Obstetrics and Gynecology
Акушерство и гинекология

UDC 618.24:616.98

CARACTERISTICS OF THE SYSTEMIC INFLAMATORY RESPONSE AND ADAPTATIVE MECHANISMS IN PUERPERAL INFECTIOUS

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Abstract. *Postpartum infectious complications are a major cause of maternal morbidity and may progress to sepsis. This study evaluated 186 women, comparing conventional therapy (82) with additional adaptive-support interventions (104). Endometritis was most frequent. Patients with complications showed increased proinflammatory cytokines, endotoxinemia, immunosuppression, and neuroendocrine disturbances. The integrative therapy improved outcomes.*

Keywords: *postpartum infectious complications, systemic inflammatory response syndrome, cytokines, endotoxinemia, immunosuppression.*

Introduction. Puerperal infectious continue to represent a major global public health concern, ranking among the leading causes of severe maternal morbidity and preventable maternal deaths [15, 17]. Maternal sepsis remains one of the principal direct causes of maternal mortality worldwide, with puerperal infections constituting a substantial component of this pathological spectrum [13].

Despite advances in antibiotic prophylaxis, refinements in surgical technique, and improvements in contemporary obstetric care, postpartum infections persist as a significant contributor to maternal morbidity, particularly in the context of rising caesarean section rates [2, 17].

Postpartum infections encompass a broad spectrum of clinical entities - puerperal endometritis, surgical site infections, mastitis, pelvic peritonitis, and obstetric sepsis - all underpinned by complex pathogenetic mechanisms involving the interplay between microbial factors, the anatomical and physiological particularities of the puerperal period, and the patient's immune status [7, 15].

The postpartum period is characterized by the presence of an extensive uterine wound surface following placental separation, associated with decidual necrosis, accumulation of lochia, and alterations in local vascularization. These conditions create a favorable environment for bacterial colonization and proliferation [15].

According to the data presented by Stephens and Barton, the principal pathogenetic mechanism underlying postpartum endometritis is the ascending spread of polymicrobial vaginal flora into the uterine cavity, particularly in situations where mechanical barriers are compromised (premature rupture of membranes, prolonged labour, repeated intrauterine manipulations) [15].

The microbiology of puerperal infections is typically polymicrobial, involving both aerobic and anaerobic bacteria - Streptococcus spp., Staphylococcus spp., members of the Enterobacteriaceae family, and Bacteroides spp. - with synergistic interactions among these microorganisms potentiating both the local and systemic inflammatory response [7, 16].

Tita and Andrews emphasize that the presence of anaerobic flora is associated with more severe clinical presentations, owing to their enhanced capacity for tissue invasion and toxin production [16].

Caesarean section constitutes the most significant risk factor for the development of postpartum infectious complications. Recent evaluations indicate that the risk of endometritis is 5–20 times higher following caesarean delivery compared with vaginal birth [2, 17]. Al-Khayat and colleagues highlight that surgical intervention results in additional tissue trauma, hematoma formation, and the creation of dead space, together with impairment of local defense mechanisms, thereby facilitating bacterial colonization and subsequent dissemination of infection [2].

In clinical practice, postpartum complications may evolve from localized infections to severe systemic involvement. Mihalcean and colleagues demonstrated a direct pathogenetic correlation between multiple perinatal infections and the development of severe sepsis associated with multiple organ dysfunction syndrome (MODS), underscoring the progressive nature of the systemic inflammatory cascade [10]. This pathogenetic model confirms that a localized infection may rapidly progress to a systemic process in the absence of timely therapeutic intervention.

Beyond local factors, the pathogenesis of postpartum infectious complications is closely linked to the specific characteristics of the maternal immune response. Pregnancy is characterized by a distinct immunological adaptation aimed at promoting fetal tolerance, with a predominance of humoral immunity and relative attenuation of cell-mediated immune reactivity [8]. Mor and Cárdenas describe the postpartum period as a phase of immunological readjustment, during which transient imbalances in host defense mechanisms may occur, thereby increasing susceptibility to infection [11].

Pro-inflammatory cytokines play a central role in pathogenesis, particularly interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), which mediate the systemic inflammatory response [14]. Rudd and colleagues emphasize that excessive activation of these mediators may precipitate systemic inflammatory response syndrome (SIRS) and subsequently obstetric sepsis [14].

