in the degradation of type II collagen and the synovial matrix, contributing to the chronicity of the inflammatory process and the initiation of degenerative changes.

LIST OF REFERENCES:


1. Berdyugina O.V. Hemarthrosis of the knee joint: the importance of laboratory immunological studies. In: Makarov MA, et al., editors. V International Congress of the Association of Rheumatoid Orthopedists: Abstracts of reports*;* 2021 Sep 17–18; Moscow. Voronezh: Nauchnaya kniga; 2021. p. 16–18.
2. Berdyugina O.V, Solomatina L.V. Clinical and laboratory evaluation of aspirate in post-traumatic hemarthrosis of the knee joint. Traumatology and Orthopedics. 2020;(3–4):187–191.
3. Labuzova NA. PRP therapy for hemarthrosis of the knee joint in children. In: Youth Science Week – 2023: Proceedings of the All-Russian Forum; 2023 Mar 23–25; Tyumen. Tyumen: RIC “Aivex”; 2023. p. 188–189.
4. Roskidaylo A.A. Differential diagnostics of non-traumatic hemarthrosis of the knee joint (case report). Modern Rheumatology. 2025;19(1):110–115. doi:10.14412/1996-7012-2025-1-110-115.
5. Eshmurzaev A.A, Marufkhanova H.M. Immunopathogenesis of posttraumatic arthritis: features of the cytokine response. Uzbekiston shifokori. 2022;(1):48–51.
6. Khamdamov B.Z, Napasov I.Z. The role and place of methods for predicting bone regeneration disorders in closed fractures of tubular bones. American Journal of Medicine and Medical Sciences. 2024;14(6):1588–1602. doi:10.5923/j.ajmms.20241406.30.
7. Khamdamov B.Z, Napasov I.Z. Results of application of traditional methods for predicting impairments of bone regeneration in closed fractures of tubular bones. Naturalista Campano. 2024;28(1):1512–1524.
8. Hamdamov B.Z, Napasov I.Z, Hamdamov A.B. The role and place of cellular immunity in bone regeneration. Journal of Education & Scientific Medicine. 2024;1(1):47–57.
9. Napasov I.Z, Hamdamov B.Z. The role and place of humoral immunity in bone regeneration. Journal of Education Scientific Medicine. 2024;1(2):9–16.
10. Напасов И.З., Хамдамов Б.З., Хамдамов А.Б. Results of applying traditional methods for predicting bone regeneration disorders in closed fractures of tubular bones. Тиббиётда янги кун. 2024;3(65):561–576.


**Entered 20.02.2026**