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Original Article

Postoperative hyperlactatemia is linked to acute kidney injury after type A aortic dissection surgery: a retrospective study

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Abstract: Purpose: Acute kidney injury (AKI) could increase the risk of mortality after cardiac surgery. We aimed to explore the predictive value of hyperlactatemia in AKI in patients undergoing type A aortic dissection (TA-AAD) operation. Methods: A total of 337 consecutive patients who underwent TA-AAD surgery in Beijing Anzhen Hospital from February 2009 to December 2013 were included into this study. Based on blood lactate level at 6 hours after surgery (median, 4.1 mmol/L), patients were divided into two groups: high (≥4.1 mmol/L) and low lactic (<4.1 mmol/L) groups. We examined the association between risk factors and AKI, especially the impact of postoperative lactate level on the incidence of AKI. Results: Of 337 patients, 152 (45.1%) patients had early postoperative AKI. 29 (8.6%) patients needed Continuous Renal Replacement Therapy (CRRT). There were 164 patients in the high lactate group and 173 patients in the low lactate group. Additionally, the rates of the AKI Stage 2-3 were also markedly different between the two groups (P<0.05). Furthermore, postoperative lactate level at 6 h was an effective predicator for AKI (OR, 1.284; 95% CI: 1.129-1.460; P<0.001; AUC, 0.767) and RRT (OR, 1.295; 95% CI: 0.737-0.891; P<0.001; AUC, 0.814). Lactate level of 5.05 mmol/L was the boundary value for AKI Stage 2-3 (sensitivity 71.0%, specificity 69.3%). Meanwhile, lactate level of 7.0 mmol/L was the boundary value for CRRT (sensitivity 73.9%, specificity 79.5%). Conclusions: Our study found that early postoperative lactate level was an effective predictor of AKI and CRRT for patients with acute type A aortic dissection.

Keywords: Hyperlactatemia, type A aortic dissection, acute kidney injury, renal replacement therapy

Introduction

Acute kidney injury (AKI) is a common serious complication for patients following the aorta operation. It was reported that the incidence of AKI was approximately 28.6%-54.0%, of which 1.7%-11% patients had to receive renal replacement therapy (RRT) [1]. Moreover, AKI was an independent predictor of increased risk of mortality irrespective of the perioperative risk factors [2]. In a Sweden nationwide population-based study of 27,929 patients undergoing cardiac surgery, Rydén et al showed that AKI was associated with long-term risk of mortality and myocardial infarction [3]. Additionally, Pickering et al found that the pooled rate of cardiopulmonary bypass (CPB)-related AKI was 18.2%, and of RRT was 2.1%. Moreover, AKI was associated with a more than 2-fold increase in early mortality [4]. Hyperlactatemiain patients who have undergone type A aortic dissection (TA-AAD) surgeryis fairly common andis associated with tissue hypoxia [5], which is associated with increased risk of AKI [6, 7]. Several studies have confirmed that postoperative hyperlactatemia was related to incidence of AKI in patients with cardiac surgery. However, no study evaluates the predictive value of hyperlactatemia for AKI and RRT in patients with aortic dissection surgery. Therefore, our study is to evaluate the relationship of the postoperative lactate level with the rate of AKI and RRT.

Materials and methods

Study population

We retrospectively analyzed the clinical data of 342 participants with TA-AAD undergoing aorta replacement in Beijing Anzhen Hospital, Capital Medical University, from February 2009 to February 2013. Five patients were excluded

owing to incomplete data. Our study was approved by the Beijing Anzhen Hospital Review Board. On the basis of the Stanford classification, acute type A dissection was separated into 3 subtypes [8, 9]. Subtype A_1: normal aortic root type; Subtype A_2: mild-to-moderately involved aortic root type; and Subtype A_3: severely involved aortic root type. Additionally, according to the pathological characteristics of aortic arch, it divided into subtype C (complex type) and subtype S (simple type). Meanwhile, there are several different operation types, including aortic root plastic or replacement; ascending aorta replacement; partial or full aortic arch replacement and aortic arch replacement plus stented elephant trunk implantation surgery.

CPB is established based on different surgery circumstances, including moderate hypothermal CPB, hypothermal CPB, deep hypothermia circulatory arrest (DHCA) and selective cerebral perfusion (SCP). Arteriovenous cannulation methods varied according to different types of surgeries. When lesions affect aortic arch, cerebral perfusion or separated perfusion for upper and lower body was performed to protect brain function.

