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REVIEW

Beyond the Scalpel: Understanding Acute Kidney Problems after Heart Surgery

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ABSTRACT

Acute renal injury (ARI) post cardiac surgery leads to substantial difficulty in going about the treatment, that often results in aggravated morbidity and mortality rates. Through this manuscript, we have the opportunity to cover a basic assessment of ARI complexity, including comorbidities which have been categorized as preoperative, intraoperative, and postoperative factors. The risk of all these ailments may also increase which is due to advanced age and comorbidities such as diabetes and hypertension and may lead to procedures like cardiopulmonary bypass (CPB) and duration of cardiopulmonary bypass. The cardiac procedures under discussion like the Coronary artery bypass surgery (CABG) and valvular surgeries are instigators of ARI with separate risk profiles discovered for each intervention type. Secondly, the paper advocates approaches and approaches to controlling the risk of both eosinophilia and erythropoietin and most importantly the techniques used in fluid management. The conversation centres on what is the clinical effect of AKI; it examines how it negatively influences patient recovery by requiring prolonged hospitalization, increased costs of healthcare, and progression of renal complications in the long-term. Moreover, early identification is followed as well as individualized postoperative care regimens are highlighted as the necessary components of the initial minimization of late postoperative ARI complications. Through a thorough assessment of the current knowledge together with the latest development in interventions, this paper offers a unique and useful guild for clinicians and researchers that seek to enhance management of ARI and prevent its poor outcomes specifically in pediatric cardiac surgery.

Keywords: Acute renal injury, Cardiac surgery, Risk factors, Prevention strategies, Patient outcomes

1. Introduction

Acute renal injury or kidney failure takes place when there is a sudden loss of approximately 50% of the kidney's function reflected by the increasing serum creatinine level and also the fall of glomerular filtration rate (GFR) [1, 2]. Elevated risk of AKI manifested by nephrotoxins, metabolic abnormalities, ischemic injury, perfusion injury, the general presence of chronic diseases, inflammation, oxidative stress, sepsis, detectable gaseous particulate emboli, use of contrasted imaging agents and reduced renal flow can be considered second only to heart surgery [3]. To address this concern, numerous studies have been conducted to evaluate the occurrence of acute kidney injury following cardiothoracic surgery. One study conducted by Cruz and colleagues evaluated 133 patients with a clinical diagnosis of acute

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kidney injury and a clinical presentation suggestive of acute tubular necrosis [4, 5]. According to the kind of procedure, the incidence might range from 3% for thoracic surgery to 94% for cardiac transplants. With an ARI incidence of up to 80%, patients who require extracorporeal membrane oxygenation following heart surgery are at significant risk. RIFLE occur in 10% and 30% of juvenile patients who have had heart surgery, respectively [6].

Pre-, intra-, and postoperative risk factors for CSA-ARI include chronic kidney disease, albuminuria, hypertension, diabetes, geriatrics, and obesity. The etiology of CSA-ARI is complicated. Investigators most frequently employ the following classifications: AKIN, KDIGO, ESRD, RIFLE, Risk, Injury, Failure, Loss of renal function, and RIFLE [7, 8]. While KDIGO is predicated on the combination of RIFLE and AKIN having greater sensitivity to diagnosis AKI postoperatively as compared to RIFLE and AKIN, RIFLE and AKIN employ changes in serum creatinine levels to diagnose CSA-AKI [9, 10].

This review paper describes the advances and limitations in the current state of medical knowledge about risk prediction, and effective prevention of CSA-ARI. This review also discusses various novel and innovative prevention strategies for ARI linked to heart surgery. In a nutshell this study highlights the steps that can be taken to lessen the acute renal injury that results from heart surgery [11]. These steps can also dramatically reduce morbidity and medical costs, as well as avoid long-term cardiovascular disease, end-stage renal disease, and CKD.

1.1. Patient population and cardiac procedures

ARI following cardiac procedures is caused by a complex mechanism that includes tubular damage, endothelial dysfunction, microcirculatory dysfunction, production of microvascular thrombi, and intrarenal inflammation, all of which can change renal perfusion and cause ARI [12, 13]. Renal hypoperfusion can occur during surgery as a consequence of lowflow, low pressure, abrupt temperature drops, CPB use, and vasopressor use.

Various risk factors contribute to the growth of AKI post-cardiac surgery. These comprise demographic elements such age and gender, comorbidities such as DM, HTN, CKD, COPD, and cardiovascular diseases like IHD, as well as procedural factors like the duration of CPB and aortic cross-clamp time [14, 15]. Some studies propose predictive models to identify patients at increased risk of acquiring AKI after cardiac surgery. These models often incorporate a combination of patient demographics, comorbidities, procedural factors, and biomarkers such as serum creatinine, NGAL (neutrophil gelatinase-associated lipocalin), and interleukin 6. Additionally, AKI is linked with unfavourable consequences like deep sternal wound and extended ventilation, reoperation, and major complications [16, 17].

