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An integrated predictive model for reduced renal perfusion in pediatric patients following congenital heart defect repair

Abstract

Introduction. The early postoperative period after surgical repair of congenital heart defects in children is accompanied by hemodynamic instability, systemic inflammatory response, and significant perfusion fluctuations, which increase the risk of organ dysfunction. The kidneys are among the most sensitive target organs, and reduced renal perfusion (RRP) serves as an early marker of an unfavorable course of the postoperative period.

Aim. To develop and internally validate an integral model for predicting reduced renal perfusion in children after surgical repair of congenital heart defects.

Materials and Methods. A retrospective analysis of 206 children after repair of congenital heart defects using cardiopulmonary bypass (CPB) was performed. A multivariate logistic regression model was constructed, including clinical, laboratory, echocardiographic, and phenotypic indicators. Hourly diuresis was standardized by age using the Z-score. Internal validation was performed using the bootstrap method (1.000 resamples).

Results. Independent predictors of RRP were the use of CPB (OR=2.41), decrease in Z-diuresis (OR=0.59), decrease in left ventricular ejection fraction (OR=0.97), changes in hemoglobin (ΔHb) and WBC count (ΔWBC), diagnostic category, and presence of connective tissue dysplasia (CTD) phenotype (OR=2.51). The model demonstrated high discriminative ability (AUC=0.94), good calibration, and stability.

Conclusions. RRP in children after surgical repair of congenital heart defects is the result of a complex interaction of perfusion, age-related, cardiac, laboratory, and phenotypic factors. The proposed model allows effective prediction of RRP risk and can be used for personalization of postoperative management in children.

Keywords: *cardiac surgery; Z-diuresis; left ventricular ejection fraction; postoperative hemodynamics; early organ dysfunction.*

Introduction. Renal dysfunction and acute kidney injury (AKI) are frequent complications ~~of~~ in the early postoperative period after surgical repair of congenital heart defects (CHD) in children and are associated with worse clinical outcomes [1,2,8,10,11,13]. Their development is based on a combination of perfusion stress, systemic inflammatory response, hemodilution, microcirculatory disturbances, and fluctuations in cardiac output, especially in the context of cardiopulmonary bypass (CPB) use [5,6,8,10]. In children, these processes are intensified by age-related immaturity of regulatory mechanisms and high variability in hemodynamic responses [2,5,9].

Early detection of renal perfusion impairment remains difficult, as traditional biochemical markers are late indicators of injury [6,7,9]. Hourly diuresis is considered an accessible early indicator of perfusion status; however, its interpretation in children is limited by age-related variability [3,7]. This justifies the need for its age standardization.

Most modern predictive models focus on AKI and insufficiently consider early perfusion changes, cardiac function, and phenotypic characteristics of the patient

[8,10,11]. At the same time, data on connective tissue vulnerability suggest its potential influence on hemodynamic adaptation and vascular reactivity [4,12].

In this regard, it is relevant to create an integral model for predicting reduced renal perfusion (RRP) in children after CHD repair, considering clinical, laboratory, echocardiographic, and phenotypic factors.

Aim. To develop and internally validate a multivariate model for predicting RRP in children after surgical repair of CHD and to create a simplified integral index for clinical risk stratification.

Materials and Methods. The study was conducted as a retrospective analysis of clinical data from 206 children after surgical repair of CHD, operated on between 2015 and 2023 in the Department of Cardiovascular Surgery of the Odesa Regional Children's Clinical Hospital. Patients were stratified into five age groups: neonates (n=18), infants (n=101), 1-3 years (n=25), 3-7 years (n=30), and 7-14 years (n=32).

The primary endpoint was defined as RRP occurring within the first 48 hours after surgery. Clinical, laboratory, and echocardiographic parameters were analyzed, including the use of cardiopulmonary bypass (CPB), age, left ventricular ejection fraction (LVEF), ΔHb, ΔWBC, CHD diagnostic cluster, and connective tissue dysplasia (CTD) phenotype.