After going back to ICU, the mechanical ventilation mode was set as control mode ventilation: rate was 12/min; tidal volume 6-8 mL/kg; PEEP 4-8 mmHg, FiO_2 40%-60% to keep SAT ≥97%. Additionally, we adjusted the amount vasoactive drugs, such as dopamine, dobutamine, epinephrine, norepinephrine to keep hemodynamic stable.

KDIGO criteria

AKI was defined with the Kidney Disease Improving Global Outcomes Work Group (KDIGO) criteria [10], including the increase of serum creatinine (sCr) 0.3 mg/dl within 48 h, or increase of sCr by 1.5-fold above baseline, known orassumed to have occurred within 7 days. Meanwhile, participants with AKI was divided into 3 stages by postoperative sCr level. Stage 1: sCr≥1.5 times baseline or sCr≥0.3 mg/dL increase; Stage 2: sCr≥2.0 times baseline; Stage 3: ≥3.0 times baseline or increase of sCr to ≥4.0 mg/dl or initiation of RRT.

Continuous renal replacement therapy (CRRT)

The indications of CRRT include volume overload, serious kidney dysfunction and electrolyte disturbances. Based on the participants' renal function, cardiopulmonary function, inflammatory response, water-electrolyte status and acid-base balance, surgeons, ICU doctors and renal physicians will decide whether the RRT was performed. For patients with CRRT, we used the continuous veno venous hemo dia filtration (CVVHDF) mode with the Prismaflex system (Barkey Gambro, Sweden). The treatment mode, dose, anticoagulation methods and dehydrated volume were performed according to the patients' conditions.

Data collection

We used the BECKMAN COULTER-AU5400 automatic biochemical analyzer to assess serum creatinine, urea nitrogen and alanine aminotransferase (ALT). Additionally, RADIOMETER-ABL800 blood gas analyzer was used to detect lactate level and other items. We also recorded patients' central venous pressure (CVP), mean arterial pressure (MAP), arterial blood gas analysis (including PaO_2, glucose, lactate) at 6 h post-operation, in-hospital rate of AKI and CRRT.

Statistical analysis

All statistical analysis was performed with SPSS 19.0 (SPSS, Inc., Chicago, Ill). Continuous variables were expressed as mean ± SD or the median with interquartile range. Independent continuous variables were compared with 2-tailed Student t tests. Categorical variables were expressed as frequency or ratio and compared with the Pearson c^2 statistic. Stepwise multivariate logistic regressions were used to analyze the predictors of AKI and RRT. Receiver operating characteristic (ROC) curves were constructed, and the area under the ROC curve (AUC) was used to assess discriminant ability of the lactate levels measured 6 hours after surgery to predict the rate of AKI and RRT. The cut-off was the value that maximized the sensitivity and specificity. $P<0.05$ was statistically significant.

Result

Study population

There were 337 participants in our study, including 267 (79.2%) males and 68 females

Table 1. The Characteristics of the high-LAC versus low-LAC groups

Characteristic and comparison factor	High-LAC (≥4.1 mmol/L) (n=164)	Low-LAC (<4.1 mmol/L) (n=173)	*t* value or χ^2 value	*P* value
Sex (male)	129 (48.1)	139 (51.9)	0.076	0.783
Age (year)	49.0±10.9	47.0±11.1	1.678	0.094
BMI	25.8±3.4	25.6±3.7	0.385	0.701
Smoking	84 (49.4)	86 (50.6)	0.112	0.738
Diabetes	5 (31.3)	11 (68.8)	2.004	0.157
Hypertension	113 (50.7)	110 (49.3)	1.239	0.266
CHD	2 (25.0)	6 (75.0)	1.814	0.178
Stanford			8.420	0.394
A1C	54	49		
A2C	49	42		
A3C	66	64		
A1S	2	4		
A2S	1	1		
A3S	2	3		
CPB (min)	202±53	184±48	3.149	0.002
DHCA (min)	111±34	103±35	2.121	0.035
Bleeding (ml)	2236±1460	1849±848	2.947	0.003
MAP (mmHg)	79±13	84±12	-3.969	0.000
PO2 (mmHg)	123±64	129±63	0.076	0.940
CVP (mmHg)	12±3	10±2	4.353	0.000
Glu (mg/dl)	249±88	197±59	6.373	0.000

LAC, lactate; BMI, body mass index; CHD, coronary heart disease; CPB, cardiopulmonary bypass; DHCA, deep hypothermal circulatory arrest; LVEF, left ventricle eject fraction; MAP, mean arterial pressure; CVP, central venous pressure; Glu, glucose; Data are mean ± standard deviation or n (%).

