

Prediction of Multiple Organ Failure in Obstetric Sepsis

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Abstract With the development of multiple organ dysfunction (MOD) associated with AS, the situation changes significantly, as it is during this period that a clear and reproducible set of indicators emerges, including SIRS levels, PCT and lactate concentrations, as well as hemostasis (D-dimer) and renal function (creatinine) indicators. These parameters, taken together, not only correlate with an increase in SOFA scores but also acquire clinical and prognostic significance. In practical terms, they form the basis of a laboratory diagnostic profile that allows for the prediction of further progression of organ dysfunction, which is why they were chosen as the basis for constructing a prognostic model.

Keywords Obstetric sepsis, Multiple organ failure, Prognosis

1. Introduction

Obstetric sepsis (OS) remains one of the leading causes of maternal mortality worldwide, despite advances in antimicrobial therapy and intensive care [1,3,5]. Septic conditions account for up to 10.7% of all maternal deaths, highlighting the critical need to improve the diagnosis and treatment of this condition [2,4,6].

The development of multiple organ failure (MOF) in OS requires special study. It has now been established that not only hyperinflammation but also the development of subsequent "immunoparalysis" are critical in sepsis, contributing to the chronicity of inflammation and multiple organ dysfunction.

The characteristics of the inflammatory response in OS in the context of MOF development remain poorly understood [7,9].

Systemic neutrophil activation, accompanied by NETosis (the formation of extracellular neutrophil traps), plays a key role in the pathogenesis of septic MOF. The work of V. Brinkmann et al. (2022) showed that excessive activation of NETs in sepsis contributes to tissue damage, microvascular thrombosis, and the development of multiple organ dysfunction.

The molecular mechanisms of septic multiple organ dysfunction include activation of Toll-like receptors, dysregulation of cytokine networks, and activation of apoptotic cascades in immune cells. This is supported by data from R. Medzhitov (2018), which emphasizes the importance of an imbalance of proinflammatory and anti-inflammatory mediators in the pathogenesis of septic complications.

Early diagnosis of immune disorders in sepsis, including assessment of HLA-DR expression on monocytes, levels of proinflammatory cytokines (IL-6, TNF- α), and endothelial activation biomarkers (sTREM-1), is considered a promising approach to predicting the development of multiple organ dysfunction. This is supported by the results of a study by D. Andaluz-Ojeda et al. (2021).

Thus, the problem of multiple organ failure in obstetric sepsis requires a thorough study of the molecular and cellular mechanisms of immune dysregulation to develop effective strategies for diagnosis and correction of the immune response, which could significantly reduce maternal mortality and improve clinical outcomes [2,4,6,8,10].

Study objective: to develop methods for predicting the risk of multiple organ failure in obstetric sepsis.

2. 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 material allowed us to create a sample that reflected real-world clinical practice in different regions of our country and created the conditions for organizing a representative study.

A total of 250 women in the postpartum period were included in the study, divided into 3 groups according to the objectives:

- The reference group (50 patients) consisted of apparently healthy women after uncomplicated births who were undergoing routine observation in the postpartum ward and had no signs of infectious or inflammatory complications. This group of patients was

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used to determine conditionally physiologically normal immune status indicators in the postpartum period and served as a control background for interpreting immunological changes in patients with AS;

- The control group (100 patients) included patients with AS who received standard treatment methods in accordance with current clinical protocols approved by the Ministry of Health of the Republic of Uzbekistan, without the use of the treatment and diagnostic algorithm we developed for predicting and preventing multiple organ failure.
- The main group (100 patients) consisted of patients with AS who, in addition to standard therapy, received our developed treatment and diagnostic algorithm for predicting and preventing multiple organ failure.

An analysis of the demographic distribution of patients revealed that the largest number of women from the Bukhara region, comprising 98 (39.2%) of the total sample, were from the Bukhara region. However, despite the large number of patients in this region, it still did not represent even half of the entire study population.