Bacterial endotoxins - especially lipopolysaccharides derived from Gram-negative organisms - are pivotal in triggering macrophage and endothelial cell activation, leading to a massive release of inflammatory mediators, complement activation, and endothelial dysfunction [10]. Singer and colleagues demonstrated that this systemic activation may result in increased vascular permeability, hypotension, coagulopathy, and multiple organ dysfunction - hallmark features of sepsis [3].

In the obstetric context, Bauer and colleagues demonstrated that the physiological hemodynamic changes of pregnancy and the postpartum period may mask the early manifestations of sepsis, thereby delay diagnosis and adversely affecting prognosis [9]. The clinical cases reported by Mihalcean and colleagues further corroborate this observation, describing the evolution of severe postpartum sepsis secondary to a deep vaginal hematoma, in which the infectious process progressed rapidly and required complex multidisciplinary management [5]. These findings underscore the critical importance of early recognition of occult infectious foci during the postpartum period.

Another important element is the role of vaginal and uterine microbiota. Chen and colleagues demonstrated that vaginal dysbiosis and colonization with antibiotic-resistant strains may disrupt the local ecological balance and increase the risk of persistent or recurrent postpartum infections [1]. Furthermore, coinfections and complex microbial interactions may potentiate the inflammatory response and complicate the clinical course.

Maternal comorbidities significantly influence pathogenesis. Diabetes mellitus, obesity, anemia, and preeclampsia are associated with endothelial dysfunction, impaired microcirculation, and reduced phagocytic capacity, thereby increasing both susceptibility to infection and disease severity [9, 13]. Furthermore, obesity is correlated with a chronic pro-inflammatory state, which may amplify the inflammatory response in the setting of a postpartum infection [14].

Regarding therapeutic management, several authors have highlighted the importance of organ-preserving surgical approaches in women presenting with puerperal complications following caesarean section, emphasizing the need for an individualized strategy aimed at maintaining reproductive function whenever feasible [8, 12].

The imbalance between pro-inflammatory and anti-inflammatory mechanisms represents a critical turning point during postpartum infectious complications. Several authors describe a phase of secondary immunosuppression that may follow an intense inflammatory activation, thereby increasing the risk of secondary infections and a protracted clinical course. In obstetrics, this phenomenon may account for the persistence of infection or the occurrence of late complications [4, 8].

Therefore, the pathogenesis of postpartum infectious complications must be approached in an integrative manner, encompassing the interplay between microbial factors, postpartum tissue changes, the specific features of the maternal immune response, endotoxin-mediated activation, and the influence of maternal comorbidities. A comprehensive understanding of these mechanisms is essential for the development of modern therapeutic strategies aimed not only at eradicating the infectious agent, but also at modulating the inflammatory response and preventing progression to sepsis and multiple organ dysfunction.

Collectively, these considerations indicate that current concepts regarding the pathogenesis and mechanisms underlying the development of inflammatory processes in puerperal women warrant further in-depth investigation to establish an optimal therapeutic framework. This constituted the primary objective of our research.

Aim of the study: to analyze the development of postpartum infectious complications not solely as a progression of infection, but also from the perspective of the host response, as a clinical expression of systemic inflammatory response syndrome (SIRS).

Materials and Methods. The study was designed as a comparative clinical investigation evaluating the effectiveness of therapeutic interventions administered to patients with postpartum infectious complications. The research aimed to assess the impact of incorporating additional therapeutic measures intended to enhance the organism's adaptive capacity, in comparison with standard conventional treatment.

Study Population. A total of 186 puerperal women diagnosed with postpartum infectious complications were enrolled in the study. The patients were allocated into two groups: comparison group – 82 patients who received exclusively conventional treatment, in accordance with standard therapeutic protocols applied in postpartum infectious pathology and main group – 104 patients in whom, in addition to the standard therapeutic regimen, adjunctive measures aimed at enhancing the organism's adaptive capacity were incorporated.

From a socio-biological perspective (age, area of residence, obstetric status) and a clinical standpoint (type and severity of infectious complications, timing of symptom onset), the two groups were comparable, with no statistically significant differences identified at baseline evaluation.

