# **CONTRAST INDUCED ACUTE KIDNEY INJURY** MID-TERM KIDNE **DOES NOT AFFECT** FUNCTION AND MORTALITY

# Medical University of Graz

<u>Werner Ribitsch<sup>1</sup>, Joerg H Horina<sup>1</sup>, Franz Quehenberger<sup>2</sup>, Alexander R Rosenkranz<sup>1</sup>, Gernot Schilcher<sup>1</sup></u>

<sup>1</sup>Clinical Division of Nephrology, Department of Internal Medicine, Medical University of Graz, Austria <sup>2</sup> Institute for Medical Informatics, Statistics and Documentation, Medical University of Graz, Austria

# **Introduction and Objectives**

Contrast induced acute kidney injury (CI-AKI) is considered a serious

complication of contrast enhanced procedures. We investigated the

incidence of CI-AKI in patients undergoing intra-arterial angiography and its

impact on mid term kidney function and mortality.

### **Methods**

We conducted a prospective observational trial on patients undergoing intra-arterial angiography. All subjects received standardized intravenous hydration pre-intervention without any other concomitant protective measure. Monomeric, non-ionic, low osmolar iomeprol was used as contrast media. CI-AKI was defined according to a  $\geq 25\%$  increase of creatinine from

### Table 1

	No CI-AKI (n=678)	CI-AKI (n=53)	р
Age (yrs)	74 (37-91)	76 (52-89)	0.13
Female, n (%)	309 (46)	18 (43)	0.76
Body mass index, kg/m <sup>2</sup>	27.3 ± 4.4	26.6 ± 3.6	0.26
Diabetes, n (%)	270 (40)	18 (34)	0.4
Contrast volume, mL	108.3 ± 52.4	115 ± 48	0.37
Creatinine d <sub>-1</sub> (mg/dl)	1.31±0.42	1.20±0.29	0.056
Creatinine d <sub>1</sub> (mg/dl)	1.24±0.38	1.51±0.4	<0.001
Creatinine d <sub>2</sub> (mg/dl)	1.28±0.40	1.64±0.51	<0.001
Creatinine m1 (mg/dl)	1.17±0.42	1.23±0.45	0.41
Creatinine m3 (mg/dl)	1.14±0.41	1.16±0.35	0.72
Urinary protein (mg/gCreatinine)	100 (0 – 7291)	123 (37 – 3135)	0.13
NT-pro-BNP d <sub>-1</sub> (ng/L)	627 (6 – 35001)	732 (61 – 35001)	0.38
Hospital stay (days)	2 (1 – 76)	3 (1 – 35)	0.003
Mortality, n (%)	100 (15)	5 (10.2)	0.38

baseline to either 24hrs or 48hrs after angiography. Univariate predictors of

CI-AKI were derived by logistic regression analysis. Plasma creatinine was measured one month (m1) and three months (m3) after hospital discharge. Patients were followed up for two years to investigate the long term effects of CI-AKI on mortality.

# Results

We studied 731 (332 female) patients with a mean eGFR of 52.1±15.3 ml/min. The incidence of CIN was 7.3% (53 patients). Patients with CI-AKI had a lower creatinine and a higher eGFR at baseline, but no other independent predictors of CI-AKI could be identified by logistic regression



analysis. Patients who developed CI-AKI were significantly longer hospitalized

(median three days, Q1-Q3 2-6 days) than patients without CI-AKI (median two days, Q1-Q3 2-4 days; p = 0.009). Kidney function was not different between both groups one and three months after discharge. Median follow up was 25.5 months and mortality was not different between patients with CI-AKI (10.2%) and without CI-AKI (15%; p=0.38, Tab.1)

Overall survival of patients with CI-AKI and without CI-AKI (p = 0.38)

# Conclusions

The incidence of CI-AKI in our cohort was much lower than frequently reported without any negative impact on mid-term kidney function and mortality. Therefore, clinically indicated angiographic studies should not be withheld.

## 54<sup>th</sup> ERA-EDTA Congress June 3<sup>rd</sup> - June 6<sup>th</sup>, Madrid, Spain



DOI: 10.3252/pso.eu.54ERA.2017



•com