Original Article

Urine microscopy and neutrophil–lymphocyte ratio are early predictors of acute kidney injury in patients with urinary tract infection

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KEYWORDS
Acute kidney injury; kidney injury molecule-1; Neutrophil–lymphocyte ratio; Quantitative urine microscopy score; Urinary tract infection

Abstract
Objective: Urinary tract infection (UTI) is a common cause of morbidity and hospitalisation in the population worldwide. Upper UTI is indolent and causes subclinical acute kidney injury (AKI) resulting in preventable cause of scarring of renal parenchyma. We explored urinary and serum levels of kidney injury molecule-1 (KIM-1), haematological parameters and quantitative urine microscopy parameters to predict kidney injury.

Methods: Neutrophil–lymphocyte ratio (NLR) is obtained by dividing absolute neutrophil count with absolute lymphocyte count. Quantitative urine sediment microscopy was performed and correlated with clinical, biochemical and haematological findings to predict AKI in patients with UTI. Quantitative ELISA was performed for serum and urine levels of KIM-1. Seventy two adult patients with UTI were enrolled, 45 of whom had AKI while 27 were in the non-AKI group.

Results: NLR (p=0.005) and renal tubular epithelial cell-granular cast score in quantitative urine microscopy (p=0.008) are strong predictors of AKI in patients with UTI while rest of

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quantitative urine microscopy parameters and serum and urinary levels of KIM-1 molecule were not found to be useful in prediction of AKI.

Conclusion: NLR in haemogram is a novel and useful biomarker for predicting AKI in patients with UTI.

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1. Introduction

Urinary tract infection (UTI) is the second most common infectious disease in the community. About 150 million people around the world are diagnosed with UTI per annum, among which 35% are hospital acquired infection [1,2]. The incidence of UTI in adult men up to the age of 65 years is extremely low. Women in this age group, however, more commonly experience UTI. Incidence of UTI in patients more than 65 years old increases dramatically for both genders, with a progressive decrease in female to male ratio [3]. Many microorganisms cause UTI, among which 35% are hospital acquired infection [1,2]. The incidence of UTI in adult men up to the age of 65 years is extremely low. Women in this age group, however, more commonly experience UTI. Incidence of UTI in patients more than 65 years old increases dramatically for both genders, with a progressive decrease in female to male ratio [3]. Many microorganisms cause UTI, among which 35% are hospital acquired infection [1,2].

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2. Patients and methods

2.1. Study design

The present study was a cross sectional study, conducted in the Department of Pathology, Jawaharlal Institute of Postgraduate Medical education and Research (JIPMER), Puducherry, for a period of 1 year from January 2017 to December 2017. Study was approved by Institute Ethics committee (JIP/IEC/2016/1027). Ethical clearance was obtained as the study involved collection of venous blood and urine samples as well as following up the patient with clinical and biochemical parameters. Informed written consents were obtained from all patients under “more than minimal risk category” as per Indian Council of Medical Research 2017 guidelines which follow the Declaration of Helsinki guidelines according to the latest version in October 2013 at Fortaleza, Brazil.

2.2. Setting and participants

Seventy two consecutive patients were enrolled for the study after obtaining the informed ethical consents, among which 45 participants had UTI fulfilling criteria for AKI as per KDIGO 2012 guidelines. Remaining 27 participants with UTI did not have AKI. Serum creatinine level alone was used to define and assess the severity of AKI as well as for statistical analysis. The values of serum creatinine and urine output correlated with the severity of AKI.

Adult patients with UTI and age more than 18 years old only were included in the study. Individuals with prior
kidney transplant, end stage kidney disease or chronic dialysis therapy, prior renal replacement therapy during index hospitalization, and catheter associated UTI were excluded from the study to avoid confounding factors for urine microscopy and urine KIM value assessment.

2.3. Study definitions

AKI was defined based on KDIGO 2012 guidelines [10] and UTI was defined by clinical features and presence of more than 5 pus cells in high power field of urine sediment microscopy [18].

2.4. Study protocol and data sources

Clinical data including patient demographics (age and sex), history of diabetes mellitus, hypertension, and base line laboratory findings such as serum creatinine were obtained from medical records and hospital information system. Blood and urine samples were collected from each patient at the time of enrolment.

2.5. Urine microscopy preparation

A fresh, midstream clean catch urine sample was collected in a sterile screw capped bottle. An aliquot of 15 mL urine was centrifuged at 2000 rpm for 5 min. Urine supernatant was removed but 0.5 mL of supernatant was retained in the tube and the pellet was resuspended with a pipette. Samples were processed immediately and analysed within 4 h of sample collection.

