Risk factors of acute kidney injury after acute myocardial infarction

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ABSTRACT
Objectives: To study the risk factors for acute kidney injury (AKI) in-patients with acute myocardial infarction (AMI).
Methods: A total of 1371 cases of adult in-patients with AMI in the First People’s Hospital of Changzhou from January 2008 to December 2012 were retrospectively analyzed. Based on the occurrence of AKI diagnosed according to the 2012 KDIGO AKI criteria, they were divided into AKI group and non-AKI group and further into conservative treatment groups, coronary angiography (CAG) groups, and coronary artery bypass grafting (CABG) groups based on the timing of AKI occurrence, respectively. Related risk factors of AKI were analyzed by univariate and multivariate logistic regressions.
Results: 410 (29.9%) developed AKI. Patients with AKI had significantly increased in-hospital mortality than patients without AKI. Multivariate logistic regression analysis showed that decreased baseline eGFR, increased fasting plasma glucose (FPG), use of diuretics and Killip grade IV were independent risk factors of AKI, while increased DBP on admission was a protective factor for patients in conservative treatment group. Decreased baseline eGFR, increased FPG, use of diuretics, intraoperative hypotension and acute infection were independent risk factors of AKI for patients in the CAG group. Decreased baseline eGFR, increased FPG, use of diuretics, and low cardiac output syndrome after operation were independent risk factors of AKI for patients in the CABG group.
Conclusions: AKI is a common complication and associated with increased mortality after AMI. Decreased baseline renal function, increased FPG and use of diuretics were common independent risk factors of AKI after AMI.

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Introduction
Acute kidney injury (AKI) is acute kidney lesion due to various causes and used to replace the concept of acute renal failure by the International Kidney Disease and Emergency Medical Community in 2004.1 Acute myocardial infarction (AMI) is one of the common acute diseases in internal medicine department. Patients with AMI concomitant AKI often progress rapidly, leading to significantly higher mortality.2 Currently, the epidemiological data on the prevalence of inpatients with AMI concomitant AKI are rare and very few researches compared the prevalence of AKI in AMI patients after various treatments including conservative treatment, percutaneous coronary intervention, and coronary artery bypass grafting (CABG). In addition, The diagnostic criteria of AKI are not uniform. In this study, the criteria for AKI diagnosis and staging issued by Kidney Disease: Improving Global Outcomes (KDIGO) in 2012 were adapted and AMI patients admitted to our hospital were retrospectively analyzed for identification of risk factors for AKI, with the hope to provide a basis for effective early interventions and improving outcomes.

Subjects and methods

Study subjects
A total of 1655 in-patients diagnosed with AMI in the Third Affiliated Hospital of Soochow University (Changzhou First People’s Hospital) from 1 December 2008 to 31 December 2012 were screened using Kingstar Winning Medical Laboratory Information Management System. The inclusion criteria included: (1) being examined at least twice for renal function (Scr and Bun) during hospitalization; (2) with minimum Scr ≥ 40 μmol/L; and (3) age ≥ 18 years old. The exclusion criteria included: (1) patients who had chronic renal failure, need regular dialysis as well as with Scr ≥ 442 μmol/L at first admission; (2) patients who had undergone renal transplantation; (3) critically ill patients who died within 24 h after admission; (4) re-admitted

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patients; and (5) patients with missing or incomplete disease history.

**Study methods**

**Case collection**

Based on the occurrence timing of AKI, patients were assigned into AKI and non-AKI groups. They were further divided based on the timing of AKI occurrence into conservative treatment AKI and non-AKI groups, post-coronary angiography (CAG) AKI and non-AKI groups, as well as post-CABG AKI and non-AKI groups. Their clinical data were collected including: (1) general information including gender, age, admitted departments, days in ICU, complications, smoking history as well as systolic blood pressure (SBP) and diastolic blood pressure (DBP) on admission; (2) laboratories and auxiliary examinations, such as baseline Scr, peak Scr, fasting blood glucose (FBG), blood lipids, blood uric acid, hemoglobin, albumin, prealbumin, and left ventricular ejection fraction (LVEF); (3) treatment and prognosis including CAG (coronary stenting; intraoperative hypotension, systolic blood pressure 5 90 mmHg; post-operative hypotension, systolic blood pressure 5 90 mmHg), CABG (including the number of grafts, post-operative low cardiac output syndrome), drug intervention (ACEI/ARB, diuretics, anti-platelet aggregation drugs, lipid-lowering drugs, etc.), renal replacement therapy, and clinical outcome.