Across different study types (cohort studies, observational studies, meta-analyses, and randomized control trials), the incidence of CS-AKI varies. For instance, in cohort studies, the incidence ranges from 9.25% to 58.7%, while in meta-analyses, it ranges from 10.48% to 13.78% [18]. AKI post-Surgery is linked to higher rates of morbidity and death. Mortality rates among AKI patients vary across studies but are often more than to non-AKI patients (Table 1). Various of cardiac surgical procedures such as CABG, valvular surgeries (e.g., AVR, MVR, combined CABG and valvular surgeries, and other cardiac procedures such as closure of ASD and VSD. In that CABG appears to be the most prevalent type of surgery across the studies, followed by valvular surgeries and combinations of valvular with CABG surgeries. The data encompass research methodologies, such as, including cohort studies, observational studies, meta-analyses, and randomized controlled trials, with different numbers of samples ranging from 100 to 3605 patients [19, 22, 23]. Meta-analyses tend to have larger sample sizes compared to individual cohort or observational studies, permitting more thorough analyses and generalizable findings. The episode of AKI varies according to the procedure performed. For instance, in cohort studies and observational studies, the incidence ranges from 37 to 221 cases, while in meta-analyses and randomized controlled trials, it ranges from 9 to 296 cases. CABG + valvular surgery appears to have a relatively higher incidence of AKI compared to isolated CABG or valvular surgeries alone in several studies [20, 24–26] (Table 2).

There is a considerable variation in the death rates of various studies. In Meta-analyses the mortality rates range from 1.5% to 94.89%, and it tends to have a wider range of mortality rates, likely due to the inclusion of data from multiple studies [21, 27]. In Observational studies the mortality rates range from 18.5% to 75%, it generally reports higher mortality rates compared to cohort studies and RCTs. In cohort studies the mortality rate ranges from 3.0% to 13.5% and in RCTs the mortality is 3.0% (Table 3).

1.2. Risk factors and prevention

CS-AKI is a serious postoperative complication that can prolong the recovery process. The development of CS-AKI can be linked to a complicated interaction between procedure-related and patient factors. These

Table 1. Description of the study population who underwent cardiac surgery in various studies.

S. no.	Source	AKI	Non-AKI	Patient population with AKI	Outcome	Ref
1	Cohort study; N = 196	40	156	Male: 33 DM: 12 Ischemic heart disease: 22 HTN: 33 Preoperative CKD: 40 Inotrope use: 29 Drugs: 28 RRT: 2 Mortality: 2	One in 5 patients developed AKI post-cardiac surgery, with significantly increased morbidity and mortality.	[8]
2	Cohort study; N = 1508	885	623	Male: 720 DM: 494 HTN: 690 Stroke: 10 Deep sternal wound infection: 16 Prolonged ventilation: 63 Reopen: 29 Mortality: 34	AKI development after cardiac surgery. Patients with AKI were elderly, and more likely to be diabetic & and hypertensive. The proportion of AKI patients with RIFLE risk was greater compared to AKIN.	[11]
3	Cohort study; N = 6261	3497	2764	Male: 3554 Female: 2707 DM: 848 HTN: 3087 COPD: 690 NYHA class IV: 137 Mortality: 205	AKI development after cardiac surgery. The primary outcome was 28-day mortality in postoperative patients admitted to the ICU in different stages of AKI regarding different types of cardiac surgery.	[13]
4	Cohort study; N = 2310	1310	1000	Male: 736 DM: 250 HTN: 546 Hyperlipidaemia: 536 Chronic renal failure: 10 COPD: 2 Peripheral vascular disease: 8 Congestive cardiac failure: 82 NYHA class III – IV: 838 Previous MI: 322 Previous cardiac surgery: 4 Smoker: 422 Drugs: 492	AKI development after cardiac surgery. AKI and severe AKI after cardiac surgery as the primary and secondary outcomes. Preoperative serum creatinine, hyperlipidaemia, lipid-lowering agents, and assisted ventilation time are the predictors of both postoperative AKI and severe AKI.	[16]
5	Cohort study; N:3889	72	3817	Female:21 RACE White:66 Black:3 Asian:1 Others:2 Hypertension:61 Hyperlipidemia:60 Diabetes:30 COPD:12 Current smoker:22 NYHA class 3 & 4:28 CPB time: 152 Cross clamp time:113	AKI development after cardiac surgery. Increasing CPB duration is associated with post-operative AKI, particularly among those with pre-operative renal impairment. For patients with an estimated GFR of less than 30ml/min/1.73m^{2,} the risk increases exponentially with time.	[19]
6	Cohort study; N=1082	288	794	Female:86 Male:202 Diabetes:20 HTN:99 CPB time:186min	AKI developed after cardiac surgery. Age, gender, hypertension, CPB duration, intraoperative 5% bicarbonate solution red blood cell transfusion, and urine volume were identified as important factors.	[20]

(*Continued on next page*)

Table 1. (*Continued*).

