Scientific Research Publishing

Open Journal of Nephrology, 2026, 16(1), 60-71
https://www.scirp.org/journal/ojneph
ISSN Online: 2164-2869
ISSN Print: 2164-2842

Usefulness of Cardiac Power as a Predictor of Acute Kidney Injury in Cardiac Surgery Patients with Extracorporeal Circulation

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How to cite this paper: Nova-Meda, J.E., Narvaez-Lopez, U., Bautista-Crescencio, C.E., Solís-Loria, W.A., Beyza-Suazo, H., Herrera-Salgado, J.M., Valle-Torres, C.J., Montes-García, A.E., Rojas-Nájera, A.Z., Rojas-Nájera, A.A., Salvador-Simon, C.L., Dueñas-Sosa, M., Jiménez-Cabrera, O.G., Ocampo-Mazariegos, J.H., Marcial-Rivera, D.F., Uribe-Zapata, E.O., Reyes-Ramirez, C.E., Ruiz-Romero, D., Cáceres-Cruz, L.N., Flores-Alejo, A.K., Soza, Y.O., Valerio-Moreno, M., Malagón-Reyes, R.M., Reyes-Mendoza, L.E., Briones-Garduño, J.C., Herrera-Villalobos, J.E. and Gutierrez-Chavarria, S.A. (2026) Usefulness of Cardiac Power as a Predictor of Acute Kidney Injury in Cardiac Surgery Patients with Extracorporeal Circulation. *Open Journal of Nephrology*, **16**, 60-71.
https://doi.org/10.4236/ojneph.2026.161007

Abstract

Introduction: Acute kidney injury (AKI), identified by elevated serum creatinine or reduced urine output, is a frequent complication following cardiac surgery. It is associated with hypoperfusion, embolic events, extracorporeal circulation–related injury, and nephrotoxic exposure, affecting up to 43% of adults and 52% of children. **Methods:** Adult patients undergoing cardiac surgery with extracorporeal circulation were included. AKI was defined according to KDIGO criteria (ΔSCr > 0.3 mg/dL within 48 h, >1.5 × baseline within 7 days, or urine output < 0.5 mL/kg/h for 6 h). Data were collected prospectively, and inferential analyses (AUROC, odds ratios, chi-square with $p < 0.05$) were performed using SPSS v24.0. Sampling was conducted by convenience. The study adhered to national and international research ethics guidelines, including the Mexican General Health Law on Research. **Results:** Forty adults underwent cardiopulmonary bypass (mean age 65.8 ± 9.9 years; 75% male),

Received: November 27, 2025
Accepted: January 17, 2026
Published: January 20, 2026

with no exclusions. Serum creatinine increased from 1.11 ± 0.38 mg/dL at baseline to 2.39 ± 1.16 mg/dL at 48 hours. AKI developed in 34 patients (85%), distributed as stage I: 7.5%, stage II: 17.5%, and stage III: 60%. Cardiac power demonstrated predictive capacity for AKI, though AUROC analysis indicated negligible performance (AUC 0.093, $p = 0.002$). **Conclusions:** In patients undergoing cardiac surgery with cardiopulmonary bypass, mean cardiac output at ICU admission was 3.01 ± 1.12 L/min with cardiac power (CP) of 0.51 ± 0.26 W. AKI occurred in 85% of patients, predominantly stage III. A CP below 0.46 W doubled the risk of AKI regardless of stage, although its AUROC-based predictive performance remained negligible despite statistical significance.

Keywords

Cardiac Power, Acute Kidney Injury, Extracorporeal Circulation

1. Introduction

Acute kidney injury (AKI) is a common clinical syndrome that is characterized by abnormal renal function and structure [1]. AKI is usually diagnosed when there is a sharp decrease in glomerular filtration rate (GFR), as represented by an increase in serum creatinine (SCr) levels or a decrease in urine output over a fixed period [2]. Cardiac surgery is a significant risk factor for AKI, increasing mortality, extending hospital stays, and resulting in substantial health costs [3].