**AKI definition**

Acute kidney injury was defined according to the guidelines in the book entitled “Kidney Disease: Improving Global Outcomes” (KDIGO) issued in 2012.3 In detail patients were diagnosed to have AKI if they met one of the following criteria: (1) Scr increased by over 26.5 μmol/L (0.3 mg/dL) within 48 h; (2) Scr increased to over 1.5-fold of baseline-confirmed or presumed occurring within 7 d; (3) urine output 5 0.5 mL·h−1·kg−1 for more than 6 h. AKI staging was defined according to KDIGO guide for AKI staging. Baseline Scr was defined as the minimum Scr value measured during hospitalization. The baseline eGFR was calculated using the simplified MDRD equation: eGFR [mL·min−1·(1.73 m2)−1] = 186 × [(Scr−1.154) (mg/dL)] × [(age−0.203) (years old)] × (Female × 0.742).4

**AMI definition**

Acute myocardial infarction was defined as the occurrence of two or more of the following: chest pain, ischemic ECG change, and elevated cardiac marker, according to the unified global definition jointly issued by European Society of Cardiology (ESC), American College of Cardiology (ACC), American Heart Association (AHA), and the World Federation (WHF) in October 2007 and graded based on the Killip classification.

**Definition of low cardiac output syndrome**

Patients who need vasoactive drugs to maintain or implanted aortic balloon counterpulsation (IABP) within 48 h of post-operation.5

**Statistical method**

Statistical analysis was performed with the R 2.15.2 (http://www.r-project.org/). Count data were expressed as percentage. Differences between groups were compared using χ² test. Normally distributed measurement data were expressed as x ± s. Abnormally distributed measurement data were expressed as M (1/4, 3/4). Differences between groups were compared using rank sum test. Risk factors related to AKI and mortality during hospitalization were analyzed using univariate and multivariate logistic regression. p<0.05 was considered statistically significant.

**Results**

**Basic information of inpatients and the prevalence of AKI**

Among the 1371 (82.8%) effective inpatients, AKI prevalence was 29.9%, of which, that was 22.1% for AKI patients at stage I, 5.1% for those at stage II, and 2.6% for those at stage III; the prevalence of AKI after conservative treatment, CAG and CABG was 34.8% (228/656), 24.5% (168/686), and 57.7% (30/52), respectively. Table 1 lists the baseline information of all enrolled patients. Patients with AKI after conservative treatment or CAG had significantly decreased cardiac function and basal renal function than non-AKI patients as indicated by increased proportion of patient with Killip grade 2 and baseline eGFR 5 60 mL·min−1·(1.73 m2)−1 decreased LVEF, p<0.05 for all. Compared to control non-AKI patients, patients with AKI after CABG had poorer heart function, but only showed a statistical significant differences in LVEF (Table 2).

**Mortality of inpatients**

A total of 107 patients died during hospitalization, showing overall mortality of 7.8%. In detail, the mortality of AKI patients were significantly higher than that of
non-AKI patients [17.1% (70/410) vs. 3.9% (37/961), \( \chi^2=68.0, p<0.001 \)]. Moreover, the mortality gradually increased with the progress of AKI, that was 11.8% (36/304) for AKI patients at stage I, 22.9% (16/70) for AKI patients at stage II, and 50.0% (18/36) for AKI patients at stage III, and showed statistically significant between any two groups (\( \chi^2=4.9 \) and \( p=0.027 \) between stages I and II, \( \chi^2=32.3 \) and \( p<0.001 \) between stages III and I as well as \( \chi^2=6.8 \) and \( p=0.008 \) between stages III and II).

