# Diagnostic accuracy of procalcitonin and presepsin for infectious disease in patients with acute kidney injury

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Running title: Procalcitonin and presepsin for infection diagnosis in AKI

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***Abstract***

*Objectives:* Procalcitonin (PCT) and presepsin (PSEP) are markers of sepsis, but their diagnostic accuracy may be compromised in acute kidney injury (AKI). This study evaluated their diagnostic accuracy in patients with and without AKI.

*Methods:* This retrospective study included 91 patients with at least one of the systematic inflammatory response syndrome criteria. The AKI markers, plasma neutrophil gelatinase-associated lipocalin (NGAL), plasma cystatin C (CysC), and estimated glomerular filtration rate (eGFR) were measured at admission and on days 1, 3, 5, and 7. Patients were divided into non-AKI and AKI groups. APACHE II severity scores were determined

*Results:* PCT and PSEP levels were significantly increased in both non-AKI and AKI patients with infection ($p < 0.01$). NGAL, CysC, and eGFR in patients with infection were associated with PCT, PSEP, and APACHE II score, and the levels of PCT and PSEP were also significantly ($p < 0.001$) correlated with severity. AKI markers also correlated with severity. The diagnostic accuracies of PCT and PSEP in patients with AKI were slightly higher than in non-AKI patients, and cut-off values were increased.

*Conclusions:* PCT and PSEP are useful markers of bacterial infections in AKI but different thresholds should be applied in these patients.

***KEYWORDS:*** Acute kidney injury; Biomarker; Diagnosis; Infection; Presepsin; Procalcitonin

***Introduction***

Procalcitonin (PCT) is a precursor of the peptide hormone calcitonin, and has a molecular weight of approximately 13 kD.[1] PCT, interleukin-6 (IL-6), and tumor necrosis factor-α have been used as diagnostic markers for sepsis. PCT, in particular, has been reported to be superior to endotoxin, β-D-glucan, IL-6, and C-reactive protein (CRP) for differentiating between bacterial infections such as sepsis, and non-bacterial infections.[2] However, PCT is known to be increased in non-infectious systemic inflammatory response syndrome (SIRS).[3] Yet, the cluster of differentiation 14 (CD14) is a glycoprotein expressed on the membrane surface of macrophages and/or monocytes and serves as a receptor complex for lipopolysaccharides and signal transduction via Toll-like receptor 4.[4] CD14 is divided into two types of soluble isoforms (49 and 55 kD),[5] and we previously reported that the levels of soluble CD14 (55 kD) were elevated in patients with multiple organ failure.[6] Presepsin (soluble CD14-ST, PSEP) has been identified as a 13-kD truncated N-terminal fragment of CD14 produced by stimuli such as phagocytosis in response to bacterial infection.[7] Several studies have confirmed the usefulness of PSEP as a marker for the diagnosis of sepsis.[8–10] However, it has been recently reported that the diagnostic accuracies of PCT and PSEP in patients with acute kidney injury (AKI) were lower than in patients without AKI because both PCT and PSEP are thought to be eliminated through the kidneys and/or liver, and it was suggested that PCT and PSEP may not be reliable indicators of sepsis in patients with more advanced AKI.[11,12] Amour et al.[13] reported that after major aortic surgery, the accuracy of PCT was not significantly different between groups with or without renal dysfunction, but that the optimal cut-off value was significantly different (non-renal dysfunction 0.81 ng/mL vs renal dysfunction 2.57 ng/mL, $p < 0.05$). Thus, if the diagnostic accuracies of PCT and PSEP in patients with AKI are not lower than in patients without AKI, it would be useful for daily clinical practice to determine the optimal cut-off values for PCT and PSEP in sepsis patients with AKI. Previous stuidies[11,12] were retrospective, single center studies using only the RIFLE (risk, injury, failure, loss of kidney function, and end-stage kidney disease) criteria, and markers of AKI such as plasma neutrophil gelatinase-associated lipocalin (NGAL), plasma cystatin C (CysC), and estimated glomerular filtration rate (eGFR) were not investigated. In the present

study, therefore, patients attending the emergency room in two medical institutions, and who fulfilled at least one of the systematic inflammatory response syndrome (SIRS) criteria, were divided into non-AKI and AKI groups using these three markers of AKI. Plasma levels of PCT and PSEP were measured to evaluate their diagnostic accuracy for infection in patients with AKI.