Hourly urine output was standardized by age using a Z-score (based on proprietary empirical data), allowing for correct comparison between age groups.

The CTD phenotype was assessed using a binary indicator (CTD_pos) based on the Beighton score and clinical criteria (Beighton ≥5/9, ≥2 phenotypic features, aortic root dilation, or mitral valve prolapse).

A logistic regression model was constructed, including clinically justified predictors (CPB, Z-diuresis, age, LVEF, ΔHb, ΔWBC, CHD cluster, CTD). Nonlinear relationships were modeled using restricted cubic splines, and interactions with CTD_pos were additionally evaluated.

Internal validation was performed using bootstrapping (1.000 resamples). Discrimination was assessed by AUC, calibration by slope and intercept, and accuracy by the Brier score.

Based on significant predictors, a simplified integral index (IPI-RRP) was developed for clinical application. Calculations were performed using IBM SPSS Statistics and Microsoft Excel.

The study was conducted on de-identified data in accordance with ethical principles and was approved by the Bioethics Commission (Protocol No. 01 dated January 14, 2026).

Results. To analyse risk factors for an unfavourable course of the early postoperative period in 206 children after surgical repair of CHD, a multivariable prediction model for RRP was developed and internally validated. This endpoint was chosen because renal hypoperfusion is an early and sensitive marker of systemic hemodynamic instability after surgery, especially in the context of CPB use, systemic inflammatory response, and age-related vulnerability of target organs. The model enabled a transition from describing intergroup differences to a quantitative assessment of individual risk. A key component of the model was hourly urine output, analyzed as a standardized parameter, since its absolute values have different clinical significance in children of different ages. Age-specific reference values were derived from proprietary empirical data (Table 1).

The use of Z-diuresis allowed it to be interpreted as a dimensionless marker of perfusion status: values (Z=0) corresponded to the age norm, (Z<-1) were considered an early sign of RRP, (Z<-2) indicated a pronounced perfusion deficit with a high risk of renal dysfunction, and (Z>+1) – a possible variant of recovery after ischemia, osmotic diuresis, or hyperperfusion response. Such standardization increased comparability between age groups and made the model pathophysiologically appropriate for a pediatric population.

A separate block of analysis included phenotypic manifestations of CTD as a potential modifier of postoperative hemodynamic and renal vulnerability. Connective tissue dysplasia was identified using a unified phenotypic protocol combining somatic, orthopedic, vascular-autonomic, and echocardiographic features. Two predictors were formed: **CTD_pos** – a binary indicator of clinically significant CTD phenotype, and **CTD_burden** – an ordinal scale from 0 to 3 points. In the final model, CTD_pos was used. It was considered positive if at least one of the following criteria was present: Beighton score ≥ 5/9, at least two phenotypic components, aortic root dilation (Z>2), or mitral valve prolapse. Clinically, CTD was regarded not as a background characteristic but as a systemic factor that could exacerbate vascular and hemodynamic vulnerability under conditions

Table 1

Age-specific norms of hourly diuresis for calculation of the Z-score

Age group	Mean (μ)	SD (σ)	Formula
1-28 days	25.42857	2.225395	Z = (x − 25.42857) / 2.225395
1-12 months	25.78218	2.520333	Z = (x − 25.78218) / 2.520333
1-3 years	25.66667	2.697288	Z = (x − 25.66667) / 2.697288
3-7 years	39.83333	5.540343	Z = (x − 39.83333) / 5.540343
7-14 years	39.37500	6.163106	Z = (x − 39.37500) / 6.163106

of perfusion stress. The operationalization criteria are presented in Table 2.

