Review of: "Acute kidney injury and acute kidney recovery following Transcatheter Aortic Valve Replacement"

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References

Authors

Marilou Peillex, Benjamin Marchandot, Kensuke Matsushita, Eric Prinz, Sebastien Hess, Antje Reydel, Marion Kibler, Adrien Carmona, Antonin Trimaille, Joe Heger, Helène Petit-Eisenmann, Annie Trinh, Laurence Jesel, Patrick Ohlmann, Olivier Morel.

Reference

Peillex M, Marchandot B, Matsushita K, Prinz E, Hess S, Reydel A, et al. (2021) Acute kidney injury and acute kidney recovery following Transcatheter Aortic Valve Replacement. PLoS ONE 16(8): e0255806. https://doi.org/10.1371/ journal.pone.0255806

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Why this study – the rationale/objective?

Acute kidney injury (AKI) represents a common complication after aortic valve replacement, both transcatheter and surgical, with an estimated incidence ranging from 12% to 28%. AKI correlates strictly with in-hospital, short- and long-term mortality after TAVI and the predictors of AKI occurrence have been widely described. Recently, the novel concept of acute kidney recovery (AKR) after TAVI has been proposed, based on the theoretically improvement of renal function that follows the increase of cardiac output, renal perfusion and with the reduction of glomerular venous congestion. Available data on AKR are sparse and report chronic kidney disfunction as the major predictor. However available data on the impact of AKR on clinical outcomes are conflicting.

[1][2]

How was it executed – the methodology?

- This is a retrospective, observational, unblinded, single-center study that included 584 TAVI patients for severe aortic stenosis.
- AKI was defined as an increase in serum creatinine ≥0.3 mg/dL or ≥ 50% increase in serum creatinine up to 72 hours after TAVR.
- AKR was mirroring defined as 1) an absolute decrease ≥0.3 mg/dL or a relative decrease ≥ 50% in serum creatinine up to 72H or 2) a 25% improvement of eGFR over 72H after TAVR.
- The primary endpoint was the incidence of AKI and AKR following TAVI.
- The secondary endpoints included the incidence of all-cause mortality and a composite endpoint of cardiac death, stroke, myocardial infarction and rehospitalization for heart failure at follow-up among AKI, AKR and unchanged renal function groups.

What is the main result?

- AKI occurred in 8.3%, while AKR in 15.7 % of patients.
- At univariate analysis CKD, EuroSCORE, contrast volume and non-transfemoral approach were predictors of AKI, with CKD that confirmed significance at the multivariate analysis (HR 3.9; 95%CI 1.7-9.2, $p<0.001$).
- By univariate analysis basal creatinine, procedure duration and major bleeding were predictors of AKR, with baseline creatinine level that remained the sole independent predictor after multivariate analysis was performed (HR: 1; 95% CI 1 to 1.1 $p < 0.001$).
- At a median follow-up of 608 days AKI and AKR were both related to an increased CV mortality, compared to unchanged renal function (14.6% and 17.8% respectively, vs. 8.1%, CI 95%, $p<0.022$), while no significant difference was reported for all-cause mortality, non-CV death and MACE. By multivariate analysis COPD (HR: 2.4; 95% CI 1.17–4.95; $p = 0.017$) and 72-hours post procedural AKR (HR: 2.26; 95% CI 1.14 to 4.88; $p = 0.021$) were strong independent predictor of CV mortality.

Critical reading and relevance for clinical practice

This report increases the knowledge about AKR in an all-comers real-world TAVI population. Indeed, conflicting results have been provided on AKR predictors and clinical implications. AKR after TAVI was firstly defined by Azarbal et al[2]as a 25% improvement in eGFR at 48 hours after TAVI. Based on a population of 366 patients, AKR resulted to be predicted by lack of chronic beta-blocker use, male gender, CKD. The same authors on a 1502 TAVI population from the Northern New England registry, reported AKR to occur in 25% of the procedures and to be predicted by CKD, COPD and previous aortic valve surgery, while DM, anaemia and high STS score (>6.1) were less likely linked with AKR. In this report AKR was defined as an increase of GFR >25% at discharge as compared to the admission value[3].

Nijenhuis et al, on a 639 TAVI population, using a different definition criteria of AKR (post to pre-TAVR ratio within 48H ≤0.80) reported a potential protective on two-year mortality rate (HR 0.53, 95%CI 0.30–0.93) compared with a stable kidney function. In this report also the predictors of AKR were conflicting with previous experience: indeed independent predictors were female gender, preserved kidney function, haemoglobin level and absence of atrial fibrillation[4].

Conversely, Pighi et al on a 674 TAVI population, defining AKR as for Azarbal's definition an increase of GFR >25% at discharge, reported an incidence of AKR of 14.5%; in this analysis the association between extravalvular cardiac damage (EVCD), AKI, AKR and their interaction with clinical outcomes was analyzed. Advanced EVCD was related to a higher rate of AKR (23.8% vs. 12.8%; p<0.01), as well as CKD. AKR had no significant impact on reduction of 12-month all-cause mortality but was associated with an improvement of renal function at 12-months[5].

In conclusion, it is necessary to emphasize that at present a validated and shared definition of AKR is lacking and consequently different and conflicting predictors have been proposed, with conflicting interaction with clinical outcomes at long-term follow-up. Thus a shared and common effort is needed in order to provide a valid definition, with clear pathogenic basis and the consequent impact on cardiac and non-cardiac clinical outcomes.

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