Biomarkers in early diagnostic of acute kidney injury in patients with acute cardiorenal syndrome

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Background and Objective

Acute kidney injury (AKI) is a common complication of critical illnesses and has a significant impact on outcomes, including mortality and morbidities. Unfortunately, apart from prophylactic measures, no effective treatment for this syndrome is known. Therefore, early recognition of AKI not only can provide better opportunities for preventive interventions, but also opens many gates for research and development of effective therapeutic options. These novel AKI biomarkers complement serum creatinine (SCr) and urine output, which are the standard diagnostic tools for AKI detection.[1]

Purpose: Explore the place biomarkers damage the heart and kidneys in the development of risk assessment, early detection and prediction of short-term outcomes of acute cardiorenal syndrome.

1. Kashani K, Cheungpasitporn W, Ronco C. Clin Chem Lab Med. 2017 Jan 11. pii: /j/cclm.ahead-of-print/cclm-2016-0973/cclm-2016-0973.xml. doi: 10.1515/cclm-2016-0973.

Methods

- On admission, 109 randomly selected patients using immunoassay ELISA analysis to determine the level of biomarkers DOS / AD CHF (NT-pro BNP in serum) and kidney damage (cystatinC in serum; NGAL, KIM-1 and IL-18 in the urine). Statistical analysis was performed using statistical software application package STATISTICA 10 and SPSS 22 using standard algorithms of variation statistics. For all quantitative traits in the two groups was carried out.

