

Acute kidney injury after coronary artery bypass graft surgery: a narrative review of causes, diagnosis, and prevention

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A – Study Design, **B** – Data Collection, **C** – Statistical Analysis, **D** – Data Interpretation, **E** – Manuscript Preparation, **F** – Literature Search, **G** – Funds Collection

Summary Acute kidney injury (AKI) after coronary artery bypass grafting (CABG) is a common cardiac event associated with short and long-term consequences and occurs in 30–51% of CABG patients. AKI may be associated with many other factors and is also responsible for many other pathologies. An increased level of serum creatinine (SCr) after surgery is one of the signs of AKI that may occur more often during cardiopulmonary bypass (CPB) in susceptible individuals. Preparing preoperative checklists is a good practice for the prevention of AKI. Defining new opportunities and strategies of perioperative care is a useful procedure for decreasing the risk of AKI after cardiac surgery. Collecting more data on preoperative risk factors and improving the intraoperative practices may decrease the incidence of AKI in the aggregate population. In this review study, we are going to review the literature on the pathophysiology of AKI and introduce the discussion about the features of patients who are more at the risk of AKI than others.

Key words: coronary artery bypass, acute kidney injury, creatinine, serum, cardiopulmonary bypass.

Jannati M, Kojuri J. Acute kidney injury after coronary artery bypass graft surgery: a narrative review of causes, diagnosis, and prevention. *Fam Med Prim Care Rev* 2021; 23(3): 372–379, doi: <https://doi.org/10.5114/fmpcr.2021.108205>.

Background

Postoperative acute kidney injury (AKI) is a common postoperative complication in 30–51% of patients undergoing cardiac surgery and can increase the risk of morbidity, mortality, and the length of hospital stay [1]. Severe AKI increases the mortality rate three to eightfold and also increases the length of hospital stay and the related costs of treatment [2]. AKI is a major mortality cause of patients following cardiac surgery [3–5].

Postoperative complications have multidimensional psychological and pathological origins, including age, prior kidney injuries, “diabetes mellitus, ischemia-reperfusion injury, altered regional blood flow with vasomotor dysfunction, and inflammatory responses” [6–9]. In the case of progressive acute renal dysfunction, the survival rate is significantly lower [10]. It is also associated with a fourfold resource utilization in hospitalized individuals [11], and dramatically reduces life quality for both the patients and their families [12].

It is very important to detect patients with high-risk of progressing postoperative AKI early in order to provide them with suitable support that may include proper hemodynamic monitoring, fluid management, appropriate pharmacologic therapy, or early use of renal replacement therapy (RRT) [13, 14].

This paper is aimed to study the impact of postoperative coronary artery bypass graft surgery (CABG) on acute kidney injury (AKI) incidence. For this reason, some of the most reputable and well-known scientific medical resources were collected.

Methods

The relevant articles were searched in the PUBMED and SCOPUS databases. The following keywords were used: “Acute Kidney Injury,” “Coronary Artery Bypass,” and “Cardiopulmonary Bypass”. Articles from 1990 to 2019 were searched. Ninety-five articles were found, and their abstracts were read. Finally, 86 articles were selected for the study.

The Acute Kidney Injury (AKI) concept and definition

There are three different classifications of AKI. After decades of no standard definition of AKI, Bellomo et al. finally introduced the RIFLE classification (Risk, Injury, Failure, Loss End-Stage Kidney Disease) in 2004 that can be seen in Table 1 [15].

In 2007, the AKIN (Acute Kidney Injury Network) group revised the RIFLE criteria. This revision introduced four main changes, namely: 1) GFR has been removed from the definition; 2) the SCr period was changed from seven days to 48 hours; 3) 0.3 mg/dL of SCr was considered as the lowest measure for AKI, and 4) two stages (loss and end-stage) have been omitted from RIFLE. All of these changes can be found in Table 2 [16]. Finally, the KDIGO (The Kidney Disease Improving Global Outcome) workshop defined AKI according to three factors: an increase in SCr by 50% in seven days, an increase in SCr by over 0.3 mg/dL in 48 hours, or oliguria [17].

