

Euro Nephrology 2019: Acute Kidney Injury: Do The Patients Recovery Their Renal Function After a Cardiac Surgery? - Bruno Côte Santana, University of Brasilia

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The incidence of acute renal injury (AKI) affects up to 40% of patients during hospitalization, due to several factors, including their own submission to a surgical procedure, as well as the use of mechanical ventilation and prolonged cardiopulmonary bypass. In this way it is our objective to identify if the recovery of the renal function occurs after complications in the postoperative period following cardiac surgery. To answer the objective, a quantitative, retrospective, longitudinal study was performed with 62 patients. Data were collected using a questionnaire. The chi-square test was used for statistical analysis, at a significance level of $p < 0.05$. The results showed that 71% of our patients developed AKI in the postoperative period following cardiac surgery, the use of noradrenaline ($p = 0.002$) such as use of mechanical ventilation ($p = 0.02$) contributed significantly to the occurrence of acute renal injury. After discharge from the Intensive Care Unit (ICU), approximately half of the patients (40.5%) recovered their renal function. It is important to emphasize that patients with acute kidney injury may develop with Chronic Kidney Disease (CKD), the percentage of renal recovery may be an indicator of greater or lesser risk of future aggravation, besides contributing to the adoption of early preventive measures and reduction of risk of death. Our findings should be considered, since patients who develop AKI in the postoperative period of cardiac surgery are at risk of progressing, even after hospital discharge, to CKD.

The development of acute kidney injury (AKI) after cardiovascular surgery (CVS) has been well recognized in the past and is implicated as a contributing factor in the elevated mortality and poor outcomes of these patients. AKI following major CVS has a complex and multifactorial etiology. Despite advances in its management, AKI continues to contribute to the poor short- and long-term outcomes of CVS. The identification of high-risk individuals, development of protective maneuvers, and use of markers of early kidney injury are important in the early detection and proper treatment of this serious complication. The definition of AKI after CVS differs in published studies; some describe it as a percent of increment from baseline creatinine, while others call it a doubling of the preoperative creatinine. As a rule, this syndrome is characterized by a deterioration of kidney function over a period of hours or days following surgery, with symptoms including oliguria (urine output less than 400 ml/day, which usually results in volume overload), paralleling elevations of serum blood urea nitrogen (BUN) and creatinine (Cr), and the development of serious electrolyte and acid-base disorders.

Recently, the Kidney Disease Improving Global Outcomes (KDIGO) developed a staging classification of AKI that includes three stages based on elevation of serum creatinine from baseline and urinary output. The hope is that this system will be universally adopted to enable future understanding of the incidence, outcomes, and effectiveness of therapeutic interventions for AKI.

AKI is a complex process involving apoptosis and necrosis of injured tubular cells with simultaneous repair and proliferation of the surviving tubular cells. In addition to general maneuvers designed to optimize the patient's overall condition, such as discontinuation of potentially detrimental drugs at least 48 hours prior to surgery (including ACE-Is, ARBs, NSAIDs, metformin, diuretics, when possible) and achieving normovolemia, there have been many different attempts to prevent AKI after CVS. Observational studies using calcium channel blockers such as nifedipine, diltiazem, and nicardipine showed beneficial effects, but their use never qualified as accepted standards of care. A meta-analysis of randomized studies showed beneficial effects using Fenoldopam, which appears to reduce the need for dialysis and mortality in critically ill patients with or at risk of AKI. Mannitol and dopamine failed to protect against AKI during thoracic aortic cross-clamping. Likewise, dopamine and furosemide were shown to lack Reno protective effects during cardiac surgery. The use of atrial natriuretic peptide in AKI also failed to show beneficial renoprotective effects. Low-dose dopamine has been found to have negative results. However, at times dopamine can help to initiate diuresis when a loop diuretic alone is insufficient. Although ACE-I/ARB therapy should be avoided in most cases, the use of intravenous enalaprilat³⁶ has improved kidney performance in patients who have undergone coronary artery bypass complicated by left ventricular dysfunction. Intravenous pentoxifylline in elderly patients showed a prophylactic beneficial effect on postoperative organ function, but more studies are needed to assess its efficacy moreover, this compound is not available in the United States. The use of automatic pulsatile intra-aortic balloon pumps during cardiopulmonary bypass has been associated with better kidney function. Off-pump coronary artery bypass may be associated with a lower incidence of postoperative AKI but did not affect the need for dialysis. In a randomized, single-blind, controlled pilot trial of 120 adult patients undergoing cardiopulmonary bypass, remote ischemic preconditioning resulted in a 27% absolute risk reduction of AKI.⁴¹