Table 2. Outcomes and comparisons of high- versus low-LAC groups

Characteristic	High-LAC (≥4.1 mmol/L) (n=164)	Low-LAC (<4.1 mmol/L) (n=173)	*t* value or χ^2 value	*P* value
Ventilation time (h)	68±103	38±50	3.472	0.001
ICU LOS (day)	4.6±6.3	2.5±2.4	4.143	0.000
Hospital LOS (day)	23.4±15.3	17.9±8.0	3.640	0.000
Hospital mortality	18 (94.7)	1 (5.3)	17.228	0.000
CRRT (n/%)	26 (89.7)	3 (10.3)	21.51	0.000
AKI	92 (60.5)	60 (39.5)	16.04	0.000
Stage 1	40 (48.2)	43 (51.8)	0.004	0.947
Stage 2	28 (66.7)	14 (33.3)	6.334	0.012
Stage 3	24 (88.9)	3 (11.1)	19.163	0.000

ICU, intensive-care unit; LOS, length of stay; CRRT, continuous renal replacement therapy; AKI, acute renal injury; Data are median (interquartile range), mean ± standard deviation or n (%).

(21.2%). According to serum lactate level within 6 h after operation (median, 4.1 mmol/L), all participants were separated into high lactic (≥4.1 mmol/L) and low lactic (<4.1 mmol/L) groups. The characteristics of two groups are illustrated in **Table 1**. There were no significant differences between two groups in age, BMI, rates of smoking, diabetes, coronary heart disease, hypertension and the Stanford types of aorta dissection. However, the two groups were statistically different in duration of CPB (202±53 min vs. 184±48 min), DHCA (111±34 min vs. 103±35 min), blood loss (2236±1460 mL vs. 1849±848 mL), MAP (79±13 mmHg vs. 84±12 mmHg), CVP (12±3 mmHg vs. 10±2 mmHg) and blood glucose (249±88 mmol/L vs. 197±59 mmol/L).

Outcomes

AKI occurred in 152 (45.1%) patients, of which 29 patients (8.6%) needed CRRT. The average length of CRRT was 5.9±3.1 d, and average length in ICUwas 3.5±4.8 d. There were significant differences between the two groups in the length of intubation (68±103 h vs. 38±50 h), ICU length of stay (4.6±6.3 d vs. 2.5±2.4 d), hospital length of stay (23.4±15.3 d vs. 17.9±8.0 d) and rate AKI (60.5% vs. 39.5%), CRRT (89.7% vs. 10.3%), stage 2 AKI (66.7% vs. 33.3%) and stage 3 AKI (88.8% vs. 11.1%). However, the risk of Stage 1 AKI was comparable between the two groups. The outcomes are illustrated in **Table 2**.

All factors possibly related to AKI were taken into logistic regression analysis (**Table 3**). Results showed that postoperative lactate level (OR 1.284, 95% CI 1.129-1.460, P<0.001) and blood loss (OR 1.000, 95% CI 1.000-1.001, P<0.001) were associated with the occurrence of AKI, and the lactate are the risk factors for AKI.

Table 3. Correlation analysis of factors may effect LAC behaviors

Variable	*P* value	R value
Bleeding	<0.001	0.239
Age	0.509	0.036
BMI	0.04	0.116
LVEF	0.231	-0.72
CPB	<0.001	0.206
Clamping	0.015	0.133
DHCA	0.74	0.018
MAP	<0.001	-0.209
CVP	<0.001	0.336
PO2	0.162	-0.076
ALT	0.001	0.180
GLU	<0.001	0.503

LVEF, left ventricle eject fraction; ALT, alanine aminotransferase; GLU, glucose.

Table 4. The comparisons of the stage of AKI in CRRT

	No-CRRT	CRRT	χ^2 value	*P* value
AKI	123 (80.9)	29 (19.1)	38.16	0.000
Stage 1	78 (94.0)	5 (6.0)	0.933	0.334
Stage 2	35 (83.3)	7 (16.7)	3.690	0.050
Stage 3	10 (37.0)	17 (63.0)	110.272	0.000

CRRT, continuous renal replacement therapy; AKI, acute kidney injury.