Research Methods. Patient assessment included comprehensive general and obstetric clinical examination, standard paraclinical investigations (hematological and biochemical analyses, inflammatory markers), evaluation of clinical evolution under treatment, monitoring of complications and duration of hospitalization.

Therapeutic efficacy was assessed by comparing the dynamics of clinical and paraclinical parameters between the two groups, as well as by evaluating the duration of symptom resolution and the frequency of potential complications. Statistical data processing was performed using standard statistical methods, with determination of the significance of intergroup differences.

Results. The most frequent postpartum complication was endometritis (86.2% in the main group and 81.4% in the comparison group), followed by perineal wound infection. The control group consisted of 48 puerperal women with a physiological course of the postpartum period. In addition to standard clinical investigations, all patients underwent specialized assessments, including measurement of serum pro-inflammatory cytokines (IL-1, IL-6, TNF) using solid-phase enzyme-linked immunosorbent assay (ELISA), detection and quantification of endotoxin by the activated particle method, determination of total serum protein and protein fractions, as well as C-reactive protein levels. Immune status was evaluated based on immunogram reflecting both humoral and cellular immunity, together with assessment of complement component C5. The leukocytic intoxication index (LII) was calculated according to the I. I. Kalf-Kalif formula.

Neuroendocrine regulatory status was assessed by electroencephalography and by measuring serum levels of ACTH, prolactin, cortisol, estrogens, and progesterone.

The obtained results demonstrated a significant increase in serum concentrations of pro-inflammatory cytokines in affected patients compared with healthy puerperal women. Particularly notable was the elevation of TNF, whose concentration increased 2.5-fold.

Endotoxin was detected in minimal concentrations in only 4 (14.2%) clinically healthy puerperal women, whereas in most patients (85.6%) with a complicated postpartum course, endotoxin levels reached activation grades II–III (30–125 IU/mL). The presence of endotoxemia was associated with an increased leukocytic intoxication index (LII) and elevated C-reactive protein levels.

Immunogram parameters revealed a reduction in immune defense even among clinically healthy puerperal women, while the development of infectious complications was accompanied by a pronounced aggravation of immunodepression.

Electroencephalographic changes and alterations in hormonal levels indicated disturbances in neuroendocrine regulation of a compensatory nature, with the most significant changes observed in prolactin and cortisol concentrations.

Discussion. The findings of our study confirm the complex and multifactorial nature of postpartum infectious complications, supporting the contemporary concept that these conditions should be analyzed not solely as an expression of microbial aggression, but also as a manifestation of a systemic host response involving inflammatory, immune, and neuroendocrine mechanisms.

The high prevalence of postpartum endometritis observed in both groups (over 80%) is consistent with data reported in the literature, which identify this entity as the most common form of puerperal infection [7, 15]. The mechanism of ascending polymicrobial vaginal flora into the uterine cavity, as described by Stephens and Barton, accounts for the high incidence of this pathology, particularly in the setting of disrupted anatomical barriers and the presence of an extensive uterine wound surface following placental separation. The polymicrobial nature of postpartum infections, emphasized by Tita and Andrews, provides a pathophysiological explanation for the intensity of the systemic inflammatory response observed in our study [15, 16].

The significant elevation in serum concentrations of pro-inflammatory cytokines (IL-1, IL-6, TNF-α), with a 2.5-fold increase in TNF levels, confirms intense activation of the systemic inflammatory cascade. These findings are consistent with the pathogenetic model described by Rudd and colleagues [14], which highlights the central role of cytokines in triggering systemic inflammatory response syndrome (SIRS).

Concurrently, the elevated endotoxin levels detected in most patients (85.6%) support the involvement of endotoxin-mediated mechanisms in pathogenesis. This observation aligns with the findings of Singer and colleagues [6, 7], who demonstrated that lipopolysaccharides derived from Gram-negative bacteria may initiate an amplified systemic inflammatory response, with the potential to progress to sepsis and multiple organ dysfunction.

The presence of endotoxemia, associated with an increased leukocytic intoxication index and elevated C-reactive protein levels, confirms the existence of an active systemic inflammatory response. These findings support the progressive model described by Mihalcean and colleagues, according to which a localized postpartum infection may rapidly evolve into severe systemic forms in the absence of effective control of the inflammatory process [10].

