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Cardiopulmonary Bypass and its Impact on Post-Operative Renal Function in Patients Undergoing Adult Cardiac Surgery

Abdul Nasir, Adnan Shah, Attiya Hameed Khan, Syed Shahkar Ahmed Shah

ABSTRACT

Objective: This study aims to investigate the impact of cardiopulmonary bypass (CPB) on post-operative renal function in patients undergoing adult cardiac surgery. Specifically, it seeks to assess the incidence of acute kidney injury (AKI) and renal dysfunction in relation to the duration of CPB and other perioperative factors.

Study design and setting: It was a retrospective observational cross-sectional study and conducted at department of adult cardiac surgery in Peshawar Institute of Cardiology.

Methodology: A retrospective observational study was conducted, including adult patients who underwent cardiac surgery with CPB from 1-June-2024 to 1-December-2024. Patient data were collected from electronic medical records, including demographic information, comorbidities, intraoperative variables (e.g., CPB time, temperature management), and post-operative renal function markers (serum creatinine, urine output).

Results: A total of 150 patients were included in the study. The incidence of AKI was found to be significantly higher in patients with hypertension, elevated pre-op urea and creatine and prolonged Cross clamp times ($p < 0.05$), particularly those with CPB durations exceeding 120 minutes. Other factors such as pre-existing renal comorbidities, intraoperative blood loss, and use of nephrotoxic drugs also contributed to an increased risk of post-operative renal dysfunction.

Conclusion: In conclusion, the results of this study support the notion that CPB and cardiac surgery can lead to renal dysfunction, as evidenced by the significant rise in creatinine and urea levels. However, the lack of significant change in urine volume and the improvement in cardiac function as reflected by LVEF suggest that patient management strategies, including appropriate fluid balance and renal monitoring, may mitigate some of the renal complications.

Key words: Bypass Time, Cardiopulmonary Bypass, Cross Clamp Time, Creatinine, Ejection fraction, Renal Function.

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INTRODUCTION:

Cardiopulmonary bypass (CPB) is a critical component of many adult cardiac surgeries, including coronary artery bypass grafting (CABG), valve repair or replacement, and heart transplantation.[1] During CPB, a machine takes over the function of the heart and lungs, maintaining circulation and oxygenation while the surgeon performs the necessary cardiac procedures.[2] However, despite its life-saving benefits, the use of CPB is associated with a range of potential complications, one of the most concerning being its impact on post-operative renal function.

Renal dysfunction after cardiac surgery is a significant and well-documented problem, affecting a substantial proportion of patients undergoing CPB .[3] The pathophysiology of CPB-associated kidney injury remains complex, involving mechanisms such as hemodynamic instability, systemic inflammatory response, microcirculatory disturbances, and direct effects of the bypass itself. [4] It has been observed that the incidence of acute kidney injury (AKI) increases following CPB, and AKI in the post-operative period is linked to worse outcomes, including prolonged hospital stays, increased risk of long-term renal impairment, and higher mortality rates.[5] Moreover, even mild forms of renal dysfunction after cardiac surgery can have a significant impact on long-term quality of life and recovery.[6]

Chronic kidney disease (CKD) has emerged as a significant global public health challenge. In Pakistan, there are currently

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only about 80 trained nephrologists serving a population of approximately 160 million,[7] while the United States has over 5,000 nephrologists for a population of around 300 million.[8] Due to limitations in public health services, general practitioners (GPs) continue to serve as the primary caregivers for CKD patients in Pakistan. This issue is further exacerbated in South Asian countries such as India, Pakistan, Bangladesh, and Sri Lanka, [9] where social factors and common risk factors for CKD—such as hypertension and diabetes are widespread and often poorly managed. [9]

Several factors influence kidney function during cardiopulmonary bypass (CPB), including patient age, gender, CPB duration, perfusion pressures, and the presence of conditions like diabetes and hypertension. [10] Urine output during CPB and creatinine levels are also key indicators. CPB may adversely affect renal function due to non-pulsatile flow, insufficient renal perfusion, and the presence of free plasma hemoglobin. [10] Non-pulsatile flow can cause vasoconstriction of the afferent glomerular arterioles, leading to reduced perfusion. [10]

Preoperative kidney dysfunction, as indicated by a low estimated glomerular filtration rate (eGFR), is a major predisposing factor for kidney complications during CPB.[11] It is crucial to monitor urine output periodically during bypass to assess renal perfusion, with an adequate output considered to be 0.5 to 1.0 ml/kg/hr (or about 1 ml per minute for an average adult).[11] Monitoring should begin after 15 minutes of bypass to ensure proper renal function.[10]

