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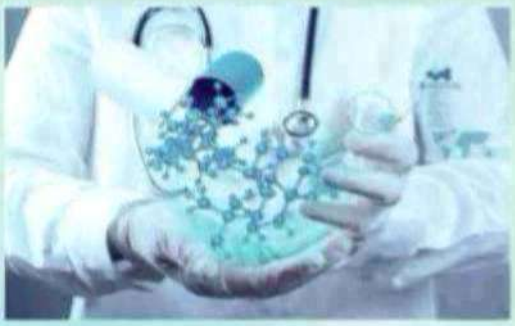

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НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

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COMPREHENSIVE TREATMENT OF MULTIPLE ORGAN FAILURE IN OBSTETRIC SEPSIS

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✓ ***Resume***

Integration of immunological markers of the treatment and diagnostic algorithm into the treatment strategy system improves prognosis accuracy, accelerates the selection of therapeutic options, and helps reduce the risk of adverse outcomes.

Keywords: obstetrics, sepsis, multiple organ failure, immunocorrection.

КОМПЛЕКСНОЕ ЛЕЧЕНИЕ ПОЛИОРГАННОЙ НЕДОСТАТОЧНОСТИ ПРИ АКУШЕРСКОМ СЕПСИСЕ

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✓ ***Резюме***

Интеграция иммунологических маркеров лечебно-диагностического алгоритма в систему лечебной тактики повышает точность прогнозирования, ускоряет выбор терапевтического направления и способствует снижению риска неблагоприятных исходов.

Ключевые слова: акушерство, сепсис, полиорганная недостаточность, иммунокоррекция.

AKUSHERLIK SEPSISDA POLIORGAN YETISHMOVCHILIGINI KOMPLEKS DAVOLASH

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✓ ***Rezyume***

Davolash va diagnostika algoritmining immunologik markerlarini davolash strategiyasi tizimiga integratsiya qilish prognoz aniqligini oshiradi, terapevtik variantlarni tanlashni tezlashtiradi va nojo'ya oqibatlar xavfini kamaytirishga yordam beradi.

Kalit so'zlar: akusherlik, sepsis, ko'p organli yetishmovchilik, immunokorreksiya.

Relevance

The problem of obstetric sepsis (OS) as a leading cause of maternal mortality has been discussed in the literature for decades. Research shows that septic conditions consistently rank among the top three causes of mortality in pregnant and postpartum women, emphasizing the importance of systematically studying this pathology [1, 2, 5].

OS has characteristics due to physiological changes in the immune status during pregnancy.

The molecular mechanisms of the development of multiple organ dysfunction (MOD) in sepsis have been extensively studied in the general clinical population. R.S. Hotchkiss et al. (2023) described in detail the processes of immune cell apoptosis, impaired phagocytosis, and antigen presentation. However, the specific features of these processes in OS remain insufficiently studied [3, 4, 6].

Immunoparalysis in sepsis is considered a key mechanism for worsening the prognosis. In a review by J.S. Boomer et al. (2021) emphasized that decreased HLA-DR expression on monocytes is a marker of severe septicemia. However, insufficient data are available on the dynamics of HLA-DR expression in patients with AS, limiting early prognosis and, consequently, prevention.

Studies on the use of immunotherapy in pregnant and postpartum women are extremely limited, creating gaps in the development of effective and safe treatment protocols for AS [7, 8, 9, 10].

Despite advances in the study of sepsis pathogenesis in the general population, the specific molecular and cellular mechanisms of immune disorders in AS, their impact on the development of multiple organ failure, and the potential for immunoprophylaxis and immunocorrection remain poorly understood. This necessitates targeted research aimed at elucidating the immunopathogenesis of multiple organ failure in patients with AS and developing effective diagnostic and treatment strategies.

Study objective: to develop a therapeutic and diagnostic algorithm for immunoprophylaxis and immunocorrection of multiple organ failure in obstetric sepsis.

Materials and methods

This study was conducted at specialized maternity centers and intensive care units (ICUs), where patients diagnosed with AS were hospitalized between 2021 and 2025. Clinical data were collected in five regional centers (Bukhara, Navoi, Kashkadarya, Samarkand, and Khorezm) and one national center (the Republic of Karakalpakstan). This geographical distribution of clinical data allowed us to create a sample that reflected real-world clinical practice in different regions of the country and created the conditions for conducting a representative study. A total of 250 postpartum women were included in the study, divided into three groups according to the objectives. The reference group (50 patients) consisted of apparently healthy women after uncomplicated deliveries who underwent routine observation in the postpartum unit and had no signs of infectious or inflammatory complications.