1p <0,05, 2p <0,01, 3p <0,001

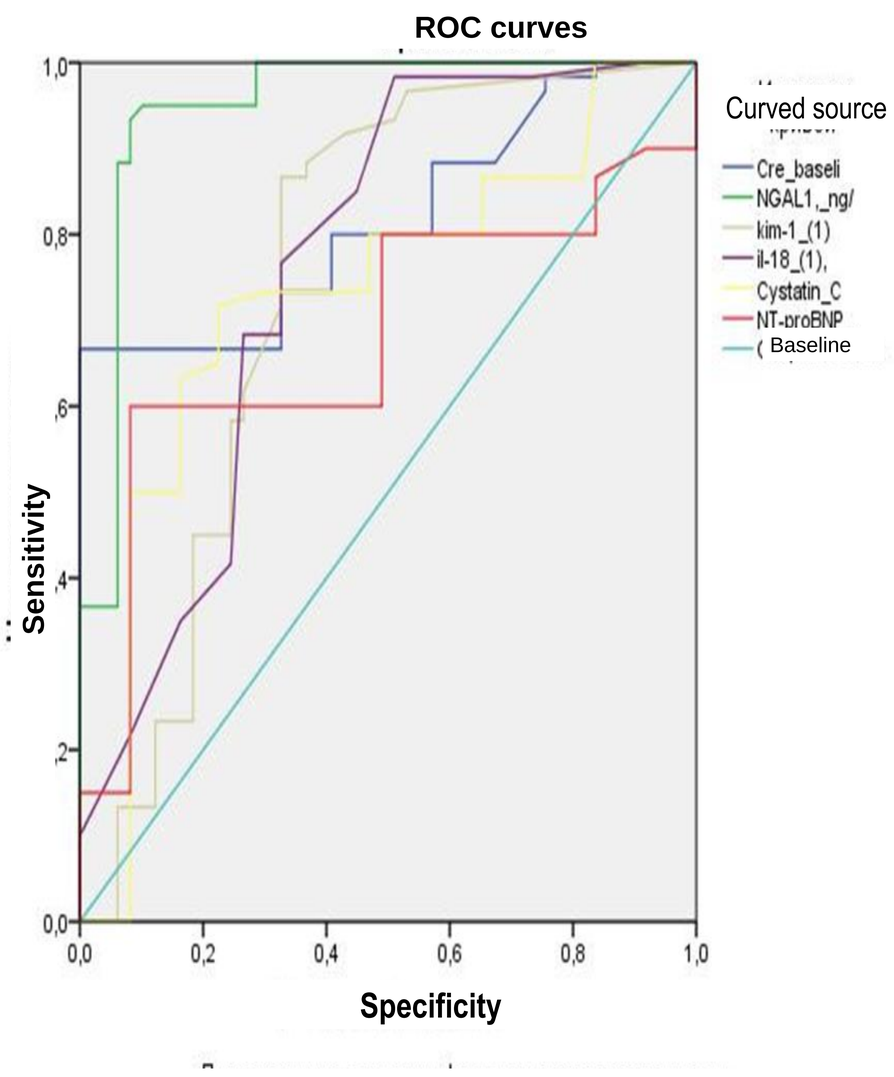
Table 1. Baseline characteristics

Biomarkers	AD CHF		ACS nST		AD CHF+	ACS nST
	AKI+ n=27	AKI- n=24	AKI+ n=33	AKI- n=25	AKI+ n=60	AKI- n=49
NGAL, ng|ml	307,8±262,1	21±16,53³	373,7±343,5	54±87,53³	344±308,8	37,9±65,13³
KIM-1,ng|ml	0,496±0,27	0,236±0,322²	0,982±0,24	0,561±0,742²	0,774±0,36	0,402±0,593³
IL-18 pg|ml	571,6±5,95	551,9±31,32²	569,8±12,7	539,5±25,23³	570,6±10,2	545,5±28,83³
Cystatin C,ng|ml	11344,9± 3049,7	9839,6± 3075,82²	12122,5± 1549,7	9265,1± 2564,63³	11772,6± 2356,5	9546,5± 2811,83³
NT-proBNP, fmol|ml	13251,1± 4467,6	11836,7± 2828,7	12857,1± 3108,8	10134,2± 2479,43³	13034,4± 3751,9	10968,1± 2765,42²

Results

- In 109 patient We examined biomarkers heart damage (NT-pro BNP) and kidney (cystatin C in serum, NGAL, and IL-18 KIM-1 in urine).
- Patients with AKI as compared with patients with stable renal function were detected higher levels of NGAL (p <0.001) and KIM-1 (p <0.01) in all groups, the differences of IL-18 levels in the urine and cystatin C in blood were higher in ACS without elevation ST than in patients with AD CHF (p <0.001 and p <0,01), NT-pro BNP levels were higher in ACS without elevation ST (p <0.001) in patients with AKI compared with patients with stable renal function and no difference in AD CHF group.
- Based on the identified association diagnostically significant markers connection with the development of renal damage AKI, all patients were divided into 4 groups depending on the level of structural damage markers (NGAL ≥60,1 ng / ml and / or a KIM-1 ≥ 0,519 ng / ml) and presence AKI: in group 1 included patients with stable renal function and the level of biomarkers following diagnosis, group 2 included patients with stable renal function and diagnostically relevant levels of biomarkers in the third group were patients with AKI and low levels of biomarkers in 4th group included patients with AKI and diagnostically relevant levels of markers
- The test includes two markers of kidney damage (NGAL and / or KIM-1) has 95% sensitivity and 59% specificity.

Figure 1. The diagnostic value of biomarkers



Diagonal segments are formed by coincidences

Figure 2. Comparative analysis of the quality of regression test models for detecting AKI

Test variable	Area under crooked	Standard error	Asymptotic Significance	Asymptotic 95% Confidence interval	
				Lower border	Upper border
Serum creatinine	0,817	0,040	0,000	0,738	0,897
NGAL	0,948	0,023	0,000	0,903	0,994
KIM-1	0,760	0,052	0,000	0,649	0,851
IL-18	0,750	0,048	0,000	0,666	0,854
Cystatin C	0,730	0,051	0,000	0,630	0,829
NT-proBNP	0,680	0,053	0,001	0,576	0,784

Note: NGAL - associated with neutrophil gelatinase lipokalin, KIM-1 -, IL-18 - interleukin-18, NT-proBNP - NT-terminal fragment of the precursor of the natriuretic peptide.

Figure 3. 7 Distribution of patients with acute cardiovascular pathology, depending on the stability of kidney function and the level of NGAL and / or KIM-parameters

No renal dysfunction	Renal dysfunction
AKI- n=15 (13,8%)	AKI+ NGAL <60,1 ng/ml and KIM-1 <0,519 ng/ml n=3 (2,8%)
Cr n-=34 (31,2%)	AKI+ NGAL-≥ 60,1 ng|ml KIM-1-≥ 0,519 ng|ml n= 57 (53,3%)

Conclusions

Patients with subclinical clinical and laboratory changes (changes in serum creatinine by 10-50% from baseline) did not differ from patients with acute kidney injury on major risk factors, hospital mortality in these patients is lower than in patients with AKI (30% and 12% , p <0.05), but higher than in the group with stable renal function. Defining two structural renal damage markers (NGAL and KIM-1) in high-risk patients to diagnose AKI 95%

Declaration of interest: nothing to declare

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