The pathogenesis of cardiac surgery-associated AKI (CSA-AKI) is multifaceted, involving reduced renal flow, dislodged emboli obstructing renal arteries, and detrimental effects from cardiopulmonary bypass (CPB) (ischemia, hemolysis, inflammation, oxidative stress) [4]. The medications used during and after surgery can contribute to kidney injury; these factors lead to renal dysfunction and characteristic electrolyte imbalances [5].

The incidence of post-cardiac surgery AKI ranges from 5% to 43%, with 1% to 7% requiring dialysis. The wide variation in the incidence rate depends on the type of surgical procedure performed, from 94% in heart transplantation to 3% in thoracic surgery [6]. Furthermore, up to 52% of children are diagnosed with AKI after cardiac surgery, which can create enormous socioeconomic burdens for clinical institutions [7].

The classifications most frequently used by researchers are Risk, Injury, Failure, Loss of kidney function, End-stage kidney disease (RIFLE), Acute Kidney Injury Network (AKIN), and Kidney Disease: Improving Global Outcomes (KDIGO). They use criteria such as change in serum creatinine (SCr) level, an increase of at least 1.5 times from baseline, and urine output of <0.5 ml/kg/h for at least six hours [7] [8].

2. Pathophysiology

The pathophysiology of CSA-AKI is multifactorial and thus far is not fully under-

stood. Several major pathways may be involved, including renal hypoperfusion, ischemia-reperfusion injury, activation of the inflammatory cascade, oxidative stress, nephrotoxin exposure, and genetic polymorphism; all of these can occur at any time during the perioperative period [9].

Renal hypoperfusion can occur throughout the perioperative period due to hypotension, decreased cardiac output, sympathetic stimulation, the administration of vasoconstrictive medications, and activation of the renin-angiotensin-aldosterone system. These events can interfere with renal autoregulation and reduce glomerular filtration rate [10] [11].

Cardiopulmonary bypass is associated with non-pulsatile flow, altered hemodynamics, decreased oxygen delivery, inflammation, and oxidative stress [10] [11]. Renal perfusion while on cardiopulmonary bypass is directly proportional to mean arterial pressure [12]. Rewarming from cardiopulmonary bypass provides a period of time when the kidney is susceptible in the renal medulla, and can exceed available supply [13].

Perioperative medications associated with nephrotoxicity include antibiotics, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, non-steroidal anti-inflammatory drugs, diuretics, and intravascular contrast agents [14].

Patient factors associated with AKI after cardiac surgery are similar to those associated with AKI in other patient populations [15]. These factors include the following preoperative and intraoperative characteristics: chronic kidney disease, advanced age, diabetes, anemia, heart failure, and hypotension. Findings within cardiac surgery overlap mechanistically with other forms of AKI [15]-[17].

Patients undergoing cardiac surgery are particularly susceptible due to the unique physiology and underlying procedures involved, including aortic cross-clamping (ACx) and CPB, as well as the use of frequent transfusions and vasopressors [18] [19].

3. Biomarkers

Current diagnostic criteria for cardiac surgery-associated AKI utilize increases in serum creatinine and decreases in urine output. While creatinine provides a good approximation of glomerular filtration rate when kidney function is normal, its accuracy is diminished in non-steady-state conditions such as the perioperative period [20].

In 2014, the U.S. Food and Drug Administration (FDA) approved the production of tissue inhibitor of metalloproteinases-2 (TIMP2) and insulin-like growth factor-binding protein 7 (IGFBP7) markers, which are involved in cell cycle arrest at the time of tubular epithelial cell growth phase, for their usefulness in the early detection of moderate-to-severe AKI defined as KDIGO stages 2 and 3 [21]. The product of both markers can be detected as early as four hours after surgery, and a decrease in these markers was the strongest predictor of kidney recovery. TIMP2 and IGFBP7 can be a bedside test, as they are easily measured with the FDA-approved Point of Care (NephroCheck) kit [21].