Results and discussion

Our analysis of the immune response in women with AS revealed that a complex of persistent abnormalities in the congenital component develops early in the disease. The most revealing finding was a combination of a quantitative increase in $CD14^+$ monocytes with a simultaneous decrease in HLA-DR expression, indicating a paradoxical inflammatory profile: cells are activated but lose their antigen-presenting function. We believe that an additional marker of hypersensitivity was TLR4 overexpression, which increased the transcription of proinflammatory mediators and maintained an imbalance in the inflammatory response. The changes described above became the starting point of the cascade leading to multiple organ dysfunction in AS.

We also observed equally significant changes in the adaptive component. A decrease in the $CD4^+$ lymphocyte count and a drop in the $CD4^+/CD8^+$ ratio, we believe, signified a loss of coordination between the main T cell subpopulations. At the same time, the percentage of $CD8^+$ remained relatively stable, which led to a shift in the balance towards the cytotoxic profile.

Against the backdrop of a T-helper deficiency, NK cell activity increased compensatorily. Deprived of regulatory control, NK cells became an additional source of tissue destruction. Overall, dysregulation of the adaptive component only exacerbated the inherent paradox.

The cytokine profile played a special role in our research and in the results obtained. High IL-6 concentrations combined with insufficient IL-10 levels resulted in a persistent shift toward proinflammatory activation. The IL-6/IL-10 ratio reliably reflected the imbalance between destructive and compensatory mechanisms and directly correlated with the severity of the condition. The integrated marker of systemic inflammation (NLR) complemented the picture, remaining elevated even in the face of partial clinical stabilization. All of this combined created a functional background conducive to the development of multiple organ dysfunction in AS. In our opinion, it is also important to emphasize that clinical and laboratory parameters (PCT, lactate, D-dimer, creatinine, and others) largely reflected the fact that MOF had already occurred. In contrast, immunological parameters

allowed us to predict the development of complications at the preclinical stage. However, they are relatively nonspecific. Therefore, the integration of immunological determinants with clinical and laboratory markers, confirmed by the ROC analysis in the previous chapter, ensured the highest accuracy in predicting the risk of MOF in AS.

The combination of these data allowed us to identify key pathogenetic points that should be targeted for therapeutic intervention. Activation of the monocytic link in the case of functional failure of HLA-DR, imbalance of T-cell regulation, compensatory hyperreactivity of NK cells, cytokine shift towards IL-6 and growth of NLR have become those key mechanisms that not only determine the likelihood of multiple organ decompensation, but can also be considered as direct targets for immunoprophylaxis and immunocorrection as part of the complex treatment of AS. Cytokine regulation constitutes a special group of methods. We considered IL-6 to be a key coordinator of septic inflammation, and its excessive production is closely linked to the development of multiple organ failure. Therefore, the use of anti-IL-6 therapy is justified. In our study, we used tocilizumab (8 mg/kg intravenously, administered once), which binds IL-6 receptors and blocks proinflammatory signaling. Intravenous immunoglobulins were used to correct the humoral component; in particular, IVIG (0.4 g/kg/day for 3 days) was administered in cases of severe immunodeficiency, which compensated for the lack of antibodies, improved opsonization, and reduced the risk of secondary infections. For more severe depletion of mucosal immunity (decreased sIgA), pentaglobin (5 ml/kg/day for 3 days), enriched with IgM and IgA, was used. As is known, this drug has a pronounced neutralizing effect on bacterial toxins and enhances the antimicrobial response.

It is important to note that immunoprophylaxis and immunocorrection are not opposed to each other. While immunocorrection is used in the presence of clinical and immunological signs of decompensation, immunoprophylaxis can be administered "one step earlier," that is, when initial deviations from AS without multiple organ failure are detected. In such cases, we used shortened GM-CSF regimens (125 mcg/m²), early administration of immunoglobulins, and dynamic monitoring of IL-6/IL-10, which in turn allowed us to intervene at a critical moment when the clinical picture was still reversible.

The development of the treatment and diagnostic algorithm was based on a combination of the prognostic risk model represented by the PREVAS scale and pathogenetically based immune therapy methods. Unlike traditional approaches focused on clinical manifestations and the SOFA score, the new algorithm allows for patient stratification even before the development of advanced MOF and tailors management to specific immunological disorders. The diagnostic and treatment algorithm we developed thus fulfills a triple function: prognosis, treatment, and prevention.

The treatment and diagnostic algorithm we developed is based on the "stepwise control" principle. At each stage, the risk level is recorded on the PREVAS scale, and immunological, clinical, and laboratory parameters are compared. Depending on the risk category, a set of measures was prescribed, either prophylactic approaches aimed at preventing the progression of immune disorders, or corrective measures applied when signs of MOF were present. After the selected treatment block, a reassessment of the parameters is mandatory, allowing for dynamic adjustments to the treatment plan and progression to the next level of intervention.