## *Methods*

### Patients

Blood samples were collected from patients admitted to the emergency rooms at Iwate Medical University Hospital, and Kochi Health Sciences Center between June 2010 and June 2011 who had at least one of the SIRS criteria (body temperature > 38°C or < 36°C, heart rate > 90 beats per minute, respiratory rate > 20 breaths per minute or $PaCO_2$ < 32 mm Hg, abnormal white blood cell count).[14] Informed consent was obtained from all 91 patients enrolled, in accordance with the guidelines of each institution. PCT, PSEP, NGAL, CysC, and creatinine (Cr) levels were measured in blood specimens collected upon admission (on arrival; day 0) and on days 1, 3, 5, and 7 (at 07:00 am). The blood specimens were retrospectively categorized into six groups according to the confirmed diagnosis of each patient (SIRS, non-SIRS, infection without SIRS, sepsis, severe sepsis, and septic shock).[15,16] The diagnoses of SIRS, sepsis, severe sepsis, and septic shock were made according to the seven criteria set by the American College of Chest Physicians/Society of Critical Care Medicine.[14] Non-SIRS was defined as patients without infection and a SIRS score < 2, while infection without SIRS was defined as patients with infection but a SIRS score < 2. The Acute Physiology and Chronic Health Evaluation II (APACHE II) score was used as an index of the severity of disease,[17] and the SOFA (Sepsis-related Organ Failure Assessment) score was calculated as an index of the severity of organ dysfunction.[18] The definitive diagnoses were deliberated and decided by two infection control doctors certified by the Japanese College of Infection Control Doctors and the clinical research coordinator. The exclusion criteria were: age under 13 years, undergoing chronic hemodialysis, or experiencing

cardiopulmonary arrest on arrival. Blood collection was suspended when patients recovered with a SIRS score <1 or died.

**Measurements**

PSEP concentrations were measured using a compact automated immunoanalyzer, PATHFAST, based on a chemiluminescent enzyme immunoassay (Mitsubishi Chemical Medience Co., Tokyo, Japan). Briefly, whole blood was collected into a conventional blood collection tube (Terumo Co., Tokyo, Japan) containing EDTA-2K as an anticoagulant, and the sample was assayed within 4 h after collection using the PATHFAST PSEP assay. PCT concentrations were measured using an Elecsys BRAHMS PCT assay (Roche Diagnostics, Tokyo, Japan). NGAL concentrations were measured using an NGAL Rapid ELISA Kit (human) from Bioport (Denmark). Cr concentrations were measured using Iatoro LQ CRE(A) II (LSI Medience Corp., Tokyo, Japan). CysC concentrations were measured using Iatoro Cys-C (LSI Medience). IL-6 concentrations were measured using an Immulyze 2000 assay system (Siemens Healthcare Diagnostics K.K., Tokyo, Japan). CRP concentrations were measured using a CRP-LATEX(II)X2 assay kit (Denka Seiken Co., Tokyo, Japan). EDTA-treated plasma was used for control samples.

**Classification of AKI**

NGAL is expressed in a variety of human tissues, and has a molecular weight of 25 kDa. Because the 25 kDa monomeric NGAL form is secreted by injured kidney tubule epithelial cells, NGAL is reported to be a useful marker for AKI.[19] We applied the cutoff value of 150 ng/mL[20–22] to discriminate between non-AKI and AKI. It has been reported that the diagnostic accuracy of AKI (area under the concentration curve [AUC]) for NGAL is 0.78–0.82.[20–22] CysC is a 13 kD cysteine protease inhibitor that is freely filtered through the glomerular membrane and is completely reabsorbed and metabolized by proximal tubular cells without secretion. pCysC has been reported to be an early predictor of AKI.[23,24] A cut-off value of 0.98 mg/L[24] was used to

discriminate between non-AKI and AKI, and it has been reported that the diagnostic accuracy of AKI (AUC) is 0.87.[25] A low eGFR is also a predictor of AKI,[26,27] and eGFR < 60 mL/min/1.73 $m^2$ has been reported to be a risk factor for AKI,[28,29;] therefore this value was used as a cut-off to discriminate between non-AKI and AKI. eGFR was calculated using equations for Japanese patients as follows:[30]