Systolic blood pressure (SBP), ejection fraction, cardiac index, stroke volume (SV), and cardiac power (CP) have been found to be associated with mortality in multivariate analysis [22]. Other studies have shown that heart rate (HR) variation can be used to predict the risk of septic patients developing septic shock and multiple organ dysfunction [23]. However, since their clinical presentation does not predict their deterioration, it is crucial to find more objective biomarkers or parameters that predict the prognosis of patients with septic shock [24].

Cardiac energy expenditure measured during exercise reflects the maximum cardiac output the heart can achieve. Comparing maximum cardiac power output with cardiac power output at rest represents the heart's cardiac reserve [25].

The heart is a pump; it consumes energy and produces work. The pumping power of the heart can be assessed by the CP, that is, the relationship between the mean arterial pressure (MAP) and the cardiac output (CO, measured in flow). With this, we obtain the measurement of the capacity of the heart to impart energy to the arterial system, which maintains a flow with two components, one pulsating and the other constant [26].

The determination of cardiac power (CP) dates back to 1969, when Bergel *et al.* published an assessment of the mechanical energy used during the pumping function of the ventricles. Despite having been applied by other researchers, its use in cardiology practice was not popular at that time. This was possibly because it was considered a mere variable or an index proposed as an ideal of contractility or ventricular function capacity. Authors such as Tan *et al.* point out the importance of considering it as an index of cardiac reserve and also as an indicator of the functional capacity of the heart, which makes it important to apply it as a fairly specific measure in the field of clinical practice, indicating the overall ability of this organ to perform its function [26].

Cardiac power can be represented by how well it can deliver hydraulic energy to maintain a circulation that can meet the most demanding physiological stresses. The variable that represents this entity is the cardiac power expenditure at peak stress, which can be calculated by the CO and MAP, for example, at maximal exercise. The average CP value is approximated by multiplying the CO (in L/min) with the MAP (mmHg), and a factor to convert to watts ($CP = CO \times MAP \times 2.2167 \times 10^{-3}$ Watts or CP (W) = MAP × CO/451) [27].

According to Fick *et al*, peak oxygen consumption rate (VO_2) is presumed to be an important prognostic factor that provides an indirect measure of CO, since peak VO_2 is derived from the product of CO and the arteriovenous difference in oxygen content ($C(av)O_2$). Normal values for cardiac output are between 2 and 5 L/minute. To use this method, O_2 is used as an indicator, thus deriving the following formula: $CO = (DavO_2 \times 100/CaO_2)/DavO_2$ [28].

The delta cardiac power (ΔCP) could be defined as the spontaneous magnitude of the basal cardiac power at maximum under stress conditions; it is used in various studies as an objective value of cardiac contractile reserve to deduce the prognostic and predictive value of unfavorable evolution [28].

AKI represents a potentially life-threatening complication in patients following cardiac surgery, often leading to an increased risk of death. In patients with severe coronary artery disease, surgical revascularization using HNSCC is currently the preferred perfusion technique for cardiopulmonary bypass (CPB) in most centers worldwide.

However, the use of HNSCC circuits has been associated with varying degrees of systemic inflammatory response syndrome (SIRS), possibly contributing to adverse clinical outcomes such as AKI.

This study is justified because accurate preoperative risk prediction of perioperative complications such as AKI can better inform patients and their families about their risk before surgery, assist with planning resource requirements, and assist with cohort enrichment. It may offer a potential therapeutic target to reduce risk.

4. Methods

This study included all clinical records of adult patients, regardless of gender, who underwent cardiac surgery with cardiopulmonary bypass between January 2023 and January 2024. To ensure consistency in diagnostic criteria, the definition of acute kidney injury (AKI) was reviewed according to the KDIGO guidelines: an increase in serum creatinine > 0.3 mg/dL within 48 hours of the initial insult; or an increase > 1.5% from baseline, known or presumed to have occurred within the 7 days prior to the initial insult; or urine output < 0.5 mL/kg/hour for 6 or more hours.

