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The correlation of post-operative acute kidney injury and perioperative anaemia in patients undergoing cardiac surgery with cardiopulmonary bypass

Tautvydas Baranauskas[1*],

Agnė Kaunienė[2],

Milda Švagždienė[3],

Edmundas Širvinskas[3],

Tadas Lenkutis[4]

[1] *Faculty of Medicine, Lithuanian University of Health Sciences, Kaunas, Lithuania*

[2] *Department of Internal Medicine, Lithuanian University of Health Sciences, Kaunas, Lithuania*

[3] *Institute of Cardiology, Lithuanian University of Health Sciences, Kaunas, Lithuania*

[4] *Clinic of Anaesthesiology, Lithuanian University of Health Sciences, Kaunas, Lithuania*

Background and objective. Acute kidney injury (AKI) following cardiac surgery with cardiopulmonary bypass (CPB) is polyethiological clinical syndrome. During CPB haemodilution develops, which is useful in reducing the risk of thrombosis; however, haemodilutional anaemia decreases oxygen transfer and provokes tissue hypoxia, which can lead to acute organ damage. The aim of the study was to find out the impact of perioperative anaemia on AKI after cardiac surgery with CPB.

Materials and methods. This prospective study included 58 adult patients undergoing elective cardiac surgery with CPB, without any preoperative chronic renal disease or any systemic autoimmune disease. Serum concentrations of NGAL had been tested before the surgery, 2 hours, 6 hours, and one day after the surgery. Perioperative anaemia was assessed according to the Ht value before the surgery, the Ht value during CPB, and immediately after the surgery.

Results. The rate of haemodilutional anaemia is 77.59% in this study. The average of serum NGAL concentration before CPB was 63.95 ± 33.25 ng/mL and it was significantly lower than the average concentration 2 hours after the surgery, 6 hours after the surgery and one day after the surgery (respectively 148.51 ± 62.39, 119.44 ± 55, 128.70 ± 59.04 ng/mL, $p < 0.05$). AKI developed in 46.55% of the patients. A significant positive reasonable correlation between the development of perioperative anaemia and AKI was determined ($r = 0.50$, $p < 0.05$).

Conclusions. Post-operative AKI after cardiac surgery with CPB has a moderate positive correlation with perioperative haemodilutional anaemia. A longer CPB time and aortic cross-clamping time were found to be the risk factors for the development of AKI.

Keywords: acute kidney injury, cardiopulmonary bypass, haemodilutional anaemia, NGAL

* Correspondence to: Tautvydas Baranauskas, Faculty of Medicine, Lithuanian University of Health Sciences, 2 Eivenių St., Kaunas 50009, Lithuania. Email: tautvydas.baranauskas@gmail.com

INTRODUCTION

Acute kidney injury (AKI) after cardiac surgery with cardiopulmonary bypass (CPB) is associated with an increased risk for chronic kidney diseases, an increased incidence of myocardial infarction, stroke, respiratory failure, serious infections, blood product transfusions, and mortality (1). The development of AKI is polyethiologic. During CPB haemodilution develops, which is a risk factor for AKI. Throughout cardiac surgery haemodilution is useful in reducing the risk of thrombosis, however, haemodilutional anaemia decreases oxygen transfer and provokes tissue hypoxia, which can lead to organ ischemia and acute damage (2). It was noticed that it was the outer layer of renal cortex that suffered from early injury and hence ischemic AKI developed (3).