If the patient's parameters are within physiological norms and the PREVAS score does not exceed 3 points, there is no risk of MOF developing. For such women, standard therapy was sufficient, including antibiotics aimed at the identified or suspected pathogen, treatment of the infectious source, and infusion and anticoagulant support. At this stage, immunoprophylactic measures were not required, but dynamic monitoring was mandatory. Thus, after 72 hours, a reassessment of clinical, laboratory, and immunological parameters was performed.

If normal values were maintained, the patient remained in the "no risk" category. If the first abnormalities appeared, she was transferred to the "low risk" group. This approach avoided unnecessary interventions while maintaining the principle of rational and safe management.

A "low-risk" group of patients was formed when moderate immunological abnormalities were detected, including decreased HLA-DR expression to 60-67%, an increase in NLR to 3-5 units, and fluctuations in the IL-6/IL-10 ratio within 1.5-2 units. These indicators are known to be unaccompanied by significant organ dysfunction but do indicate a tendency toward immune dysregulation. In this situation, standard therapy was insufficient, so an immunoprophylactic regimen

was added. The most effective treatment was GM-CSF at a prophylactic dose of 125 mcg/m² subcutaneously once daily for 5 days, stimulating HLA-DR restoration and preventing further monocytic insufficiency. Additionally, a short course of intravenous immunoglobulin (IVIG 0.4 g/kg per day for 2 days) was administered to offset the initial signs of humoral depletion and increase resistance to bacterial load. IL-6 and IL-10 levels were also dynamically monitored to detect any potential transition to a higher risk category. A crucial element of this step of the algorithm was mandatory reassessment of these parameters after 72 hours.

The group of patients with a "high risk" of developing MOF due to AS included those who demonstrated significant immunological abnormalities, such as a drop in HLA-DR expression below 60%, a decrease in $CD4^+$ lymphocyte count to 28-33%, a decrease in the $CD4^+/CD8^+$ ratio to less than 1.3 units, an increase in NLR to 5-7 units, and an increase in the IL-6/IL-10 ratio above 2.5 units. These changes in these parameters directly indicated a breakdown in the coordination of the innate and adaptive responses, i.e., the critical stage when predicting MOF acquired real clinical significance. While maintaining standard therapy in these patients, the risk of decompensation reached its maximum, necessitating intensive immunoprophylaxis and, at the first signs of organ dysfunction, early immunocorrection.

At this stage, we administered full therapeutic doses of GM-CSF (250 mcg/m² subcutaneously once daily for 5 days), which facilitated accelerated restoration of HLA-DR and partially reversed the phenomenon of "immune paralysis." A standard course of immunoglobulins was additionally prescribed (IVIG 0.4 g/kg/day for 3 days), and if sIgA levels were significantly reduced, pentaglobin was added at a dose of 5 ml/kg/day for 3 days. Such measures not only enhanced opsonization and neutralization of bacterial toxins, but also provided additional protection in cases of weakened adaptive regulation.

Cytokine correction was particularly important in the high-risk group. Patients with critically elevated IL-6 levels were treated with tocilizumab (8 mg/kg intravenously, administered once), which blocked one of the central mediators of inflammatory progression. In cases with severe $CD4^+$ deficiency, recombinant IL-7 was used (10 μg/kg subcutaneously daily for 7 days). This drug stimulated T-helper cell proliferation and restored the coordinating function of the adaptive component.

Strict monitoring of parameters every 48 hours remained a mandatory component of this stage. If parameters stabilized and the risk gradually declined, the patient could be transferred to the "low-risk" category, with a subsequent reduction in the scope of immune interventions. If signs of increasing multiorgan dysfunction (increasing SOFA, worsening immune disorders) emerged during therapy, the patient was automatically classified as "critically risk," requiring the most aggressive immunocorrection regimen. These patients had immune profiles consistent with established MOF. The most characteristic signs included a drop in HLA-DR below 45%, a decrease in $CD4^+$ lymphocytes to less than 28% with a $CD4^+/CD8^+$ ratio of <1.0 units, hyperactivation of the monocyte component with a $CD14^+$ share of more than 22%, persistent growth of NLR (≥ 8 units), and a pronounced cytokine imbalance with an IL-6/IL-10 ratio exceeding 3 units.