Males: eGFR (mL/min/1.73 $m^2$) = 194 × (serum Cr)$^{-1.094}$ × (Age)$^{-0.287}$

Females: eGFR (mL/min/1.73 $m^2$) = 194 × (serum Cr)$^{-1.094}$ × (Age)$^{-0.287}$ × 0.739

**Statistical analysis**

Comparisons between two groups were made using the Mann–Whitney *U* test. The Spearman rank order correlation coefficient was employed for the analysis of correlations. We used the cutoff value obtained by receiver operating characteristic (ROC) analysis with the Youden index. A *p*-value < 0.05 was considered statistically significant. Two-group comparisons, correlations, and multiple liner regression analyses were performed using JMP software (SAS Institute, Cary, NC, USA). ROC analysis was performed using Dr. SPSS II software (SPSS, Chicago, IL, USA). Two AUC comparisons were performed using EZR (Saitama Medical Center, Jichi Medical University, Saitama, Japan).

**Ethics**

Ethical approval for this study (Ethical Committee No. H24-42) was provided by the Ethical Committee of Iwate Medical University, Morioka, Japan (Chairperson: Prof. K. Suzuki) on 6 May 2010.

***Results***

**Patient characteristics**

Ninety-one patients, including 58 men (mean age, 68.28 years) and 33 women (mean age, 73.93 years) were enrolled in this study. The confirmed diagnoses were as follows: gastroenterological disease (*n* = 20); respiratory disease (*n* = 11); trauma (*n* = 12); circulatory disease (*n* = 3); renal disease (*n* = 8); burns (*n* = 8); cerebral hemorrhage (*n* = 2); pancreatitis (*n* = 1); hepatobiliary disease (*n* = 9); cellulitis/phlegmon (*n* = 6); drug poisoning (*n* = 1); and others (*n* = 10). A total of 403 blood specimens were collected at different time points and categorized into six groups according to patient condition (non-SIRS, SIRS, infection without SIRS, sepsis, severe sepsis, and septic shock). The characteristics and diseases of the patients are shown in Table 1. Of the 403 blood specimens, 174 indicated a diagnosis of non-infection (non-SIRS and SIRS) and 229 samples indicated a diagnosis of infection (infection without SIRS, sepsis, severe sepsis, and septic shock).

**Comparison of median PCT and PSEP levels between patients without and with infection in AKI patients**

Patients were divided into non-AKI and AKI groups according to the three AKI makers, Using NGAL, there were 171 samples classified as non-AKI (<150 ng/mL), 232 as AKI (≥ 150 ng/mL). Using CysC, there were 196 samples classified as non-AKI (< 0.98 mg/L) and 207 as AKI (≥ 0.98 mg/L). There were 225 samples from patients with eGFR ≥ 60 mL/min/1.73 $m^2$, and 178 samples from patents with < 60 mL/min/1.73 $m^2$. Both PCT and PSEP levels in patients with infection were significantly ($p < 0.01$) higher than in non-infectious patients in both non-AKI and AKI groups (Fig. 1).

**AUC and cut-off values of PCT and PSEP for diagnosing infection in non-AKI and AKI patients**

Using NGAL, CysC, and eGFR for AKI classification, the AUCs of PCT and PSEP for diagnosing infection were slightly higher in AKI patients than in non-AKI patients (Table 2). AUCs of PSEP

were higher than those of PCT in both non-AKI and AKI groups and some differences were significant (NGAL: AKI PSEP 0.83 vs PCT 0.72, $p < 0.05$; CysC: non-AKI PSEP 0.77 vs PCT 0.67, $p < 0.01$; eGFR: non-AKI PSEP 0.79 vs PCT 0.69, $p < 0.05$). The cut-off values of both PCT and PSEP were increased in AKI patients (Table 2).

**Multiple logistic regression analysis of AKI markers in infection**

The levels of PCT and PSEP were markedly higher in patients with infection and AKI (Fig. 1). Therefore, using logistic regression analysis with stratification by AKI severity scores, we investigated whether the three AKI makers were independently associated with markers of infection, including PCT, PSEP, IL-6, and CRP (Table 3). NGAL was significantly associated with PCT ($p = 0.03$) and PSEP ($p = 0.003$) in patients with APACHE II ≥ 14. CysC and eGFR were significantly associated with only PSEP in patients with APACHE II ≥ 14 ($p = 0.002$, 0.002, respectively). However, NGAL, CysC, and eGFR were not significantly associated with PCT nor PSEP in patients with APACHE II < 14.