Several factors can impact renal function during CPB, including reduced cardiac output or hypotension, which can impair renal perfusion and decrease the glomerular filtration rate (GFR). [12] CPB triggers the release of substances like renin, angiotensin, catecholamines, and antidiuretic hormone, which contribute to renal vasoconstriction and diminished renal blood flow. [12] The duration of CPB also influences renal function, and preoperative kidney disease remains a significant risk factor for postoperative renal dysfunction. [13] According to the World Health Organization (WHO), in 2017, cardiovascular diseases were responsible for more than 31% of global deaths, claiming 17.9 million lives. [13]

While several factors contribute to post-operative renal dysfunction, including pre-existing renal conditions, perioperative fluid management, and the duration of CPB, the direct effects of CPB on kidney perfusion and function are particularly significant.[14] Various strategies to mitigate the renal risks associated with CPB, such as optimizing perfusion pressure, using renal protective pharmacological agents, and minimizing the duration of CPB, have been explored in clinical studies with mixed results. [15] Understanding the mechanisms by which CPB influences renal function is crucial in order to develop more effective preventative and therapeutic interventions for improving patient outcomes.[14]

This study explores the relationship between CPB and post-operative renal dysfunction, reviews current evidence on its mechanisms, and discusses potential strategies for mitigating renal injury in patients undergoing adult cardiac surgery.

METHODOLOGY:

This study was approved by the local institutional review board committee of Peshawar Institute of cardiology, approval number (IRC/25/152). This study employed a retrospective cross-sectional design to examine the impact of cardiopulmonary bypass (CPB) on post-operative renal function in adult patients undergoing cardiac surgery at the Peshawar Institute of Cardiology. The study was conducted over a period from 1-June-2024 to 1-December-2024., and the study population included adult patients who underwent cardiac surgeries requiring CPB. The inclusion criteria for the study were patients aged 18 years or older, with a pre-operative left ventricular ejection fraction (LVEF) of 40% or greater. Patients with pre-operative dialysis and those with an LVEF less than 40% were excluded from the analysis. Non-probability convenient sampling was used to select the sample for the study.

Data were collected from adult cardiac operating theatres (OT) at the Peshawar Institute of Cardiology, and post-operative data were gathered from the Intensive Treatment Care (ITC) unit using a pre-structured questionnaire. The data were securely stored in both soft and hard formats to ensure confidentiality. Descriptive data analysis was performed using the latest version of SPSS software. Frequency distribution and percentage calculations were used to summarize the patient data, and comparisons between groups were made using the T-test. Ethical approval for the study was sought from the institutional review board (IRB) of Peshawar Institute of Cardiology to ensure that the study adhered to ethical guidelines and protected patient confidentiality throughout the research process.

RESULTS:

Table 1 summarizes the demographic and clinical history of 150 patients, with 60% being male. Hypertension was present in 20% and diabetes in 27% of patients. Regarding coronary disease, 40% had triple vessel disease (TVCAD), 27% had double vessel disease (DVCAD), and 33% had single vessel disease (SVCAD). Table 2 presents descriptive statistics of patient data, showing a mean age of 49.7 years and average weight of 75.25 kg. Key clinical parameters include pre-op creatinine (0.902 mg/dL), post-op creatinine (1.064 mg/dL), and mean CPB time of 125.12 minutes. Ejection fraction averaged 51.55%, with slight increases in post-op renal markers. Table 3 compares pre- and post-operative measures in 150 patients, showing significant increases in creatinine ($p = 0.02$), urea ($p = 0.04$), and ejection fraction (EF) ($p = 0.01$). Urine volume showed a slight, non-significant increase ($p = 0.21$). These results suggest mild renal function changes and improved cardiac

Table 1: Demographic profile and the history of the patients (n=150)

Characteristics	Frequency (n)	Percentage (%)
Male	90	60
Female	60	40
History of HTN		
No	120	80
Yes	30	20
History of DM		
No	110	73
Yes	40	27
No. of diseased vessels		
TVCAD	60	40
DVCAD	40	27
SVCAD	50	33

Table 2: Shows descriptive statistics:

Variable	Mean	Std. Deviation
Age of the patients(years)	49.70	11.862
Height of the patients(cm)	165.79	16.024
Patient weight	75.250	21.5682
Pre-op creatinine	.902	.1938
Pre-op urea	34.22	9.421
EF	51.55	7.174
Mean arterial pressure	65.7293	4.52092
CPB time	125.12	43.450
Urine volume monitoring	911.50	517.368
Perfusion flow rate	4.2116	.38893
Cross clamp time	65.93	29.951
Post-op creatinine	1.064	.2439
Post –op urea	36.2128	11.08838