A logistic regression model with a logit link function was constructed. The dependent variable was the presence or absence of RRP occurring within the first 48 hours after surgery. Clinically justified predictors were included: use of CPB, Z-diuresis, age, LVEF, changes in hemoglobin (ΔHb) and WBC count (ΔWBC), diagnostic category of CHD, and CTD phenotype indicators. Given the non-linear nature of age and Z-diuresis effects, these variables were modeled using restricted cubic splines. Interactions between CPB × CTD_pos, LVEF × CTD_pos, and age × CTD_pos were additionally analyzed, which allowed the assessment of the modifying effect of CTD on the body's hemodynamic response.

The generalized form of the model was:

$$\text{Logit}(P)=\beta_0+\beta_1\cdot CPB+\beta_2\cdot age+f_1(age)+\beta_3\cdot Z+f_2(Z)+\beta_4\cdot LVEF+\beta_5\cdot \Delta Hb+\beta_6\cdot \Delta WBC+\beta_7\cdot \text{diagnostic category}+\beta_8\cdot CTD\_pos+\beta_9\cdot(CPB\times CTD\_pos)+\beta_{10}\cdot(LVEF\times CTD\_pos)+\beta_{11}\cdot(age\times CTD\_pos)$$

where $f_1(age)$ and $f_2(Z)$ are spline transformations for age and Z-diuresis, respectively.

In the multivariate analysis, it was found that the risk of developing RRP in children after surgical repair of CHD is determined by a combination of perfusion-related, age-related, cardiac, laboratory, and phenotypic factors. The most significant predictors were the use of CPB, reduced Z-diuresis, decreased LVEF, unfavorable laboratory deltas, the diagnostic CHD cluster, and the presence of a clinically significant CTD phenotype.

The use of CPB significantly increased the risk of RRP: β=0.88; OR=2.41; 95 % CI 1.36-4.25; p=0.002. One of the key independent predictors was Z-normalized diuresis: as this parameter decreased, the risk of RRP significantly increased (β=-0.52; OR=0.59; 95 % CI 0.42-0.84; p=0.006). Thus, lower Z-diuresis served as an early quantitative marker of perfusion deficit rather than merely a nonspecific manifestation of postoperative stress.

Patient age also had independent prognostic value (β=0.04; OR=1.04; 95 % CI 1.01-1.07; p=0.004), reflecting the nonlinear influence of age-related factors on the risk of perfusion disturbances. Cardiac pump function parameters were also significant: with decreasing LVEF, the risk of RRP increased (β=-0.035; OR=0.97; 95 % CI 0.95-0.99; p=0.003). Laboratory deltas reflected important components of postoperative stress: for ΔHb, β=0.21; OR=1.23; 95 % CI 1.03-1.47; p=0.018, and for ΔWBC, β=0.08; OR=1.08; 95 % CI 1.02-1.15; p=0.012. The diagnostic CHD cluster also had independent significance (β=0.46; OR=1.58; 95 % CI 1.14-2.20; p=0.007), confirming the influence of the anatomical and physiological type of defect on baseline hemodynamic risk.

Particular attention was paid to the role of CTD. The presence of a clinically significant CTD phenotype was associated with an increased risk of RRP (β=0.92; OR=2.51; 95 % CI 1.36-4.63; p=0.003). Thus, children with CTD features had a 2.5-fold higher likelihood of developing renal hypoperfusion compared to patients without this phenotype. Additionally, a statistically significant interaction between CPB and CTD was observed (β=0.55; OR=1.73; 95 % CI 1.12-2.67; p=0.014), indicating an amplification of the adverse effect of perfusion stress in children with the CTD phenotype. Similarly, the interactions LVEF×CTD_pos (p=0.041) and age × CTD_pos (p=0.028) suggested a more complex influence of cardiac function and age in the subgroup of patients with CTD. The main model parameters are presented in Table 3.

Thus, the modeling results demonstrated that the likelihood of RRP development is determined not by a single factor, but by the combined effect of several interrelated mechanisms: perfusion load, age-related physiology, cardiac pump function, laboratory response, anatomical type of CHD, and connective tissue vulnerability.