According to the severity, AKI is classified into (1) severe (serum creatinine level increase >3 times of the initial value), requiring kidney replacement therapy, (2) moderate (serum creatinine level 2–3 times higher than the initial value), and (3) mild (serum creatinine level increases up to 1.5 times from the initial value) postoperative AKI. However, serum creatinine concentration is not an accurate and early indicator of the kidney condition, thus it is often suggested to use newer biomarkers instead, which allow earlier diagnosis and detect injuries of a minor degree (4). AKI can be diagnosed by serum level of neutrophil gelatinase-associated lipocalin (NGAL), which is a biomarker reflecting early kidney injury in the distal nephrons (5). It reflects kidney injury caused by ischemia or nephrotoxins (6). The increase of serum NGAL concentration under 150 nmol/L is considered a normal reaction to intervention, stress, and other factors. The level of serum NGAL over 150 nmol/L shows minimal kidney injury. However, when it exceeds 250 nmol/L, it is considered a serious injury. Biological action of NGAL relies on specific receptors found in the brain or other tissues. Most often the interface of receptors is observed in kidney distal tubule cells on the brush layer (3). Other studies have shown that the values of serum NGAL 2 hours after the surgery have a significant correlation with AKI (7). During cardiac surgery, it is essential to assess haematocrit (Ht), though its minimum value, which should be maintained during CPB, is not defined due to the lack of clinical studies (9, 10). A decrease in Ht is associated with a higher risk of complications, mortality, and higher rates of return to CPB (8). Each hospital has its own regulations on acceptable values of haematocrit and indications for transfusion during CPB. Some authors recommend to keep haematocrit >25% while others suggest higher limits (>30%). A study with animals showed 19% to be the critical point. The goals of the present study were: (1) to detect the rate of AKI and the rate of perioperative anaemia in patients undergoing elective cardiac surgery with CPB; (2) to find the relationship between intraoperative haemodilutional, preoperative, and postoperative anaemia, and the rate of postoperative AKI; (3) to determine the impact of various perioperative factors for the development of AKI.

MATERIALS AND METHODS

A prospective study was conducted from November to December 2015 at the Department of Cardiothoracic and Vascular Surgery, of the Kauno klinikos Hospital of the Lithuanian University of Health Sciences. The study was approved by Kaunas Ethics Commission (protocol No. BEC-MF-146) and research was carried following the Declaration of Helsinki II.

The study included 58 patients undergoing elective cardiac surgery with CPB, without chronic renal disease and any systemic autoimmune disease preoperatively, which could have been the reason for any preoperative kidney injuries.

All patients received a standardized anaesthetic and CPB management. Patients were pre-medicated with midazolam or diazepam and morphine. Anaesthesia was induced with propofol, fentanyl, and rocuronium and maintained with sevoflurane, fentanyl, and rocuronium. All patients underwent median sternotomy, the aorta was cross-clamped, and anterograde cold crystalloid cardioplegia (4°C St. Thomas' hospital solution) was applied for myocardium protection. The CPB machine was primed with 1.5 L of crystalloid solution containing 10,000 units of heparine. A roller-pump with a membrane oxygenator (Dideco D703, Mirandola, Italy) and a venous reservoir were used. The non-pulsatile pump flow rates were maintained between 2.4 and 2.6 L/min/m^2 body surface area.

The following risk factors have been assessed: diabetes mellitus (DM), the use of diuretics before and after the surgery.

Perioperative anaemia was assessed according to Ht values before the surgery, Ht value during CPB, and immediately after the surgery. Ht < 25% was considered as haemodilutional anaemia (11).

AKI was assessed according to the serum concentration of NGAL, which had been tested before surgery, 2 hours, 6 hours, and one day after surgery. Patients were included into the group with AKI when at least one postoperative value of NGAL was >150 ng/mL. Serum creatinine concentration was also assessed before and after surgery within the prior 7 days in the cardiac surgery department. According to KDIGO criteria, AKI was diagnosed when creatinine increased at least by 50% during the first seven days after surgery (12).

Statistical analysis was performed with the Statistical Package for the Social Science (SPSS) v.23. Quantitative variables were described as a mean and standard deviation (SD). The Kolmogorov-Smirnov test was used to determine the distribution of quantitative data. When the distribution of variables was normal, Student's *t*-test was used to compare the quantitative sizes of two independent samples, and the non-parametric Mann-Whitney U test was used to compare non-normally distributed variables. Qualitative data were compared between the two groups by using chi-square (χ^2) tests. Linear dependence between variables was evaluated by using correlation coefficients. The Pearson's or Spearman's correlation coefficient was used taking into account the distribution of variables. The difference was considered statistically significant when $p < 0.05$.

RESULTS

The study included 58 patients assigned to ASA class III. Their demographic and surgery-related parameters are presented in Table 1. Preoperatively, the values of Ht, serum creatinine, and serum NGAL were within normal ranges in all the patients. Moreover, creatinine and NGAL before the surgery were not significantly different in patients who developed AKI postoperatively and those who did not.