S. no.	Source	AKI	Non-AKI	Patient population with AKI	Outcome	Ref
7	Cohort study; N=380	60	320	Female:8 Diabetes:14 Hypertension:51 COPD:12 Smoking; Ex-smoker:39 Current smoker:5 NYHA class; Class I:7 Class II:17 Class III:32 Class IV:4 Elective:33 Urgent:24 Emergency:3 Mortality: 8	In patients undergoing redo CABG, the occurrence of postoperative AKI is associated with increased 30-day mortality and major complications and with reduced long-term survival.	[21]
8	Observational Study; N = 400	37	363	Male: 33 Female: 4 DM: 10 HTN: 34 Dyslipidaemia: 29 Stroke: 5 CPB: 13 Atrial fibrillation: 16 NYHA class I & II: 29 NYHA class III & IV:8 Re-intubation: 7 Re-exploration: 1 Mortality: 50–60 %	AKI development after cardiac surgery. There is a need for quality improvement in the care of patients with CS-AKI in its most severe forms since mortality risks posed by the development of stage 3 AKIN.	[9]
9	Observational study; N = 134	66	68	Male: 32 Female: 34 Aortic cross-clamping time: 79.62 min CPB time: 103.71 min Urine output: 1.15 L	AKI development after cardiac surgery. Lower preoperative eGFR does not predict a higher incidence of CS-AKI compared to higher preoperative eGFR. Lower height is independently associated with a higher incidence of CS-AKI.	[10]
10	Observational study; N = 435	54	381	Female: 9 BMI: 44 DM: 18 HTN: 48 Peripheral-vascular disease: 22 Stroke: 3 COPD: 5 Dyslipidaemia: 34 Acute MI: 11 Left-ventricular dysfunction: 9 NYHA class III & IV: 12 Drugs: 72 RRT: 4 Smoking: 32 Mortality: 10	AKI development after cardiac surgery. Age, hypertension, low ejection fraction, eGFR, Euro SCORE II, and no intake of calcium channel blockers were independent risk factors for postoperative AKI. These factors provide an easy and accurate model to predict postoperative AKI in patients undergoing cardiac surgery.	[14]
11	Observational study; N = 443	221	222	Female: 72 HTN: 173 DM: 56 COPD: 45 MI: 49 LVEF: 56 Aortic cross-clamp time: 58 min CPB time: 90 min ICU stay: 2 Hospital length of stay: 13	AKI development after cardiac surgery. The use of standard RIFLE classification in patients undergoing cardiac surgery resulted in a 50% incidence of AKI. Patients with co-morbidities were associated with increased mortality at 1-yearpost-cardiac surgery.	[22]

(*Continued on next page*)

Table 1. (*Continued*).

S. no.	Source	AKI	Non-AKI	Patient population with AKI	Outcome	Ref
12	Observational study; N = 3605	856	2749	Male: 332 DM: 184 HTN: 321 Chronic kidney disease: 263	AKI development after cardiac surgery. Acute kidney disease after cardiac surgery is associated with 90-day mortality, 90-day renal prognosis, and 2-year follow-up.	[23]
13	Meta-analysis; N = 1601	318	1283	Male: 187 BMI: 318 DM: 52 HTN: 146 Hyperlipemia: 8 Previous surgery history: 145 Smoking: 9 NYHA class I: 29 NYHA class II: 73 NYHA class III: 189 NYHA class IV: 27	AKI development after cardiac surgery. Abnormal body mass index is an independent risk factor for predicting the occurrence of AKI after cardiac surgery.	[12]
14	Meta-analysis; N = 9656	2331	7325	Male: 26 RRT: 539 ICU stay: 161 Lower sCr: 1596 Hospital Stay: 1009 Mortality: 2212	AKI development after cardiac surgery. The worse outcomes of AKI in patients after cardiac surgery include higher RRT rates, mortality, and longer hospital length of stay than those in non-AKI patients.	[15]
15	Meta-analysis; N = 1372	153	1219	Males: 1101 DM: 302 HTN: 587 Dyslipidaemia: 656 Prior MI: 159 Smoker: 678 eGFR: $\leq$60 ml/min/1.73 m^2	AKI development after cardiac surgery. Among STEMI patients undergoing primary PCI, serum UA levels were found to be associated with a higher rate of AKI. It appears that patients with both preexisting moderate renal insufficiency (eGFR $\leq$ 60 ml/min/1.73 m 2) and hyperuricemia are at the highest risk for developing AKI.	[24]
16	Meta-analysis; N = 1575	165	1410	Mechanical ventilation: 1229 ICU stay: 1363 Hospital length of stay: 878 Mortality: 15	The primary outcome is the development of AKI. Secondary outcomes are mechanical ventilation duration, ICU stay, hospital length of stay, and in-hospital mortality within 30 days.	[25]
17	Meta-analysis; N = 2148	296	1852	Mechanical ventilation: 758 ICU stay: 762 Hospital length of stay: 762 Mortality: 711	AKI development after cardiac surgery. Dexmedetomidine has a positive effect on preventing AKI and postoperative delirium after cardiac surgery and significantly reduces the length of stay in the ICU.	[26]
18	Meta-analysis; N = 671	163	508	Male: 454 Smoking: 244 Previous cardiac surgery: 96 DM: 215 HTN: 453 Dyslipidaemia: 258 COPD: 73 Chronic kidney disease: 207 Congestive heart failure: 131 MI: 104 NYHA class I: 113 NYHA class II: 365 NYHA class III: 161 NYHA class IV: 32 Drugs: 684	AKI development after cardiac surgery. Intraoperative time- series is crucial for AKI prediction.	[18]
19	Randomized control trial; N = 100	9	91	Female: 2 HTN: 8 COPD: 3 Left ventricular dysfunction: 4 RRT: 3 Mortality: 3	The primary outcome is major adverse kidney events and the secondary outcome is AKI development after cardiac surgery. Biomarkers NGAL and interleukin 6 are useful in predicting AKI post-cardiac surgery.	[17]

Table 2. Examination of the types of cardiac procedures and their potential association with acute renal failure.