One drop (about 25 μL) of sediment was transferred on to a microscopic glass slide and a cover slip was placed over it. The slide was examined under a standard light microscope and a phase contrast microscope at 10×, 20× and 40×. RBCs, pus cells, renal tubular epithelial (RTE) cells, epithelial cells, RTE cell casts, red cell casts, white cell casts, hyaline casts, granular casts and microorganisms were identified based on standard definition and their numbers were recorded [14]. The urinary scoring system by Perazella et al. [14] (Table 1) was used for subsequent analysis. All the investigators who performed microscopy were blinded to the clinical history and outcome.

2.6. Serum and urine KIM-1

Two millilitres of blood was collected and transferred into a non-vacuum plastic tube without any anticoagulants for serum KIM-1. Blood was centrifuged after clot formation at 3000 rpm for 5 min and the serum and the urine supernatant for urine KIM-1 was stored at –80°C for batched analysis. ELISA was done with Human Kidney Injury Molecule-1 ELISA Kit (Bioassay Technology Laboratory, Shanghai, China).

Results were calculated from the standard curve constructed by plotting the average optical density for each standard on vertical Y axis against the concentration on the X axis. The curve was plotted and results were calculated by using Microsoft Excel software.

2.7. NLR

Complete haemogram was performed in Sysmex XT-2000i (Sysmex Corporation, Kobe, Japan) from ethylene diamine tetra acetic acid anticoagulated blood and NLR was obtained by dividing absolute neutrophil count with absolute lymphocyte count.

2.8. Statistical analysis

Continuous variables were summarized as mean (standard deviation [SD]) or median (inter-quartile range) based on normality. Categorical variables were summarized as frequency with proportions. Association of continuous variables that satisfied normality was compared between AKI and non-AKI group using independent t-test. Non-normally distributed data were compared using McNemar test. Association of various categorical variables between AKI and non-AKI groups were assessed using Chi-square test. A p-value <0.05 was considered as statistically significant. The agreement between techniques (light microscopy and phase contrast microscopy) was analyzed with kappa statistics. Similarly, agreement between methods for confirming AKI (serum creatinine and urine microscopy scoring system) was assessed with kappa statistics. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), diagnostic accuracy, etc. were summarized as percentages with 95% confidence interval (CI). Association of clinical and laboratory markers with the binary presence/absence of AKI were assessed by stepwise multivariate logistic regression. Odds ratio and CI were calculated with 95% confidence. Variables having a p-value <0.05 in univariate analysis were considered for multivariate logistic regression. The sensitivity and specificity of NLR in predicting AKI in patients with UTI were obtained from receiver operating characteristic (ROC) curve. All the statistical analysis was performed by IBM SPSS Statistics Ver. 20.0 (IBM SPSS Inc., Chicago, IL, USA) and Open-Epi version 3.01 Software (Open source, MIT, Atlanta, GA, USA).

3. Results

3.1. Age and gender

Seventy two consecutive study participants were enrolled in the study. Among 72 study participants, 45 had UTI with AKI and their mean (SD) age was 53.4 (SD: 14.8) years. In this group, 58% were male and 42% were female. Mean (SD)

| Table 1 | RTE cell-granular cast scoring systema. |
|---------|----------------------------------------|
| Granular casts (per LPF) | RTE cells (per HPF) | 0 (0 point) | 1–5 (1 point) | ≥6 (2 points) |
| 0 (0 point) | 0 | 1 | 2 |
| 1–5 (1 point) | 1 | 2 | 3 |
| ≥6 (2 points) | 2 | 3 | 4 |
| HPF, high power field; LPF, low power field; RTE, renal tubular epithelial. | a Adapted from Perazella et al. [14]. |
age of 27 UTI patients in the non-AKI group was 52.2 (SD: 12.4) years, with 41% male and 59% female patients. There was no statistical significance in the age (p=0.71) and gender (p=0.16) of patient in two groups (Table 2).

3.2. Serum creatinine

Patients were followed for 5 days of hospital admission and their serial rises in creatinine were noted and analyzed statistically by using independent t-test. In the study group with AKI, follow-up data of all 45 patients could not be collected as they had left the hospital against medical advice before complete recovery. Hence, the follow-up data on Days 3 and 5 were available only for 36 patients on Day 3 and 35 patients on Day 5 in the AKI group. Similarly in the non-AKI group, of the 27 patients Day 3 follow-up data were available for 21 patients and Day 5 data were available for 17 patients. As expected the p-value shows the higher significance (p<0.001) of rise in creatinine level during AKI (Table 3). No patient developed septicaemia and intensive care was not required.

