RELATIONSHIP BETWEEN SYSTEMIC INFLAMMATORY RESPONSE AND HEMODYNAMIC DISTURBANCES IN CHILDREN WITH SEVERE BURN TOXEMIA

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Abstract. *This study evaluated age-related relationships between systemic inflammatory response and hemodynamic disturbances in 76 children with severe burn toxemia. The pediatric cohort included 31 children aged 6 months–3 years, 20 children aged 3.1–7 years, and 25 children aged 7.1–18 years. Prolonged hourly monitoring included systolic, diastolic, pulse, and mean arterial pressure, cardiac output, stroke volume, total peripheral vascular resistance, heart rate, autonomic tone, and myocardial oxygen demand. Laboratory assessment included leukocyte count and differential, erythrocyte sedimentation rate, urea, creatinine, total protein, albumin, and medium-molecular-weight peptides. In the younger age groups, mean arterial pressure was generally maintained within approximately 80–85 mmHg, whereas in children aged 7.1–18 years a gradual decrease in the mean arterial pressure mesor was observed on days 6–10. Correlation analysis demonstrated age-dependent relationships between mean arterial pressure and leukocyte indices. Leukocytosis was associated with an unfavorable hemodynamic tendency in all three pediatric age groups. The direction of the relationship between mean arterial pressure and band neutrophils differed by age: it was positive in children younger than 3 years and negative in the 3.1–7- and 7.1–18-year groups. In younger children, increasing endogenous intoxication and impaired renal excretory function during the first 10 days were associated with lower mean arterial pressure and reduced circulatory efficiency, whereas older children showed a greater ability to increase cardiac output and mean arterial pressure under hypercatabolic inflammatory stress.*

These findings indicate that the hemodynamic consequences of systemic inflammation during burn toxemia are age dependent. Combined interpretation of inflammatory markers and hemodynamic variables may reveal early signs of

reduced cardiovascular compensation that can be missed when arterial pressure is assessed in isolation.

Keywords: *severe burn toxemia, children, systemic inflammatory response, hemodynamics, mean arterial pressure, leukocytosis, cardiac output, endogenous intoxication, age-related response, intensive care.*

Relevance. Severe burn injury triggers a complex systemic inflammatory response that affects vascular permeability, circulating volume, myocardial function, peripheral vascular tone, and tissue perfusion. In children, these mechanisms are especially important because cardiovascular reserve, circulating blood volume, and compensatory responses vary substantially with age [1, 2]. Pediatric burns involving a large body surface area may produce systemic inflammatory response syndrome, burn shock, hypermetabolism, and subsequent cardiovascular stress [2–4]. Modern reviews emphasize that inflammatory mediators, endothelial dysfunction, capillary leak, and altered vascular resistance interact closely with the hemodynamic response after severe thermal injury [1, 5]. At the same time, no single hemodynamic endpoint fully characterizes the circulatory status of a burned child, which supports the use of combined physiological and laboratory monitoring [3, 6].

Accordingly, simultaneous assessment of systemic inflammatory markers and hemodynamic parameters may help identify age-related differences in compensatory cardiovascular responses during burn toxemia. The present study evaluates the relationship between the inflammatory response and central hemodynamics in children younger than 18 years with severe thermal injury.

Objective. To assess age-related relationships between systemic inflammatory response and hemodynamic disturbances during severe burn toxemia in children and to characterize differences in cardiovascular compensation across pediatric age groups.

Materials and Methods. The analysis included 76 pediatric patients with severe thermal burns treated in the burn intensive care unit. According to age, patients were divided into three groups: 31 children aged 6 months–3 years, 20 children aged 3.1–7 years, and 25 children aged 7.1–18 years. Within pediatric age groups, patients were additionally stratified according to burn severity and duration of intensive care. Hemodynamic monitoring was performed hourly and included systolic arterial pressure, diastolic arterial pressure, pulse pressure, mean arterial pressure (MAP), stroke volume, cardiac output (CO), total peripheral vascular resistance (TPVR), heart rate, overall autonomic tone, and myocardial oxygen

demand. Circadian characteristics of the monitored variables were assessed using mesor, amplitude, acrophase, and bathyphase. Laboratory evaluation included complete blood count with leukocyte differential, erythrocyte sedimentation rate (ESR), urea, creatinine, total protein, albumin, and medium-molecular-weight peptides. The relationship between hemodynamic variables and laboratory indicators of systemic inflammation and endogenous intoxication was assessed by correlation analysis. Particular attention was paid to the first 10–12 days of burn toxemia, when inflammatory and hypercatabolic changes were most clinically relevant.

Results. The relationship between systemic inflammatory response and hemodynamics differed substantially with age. In children younger than 3 years and in the 3.1–7-year group, MAP was generally lower than in older patients and remained mainly within 80–85 mmHg, consistent with age-related physiological characteristics. In children aged 7.1–18 years, a gradual reduction in the circadian mesor of MAP was observed on days 6–10 of treatment. In this group, the Frank Index was 76.3±19.1 units, and the decline in MAP coincided with a reduction in the systemic inflammatory response during anti-inflammatory and stress-limiting therapy.

Correlation analysis showed a tendency toward a direct relationship between MAP and ESR, suggesting that the intensity of the inflammatory response was associated with arterial pressure dynamics. At the same time, leukocytosis was associated with an unfavorable hemodynamic tendency in all three pediatric age groups. The relationship between MAP and band neutrophils was age dependent: a positive association was observed in children younger than 3 years, whereas inverse associations were found in the 3.1–7- and 7.1–18-year groups. These findings indicate that similar laboratory signs of inflammation may correspond to different cardiovascular responses at different stages of physiological maturation. Age-related differences were also apparent when inflammatory stress was accompanied by hypercatabolism and endogenous intoxication. In children younger than 3 years, deterioration of renal excretory function during the first 10 days of burn toxemia was associated with increasing intoxication, lower MAP, and reduced circulatory efficiency. In contrast, children aged 7.1–18 years, whose cardiovascular system was anatomically and functionally more mature, demonstrated a compensatory response characterized by increased cardiac output and higher MAP under systemic inflammatory and hypercatabolic stress.

The inflammatory response was closely linked to endogenous intoxication in the younger groups. On days 10–12, a strong direct correlation between leukocyte count and medium-molecular-weight peptides was found in children younger than 3 years (r=0.79) and in children aged 3.1–7 years (r=0.99). These relationships support the concept that progressive endogenous intoxication and cellular inflammatory activation develop in parallel and may contribute to destabilization of cardiovascular compensation. Across the pediatric groups, persistent hypersympathicotonia was associated with a hypercirculatory tendency and increased myocardial oxygen demand, indicating that systemic inflammatory stress imposes an additional functional load on the cardiovascular system.

Conclusion. During severe burn toxemia in children, the relationship between systemic inflammatory response and hemodynamic status is age dependent. Leukocytosis and changes in leukocyte differential are associated with MAP dynamics, but the direction and apparent compensatory significance of these relationships vary across age groups. Younger children demonstrate greater vulnerability to circulatory inefficiency when endogenous intoxication and renal dysfunction increase, whereas older children retain a greater ability to respond through increased cardiac output and arterial pressure. Therefore, assessment of burn toxemia in pediatric intensive care should combine dynamic hemodynamic monitoring with inflammatory and biochemical markers rather than relying on arterial pressure alone. Such an approach may improve early recognition of declining cardiovascular compensation and support age-oriented correction of intensive therapy.

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