Perioperative Anaemia is a Risk Factor of Acute Kidney Injury in Patients Undergoing Partial Nephrectomy

Shun Zhang
Shanghai Jiaotong University School of Medicine Xinhua Hospital
Huangqi Duan
Shanghai Jiaotong University School of Medicine Xinhua Hospital
Yu Wu
Shanghai Jiaotong University School of Medicine Xinhua Hospital
Weimin Xia
Shanghai Jiaotong University School of Medicine Xinhua Hospital
Subo Qian
Shanghai Jiaotong University School of Medicine Xinhua Hospital
Haibo Shen (shenhaibo@xinhuamed.com.cn)
Department of Urology, Xinhua Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai 200092, China https://orcid.org/0000-0002-1183-6570

Research

Keywords: acute kidney injury (AKI), anaemia, haematocrit (Ht), neutrophil gelatinase-associated lipocalin (NGAL), partial nephrectomy (PN)

Posted Date: October 22nd, 2020

DOI: https://doi.org/10.21203/rs.3.rs-94939/v1

Abstract

Background Acute kidney injury (AKI) secondary to partial nephrectomy (PN) is a challenging clinical issue. During PN anaemia develops due to haemodilution and blood loss, which decreases oxygen transfer and provokes tissue hypoxia, leading to postoperative adverse outcomes. The aim of this study was to investigate the impact of perioperative anaemia on postoperative AKI after PN.

Methods This retrospective cohort study included 68 adult patients undergoing PN for a single nonmetastatic renal tumor. Detailed clinical information was systematically reviewed and analyzed. Serum concentrations of neutrophil gelatinase-associated lipocalin (NGAL) and creatinine (sCr) were tested before, 2 hours and 1 day after PN surgery. Perioperative anaemia was assessed according to haematocrit (Ht) value at the same timepoint. Association between perioperative anaemia and postoperative AKI were explored by logistic regression analyses and pearson correlation analysis.

Results The rate of perioperative anaemia in patients undergoing PN was 35.3% when the criteria of Ht < 30% was adopted. AKI developed in 32.4% of the patients when KDIGO criteria was applicated and in 51.5% of the patients when the criteria of serum NGAL >150 ng/mL was used. The incidence of AKI in patients with perioperative anaemia was higher than that in patients without anaemia. Univariate logistic regression analyses showed perioperative anaemia was a relevant factor of postoperative AKI in patients undergoing PN.

Conclusions Perioperative anaemia might be a risk factor of postoperative AKI after PN. But the detailed interrelation still needs to be verified by large-scale prospective studies.

Background

Partial nephrectomy (PN) is considered the reference standard for the management of T1a renal tumors and its indications have recently been expanded to include T1b/T2[1, 2]. A primary goal of PN is to preserve as much renal function as possible. However, it requires excision of functioning nephrons adjacent to the tumor and reconstruction, which will lead to focal devascularization[3]. Besides, traditional PN has been performed in the setting of hilar occlusion, which will induce ischemia-reperfusion injury (IRI). All of these will contribute to the loss of residual renal function and increase the risk of postoperative acute kidney injury (AKI).

AKI is a common critical illness with high risks of morbidity and mortality. Around 50% will eventually develop to chronic kidney disease (CKD), and 8.1% of these patients will progress to end-stage renal disease[3–5]. The development of AKI is due to polyetiological mechanisms. Recent studies reported that the model to predict eGFR after PN included renal ischemia time, age, presence of a solitary kidney, diabetes, hypertension, preoperative eGFR, preoperative proteinuria, surgical approach, time from surgery, etc.[6]. In addition, it has been reported that during cardiopulmonary bypass (CPB) haemodilution develops, which is useful in reducing the risk of thrombosis, but decreases oxygen transfer and provokes tissue hypoxia, which can lead to acute organ damage. Postoperative AKI after CPB has a moderate positive correlation with perioperative haemodilutional anaemia[4, 7, 8]. However, whether perioperative anaemia also an indicator of AKI after PN is still unknown. As a result, we conducted this retrospective observational study to investigate the association between perioperative anaemia and postoperative AKI in a cohort of patients underwent PN.

