INTERNATIONAL JOURNAL OF MEDICAL SCIENCE AND PUBLIC HEALTH RESEARCH
IJMSPHR
EST 2020

TYPE Original Research
PAGE NO. 7-11
DOI 10.37547/ijmsphr/Volume07Issue01-02

Cytokine Dysregulation As A Key Mechanism Of Dic Syndrome Formation In Children With Severe Pneumonia: Clinical And Immunological Analysis

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OPEN ACCESS

SUBMITTED 12 November 2025
ACCEPTED 05 December 2025
PUBLISHED 07 January 2026
VOLUME Vol.07 Issue01 2026

CITATION

Mirrahimova Maktuba Khabibullayevna, Tashmatova Gulnoza A'loevna, & Tangriberdiev Abdulla Rayimberdiyevich. (2026). Cytokine Dysregulation As A Key Mechanism Of Dic Syndrome Formation In Children With Severe Pneumonia: Clinical And Immunological Analysis. International Journal of Medical Science and Public Health Research, 7(01), 7–11. https://doi.org/10.37547/ijmsphr/Volume07Issue01-02

Abstract: The aim of the study: To determine the clinical and immunological characteristics and elucidate the role of cytokine dysregulation in the development of DIC syndrome in children aged 1–3 years with severe pneumonia in order to improve early diagnosis and prognosis of complications.

Materials and methods. The study was conducted in 2024–2025 at the Intensive Care and Pediatric Pulmonology Department of the Multidisciplinary Clinic of TSMU. Thirty-nine children aged 1–3 years diagnosed with severe pneumonia were observed. To elucidate the role of cytokine dysregulation in the development of DIC syndrome, the patients were divided into two groups. The first group consisted of 17 children (43.6%) who developed DIC syndrome against a background of severe pneumonia. Among them, there were 10 boys (58.8%) and 7 girls (41.2%). The second group consisted of 22 children (56.4%) who did not have hemostasis disorders. Of these, 12 were boys (54.5%) and 10 were girls (45.5%).

Results and discussion. The analysis of the examined children revealed significant differences in clinical and laboratory parameters between the group with DIC and patients without coagulopathic complications. Already

at admission, children with DIC demonstrated a more pronounced inflammatory response: their C-reactive protein levels averaged 118.4 mg/L (95% CI: 103–132 mg/L), which was statistically significantly higher than those in the second group, where the average values were 62.7 mg/L (95% CI: 52–73 mg/L; $p < 0.01$).

Conclusions. In children aged 1–3 years with severe pneumonia, the development of DIC syndrome is accompanied by more pronounced systemic inflammation, as evidenced by significantly elevated levels of C-reactive protein and procalcitonin compared to patients without DIC. The development of DIC is associated with pronounced cytokine dysregulation: IL-6 concentrations in children with DIC were more than twice as high, while IL-10 levels were significantly lower, indicating an insufficient anti-inflammatory response.

Keywords: Children, severe pneumonia, DIC syndrome, cytokines, TNF-α, IL-6, IL-8, IL-10, immune regulation, hemostasis.

1. Introduction: Severe pneumonia in young children (aged 1–3 years) remains one of the most significant challenges in modern pediatrics. At this age, children have functional immaturity of the immune, respiratory, and hemostatic systems, which increases the risk of rapid infection progression and complications. One of the most dangerous and life-threatening complications is disseminated intravascular coagulation (DIC), which develops rapidly in young children and is characterized by severe microcirculatory impairment, thrombus formation, and subsequent consumptive coagulopathy [1, 4].

Studying the immunological mechanisms underlying the development of DIC is particularly important, as children aged 1–3 years exhibit different immune system reactivity compared to older age groups. This period is characterized by the development of an imbalance between proinflammatory and anti-inflammatory cytokines (IL-1β, IL-6, TNF-α, IL-10, etc.), which determines the severity of the inflammatory response in pneumonia. Hyperproduction of proinflammatory cytokines can trigger a cascade of coagulation activation, endothelial dysfunction, and microcirculatory disorders, creating the conditions for the development of DIC early in the disease [6, 10]. At the same time, insufficient production of regulatory cytokines in young children limits the body's ability to control excessive inflammation, increasing the risk of immune response decompensation. In the setting of severe pneumonia, a cytokine storm becomes a critical factor in vascular endothelial damage, platelet activation, and impaired fibrinolysis. Therefore, the study of cytokine regulation in children aged 1–3 years is of particular scientific and practical importance [2, 3, 7].