Table 1. The RIFLE (Risk, Injury, Failure, Loss End-Stage Kidney Disease) classification

	Class risk	Injury	Failure	Loss	ESKD
RIFLE	↑SCr × 1.5 or ↓GFR over 25%	↑SCr × 2 or ↓GFR over 50%	↑SCr × 3 or ↓GFR over 75%	Persistent acute renal failure with complete loss of kidney function over four weeks	RRT required for over three months

EDKD – End-Stage Kidney Disease, GFR – glomerular filtration rate, RRT – renal replacement therapy, SCr – serum creatinine concentration.



Table 2. The AKIN (Acute Kidney Injury Network) classification

	Stage 1	Stage 2	Stage 3
AKIN	SCr > 1.5 baseline or > 0.3 mg/dL increase	SCr > 2 baseline	SCr > 3 baseline or ↑SCr to 4.0 mg/dL (with an acute increase of at least 0.5 mg/dl) or ↑ of RRT

AKIN – Acute Kidney Injury Network, RRT – renal replacement therapy, SCr – serum creatinine concentration.

Table 3. The KDIGO (The Kidney Disease Improving Global Outcome) definition of AKI

	Stage 1	Stage 2	Stage 3
KDIGO	SCr > 1.5 baseline or > 0.3 mg/dL increase	SCr > 2 baseline	SCr > 3 baseline or ↑SCr to 4.0 mg/dL or ↑ of RRT

RRT – renal replacement therapy, SCr – serum creatinine concentration.

Risk markers, prevention, pathophysiology, and survival implications

When hypotension or a low cardiac output state persists, the kidney compensatory reserves become gradually exhausted, filtration reserve is exceeded, and endogenous and/or exogenous vasopressors increase afferent arteriolar resistance that consequently reduces the GFR. At this point of pre-renal azotemia, oliguria may occur, but tubular function may remain intact. A longer period of ischemia will cause structural tubular injury with cell disruption that may obstruct the tubules with back leakage of fluid into the circulation. There are also oxidant injury and inflammatory responses that result in further hypoperfusion and damage to tubular cells [18, 19].

Cardiac surgery is recognized as one of the significant risk factors for postoperative AKI. It may also induce “bleeding, postoperative pro-coagulable state,” and the consequent “macro thrombotic” events, including “preoperative stroke or myocardial infarction” [12]. Cardiopulmonary bypass (CPB) could accelerate microemboli formation which consists of fibrin, activated leukocytes, and aggregated platelets. Subsequently the resulted microvascular plugging with leukocytes and activated platelets may have critical roles in the pathogenesis of postoperative AKI and post-infection kidney injury [20]. However, post-CABG and CPB incidence of AKI is partly associated with a platelet count drop, but still little is known about the implications due to relatively fewer comprehensive studies in a “cardiac surgical patient cohort”. In addition to the increased medical costs, chronic kidney disease and dialysis, elevated hospital mortality, and a decline in long term-survival are caused by postoperative AKI [21–23]. AKI is an indicator of “all-cause mortality” in post-CABG. Consequently, it is critical in post-CABG hospitalization to develop a reliable AKI prediction model for improving clinical and medical effectiveness. The number of diagnostic models for post-CABG AKI risk factors has been increasing in recent years, with “European System for cardiac operative risk evaluation (EuroSCORE)” (1999, revised in 2012) [24], and the “society of thoracic surgeons (STS) score” (2008) being the most frequently used models [25]. Another model is “The patient’s age, creatinine, and ejection fraction (ACEF) score,” first developed in 2009 for quick clinical tests [26]. Regardless of the frequent use of these models for postoperative mortality prediction, several preoperative “demographic and clinical variables” have also been reported as influencing postoperative mortality and morbidity caused by AKI. Evidence shows that STS scoring and database was the only most reliable test to predict end-point renal failure [27]. The potential very limited outcomes for risk prediction model(s) may be attributed to low dialysis rates. This accounts just for 1–5% of studied patients [28]. Since AKI is a prevalent event in post-operative CABG and may cause severe outcomes, for instance, it may increase the risk of end-stage renal disease (ESRD) threefold [29], different models are required to overcome this problem. In addition, more research is needed to develop validated, reliable models to determine the severity of postoperative AKI [30].