**Multiple linear regression analysis of AKI makers**

To confirm the results of the multiple logistic regression analysis (Table 3), multiple linear regression analysis was performed to determine whether the three AKI makers were associated with the infection markers PCT and PSEP, or the severity score. The standardized β coefficients and *p*-values are presented in Table 4. NGAL, CysC, and eGFR were each significantly associated with PCT, PSEP, and APACHE II score. Then, to investigate confounding factors, we analyzed the correlation between the levels of PCT or PSEP and APACHE II score. Both PCT and PSEP were also significantly correlated with APACHE II score ($p < 0.001$; $r = 0.42$, and $p < 0.001$; $r = 0.48$, respectively).

***Discussion***

This study revealed five main findings: 1) the levels of PCT and PSEP in patients with infection were significantly higher than in patients without infection in both non-AKI and AKI groups; 2) the diagnostic accuracies of PCT and PSEP in patients with AKI were not lower than in non-AKI patients and were indeed slightly higher than in non-AKI patients, and AUCs of PSEP were higher than that of PCT; 3) the cut-off values of both PCT and PSEP were increased in patients with AKI; 4) the AKI markers were not independently associated with PCT and PSEP in infectious patients, and depended on severity; 5) the AKI markers NGAL, CysC, and eGFR in patients with infection were significantly associated with PCT, PSEP, and APACHE II severity score, and the levels of PCT and PSEP were also significantly correlated with APACHE II score. Therefore, higher levels of PCT and PSEP in patients with AKI may, at least in part, be associated with disease severity.

It was reported that the diagnostic accuracy of PSEP in patients with AKI was slightly but not significantly lower than in patients without AKI (AKI 0.698 vs. non-AKI 0.784), and they suggested that the kidney is the main organ responsible for cleansing blood of PSEP because there was a positive correlation between PSEP and serum Cr, and the significant negative correlation between PSEP and eGFR was similar in non-sepsis and sepsis groups.[12] Therefore, we suggest the lower AUC in AKI may be responsible for the lack of a difference in PSEP levels between non-sepsis and sepsis groups in their study. In contrast to that report, our study found that PSEP levels in patients with infection and AKI were significantly higher than in patients without infection, and that the AUC of PSEP in AKI was higher than in non-AKI patients (Table 4). The higher AUC in AKI may be related to the enhancement of the PSEP levels in the infection group. This enhancement may be related to disease severity, and this hypothesis was supported by logistic regression analysis and multiple linier regression analysis, which indicated that the AKI markers were not independently associated with PSEP in infectious patients, but depended on the severity of illness. Meanwhile, there is little increase in PSEP levels in patients without infection even if they are severely ill, as these are primarily markers of infection.[8, 16] Indeed, the ratio of PSEP between non-infection and infection groups with AKI classified by NGAL was 2.21 compared with 2.02 in non-AKI patients (Fig. 1).

Amour et al.[13] reported that the AUC of PCT for infection in patients with renal dysfunction was higher than in patients without renal dysfunction (0.80 vs 0.70) in line with our view. In contrast, in a study by Nakamura et al.,[12] no differences in PSEP levels were found between non-sepsis and sepsis in *Failure* group, Nakamura et al. also reported that PCT levels in sepsis patients were significantly higher than in non-sepsis patients.[11] However, the AUC of PCT for diagnosis of sepsis in *kidney failure* patients was significantly lower than that in non-AKI patients (*kidney failure* 0.857 vs. non-AKI 0.958).[11] The reported AUC of PCT in non-AKI patients was 0.958,[11] and might be a little higher than that found in our study (0.67–0.79) and elsewhere (0.70[13]). Although *kidney failure* represents a greater degree of AKI than *kidney injury*, the median value of PCT in sepsis patients with *kidney failure* was reported to be lower than that in sepsis patients with *kidney injury* (11.64 vs. 17.10[11]). Further studies are necessary to confirm these findings.