The average concentration of serum NGAL before CPB was 64.38 ± 33.55 ng/mL and it was significantly lower than the average concentration 2 hours after surgery, 6 hours after surgery, and one day after surgery (respectively, 150.37 ± 62.36, 119.57 ± 54.64, 129.22 ± 58.74 ng/mL, $p < 0.01$).

When evaluating NGAL values, in 46.55% of the patients a significant increase of NGAL concentration from the initial concentration was observed. Mild AKI occurred in 32.76% patients (NGAL serum level 150–250 ng/mL), and in 13.79% of the patients serious AKI was observed (NGAL serum level >250 ng/mL). The average serum creatinine level after surgery was significantly higher than that before surgery (respectively 106.53 ± 38.63 μmol/L vs 95.09 ± 28.18 μmol/L, $p < 0.01$). According to the changes in the creatinine level after surgery, AKI was detected in 12.07% of the patients. Renal replacement therapy was needed in two patients (3.33%) after surgery.

During surgery, 27.59% of the patients had red blood cell transfusions. Of those who developed AKI, 33.33% underwent a transfusion. In comparison with the patients who did not develop AKI, only 22.58% underwent a transfusion. Although those patients, who had transfusions, experienced AKI more often, there was no significant correlation between AKI and transfusion.

The average haematocrit before CPB was 37.16 ± 6.52%. The average haematocrit during CPB was significantly lower than that after surgery (respectively, 22.66 ± 4.05% and 27.36 ± 4.32%, $p < 0.01$). During the perioperative period, anaemia was observed in 77.59% of the patients (Ht < 25%) (Table 2). A significant positive correlation between the perioperative anaemia and AKI was determined. In addition, intraoperative (Ht < 25%) anaemia occurred in 75.86% of the patients and had a moderate significant correlation with postoperative AKI. Postoperative (Ht < 25%) anaemia separately did not significantly the development of AKI.

The duration of surgery, a red blood cell (RBC) transfusion, the use of diuretics before surgery and after surgery in the ICU, fluid balance, and diabetes mellitus did not show a significant correlation with the development of AKI. However, the duration of aortic cross-clamping and the CPB duration had a moderate correlation with the development of AKI (Table 3). However, in the group of the patients with AKI, the duration of aortic cross-clamping and the duration of CPB were not significantly higher in comparison with the patients without AKI (Table 1).

Table 1. Patients' demographic and clinical characteristics (n = 58)

	Patients with postoperative AKI (n = 27)	**Patients without postoperative AKI (n = 31)**	***p* value**
Anthropometric parameters			
Age (years)	69.19 ± 10.09	64.68 ± 15.20	0.21
Sex (male, %)	70.37	51.61	0.15
Surgery parameters			
Duration of surgery (min)	204.26 ± 51.75	199.52 ± 39.36	0.62
	Patients with postoperative AKI (n = 27)	Patients without postoperative AKI (n = 31)	p value
Duration of CPB (min)	99.63 ± 26.57	80.06 ± 27.50	0.44
Duration of aortic cross-clamping (min)	56.70 ± 19.82	44.29 ± 18.74	0.35
Surgery type			
CABG, %	70.37	58.06	0.33
Valve repair, %	29.63	35.48	
Combined, %	0	6.45	
Perfusion pressure (mmHg)	67.56 ± 5.60	68.00 ± 5.24	0.61
Haematocrit			
Before surgery, %	37.70 ± 6.11	36.68 ± 6.92	0.38
Within surgery (lowest value), %	22.67 ± 4.17	22.65 ± 4.01	0.37
After surgery, %	27.63 ± 4.01	27.13 ± 4.63	0.53
Blood components			
RBC transfusion, %	33.33	22.58	0.36
Blood plasma, %	7.41	6.45	0.89
Diuretics			
Before surgery, %	37.04	22.58	0.23
After surgery, %	74.07	64.52	0.50
ICU time			
Duration in ICU (days)	4.44 ± 6.67	3.23 ± 1.33	0.12
Fluid balance 1st day after surgery, mL	462.22 ± 1344.54	287.42 ± 1063.00	0.43
Diabetes mellitus, %	11.11	12.90	0.83
Haemoglobin preoperative, g/L	131.44 ± 18.04	132.97 ± 13.12	0.28
Creatinine preoperative, μmol/L	107.52 ± 34.00	84.26 ± 15.70	0.42
NGAL preoperative, ng/mL	87.73 ± 31.81	44.05 ± 18.54	0.40
Fluid balance during surgery, mL	2073.52 ± 786.88	2002.26 ± 647.60	0.55