S. no.	Source	Type of surgery	AKI	Ref
1	Cohort study; N = 196	CABG: 13 Valvular surgery: 13 CABG + valvular surgery: 14	40	[8]
2	Cohort study; N = 1508	CABG: 1508	885	[11]
3	Cohort study; N = 2310	CABG + valvular surgery: 100 Elective surgery: 1014 CPB: 422	1310	[16]
4	Cohort study; N = 3889	Isolated CABG:21 Isolated AVR:13 Isolated MVR:4 AVR/CABG:15 MVR/CABG:4 AVR/MVR:5 MV repair:5 MV repair/CABG:5	72	[19]
5	Observational study; N = 400	CABG: 27 Valvular surgery: 4 CABG + Valvular surgery: 4 Other surgeries: 2	37	[9]
6	Observational study; N = 134	CABG/ CABG + valvular surgery: 3 ASD closure: 5 VSD closure: 3 Aortic valve replacement: 17 MVR: 23 DVR: 9 Other surgeries: 6	66	[10]
7	Observational study; N = 435	CABG: 435 Emergent surgery: 14	54	[14]
8	Observational study; N = 443	CABG: 80 Off-pump CABG: 21 Valvular surgery: 74 CABG + valvular surgery: 46	221	[22]
9	Observational study; N = 3605	CABG: 170 Valvular surgery: 216 CABG + Valvular surgery: 67 Cardiopulmonary bypass surgery: 385 Non-elective surgery: 7178	856	[23]
10	Meta-analysis; N = 671	CABG: 250 Valvular surgery: 347 CABG + valvular surgery: 74	163	[18]
11	Meta-analysis; N = 1575	CABG: 95 CABG + valvular surgery: 621 Aortic vascular surgery: 75	165	[25]
12	Meta-analysis; N = 2148	CABG: 170 CABG + valvular surgery: 743 AV repair: 15 Aortic vascular surgery: 75 Cardiac valve replacement: 74	296	[26]
13	Randomized control trial; N = 100	CABG: 2 Valvular surgery: 4 CABG + valvular surgery: 3 Redo cardiac surgery: 2	9	[17]

factors can be divided into preoperative, intraoperative, and postoperative factors shown in Fig. 1 [28].

1.3. Preoperative risk factors

Within seven days after heart surgery, acute kidney injury typically starts due to preoperative and postoperative factors. Preoperative factors include diabetes mellitus, congestive heart failure, anemia, large delta MAP, peripheral vascular disease, poor ejection fraction, and contrast medium. Additionally, there may be proteinuria, hyperuricemia, use of an intra-aortic balloon pump IABP, anemia, prolonged CPB, perioperative red blood cell transfusion, left ventricular

Table 3. Mortality rate in the different types of studies.

S. no.	Source	Total population	AKI	Non-AKI	Mortality rate	Ref
1	Cohort study	196	40 [20.4%]	156 [79.6%]	11.5%	[8]
2	Cohort study	1508	885 [58.7%]	623 [41.3%]	3.8% [34]	[11]
3	Cohort study	6261	3497 [55.9%]	2764 [44.1%]	3% [205]	[13]
4	Cohort study	3889	60	320	13.3% [8]	[21]
5	Observational study	400	37 [9.25%]	363 [90.75%]	75%	[9]
6	Observational study	435	54 [12.4%]	381 [87.6%]	18.5% [10]	[14]
7	Meta-analysis	9656	2331	7325	94.89% [2212]	[15]
8	Meta-analysis	1575	165 [10.48%]	1410 [89.52%]	1.5% [15]	[25]
9	Meta-analysis	2148	296 [13.78%]	1852 [86.22%]	1.5% [20]	[26]
10	Randomized control trial	100	9 [9%]	91 [91%]	3% [3]	[17]

The above data (Table 1, Table 2 and Table 3) presents mortality rates in different types of studies related to ARI (Acute Renal Injury) in patients during heart surgery patients.