The study's AS diagnosis was based on the international Sepsis-3 definitions, which define sepsis as life-threatening organ damage resulting from a dysregulated response to infection.

The SOFA score was used to quantitatively stratify the severity of the condition, providing an objective measure of organ dysfunction. An increase of ≥ 2 points was used as a diagnostic criterion for sepsis and was used for follow-up.

Clinical diagnostics included signs of SIRS such as a temperature above 38 °C or hypothermia below 36 °C, tachycardia above 90 bpm, tachypnea greater than 20 breaths per minute, altered consciousness, as well as manifestations of hypotension and tissue hypoperfusion. These parameters were used as screening parameters, while final stratification was performed using the SOFA score.

3. Results and Discussion

Women with AS without signs of MOD were more likely to have vaginal deliveries, while in the group with MOD and AS, the rate was more than 2.5 times lower, reflecting the influence of the obstetric scenario itself. That is, during a natural birth, the infectious process was limited primarily to local manifestations, without progressing to the stage of systemic dysfunction. A completely different trend was observed with regard to emergency cesarean sections. In the group with MOD, they occurred 3.7 times more often ($p=0.0001$) than in women without organ failure, which was confirmed by a statistically significant correlation. In clinical practice, the following observation of a 32-year-old patient can be described as an example. She underwent surgery for chorioamnionitis and labor weakness. On the first day after the procedure, her condition remained satisfactory, but by the third day, signs of respiratory failure developed, coagulation changes were noted, and lactate levels increased.

A decrease in HLA-DR expression and an increase in SOFA scores were simultaneously observed, indicating the progression of the infection to multiple organ dysfunction.

This case demonstrates how emergency surgical intervention can accelerate the progression of a septic process in an unfavorable immunological environment.

Induction of labor was observed 1.6 times more frequently in women with multiple septal failure than in the uncomplicated group, and cervical suturing was more than 3.3 times more common. Although the statistical significance of these differences was limited ($p=0.531$ and $p=0.724$, respectively), the trend was clear. The sample included a 28-year-old patient whose pregnancy was accompanied by cervical insufficiency. After delivery, she developed endometritis followed by fever and tachycardia, and on the third day, signs of initial renal dysfunction appeared. This case demonstrates that even relatively rare obstetric interventions, when the pregnancy course is unfavorable, can exacerbate the septic cascade and increase the likelihood of organ dysfunction.

Elective cesarean sections were distributed almost equally in both groups, highlighting the lack of a direct link between the fact of operative delivery and the severity of the septic process. In the context of a stable maternal condition and standard antibacterial prophylaxis, the planned intervention did not become an independent risk factor for MOF.

The dynamics of clinical and laboratory parameters in patients with AS without MOF had distinct characteristics. Thus, as early as the first day of illness, parameters indicated systemic inflammation and metabolic destabilization.

We detected an increase in PCT levels, recorded an increase in blood lactate concentrations, an increase in D-dimer, and an increase in creatinine. These changes were accompanied by pronounced clinical manifestations, reflected in an average SIRS level exceeding the physiological baseline by more than 9 times. This combination of abnormalities corresponded to the early phase of a septic reaction but had not yet been accompanied by the development of multiple organ dysfunction. However, a tendency toward normalization of most parameters was observed subsequently.

As early as the third day, PCT decreased more than twofold, and lactate levels decreased by approximately one-third compared to the initial values. At the same time, partial platelet recovery and stabilization of hemostasis parameters occurred, as evidenced by a decrease in the INR and a return of fibrinogen to values close to reference values. The clinical picture also became less pronounced, as reflected by a decrease in the SIRS score.

On the seventh day, the dynamics were even more favorable, with most parameters reaching values that were statistically indistinguishable from reference ranges. PCT concentrations approached the baseline level in healthy women, lactate decreased to 1.3 ± 0.1 mmol/L, and creatinine and bilirubin levels returned to the reference range.