Inflammation and genetics

Modification of genetic factors and many clinical factors is a hard or impossible task. However, other clinical risk factors for AKI could be prevented, and could help in identifying a patient with a high risk of AKI and ARF. The balance between “pro-inflammatory response and the compensatory anti-inflammatory response” is the key to modifying clinical AKI risk factors. Administration of intra-operative corticosteroids is the treatment for the systemic inflammatory response syndrome (SIRS) as an AKI risk factor [31]. However, biological active mediators of natural body compensatory anti-inflammatory response to SIRS as pro-inflammatory proteins resulting in kidney dysfunction plays a critical role in postoperative AKI prevention [32]. Inflammation is potentially caused by kidney injury in post-coronary artery bypass graft (CABG) patients [33, 34], which explains “endothelial responses and neutrophil recruitment in ischemia-reperfusion injury including distant organ injury” [35] and “post-ischemic renal failure” [36]. Thrombocytopenia is a prevalent event in acute AKI patients and is attributed to elevated platelet destruction (immune and non-immune), hemodilution, platelet sequestration, and decreased production. Thrombocytopenia may be related to prolonged medical health care and hospitalization, a drop in survival rates, and an elevated incidence of AKI [37, 38].

Classification

Regarding etiology, AKI has been classified into pre-renal (reduced renal perfusion), renal (intrinsic renal insult), and post-renal (obstructive uropathy). Pre-renal etiology, followed by renal etiology are the most common causes of AKI. As volume changes are common during cardiac surgery, CSA-AKI can be divided into volume responsive and non-volume responsive. Various factors, including ischemia and ischemia-reperfusion injury, inflammation and oxidative stress, exogenous and endogenous toxins, metabolic abnormalities, and neurohormonal activation may cause renal CSA-AKI. They can be divided into three factors: hemodynamic, inflammatory, and nephrotoxic [39].

Diagnosis, renal biomarkers

There are three well-established measures for AKI diagnosing and staging; RIFLE, AKIN, and KDIGO. However, as they are based on changes in SCr occurring 48 hours to seven days after the original insult, they all have significant limitations for early preoperative AKI diagnosis. Another limitation is that there are various factors such as age, sex, ethnicity, muscle mass, diet, drug use, and intravascular volume loading affecting SCr, which are not connected with kidney function and are not incorporated into these measures [40]. The other concern is that elevated creatinine levels are found only after 50% kidney function loss [41, 42]. Consequently, RIFLE, AKIN, and KDIGO are only

instituted for postoperative diagnosis, which may be far too late for preoperative or intraoperative AKI treatment. On the other hand, urine output criteria are not a reliable measure for AKI pre-diagnosis as they do not differentiate pre-renal from intra-renal oliguria or ischemic from nephrotoxic kidney injury [43]. AKI may occur with no oliguria, and extra-renal obstruction may cause oliguria with no AKI symptoms. The SCr frequently drops post-CBP due to hemodilution, which can be followed by a potential delayed rise, even after GFR significantly decreases. These limitations encouraged clinicians and academics to search for new biomarkers for early AKI diagnostics to ensure timely intervention and avoid kidney function loss or failure. The number of clinical centers where renal function is evaluated using different measures than SCr is currently small. In the majority of cases, there is little significance of subtle changes in tubular function as long as the kidneys produce a satisfactory urine output with or without diuretics with minimal change in the SCr. For early AKI diagnostics, biomarkers should be the most sensitive means for early reliable detection. CABG could potentially raise all kidney-specific proteins such as tubular damage markers, some of which, such as neutrophil gelatinase-associated lipocalin (NGAL), cystatin C, kidney injury molecule 1 (KIM-1), and interleukin-18 (IL-18) can be indicators of early AKI, its severity and duration [44-47]. In addition, other AKI biomarkers, such as NGAL, cystatin C, KIM-1, and IL-18 are found after two to six hours post-CABG [48]. Biomarkers are more reliable early AKI indicators than SCr. Cystatin concentration indicates a more accurate baseline GFR compared to SCr; nevertheless, NGAL is markedly increased in renal tubular cells in response to ischemic injury and detected easily in the blood and urine because of its small molecular size. Early NGAL detection is a common indicator of subsequent GFR decline; however, it is initially independent of GFR [49]. Injured distal tubule epithelial cells secrete NGAL (lipocalin-2) and enter the urine through the tubular back leak. NGAL measurement can be a consistent predictor of AKI incidence and severity. However renal baseline function significantly influences the diagnostic performance of NGAL [50, 51]. Another tubular factor which is useful in the diagnostic differentiation between ischemic AKI and pre-renal azotemia and chronic kidney disease is KIM-1. Another AKI marker is a pro-inflammatory cytokine called IL-18, which is detectable four to six hours after CABG in the urine and peaks twelve hours after the surgery. Important outcomes such as duration of stay in the ICU and hospital, dialysis, and mortality, can be predicted by NGAL and IL-18 [52, 53]. Cystatin C, as a low-weight-molecule protease inhibitor, facilitates predicting AKI two days before creatinine and is a reliable indicator reflecting GFR changes. There are also two new urinary cell-cycle biomarkers released by cellular stress in the tubular cell injury initiation phase (promoted by different factors such as inflammation, ischemia, oxidative stress, drugs, and toxins), called metalloproteinase-2 tissue inhibitor and insulin-like growth factor-binding protein 7. Both are involved in the process of G1 cell-cycle arrest, which prevents cell proliferation in the case of DNA damage until the damage is repaired. They act as alarm proteins for adjacent cells in a paracrine fashion, and they can predict AKI better than NGAL, KIM-1, IL-18, or cystatin C [54]. However, their usefulness will depend on the development of a cost-effective and easy-to-use kit applicable in the surgery room or ICU. Although every single biomarker provides some predictive information on AKI incidence, a combination of biomarkers as a diagnostic panel in the early preoperative stage is required for an accurate and reliable diagnosis of AKI risk and severity [55, 56]. An ideal measure for predicting AKI within 24 hours is a non-invasive, specific, target-oriented, and sensitive biomarker which can be detected and measured in a fast and reproducible way to determine AKI risk and subtypes [42, 56].