Methods

This was a retrospective cohort study conducted on patients who underwent PN for a single nonmetastatic renal tumor between April 2019 and June 2020 at our institution. Exclusion criteria were (1) anatomic or functional solitary kidney; (2) abnormal contralateral renal function; (3) abnormal preoperative sCr or serum NGAL; (4) preoperative anaemia; (5) venous tumor thrombus; (6) systemic autoimmune disease; (7) sepsis or urinary tract infection; (8) urolithiasis; (9) hydronephrosis; (10) diabetes mellitus or uncontrolled hypertension. A total of 68 patients were reviewed. Detailed information of age, gender, R.E.N.A.L. score, operation (laparoscopic or open surgical approaches, ischemia time, blood loss and blood transfusion) were recorded and analyzed.

Perioperative anaemia was assessed according to Ht values before PN and 2 hours and 1 day after the surgery. Some researchers suggested Ht < 25% as the standard of anaemia, while others recommended Ht < 30%[9, 10]. In general, it is agreed that tissue oxygen supply won't be influenced obviously when Ht > 30%. Therefore, the criteria of Ht < 30% was chosen in our research.

AKI was assessed according to the sCr and serum concentrations of NGAL, which was recorded in the baseline, 2 hours and 1 day after PN. NGAL is a lipocalin located in the distal renal tubular cells and can be rapidly released after renal insults. Increases in NGAL levels predict AKI 24 to 72 hours before diagnostic sCr increases and are of prognostic value in response to renal injury[11, 12]. In a meta-analysis of 2538 patients, Haase et al. found that the NGAL cutoff value for predicting AKI was > 150 ng/mL [13]. Therefore, serum NGAL > 150 ng/mL was used as the standard for postoperative AKI diagnosis in our research. Patients were classified into the AKI group when at least one postoperative serum NGAL was > 150 ng/mL or the sCr concentration increased at least by 50% or 26.5μ mol/L according to the KDIGO criteria [14].

Statistical analysis

Statistical comparisons were performed using the statistical software GraphPad Prism 5 (GraphPad Software, CA, USA) and PASW Statistics 18.0 (IBM Corp., NY, USA). Continuous variables were expressed as mean ± standard deviation (SD). The intergroup differences were tested using the Student's t test. Categorical variables were expressed as frequency (percentage), and Chi square test was used to test the intergroup differences. Pearson correlation analysis was used to analyze the interdependency between variables. Logistic regression analyses were used to investigate the relevant factors of postoperative AKI and perioperative anaemia. Statistical significance was defined as $p < 0.05$.

Results

Preoperatively, the values of Ht, sCr, and serum NGAL were all within normal ranges in all patients. Moreover, sCr and serum NGAL before PN were not statistically different in patients who developed AKI postoperatively and those who did not.

A total of 24/68 (35.29%) patients developed perioperative anaemia when the criteria of at least one of the postoperative Ht < 30% was adopted. Patients with or without perioperative anaemia were comparable in terms of demographic and biochemical characteristics, including age, gender, baseline Ht, baseline sCr, and baseline serum NGAL, as well as R.E.N.A.L. Score (Table 1).

Table 1
Characteristics of the study population

	Patients with perioperative anaemia (n = 24)	**Patients without perioperative anaemia (n = 44)**	**p value**
AKI (%)	14 (58.3)	8 (18.2)	0.0011**‡
Mean (SD) age (years)	62.9 (8.2)	62.7 (6.6)	0.8878†
Gender	-	-	0.3112‡
Male (%)	10 (41.7)	25 (56.8)	-
Female (%)	14 (58.3)	19 (43.2)	-
Mean (SD) Ht-BL (%)	38.02 (2.94)	36.76 (3.38)	0.1286†
Mean (SD) Ht-2 h (%)	30.64 (3.61)	34.48 (3.02)	<0.0001***†
Mean (SD) Ht-1d(%)	27.64 (2.81)	33.46 (3.13)	<0.0001***†
Mean (SD) sCr-BL (μmol/L)	67.8 (16.0)	65.8 (16.1)	0.6114†
Mean (SD) sCr-2 h (μmol/L)	69.8 (20.0)	66.0 (17.3)	0.4321†
Mean (SD) sCr-1d (μmol/L)	113.8 (42.6)	95.8 (35.2)	0.0661†
Mean (SD)NGAL-BL (ng/mL)	75.04 (22.2)	69.52 (22.6)	0.3364†
Mean (SD) NGAL-2 h (ng/mL)	197.4 (114.0)	135.5 (60.5)	0.0046**†
Mean (SD) NGAL-1d (ng/mL)	183.1 (90.4)	141.9 (96.9)	0.0906†