**Logistic regression analysis of AKI patients**

Multivariate logistic regression analysis of the meaningful factors in univariate analysis after adjusted with age and gender showed that (1) for AKI patients in conservative treatment group, decreased baseline eGFR (\( OR = 2.049, 95\% CI: 1.246-3.370 \)), increased fasting blood glucose (\( OR = 1.070, 95\% CI: 1.018-1.124 \)), diuretics (\( OR = 1.867, 95\% CI: 1.220-2.856 \)) and myocardial infarction Killip grade IV (\( OR = 1.362, 95\% CI: 1.059-3.170 \)) were
Acute kidney injury is a sudden deterioration of renal function. Because of its mild manifestation at early stage, AKI is easily missed in diagnosis. Once symptoms worsened, patients often has entered the moderate and severe AKI stages, which not only extends days of hospitalization but also increases mortality. Therefore, early identification and intervention of AKI is crucial to patient outcomes.

### Discussions

Acute kidney injury is a sudden deterioration of renal function. Because of its mild manifestation at early stage, AKI is easily missed in diagnosis. Once symptoms worsened, patients often has entered the moderate and severe AKI stages, which not only extends days of hospitalization but also increases mortality. Therefore, early identification and intervention of AKI is crucial to patient outcomes.

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**Table 3. Multivariate logistic regression analysis of AKI occurrence after conservative treatment adjusted with gender and age.**

| Variables                        | B-value | Adjusted OR value | 95% CI     | p-Value |
|----------------------------------|---------|-------------------|------------|---------|
| Diastolic blood pressure at admission | −0.018  | 0.986            | 0.974–0.998 | 0.029   |
| History of stroke                | 0.242   | 1.286            | 1.094–1.512 | 0.034   |
| Acute infections                 | 0.168   | 1.332            | 1.093–1.625 | 0.017   |
| Baseline eGFR < 60 (ml/min/(1.73 m²)^ −1) | 6.717   | 2.049            | 1.246–3.407 | 0.005   |
| Fasting plasma glucose (mg/dl)    | 0.015   | 1.070            | 1.018–1.124 | 0.007   |
| Albumin (g/dl)                   | −0.021  | 0.979            | 0.928–1.009 | 0.118   |
| Hyperuricemia (mg/dl)            | 0.373   | 1.133            | 0.717–1.728 | 0.631   |
| Killip grade                      |         |                   |            |         |
| Killip grade II                  | 0.169   | 1.291            | 0.702–2.374 | 0.309   |
| Killip grade III                 | −0.275  | 0.725            | 0.425–1.378 | 0.468   |
| Killip grade IV                  | 0.366   | 1.636            | 1.055–3.167 | 0.047   |
| Diuretics                        | 0.646   | 1.867            | 1.220–2.856 | 0.019   |
| LVEF                             | −0.005  | 0.999            | 0.975–1.025 | 0.950   |

**Table 4. Multivariate logistic regression analysis of AKI occurrence after CAG adjusted with gender and age.**

| Variables                        | B-value | Adjusted OR value | 95% CI     | p-Value |
|----------------------------------|---------|-------------------|------------|---------|
| History of stroke                | −0.445  | 0.641            | 0.324–1.267 | 0.200   |
| Acute infections                 | 0.518   | 1.678            | 1.023–2.754 | 0.040   |
| baseline eGFR < 60 (ml/min/(1.73 m²)^ −1) | 0.863   | 2.371            | 1.500–3.747 | <0.001 |
| Fasting plasma glucose (mg/dl)    | 0.009   | 1.009            | 1.005–1.012 | <0.001 |
| Albumin (g/dl)                   | −0.022  | 0.978            | 0.919–1.220 | 0.220   |
| Hyperuricemia (mg/dl)            | 0.173   | 1.189            | 0.647–2.185 | 0.576   |
| Killip grade                      |         |                   |            |         |
| Killip grade II                  | 0.331   | 1.343            | 0.708–2.241 | 0.335   |
| Killip grade III                 | −0.333  | 0.765            | 0.186–3.738 | 0.626   |
| Killip grade IV                  | −0.054  | 0.948            | 0.277–3.237 | 0.172   |
| Diuretics                        | 0.341   | 1.674            | 1.042–2.690 | 0.032   |
| LVEF                             | −0.012  | 0.988            | 0.972–1.026 | 0.908   |
| Coronary stenting                | 0.019   | 1.019            | 0.673–1.544 | 0.928   |
| Intraoperative hypotension       | 0.621   | 2.276            | 1.324–3.575 | 0.008   |