Despite the active study of the mechanisms of DIC syndrome in general pediatrics, data on the pathogenetic role of cytokines in early childhood remain limited. Prognostic markers reflecting the transition from compensated forms of hemostatic disorders to decompensation remain insufficiently defined, and the clinical and immunological features of severe pneumonia in young children have not been fully studied [5, 8, 9]. Thus, research into cytokine dysregulation as a key mechanism for the development of DIC in children aged 1–3 years is a relevant and in-demand scientific area. The results obtained could improve early diagnosis, enhance treatment effectiveness, optimize prognosis, and reduce the risk of severe complications and mortality in this age group.

Purpose of the research

To determine the clinical and immunological characteristics and elucidate the role of cytokine dysregulation in the development of DIC syndrome in children aged 1–3 years with severe pneumonia, in order to improve early diagnosis and prognosis of complications.

2. Methods

The study was conducted in 2024–2025 in the Intensive Care and Pediatric Pulmonology Department of the Multidisciplinary Clinic of TSMU. Thirty-nine children aged 1–3 years diagnosed with severe pneumonia were observed. To elucidate the role of cytokine dysregulation in the development of DIC syndrome, patients were divided into two groups.

The first group consisted of 17 children (43.6%) who developed DIC syndrome against a background of severe pneumonia. Among them were 10 boys (58.8%) and 7 girls (41.2%). The second group consisted of 22 children (56.4%) who did not have hemostasis disorders. Of these, 12 were boys (54.5%) and 10 were girls (45.5%). Thus, the gender distribution in both groups was comparable, although a higher proportion of boys was observed in the group with a complicated course.

Clinical and laboratory examination included an assessment of the severity of respiratory failure, the severity of intoxication syndrome, respiratory rate, oxygen saturation, and radiographic signs of lung

damage. Laboratory parameters of systemic inflammation—C-reactive protein, ESR, and procalcitonin—were used as markers of the severity of the inflammatory process. Hemostasis was simultaneously analyzed, which allowed us to confirm the presence or absence of DIC according to the ISTH diagnostic criteria.

Particular attention was paid to the cytokine profile, which reflects the balance of proinflammatory and regulatory mediators. Levels of key cytokines were determined using standard certified methods. The obtained values made it possible to identify differences in the strength of the immune response between the groups and assess their association with the risk of developing DIC.

All parameters were processed using statistical analysis methods, calculating mean values, standard errors, and comparing groups at a significance level of $p < 0.05$. This ensured the reliability of the obtained data and allowed us to determine the role of cytokine dysregulation in the development of severe complications.

3. Results And Discussion

The analysis of the examined children revealed significant differences in clinical and laboratory parameters between the group with DIC syndrome and patients without coagulopathic complications. Already at the admission stage, children with DIC were distinguished by a more pronounced inflammatory response: their C-reactive protein level reached an average of 118.4 mg/L (95% CI: 103–132 mg/L), which was statistically significantly higher than the indicators of the second group, where the average values were 62.7 mg/L (95% CI: 52–73 mg/L; $p < 0.01$). Similar differences were observed for procalcitonin, with concentrations in children with DIC ranging from 5.4 ng/ml (95% CI: 3.9–6.8 ng/ml) to 1.9 ng/ml (95% CI: 1.2–2.7 ng/ml; $p < 0.001$) in patients without DIC. These data confirm a higher intensity of systemic inflammation in children with complicated pneumonia.

Significant differences were also found in hemostasis. Children with DIC demonstrated a prolonged APTT ranging from 42–58 seconds (mean 49.6 sec), which was significantly higher than in the second group (31–39 seconds, mean 34.2 sec; $p < 0.01$). Fibrinogen levels decreased to 1.4 g/L (95% CI: 1.1–1.8 g/L), while in children without complications, they remained within the range of 2.4–3.3 g/L ($p < 0.05$). The most significant differences concerned D-dimer concentrations: in patients with DIC, its level reached 3.8 µg/mL (95% CI: 3.1–4.6 µg/mL), which was almost three times higher than the values in the second group (1.2 µg/mL; 95% CI: 0.8–1.6 µg/mL; $p < 0.001$).