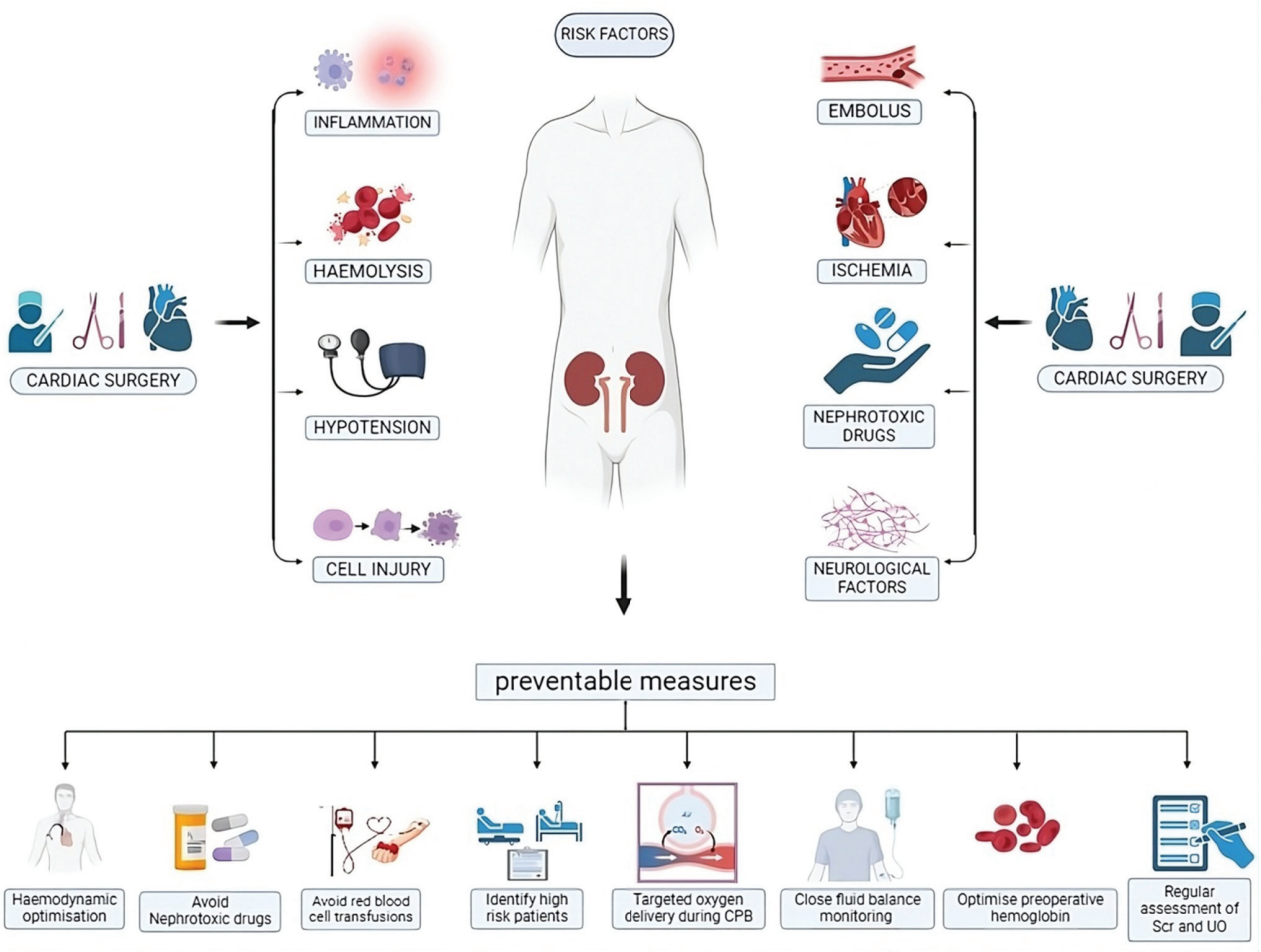

Fig. 1. Navigating the complexities of acute renal injury in cardiac surgery towards targeted prevention and management.

ejection fraction LVEF, and contrast medium [29–31].

Renal function declines with aging resulting in reduced GFR rates which causes the development of Acute Renal Injury. Studies have shown that compared to men, women are more likely to experience AKI. The key factors that influence the likelihood of developing acute respiratory distress in patients after heart surgery are their operational status and baseline co-morbidities [32–34]. Individuals who suffer from co-morbid conditions such diabetes mellitus, hypertension, congestive heart failure, peripheral vascular disease, and chronic obstructive pulmonary disease are more susceptible to acute respiratory illness. Additionally, nephrotoxic medications such as angiotensin receptor blockers, ACEIs, and non-steroidal

anti-inflammatory drugs are often given to patients with these co-morbidities [35]. These medications further alter glomerular filtration and may cause nephropathy. As a result, there is an increase in post-operative ARI susceptibility and an elevation in renal function [33, 36, 37].

Patients have, an augmented risk of ARI exists when cardiac catheterization and heart surgery are performed on the same day. There are numerous resources available to assess the preoperative risk factors for acute respiratory distress (ARI), such as plasma, urine biomarkers, clinical data, and sophisticated imaging methods.

Several studies have evaluated the clinical and surgical risk factors for ARI-related heart surgery; the risk prediction variables found are reliable for predicting stage 3 ARI, which is linked to a rise in blood creatinine or the need for renal replacement therapy. The Mehta score, the Simplified Renal Index, and the Cleveland Clinic score are some of these models. These models forecast the probability of ARI following heart surgery based on preoperative risk variables [1, 38].

1.4. Intraoperative risk factors

Intraoperative risk factors include the aortic cross-clamp time, intraoperative systolic blood pressure, hypotension, excursions of MAP beyond the limit of autoregulation, cardiopulmonary bypass, lower CPB flow, duration of CPB, use of inotropes, mitral valve surgery, reoperation, blood transfusion, reinitiation of CPB, frequent use of blood transfusions, vasopressors, inflammation, ischemic injury, fluid balance, maximum lactate level during CPB, lowest oxygen delivery, lowest hematocrit values [7, 39].