**Table 5. Multivariate logistic regression analysis of AKI occurrence after CABG adjusted with gender and age.**

| Variables                        | B-value | Adjusted OR value | 95% CI     | p-Value |
|----------------------------------|---------|-------------------|------------|---------|
| History of stroke                | 0.326   | 1.386            | 0.903–2.125 | 0.137   |
| Acute infections                 | 0.287   | 1.331            | 0.903–1.964 | 0.175   |
| baseline eGFR < 60 (ml/min/(1.73 m²)^ −1) | 0.825   | 2.286            | 1.340–3.981 | <0.001 |
| Fasting plasma glucose (mg/dl)    | 0.068   | 1.059            | 1.018–1.124 | 0.006   |
| Albumin (g/dl)                   | −0.029  | 0.972            | 0.923–1.012 | 0.129   |
| Hyperuricemia (mg/dl)            | 0.107   | 1.109            | 0.706–1.734 | 0.631   |
| Killip grade                      |         |                   |            |         |
| Killip grade II                  | 0.261   | 1.298            | 0.706–2.387 | 0.409   |
| Killip grade III                 | −0.189  | 0.828            | 0.425–1.612 | 0.668   |
| Killip grade IV                  | 0.524   | 1.689            | 0.855–3.325 | 0.172   |
| Diuretics                        | 0.541   | 1.723            | 1.122–2.650 | 0.012   |
| LVEF                             | −0.006  | 0.945            | 0.971–1.032 | 0.908   |
| Number of grafts                 | 0.871   | 2.490            | 0.765–3.121 | 0.428   |
| Postoperative low cardiac output | 0.721   | 2.331            | 1.277–3.286 | 0.007   |

**Table 6. Multivariate logistic regression analysis of mortality during hospitalization.**

| Variables                        | B-value | Adjusted OR value | 95% CI     | p-Value |
|----------------------------------|---------|-------------------|------------|---------|
| Model I                          | 1.790   | 5.989            | 2.658–13.494 | <0.001 |
| Model II                         |         |                   |            |         |
| KDIGO staging                    |         |                   |            |         |
| AKI-I stage                      | 1.686   | 5.396            | 2.263–12.869 | <0.001 |
| AKI-II stage                     | 1.352   | 3.865            | 1.077–13.873 | 0.038   |
| AKI-III stage                    | 2.778   | 16.073           | 5.038–51.273 | <0.001 |

Notes: Adjusted variables include gender, age, diastolic pressure at admission, stroke history, acute infection, serum albumin level, baseline eGFR <60 [ml/min/(1.73 m²)^ −1], hyperuricemia, fasting glucose, LVEF level and diuretics.
hospitalization and increases costs, but also increases mortality. Among patients with AMI, congestive heart failure and sepsis, those who underwent cardiac surgery and other high-risk groups, AKI prevalence is very high (10% to 25%). Our study showed that the prevalence of patients with AMI was 29.9%, of which, that was 22.1% for AKI patients at stage I, 51% for those at stage II, and 2.6% for those at stage III. The overall mortality of AMI patients with AKI was 7.8%, which was 4.39-fold of that of AMI patients without AKI. In detail, the mortality of AMI patients with stage I, II, and III AKI was 3.03-, 5.87-, and 12.82-times of that of AMI patients without AKI, consistent with previous reports. In addition, multivariate regression analysis indicated that incremental AKI staging is an independent risk factor for increased mortality during hospitalization.

It is widely accepted that AKI on the basis of CKD is the third etiology of AKI. Hsu et al. pointed out that different eGFR stratification is closely associated with the risk of AKI. The risk of AKI for patients with baseline eGFR at 15-29 mL/min/(1.73 m²)⁻¹ and 45-59 mL/min/(1.73 m²)⁻¹ is 29- and 2-fold of that for patients with baseline eGFR ≥ 60 mL/min/(1.73 m²)⁻¹. Our results suggest that the risk for AKI for AMI patients with baseline eGFR < 60 mL/min/(1.73 m²)⁻¹ was significantly increased and decline in basic renal function is the strongest risk factor for AKI prevalence for AMI patients in conservative treatment and post-operative CAG groups (adjusted OR was 2.049 and 2.371, respectively). AKI is induced by multiple factors and often co-regulated by basal renal function and other nerve-humoral and endocrine factors. Patients with poor basal renal function had weak kidney reserve capacity and low compensatory ability. After AMI, they further suffered from heart and kidney hypoperfusion and strong stress response, therefore, leading to an increased renal damage. Thus, early screening, early diagnosis, and early treatment have a very important clinical significance for CKD patients.