It may be necessary to pay attention to patients receiving hemodialysis (HD) as well as patients with renal dysfunction. Recently, Nagata et al.[31] reported that the median PSEP level in non-infectious patients receiving HD was 1160.0 pg/mL, which was similar to values seen in patients with severe sepsis or septic shock. Meanwhile, PSEP levels in renal dysfunction without infection were inversely correlated with the measured GFR, and the median PSEP for GFR < 15 mL/min/1.73 $m^2$ in patients without HD was 251.0 (213.0–297.5) pg/mL, and less than the cut-off value for sepsis (500 pg/mL[32]). Therefore, mechanism for the increase in PSEP related to HD may be different from that related to renal dysfunction, and associated with activation of neutrophils and/or monocytes. It has been reported that release of PSEP occurred in monocytes, and activation was necessary to release PSEP from monocytes.[33] Therefore, the PSEP levels in patients receiving HD would be markedly higher than that in patients with renal dysfunction because HD activates monocytes and/or neutrophils. More studies are needed to investigate this further.

There are some limitations to our study. This study was a two-center retrospective study. Therefore, a multicenter prospective cohort study with a larger number of patients is required. Moreover, only Japanese patients were enrolled in this study, and studies in other populations will also be necessary.

In conclusion, higher levels of PCT and PSEP were found in infectious patients with AKI, but the increase in PCT and PSEP levels in AKI patients was at least, in part, related to the severity of infectious patients as well as renal dysfunction. The diagnostic accuracies of PCT and PSEP for infection in patients with AKI were not lower than in patients without AKI. Both PCT and PSEP are useful markers of bacterial infections in patients with AKI, but different thresholds should be applied in these patients.

***Conflict of interest***

The authors have no conflicts of interest to declare.

***Acknowledgements***

We sincerely thank Prof. Kazuyoshi Itai for help with the statistical analysis.

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**Figure Legend**

**Figure 1** Comparison of PCT and PSEP levels between non-infection and infection groups in non-AKI and AKI patients. AKI was classified by NGAL (A), CysC (B), and eGFR (C). The median values are indicated in the boxes. PCT, procalcitonin; PSEP, presepsin; AKI, acute kidney injury; NGAL, plasma neutrophil gelatinase-associated lipocalin; CysC, plasma cystatin C; eGFR, estimated glomerular filtration rate. * $p < 0.01$.

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Fig. 1

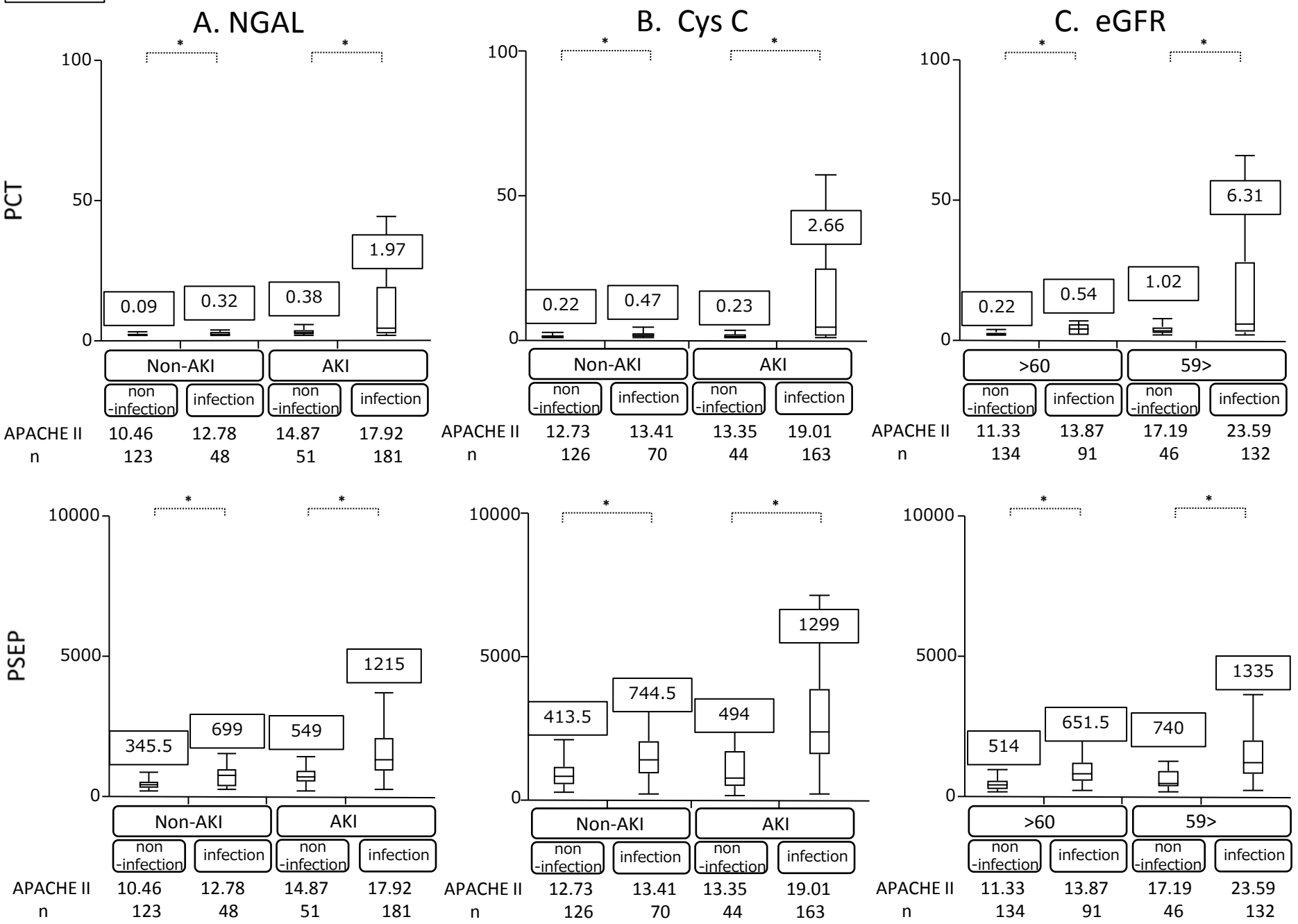
A. NGAL
B. Cys C
C. eGFR
PCT
PSEP
100
50
0
10000
5000
0
*
0.09
0.32
0.38
1.97
0.22
0.47
0.23
2.66
0.22
0.54
1.02
6.31
345.5
699
549
1215
413.5
744.5
494
1299
514
651.5
740
1335
Non-AKI
AKI
>60
59>
non -infection
infection
APACHE II
n
10.46
12.78
14.87
17.92
123
48
51
181
12.73
13.41
13.35
19.01
126
70
44
163
11.33
13.87
17.19
23.59
134
91
46
132