The immunological part of the study demonstrated a clear cytokine imbalance in children with DIC. The IL-6 concentration in the first group reached 189 pg/ml (95% CI: 160–215 pg/ml), which was twice as high as in children without complications, where the figure was 92 pg/ml (95% CI: 78–104 pg/ml; $p < 0.001$). TNF-α levels showed a similar trend, exceeding normal values by almost three times. Whereas the regulatory cytokine IL-10 was relatively lower - 6.8 pg/ml in the DIC group versus 11.4 pg/ml in children without complications, which confirms a weakening of the anti-inflammatory response. The cytokine imbalance index (IL-6/IL-10) in the DIC group reached 27.7, which is almost four times higher than in the second group (7.9), and these differences were statistically significant ($p < 0.001$). Thus, the obtained data demonstrate that the development of DIC syndrome in young children is closely linked to a pronounced inflammatory response and dysregulated cytokine activity. Hyperproduction of proinflammatory cytokines, accompanied by an insufficient increase in regulatory mediators, contributes to the activation of the coagulation cascade and the development of systemic microcirculatory disorders. These results confirm the key role of cytokine dysregulation in the pathogenesis of DIC in severe pneumonia and emphasize the need for early monitoring of immunological and hemostasis parameters for the timely prevention of complications.

This study identified key clinical and immunological mechanisms underlying the development of DIC syndrome in children aged 1–3 years with severe pneumonia. The obtained data convincingly demonstrated that the development of DIC is directly linked to pronounced cytokine dysregulation and profound impairments of the hemostatic system. Children with DIC showed significantly higher concentrations of proinflammatory cytokines (IL-6, TNF-α) against a background of decreased levels of anti-inflammatory IL-10, leading to a pronounced cytokine imbalance. The IL-6/IL-10 ratio in the DIC group was almost four times higher than in patients without coagulopathic complications, confirming the leading role of immune hyperactivation in triggering the cascade of hemostatic disorders.

Concurrently, characteristic hemostatic changes were noted—decreased fibrinogen levels, prolonged APTT, and a significant increase in D-dimer concentrations. These parameters significantly correlated with the severity of the inflammatory response and cytokine

levels, confirming the close relationship between the immune and coagulation mechanisms in the pathogenesis of DIC. Thus, the study results highlight that severe pneumonia in young children is associated with a high risk of developing DIC, caused by both the overproduction of proinflammatory cytokines and the insufficiency of anti-inflammatory mechanisms. These findings suggest that cytokine levels may be early prognostic markers for the risk of developing DIC.

These findings support the need for early monitoring of cytokine profiles and hemostatic parameters in children with severe pneumonia, which will enable timely identification of high-risk groups, tailored treatment strategies, and the prevention of life-threatening complications.

4. Conclusions

1. In children aged 1–3 years with severe pneumonia, the development of DIC is accompanied by more pronounced systemic inflammation, as evidenced by significantly elevated levels of C-reactive protein and procalcitonin compared to patients without DIC.

2. The development of DIC is associated with pronounced cytokine dysregulation: IL-6 concentrations in children with DIC were more than twice as high, while IL-10 levels were significantly lower, indicating an insufficient anti-inflammatory response.

3. The IL-6/IL-10 cytokine imbalance index in children with DIC was almost four times higher than in the comparison group, suggesting it may be a potential marker for the early risk of developing coagulopathic complications.

4. Hemostasis disorders in children with DIC were manifested by decreased fibrinogen levels, prolonged APTT, and a sharp increase in D-dimer, reflecting the activation of thrombus formation and consumption of coagulation factors.

5. The severity of immunological and hemostatic disorders was directly correlated, confirming the pathogenetic role of cytokine hyperproduction in triggering the cascade of hemocoagulation disorders in severe pneumonia.

6. The obtained data indicate the need for early comprehensive monitoring of the cytokine profile and hemostatic parameters in young children with severe pneumonia for the timely prevention and correction of DIC syndrome.

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