3.3. Urine microscopy score

RTE cell-granular cast score shows greater significance (p = 0.008) for predicting AKI in patients with UTI. A microscopy score greater than or equal to 2 (≥2) is suggestive of AKI (Table 4). Out of the 45 patients in group 1, AKI was identified in 31 (68.9%) by RTE-granular cast scoring system. Among the 27 (37.5%) patients in UTI group without AKI, the RTE-granular cast scoring system identified 26 (96.3%) as not having AKI. Kappa statistics across the binary variables in the test group was found to be 0.59, which indicates that there was a moderate agreement between RTE-granular cast scoring system and serum creatinine. RTE-granular cast scoring system has a sensitivity of 68.9% (95% CI: 54.3−80.5) and specificity of 96.3% (95% CI: 81.7−99.3). PPV and NPV were 96.9% (95% CI: 84.3−99.5) and 65% (95% CI: 49.5−77.9) respectively, for the prediction of AKI in patients with UTI. The score has a diagnostic accuracy of 79.2% (95% CI: 68.4−86.9). The likelihood ratio of positive test was 18.6 (95% CI: 2.5−135.9).

3.4. Serum and urine KIM-1

Both serum and urine KIM-1 showed a normal distribution of data. However, both serum and urine levels of KIM-1 could not distinguish patients with and without AKI and results were not statistically significant (Table 5). The p-value for serum and urine KIM-1 were 0.85 and 0.75, respectively. ELISA could not be done in three serum samples due to visible lipemia after storage and thawing of the sample. Since lipemic, icteric and haemolysed samples affect the optical density and test values, these samples were not processed and considered for analysis.

3.5. NLR

NLR on first day of haemogram sample, predicted AKI in patients with UTI. NLR was expressed as median (inter-quartile range). Median (inter-quartile range) for NLR in AKI group was 7.2 (4.1−10.8) and in the non-AKI group, it was 3.2 (2.3−6.1). NLR was found to significantly predict AKI (p = 0.005). ROC curve was designed for NLR to identify sensitivity and specificity in prediction of AKI (Fig. 1). AUC to predict AKI using NLR was 0.704, with 95% confidence interval of 0.574−0.835. The sensitivity and the specificity were 72.1% and 65.9% respectively using cut-off of 4.2 for NLR to predict AKI. If the NLR is considering as a screening tool, then 3.4 can be taken as the cut-off with a high sensitivity (Table 6). NLR with a cut-off with 4.2 is considered in this

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### Table 2

Basic demographic data of patients in study group.

| Characteristics         | AKI (n=45)          | No AKI (n=27)          | p-Value |
|-------------------------|---------------------|------------------------|---------|
| Age, year, mean±SD      | 53.4±14.8           | 52.2±12.4              | 0.71    |
| Male sex, n (%)         | 26 (58)             | 11 (41)                | 0.16    |
| Female sex, n (%)       | 19 (42)             | 16 (59)                | 0.16    |
| ANC, median (IQR for ANC) | 8900 (7400−12 180) | 7350 (5825−10 140)     | 0.14    |
| ALC, median (IQR for ALC) | 1400 (1100−2005)   | 1820 (1520−2430)       | 0.04    |
| Diabetes, n (%)         | 22 (48.9)           | 14 (51.9)              | 0.81    |
| Hypertension, n (%)     | 6 (13.3)            | 5 (18.5)               | 0.55    |

AKI, acute kidney injury; ANC, absolute neutrophil count; ALC, absolute lymphocyte count; IQR, inter-quartile range; SD, standard deviation.

### Table 3

Significance of rise in creatinine in AKI.

| Creatinine Mean±SD | No AKI (n=27) | p-Value |
|-------------------|---------------|---------|
| Day 1             | 3.8±3.1       | 1.14±0.5 | <0.001 |
| Day 3             | 3.22±2.4      | 1.10±0.04| <0.001 |
| Day 5             | 2.83±1.2      | 1.18±0.4 | 0.001  |

AKI, acute kidney injury; SD, standard deviation.