From the selected cases, observation units and variables documented in each clinical record were systematically collected through comprehensive data abstraction.

Inferential analysis was performed using the area under the receiver operating characteristic curve (AUROC). Based on odds ratio (OR) values, a scoring system was developed to predict mortality. The risk probability coefficient was calculated by constructing a 2 × 2 contingency table and applying the Chi-square test, with statistical significance established at $p < 0.05$. Data analysis was conducted using SPSS version 24.0.

This research project was conducted in strict adherence to international and local research ethics guidelines, as well as the Mexican General Health Law on Research.

5. Results

During the study period, 40 records of patients undergoing cardiac surgery with cardiopulmonary bypass were identified. No reasons were identified for their exclusion from the analysis. The clinical characteristics of the selected patients are presented in Table 1.

The mean age was 65.83 ± 9.90 years, with a 25.0% female patient distribution versus a 75.0% male distribution (3:1 ratio).

Cr records reported mean baseline Cr levels of 1.11 ± 0.38 mg/dL, admission Cr levels of 1.66 ± 0.77 mg/dL, 24-hour Cr levels of 2.21 ± 1.07 mg/dL, and 48-

hour Cr levels of 2.39 ± 1.16 mg/dL. Based on these, it was found that AKI developed in 34 patients. The stage distribution is presented in Figure 1 below. Stage I AKI was observed in 7.5%, stage II in 17.5%, and stage III in 60.0% of patients.

Table 1. Clinical characteristics of the patients selected for the study.

	Average, frequency	*SD*	*%*
Age	65.83	9.90	
Sex			
Feminine	10		25.00%
Masculine	30		75.00%
SCr basal (mg/dL)	1.11	0.38	
SCr income (mg/dL)	1.66	0.77	
SCr 24 h (mg/dL)	2.21	1.07	
SCr 48 h (mg/dL)	2.39	1.16	
AKI			
KDIGO I	3		7.50%
KDIGO II	7		17.50%
KDIGO III	24		60.00%

%: Percentage. SD: Standard deviation. GBS: Glasgow-Blatchford Scale.

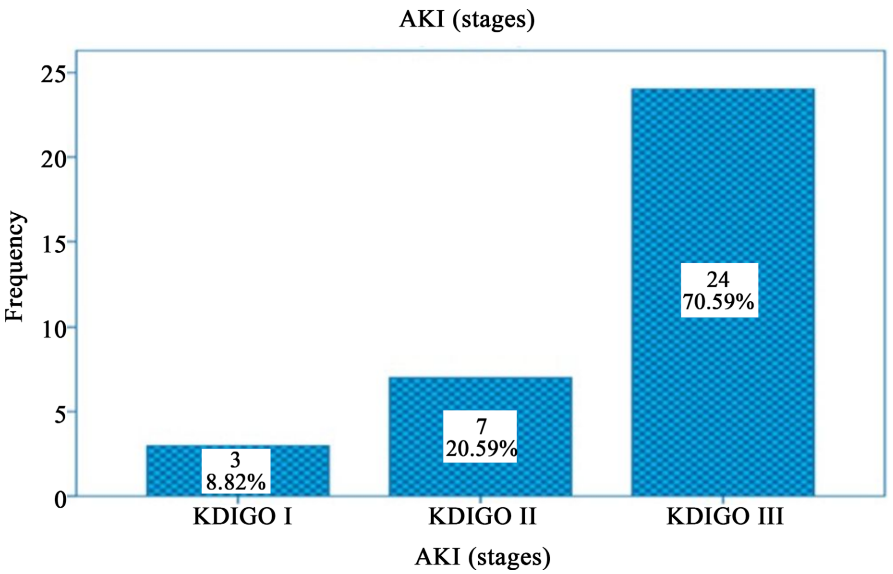

Figure 1. Distribution of AKI by stage in the study population.

