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Clinical and Pathophysiological Determinants of Cardiac Surgery–Associated Acute Kidney Injury in Young Children: The Role of Preoperative and Intraoperative Factors

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Abstract

Cardiac surgery–associated acute kidney injury (CSA-AKI) in young children is a common and clinically significant complication following surgical correction of congenital heart defects, developing as a result of the combined impact of baseline renal vulnerability and intraoperative perfusion stress.

Objective. To identify clinical and pathophysiological risk factors for CSA-AKI and to determine independent predictors of its development in young children. The study included 585 children under 3 years of age who underwent cardiac surgery with cardiopulmonary bypass. CSA-AKI was diagnosed according to KDIGO criteria. Preoperative and intraoperative

factors were analyzed using univariate and multivariate statistical methods. CSA-AKI developed in 224 patients (38.3%). At the preoperative stage, significant risk factors included heart failure, hypoxemia, low body weight, and high complexity of congenital heart defects. In the intraoperative period, the risk of CSA-AKI was significantly associated with cardiopulmonary bypass duration >90 minutes, hyperlactatemia, hematocrit <25%, and reduced urine output. Multivariate analysis identified elevated lactate levels, prolonged perfusion duration, hemodilution, and early renal functional response as independent predictors. CSA-AKI in young children is determined by a combination of preoperative and intraoperative factors reflecting impaired systemic perfusion and oxygen delivery. Early risk stratification and targeted control of modifiable perfusion parameters are essential for prevention.

Keywords: Congenital heart defects, cardiopulmonary bypass, acute kidney injury, urine output, perfusion.

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1. Introduction

Acute kidney injury (AKI) after pediatric cardiac surgery remains one of the most challenging problems in pediatric cardiac intensive care because of its high incidence, the difficulty of early diagnosis, and its pronounced impact on clinical outcomes. According to the prospective multicenter study by Li et al. (2011), the incidence of AKI after pediatric cardiac surgery remains high and is associated with worse early outcomes, prolonged intensive care unit stay, and increased mortality. Yuan (2019) likewise emphasized that this complication is particularly common in neonates and infants due to the immaturity of the renal parenchyma and the limited adaptive capacity of the kidneys under systemic stress.

In young children, AKI cannot be regarded solely as a local renal complication. In clinical reality, it develops in the context of a complex interaction between systemic hemodynamics, chronic hypoxemia, congenital anatomical complexity of the defect, CPB parameters, and the severity of postoperative cardiorenal interaction. Current KDIGO recommendations continue to rely on serum creatinine and urine output as the diagnostic foundation; however, these parameters often reflect already established injury rather than the subclinical phase of the process.

In recent years, increasing attention has been paid to identifying early risk factors and modifiable determinants capable of preventing the transition from functional instability to structural nephron injury. Studies by Krawczeski et al. (2010), Parikh et al. (2011), and Dreher et al. (2023) have shown that clinical value is associated not only with early biomarkers, but also with perfusion-related parameters reflecting oxygen delivery, tissue hypoperfusion, and the adequacy of systemic hemodynamics.

However, the relationship between preoperative vulnerability and intraoperative perfusion stress as a unified pathogenetic cascade in young children remains insufficiently characterized. This makes it relevant to distinguish between baseline and triggering factors and to provide their clinical and pathophysiological interpretation.

Objective of the study: to determine the clinical and pathophysiological determinants of CSA-AKI in young children after surgical correction of congenital heart defects and to identify the most significant preoperative and intraoperative predictors of its development.

2. Methods

The study included 585 children under 3 years of age who underwent surgical correction of congenital heart defects with cardiopulmonary bypass. CSA-AKI was diagnosed in the early postoperative period according to the KDIGO criteria. In the study cohort, CSA-AKI was identified in 224 patients, whereas 361 children showed no signs of renal dysfunction. The baseline numerical data and cohort composition corresponded to the main text of the article.

The analysis was structured according to a two-stage design. First, the baseline preoperative patient characteristics were evaluated: age, body weight, anatomical complexity of the defect according to the RACHS classification, presence of hypoxemia, and presence of heart failure. Second, intraoperative parameters were analyzed, including CPB duration, aortic cross-clamp time, perfusion pressure, severity of hemodilution, lactate level, and urine output.

