## 208 PREDICTION OF CONTRAST INDUCED NEPHROPATHY USING NOVEL BIOMARKERS FOLLOWING ELECTIVE CONTRAST CORONARY ANGIOGRAPHY

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**Introduction** Chronic Kidney Disease (CKD) is a risk factor for contrast induced nephropathy (CIN), defined as an increase in serum creatinine of >25% from baseline or a delta rise of >26.5 µmol/L within 48 h. Early diagnosis of CIN requires validated novel biomarkers.

**Methods** A prospective observation study of 301 consecutive CKD patients undergoing elective invasive coronary angiography was performed. Low-osmolar contrast was standard. Demographics and Mehran risk score were recorded. Samples for plasma neutrophil gelatinase-associated lipocalin (NGAL), serum liver fatty acid-binding protein (L-FABP), serum kidney injury marker 1 (KIM-1), serum interleukin 18 (IL-18) and serum creatinine were taken at 0, 1, 2, 4, 6 and 48 h post contrast. Urinary NGAL and urinary cystatin C (CysC) were collected at 0, 6 and 48 h. Incidence of major adverse clinical events (MACE); acute myocardial infarction, heart failure hospitalisation, stroke and death were recorded at 1 year.

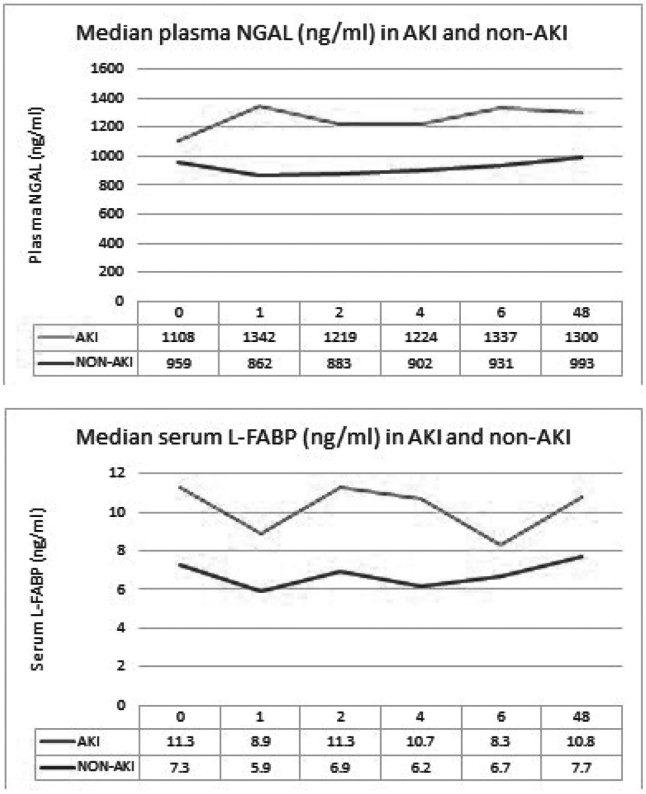
**Results** CIN occurred in 28 (9.3%) patients and were independently associated with older age, diabetes, higher Mehran score, larger contrast volume and anaemia ($p < 0.05$). Logistic regression analysis showed diabetes, CKD stage and GFR to be most predictive of CIN. The predictive power of plasma NGAL was greatest at 6 h with median levels of 1,337 ng/ml in CIN patients compared with 931 ng/ml in non-CIN patients ($p = 0.002$, AUC 0.71, sensitivity 75.0%, specificity 96.1%, OR 2.86), see figure 1 and table 1. L-FABP performed best at 4 h with median levels of 10.7 ng/ml in CIN patients compared with 6.2 ng/ml in non-CIN patients, $p = 0.001$, AUC 0.69, sensitivity 42.3%, specificity 90.2%, OR 6.75, Figure 1 and Table 1. Median urinary NGAL was higher only after 48 h, 487 ng/ml in CIN patients versus 155 ng/ml in non-CIN patients, $p = 0.008$, AUC 0.63. CysC, IL-18 and KIM-1 were not predictive at any time-point ($p > 0.05$). A Mehran score â%¥10 performed prior to procedure achieved an AUC of 0.65, $p = 0.006$. MACE occurred in 7 (25.0%) CIN patients but only 17 (6.2%) non-CIN patients ($p < 0.001$). CIN cases also had considerably higher mortality (10.7% compared to 3.3%, $p = 0.037$). Exploratory analysis showed that the combination of Mehran score >10, 6 hr NGAL and 4 hr L-FABP improved specificity to 96.7%. Figure 2 highlights a proposed pathway of how biomarkers could be used to identify CIN early and facilitate timely therapeutic intervention to reduce morbidity and mortality.

**Conclusions/implications** Mehran risk score, 6 h plasma NGAL and 4 h serum L-FABP performed best at early CIN prediction. CIN patients were four times more likely to develop MACE and had a trebling of mortality risk at 1 year. The implications of our results, translated to the design of safer elective coronary intervention services able to more efficiently manage the increasing volume of contrast studies, should be a key health priority for cardiac and renal services.

**Abstract 208 Table 1** Summary of NGAL (ng/ml) and L-FABP (ng/ml) in AKI and non-AKI patients

| Biomarker | Time (hr) | Median CIN | Median non-CIN | AUC | P value |
|---|---|---|---|---|---|
| NGAL | 0 | 1108 | 959 | 0.62 | 0.046 |
| | 2 | 1219 | 883 | 0.68 | 0.004 |
| | 4 | 1224 | 902 | 0.65 | 0.014 |
| | 6 | 1337 | 931 | 0.71 | 0.002 |
| L-FABP | 0 | 11.3 | 7.3 | 0.65 | 0.011 |
| | 2 | 11.3 | 6.9 | 0.67 | 0.007 |
| | 4 | 10.7 | 6.2 | 0.69 | 0.001 |
| | 6 | 8.3 | 6.7 | 0.63 | 0.045 |

Sensit: sensitivity; specif: specificity; PPV: positive predictive value; NPV: negative predictive value; RR: relative risk; OR: odds ratio




**Abstract 208 Figure 1** Median plasma NGAL (ng/ml) and serum L-FABP (ng/ml) in AKI and non-AKI

## 209 WHOLE EXOME SEQUENCING IDENTIFIES GENETIC CAUSE OF HISTIOCYTOID CARDIOMYOPATHY

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