Copeptin as a novel biomarker for detecting early renal dysfunction after TAVI

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Background: Acute kidney injury (AKI) is one of the most prevalent (10–30%) complications after transcatheter aortic valve implantation (TAVI). Furthermore, AKI is accompanied by increased mortality, a higher incidence of dialysis and blood transfusion, and a prolonged hospital stay. Although measurement of serum creatinine is the gold standard in diagnosing AKI, changes in serum creatinine may lag behind compromised renal function. Arginine vasopressin (AVP), or antidiuretic hormone, is a nine-amino acid peptide member of the hypothalamo-neurohypophysial axis. Copeptin is the C-terminal moiety of the AVP precursor pre-proAVP that is secreted into the circulation. Recently, copeptin has been suggested to play a role in chronic kidney injury. We evaluated the value of copeptin in the prediction of AKI in patients undergoing TAVI.

Methods: All patients with severe aortic valve stenosis undergoing TAVI between May 2011 and May 2016 were included in our study. AKI was defined by the VARC-2 definition. Patients with no AKI and stage 1 AKI were compared with patients with stage 2 or 3 AKI. Routine laboratory parameters, including creatinine, were measured immediately after blood draw. Additionally, venous blood samples were collected on admission and after 24, 48, and 72 hours, processed immediately, and stored at –80°C until assay. The copeptin concentration in serum was measured by a sandwich immunoluminometric assay.

Results: Copeptin levels were available in 642 patients who were treated by TAVI in our centre from 2012–2016. AKI was detected in 113 patients (17.6%), including 61 patients with stage 1 (9.5%), 29 with stage 2 (4.5%), and 23 with stage 3 (3.6%).

There were no differences among these patients in baseline measurements, but serum copeptin increased in all patients with AKI 24 h post-procedure according to the AKI stage: no AKI 34.5 (18.0–59.3 pmol/L), AKI stage 1: 68.7 (34.6–130.1 pmol/L); AKI stage 2: 96.0 (48.1–185.1 pmol/L); AKI stage 3: 154.9 (79.5–280.7 pmol/L); ANOVA $p<0.001$ (Fig. 1). Copeptin showed an earlier and sharper increase than creatinine (Fig. 1), with a negative predictive value of 0.97 to rule out AKI after 24 h.

Conclusion: AKI subsequent to TAVI is a common and harmful complication that occurred in almost every 5th patient (17.6%) in our cohort. AVP is secreted in response to hypotension, which commonly occurs during TAVI. In our cohort of TAVI patients, those who developed AKI after TAVI showed a rapid increase in copeptin that was earlier than that of creatinine. In light of these observations, copeptin could be a new parameter for detecting early renal dysfunction.

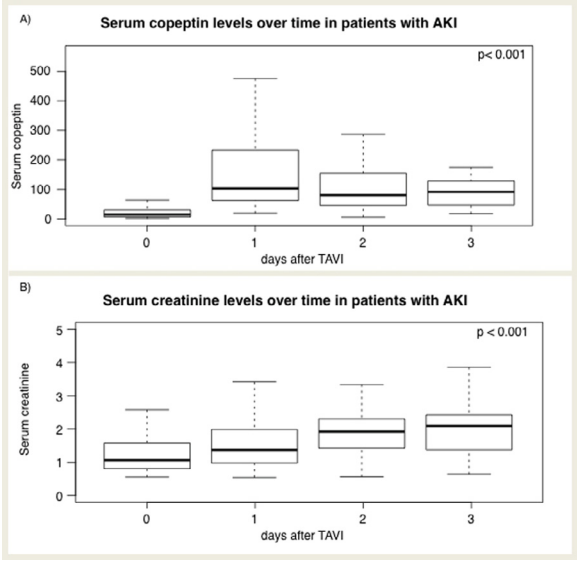

Figure 1