

Evaluation of Neutrophil Gelatinase-associated Lipocalin as a Marker of Open Heart Surgery Related Acute Kidney Injury in Children

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Introduction. Cardiac surgery with cardiopulmonary bypass (CPB) is commonly perceived as a risk factor for decline in renal function. Hypothermia, hypoxia, hypotension, non-pulsatile blood flow during CPB, use of ACE inhibitors, inotropic and (or) vasoactive support affects kidney and contributes to the acute kidney injury (AKI). The conventional biomarker creatinine is not sensitive enough to detect AKI until a significant decline in renal filtration has occurred. Urine neutrophil gelatinase-associated lipocalin (NGAL) is an early biomarker and a predictor of AKI in a variety of clinical settings. Earlier detection of AKI could facilitate evaluation of novel therapeutic strategies.

Aim. The aim of the study is to compare different functional and biological markers of kidney in children undergoing surgical correction of congenital heart lesions.

Material and methods. Between 2012–2013, prospective uncontrolled cohort study was conducted. 32 children with various congenital heart lesions undergoing CPB were enrolled. Median age of patients was 8.5 months, median weight – 7.3 kg. Median duration of CPB was 148 min, median aortic cross-clamp time – 88 min, the lowest median temperature during cooling was 30°C. Urine output, doses of diuretics, vasopressors and inotropics were recorded. Serum creatinine (SCr) level was determined by Jaffé's method (Cobas 6000 analyzer, Roche), serum Cystatin C (CysC) was determined by particle-enhanced nephelometric immunoassay, urine NGAL was determined by ARCHITECT system (Abbott Diagnostics, Illinois, USA). All samples (blood and urine) were collected at designated time periods: on the following morning after completion of surgical repair (1), after 24 hours (2) and after 48 hours (3).

Results. AKI developed in 8 patients (33.3%), with SCr rise by more than 50% from the baseline, but the diagnosis using serum creatinine was delayed by 48 hours after CPB. In contrast, maximum increase in urine NGAL levels was 316-fold within 12 hours after CPB in patients having AKI. Median level of urine NGAL in the first sample was 202.7 ng/ml. In the group of patients with normal renal function the corresponding level of NGAL was 15.7 ng/ml ($p = 0.016$). Median CysC level in first sample in AKI group was 1.195 mg/l vs. 1.055 mg/l in non-AKI group, ($p < 0.1$).

Conclusions. Accurate measurements of urine NGAL are obtained using the ARCHITECT system. Urine NGAL is an early and sensitive biomarker of CPB related AKI.