COMENTÁRIO Editorial

Paulo Neves Serviço de Cirurgia Cardiotorácica Centro Hospitalar de Vila Nova de Gaia/Espinho, EPE Vila Nova de Gaia, Portugal pauloacn@gmail.com

Post cardiopulmonary bypass renal failure – do we need goal directed perfusion?

Cardiopulmonary bypass - associated acute kidney injury (CPB-AKI) is a very common complication in cardiac surgery, occurring in up to 50% of patients.¹ CPB-AKI is now recognized as an independent risk factor for mortality after cardiac surgery.² The pathophysiology of CPB-AKI is multifactorial and includes perioperative inflammation, hemolysis, changes in renal perfusion, oxidative stress, and ischemia-reperfusion injury.³ All these factors are causing glomerular damage and ultimately necrosis, resulting in a decrease of glomerular filtration rate (GFR).

Assessment of serum creatinine (SCr) levels is the current gold standard for diagnosis of GFR changes and AKI. However, it is very important to keep in mind that SCr levels only start to increase after loss of more than 50% of glomerular function. Therefore, most of the times, both diagnosis and therapy of AKI are delayed. Nevertheless, it is well validated that early recognition and treatment is critical for the recovery of kidney function.^{4,5}

Therefore, to early recognize and treat this potential lethal complication, it is essential to understand the risk factors associated with CPB-AKI. *Moreira et al.*, in the paper published in this issue of the Journal, found that age, CPB time, urine output during CPB, mannitol and furosemide administration during CPB were risk factors for CPB-AKI development. Additionally, it was reported that CPB had an influence on renal function's evolution in postoperative period after cardiac surgery, and that it may lead to the development of AKI. The percentage of patients who developed this complication (19%) is aligned with the literature. While some of these identified risk factors and non-modifiable ones, others can be modified, such as loop diuretic and mannitol administration.

Moreira et al. reported no statistically significant differences between the two groups (no AKI vs AKI) regarding preoperative values of hemoglobin and hematocrit (HCT), nor with respect to minimum hemoglobin values during CPB, and hematocrit, or as to addition of blood in the priming, blood transfusion during CPB or administration of blood collected in cell-saver. Regarding this issue, a

relationship between the nadir HCT value during CPB and postoperative AKI was first reported in 1994.⁶ This finding was further confirmed, and some authors have hypothesized that inadequate oxygen delivery (DO2) may be the associated mechanism between severe hemodilution on CPB and CPB-AKI.7 Plus, it was subsequently identified a critical DO2, around 260 to 272 mL·min⁻¹·m⁻² for patients undergoing mild hypothermic (>32°C) CPB. Based on these observations, there was developed the concept of goal--directed perfusion (GDP), shifting the target of the CPB from the cardiac index to the DO2, which should be maintained above the aforementioned critical level.⁸ To validate these findings in a higher level, Goal-Directed Perfusion Trial (GIFT) was designed to test the hypothesis that the GDP approach to avoid a DO2 nadir <280 mL·min⁻¹·m⁻² will decrease the degree of CPB-AKI in patients undergoing CPB with mild hypothermia. GIFT was able to demonstrate that GDP is effective in reducing risk of early stages of CPK-AKI, namely any SCr increase and Acute Kidney Injury Network (AKIN) stage 1.9

In conclusion, paper from Moreira et al. brings another contribution to this very important topic. GIFT results do not unquestionably advise a change in clinical practice, given the fact that GDP was only able to reduce minor degrees of CPB-AKI. Therefore, further studies are needed to define perfusion interventions that may decrease more severe degrees of CPB-AKI.

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