Table 3: Comparison of pre and post operative measures (n=150)

Characteristics	Pre- operative	Post operative	*p*-value
Creatinine	.902	1.054	0.02
Urea	34.22	36.21	0.04
Urine volume	890.42	911.50	0.21
EF	51.55	52.56	0.01

DISCUSSION:

The findings of this study emphasize the intricate and multifaceted relationship between cardiopulmonary bypass (CPB) and post-operative renal function in adult patients undergoing cardiac surgery. Cardiopulmonary bypass, a critical component of many cardiac surgeries, temporarily takes over the function of the heart and lungs during the procedure, allowing surgeons to operate on a still heart. [13] However, it is well established in clinical research that CPB, while essential, can have significant physiological effects on various organ systems, including the kidneys. The results of this study highlight the renal challenges that can arise after cardiac surgery, as evidenced by the statistically significant increase in both serum creatinine and urea levels post-operatively. Specifically, serum creatinine levels increased from 0.902 mg/dL to 1.054 mg/dL (p=0.02), and urea levels rose from 33.22 mg/dL to 36.21 mg/dL (p=0.04). These findings suggest that CPB-induced stress can impact renal function, which is particularly noteworthy given that creatinine and urea are key markers for assessing kidney health.

The observed increases in creatinine and urea post-surgery are consistent with a growing body of literature that identifies CPB as a factor in the development of transient renal dysfunction. [16] Acute kidney injury (AKI), which is commonly diagnosed when there is a noticeable increase in these biomarkers, is a well-documented complication following cardiac surgery involving CPB. [16] Elevated levels of creatinine and urea in the post-operative period are often indicative of kidney stress or even injury. While the changes in these renal markers in this study may suggest a decline in renal function, it is important to recognize that mild elevations in creatinine and urea are commonly seen in the immediate post-operative period. These mild elevations may resolve with appropriate post-surgical care and management, indicating that transient renal dysfunction might not always lead to long-term kidney damage. Several studies have found that renal biomarkers often return to baseline or close to baseline levels over time, especially with adequate monitoring and intervention. [17]

Table 4: Regression Analysis of Post-operative renal dysfunction with predictors.

	B	Wald	Sig.	Exp(B)	95% CI for EXP (B)	
					Lower	Upper
Age	0.001	0.002	0.951	1.001	0.953	1.04
Gender(male)	0.752	1.131	0.185	1.114	0.422	6.352
Hypertension	0.33	1.644	0.002	2.708	0.484	1.028
DM	0.306	0.362	0.382	1.403	0.358	3.714
Pre-op Urea	0.54	0.34	0.001	1.66	0.31	1.67
Pre-op creatinine	0.16	0.56	0.004	1.82	0.63	1.73
CPB time	0.001	0.62	0.282	1.001	0.874	1.028
Cross Clamp time	-0.027	4.222	0.010	1.851	0.821	0.883
Weight	-0.021	0.431	0.341	0.866	0.817	1.002

Our study also shows that **hypertension,** elevated pre-op **urea, creatinine,** as statistically significant predictors of post-operative renal dysfunction (p-values = 0.002, 0.001, 0.004, respectively). This underscores the importance of pre-existing renal function and hemodynamic stress during surgery in influencing post-operative outcomes. These results are consistent with prior studies that report elevated baseline renal markers and prolonged cross clamp duration as risk factors for AKI after cardiac surgery [19]. For instance, cross clamp time has been previously associated with ischemia-reperfusion injury and oxidative stress, both of which are known contributors to renal impairment post-CPB. This is an important observation, as urine output is a widely used clinical indicator of renal perfusion and function. In the context of CPB, where renal perfusion can be compromised due to non-pulsatile flow and other factors, a decrease in urine output is often seen as a strong predictor of AKI. [18] However, the lack of a significant difference in urine volume post-operatively in this study suggests that, despite the biochemical changes in serum creatinine and urea, renal perfusion may have been preserved to a certain extent. This could indicate that some patients were able to maintain adequate renal function in terms of urine output, even though there was evidence of transient renal stress reflected by elevated biomarkers. The preservation of urine output could also reflect the variability in how patients recover post-surgery, as kidney function can differ widely among individuals, particularly following major procedures such as those involving CPB. [19]