Table 2. Correlations between AKI and pre-operative, intra-operative, post-operative, and perioperative anaemias

	Patients (n = 58)	***p* value, *r* value**
Intraoperative anaemia, n (%)	44 (75.86)	$p < 0.01$, $R = 0.45$
Postoperative anaemia, n (%)	16 (27.59)	$p = 0.96$, $R = 0.01$
Perioperative anaemia, n (%)	45 (77.59)	$p < 0.01$, $R = 0.50$

Table 3. The correlation of baseline characteristics and post-operative AKI

Characteristics	Patients (n = 58)	Correlation with AKI
Duration of surgery (min)	202 ± 45	$p = 0.99$, $r < 0.01$
Duration of CPB (min)	89 ± 29	$p < 0.01$, $r = 0.38$
Duration of aortic cross-clamping (min)	50 ± 20	$p = 0.02$, $r = 0.30$
RBC transfusion n (%)	16 (27.59)	$p = 0.37$, $r = 0.12$
Usage of diuretics n (%)		
Before surgery	17 (29.31)	$p = 0.24$, $r = 0.16$
After surgery	40 (68.97)	$p = 0.67$, $r = 0.07$
Fluid balance after surgery (mL)	2035 ± 710	$p = 0.84$, $r = 0.03$
Diabetes mellitus n (%)	7 (12.07)	$p = 0.84$, $r = -0.03$

DISCUSSION

The aim of this study was to find out the effect of perioperative anaemia on AKI after cardiac surgery with CPB. The data regarding the minimum Ht value presented in the scientific literature are contradictory. It has been decided to regard Ht value as harmful when it is below 25%. In the study by Hajjar et al., it is proposed to regard 24% as the minimum Ht value. However, there are sources suggesting that 30% should be viewed as the minimum value, which may require a transfusion (9, 13). In most publications, this value of haematocrit is associated with tissue hypoxia that affects target organs – the lungs, the kidneys and the heart (13). Usually, even the affected renal function recovers to the normal. However, there are data confirming that AKI is a significant factor for the development of the chronic kidney disease (14). In the study by Pannu et al., it was noticed that approximately 11% of AKI result in the chronic kidney disease (15).

Usually the evaluation of the kidney function is based on serum creatinine levels and diuresis. We used NGAL to assess the AKI. It is produced in the distal nephron and its synthesis is up-regulated in response to kidney injury. NGAL acts to bind iron-chelating proteins secreted by bacteria and thus has an anti-inflammatory and bacteriostatic effect (30). NGAL is superior to creatinine because, unlike creatinine, it gives no value to age, sex, muscle mass, metabolic disorders, and dehydration. NGAL, opposite to creatinine or oliguria, may show whether the emerging kidney injury is reversible or not (14). It is important that the serum creatinine level remains normal when the kidney function is affected slightly (5). The most visible drawback of serum creatinine is that it is distributed throughout the whole body; therefore, such changes as synthesis or elimination in the blood plasma are noticeable only after two days. Moreover, the decline of the creatinine concentration in serum after the cardiac surgery due to haemodilution is observed, which further complicates the diagnostics of AKI (16). On the other hand, decreased GFR may be masked by loop diuretics, and, as a result, AKI may not be diagnosed (17). So, NGAL contributes to the earlier diagnosis of AKI, thus, enabling the application of early treatment and prophylaxis. The definition of AKI is still not standardized (18). This study revealed that the frequency of AKI is higher when assessing changes in serum NGAL concentration than that assessed by the changes in the concentration of serum creatinine. However, NGAL is still not routinely used in clinical practice because overall results are inconsistent, with variations in sample collection protocols, procedure type, and the patient population.

A low Hb level of reduces oxygen transfer to the tissues. The emerging hypoxia plays a critical role in the development of AKI. Therefore, preoperative haemoglobin should be optimized with iron supplementation (19). This study observed that AKI after cardiac surgery is associated with moderate anaemia. The study of Santo et al.

suggests that anaemia is independently associated with AKI. Other references also found that the haemodilution of CPB duration increases the risk of developing AKI (20).