Categorical variables were analyzed using Pearson's chi-square test in order to determine the statistical significance of differences in the distribution of variables between groups. To assess the strength of association between a factor and the outcome, odds ratios (OR) with 95% confidence intervals (95% CI) were calculated. At the second stage, multivariate logistic regression was applied to identify independent predictors while minimizing the mutual influence of associated variables.

3. Results

The results of the study demonstrated that CSA-AKI is not an episodic complication but rather a predictable manifestation of a systemic pathological process developing in the setting of cardiac surgery in young children.

In the study population, CSA-AKI was diagnosed in 224 of 585 children, accounting for 38.3%. This figure alone confirms the high clinical significance of renal dysfunction within the structure of postoperative complications in young children. The observed incidence is comparable to the range reported in the international literature for pediatric cardiac surgery, especially in high-risk groups.

Importantly, the development of CSA-AKI did not appear to be a random event. Even at the initial comparison, it was evident that children with AKI had a different risk profile, and these differences were already present at the preoperative stage and became more pronounced under the conditions of intraoperative perfusion stress.

Analysis of preoperative risk factors showed that the development of CSA-AKI begins long before surgery and is largely determined by the patient's baseline condition (Tabl.1).

Table 1. Clinical, demographic, and anatomical determinants of CSA-AKI risk

Parameter	No AKI (n=361)	AKI (n=224)	χ^2	OR (95% CI)	p
Age <1 month	68 (18.8%)	76 (33.9%)	15.2	2.2 (1.5–3.2)	<0.001
Body weight <5 kg	60 (16.6%)	82 (36.6%)	22.6	2.9 (1.9–4.2)	<0.001
RACHS ≥3	199 (55.1%)	152 (67.9%)	9.4	1.7 (1.2–2.4)	0.002
SpO_2 <90%	108 (29.9%)	128 (57.1%)	38.5	3.0 (2.1–4.3)	<0.001

Heart failure, NYHA/Ross class II–III*	122 (33.8%)	154 (68.8%)	52.1	3.3 (2.3–4.8)	<0.001

Analysis of preoperative factors demonstrated that the baseline probability of CSA-AKI was distributed крайне unevenly across the cohort. The most pronounced differences were observed for heart failure of functional class II–III: its frequency in the AKI group reached 68.8% versus 33.8% in patients without AKI, with χ^2=52.1 and the highest odds ratio among the preoperative variables, OR 3.3 (95% CI 2.3–4.8; p<0.001). This allows heart failure to be regarded not merely as a comorbid condition, but as one of the central mechanisms underlying baseline renal vulnerability. From a pathophysiological perspective, this is understandable: chronic reduction in effective cardiac output leads to persistent renal hypoperfusion, venous congestion, activation of neurohumoral systems, and increased sensitivity of the nephron to any additional ischemic insult.

Hypoxemia proved to be another highly significant factor. The frequency of SpO_2 <90% in the CSA-AKI group was 57.1% compared with 29.9% in the group without renal dysfunction, with χ^2=38.5 and OR 3.0 (95% CI 2.1–4.3; p<0.001). Such a strong association indicates that baseline oxygen deficit is one of the major components of preoperative risk. Chronic hypoxemia, characteristic of several complex congenital heart defects, reduces oxygen delivery reserve to organs and creates conditions for subclinical ischemia of renal tissue even before surgery.

Age below 1 month was also significantly associated with the development of CSA-AKI: 33.9% in the AKI group versus 18.8% in the non-AKI group, χ^2=15.2, OR 2.2 (95% CI 1.5–3.2; p<0.001). This finding has an important biological basis. During the neonatal period, the kidneys are structurally and functionally immature, glomerular filtration rate is reduced, and the capacity for autoregulation of renal blood flow is limited. Consequently, any reduction in systemic perfusion is tolerated substantially worse in this age group than in older children.

Body weight below 5 kg demonstrated an even stronger association with the outcome: χ^2=22.6, OR 2.9 (95% CI 1.9–4.2; p<0.001). Low body weight reflects not only age-related immaturity, but also limited physiological reserves, including lower circulating blood volume, greater vulnerability to hemodilution, and a stronger dependence of organ blood flow on systemic hemodynamics.