Values are in mean (SD) or n (%)

Abbreviations SD standard deviation, *sCr* serum creatinine, *NGAL* neutrophil gelatinase-associated lipocalin, *Ht* haematocrit

Ht-BL, sCr-BL, NGAL-BL represented Ht, sCr and serum NGAL before PN; Ht-2 h, sCr-2 h, NGAL-2 h represented Ht, sCr and serum NGAL 2 hours after PN; Ht-1d, sCr-1d, NGAL-1d represented Ht, sCr and serum NGAL 1 day after PN

† Student's t-test. ‡ Chi-square test.

*** meant p < 0.001, ** meant p < 0.01, * meant p < 0.05, were assumed as statistically significant.

	Patients with perioperative anaemia (n = 24)	Patients without perioperative anaemia (n = 44)	p value
Ischemia time (mins)	32.2 (8.9)	28.3 (6.3)	0.0386*†
Blood loss (mL)	331.3 (234.0)	92.3 (88.1)	<0.0001***†
Blood transfusion	-	-	0.0579‡
Yes (%)	6 (25.0)	3 (6.8)	-
No (%)	18 (75.0)	41 (93.2)	-
Surgical approaches	-	-	0.0022**‡
Laparoscopic (%)	17 (70.8)	43 (97.7)	-
Open (%)	7 (29.2)	1 (2.3)	-
R.E.N.A.L. Score	6.7 (1.5)	6.1 (1.1)	0.0869†
Values are in mean (SD) or n (%)			
Abbreviations SD standard deviation, *sCr* serum creatinine, *NGAL* neutrophil gelatinase-associated lipocalin, *Ht* haematocrit			
Ht-BL, sCr-BL, NGAL-BL represented Ht, sCr and serum NGAL before PN; Ht-2 h, sCr-2 h, NGAL-2 h represented Ht, sCr and serum NGAL 2 hours after PN; Ht-1d, sCr-1d, NGAL-1d represented Ht, sCr and serum NGAL 1 day after PN			
† Student's t-test. ‡ Chi-square test.			
*** meant p < 0.001, ** meant p < 0.01, * meant p < 0.05, were assumed as statistically significant.			

Postoperative AKI was diagnosed in 22/68 (32.4%) patients when KDIGO criteria was applicated. AKI was more frequently diagnosed in patients with perioperative anaemia (14/24) than those without (8/44), p = 0.0011. Intriguingly, patients with perioperative anaemia suffered longer ischemia time (32.2 ± 8.9 vs. 28.3 ± 6.3 min, p = 0.0386), representing severer renal ischemia-reperfusion injury (IRI). Besides, the blood loss of patients with perioperative anaemia was significantly higher when compared with those who without anaemia (331.3 ± 234.0 vs. 92.3 ± 88.1 ml, p0.0001).

sCr and serum NGAL significantly increased after PN. But sCr was not a sensitive biomarker of AKI. sCr didn't present an obvious elevation after PN surgery until 1 day later, even in the subgroup of patients with postoperative AKI (64.36 ± 16.70 μmol/L in the baseline, 72.23 ± 22.87 μmol/L 2 h after PN, 126.6 ± 53.19 μmol/L 1d after PN). However, serum NGAL presented a dramatical increase as early as 2 h after PN (71.47 ± 22.44 ng/mL in the baseline, 157.4 ± 87.72 ng/mL 2 h after PN, 156.4 ± 96.03 ng/mL 1d after PN), and even in the subgroup of patients without AKI (69.43 ± 20.51 ng/mL in the baseline, 126.6 ± 50.33 ng/mL 2 h after PN, 125.7 ± 46.74 ng/mL 1d after PN). Serum NGAL was a more sensitive biomarker of AKI. As shown in **Supplement** Table 1, when the criteria of postoperative serum NGAL > 150 ng/mL was used, 13 more patients with postoperative AKI can be diagnosed. In univariate analysis, perioperative anaemia, ischemia time, blood loss, blood transfusion, surgical approaches and R.E.N.A.L. score were all confirmed as relevant factors of postoperative AKI in patients undergoing PN (Table 2). Meanwhile, perioperative anaemia was associated with ischemia time, blood loss, blood transfusion and surgical approaches (Table 3).