This study did not reveal that blood transfusion increases the possibility of developing AKI. Recently, major studies found transfusion to be an independent predictor of AKI (21). But numerous sources maintain that dilution anaemia is the main cause of AKI and the transfusions performed are only the consequence (20).

In the study conducted by Fakhari et al., it was found that the rate of AKI after cardiac surgery can be reduced by using furosemide during surgery and in early postoperative period (22). In our study, 36.21% of patients received diuretic therapy during the early postoperative period in the ICU, but it did not influence the rate of AKI. It could be explained by the dose and the choice of diuretic, and more intense infusion therapy during surgery (23). However, further multi-centre studies with larger number of patients and different doses of furosemide are required to confirm these results.

Diabetes mellitus is an independent risk factor for AKI. Patients with the disease suffer from impaired vasculature, therefore there is a higher risk of kidney hypoperfusion and ischemia periods, which may cause AKI (24). In patients with DM, AKI is often underdiagnosed after heart surgery (25, 26). This study did not find any relation between DM and AKI, possibly due to a relatively small sample size.

Surgical factors such as CPB duration and aortic cross-clamping time may help to assess the risk of AKI (27). The longer CPB, the more intense haemolysis is. Free haemoglobin released from damaged erythrocites is nephrotoxic. In the study by Yunus it was found that CPB duration of 71–140 min and >140 min increases the risk of AKI by an OR of 4.76 and 6.30, respectively (28). It was also observed that the patients who had no history of kidney diseases prior to surgery but developed it after it, had a longer CPB duration, a lower CPB perfusion flow, and CPB perfusion pressure was <60 mmHg (29). In our study, surgery duration and mean perfusion pressure did not influence the rate of AKI. Aortic cross-clamping time and CPB duration were not significantly higher in patients with AKI, but both factors moderately correlated with the development of AKI.

Our data indicates that exposure to haemodilutional anaemia during surgery was associated with an increased risk of AKI.

There are several limitations in this study. The small sample size causes concerns for type II error and the over signification of individual patients in the analysis. Furthermore, the high incidence of anaemia leaves only 22.41% patients without perioperative anaemia. These small subgroup sample sizes limit the utility of the statistical comparisons made between them.

CONCLUSIONS

The rate of perioperative anaemia is 77.59% in our study. AKI developed in 46.55% of the patients. Post-operative AKI after cardiac surgery with CPB has a moderate positive correlation with perioperative haemodilutional anaemia. Longer CPB time and aortic cross-clamping time were found to be the risk factors for the development of AKI.

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References

1. Gude D, Jha R. Acute kidney injury following cardiac surgery. Ann Card Anaesth. 2012; 15: 279–86.
2. Hanumantha RM, Aloka S, Muralidhar A. Blood transfusion practices in cardiac anaesthesia. Indian J Anaesth. 2014; 58(5): 616–21.
3. David PB, Yoder MC. Renal endothelial dysfunction in acute kidney ischemia reperfusion injury. Cardiovasc Hematol Disord Drug Targets. 2014; 14(1): 3–14.
4. De Loor J, Herck I, Francois K, Van Wesemael A, Nuytinck L, Meyer E, et al. Diagnosis of cardiac surgery-associated acute kidney injury: differential roles of creatinine, chitinase-3-like protein 1 and neutrophil gelatinase-associated lipocalin: a prospective cohort study. Ann Intensive Care. 2017; 7(1): 24.
5. Devarajan P. Neutrophil gelatinase-associated lipocalin (NGAL): a new marker of kidney disease. Scandinavian Journal of Clinical and Laboratory Investigation. Supplementum. 2008; 241: 89–94.