Hemodynamic measurements were collected from the study population upon admission to the ICU and at 48 hours (Table 2). The CO at admission was 3.01 ± 1.12 L/min with a PC of 0.51 ± 0.26 Watts, while at 48 hours it was 2.88 ± 0.93 L/min with a PC of 0.51 ± 0.23. The average ΔPC was 0.01 ± 0.18 Watts. The average length of stay in the ICU was 4.55 ± 2.26 days.

Table 2. Hemodynamic measurements.

	Average	*SD*
Input GC (L/min)	3.01	1.12
Income SBP (mmHg)	103.30	19.19
Income DBP (mmHg)	58.60	13.16
Income MAP (mmHg)	73.50	14.08
PC input (Watts)	0.51	0.26
GC 48 hrs (L/min)	2.88	0.93
SBP 48 hours (mmHg)	106.35	20.65
DBP 48 hours (mmHg)	61.25	12.91
MAP 48 hrs (mmHg)	76.33	14.80
PC 48 hrs (Watts)	0.51	0.23
Delta PC 48 hours	0.01	0.18
Days in ICU	4.55	2.26
Input GC (L/min)	3.01	1.12

Receiver operating characteristic curve analysis was performed for CP as a predictor of AKI in patients undergoing cardiac surgery with extracorporeal circulation, identifying poor to no predictive capacity with statistically significant findings (AUC = 0.93, 95% CI 0.50 - 0.197, $p = 0.002$) (Table 3) (Figure 2).

Table 3. AUC test of PC at admission to predict AKI in the study population.

COR Curve - PC Income (Watts)			95% asymptotic confidence interval	
Area	Standard Error	Asymptotic Significance	Lower Bound	Upper Bound
0.93	0.053	0.002	0	0.197

An ideal cut-off value for CP at admission of 0.46 Watts was established, and the association analysis was performed, where up to two times more association of PC values < 0.46 with the development of AKI at any stage was observed, with statistically significant findings (OR 2.00, 95% CI 1.42 - 2.79, $p = 0.022$) (Figure 3).

6. Discussion

Our study is relevant because CS-AKI is a complex disease spectrum. Identifying, preventing, and modifying surgical and patient risk factors can help reduce cases and, therefore, the disease burden. However, diagnosis remains a challenging area, and the use of novel biomarkers appears more promising for identifying