Table 2
Univariate analysis of relevant factors of postoperative AKI

Postoperative AKI				
Variables	Estimate	SE	p value	OR (95% CI) †
Age (years)	-0.007	0.037	0.840	0.993 (0.924–1.066)
Gender	0.630	0.526	0.231	1.878 (0.670–5.263)
Perioperative anaemia	-1.841	0.569	0.001**	0.159 (0.052–0.485)
Ischemia time (mins)	0.219	0.059	<0.001***	1.245 (1.109–1.397)
Blood loss (mL)	0.006	0.002	0.003**	1.006 (1.002–1.010)
Blood transfusion	-1.682	0.765	0.028*	0.186 (0.042–0.834)
Surgical approaches	-3.247	1.104	0.003**	0.039 (0.004–0.338)
R.E.N.A.L. Score	0.939	0.287	0.001**	2.559 (1.457–4.492)

Abbreviations AKI acute kidney injury, *CI* confidence interval, *OR* odds ratio, *SE* standard error

† Binary univariate logistic regression analysis

*** meant $p < 0.001$, ** meant $p < 0.01$, * meant $p < 0.05$, were assumed as statistically significant.

Table 3
Univariate analysis of relevant factors of perioperative anaemia

Perioperative anaemia				
Variables	Estimate	SE	p value	OR (95% CI) †
Age (years)	0.005	0.036	0.886	1.005 (0.937–1.079)
Gender	0.661	0.514	0.235	1.842 (0.673–5.043)
Ischemia time (mins)	0.071	0.036	0.049*	1.074 (1.000-1.153)
Blood loss (mL)	0.015	0.003	<0.001***	1.015 (1.008–1.022)
Blood transfusion	-1.516	0.762	0.046*	0.220 (0.049–0.977)
Surgical approaches	-3.068	1.100	0.005**	0.047 (0.005–0.402)
R.E.N.A.L. Score	0.355	0.211	0.093	1.426 (0.943–2.156)

Abbreviations CI confidence interval, *OR* odds ratio, *SE* standard error

† Binary univariate logistic regression analysis

*** meant p < 0.001, ** meant p < 0.01, * meant p < 0.05, were assumed as statistically significant.

Furthermore, the relationship between the variation of sCr after PN and demographic characteristics of the 68 patients were analyzed by Pearson correlation analysis. Results revealed that Ht 1d after PN (Pearson r=-0.3171, p = 0.0084) and the variation of Ht (Pearson r=-0.3075, p = 0.0107) showed a weak negative correlation, blood loss (Pearson r = 0.4871, p<0.0001) and R.E.N.A.L. score (Pearson r = 0.5025, p<0.0001) presented a mild positive correlation, and ischemia time (Pearson r = 0.6616, p<0.0001) exhibited a strong positive correlation with the variation of sCr after PN. When the variate was changed from variation of sCr to serum NGAL 1d after PN, the semblable relationships were observed (Fig. 1).

Discussion

The aim of this study was to find out the relationship between perioperative anaemia and postoperative AKI after PN surgery. In present findings, we found perioperative anaemia occurred in 35.29% of the patients undergoing PN for a single nonmetastatic renal tumor, and postoperative AKI developed in 32.4% of the patients when KDIGO criteria was applicated. In addition, we found that the patients with perioperative anaemia had a higher risk of AKI than those without anaemia (58.3% (14/24) vs. 18.2% (8/44)). Moreover, univariate logistic regression analyses revealed that anaemia was a risk factor of postoperative AKI.

Anaemia is a common complication within the surgical populations for various reasons. Numbers of studies suggested an association of anaemia in the perioperative period with postoperative adverse outcomes, like AKI, stroke and myocardial infarction[7, 8]. Our data exhibited the high prevalence of postoperative anaemia in patients undergoing PN. Multifactorial in origin, preoperative anemia, perioperative blood loss (surgical bleeding, coagulopathy, phlebotomy, etc.) and progressive blunted erythropoiesis are the main factors that cause postoperative anaemia after clinical surgery[7]. Likewise, our univariate analysis results showed that the occurrence of postoperative anemia was related to the ischemia time, blood loss and surgical approaches. Furthermore, haemodilution caused by excessive infusion may lead to "dilutional" anemia or aggravate previous anaemia.