In recent years, more and more researches began to focus on the decisive role of hyperglycemia in AMI prognosis. Acute blood sugar elevation at admission can lead to deterioration of renal function after cardiac surgery (including coronary angiography or bypass surgery), and increased mortality. For patients without diabetes, acute blood sugar elevation at admission is also very common at early stage of AMI. Our study found that hyperglycemia at admission was an independent risk factor for AMI complicated with AKI, regardless of AKI occurring after surgery or conservative treatment. A large number of physiological studies have revealed that oxidative stress, inflammation, apoptosis, endothelial injury, hypercoagulable state, and platelet aggregation are the mechanisms underlying the effects of hyperglycemia on myocardial ischemic injury. Therefore, the emphasis of blood glucose management in patients with AMI may contribute to recovery of renal function and decreases of mortality.

Our findings suggest that, AMI patients treated with either conservative method modality or surgical intervention, use of diuretics is one of the independent risk factor for AKI prevalence. For patients with conservative treatment, elevated diastolic blood pressure at admission was a protective factor of AKI. For AMI patients underwent post-operative intervention, intra-CAG hypotenion and post-CABG low cardiac output were independent risk factors for AKI. These results are presumably related to heart and kidney hypoperfusion after AMI, use of diuretics, surgery-induced stress response, over long operation time, hemodynamic instability during surgery, post-operative restrictions on the amount of fluid, ischemia-reperfusion injury, sepsis, heart failure, drug-related injuries (diuretics, ACEI or ARB use, etc.), multiple organ failure and other factors. Therefore, it is necessary to strictly monitor patient’s hemodynamics to reduce volatility of renal perfusion. In addition, for high-risk patients with insufficient effective circulating blood volume, these drugs should be used with caution at early stage of post-operation in addition to closely monitoring changes in renal function.

Hillege et al. found that patients with chronic heart failure had renal failure rate of about 25% and increased mortality. Ronco et al. speculated that this may related to impaired cardiac function-induced changes in hemodynamics, renal perfusion reduction, long-term use of nephrotoxic drugs in addition to changes in body fluid endocrine regulation and imbalances of immune regulation, and others. In this study, baseline data suggest that heart function and basal renal function were significantly lower for AKI patients in conservative treatment group and post-CAG group than patients in the control group as indicated by increased proportion of patients with Killip grade 2-4, low LVEF, and decreased baseline eGFR. In addition, cardiac function was poorer for AKI patients in the CABG group than patients in the control group as shown by only significant decreased in LVEF level. Multivariate logistic regression analysis indicated that Killip IV was an independent risk factor for AKI disease only for patients in the conservative treatment group and post-operative risk of AKI was not independently associated with pre-operative cardiac function level. Whether these results for patients in the surgery group are associated with confounding factors need to be further studied.

Acute kidney injury is less appeared as an isolated organ dysfunction. For critically ill patients, AKI is often
only a part of multiple organ dysfunction syndrome. For example, it is often associated with sepsis, shock, cardiovascular events, and other acute acute stresses. Large-scale multi-center observational study found that critically ill patients with severe infections (e.g. sepsis) is a major cause of AKI. 23 Logistic regression showed that critically ill patients with severe infections (e.g. sepsis) is a major cause of AKI in patients after CAG. Therefore, a good prevention of infectious diseases may to some extent prevent the occurrence of AKI.

Although hyperuricemia, hypertension, history of stroke, serum albumin levels, decreased LVEF, and coronary stenting are independent risk factors with statistical significance in univariate analysis, they are not independent risk factors in multiple regression analysis, presumably due to the influence of other confounding factors, which need to be further explored.

In summary, this study is a single center retrospective study. Although the study described the AKI distribution in hospitalized AMI patients and its related risk factors, it cannot determine the causal relationship between AKI prevalence and these risk factors. Nevertheless, our results suggest that early prevention of AKI in AMI patients has clinical significance for improving the prognosis of patients.

Disclosure statement
The authors have no conflicts of interest to disclose.