1.5. Postoperative risk factors

Between seven- and thirty-days following surgery, acute renal injury can happen. It is frequently associated with low cardiac output, sepsis, erythrocyte transfusion, vasoconstriction, inotropes, hemodynamic instability, preexisting anemia, congestive heart failure, decreased cardiac output, diuretics, and anti-arrhythmic drugs [40]. When intraoperative haemoglobin falls by more than 50% from baseline, the incidence of ARI rises noticeably. An elevated incidence of ARI was linked to preexisting isolated systolic hypertension and blood pressure following cardiac surgery. Another independent risk factor for ARI following heart surgery is the length of CPB. Many people believe that decreased urine production is a component of the stress reaction following heart surgery [41, 42].

The probability of ARI does appear to be higher in patients who receive intravenous contrast before heart surgery. For non-diabetic patients undergoing heart surgery, keeping perioperative blood glucose levels between 80 and 110 mg/dl is linked to a considerable decrease in ARI. Patients undergoing heart surgery may have different renal outcomes depending on the intravenous fluid selection. When crystalloid hydroxyethyl starch was administered to critically ill patients instead of normal saline or HES, more kidney damage occurred and more RRT was required [31, 43–46].

2. Discussion on potential preventive measures and interventions

Although a number of methods and approaches have been employed, their effectiveness in preventing acute renal failure during cardiac operations has been uneven. In order to accomplish these objectives, preoperative, intraoperative, and postoperative care are essential [31, 44–46].

Systemic inflammation increases the risk of acute renal injury (ARI) and postoperative death during cardiac surgery. The steroids have the ability to up- and down-regulate pro- and anti-inflammatory cytokines, and dexamethasone is associated with a decreased incidence of ARI necessitating replacement therapy, especially in patients with more severe chronic kidney disease. When patients undergoing off-pump cardiac surgery get 20% of human albumin right before the procedure, their urine production increases and their risk of acute kidney injury (ARI) decreases [47].

Despite the reduction of polymorph nuclear leukocytes, which lower systemic low-grade inflammation and oxidative stress, erythropoietin demonstrates a protective impact on the kidneys. Patients with high-risk factors for ARI benefit from a low-dose infusion of erythropoietin (200–300 IU/kg) prior to surgery to lower their chance of developing ARI [48]. Contrast exposure increases the risk of CSA-ARI, so cardiac surgery should be postponed until 24 to 72 hours following contrast injection to minimize the nephrotoxic burden [49]. In certain high-risk patients having CPB, an intra-aortic balloon pump (IABP) can reduce endothelial activation, raise whole-body perfusion, decrease the incidence of AKI, and reduce the requirement for RRT [50].

For patients undergoing heart surgery, preoperative aspirin medication has been linked to a decreased risk of postoperative renal failure and 30-day mortality. However, aspirin does not lower the risk of acute kidney injury (AKI) in patients having significant

non-cardiac surgery. Statins, known for attenuating inflammation and improving endothelial dysfunction, have been shown to reduce the incidence of acute renal injury (ARI) in patients undergoing cardiac surgery. There is a correlation between a lower risk of ARI and the usage of statins on the first postoperative day. But there's no proof that using them lowers the frequency of CSA-ARI [51]. A meta-analysis suggested that fenoldopam may reduce acute kidney injury (AKI) after cardiac surgery but not RRT or 30-day mortality risk [52].

Prolonged CPB and aortic cross-clamp times are linked to an increased risk of acute renal injury (ARI) [53]. Remote ischemic preconditioning (RIPC) is a technique that involves applying mild, non-lethal ischemia followed by reperfusion to protect other organs from ischemia-reperfusion episodes [54]. Goal-directed therapy (GDT) is a strategy to improve cardiac output and reduce acute renal injury post-cardiac surgery, shifting focus from traditional endpoints to modern ones [55]. A positive fluid balance is linked to an increased need for RRT after cardiac surgery, and a positive fluid balance is associated with acute renal injury (ARI) [16]. Pharmacologic agents like theophylline, pentoxifylline, and diltiazem have been used to alleviate AKI associated with cardiopulmonary bypass, but none showed any benefit. Sodium bicarbonate, a potential renal protective agent, has been terminated due to increased kidney injury and mortality. Other clinical trials include THR-184, erythropoietin, and curcumin [56].

3. Clinical implications

3.1. Renal injury of the acute phase of the disease on the overall patient outcome will be examined

Post-surgery heart patients suffering of Acute renal injury (ARI) are the ones with the worst prognosis as they have higher risk of morbidity and mortality as well as an extended stay with higher medical costs. Knowing the influence of ARI on patients outcomes might aid in improving clinical management and in the implementation of preventive strategy [57]. ARI is described as leading to multiple adverse effects, for instance, electrolyte disturbances, hypervolemia, metabolic acidosis, and uremia. They may affect the heart at cellular level, leading to cardiac arrhythmias; they may cause oxygen deficiency and water retention in the lung; there may be disorders in the blood cell production or in the blood-clotting process; also, they may affect the nervous system. As an IRI patients respond by being admitted to the intensive care unit (ICU) and put on a prolonged ventilator. This increases the chance of ventilator-associated pneumonia and other ICU related complications [41].