Complexity of the defect according to RACHS ≥3 also retained statistical significance, although its magnitude was less pronounced than that of heart failure and hypoxemia: χ^2=9.4, OR 1.7 (95% CI 1.2–2.4; p=0.002). This observation is fundamentally important. It shows that anatomical complexity is indeed relevant, but its impact is inferior to that of functional disturbances, particularly heart failure and hypoxemia. Therefore, risk assessment for CSA-AKI in young children should not rely solely on surgical classification; integration of both anatomical and functional patient characteristics is more informative.

Thus, the preoperative stage identifies a subgroup of patients at high risk. The most important factors are those reflecting chronic deficits in perfusion and oxygenation rather than anatomical complexity alone. This supports the concept of the preoperative phase as the period during which baseline renal vulnerability is formed.

If the preoperative stage determines the degree of predisposition, then the intraoperative period becomes the key phase in which renal injury is realized. It is during CPB that hemodilution, reduced oxygen-carrying capacity of blood, microcirculatory disturbances, systemic inflammatory response, and mismatch between oxygen delivery and consumption develop (tabl.2).

Table 2. Univariate analysis of associations between intraoperative parameters and the development of CSA-AKI

Factor	χ^2	OR	95% CI	p
CPB duration >90 min	18.4	2.9	1.6–5.0	<0.001

Aortic cross-clamp time >60 min	15.2	2.7	1.5–4.8	<0.001
Mean perfusion pressure <40 mmHg	9.1	2.5	1.4–4.4	0.002
Hematocrit during CPB <25%	17.3	2.8	1.6–4.9	<0.001
Lactate at the end of surgery >4.0 mmol/L	22.8	3.1	1.8–5.3	<0.001
Intraoperative urine output <1.0 mL/kg/h	14.6	2.6	1.5–4.5	<0.001

All analyzed factors demonstrated statistically significant associations with the development of CSA-AKI, although their contribution varied.

The strongest association was observed for lactate level >4.0 mmol/L: χ^2=22.8, OR 3.1 (95% CI 1.8–5.3; p<0.001). This allows hyperlactatemia to be considered the most integral marker of inadequate perfusion. Unlike isolated hemodynamic parameters, lactate reflects the global mismatch between oxygen delivery and oxygen consumption at the tissue level. That is why its elevation is especially important in the context of CSA-AKI prognosis: it represents not merely an unfavorable number, but a systemic metabolic trace of perfusion inadequacy.

CPB duration greater than 90 minutes was also associated with a marked increase in the risk of CSA-AKI: χ^2=18.4, OR 2.9 (95% CI 1.6–5.0; p<0.001). Clinically, this means that the longer a child remains under conditions of non-pulsatile perfusion, hemodilution, and blood contact with the extracorporeal circuit, the higher the probability of renal injury. Several mechanisms are involved here, including decreased oxygen-carrying capacity, activation of inflammatory response, microvascular disturbances, and reperfusion stress.

Marked hemodilution, reflected by hematocrit below 25%, also showed a strong association with the outcome: χ^2=17.3, OR 2.8 (95% CI 1.6–4.9; p<0.001). In young children, this is of particular importance, since a reduction in hematocrit directly diminishes oxygen transport and worsens tolerance to hypoperfusion. In fact, this parameter may be regarded as a modifiable marker of intraoperative perfusion quality.

Aortic cross-clamp time >60 minutes retained significant statistical importance as well (χ^2=15.2, OR 2.7; 95% CI 1.5–4.8; p<0.001). Although this factor primarily reflects surgical complexity and the depth of ischemic stress, it also indirectly characterizes the duration of systemic burden imposed on the child's body.

Reduced intraoperative urine output <1.0 mL/kg/h was likewise significantly associated with CSA-AKI. This parameter deserves particular attention, since it reflects the earliest functional renal response to systemic hypoperfusion and may be interpreted not only as a marker, but also as an early manifestation of ongoing injury.

Thus, the intraoperative stage is characterized by the development of a complex perfusion stress. The most powerful predictors are hyperlactatemia, prolonged CPB duration, and the severity of hemodilution, emphasizing the leading role of impaired oxygen transport.