A particularly important aspect highlighted by our study is the aggravation of immunodepression in the context of infectious complications. While clinically healthy puerperal women already exhibited a degree of reduced immune reactivity—an observation that may be explained by the postpartum immunological readjustment described by Mor and Cárdenas—this immunodepression was markedly more pronounced in patients with a complicated clinical course [11]. These findings support the hypothesis of a secondary immunosuppressive phase following the initial inflammatory activation, a phenomenon described in recent literature, which may account for the persistence or progression of the infectious process [4, 8].

The neuroendocrine disturbances identified through EEG changes and significant variations in prolactin and cortisol levels confirm the interdependence between the immune and neuroendocrine systems. Cortisol, known for its immunomodulatory effects, may further exacerbate immunosuppression in the context of an intense systemic inflammatory response.

These observations are consistent with the findings of Bauer and colleagues, who emphasize that the physiological hemodynamic and hormonal particularities of the postpartum period may influence the clinical presentation and potentially mask the onset of severe complications [9].

The role of endotoxin and complement activation (C5) identified in our study supports the involvement of endothelial dysfunction and complement system activation in the progression of complications, a mechanism described by Singer and colleagues [3]. In this context, it becomes evident that mere eradication of the causative pathogen is not always sufficient to halt the inflammatory cascade; modulation of the host response is likewise required.

By incorporating adjunctive measures aimed at enhancing the organism's adaptive capacity in the main group, our study addresses the need, highlighted in literature, for an integrative therapeutic approach. The data reported by Al-Khayat and colleagues regarding the impact of surgical interventions on tissue integrity, together with observations on the role of maternal comorbidities, support the concept that maternal vulnerability results from a complex interplay between local and systemic factors [2, 9, 13].

Overall, the findings of our research confirm that the development of postpartum infectious complications cannot be explained solely by the presence of a microbial pathogen. Rather, they reflect a profound imbalance between pro-inflammatory and anti-inflammatory mechanisms, between immune activation and the subsequent phase of secondary immunosuppression, as well as a significant involvement of the neuroendocrine system.

Thus, the data obtained supports the contemporary concept outlined in the Introduction, according to which the management of postpartum infectious complications should be directed not only toward infection control, but also toward modulation of the inflammatory response and support of the organism's adaptive mechanisms. Such an approach may contribute to preventing obstetric sepsis and multiple organ dysfunction syndrome, thereby improving maternal prognosis.

Conclusions. The results of the conducted research demonstrate the involvement of all major regulatory systems of the puerperal organism in response to the invasion of an infectious agent and allow postpartum complications to be interpreted as a manifestation of the early stage of systemic inflammatory response syndrome (SIRS).

Approaching the pathogenesis of postpartum infectious complications from the perspective of SIRS enabled us to develop a therapeutic complex which, while maintaining adequate antimicrobial therapy, placed particular emphasis on modulation of the regulatory systems of the puerperal organism. Clinical validation of this therapeutic approach demonstrated superior efficacy compared with conventional treatment modalities.

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Материал поступил в редакцию 25.02.26

ХАРАКТЕРИСТИКА СИСТЕМНОГО ВОСПАЛИТЕЛЬНОГО ОТВЕТА И АДАПТАЦИОННЫХ МЕХАНИЗМОВ ПРИ ПОСЛЕРОДОВЫХ ИНФЕКЦИОННЫХ ОСЛОЖНЕНИЯХ

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Аннотация. *Инфекционные осложнения в послеродовом периоде являются одной из основных причин материнской заболеваемости и могут прогрессировать до сепсиса. В исследовании оценивались 186 женщин, сравнивались конвенциональная терапия (82) и дополнительное вмешательство, направленное на поддержку адаптационных механизмов (104). Наиболее частой был эндометрит. У пациенток с осложнениями выявлены повышенные уровни провоспалительных цитокинов, эндотоксинемия, иммуносупрессия и нарушения нейроэндокринной регуляции. Интегративная терапия улучшила клинические исходы.*

Ключевые слова: *послеродовые инфекционные осложнения, синдром системного воспалительного ответа, цитокины, эндотоксинемия, иммуносупрессия.*