In women with AS complicated by multiple organ failure, significant changes in all clinical and laboratory parameters were observed as early as the first day of illness. A high SIRS level reflected not simply the presence of a systemic

inflammatory response, but its maximum intensity. Average values for this indicator exceeded the physiological range by almost 20 times, and even by the seventh day, it was not possible to reduce the level below 2.8 points. This level of values indicated that the clinical manifestations of systemic inflammation remained persistent and were poorly responsive to standard therapy. For comparison, in the group without multiple sclerosis, a clear trend toward normalization of SIRS was observed as early as day 3, highlighting the fundamental difference in dynamics.

PCT demonstrated a similarly unfavorable picture. Its concentration at the onset of the disease exceeded normal values by more than 100 times and remained high even by the end of the first week of observation, remaining at an average level of 5.1 ± 1.3 ng/ml. A lack of decrease in PCT levels serves as a reliable indicator of ongoing systemic infection and correlates with a poor prognosis. In our sample, the case of a 29-year-old patient whose delivery ended in an emergency cesarean section due to chorioamnionitis is illustrative. Already on the first postoperative day, her PCT level exceeded 6 ng/ml and did not decrease over the following days, despite extensive antibiotic therapy. Against the background of persistent systemic inflammation, the woman developed progressive multiorgan dysfunction, ultimately requiring long-term vasopressor support. The lactate dynamics were no less revealing. In patients with multiple organ failure, its concentration on the first day was almost four times higher than normal and remained within the abnormal range even on the seventh day, decreasing only partially.

The described changes were likely associated with persistent tissue hypoperfusion and metabolic destabilization. One clinical case showed a 35-year-old female patient admitted in critical condition after childbirth with rupture of membranes and endometritis. Upon admission, lactate was 4.8 mmol/L, decreased only to 4.1 mmol/L on the third day, and remained above 3.5 mmol/L by the seventh day. Even with partial improvement, persistent hyperlactacidemia reflected profound microcirculatory disturbances that underlay the development and progression of multiple organ failure. Hemostasis parameters, however, deserve special attention.

We observed thrombocytopenia and a sharp increase in D-dimer levels in women with multiple organ failure secondary to AS. The latter was more than five times higher than normal and remained virtually unchanged throughout the first week, indicating the development of severe coagulopathy with activated fibrinolysis and a high risk of developing DIC. Combined with elevated INR and persistently high fibrinogen levels, a hemostatic disorder characteristic of sepsis developed. Coagulation factors were mobilized and subsequently depleted. Clinically, this was manifested by frequent bleeding from invasive sites, hemorrhagic impregnation of postoperative sutures, and instability of laboratory parameters.

Renal and hepatic dysfunction were confirmed by increased creatinine and bilirubin. Creatinine levels, on average, exceeded normal values by more than two times and

remained elevated throughout the week, consistent with acute renal failure with a marked decrease in urine output. Bilirubin also remained elevated, although it showed a slight positive trend (from 38 ± 12 $\mu\text{mol/L}$ to 34 ± 10 $\mu\text{mol/L}$).

This trend demonstrates that even with partial compensation, liver function did not return to physiological levels. In practice, these laboratory changes were accompanied by increasing uremia, hyperkalemia, and signs of encephalopathy.

Thus, unlike the group of patients with AS without MOD, where most parameters returned to normal by day 7, in patients with complicated AS, clinical and laboratory parameters showed persistent abnormalities.

The performed correlation analysis allowed us to quantitatively evaluate the relationship between the severity of MOD, expressed as SOFA scores, and the main clinical and laboratory parameters in patients with AS. This approach is crucial, as many parameters themselves reflect only individual aspects of the pathological process (inflammation, metabolic disorders, or organ dysfunction). Only their statistical correlation with the integrated severity scale allows us to speak of their true prognostic value. This was the main goal of the analysis, which was built around identifying from the many parameters those that truly accompany the progression of organ dysfunction.

In the group of women with AS without MOD, the identified associations were relatively weak.