### Table 4

RTE cell-granular cast score (Perazella scoring system).

| RTE-granular cast score | AKI, n (%) | No AKI, n (%) | p-Value |
|-------------------------|------------|--------------|---------|
| <2                      | 54 (100)   | 29 (53.7)    | 25 (46.3) | 0.008    |
| ≥2                      | 18 (100)   | 16 (88.9)    | 2 (11.1)  |          |

AKI, acute kidney injury; RTE, renal tubular epithelial.

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study for the prediction of AKI in patients with UTI. NLR shows statistically significant association with AKI \( (p = 0.003) \) with a relative risk of 1.77. A total of 45 (62.5%) UTI patients were diagnosed with AKI using serum creatinine levels as per KDIGO 2012 guidelines. Out of the 45 patients in group I, AKI was identified in 31 (68.9%) by NLR. Among the 27 (37.5%) patients in the non-AKI group, the NLR could independently identify 18 (66.6%) as not having AKI (Table 7).

### 4. Discussion

The prevalence of UTI in India is increasing due to higher incidence of co-existing morbidities such as diabetes mellitus, urolithiasis and iatrogenic causes such as catheterization \[19,20\]. Infection in the lower urinary tract ascends to the kidney parenchyma directly or through haematogenous route causing AKI. In some instances, AKI may be caused indirectly due to reflux of urine from lower urinary tract. AKI assessment is delayed due to high residual functional capacity of kidney and non-specific symptomatology thereby leading to delay in the diagnosis. This can result in permanent residual scarring of the kidney. Hence we attempted to identify simple diagnostic tools to predict AKI in the patients with UTI. Urine microscopy is a simple but often neglected tool which if performed sincerely can be useful in predicting renal dysfunction. In our study, we adopted the scoring system of Perazella et al. \[14\] who advocated the role of renal tubular epithelial cell-granular cast in prediction of AKI.

In our study, from urinalysis we found that RTE-granular cast score could predict AKI in patients with UTI. In univariate analysis, the \( p \)-values of RTE-granular casts score was 0.008. Subsequently these parameters were studied for agreement statistics using serum creatinine as the gold standard for predicting AKI. Renal tubular epithelial cell-granular cast (RTE-granular cast) scoring system showed moderate agreement with serum creatinine levels with a Kappa score of 0.59 to predict AKI in patients with UTI. In our study, the sensitivity and specificity of RTE-granular cast score in predicting AKI was 68.9% and 96.3%, respectively. Positive predictive value was 96.9% while the negative predictive value was 65% in the prediction of AKI in patients with UTI. The results were partially similar to that of Perazella et al. \[14\] who had a sensitivity of 73% and specificity of 75%. In our study, the specificity of RTE-granular cast score was much higher in predicting AKI in patients with UTI. Positive predictive value in our study was 96.9% in comparison with 100% in the study by Perazella et al. \[14\], while the negative predictive value in our study was much lesser, constituting 65% in comparison with 91% in the study by Perazella et al. \[14\]. The difference is most likely due to the variation in sample size. The utility of RTE-granular cast in predicting renal tubular injury and sepsis induced AKI is also described in other studies from Lakhmir Chawla et al. \[21\] and several other authors \[8,14,15\].

In our study, we did not find significant association with either urinary or serum levels of KIM-1 molecule with renal dysfunction. All the patients in our study group with AKI had significant recovery of serum creatinine and renal function within 5 days after diagnosis and administration of appropriate treatment. Thus our results are different from the studies from Vaidya et al. \[12\] and several other authors \[22–26\]. However, Petrovic et al. \[27\] have highlighted that urine and serum KIM-1 levels are not predictive of AKI. KIM-
tubular necrosis as renal biopsies were not assessed in our study. However, in our study, we could not validate the prognostic aspect due to lack of long-term follow-up. However, within the duration of the first 5 days of treatment and monitoring of our patients, we found that all of them had significant recovery of serum creatinine and renal function. Thus, it is possible that in our study, urine and serum KIM-1 levels did not have any prognostic significance as we did not have poor outcome patients, with the limitation of smaller sample size and lack of long-term follow-up.

In our study, NLR was a significant and an independent predictor of AKI. NLR had a cut-off value of 4.2 with an area under the curve of 0.704 (95% CI: 0.574–0.835) having the maximum sensitivity and specificity of 72.1 and 65.9 respectively to predict AKI in patients with UTI. Our results on the value of NLR were similar to the results of Yilmaz et al. [17]. However, we did not find any other reference on the value of absolute lymphocyte count having a predictive value on AKI. In Pearson correlation analysis, there was moderate correlation ($r = 0.482$, $p = 0.01$) between NLR and serum creatinine in non-AKI group, but there is a high correlation ($r = 0.678$, $p < 0.001$) between NLR and serum creatinine in AKI group.