The comparisons of the stage of AKI versus RRT in **Table 4**. there were significant differences observed between the No-CRRT versus CRRT groups in AKI (80.9% vs. 19.1%, respectively, P=0.001), stage 2 of AKI (83.3% vs. 16.7%, P=0.050) and stage 3 of AKI (37.0% vs. 63.0%, P<0.001).

The receiver operating characteristics (ROC) curve of lactate predicting stage 2 to stage 3 of AKI after surgery of aortic dissection type A was illustrated in **Figure 1**. The AUC was 0.767 (OR 1.284; 95% CI 1.129-1.460, P<0.001). Lactate levels of 5.05 mmol/L was the boundary value of AKI (sensitivity 71.0%, specificity 69.3%). Additionally, the AUC of lactate predicting CRRT 0.814 (95% CI 0.737-0.891, P<0.001). Lactate levels of 7.0 mmol/L was the boundary value of CRRT (sensitivity, 75.9%; specificity, 79.5%) (**Figure 2**).

Discussion

In consistent with previous studies, our study demonstrated that lactate levels at 6 hours after aortic dissection surgery was significantly related to postoperative adverse events, including AKI and CRRT. A threshold of 5.05 mmol/L at 6 hours after surgery is independently associated with the risk of AKI. Additionally, a lactate level of 7.0 mmol/L identifies patients needing CRRT. More studies are needed to confirm our findings.

Hyperglycemia was associated with increased risk of AKI. Cardiac surgery is the second leading causes of AKI in the ICU, just following sepsis. Although the pathophysiology of AKI following cardiac surgery is multifactorial, the dominant mechanism of injury is thought to be intraoperative ischemia-reperfusion injury. Since lactate is a sensitive biomarker of tissue hypoxia, it can be used as a marker of AKI. Dividing 117 patients in the AKI group (n=17) and non-AKI group (n=100), Zhang et al reported that normalized lactate load was independently associated with postoperative AKI in patients undergoing CPB [11]. The AUC was 0.63 (95% CI 0.47-0.79). The best sensitivity and specificity were 41.2% and 87% at the cutoff point of 4.4 mmol/L. Moreover, they found that at different time points, serum lactate showed markedly different diagnostic performance. While lactate measured early may be timely enough but also lacks accuracy, and lactate measured late may be accurate but also may be too late. They found that the time point of 8.4 hours after ICU entry showed better diagnostic performance (AUC 0.73; 95% CI, 0.64-0.81) [11]. Using a modified RIFLE (risk, injury, failure, loss of kidney function, and end-stage renal failure), Lopez-Delgado and coworkers demonstrated that serum lactate 24 hours after admission is an independent risk factor for AKI [12]. However, patients enrolled in those studies are mainly with coronary artery bypass surgery (CABG) or valve surgery. Meanwhile, variable adherence to AKI definitions (RIFLE, AKIN and KDIGO criteria) can lead to significant differences in estimates of test discrimination. In consistent with these studies, we confirmed that postoperative hyperlactatemia was related to AKI according to KDIGO criteria. However, we recommended that lactate level (5.05 mmol/L) at 6 h after aortic dissection surgery was an effective predictor of AKI.

Hyperlactatemia was also related to severe AKI. For patients with severe AKI, several independently validated models have been developed to predict RRT after cardiac surgery. Of