In particular, PCT levels had a correlation coefficient of $r=0.298$ ($p=0.022$), while lactate had a correlation coefficient of $r=0.334$ ($p=0.012$). These values indicate only a moderate correlation between increases in these markers and increases in SOFA scores. In fact, laboratory changes were recorded in these patients, but they were not always accompanied by an increase in clinical severity. CRP demonstrated an even weaker correlation ($r=0.226$; $p=0.071$), platelets had a negative but non-significant correlation ($r=-0.214$; $p=0.086$), and creatinine had a correlation coefficient of $r=0.268$ ($p=0.038$).

Thus, in most cases of AS without MOF, laboratory changes did not directly reflect a critical condition, but could rather be considered "signals" of inflammation that tend to normalize during therapy. A completely different pattern of relationships was observed in patients with MOF against the background of AS. Here, virtually every indicator demonstrated a high degree of association with the severity of the condition. Particularly impressive were the data on the lactate level, where the coefficient was $r = 0.842$ ($p < 0.001$). This result essentially confirms the key role of tissue hypoperfusion and metabolic destabilization in the progression of AS. PCT had no less convincing indicators ($r = 0.785$; $p < 0.001$), which reflects a stable relationship between the severity of systemic bacterial aggression and the development of MOF. D-dimer was closely associated with SOFA ($r = 0.804$; $p < 0.001$), which is consistent with the known role of disseminated hemostatic disorders in the clinical picture of the septic process. In turn, creatinine ($r = 0.766$; $p < 0.001$) demonstrated the significance of renal dysfunction as one of the leading components of MOF.

Finally, the total SIRS level had $r=0.812$ ($p<0.001$), which highlights the high value of clinical assessment of the systemic inflammatory response in predicting severe outcomes.

Interestingly, other parameters, usually considered secondary, demonstrated convincing correlations in the context of MOF. For example, total bilirubin correlated with SOFA at $r=0.731$ ($p<0.001$), urea at $r=0.695$ ($p<0.001$), and INR at $r=0.602$ ($p<0.001$). Even markers such as CRP demonstrated significant association ($r=0.564$; $p<0.001$). However, the strength of these correlations was lower than that of the "five" key determinants, which allows us to consider them more as additional than as leading prognostic indicators. This can be illustrated by the observation of a 34-year-old patient who developed AS after an emergency cesarean section. On the first day, her lactate level was 4.8 mmol/L, PCT exceeded 6 ng/mL, and D-dimer was above 1600 ng/mL. On the third day, these values remained virtually unchanged, and by the seventh day, creatinine increased from 160 to 175 $\mu\text{mol/L}$. All these changes were accompanied by an increase in the SOFA score to 9 points, confirming critical multiple organ failure.

This case demonstrates that these parameters reflect the actual progression of the condition and are reliable indicators of an unfavorable outcome.

Thus, the results of the correlation analysis showed that in AS without multiple organ failure, clinical and laboratory parameters reflect only fragmentary changes and do not demonstrate a consistent relationship with the severity of the condition. With the development of MOF in the setting of AS, the situation changes significantly, as it is during this period that a clear and reproducible set of indicators is identified, including SIRS levels, PCT and lactate concentrations, as well as hemostasis (D-dimer) and renal function (creatinine) indicators. These parameters, taken together, not only correlate with an increase in SOFA scores but also acquire clinical and prognostic significance. In practical terms, they form the basis for a laboratory diagnostic profile that allows for the prediction of further progression of organ dysfunction, which is why they were chosen as the basis for constructing a prognostic model.

4. Conclusions

1. With the development of MOF in the setting of AS, the situation changes significantly, as it is during this period that a clear and reproducible set of indicators is identified, including SIRS levels, PCT and lactate

concentrations, as well as hemostasis (D-dimer) and renal function (creatinine) indicators.

2. In practical terms, they form the framework of a laboratory diagnostic profile that allows for the prediction of further progression of organ dysfunction, and for this reason they were chosen as the basis for constructing a prognostic model.

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