



# Postoperative saline administration following cardiac surgery: impact of high versus low-volume administration on acute kidney injury

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*Comment on:* Lim JY, Kang PJ, Jung SH, *et al.* Effect of high- versus low-volume saline administration on acute kidney injury after cardiac surgery. *J Thorac Dis* 2018;10:6753-62.

Submitted Jan 31, 2019. Accepted for publication Apr 02, 2019.

doi: 10.21037/jtd.2019.04.28

View this article at: <http://dx.doi.org/10.21037/jtd.2019.04.28>

Acute kidney disease (AKI) is a known complication following cardiac surgery seen in almost one third of patients (1-3). AKI increases morbidity and mortality and of course affects the financial burden. Age, sex, preoperative renal function, diabetes mellitus, postoperative hypoperfusion, chronic hypertension and prolonged cardiopulmonary bypass (CPB) time contribute in varying proportions for the development of renal injury following heart surgery (4,5). Although CPB time is an independent risk factor, off-pump coronary artery bypass grafting surgery can lead to AKI and many studies suggest that perioperative intravenous fluid administration could be considered also as an important factor for better preservation of postoperative renal function (6,7).

Fluid resuscitation is a common strategy for these patients after intracardiac repair in order to optimize stroke volume and maintain efficient circulation. Normal saline solution is one of the most commonly used fluids for these circumstances (8). However, there are studies suggesting that increasing use of this may contribute to acidosis, especially with hyperchloremia further precipitating AKI (9,10). The current British Consensus Guidelines on intravenous fluid administration for adults state that use of balanced salt solutions should replace 0.9% saline for fluid resuscitation where non-colloidal fluid administration become important, in order to minimize the risk of development of hyperchloremic acidosis (11).

Lim *et al.* tried to elucidate the blur field regarding

applicability of intravenous fluid administration in cardiac surgical patients under CPB and they managed to compare the impact of crystalloid use in two categories between high and low-volume saline therapy on renal function following surgery in this group of patients (12). Although the study is retrospective and not randomized controlled, Lim *et al.* showed that high-volume saline administration after cardiopulmonary bypass was not related in particular to the development of renal injury. They compare two groups and although the bias of potential confounding factors always exists, a well propensity score matching and multivariable adjustment analysis was performed in order to eliminate this and the authors need to be congratulated for this. However, the groups of high and low volume were categorized in an arbitrary manner as the limit for high and low volume administration was the amount of 1l. We think that the power would be stronger, if this could be planned according to the BSA of each patient in order to define more accurately the high and the low level of fluid administration.

Two kinds of crystalloid solution were used: a chloride rich solution (0.9% saline, chloride concentration 150 mmol/L) and a balanced buffered solution Plasma Solution). We have to mention that in the study, the infusion chosen was at the physician's discretion. In situations where this was found to be ineffective, 6% HES solutions (Hextend 670/0.75) were preferred up to 20 mL/kg as a second preference according to the clinical criteria, that includes renal parameters and coagulation.

Furthermore, the authors stress that the volume of saline administration was 2.47 L on average in the high-volume group. This could have also been related to higher use of blood products or other colloids in order to expand the intravascular volume. In that way, the volume of crystalloid used will not be significantly harmful for the renal function when other preoperative and intraoperative volumes were taken into consideration. On the other hand, Bhaskaran *et al.* found that chloride restricted use of intravenous fluids was found to decrease the overall incidence of renal impact following off-pump coronary artery bypass grafting (6).

Moreover, Mukaida *et al.* recently described that time-dose response of oxygen delivery during cardiopulmonary bypass predicts acute kidney injury (1) and this significant factor always needs to be considered in patients undergoing cardiac operations with CPB. Mukaida *et al.* aimed to examine whether the time-dose response of the oxygen delivery during CPB can be used to estimate the risk of postoperative AKI. They concluded that maintaining oxygen delivery levels  $>300 \text{ mL/min/m}^2$  may reduce the risk for postoperative AKI development (1).

In addition, high central venous pressure (CVP) is associated with AKI in patients after CPB operations. Increased CVP at the end of surgery was associated with the development of AKI, which was especially observed in patients with a CVP  $>10 \text{ cmH}_2\text{O}$  (13). Yang *et al.* showed that an increased CVP was a strong and independent determinant of all-cause mortality among patients who underwent CPB procedures (13).

Another important issue to be mentioned in patients undergoing heart operations under CPB, is the portal flow pulsatility and intrarenal flow alterations. These are markers of venous congestion and are independent risk factors for AKI after CPB. Prevention of congestive cardiorenal syndrome needs to be pursued (14). These Doppler features are non-invasive procedures available at a minimal cost. A personalized treatment strategy aimed at preventing or reversing portal and intrarenal flow alterations along with other clinical characteristics of each patient may prevent AKI in this group of patients.

Our experience from paediatric cardiac surgery shows that AKI is a common complication, associated with increased morbidity and mortality. Renal and extrarenal risk factors can be responsible for AKI after cardiac surgery in children. Preoperative serum albumin level, age  $<12$  months, preoperative pulmonary hypertension and CPB duration have been associated with AKI in children undergoing congenital heart surgery (15). Early fluid overload is a

significant factor that contributes to the occurrence of AKI. Recent studies suggest that there are sensitive urine biomarkers such as neutrophil gelatinase-associated lipocalin, cystatin C, liver fatty acid-binding protein, kidney injury molecule-1 and interleukin-18, which can be used for an early identification of postoperative AKI. The first three markers increase at 2–4 hours and the last two increase at 6–12 hours after surgery (16). These markers can be very useful tools for early detection of AKI in order to prevent its occurrence or to target the treatment modality for these patients. In addition, again in paediatric population, Volovelsky *et al.* described that pre- and postoperative serum fibroblast growth factor 23 (FGF23) are higher in children who develop AKI after cardiac surgery. In that way, FGF 23 may be able to detect subclinical kidney injury and may be used as a prognostic factor for AKI development after paediatric cardiac surgery (17). From these studies in paediatric population, we can learn and expect things that can be focused on cardiac surgery in adults.

Risk for AKI after cardiac surgery is undoubtedly multifactorial and it is very difficult to isolate independent risk factors for this severe complication. Lim *et al.* described that high volume administration of saline in the period following cardiac surgery is not associated with a significant increase in the risk of AKI (12). However, randomized controlled and prospective studies are warranted in order to clarify these well documented results.

## Acknowledgements

None.

## Footnote

*Conflicts of Interest:* The authors have no conflicts of interest to declare.

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**Cite this article as:** Kanakis M, Martens T, Muthialu N. Postoperative saline administration following cardiac surgery: impact of high versus low-volume administration on acute kidney injury. *J Thorac Dis* 2019;11(Suppl 9):S1150-S1152. doi: 10.21037/jtd.2019.04.28