Prevention from acute kidney injury after CABG

Fluid overload, electrolyte abnormalities, and metabolic acidosis are among the potential risks of chronic kidney disease.

Patients with acute renal failure (ARF) should undergo dialysis immediately before and after kidney replacement surgery. Positive fluid balance, as a kidney dysfunction risk factor, should be reduced by intraoperative hemofiltration. The mortality risk is estimated at 10%–15% for post-CABG patients with AKI or ARF. If applicable, the level of urea, nitrogen, and creatinine in the blood should return to baseline as part of pre-CABG clinical care. Using non-steroidal anti-inflammatory drugs, ACE inhibitors, and angiotensin II receptor blockers (ARBs) should be withheld one day prior to surgery to minimize their intra-CABG drug hypotensive effects [57, 58]. Off-pump CABG may reduce AKI incidence; however, it has no effect on the RRT requirement [59]. Intraoperative measures should be used to try to augment renal reserve by improving renal blood flow. It elevates the GFR and prevents tubular damage in patients with renal dysfunction or with risk factors in medical history. Using heparin-coated circuits or miniaturized circuits prevents intra-CABG hyperglycemia, which may decrease the systematic inflammatory response. Glomerular and tubular injury may be alleviated by leucocyte filters [60, 61], which consequently decrease microalbumin and creatinine-indexed retinol-binding protein. Free water and solutes may be removed by hemofiltration, which is beneficial in removing excess fluid in heart failure (HF) patients, and in improving pulmonary function. Antifibrinolytics may also minimize bleeding diathesis, which is a common event in renal dysfunction. ϵ -Aminocaproic acid is a commonly used safe agent although it is associated with some degree of renal tubular dysfunction without a significant change in creatinine clearance. Tranexamic acid is another better alternative. Aprotinin was removed from the market in 2007 because it increases renal failure risk and has other adverse side effects.

High perfusion pressure is a key AKI preventing factor, but optimal average intra-CABG arterial pressure is not yet well determined.