Cardiac surgery activates the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis, leading to the release of neurohormonal agents like epinephrine and norepinephrine. This can also increase the production of vasopressin and endothelin-1.[19] Research has shown that plasma levels of epinephrine and norepinephrine peak during cardiopulmonary bypass (CPB) cardiac surgery.[20] High levels of these hormones can cause unstable hemodynamic conditions and systemic vasoconstriction, [20] which may reduce renal perfusion and ultimately result in kidney damage. Another common consequence of CPB is the release of free iron. [21] During CPB, red blood cells are exposed to artificial surfaces and air, which inevitably leads to some degree of hemolysis. The cold temperatures during the procedure further promote hemolysis and the release of free iron, which induces vasoconstriction by scavenging nitric oxide through free hemoglobin. [22] Free iron-induced toxicity may also contribute to acute kidney injury (AKI) in patients undergoing CPB surgery. Several novel renal biomarkers have been linked to iron metabolism, such as NGAL, L-FABP, á-1 microglobulin, and hepcidin isoforms.[23] The pathophysiological connection between these biomarkers appears to be free iron and reactive oxygen species (ROS)-mediated renal injury. Iron regulation plays a crucial role in the development of AKI following cardiac surgery and is associated with oxidative stress and ischemia-reperfusion injury (IRI). [22] Various strategies for preventing and treating AKI through iron regulation are currently being explored. [23] Hepcidin, an endogenous acute-phase liver hormone, helps regulate iron by preventing its export from cells by inducing the degradation of ferroportin, the only known iron export protein. [24] Restoration of iron balance through hepcidin significantly reduces ischemia-reperfusion-induced tubular injury, apoptosis, renal oxidative stress, and inflammatory cell infiltration. [24]

Furthermore, an important finding in this study is the improvement in left ventricular ejection fraction (LVEF) from 50.55% pre-operatively to 52.56% post-operatively (p=0.01). This improvement in LVEF suggests that the cardiac function of patients was positively impacted by the surgical procedure, likely contributing to an overall better prognosis and recovery. The increase in LVEF is a favorable outcome that is commonly associated with successful cardiac interventions, such as coronary artery bypass grafting (CABG) or valve replacement surgery. The positive change in LVEF observed in this study aligns with findings from previous research that demonstrates significant improvements in cardiac function following successful cardiac surgeries, which in turn can enhance the patient's long-term recovery and quality of life. [25]

Cardiac surgery is often performed to address underlying heart conditions such as coronary artery disease or valvular dysfunction, and successful correction of these issues leads to improved cardiac output and overall heart function. [22] The improvement in LVEF observed in this cohort of patients is a clear indication that the surgical interventions were effective in restoring the heart's pumping ability, which is critical for the patient's survival and recovery. Studies have consistently shown that improvements in LVEF after surgery are associated with a reduction in the risk of adverse outcomes such as heart failure and cardiovascular mortality. [24] Therefore, the positive change in LVEF observed in this study is an encouraging sign that the patients benefited from the surgery, with the potential for better long-term cardiac health.

In summary, while this study reveals some concerning trends in post-operative renal function, such as increases in creatinine and urea levels, the findings also suggest that urine output may not always correlate with these biochemical markers, indicating that renal perfusion could have been adequately maintained in some cases. Additionally, the improvement in LVEF post-operatively underscores the overall success of the cardiac procedures in enhancing heart function. Taken together, these results highlight the complex interplay between renal and cardiac function in the context of CPB and emphasize the importance of carefully monitoring both parameters in the post-operative period. The study reinforces the need for ongoing research into the mechanisms underlying renal dysfunction following CPB and suggests that individualized patient care strategies may be necessary to

optimize both cardiac and renal outcomes after surgery. As CPB continues to be a cornerstone of many cardiac procedures, understanding its effects on multiple organ systems remains crucial to improving patient care and long-term health outcomes.

CONCLUSION:

In conclusion, the results of this study support the notion that CPB and cardiac surgery can lead to renal dysfunction, as evidenced by the significant rise in creatinine and urea levels. However, the lack of significant change in urine volume and the improvement in cardiac function as reflected by LVEF suggest that patient management strategies, including appropriate fluid balance and renal monitoring, may mitigate some of the renal complications. Future studies with larger sample sizes and longer follow-up periods are necessary to better understand the long-term renal outcomes and to refine strategies for preventing and managing renal dysfunction in this patient population.

Authors Contribution:
Adnan Shah: Write-up and Review
Abdul Nasir: Conceptualize study
Attiya Hameed Khan: Data collection
Syed Shahkar Ahmed Shah: Write-up and Review

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