The incidence of postoperative AKI in our research was high. An important cause of AKI in PN surgery is cellular ischemia, which results in tubular epithelial and vascular endothelial injury and activation[15, 16]. The higher NGAL level in the anaemia group confirmed this in our research. As reported, the decline in renal function after PN averages about 20% in the operative kidney[3]. In fact, the patients enrolled in our study underwent more complicated surgery (R.E.N.A.L. score was 6.7 ± 1.5 for patients in perioperative anaemia group and 6.1 ± 1.1 in non-anaemia group, which meant middle complexity and is concerned with outcome of PN[17]) and suffered more blood loss and longer ischemia time that culminated in renal hypoxaemia [1, 8], which might increase the risk of postoperative anaemia and AKI. In the past, postoperative AKI was thought to be a transient injury, which had no effect on prognosis after recovery. However, studies have demonstrated that postoperative AKI, even short-term AKI (lasting 48–72 hours), could increase the risk of long-term functional impairment[18].

However, it is not clear whether the presence of anaemia is a direct contributor to AKI or simply a co-morbid disease that indirectly leads to AKI through potential factors. In previous study, Hales[19] found that AKI could contribute to the development of anaemia as a result of reduced EPO production, an increased risk of bleeding and reduced red cell life span. It has also been demonstrated that anaemia was a risk factor of AKI in patients underwent major surgery, leading to increased postoperative adverse outcomes[7, 20, 21]. Postoperative anemia may affect renal function via either a higher incidence of hypotension or a decrease in oxygen-carrying capacity to enhance renal oxidative stress[22]. Although a blood transfusion might improve oxygen delivery, less transfusion is consistently related to a decreased risk of morbidity and mortality[23–25]. Moreover, red cell viability is impaired during storage, resulting in excess hemolysis after transfusion. As a result, transfusions may induce iron release from macrophages that results in oxidative renal injury[26, 27]. Therefore, transfusion itself may have affected the incidence of AKI [28, 29]. In our study, 3 patients had blood loss and adequate transfusion to correct their hematocrit to > 30%,of which 1 and 2 developed AKI when KDIGO and NGAL criteria was used respectively. The adverse consequences of anaemia are likely to be severer during PN surgery, during which, for reasons outlined earlier, postoperative AKI is more prone to occurrence.

Our results suggest that postoperative anaemia is a risk factor for the development of AKI in patients undergoing PN, though the correlation is weak, which is probably compromised by other stronger predictors of AKI, such as ischemia time and blood loss. Nevertheless, our study still has a few limitations: Firstly, it was a single-center retrospective study, which might undergo selection bias. Secondly, the sample size was small, so we cannot perform a multivariable analysis to determine the independent predictive ability of perioperative anemia. Thirdly, we did not evaluate the long-term effect on patient's kidney function and could not draw any conclusion if perioperative AKI can be reversed. As a result, further large-scale, long-term and prospective studies should be conducted in multi-centers to verify the detailed correlation between anaemia and postoperative AKI.

Conclusions

In conclusion, patients who suffered anaemia after PN presented a higher risk of developing AKI. Unanswered questions remain and further investigation is warranted. Since perioperative anaemia is a risk factor of postoperative AKI and could have long-term effects, efforts should be made to control the anaemia-related risk factors and thus avoid postoperative AKI. If anaemia occurs after operation, AKI should be on the alert. NGAL or other sensitive biomarkers need to be monitored to detect AKI in early stage.

List Of Abbreviations

PN, partial nephrectomy

NGAL, neutrophil gelatinase-associated lipocalin

sCr, serum creatinine

Ht, haematocrit

AKI, acute kidney injury

CKD, chronic kidney disease

CPB, cardiopulmonary bypass

BUN, blood urea nitrogen

GFR, glomerular filtration rate

IRI, ischemia-reperfusion injury

SD, standard deviation

Declarations

Ethics approval and consent to participate

The experimental protocol was established, according to the ethical guidelines of the Helsinki Declaration and was approved by the Human Ethics Committee of Xinhua Hospital, School of Medicine, Shanghai

Jiao Tong University. Written informed consent was obtained from individual or guardian participants.

Consent for publication:

Written informed consent for publication was obtained from all participants.