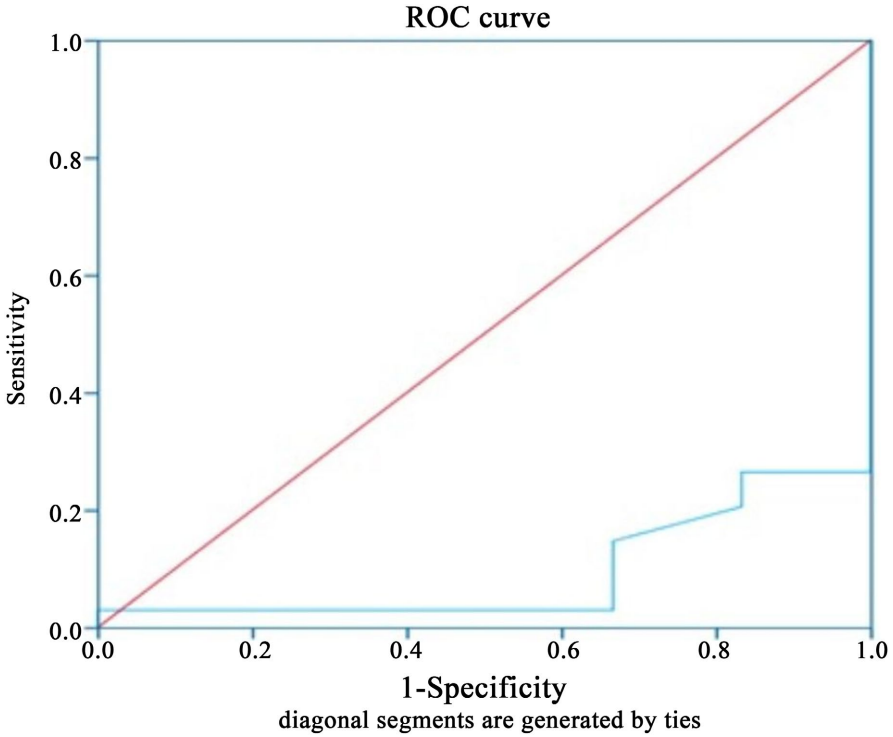

Figure 2. AUROC diagram of PC at admission to predict AKI in the study population.

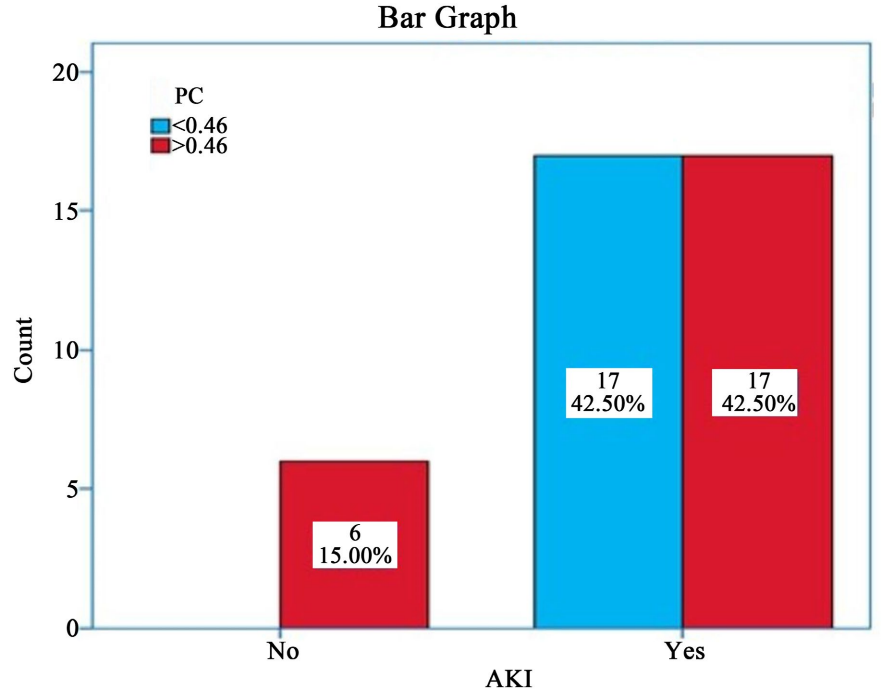

Figure 3. Distribution of AKI according to the CP cut-off value upon admission to the ICU in the study population.

at-risk patients earlier than conventional methods, such as serum creatinine levels and urine output measurements.

In patients with AKI after cardiac surgery, new biomarkers have been recog-

nized as reliable diagnostic indicators, predicting adverse outcomes and even mortality from postoperative AKI.

The use of biomarkers for preoperative risk stratification is not new. In fact, the Canadian Society of Cardiology Guidelines on Perioperative Cardiac Risk Assessment and Management in Patients Undergoing Noncardiac Surgery strongly recommends natriuretic peptide measurement to improve preoperative risk stratification of adverse cardiac outcomes in at-risk patients.

Han *et al.* found that the survival rate of patients with oliguric AKI was significantly lower than that of those with non-oliguric AKI. Oliguric AKI, along with sustained hypotension, the number of failing organs, and the need for dialysis, was a risk factor closely associated with mortality.