AKI drug prevention

The intraoperative renoprotective advantages of Nesiritide (β -type natriuretic peptide), such as dilating renal afferent arterioles, and to a lesser extent, the efferent arterioles, have been demonstrated in several studies [62–65]. Showing strong natriuretic and diuretic properties, the drug, acting as a renin-angiotensin-aldosterone axis inhibitor, results in increased glomerular filtration. An initial clinical trial with human atrial natriuretic peptide in CABG patients with left ventricular dysfunction demonstrated the same protective benefits. The intraoperative administration is a bolus dose of 2 $\mu\text{g}/\text{kg}$ over 1 min, with a following infusion of 0.01–0.03 $\mu\text{g}/\text{kg}/\text{min}$. Fenoldopam, a selective agonist of the dopamine 1 (DA1) receptor produces a dose-dependent increase in renal plasma flow, decreases renal vascular resistance, and maintains GFR. Consequently, blood flow to the renal cortex and medulla increases, and sodium tubular reabsorption is inhibited, resulting in diuresis, natriuresis, and kaliuresis [66, 67]. It is a CBP-CABG renoprotection drug used as a nesiritide alternative. Patients with a SCr over 1.4 mg/dL should be administered fenoldopam, a pre-CABG infusion of 0.03–0.1 $\mu\text{g}/\text{kg}/\text{min}$, and continue in the ICU for an estimated 12-hour period post-CABG. There is inconsistent data about therapeutic interventions such as preoperative statins, acetylsalicylic acid, N-acetylcysteine, and sodium bicarbonate [68–70].

Literature review

Stallwood et al. (2004) studied acute renal failure in coronary artery bypass surgery regarding the independent effect of cardiopulmonary bypass (CPB). The statistical sample was 2199 post-CABG patients from 2000–2002, in which patients with remarkable kidney function loss were removed using retrospec-

tive analysis. AKI independent risk factors were analyzed using a multivariate regression analysis model. The results showed that 53 patients (2.4%) developed AKI during hospitalization. The research also revealed 2.9% incidence of AKI in the patients who underwent on-pump surgery (among a total of 1483), compared with 1.4% in the off-pump CABG group (among a total of 716). The findings also suggested a correlation between the use of cardiopulmonary bypass and the risk of the development of acute renal failure [71].

Benedetto et al. studied miniaturized cardiopulmonary bypass and acute kidney injury among patients undergoing coronary artery bypass graft surgery. A statistical sample of this study included 705 mini-CPB (104) and conventional-CPB (601) postoperative patients. A propensity score matched-pair analysis was used to determine AKI in the patients based on the current acute kidney injury network classification. The findings revealed that 274 (38.8%) of 705 patients developed postoperative AKI, and a total of 27 patients (3.8%) required immediate kidney replacement therapy. The study also indicated that AKI could prolong average postoperative recovery from two days for patients without AKI up to 5.4 days for patients with AKI. On the other hand, in the comparison between mini-CPB and CPB, the study revealed that the overall incidence of AKI was 30 out of the 104 patients who underwent mini CPB (28.8%), and 244 out of the 601 patients who underwent CPB (40.5%), which indicates a remarkable decline in AKI incidence rate for mini-CPB postoperative patients [60].

Olson et al. (2012) studied acute kidney injury following coronary artery bypass surgery and long-term risk of heart failure. The study sample was a total of 24 018 Swedish patients undergoing isolated CABG from 2000–2008 with no prior history of heart disease. The level of serum creatinine (Scr) was used in this study as an indicator for AKI diagnosis in three different stages. The mean follow-up duration was 4.1 years, in which 1325 subjects (5.5%) developed heart failure. The hazard ratios were 1.60% for stage 1 AKI, 1.87% for stage 2 AKI, and 1.98% for stage 3 AKI. The results indicated a correlation between post-CABG, AKI, long-term heart failure, and kidney function loss [72].

Chen et al. (2015) compared contemporary preoperative risk models for predicting acute kidney injury after isolated coronary artery bypass grafting in a retrospective cohort study. The statistical sample of this study included 353 post-CABG patients from 2010 to 2012. This study mainly aimed to determine AKI in post-CABG patients, defined based on the Kidney Disease Improving Global Outcome (KDIGO) Clinical Practice Guideline for AKI, from 2012. The results showed that 102 patients developed AKI (28.9%), and EuroSCORE II, STS score, and ACEF score were all good tools for diagnosing stage-3 AKI. Among them, the ACEF score performed significantly better. The study benefited from multivariate logistic regression analysis to examine the effect of CABG on AKI development. A drop in ejection fraction and elevated serum creatinine were identified as independent AKI risk factors [73].

Kertai et al. (2016) studied the relationship between platelet counts, acute kidney injury, and mortality after coronary artery bypass grafting surgery. The statistical sample of this study comprised 4217 post-CABG adult patients. To determine postoperative AKI and mortality, the postoperative nadir platelet counting method was used. The results showed that nadir platelet counts are a predictor of AKI and of mortality in post-CABG patients. The study also revealed a 54% overall AKI incidence rate in postoperative CABG patients. A reverse correlation was found between platelet counts and postoperative AKI. The findings also suggested an elevated mortality risk in AKI patients immediately after CABG surgery [74].