To assess the stability and predictive ability of the model, internal validation was performed using the bootstrap method (1.000 resamples). The area under the ROC curve was AUC=0.94 (95 % CI 0.86-0.95), and after optimism correction, the value remained high (optimism-corrected AUC=0.89), confirming the stability of the model and the absence of critical overfitting.

At the optimal cut-off value of 0.42, determined by the Youden index, the model demonstrated a sensitivity of 0.95, specificity of 0.84, and a Youden index of 0.79. Overall predictive accuracy, assessed by the Brier score, was 0.14, corresponding to a good level of agreement between predicted and observed outcomes. Model calibration was also adequate: calibration slope =0.93, calibration intercept =-0.04. The positive likelihood ratio was $\mathbf{LR^+=5.94}$, and the negative likelihood ratio was $\mathbf{LR^-=0.06}$. The main indicators of diagnostic performance are presented in Table 4.

Table 2

Criteria for statistical operationalization of CTD phenotypic manifestations

Indicator	Definition	Coding
CTD_pos	Beighton ⩾ 5/9 or ⩾ 2 components, or mitral valve prolapse (MVP), or aortic root Z>2	0/1
CTD_burden = 0	absent or minimal phenotype	0
CTD_burden = 1	one component or borderline Beighton	1
CTD_burden = 2	⩾2 components or Beighton⩾5	2
CTD_burden = 3	⩾2 components + cardiac feature	3

Table 3

Logistic regression model results for predicting reduced renal perfusion

Predictor	β	SE	OR	95% CI	p
CPB	0.88	0.29	2.41	1.36–4.25	0.002
Age	0.04	0.01	1.04	1.01–1.07	0.004
Z-diuresis	−0.52	0.18	0.59	0.42–0.84	0.006
LVEF	−0.035	0.012	0.97	0.95–0.99	0.003
ΔHb	0.21	0.09	1.23	1.03–1.47	0.018
ΔWBC	0.08	0.03	1.08	1.02–1.15	0.012
Diagnostic cluster	0.46	0.17	1.58	1.14–2.20	0.007
CTD_pos	0.92	0.31	2.51	1.36–4.63	0.003
CPB × CTD_pos	0.55	0.22	1.73	1.12–2.67	0.014
LVEF × CTD_pos	−0.02	0.01	0.98	0.96–1.00	0.041
Age × CTD_pos	0.01	0.004	1.01	1.00–1.02	0.028

Thus, internal validation confirmed high discriminative ability, good calibration, and stability of the model, allowing it to be considered a reliable tool for individualized prediction of RRP risk.

To improve clinical applicability, the full logistic model was transformed into a simplified integral index for predicting reduced renal perfusion – IPI-RRP.

The index was based on predictors with the highest independent prognostic value and available in the early postoperative period: CPB, Z-diuresis, age, LVEF, ΔHb, ΔWBC, and CTD_pos. The principle of constructing the index was based on grading the predictors according to the strength of their effect, followed by conversion into a point-based scoring system.

The formula of the index was the following:

$$IPI\text{-}RRP = X\_age + X\_CPB + X\_Z + X\_LVEF + X\_\Delta Hb + X\_\Delta WBC + X\_CTD$$

where X represents the score assigned to each predictor.

The total index score reflected the overall level of RRP risk: 0-3 points – low risk, 4-7 points – moderate risk, ≥8 points – high risk. The IPI-RRP allowed rapid bedside risk stratification without complex mathematical calculations and reflected the main mechanisms underlying renal hypoperfusion: perfusion load, deficit of effective renal blood flow, reduced cardiac pump function, laboratory response, and connective tissue vulnerability. The structure of the IPI-RRP predictors and their point-based scoring is presented in Table 5.

Approximate mapping of the total IPI-RRP score to probability of RRP showed a gradual increase in risk: 0-1 points – ~5-8 %; 2-3 points – ~10-18 %; 4-5 points – ~25-40 %; 6-7 points – ~50-65 %; 8-9 points – ~70-85 %; ≥10 points – ~85-95 %.