The primary focus of treatment remained restoration of monocyte function. GM-CSF was administered at a standard dose of 250 μg/m² subcutaneously daily, which partially restored HLA-DR expression and reduced the level of immune paralysis. Recombinant IL-7 (10 μg/kg subcutaneously daily for 7 days) was used in combination, increasing the $CD4^+$ lymphocyte count and restoring the $CD4^+/CD8^+$ index. IVIG immunoglobulins (at a dose of 0.4 g/kg/day for 3 days) and/or pentaglobin (5 ml/kg/day × 3 days) were used as additional therapy, allowing us not only to compensate for the humoral deficiency but also to neutralize bacterial toxins. In cases of severe sIgA reduction, pentaglobin demonstrated the greatest efficacy.

Immunocorrection at this stage was always combined with a comprehensive set of intensive measures, including mechanical ventilation for respiratory failure, hemodialysis for acute renal failure, correction of coagulopathy with low-molecular-weight heparin and fresh frozen plasma, and vasopressor support for SS.

The treatment and diagnostic algorithm we developed combines three key components: the PREVAS prognostic scale, pathogenetically based immunoprophylaxis and immunocorrection measures, and a dynamic monitoring system that allows for timely adjustments to patient management.

Its distinctive feature is its stepwise structure, which begins with standard therapy in the absence of risk, progressing through early preventive interventions for moderate impairment, to active immunocorrection in the development of multiple organ failure secondary to AS. In each case, our treatment and diagnostic algorithm determines not only the set of treatment measures but also the order in which they are applied, taking into account monitoring results, ensuring a flexible and individualized approach. The treatment and diagnostic algorithm we presented allows us to link a patient's clinical and immunological profile to a specific physician decision, eliminating subjectivity and delayed interventions. Integrating immunological markers from the treatment and diagnostic algorithm into the treatment strategy system improves prognostic accuracy, accelerates the selection of treatment options, and helps reduce the risk of adverse outcomes. For example, we integrated the treatment and diagnostic algorithm we developed into an artificial intelligence module that automatically correlates PREVAS scores with risk categories and generates standardized recommendations for immunoprophylaxis or immunocorrection.

Conclusions

1. The treatment and diagnostic algorithm we developed combines three key components: the PREVAS prognostic scale, pathogenetically based immunoprophylaxis and immunocorrection measures, and a dynamic monitoring system that allows for timely adjustments to patient management strategies.

2. The treatment and diagnostic algorithm determine not only the set of treatment measures but also the order of their application, taking into account monitoring results, ensuring flexibility and individualization of the approach.

LIST OF REFERENCES:

1. Galkina DE, Makarenko TA, Ulyanova IO. Septic complications after abdominal delivery (literature review). Problems of Reproduction. 2024;30(6):108–117.
2. Kasymova ZN, Radzhabova MA, Saidova MM, et al. Purulent-inflammatory diseases in obstetrics. Mother and Child. 2020;(1):27–29.
3. Kenzhaeva ZO, Negmatullaeva MN, Tuksanova DI, Zaripova DYa. The role of some markers of the infectious process in early diagnosis and evaluation of the efficiency of antibacterial therapy for AS. Russian Bulletin of Obstetrician-Gynecologist. 2025;25(2):15–20.
4. Kulikov AV, Baranov II, Kuznetsova OV. Modern approaches to the treatment of postpartum purulent-septic diseases. Russian Bulletin of Obstetrician-Gynecologist. 2023;23(2):45–51.
5. Shalina RI, Serova OF, Kurbangaleeva EM. Maternal sepsis: modern concepts of diagnosis and treatment. Obstetrics and Gynecology. 2021;(6):15–20.
6. Andaluz-Ojeda D, Bobillo F, Iglesias V, et al. Early natural killer cell counts in blood predict outcome in severe sepsis. Crit Care. 2011;15(5):R243. doi:10.1186/cc10484.
7. Aronoff DM, Marrazzo JM. Infections caused by Clostridium perfringens and Paeniclostridium sordellii after unsafe abortion. Lancet Infect Dis. 2023;23:e48–e55. doi:10.1016/S1473-3099(22)00679-6.
8. Ashford and St Peter's NHS Foundation Trust. Maternal Sepsis Clinical Guideline. 2025. 27 p.
9. Bonet M, Brizuela V, Abalos E, et al. Implementing maternal sepsis bundles in tertiary hospitals: a prospective evaluation. Lancet Infect Dis. 2021;21(12):1663–1672. doi:10.1016/S1473-3099(21)00123-1.
10. Caldwell BA, Wu Y, Wang J, et al. Altered DNA methylation underlies monocyte dysregulation and immune exhaustion memory in sepsis. Cell Rep. 2024;43(3):113894. doi:10.1016/j.celrep.2023.113894.

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