In a randomized clinical trial, Garg et al. (2017) studied kidney function after off-pump or on-pump coronary artery bypass graft surgery. They suggested that “mild or moderate increases in the serum creatinine concentration” are a significant indicator for AKI that may persist for several days. They also believe that AKI is a side effect of chronic kidney dysfunction. The study

aimed to classify AKI risk using randomized clinical trials and to observe differences between two AKI groups that underwent surgery either with a beating-heart technique or with cardiopulmonary bypass. The study sample contained 4752 off-pump or on-pump post-CABG patients from 79 clinical sites in 19 different countries between 2010 and 2013. The results showed a 50% risk of a AKI event (with an increase in creatinine concentration) 30 days after the surgery and a 20% risk of kidney failure after one year. The research also revealed a drop in AKI risk in 1472 (17.5%) postoperative off-pump patients and in 1460 (28%) on-pump CABG patients. The between-group difference for one-year postoperative kidney failure was not significant (17.1% vs 15.3%). The findings indicated that off-pump CABG surgery reduced AKI postoperative risk and could lead to a better one-year postoperative kidney function [75].

Discussion

When the kidney partly or completely stops its routine functioning with no prior symptoms, this event is called acute kidney injury (AKI). One biomarker of this condition is elevated creatinine content in the serum [76]. AKI accounts for a significant rate of post-CABG mortality, especially in the cases with the need for urgent hemodialysis [28, 77]. Among postoperative AKI patients, mortality ranged between 7% and 38% [11, 28, 77–79]. Cardiopulmonary bypass (CPB) can increase preoperative and postoperative acute renal injury. However, results regarding the collected data are inconsistent, which was partly due to different outcomes for using off-pump or on-pump CABG [80]. Dialysis is the final prescribed treatment for severe AKI leading to long-term kidney function loss [81]. However, statistical results showed up to 30% less severe AKI, which is more likely in postoperative hospitalization. The symptom may be serum creatinine concentration that is elevated more than 50% and lasts several days before it returns to the baseline levels. Among this population, 1% of postoperative patients need dialysis [82].

Coronary artery bypass grafting (CABG) is the most frequent cardiac surgery that is used as an effective treatment for angina ischemic heart disease [52]. During the last decade, the CABG incidence rate was 1000 cases per million US population. Acute kidney injury (AKI) is a common medical complication occurring in 8% to 15% post-CABG patients annually. It is associated with short and long-term mortality. A minor 0.5 mg/dL increase in serum creatinine (Scr) was shown to be correlated with 30 days elevated mortality up to 3 times more than routine Scr [53]. While there are several studies about moderate AKI effects on long-term kidney performance manifested by microvascular injuries, capillary density decline, and “chronic renal hypoxia,” there are still gaps in this area of study [83]. On the other hand, several clinical case studies suggested a relationship between non-sever AKI and long-term kidney performance loss three months after the surgery. In the case of (on-pump or off-pump) postoperative CABG AKI, it is believed that off-pump surgery reduces postoperative AKI risk. Another important issue is whether long-term postoperative kidney function after on-pump CABG is affected after one year? According to previous studies [76, 81], there was robust evidence that off-pump CABG reduces either postoperative AKI risk immediately after surgery or kidney function three months and one year after surgery, respectively. The recent meta-analysis of 4819 randomized controlled trials revealed that off-pump CABG reduced AKI risk by up to 40% [20]. Accordingly, existing evidence indicates the reductive effect of off-pump CABG on non-sever AKI postoperative development, which appeared to be highest in preoperative CABG patients with chronic kidney disease. Nevertheless, the clear effects of one-year postoperative off-pump CABG are not yet fully known. Some studies [84, 85] focused on one-year postoperative effect of off-pump or on-pump CABG on AKI risk. There are some possible reasons for this shortcoming.

While evidence for one-year postoperative off-pump reduction in AKI risk and kidney function is sufficient [85], one year may be too short a period to study all clinical and physical effects of this surgery on kidney function. The follow-up study may provide more reliable data on kidney function if it is carried out in longer durations. However, evidence showed a one-year GFR loss in off-pump CABG patients compared to on-pump surgery patients.