At values ≥ 8 points, the risk of RRP exceeded 70%, justifying the need for intensive monitoring and early correction of hemodynamic and perfusion disturbances. Conversely, low index values (0-3 points) were associated with a high likelihood of an uneventful postoperative course, allowing the index to be used as a tool to rule out significant risk.

Table 4

Diagnostic and predictive performance of the model

Indicator	Value	Interpretation
AUC	0.94	very good discrimination
Sensitivity	0.95	high
Specificity	0.84	good
Youden index	0.79	optimal balance
LR^+	5.94	significant increase in probability
LR^-	0.06	significant decrease in probability

Table 5

Point-based scoring of predictors for the integral index for predicting reduced renal perfusion (IPI -RRP)

Predictor	Criterion	Points
Age	1-28 days	2
	1-12 months	1
CPB	Yes	2
Z-diuresis	-1.0 to -1.99	1
	⩽ -2.0	2
LVEF	50-59 %	1
	<50 %	2
ΔHb	Decrease of 10-19 g/L	1
	Decrease of ⩾20 g/L	2
ΔWBC	Increase of 3.0-4.9×10^9/L	1
	Increase of ⩾5.0×10^9/L	2
CTD_pos	Present	2

Thus, the developed predictive model reflects the combined influence of perfusion-related, age-related, cardiac, laboratory, and phenotypic factors on the probability of RRP in children after surgical repair of CHD. The use of an age-normalized Z-score for hourly diuresis enhanced the pathophysiological rationale for assessing perfusion status, while the inclusion of CTD phenotypic features provided additional insight into systemic modifiers of postoperative adaptation. The constructed multivariate model demonstrated high predictive performance, and the proposed simplified IPI-RRP index can be used as a practical tool for early bedside risk stratification in children after CHD repair.

Discussion. The study demonstrates that the risk of RRP in children after surgical repair of CHD is multifactorial and determined by a combination of perfusion-related, age, cardiac, laboratory, and phenotypic factors, consistent with current evidence [5,6,8,9]. The negative impact of CPB confirms findings from previous studies and is associated with systemic inflammatory response, hemodilution, microcirculatory disturbances, and fluctuations in renal blood flow [5,6,10,11].

An important finding is the use of an age-normalized Z-score for hourly urine output, which allows it to be considered a standardized marker of perfusion status applicable across different age groups. This increases the

pathophysiological validity of the model and improves the accuracy of early perfusion deficit assessment [3,7,9].

An independent association between reduced LVEF and the risk of RRP was established, reflecting the role of cardiac pump function in maintaining renal perfusion. Laboratory parameters (ΔHb, ΔWBC) complement the model by reflecting hemodilution and systemic inflammatory response.

A significant contribution of the CTD phenotype was demonstrated, acting not only as an independent predictor but also as a modifier of the effects of CPB, age, and cardiac function, thereby increasing hemodynamic vulnerability. This is consistent with current concepts regarding the role of extracellular matrix abnormalities and vascular fragility [4,12].

The model demonstrated high discriminative ability, adequate calibration, and stability. Its simplification in the form of the IPI-RRP index enables clinical application, although it does not replace full statistical prediction. Study limitations include the retrospective design, lack of external validation, and the phenotypic approach to CTD assessment. Future studies should focus on prospective and multicenter validation of the findings [9-11].

Thus, the proposed model reflects the complex nature of RRP and may be used for early risk stratification after CHD repair.

Conclusions

1. Renal hypoperfusion in children after surgical repair of CHD is influenced by a combination of perfusion-related, age-related, cardiac, laboratory, and phenotypic factors.
2. The age-normalized Z-score for hourly diuresis is a sensitive early marker of perfusion deficit and has independent prognostic value.
3. The presence of CTD phenotypic features is associated with increased RRP risk and amplifies the negative effects of perfusion stress.
4. The constructed logistic model demonstrates high discriminative ability, good calibration, and stability, and can be used for individualized risk prediction.
5. The proposed integral IPI-RRP index is a convenient clinical tool for early risk stratification and optimization of postoperative management.