As most concerned the possible influence of the ARI may have onto the raised mortality may be put forward. One can look at multiple studies and see that the death rate increases in the short term and over the long term for patients who develop the ARI over those who do not [58]. The risk of mortality is potentially high in patients who require renal replacement therapeutic measures (i.e. RRT) for major ARI sequelae. Factors contributing to increased mortality include hemodynamic instability, multiorgan dysfunction, and complications related to renal replacement therapy. Patients with ARI often experience prolonged hospital stays due to the need for close monitoring, management of complications, and rehabilitation [59]. Prolonged hospitalization not only increases healthcare costs but also poses risks of hospital-acquired infections, deconditioning, and psychological distress for patients and their families depicted in Fig. 2.

ARI can have long-term consequences on renal function, predisposing patients to chronic kidney disease (CKD) and end-stage renal disease (ESRD). Even mild forms of ARI have been linked to an increased risk of progressive renal impairment and cardiovascular events in the long term [60]. Patients who survive an episode of ARI may require ongoing nephrology follow-up, dietary modifications, and medication adjustments to preserve renal function and prevent further decline. ARI significantly impairs the quality of life for affected patients. Chronic symptoms such as fatigue, weakness, cognitive impairment, and reduced exercise tolerance can persist even after renal recovery. The psychological burden of dealing with a life-threatening complication and the uncertainty of long-term renal health can also impact patients' mental well-being and social functioning [43].

3.2. The outcomes of the study can be helpful in postoperative protocol decision regarding the needs of the patient

Many specialists involved in the risk analysis need to understand the complex web of potential preoperative risk factors like advanced age, comorbidities, such as diabetes and hypertension, and factors related to the surgery itself, e.g. long duration of cardiopulmonary bypass (CPB), which further helps differentiate risk profiles that affect prognosis. Prior to undergoing surgery, physicians should optimise these determinants, for instance, through regulating blood pressure and glucose levels, because there is a high possibility of an ARI. Close following of in house variables of blood pressure, duration of CPB, as well

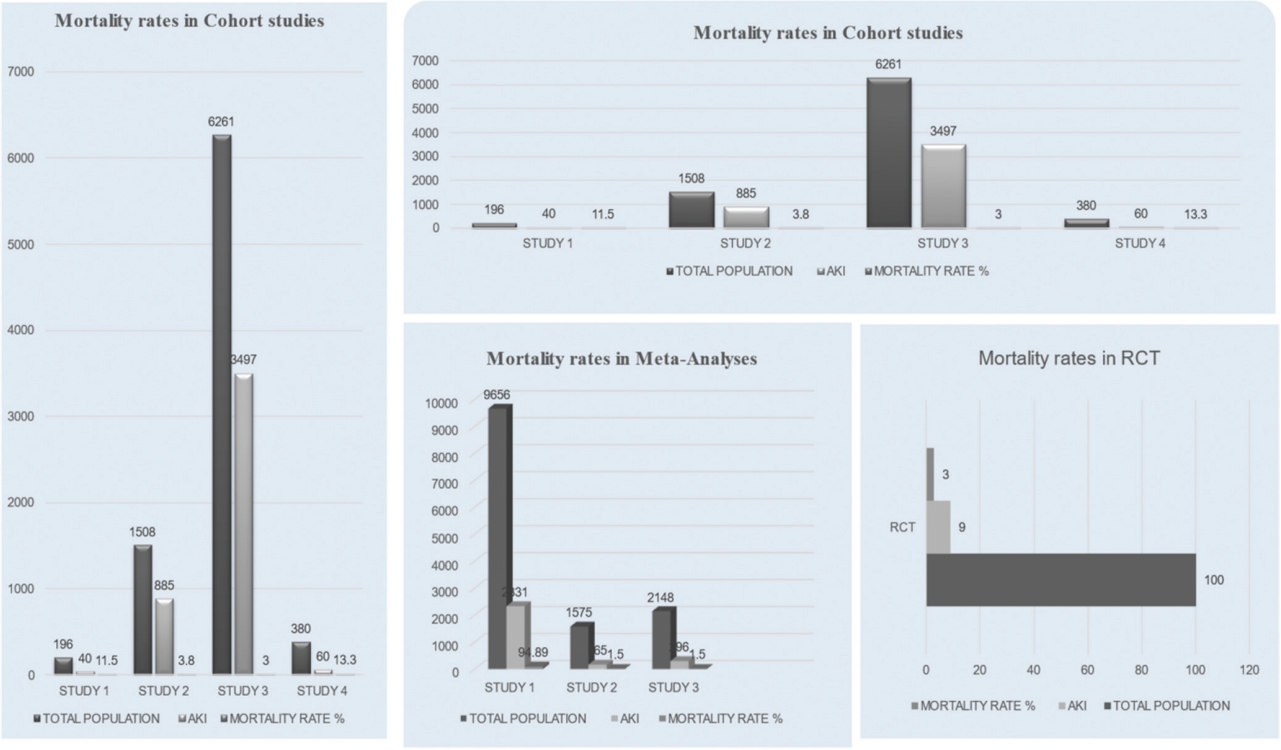

Fig. 2. Statistical analysis for understanding the landscape of acute renal injury in cardiac surgery.

as fluid balance ranks to be prudent [61]. Adequate blood pressure, proper oxygen delivery, minimizing of CPB as well as aortic cross-clamp times, are the fundamental factors of preventing ARI onset. In addition to GDT, smoothly operating fluid management and hemodynamics which are both equally important could considerably improve those results.