Our study has validated the significance of a systematic scoring system for urine microscopy with the RTE-granular cast score developed by Perazella et al. [14], having independent diagnostic and predictive value in the detection of AKI in patients with UTI. Our study also highlights the significance of NLR with a cut-off value of 4.2, which is a novel tool in prediction of AKI.

The limitation of the study is the lack of long-term follow-up, which precludes us from assessing the prognostic value of serum and urine KIM-1 levels in our subset of patients. Another limitation is the relatively smaller sample size, but it is also due to rigid inclusion and exclusion criteria.

RTE-granular cast score with a cut-off of two and NLR are potentially useful and inexpensive tools in the diagnosis of AKI in patients with UTI. The findings in our study, if analyzed and validated in a larger cohort, shall probably be helpful in detection of AKI in remote and under-privileged settings and in developing countries where expensive resources may not be readily available.

Prediction of AKI is a critical requirement not only in the setting of UTI, but also in several patients with both neoplastic and non-neoplastic conditions. Acute tubular necrosis is an important and common cause of AKI. We have not studied the diagnostic role of urine microscopic scoring systems, serum and urine KIM-1 levels as well as NLR in a setting of acute tubular necrosis as renal biopsies were not assessed in our study. Another major potential application for prediction of AKI in immediate and long-term outcome will be in patients who undergo nephrectomy for malignancies [28]. Urine microscopy scoring systems, NLR as well as serum and urine KIM-1 levels may be extremely useful and it needs to be studied and validated in different clinical settings both in neoplastic and non-neoplastic conditions [29–31].

5. Conclusion

The study validates the RTE-granular cast score as an independent predictor and diagnostic tool in the detection of AKI in patients with UTI. Urine and serum KIM-1 did not have any role in the diagnosis or prediction of AKI in a setting of UTI. The prognostic value of the same could not be assessed in our study, due to the lack of long-term follow-up in our patients. NLR was found to be a novel and independent predictor of AKI in patients with UTI.

Author contributions

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Conflicts of interest

The authors declare no conflict of interest.

