ANEMIA AS A PROGNOSTIC FACTOR IN CONTRAST-INDUCED ACUTE KIDNEY INJURY IN PATIENTS WITH STABLE CORONARY ARTERY DISEASE.

Olga Mironova1, Olga Perekosova1, Alina Ushanova1, Georgy Isaev1, Alexander Ermolaev1, Ekaterina Schelkanovceva1, Polina Lakotka1, Victor Fomin1

1Sechenov University, Internal Diseases, Moscow, Russia

Background and Aims: Contrast-induced acute kidney injury (CI-AKI) remains one of the major obstacles to perform percutaneous coronary interventions (PCI), especially in older patients and in patients with comorbidities. The number of cases of stable coronary artery disease (CAD) requiring such kind of interventions, in spite of optimal medical treatment received, remains high. Diabetes, hyperuricemia and other components of metabolic syndrome, as well as heart failure, are well known risk factors predisposing to the development of CI-AKI after contrast exposure. Anaemia is diagnosed in a number of patients without underlying chronic kidney disease (CKD), when they seek for medical help due to CAD.

The aim of our study was to assess the prevalence of CI-AKI (primary outcome) and the prognostic significance of anaemia as a its possible risk factor (secondary outcome) in different groups of patients with stable CAD requiring PCI using the contrast media.

Method: We conducted a single-centre prospective observational cohort study. 561 patients aged 18-89 with stable CAD undergoing PCI were enrolled from June 2012 until October 2013. The CI-AKI was defined as a rise in serum creatinine of ≥0.5 mg/dl (≥44 μmol/l) or a 25% increase from baseline value, assessed at 48-72 hours after PCI. Anaemia was defined according to the WHO definition – haemoglobin level <12.0 g/dl in women and <13.0 g/dl in men. The contrast media used was either iodixanol (isomolar contrast) or iopromide (low-osmolar contrast), which are both known to cause less adverse events than high-osmolar types of contrast. Nephrotoxic drugs were stopped 48 hours before PCI. The 5-year prognosis including all-cause and cardiovascular mortality, myocardial infarction, stroke, gastrointestinal bleeding, decompensation of chronic heart failure, repeat revascularizations (PCI and coronary artery bypass grafting), end-stage renal disease (ESRD) development, was assessed via phone calls and appointments according to the clinical situation and severity of the condition.

Results: The prevalence of CI-AKI in this group of patients was 104 cases (18.5%) (primary outcome). The number of patients with anaemia was higher in the group of patients who developed CI-AKI after PCI (6% [7/104] vs 4.4% [20/457]). The female patients with anaemia were more likely to develop CI-AKI (71% [5/7] vs 35% [7/20]). The number of patients who suffered from MI having anaemia at the inclusion date was 2 (28.6%) vs 6 (30%) in patients with and without CI-AKI respectively. Acute heart failure decompensation in patients with anaemia was significantly higher in patients with CI-AKI (43% [3/7] vs 10% [2/20]). This fact needs further evaluation in larger studies but anaemia may be one of the prognostic factors, worsening the kidney damage and leading to worse cardiorenal outcomes.

Conclusion: Patients with stable CAD suffering from anaemia are more likely to develop CI-AKI even without underlying CKD or ESRD. Female patients with anaemia and stable CAD have higher risk of development of CI-AKI. The combination of CI-AKI and anaemia may lead to a higher 5-year risk of acute heart failure decompensation.