TCT-148

Blood Transfusion And The Risk Of Acute Kidney Injury Following Transcathereter Aortic Valve Implantation.

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Background: Blood transfusion is associated with acute kidney injury (AKI) after transcatheter aortic valve implantation (TAVI). We sought to elucidate in more detail the relation between blood transfusion and AKI and its effects on short- and long-term mortality.

Methods: 995 patients with aortic stenosis underwent TAVI with the Medtronic-CoreValve or the Edwards Valve in 7 centers. AKI was defined by the Valve Academic Research Consortium (absolute increase in serum creatinine ≥0.3 mg/dl (≥26.4 μmol/l) or ≥50% increase ≥72 hr). Logistic and Cox regression was used for predictor and survival analysis.

Results: AKI occurred in 20.7% (n=206). The number of units of blood transfusions ≤24 hr was the strongest predictor of AKI (p<0.001; OR: 4.81 [1.45-15.95], 3-4 units, OR: 3.05 [1.24-7.53], 1-2 units, OR: 1.47 [0.98-2.21] compared to patients without anemia before TAVI (p=0.001)). AKI and life-threatening bleeding were independent predictors of 30-day mortality (OR: 3.04 [1.52-6.07], OR: 5.39 [2.14-13.57], respective) while transfusion (≥3 units), baseline anemia and AKI predicted mortality beyond 30 days.

Conclusions: AKI occurred in 21% of patients after TAVI. The number of blood transfusions but not the indication of transfusion predicted AKI. AKI was a predictor of both short- and long-term mortality whereas blood transfusion predicted long-term mortality. These findings indicate that outcome of TAVI may be improved by more restrictive use of blood transfusions.

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Impact of Chronic Kidney Disease on Myocardial Infarct Size and Adverse Events in ST-Elevation Myocardial Infarction: Results from the INFUSE-AMI Trial

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Background: Chronic kidney disease (CKD) patients have less favorable outcomes after ST-elevation myocardial infarction (STEMI) for yet unclear reasons.

Methods: The INFUSE-AMI trial randomized patients with STEMI due to proximal or mid-LAD occlusion to intracoronary budesonide (Clearway RX catheter) vs. placebo, and to thrombus aspiration (Export) vs. no aspiration. We compared infarct size as % of LV mass assessed by magnetic resonance imaging at 30-days, myocardial reperfusion and incidence of adverse events between patients with vs. patients without CKD. CKD was defined as a creatinine clearance < 60 ml/min.

Results: Patients with CKD (n=59, 14.4%) were older, more often female, diabetic and less likely to undergo angiography within 3 hours of symptom onset (50.8% vs. 72.2%, p=0.001) compared to those without CKD (n=349, 85.5%). Following PCI, final thrombolysis in myocardial infarction (TIMI) 3 flow (88.4% vs. 92.8%, p=0.12) and myocardial blush grade (MBG) 3 (64.4% vs. 70.5%, p=0.35) was observed similarly in both groups. Median infarct size was non-significantly larger in CKD patients (19.3% vs. 17.0%, p=0.34). The incidence of 30-day adverse events, were significantly higher in those with CKD (Figure). There were no significant differences in stent thrombosis, reinfarction or revascularization between groups.

Conclusions: Differences in infarct size between patients with and without CKD presenting with STEMI are modest and unlikely to account for the significantly higher short-term cardiac risk with CKD.

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Prognostic Value of Different Definitions of Contrast Induced Acute Kidney Injury in STEMI: Analysis from the HORIZONS-AMI Trial

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Background: Several definitions of acute kidney injury (AKI) are in use, and the optimal absolute and relative increase of serum creatinine increase after contrast administration to define contrast-media induced acute kidney injury (CAKI-AMI) is still a matter of debate. Moreover, the prognostic relevance of AKI according to the varying definitions in STEMI has not been established.

Methods: Serum creatinine concentration data within 48h after coronary angiography was present in 2975 STEMI pts in the HORIZONS-AMI trial. Patients were analyzed according to different AKI definitions (AKIN-, modified AKIN- (mAKIN), Waikar-Bonventre (wb), percentage-change of creatinine (<25%, 26-50%; 51-75%; >75%) and to the commonly used “standard-definition” of a relative increase in serum creatinine of ≥25% or an absolute increase of ≥0.5 mg/dl). The primary endpoint was all-cause mortality at 3 years.

Results: Depending on definitions the incidence of CI-AKI ranged from 5.0% to 15.5%. Similarly, 3-year mortality rates differed substantially with respect to the different CI-AKI definitions (Table). Absolute changes of creatinine were strongly associated with all-cause mortality, starting with an increase of 0.3mg/dl absolute increase of creatinine above baseline (HR 3.68 p<0.001). Figure – the cutoff level used in the AKIN- and modified AKIN-definitions. The increased risk associated with >0.3mg/dl absolute increase of creatinine was independent of the amount of contrast-media used.

Conclusions: The modified AKIN criteria might be the optimal definition for contrast-induced AKI after primary PCI in STEMI.