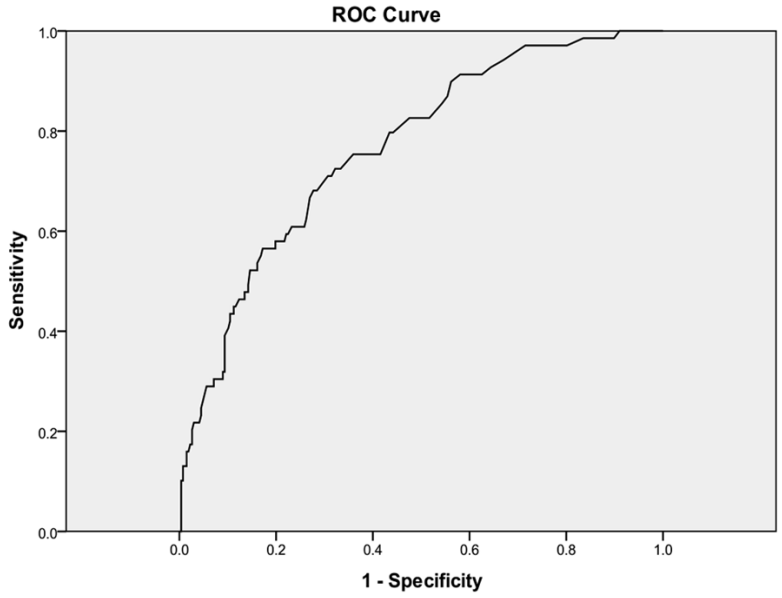

Figure 1. ROC curve of LAC alerting stage 2 and stage 3 of AKI after the operation of A type aortic dissection. ROC, receiver operating characteristic; LAC, lactate; AKI, acute kidney injury.

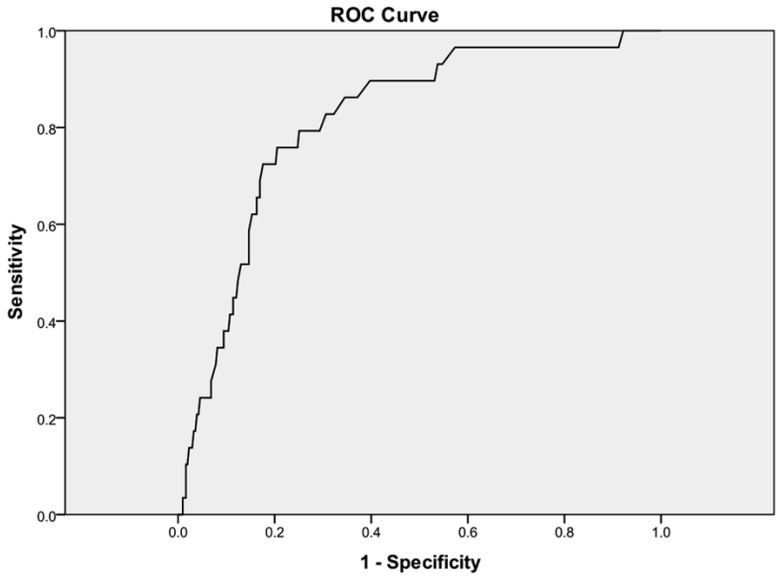

Figure 2. ROC curve of LAC alerting CRRT after the operation of A type aortic dissection. ROC, receiver operating characteristic; CRRT, continuous renal replacement therapy; LAC, lactate.

these, the Thakar score have demonstrated good to excellent discrimination in both the original derivation and validation cohorts (AUC 0.81 vs. 0.82, respectively) and also in later independent validations studies (AUC 0.86 and 0.82, respectively) for AKI requiring dialysis [13-15]. However, whether new biomarkers, such as Cyctatin C, neutrophil gelatinase-associated lipocalin (NGAL), kidney injury marker 1 (KIM-1), urine IL-18 and B-type peptide (BNP) might better predict severe AKI has not been addressed in large studies. Using the lactate alone, our study showed that a lactate level of 7.0 mmol/L at 6 h after surgery could identify patients with severe AKI. The AUC of predicting stage 2 to stage 3 of AKI after surgery was 0.767 and the AUC of predicting CRRT was 0.814. Furthermore, we also demonstrated that higher lactate levels (7.0 mmol/L) was associated with increased risk of CRRT, which was confirmed by another studies conducted in pediatric patients undergoing CPB [16].

AKI was associated with increased risk of morbidity and mortality, and several novel biomarkers have been developed for the early detection of AKI after cardiac surgery. Ideally, a biomarker would identify kidney injury as it occurs intraoperatively or at least within a few hours after surgery. Extensive studies have identified potential biomarkers for the prediction of AKI after insult from cardiac surgery [17]. Combining 28 studies reporting new biomarkers to detect AKI, Ho et al found that intraoperative discrimination by the urine biomarkers NGAL and KIM-1 demonstrated AUC<0.7, whereas cystatin c had AUC<0.75. In the 24-hour postoperative period, the composite AUC for postoperative urine cystatin C, NGAL, and IL-18 were ≤0.7. Similarly, the composite AUC for postoperative blood NGAL and cystatin-c were <0.7 [18]. Recently, growth dif-

ferentiation factor 15 has been found to be associated with the risk of AKI. Previous studies have shown that pre-operative blood GDG-15 levels significantly improve the prognostic value the the EuroSCORE for mortality after cardiac surgery [19]. Guenancia et al found that GDF-15 was the best pre-operative biomarker to predict AKI (AUC, 0.83) compared with eGFR (AUC 0.67) and NT-proBNP (AUC 0.62). Moreover, pre-operative GDF-15 was also markedly better than the EuroSCORE in predicting AKI (AUC 0.62) [20].