**Table 1** Patient characteristics

| | non-SIRS | SIRS | Infection without SIRS | sepsis | severe sepsis | septic shock | *p* |
|---|---|---|---|---|---|---|---|
| APACHE II | 11.68 | 14.01 | 15.5 | 17.13 | 23.08 | 25.0 | < 0.01 |
| SOFA | 2.29 | 4.06 | 5.14 | 5.37 | 8.16 | 9.28 | < 0.01 |
| Diagnosis | | | | | | | |
| GD | 18 | 5 | 21 | 24 | 6 | 12 | |
| Respiratory Disease | 9 | 0 | 18 | 21 | 3 | 0 | |
| Trauma | 25 | 22 | 1 | 3 | 0 | 0 | |
| CD | 2 | 6 | 0 | 7 | 0 | 0 | |
| Renal Disease | 9 | 0 | 9 | 13 | 9 | 0 | |
| Burns | 8 | 12 | 5 | 9 | 0 | 0 | |
| CH | 7 | 3 | 0 | 0 | 0 | 0 | |
| Pancreatitis | 0 | 2 | 0 | 0 | 0 | 0 | |
| HD | 6 | 0 | 12 | 16 | 3 | 0 | |
| CP | 4 | 0 | 11 | 10 | 2 | 2 | |
| Drug poisoning | 3 | 1 | 0 | 0 | 0 | 0 | |
| Others | 26 | 6 | 7 | 4 | 1 | 0 | |
| Total blood samples, *n* (%) | 117 (29.0%) | 57 (14.1%) | 84 (20.8%) | 107 (26.5%) | 24 (5.9%) | 14 (3.5%) | 403 |

Data are presented as medians. APACHE, Acute Physiology and Chronic Health Evaluation; SOFA, Sequential Organ Failure Assessment; GD, gastroenterological disease; CD, circulatory disease; CH, cerebral hemorrhage; MD, malignant disease; HD, hepatobiliary disease; CP, cellulitis phlegmon; SIRS, systemic inflammatory response syndrome.