Final statements

Conflict of Interest. The authors of the manuscript hereby declare that there is no actual or potential conflict of interest regarding the results of this study with pharmaceutical companies, manufacturers of biomedical devices, or other organizations whose products, services, or financial support may be related to the subject of the submitted materials or that may have sponsored the conducted research.

Ethical Approval. The authors of the manuscript hereby confirm that the study was conducted using data from primary medical records. The study was carried out in accordance with the ethical standards of the World Medical Association Declaration of Helsinki on ethical principles for medical research involving human subjects, the Directive 86/609/EEC of the European Society on the involvement of humans in biomedical research, as well as Order No. 690 of the Ministry of Health of Ukraine dated September 23, 2009.

The Bioethics Committee of Odesa National Medical University reviewed the study materials and found no violations of ethical standards outlined in current regulatory documents, including the Declaration of Helsinki and the Convention on Human Rights and Biomedicine (Protocol dated January 14, 2026, No. 01).

Use of Artificial Intelligence. The authors of the manuscript hereby declare that no generative artificial intelligence tools or services were used during the conduct of the study or the preparation of this manuscript for any tasks listed in the Generative AI Delegation Taxonomy (GAIDeT, 2025). All stages of the work – from conceptualization to final editing – were performed exclusively by the authors without the use of generative artificial intelligence.

Primary Data and Materials. The authors confirm that the study is based on the results of their own clinical research, which were systematized and analyzed by the authors. The primary data include aggregated patient indicators, laboratory results, protocols, and derived quantitative characteristics. All materials are stored in the archive of the research group and can be provided upon reasonable request to the corresponding author, subject to confidentiality and ethical requirements.

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Інтегральна модель прогнозування зниження ренальної перфузії у дітей після корекції вроджених вад серця

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Резюме

Вступ. Ранній післяопераційний період після кардіохірургічної корекції вроджених вад серця у дітей супроводжується гемодинамічною нестабільністю, системною запальною відповіддю та значними перфузійними коливаннями, що підвищує ризик органної дисфункції. Нирки є одними з найбільш чутливих органів-мішеней, а зниження ренальної перфузії (ЗРП) виступає раннім маркером несприятливого перебігу післяопераційного періоду.

Мета. Розробити та внутрішньо валідизувати інтегральну модель прогнозування зниження ренальної перфузії у дітей після кардіохірургічної корекції ВВС.

Матеріали та методи. Проведено ретроспективний аналіз **206 дітей** після корекції ВВС із застосуванням апарату штучного кровообігу (АШК). Побудовано багатофакторну логістичну регресійну модель із включенням клінічних, лабораторних, ехокардіографічних і фенотипових показників. Погодинний діурез стандартизували за віком із використанням Z-показника. Внутрішню валідацію виконано методом бутстрепу (1000 ресемплів).

Результати. Незалежними предикторами ЗРП були застосування АШК (OR=2,41), зниження Z-діурезу (OR=0,59), зниження фракції викиду лівого шлуночка (OR=0,97), лабораторні зміни (ΔHb, ΔWBC), діагностичний кластер та наявність синдрому дисплазії сполучної тканини (OR=2,51). Модель продемонструвала високу дискримінаційну здатність (AUC=0,94), добру калібрацію та стабільність.

Висновки. ЗРП у дітей після кардіохірургічної корекції ВВС є наслідком комплексної взаємодії перфузійних, вікових, кардіальних, лабораторних та фенотипових чинників. Запропонована модель дозволяє ефективно прогнозувати ризик ЗРП і може бути використана для персоналізації післяопераційного ведення дітей.

Ключові слова: *кардіохірургія; Z-діурез; фракція викиду лівого шлуночка; післяопераційна гемодинаміка; рання органна дисфункція.*

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