There are several effective therapeutic agents for the prevention and treatment of AKI after cardiac surgery. Maintaining adequate hydration and optimal MBP, avoiding tissue hypoperfusion, and minimizing exposure to nephrotoxic agents are effective strategies that help decrease renal injury. Preoperative hydration with half isotonic saline (1 ml/kg/h) administered for 12 hours seems to be beneficial in patients with moderate to severe kidney disease (GFR<45 ml/min/1.73 m^2) affected a 53% reduction in AKI [21]. Additionally, conservative hydration strategy in the immediate postoperative period was associated with more ventilator-free and ICU-free days without an increase in AKI [22]. Moreover, hyperglycemia induces oxidative stress and stimulates reactive oxygen species. The Society for Thoracic Surgeon guideline recommend the blood glucose level less than 180 mg/dL during cardiac surgery. The use of angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, statins, diuretics, natriuretic peptides, fenoldopam, dopamine and N-acetylcysteine in the prevention of AKI remains uncertain [23]. Therefore, more effective strategies and drugs are needed to prevent and treat AKI.

Limitations

There are several limitations in this study. First, this is a retrospective study, and more high quality studies are needed to confirm our findings. Second, the sample size of participants was relatively small, which will likely have reduced the statistical power for data analysis. Third, all patients enrolled in our study was operated by several different surgeons, which may influence the result. Finally, patients were followed by short-term, and more long-term follow-up data and hard endpoints, such as mortality are needed to explore the relationship between hyperlactatemia and clinical outcome. Therefore, large studies with longer follow-up and careful matching of key clinical variables are needed.

Conclusions

We found that hyperlactatemiaat 6 hours after type-A aortic dissection surgery was related to postoperative adverse events, including AKI and CRRT. A threshold of 5.05 mmol/L at 6 hours after surgery is independently associated with the risk of AKI. Additionally, a lactate level of 7.0 mmol/L identifies patients needed CRRT. Longer follow-up studies are needed to confirm our findings.

Disclosure of conflict of interest

None.

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References

[1] Macedo E, Mehta RL. Preventing Acute Kidney Injury. Crit Care Clin 2015; 31: 773-784.

[2] Kandler K, Jensen ME, Nilsson JC, Møller CH, Steinbrüchel DA. Acute kidney injury is independently associated with higher mortality after cardiac surgery. J Cardiothorac Vasc Anesth 2014; 28: 1448-1152.

[3] Rydén L, Ahnve S, Bell M, Hammar N, Ivert T, Sartipy U, Holzmann MJ. Acute kidney injury after coronary artery bypass grafting and long-term risk of myocardial infarction and death. Int J Cardiol 2014; 172: 190-195.

[4] Pickering JW, James MT, Palmer SC. Acute kidney injury and prognosis after cardiopulmonary bypass: a meta-analysis of cohort studies. Am J Kidney Dis 2014; 65: 283-293.

[5] Okorie ON, Dellinger P. Lactate: biomarker and potential therapeutic target. Crit Care Clin 2011; 27: 299-326.

[6] Zhang Z, Ni H. Normalized lactate load is associated with development of acute kidney injury in patients who underwent cardiopulmonary bypass surgery. PLoS One 2015; 10: e0120466.