To assess the independent contribution of individual intraoperative factors to the development of CSA-AKI, multivariate analysis was performed (tabl.3).

Table 3. Multivariate analysis of independent intraoperative predictors of CSA-AKI

Factor	β	OR	95% CI	p
CPB duration >90 min	0.88	2.4	1.3–4.5	0.003
Lactate at the end of surgery >4.0 mmol/L	0.96	2.6	1.4–4.8	0.001
Hematocrit <25%	0.79	2.2	1.2–4.0	0.008
Intraoperative urine output <1.0 mL/kg/h	0.67	1.9	1.1–3.4	0.019

Multivariate analysis makes it possible to distinguish truly independent determinants from factors that merely accompany the main mechanisms. Four parameters entered the final model, each reflecting an independent pathogenic link.

The greatest contribution to the model was made by hyperlactatemia (β=0.96; OR 2.6; 95% CI 1.4–4.8; p=0.001). This confirms that metabolic stress and global tissue hypoperfusion play a central role in the development of CSA-AKI.

CPB duration >90 minutes (β=0.88; OR 2.4; 95% CI 1.3–4.5; p=0.003) reflects the temporal component of injury: the longer the exposure to unfavorable perfusion conditions, the higher the likelihood that functional instability will progress into structural injury.

Hematocrit <25% (β=0.79; OR 2.2; 95% CI 1.2–4.0; p=0.008) confirms the independent role of hemodilution as a modifiable risk factor. Intraoperative urine output <1.0 mL/kg/h (β=0.67; OR 1.9; 95% CI 1.1–3.4; p=0.019) completes the model as an early functional indicator that the kidneys have already become involved in the pathological process.

Thus, the resulting model shows that intraoperative CSA-AKI risk cannot be reduced to a single cause. Rather, it is formed by the combination of exposure duration, adequacy of oxygen transport, severity of metabolic stress, and the early renal response.

4. Discussion

The results of the study indicate that CSA-AKI in young children develops as a consequence of sequential interaction between preoperative renal vulnerability and intraoperative perfusion stress. At the preoperative stage, the most important determinants were heart failure, hypoxemia, low body weight, and early age, all of which reflect a reduced baseline reserve of systemic and renal perfusion (p<0.001). This supports the view that CSA-AKI should not be regarded as a random complication, but rather as a predictable consequence of an unfavorable functional status.

The intraoperative period represents the phase of injury realization, in which the leading role is played by parameters characterizing the quality of oxygen transport and tissue perfusion. The most significant factors were CPB duration >90 minutes, hyperlactatemia, hematocrit <25%, and reduced intraoperative urine output (p<0.001). Importantly, these parameters, rather than anatomical complexity alone, demonstrated the greatest prognostic value, highlighting the dominant role of functional and perfusion-related mechanisms in the pathogenesis of CSA-AKI.

Of particular importance is the fact that many of the identified determinants are potentially modifiable. This has direct clinical relevance, as it supports a shift from late diagnosis of renal dysfunction to early risk stratification and active prevention. Maintenance of adequate perfusion, limitation of hemodilution, lactate control, and assessment of intraoperative urine output should be regarded as key elements of an organ-protective strategy in high-risk children.

5. Conclusion

CSA-AKI develops in 38.3% of young children after cardiac surgery and represents one of the most significant complications of the early postoperative period.

The preoperative stage is characterized by the formation of baseline renal vulnerability, the key components of which are heart failure, hypoxemia, low body weight, and early age. The intraoperative stage realizes renal injury through prolonged CPB duration, hyperlactatemia, hemodilution, and reduced intraoperative urine output.

The most significant independent intraoperative predictors are hyperlactatemia, CPB duration greater than 90 minutes, hematocrit below 25%, and decreased urine output. These parameters reflect impaired oxygen transport, severity of metabolic stress, and early renal involvement in the pathological process.

The obtained data substantiate the need for early risk stratification and a personalized organ-protective strategy focused on modifiable perfusion-related parameters.

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Declaration of Financial and Other Relationships. All authors participated in the conception and design of the study and in writing the manuscript. The final version of the manuscript was approved by all authors. The authors did not receive any fees for the study.

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