**Table 2** Cut-off values of PCT and PSEP for diagnosing sepsis in non-AKI and AKI patients

| | | | AUC | Cut-off | Sensitivity | Specificity | Youden index |
|---|---|---|---|---|---|---|---|
| NGAL | Non-AKI | PCT | 0.67 | 0.85 | 0.68 | 0.58 | 0.21 |
| | | PSEP | 0.75 | 694 | 0.69 | 0.81 | 0.45 |
| | AKI | PCT | 0.72 | 2.01 | 0.57 | 0.81 | 0.25 |
| | | PSEP | 0.83* | 828 | 0.81 | 0.71 | 0.45 |
| CysC | Non-AKI | PCT | 0.67 | 0.85 | 0.42 | 0.83 | 0.47 |
| | | PSEP | 0.77** | 684 | 0.63 | 0.88 | 0.48 |
| | AKI | PCT | 0.82 | 0.94 | 0.69 | 0.79 | 0.30 |
| | | PSEP | 0.85 | 891 | 0.83 | 0.69 | 0.49 |
| eGFR | Non-AKI | PCT | 0.69 | 0.86 | 0.45 | 0.85 | 0.49 |
| | | PSEP | 0.79* | 694 | 0.66 | 0.87 | 0.50 |
| | AKI | PCT | 0.81 | 1.14 | 0.69 | 0.79 | 0.34 |
| | | PSEP | 0.84 | 891 | 0.86 | 0.62 | 0.45 |

AUC, area under curve. * $p < 0.05$ vs PCT, ** $p < 0.01$ vs PCT

**Table 3** Multiple logistic regression analysis of AKI markers in patients with infection

| | | APACHE II ≤ 14 | | APACHE II > 14 | |
|---|---|---|---|---|---|
| | | $\chi^2$ | *p* | $\chi^2$ | *p* |
| NGAL | IL-6 | 0.74 | 0.39 | 0.41 | 0.52 |
| | CRP | 1.65 | 0.19 | 3.06 | 0.08 |
| | PSEP | 0.68 | 0.41 | 8.82 | 0.003 |
| | PCT | 1.64 | 0.19 | 4.42 | 0.03 |
| | Age | 1.61 | 0.20 | 13.3 | 0.003 |
| | Sex | 4.6 | 0.03 | 1.38 | 0.24 |
| | SOFA | 0.07 | 0.79 | 0.39 | 0.53 |
| CysC | IL-6 | 1.77 | 0.18 | 1.32 | 0.25 |
| | CRP | 0 | 0.99 | 5.67 | 0.01 |
| | PSEP | 3.56 | 0.05 | 9.71 | 0.002 |
| | PCT | 0.4 | 0.52 | 0 | 0.99 |
| | Age | 6.23 | 0.01 | 5.78 | 0.02 |
| | Sex | 0.24 | 0.62 | 0.09 | 0.76 |
| | SOFA | 3.4 | 0.06 | 0.82 | 0.36 |
| eGFR | IL-6 | 2.16 | 0.14 | 0.04 | 0.84 |
| | CRP | 0.3 | 0.58 | 5.8 | 0.01 |
| | PSEP | 1.08 | 0.29 | 9.36 | 0.002 |
| | PCT | 0.02 | 0.88 | 2.7 | 0.1 |
| | Age | 9.25 | 0.002 | 13.15 | 0.0003 |
| | Sex | 0.13 | 0.72 | 2.89 | 0.08 |
| | SOFA | 1.02 | 0.31 | 0 | 0.95 |

IL-6, interleukin-6; CRP, C-reactive protein; SOFA, sequential organ failure assessment.

**Table 4** Multiple linear regression analysis of AKI markers

| | $r^2$ | | t | *p* | β |
|---|---|---|---|---|---|
| NGAL | 0.45 | PCT | 4.07 | < 0.001 | 0.223 |
| | | PSEP | 5.6 | < 0.001 | 0.302 |
| | | APACHE II | 7.02 | < 0.001 | 0.377 |
| Cysc | 0.36 | PCT | 3.28 | 0.0012 | 0.192 |
| | | PSEP | 4.92 | < 0.001 | 0.192 |
| | | APACHE II | 5.78 | < 0.001 | 0.334 |
| eGFR | 0.26 | PCT | -2.06 | 0.041 | -0.198 |
| | | PSEP | -2.54 | 0.011 | -0.158 |
| | | APACHE II | -6.03 | < 0.001 | -0.375 |