Certain types of drug treatments, especially corticosteroids, album substituent therapy and erythropoietin infusion, may reduce morbidity associated with AKI. In variety, these agents can be put in postoperative care protocol regulations, especially in high risk patients, which may play pivotal role. Assessing the time sequence, for example, performing cardiac catheterization and contrast initiation before cardiac surgery, is of importance. Delaying the scheduled surgery after contrast medium administration until the checkup and lab testing decrease kidney stress and reduction of the kidney injury risk [62].

Identifying (renal allograft injury) in the early period can be achieved by examination and that renal function after a surgery using biomarkers such as blood creatinine and neutrophil gelatinase-associated lipocalin (NGAL). Putting in place guiding mechanisms in terms of timely interventions, which strive to build on these biomarkers, can greatly curb progression to renal damage. While every individual patient may manifest peculiar factors that raise his or her risk or response to particular interventions, customizing therapeutic approach is indispensable accordingly [63]. Cutting down on the approach as well as implementing interventions unique to the patients' variation in characteristics can help reduce the rate of ARI as well as optimize the response. Introducing these findings to comprehensive postoperative management guidelines can be the strategy of minimizing the incidence of ARI, hence, patients will have a better outcome and there will hardly be high mortality or morbidity resulting from this complication.

4. Future implications

Among all the studies on renal injury after cardiac surgery, AKI has the most encouraging matter. The multifactorial nature of this problem, LD particularly, together with the development of prophylactic approaches as the main therapeutic tools of the problem, gives the scope of practice in cardiac surgery settings. The role of technologies and interdisciplinary collaborations stakeholders in personalized medicine- which align the therapeutic regimes based on the profiles of the patients, thus inhibiting the onset of AKI- is one that has great prospect to preventing the condition as well as its severity. On the contrary, current research that entails the unraveling

of the various molecular mechanisms associated with pathogenesis of the AKI is an area of interest to the treatment of AKI through various modalities such as invention of novel therapies and biomarker characterization. Every day, precision medicine is continuously on the rise. It combines genes, proteins, and clinical data to develop better risk models and more tailored therapy solutions as it progresses. But beside these several advancements that are possible to lower morbidity and mortality of AKI post-cardiac surgery, for sure they also help us to cut the costs that are related to AKI treatment.

5. Conclusion

Throughout running a race on a difficult track acute kidney injury (AKI) post-cardiac surgery, our road has had both stumbling blocks and chances. Currently, the diagnostic use of these classifications is unquestionable, but systematic improvements continue to be made in the applications of the classifications for treatment guidance. Nevertheless, the future is imbued with hope- the one which promises the development of novel preventive strategies tackling the whole range of perioperative risks. An integrated approach, made up of research and implementation of medications, is a start to the transformation, which will allow the development of personalized medicine where the unified solutions will radically decrease the incidence and the severity of AKI. With every step we take, multidisciplinary work and conducting on-going research are of utmost importance to untangle the mystery of AKI as it concerns the cardiac surgical practice. One step at a time we tread the path that leads us to envision the future in which optimized risk prediction models and targeted interventions rise to a new level in AKI management, in turn formulating an entirely new approach to patient care in the field of cardiac surgery.

Abbreviations

ARI: Acute Renal Injury; GFR: Glomerular Filtration Rate; CABG: Coronary Artery Bypass Graft; CS: Cardiac Surgery; KDIGO: Kidney Disease Improving Global Outcomes; AKIN: Acute Kidney Injury Network; RIFLE: Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease; ESRD: End-Stage Renal Disease; CSA-AKI: Cardiac Surgery-Associated Acute Kidney Injury; CKD: Chronic Kidney Disease; CPB: Cardiopulmonary Bypass; DM: Diabetes Mellitus; HTN: Hypertension; RRT: Renal Replacement Therapy; COPD: Chronic Obstructive Pulmonary Disease; NYHA: New York Heart Association; MI: Myocardial Infarction; LVEF: Left Ventricular Ejection Fraction; ICU: Intensive Care Unit; SCr: Serum Creatinine; Egfr: Estimated Glomerular Filtration Rate; IHD: Ischemic Heart Disease; NGAL: Neutrophil Gelatinase-Associated Lipocalin; AVR: Aortic Valve Replacement; DVR: Double Valve Replacement; MVR: Mitral Valve Replacement; ASD: Atrial Septal Defect; VSD: Ventricular Septal Defect; MAP: Mean Arterial Pressure; PRBC: Packed Red Blood Cells.

Conflict of interest

No conflict of interest was encountered.

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