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References

[1] Gonzalez CM, Schaeffer AJ. Treatment of urinary tract infection: what’s old, what’s new, and what works. World J Urol 1999;17:372–82.
[2] Drekonja DM, Johnson JR. Urinary tract infections. Prim Care 2008;35:345–67.
[3] Mahon CR, Lehman DC, Manuselis G. Textbook of diagnostic microbiology. 5th ed. Saunders; Elsevier; 2015. p. 887.
[4] Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL. Harrison’s manual of medicine. 16th ed. New York: McGraw-Hill Medical; 2005. p. 724–8.
[5] Gupta K, Trautner BW. Urinary tract infections, pyelonephritis, and prostatitis. In: Fauci A, Braunwald E, Kasper D, Hauser S, Longo D, Jameson J, et al., editors. Harrison’s principles of internal medicine. 17th ed. New York: McGraw-Hill Publishers; 2001. p. 1820–7.
[6] Hsiao CY, Yang HY, Hsiao MC, Hung PH, Wang MC. Risk factors for development of acute kidney injury in patients with urinary tract infection. PLoS One 2015;10:e0133835. https://doi.org/10.1371/journal.pone.0133835.
[7] Schiff H, Lang SM. Update on biomarkers of acute kidney injury: moving closer to clinical impact? Mol Diagn Ther 2012;16:199–207.
[8] Bagshaw SM, Haase M, Haase-Fielitz A, Bennett M, Devarajan P, Bellomo R. A prospective evaluation of urine microscopy in septic and non-septic acute kidney injury. Nephrol Dial Transplant 2012;27:582–8.
[9] Lines S, Lewington A. Acute kidney injury. Clin Med 2009;9:273–7.
[10] Kidney Disease Improving Global Outcomes AKI Guideline Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012;2:1–138.
[11] Waikar SS, Liu KD, Chertow GM. Diagnosis, epidemiology and outcomes of acute kidney injury. Clin J Am Soc Nephrol 2008;3:844–61.
[12] Vaidya VS, Waikar SS, Ferguson MA, Collings FB, Sunderland K, Gioules C, et al. Urinary biomarkers for sensitive and specific detection of acute kidney injury in humans. Clin Transl Sci 2008;1:200–8.
[13] Safdar OY, Shalaby M. Neutrophil gelatinase-associated lipocalin as an early marker for the diagnosis of urinary tract infections in Saudi children. J Nephrol Therapeut 2015;5:6–9.
[14] Perazzella MA, Coca SG, Kanbay M, Brewster UC, Parikh CR. Diagnostic value of urine microscopy for differential diagnosis of acute kidney injury in hospitalized patients. Clin J Am Soc Nephrol 2008;3:1615–9.
[15] Perazzella MA. The urine sediment as a biomarker of kidney disease. Am J Kidney Dis 2015;66:748–55.
[16] Han SY, Lee IR, Park SJ, Kim JH, Shin JI. Usefulness of neutrophil-lymphocyte ratio in young children with febrile urinary tract infection. Korean J Pediatr 2016;59:139–44.
[17] Yilmaz H, Cakmak M, Inan O, Darcin T, Akcay A. Can neutrophil-lymphocyte ratio be independent risk factor for predicting acute kidney injury in patients with severe sepsis? Ren Fail 2015;37:225–9.
[18] Baerheim A, Albrektsen G, Eriksen AG, Laerum E, Sandberg S. Quantification of pyuria by two methods correlation and interobserver agreement. Scand J Prim Health Care 1989;7:83–6.
[19] Viswanathan V, Janifer J, Geethalakshmi S, Satyavani K. Prevalence of lower urinary tract infection in South Indian type 2 diabetic subjects. Indian J Nephrol 2009;19:107–11.
[20] Sangamithra V, Sneka P, Praveen S, Manonmoney J. Incidence of catheter associated urinary tract infection in medical ICU in a tertiary care hospital. Int J Curr Microbiol Appl Sci 2017;6:662–9.
[21] Chawla LS, Dommu A, Berger A, Shih S, Patel SS. Urinary sediment cast scoring index for acute kidney injury: a pilot study. Nephron Clin Pract 2008;110:145–50.
[22] Han WK, Bailly V, Abichandani R, Thadhani R, Bonventre JV. Kidney injury molecule-1 (KIM-1): a novel biomarker for human renal proximal tubule injury. Kidney Int 2002;62:237–44.
[23] Vaidya VS, Ramirez V, Ichimura T, Bobadilla NA. Bonventre JV. Urinary kidney injury molecule-1: a sensitive quantitative biomarker for early detection of kidney tubular injury. Am J Physiol Ren Physiol 2006;290:517–29.
[24] Lee HE, Lee SH, Baek M, Choi H, Park K. Urinary measurement of neutrophil gelatinase associated lipocalin and kidney injury molecule-1 helps diagnose acute pyelonephritis in a preclinical model. J Biomark 2013;6:1–6.
[25] Xie Y, Wang Q, Wang C, Che X, Shao X, Xu Y, et al. Association between the levels of urine kidney injury molecule-1 and the progression of acute kidney injury in the elderly. PloS One 2017;12:1–12.
[26] Bagshaw SM, Bellomo R. Early diagnosis of acute kidney injury. Curr Opin Crit Care 2007;13:638–44.
[27] Petrovic S, Bogavac-Stanojevic N, Peco-Antic A, Ivanisevic I, Kotur-Stevuljevic J, Paripovic D, et al. Clinical application neutrophil gelatinase-associated lipocalin and kidney injury molecule-1 as indicators of inflammation persistence and acute kidney injury in children with urinary tract infection. Biomed Res Int 2013:947157. https://doi.org/10.1155/2013/947157.
[28] Martini A, Sfakianos JP, Paulucci DJ, Abaza R, Eun DD, Bhandari A, et al. Predicting acute kidney injury after robot-assisted partial nephrectomy: implications for patient selection and postoperative management. Urol Oncol Semin Orig Invest 2019;37:445–51.
[29] Bravi CA, Vertosick E, Benfante N, Tin A, Sjoberg D, Hakimi AA, et al. Impact of acute kidney injury and its duration on long-term renal function after partial nephrectomy. Eur Urol 2019;76:398–403.
[30] Martini A, Cumarasamy S, Hemal AK, Badani KK. Renal cell carcinoma: the oncological outcome is not the only endpoint. Transl Androl Urol 2019;8:593–5.
[31] Martini A, Cumarasamy S, Bek sac AT, Abaza R, Eun DD, Bhandari A, et al. A nomogram to predict significant estimated glomerular filtration rate reduction after robotic partial nephrectomy. Eur Urol 2018;74:833–9.