[7] Roh GU, Lee JW, Nam SB, Lee J, Choi JR, Shim YH. Incidence and risk factors of acute kidney injury after thoracic aortic surgery for acute

dissection. Ann Thorac Surg 2012; 94: 766-771.
[8] Lansman SL, McCullough JN, Nguyen KH, Spielvogel D, Klein JJ, Galla JD, Ergin MA, Griepp RB. Subtypes of acute aortic dissection. Ann Thorac Surg 1999; 67: 1975-1978.
[9] Sun L, Qi R, Zhu J, Liu Y, Chang Q, Zheng J. Repair of Acute Type A Dissection: Our Experiences and Results. Ann Thora Surg 2011; 91: 1147-1152.
[10] Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012; 2: 1-138.
[11] Zhang Z, Ni H. Normalized lactate load is associated with development of acute kidney injury in patients who underwent cardiopulmonary bypass surgery. PLoS One 2015; 10: e0120466.
[12] Lopez-Delgado JC, Esteve F, Javierre C, Torrado H, Rodriguez-Castro D, Carrio ML, Farrero E, Skaltsa K, Mañez R, Ventura JL. Evaluation of Serial Arterial Lactate Levels as a Predictor of Hospital and Long-Term Mortality in Patients After Cardiac Surgery. J Cardiothorac Vasc Anesth 2015; 29: 1441-1453.
[13] Thakar CV, Arrigain S, Worley S, Yared JP, Paganini EP. A clinical score to predict acute renal failure after cardiac surgery. J Am Soc Nephrol 2015; 16: 162-168.
[14] Mehta RH, Grab JD, O'Brien SM, Bridges CR, Gammie JS, Haan CK, Ferguson TB, Peterson ED; Society of Thoracic Surgeons National Cardiac Surgery Database Investigators. Bedside tool for predicting the risk of postoperative dialysis in patients undergoing cardiac surgery. Circulation 2006; 114: 2208-2216.
[15] Wijeysundera DN, Karkouti K, Dupuis JY, Rao V, Chan CT, Granton JT, Beattie WS. Derivation and validation of a simplified predictive index for renal replacement therapy after cardiac surgery. JAMA 2007; 297: 1801-1809.
[16] Maarslet L, Møller MB, Dall R, Hjortholm K, Ravn H. Lactate levels predict mortality and need for peritoneal dialysis in children undergoing congenital heart surgery. Acta Anaesthesiol Scand 2012; 56: 459-464.
[17] Obermüller N, Geiger H, Weipert C, Urbschat A. Current developments in early diagnosis of acute kidney injury. Int Urol Nephrol 2014; 46: 1-7.
[18] Ho J, Tangri N, Komenda P,Kaushal A, Sood M, Brar R, Gill K, Walker S, MacDonald K, Hiebert BM, Arora RC, Rigatto C. Urinary, Plasma, and Serum Biomarkers' Utility for Predicting Acute Kidney Injury Associated With Cardiac Surgery in Adults: A Meta-analysis. Am J Kidney Dis 2015; 66: 993-1005.
[19] Heringlake M, Charitos EI, Gatz N, Käbler JH, Beilharz A, Holz D, Schön J, Paarmann H, Petersen M, Hanke T. Growth differentiation factor 15: a novel risk marker adjunct to the EuroSCORE for risk stratification in cardiac surgery patients. J Am Coll Cardiol 2013; 61: 672-681.
[20] Guenancia C, Kahli A, Laurent G, Hachet O, Malapert G, Grosjean S, Girard C, Vergely C, Bouchot O. Pre-operative growth differentiation factor 15 as a novel biomarker of acute kidney injury after cardiac bypass surgery. Int J Cardiol 2015; 197: 66-71.
[21] Zacharias M, Mugawar M, Herbison GP, Walker RJ, Hovhannisyan K, Sivalingam P, Conlon NP. Interventions for protecting renal function in the perioperative period. Cochrane Database Syst Rev 2013; 9: CD003590.
[22] Society of Thoracic Surgeons Blood Conservation Guideline Task Force, Ferraris VA, Brown JR, Despotis GJ, Hammon JW, Reece TB, Saha SP, Song HK, Clough ER; Society of Cardiovascular Anesthesiologists Special Task Force on Blood Transfusion, Shore-Lesserson LJ, Goodnough LT, Mazer CD, Shander A, Stafford-Smith M, Waters J; International Consortium for Evidence Based Perfusion, Baker RA, Dickinson TA, FitzGerald DJ, Likosky DS, Shann KG. 2011 update to the Society of Thoracic Surgeons and the Society of Cardiovascular Anesthesiologists blood conservation clinical practice guidelines. Ann Thorac Surg 2011; 91: 944-982.
[23] Alsabbagh MM, Asmar A, Ejaz NI, Aiyer RK, Kambhampati G, Ejaz AA. Update on clinical trials for the prevention of acute kidney injury in patients undergoing cardiac surgery. Am J Surg 2013; 206: 86-95.