Impact of point-of-care creatinine and eGFR measurements on the risk of contrast-induced acute kidney injury for patients with ST segment elevation myocardial infarction undergoing primary PCI

Grigoris V Karamasis1,2, Firas Al-Janabi1, Shah Mohdnazri1,2, Michael Parker3, Henry Seligman1, Adam Ioannou1, Tony Everitt1, Rohan Jagathesan1, Alagmir Kabir1, Jeremy W Sayer1, Nicholas M Robinson1, Rajesh K Aggarwal1, Gerald J Clesham1, Reto A Gamma, Paul A Kelly1, Kare H Tang1, John R Davies1, Thomas Keeble1,2

1Department of Cardiology, The Essex Cardiac Centre, Basildon, United Kingdom
2Anglia Ruskin University, East of England, United Kingdom

Background: Contrast-induced acute kidney injury (CI-AKI), defined as a serum creatinine increase of ≥0.5 mg/dL or ≥25%, is a recognised complication during primary percutaneous coronary intervention (PPCI) that affects short and long term prognosis. Volume of contrast media used during the procedure is a known predisposing factor for the development of CI-AKI.

Purpose: The aim of this study was to assess the impact of point-of-care pre-PPCI creatinine and eGFR measurements with immediate feedback to the operator on the risk of developing CI-AKI for patients presenting with ST segment elevation myocardial infarction (STEMI). The hypothesis was that in patients found to have renal impairment prior to the procedure, less contrast media volume would be used, leading to a decreased incidence of CI-AKI.

Methods: 160 patients presenting with STEMI in a single tertiary cardiac centre due to undergo PPCI were recruited (STATCREAT group). Presentation with cardiogenic shock was the only exclusion criterion. Point-of-care creatinine and eGFR were measured prior to the procedure with a handheld analyser (StatSensor, Nova Biomedical, MA, USA) with the results available within 30 seconds and instantly fed back to the operator. The STATCREAT group was compared with a similar retrospective cohort of STEMI patients who had PPCI from the previous 6 months (n=294). For these patients (Control group) point-of-care blood test was not performed, so the operator was unaware of their baseline creatine and eGFR at the time of PPCI. For the purpose of the analysis the two groups were further divided to subjects with chronic kidney disease (CKD) prior to PCI (eGFR < 60) and subjects with normal renal function (eGFR > 60).

Results: 21% (n=34) from the STATCREAT and 19% (n=56) from the control group were found to have CKD pre PCI. For these patients, contrast media volume used was significantly reduced for the STATCREAT group (124.6 ml vs. 152.3 ml, p = 0.04). Only 5.9% (n=2) in the STATCREAT group developed CI-AKI compared with 17.9% (n=10) in the control one. This difference did not reach statistical significance (p=0.12) possibly due to small numbers. There was no difference in the number of lesions treated (1.1 vs. 1.2, p=0.64) or number of stents used (1.18 vs. 1.25, p=0.78). For the two groups of patients with normal renal function prior to the procedure, the volume of contrast media used was numerically higher for the STATCREAT group (172.4 ml vs. 158.4 ml, p=0.07). There was no difference in the incidence of CI-AKI (16.7 vs. 13.4, p=0.4), the number of lesions treated (1.2 vs. 1.2, p=1.0) or the number of stents used (1.21 vs. 1.16, p=0.61).

Conclusions: Pre-PPCI point of care renal function measurement reduced significantly the contrast media volume used in patients who were found to have chronic kidney disease. At the same group contrast induced acute kidney injury was numerically reduced. A large randomised trial is merited to investigate further whether point-of-care creatinine testing can result in better outcomes for STEMI patients undergoing PPCI.