6. Aldo C, Claudio G, Antonio F, Claudio R. Neutrophil gelatinase-associated lipocalin (NGAL) as biomarker of acute kidney injury: a review of the laboratory characteristics and clinical evidences. Clin Chem Lab Med. 2012; 50(9): 1505–17.
7. Mishra J, Dent C, Tarabishi R, Mitsnefes MM, Ma Q, Kelly C, et al. Neutrophil gelatinase-associated lipocalin (NGAL) as a biomarker for acute renal injury after cardiac surgery. Lancet. 1999; 365(9466): 1231–8.
8. DeFoe GR, Ross CS, Olmstead EM, Surgenor SD, Fillinger MP, Groom RC, et al. Lowest hematocrit on bypass and adverse outcomes associated with coronary artery bypass grafting. Ann Thorac Surg. 2001; 71(3): 769–76.
9. Carson JL, Patel MS. Red blood cell transfusion thresholds: can we go even lower? Transfusion. 2014; 54(10): 2593–4.
10. Liam BL, Plochl W, Cook DJ, Orszulak TA, Daly RC. Hemodilution and whole body oxygen balance during normothermic cardiopulmonary bypass in dogs. J Thorac Cardiovasc Surg. 1998; 115(5): 1203–8.
11. Swaminathan M, Phillips-Bute BG, Conlon PJ, Smith PK, Newman MF, Stafford-Smith M. The association of lowest hematocrit during cardiopulmonary bypass with acute renal injury after coronary artery bypass surgery. Ann Thorac Surg. 2003; 76(3): 784–91, discussion 792.
12. KDIGO Clinical Practice Guideline for Acute Kidney Injury. 2012; 2(1).
13. Hajjar L, Vincent J. Transfusion requirements after cardiac surgery. JAMA. 2010; 304(14): 1559–67.
14. Vives M, Wijeysundera D, Marczin N, Monedero P, Rao V. Cardiac surgery-associated acute kidney injury. Interact Cardiovasc Thorac Surg. 2014; 18(5): 637–45.
15. Pannu N, Wiebe N, Tonelli M. Prophylaxis Strategies for Contrast-Induced Nephropathy. JAMA. 2006; 295(23): 2765–79.
16. Mao H, Katz N, Ariyanon W, Blanca-Martos L, Adýbelli Z, Giuliani A, et al. Cardiac surgery-associated acute kidney injury. Cardiorenal Med. 2013; 3(3): 178–99.
17. Vandenberghe W, De Loor J, Hoste EAJ. Diagnosis of cardiac surgery-associated acute kidney injury from functional to damage biomarkers. Curr Opin Anaesthesiol. 2017; 30(1): 66–75.
18. Kidher E, Harling L, Ashrafian H, Naase H, Chukwuemeka A, Anderson J, et al. Pulse wave velocity and neutrophil gelatinase-associated lipocalin as predictors of acute kidney injury following aortic valve replacement. J Cardiothorac Surg. 2014; 9(1): 89.
19. Schetz M, Bove T, Morelli A, Mankad S, Ronco C, Kellum JA. Prevention of cardiac surgery-associated acute kidney injury. Int J Artif Organs. 2008; 31(2): 179–89.
20. Ng RRG, Chew STH, Liu W, Shen L, Ti LK, Lim C, et al. Identification of modifiable risk factors for acute kidney injury after coronary artery bypass graft surgery in an Asian population. J Thorac Cardiovasc Surg. 2014; 147(4): 1356–61.
21. Murphy GJ, Reeves BC, Rogers CA, Rizvi SIA, Culliford L, Angelini GD. Increased mortality, postoperative morbidity, and cost after red blood cell transfusion in patients having cardiac surgery. Circulation. 2007; 116(22): 2544–52.
22. Fakhari S, Mirzaei BF, Bilehjani E, Abolhasani S, Mirinazhad M, Naghipour B. Prophylactic furosemide infusion decreasing early major postoperative renal dysfunction in on-pump adult cardiac surgery: a randomized clinical trial. Res Reports Urol. 2017; 9: 5–13.
23. Kunt AT, Akgün S, Atalan N, Bitir N, Arsan S. Furosemide infusion prevents the requirement of renal replacement therapy after cardiac surgery. Anadolu Kardiyol Derg. 2009; 9(6): 499–504.
24. Bienholz A, Wilde B, Kribben A. From the nephrologist's point of view: diversity of causes and clinical features of acute kidney injury. Clin Kidney J. 2015; 8(4): 405–14.
25. Kalimeris K, Nikolakopoulos N, Riga M, Christodoulaki K, Moulakakis KG, Dima C, et al. Mannitol and renal dysfunction after endovascular aortic aneurysm repair procedures: a randomized trial. J Cardiothorac Vasc Anesth. 2014; 28(4): 954–9.
26. Brinkman R, HayGlass KT, Mutch WAC, Funk DJ. Acute kidney injury in patients undergoing open abdominal aortic aneurysm repair: a pilot observational trial. J Cardiothorac Vasc Anesth. 2015; 29(5): 1212–9.
27. Fuhrman DY, Kellum JA, Epidemiology and pathophysiology of cardiac surgery-associated acute kidney injury. Curr Opin Anaesthesiol. 2017; 30(1): 65.
28. Karim HMR, Yunus M, Saikia MK, Kalita JP, Mandal M. Incidence and progression of cardiac surgery-associated acute kidney injury and its relationship with bypass and cross clamp time. Ann Card Anaesth. 2017; 20(1): 22–7.