References
1. Warnock DG. Towards a definition and classification of acute kidney injury. *J Am Soc Nephrol.* 2005;16:3149–3150.
2. Bruetto RG, Rodrigues FB, Torres US, Otaviano AP, Zanetta DM, Burdmann EA. Renal function at hospital admission and mortality due to acute kidney injury after myocardial infarction. *PloS One.* 2012;7:e35496.
3. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney Int Suppl.* 2012;2:S1–S138.
4. National Kidney Foundation. *K/DOQI* clinical practice guidelines for chronic kidney disease: Evaluation, classification and stratification. *Am J Kidney Dis.* 2002;39:S1–S266.
5. Palomba H, de Castro I, Neto AL, Lage S, Yu L. Acute kidney injury prediction following elective cardiac surgery: AKICS score. *Kidney Int. 2007;72:624–631.
6. Chertow GM, Burdick E, Honour M, Bonventre JV, Bates DW. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. *J Am Soc Nephrol.* 2005;16:3365–3370.
7. Parikh CR, Coca SG, Wang Y, Masoudi FA, Krumholz HM. Long-term prognosis of acute kidney injury after acute myocardial infarction. *Arch Intern Med.* 2008;168:987–995.
8. Oda T. Acute kidney injury after off-pump coronary artery bypass grafting. *Circ J.* 2010;74:1069–1070.
9. Cruz DN, Ricci Z, Ronco C. Clinical review: RIFLE and AKIN - time for reappraisal. *Crit Care.* 2009;13:211. doi: 10.1186/cc7759.
10. Anavekar NS, McMurray JJ, Velazquez EJ, et al. Relation between renal dysfunction and cardiovascular outcomes after myocardial infarction. *N Engl J Med.* 2004;351:1285–1295.
11. Metra M, Nodari S, Parrinello G, et al. Worsening renal function in patients hospitalised for acute heart failure: Clinical implications and prognostic significance. *Eur J Heart Fail.* 2008;10:188–195.
12. Hsu CY, Ordoñez JD, Chertow GM, Fan D, McCulloch CE, Go AS. The risk of acute renal failure in patients with chronic kidney disease. *Kidney Int.* 2008;74:101–107.
13. Bagshaw SM, George C, Gibney RT, Bellomo R. A multicenter evaluation of early acute kidney injury in critically ill trauma patients. *Ren Fail.* 2008;30:581–589.
14. Moriyama N, Ishihara M, Noguchi T, et al. Admission hyperglycemia is an independent predictor of acute kidney injury in patients with acute myocardial infarction. *Circ J.* 2014;78:1475–1480.
15. Ishihara M, Kojima S, Sakamoto T, et al. Comparison of blood glucose values on admission for acute myocardial infarction in patients with versus without diabetes mellitus. *Am J Cardiol.* 2009;104:769–774.
16. Liotta M, Olsson D, Sartipy U, Holzmann MJ. Minimal changes in postoperative creatinine values and early and late mortality and cardiovascular events after coronary artery bypass grafting. *Am J Cardiol.* 2014;113:70–75.
17. Olivero JJ, Olivero JJ, Nguyen PT, Kagan A. Acute kidney injury after cardiovascular surgery: An overview. *Methodist Debakey Cardiovasc J.* 2012;8:31–36.
18. Che M, Li Y, Liang X, et al. Prevalence of acute kidney injury following cardiac surgery and related risk factors in Chinese patients. *Nephron Clin Pract.* 2011;117:c305–c311.
19. Chao CT, Lin YF, Tsai HB, Wu VC, Ko WJ. Acute kidney injury network staging in geriatric postoperative acute kidney injury patients: Shortcomings and improvements. *J Am Coll Surg.* 2013;217:240–250.
20. Nadeau-Fredette AC, Bouchard J. Fluid management and use of diuretics in acute kidney injury. *Adv Chronic Kidney Dis.* 2013;20:45–55.
21. Hillegé HL, Nitsch D, Pfeffer MA, et al. Renal function as a predictor of outcome in a broad spectrum of patients with heart failure. *Circulation.* 2006;113:671–678.
22. Ronco C, Chionh CY, Haapio M, Anavekar NS, House A, Bellomo R. The cardiorenal syndrome. *Blood Purif.* 2009;27:114–126.
23. Bagshaw SM, George C, Bellomo R; ANZICS Database Management Committee. Early acute kidney injury and sepsis: A multicentre evaluation. *Crit Care.* 2008;12:R47–R55.