Since even a 20% loss in GFR measured one year after surgery does not indicate a significant change in “end-stage renal disease,” serum creatinine concentration used as a major kidney function indicator may be prone to errors. The errors may be caused by in-vitro or in-situ diversity at the baseline and at the one-year postoperative follow-up which possibly results in unrecognized signal events. In addition, in some post-CABG patients, the baseline serum creatinine concentration may be unstable due to renal perfusion deficiency. For this reason, there have been systematic efforts to provide better standards for serum creatinine assay during the last decade [85]. Otherwise, AKI risk in postoperative off-pump CABG patients was evidently lower than in on-pump CABG patients, but it is still significantly higher than in other patients.

Overall findings indicated that postoperative AKI risk is at least 50% greater in post-CABG patients. The risk is correlated with elevated serum creatinine concentration during 30-day follow-up after surgery. The randomized controlled trial method was the basic diagnostic tool for postoperative AKI patients. This method started with serum creatinine concentration measurement done seven days before randomization. Then postoperative concentration was collected during hospitalization. Afterwards, the highest concentration collected during 30 days was adopted for primary analysis to confirm if AKI was diagnosed after surgery or not.

Conclusions

Post-CABG patients had up to 50% more risk of AKI compared to other patients after a 30-day follow-up. It was also evident that kidney function loss is significantly higher in the one year after CABG. However, the postoperative AKI risk and one-year kidney function loss in off-pump CABG patients was apparently lower than in on-pump CABG patients. The main findings also associated postoperative AKI after CABG with an elevated risk of long-term kidney function loss and heart failure. Accord-

ing to evidence, AKI is an important predictor of long-term kidney and heart dysfunction. Moreover, a moderate 0.3–0.5 mg/dL increase in SCr indicated a significant increase in postoperative cardiac events. There is also evidence that long-term kidney function loss leads to cardiac failure. Postoperative renal failure is a key influencing factor in postoperative mortality and morbidity. Chertow et al. suggested a 63% mortality rate in postoperative hemodialysis patients due to severe AKI, compared to a 4.3% rate for patients with no AKI symptoms within 30 days after the surgery [28]. While this study was performed 15 years ago, the findings were consistent with the recent Mangano et al. study with the same mortality rates [11]. There are numerous complicated factors related to post-cardiac surgery renal failure mechanisms [11, 28, 77, 79]. Using clinical variables, prediction algorithms were developed for individual postoperative AKI risk. The clinical variables were classified into four distinct areas based on their effects:

1. Factors related to renal ischemia after atherosclerosis with cardiac output reduction symptoms.
2. Exogenous nephrotoxin-induced kidney injury, such as “amino-glycoside antibiotics, diuretics, or radiologic contrast media” [80].
3. Endogenous nephrotoxin release (e.g., myoglobin, free radicals, or pro-inflammatory cytokines such as interleukin-8, interleukin-1, and tumor necrosis factor).

Baseline renal dysfunction is recognized as a primary indicator of preoperative and postoperative renal failure. Consequently, preoperative patients with an excessive creatinine level of 200 mol/L should not be listed for CABG before their creatinine level returns to baseline concentration with renal dysfunction medical treatment. Most postoperative AKI risk factors are patient-oriented, so they are mainly beyond clinical control. The effect of CABG on AKI has been studied since the 1990s with the introduction of off-pump CABG [86].

Nesiritide (β -type natriuretic peptide), fenoldopam, nesiritide, statins, acetylsalicylic acid, N-acetylcysteine, and sodium bicarbonate are known as common pre-, intra-, and post-CABG acute kidney injury drug prevention strategies. Intraoperative hemofiltration, adverse retention of urea, nitrogen, and creatinine in the blood to the baseline using nonsteroidal anti-inflammatory drugs, ACE inhibitors, angiotensin II receptor blockers (ARBs) one day prior to surgery to minimize the intra-CABG drug hypotensive effects are among the other AKI prevention strategies for post-CABG patients.

Source of funding: This work was funded from the authors' own resources.

Conflicts of interest: The authors declare no conflicts of interest.

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Tables: 3

Figures: 0

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Received: 25.08.2020

Reviewed: 06.09.2020

Accepted: 22.09.2020

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