29. Fischer UM, Weissenberger WK, Warters RD, Geissler HJ, Allen SJ, Mehlhorn U. Impact of cardiopulmonary bypass management on postcardiac surgery renal function. Perfusion. 2002; 17(6): 401–6.
30. Chakraborty S, Kaur S, Guha S, Batra SK. The multifaceted roles of neutrophil gelatinase associated lipocalin (NGAL) in inflammation and cancer. Biochim Biophys Acta – Rev Cancer. 2012; 1826(1): 129–69.

Tautvydas Baranauskas, Agnė Kaunienė, Milda Švagždienė, Edmundas Širvinskas, Tadas Lenkutis

PACIENTŲ PO ŠIRDIES OPERACIJŲ DIRBTINĖS KRAUJO APYTAKOS SĄLYGOMIS POOPERACINIO ŪMINIO INKSTŲ PAŽEIDIMO IR PERIOPERACINĖS ANEMIJOS SĄSAJA

Santrauka

Įvadas. Ūminis inkstų pažeidimas (ŪIP) po širdies operacijų dirbtinės kraujo apytakos sąlygomis yra polietiologinis klinikinis sindromas, kurio vienas iš etiologinių veiksnių yra dirbtinė kraujo apytaka (DKA). Susidaro praskiedimo anemija, kuri kardiovaskulinių operacijų metu yra naudinga, nes mažina trombozės riziką, tačiau mažėja deguonies pernaša į audinius ir tai nulemia ūmius audinių bei organų pažeidimus. Šio tyrimo tikslas – išsiaiškinti perioperacinės anemijos įtaką ŪIP išsivystymui atliekant širdies operacijas DKA sąlygomis.

Medžiaga ir metodika. Į prospektyvinį tyrimą buvo įtraukti 58 pacientai, neturintys sisteminių autoimuninių bei inkstų ligų ir kuriems planine tvarka buvo atliekamos širdies operacijos DKA sąlygomis. Perioperacinė anemija vertinta atsižvelgiant į hematokrito reikšmes prieš operaciją, operacijos metu ir po operacijos. Inkstų pažeidimas vertintas pagal *biomarkerio* NGAL koncentraciją kraujo plazmoje, kuri buvo tirta prieš operaciją, po operacijos praėjus 2 val., 6 val. ir vienai parai.

Rezultatai. Tyrime nustatyta 77,59 % praskiedimo anemija. Serumo NGAL koncentracijos vidurkis prieš operaciją buvo 63,95 ± 33,25 ng/mL. NGAL koncentracijos vidurkis po operacijos praėjus 2 val., 6 val. ir vienai parai statistiškai reikšmingai didėjo (atitinkamai 148,51 ± 62,39, 119,44 ± 55, 128,70 ± 59,04 ng/mL, $p < 0,05$). ŪIP pasireiškė 46,55 % pacientų. Tarp perioperacinės anemijos ir ŪIP pasireiškimo nustatyta statistiškai reikšminga teigiama koreliacija ($r = 0,50$, $p < 0,05$).

Išvados. Atliekant širdies operacijas DKA sąlygomis perioperacinė anemija vidutiniškai koreliuoja su pooperaciniu ŪIP. Ilgesnė DKA trukmė bei aortos užspaudimo laikas buvo nustatyti kaip pooperacinio ŪIP išsivystymo rizikos veiksniai.

Raktažodžiai: ūminis inkstų pažeidimas, dirbtinė kraujo apytaka, praskiedimo anemija, NGAL