Meanwhile, Tseng *et al.* found that the development of CS-AKI was observed in 163 patients (24.3%) during the first postoperative week. Regarding the efficacy of the single model that most accurately predicted outcome, the RF model exhibited the highest AUC (0.939, 95% confidence interval [CI]: 0.772 - 0.898), while the AUC (0.843, 95% CI: 0.778 - 0.899) of the joint model (RF + XGboost) was even higher than that of the RF model alone.

The high rate of CS-AKI in this study could be due to a more severe presentation to the referral hospital, thus increasing the likelihood of mortality due to a delay or failure to achieve hemodynamic stability in patients undergoing cardiac surgery with cardiopulmonary bypass who are admitted to the ICU, likely associated with supportive care. Furthermore, there is a widespread lack of available interventional measures, such as the implementation of effective supportive care, and this may have contributed to the high incidence of CS-AKI.

This may reflect the observed impairment among these patients, but it could also reflect that some patients did not receive care according to guidelines. Therefore, the incidence of CS-AKI observed in our study is much higher compared to that in developed countries. Factors that may have contributed to the high mortality rate observed include greater disease severity, disposition status, and gaps in the management of these patients in the current setting due to non-medical causes.

The advantage of our study is the use of hemodynamic values and CP to predict CS-AKI. Although several risk factors have been identified using previously used risk scoring models, such as preoperative HGB, preoperative renal function, age, operative time, left ventricular ejection fraction, body mass index, and hypertension, and the recognition of intraoperative urine output, dynamic changes in hemodynamic characteristics are important risk factors that have been ignored by traditional risk scoring models.

Surprisingly, very few studies have studied biomarkers in cardiac surgery patients with cardiopulmonary bypass in such a heterogeneous manner as the present one. This could be important since hemodynamic-based variables are recognized as important variables in addition to clinical variables for identifying patients at high risk for CS-AKI. Furthermore, their variation over time can guide early resuscitation, as demonstrated by the concept of Cr clearance, which repre-

sents an independent prognostic factor that provides additional critical information.

Another factor to consider is that the definition of AKI used is broadly consistent with the creatinine-based Kidney Disease Improving Global Outcomes (KDIGO) criteria. However, its use of a 48-h sliding window, in which an increase in creatinine over a 48-h period within 7 days of surgery could define AKI, may be particularly susceptible to misclassification in patients undergoing cardiac surgery.

Although the stated objectives were met, this study had several limitations. First, the study was conducted in a heterogeneous group of ICU patients. Second, the small population size may have underestimated or overestimated the incidence observed in the present results.

7. Conclusions

This study demonstrates that cardiac power is a useful and reliable parameter for predicting the onset of acute kidney injury (AKI) in patients undergoing cardiac surgery with cardiopulmonary bypass. The findings confirm the alternative hypothesis, showing a statistically significant association between reduced cardiac power values and the development of AKI.

Upon admission to the intensive care unit, patients presented with a mean cardiac output of 3.01 ± 1.12 L/min and a cardiac power of 0.51 ± 0.26 Watts. These initial values allow for the establishment of a hemodynamic profile which, in combination with other clinical factors, may contribute to early risk stratification.

The incidence of AKI reached up to 85.0%, with a predominance of stage III AKI, reflecting the high vulnerability of this population and the need to implement intensive prevention and monitoring strategies. Notably, a cardiac power < 0.46 Watts was associated with a twofold increased risk of developing AKI, regardless of stage, with a higher prevalence in stage III.

These results suggest that cardiac power could be integrated as a prognostic marker in clinical practice, enabling early intervention in patients at higher risk. Furthermore, they reinforce the importance of advanced hemodynamic monitoring in the context of cardiac surgery with cardiopulmonary bypass, given its direct impact on renal function and postoperative outcomes.

In conclusion, cardiac power emerges as a clinically valuable parameter for predicting acute kidney injury in patients undergoing cardiac surgery, with significant implications for the prevention of complications and the improvement of clinical outcomes.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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