Availability of data and material:

The datasets used and analyzed during the current study are available from the corresponding author Subo Qian and Haibo Shen on reasonable request.

Competing interests:

The authors declare that they have no competing interests.

Funding

Not applicable.

Authors' contributions

SZ carried out the main part of the experiments, analyzed the data, wrote and edited the manuscript. HD carried out the main part of the experiments, analyzed the data and edited the manuscript as well. YW helped to analyze the data and was also a major contributor in editing the manuscript. JG and WX helped to perform the experiments and participated in the data analysis. SQ and HS designed and supervised the progress of this research and were also major contributors in writing and editing the manuscript. All the authors have read and approved the final version of the manuscript.

Acknowledgements

Not applicable.

References

1. Mir MC, Ercole C, Takagi T, Zhang Z, Velet L, Remer EM, Demirjian S, Campbell SC: **Decline in renal function after partial nephrectomy: etiology and prevention**. *The Journal of urology* 2015, **193**(6):1889-1898.
2. Campbell SC, Novick AC, Belldegrun A, Blute ML, Chow GK, Derweesh IH, Faraday MM, Kaouk JH, Leveillee RJ, Matin SF *et al*: **Guideline for management of the clinical T1 renal mass**. *The Journal of urology* 2009, **182**(4):1271-1279.
3. Mehta RL, Cerdá J, Burdmann EA, Tonelli M, García-García G, Jha V, Susantitaphong P, Rocco M, Vanholder R, Sever MS *et al*: **International Society of Nephrology's 0by25 initiative for acute kidney injury (zero preventable deaths by 2025): a human rights case for nephrology**. *Lancet (London, England)* 2015, **385**(9987):2616-2643.

4. Hu J, Liu S, Jia P, Xu X, Song N, Zhang T, Chen R, Ding X: **Protection of remote ischemic preconditioning against acute kidney injury: a systematic review and meta-analysis**. *Critical care (London, England)* 2016, **20**(1):111.
5. Luo F, Shi J, Shi Q, Xu X, Xia Y, He X: **Mitogen-Activated Protein Kinases and Hypoxic/Ischemic Nephropathy**. *Cellular Physiology and Biochemistry* 2016, **39**:1051-1067.
6. Bhindi B, Lohse CM, Schulte PJ, Mason RJ, Cheville JC, Boorjian SA, Leibovich BC, Thompson RH: **Predicting Renal Function Outcomes After Partial and Radical Nephrectomy**. *European urology* 2019, **75**(5):766-772.
7. Fowler A, Ahmad T, Phull M, Allard S, Gillies M, Pearse R: **Meta-analysis of the association between preoperative anaemia and mortality after surgery**. *The British journal of surgery* 2015, **102**:1314-1324.
8. Baron DM, Hochrieser H, Posch M, Metnitz B, Rhodes A, Moreno RP, Pearse RM, Metnitz P: **Preoperative anaemia is associated with poor clinical outcome in non-cardiac surgery patients**. *British journal of anaesthesia* 2014, **113**(3):416-423.
9. Baranauskas T, Kaunienė A, Švagždienė M, Širvinskas E, Lenkutis T: **The correlation of post-operative acute kidney injury and perioperative anaemia in patients undergoing cardiac surgery with cardiopulmonary bypass**. *Acta medica Lituanica* 2019, **26**(1):79-86.
10. Musallam KM, Tamim HM, Richards T, Spahn DR, Rosendaal FR, Habbal A, Khreiss M, Dahdaleh FS, Khavandi K, Sfeir PM *et al*: **Preoperative anaemia and postoperative outcomes in non-cardiac surgery: a retrospective cohort study**. *Lancet (London, England)* 2011, **378**(9800):1396-1407.
11. Qian S, Huang Y, Wang N, Ding Y, Qi J, Yu Y: **Urinary neutrophil gelatinase-associated lipocalin as a biomarker to monitor renal function in patients with obstructive ureteral calculi**. 2019, **37**(6):1197-1204.
12. Haase M, Bellomo R, Devarajan P, Schlattmann P, Haase-Fielitz A: **Accuracy of neutrophil gelatinase-associated lipocalin (NGAL) in diagnosis and prognosis in acute kidney injury: a systematic review and meta-analysis**. *American journal of kidney diseases : the official journal of the National Kidney Foundation* 2009, **54**(6):1012-1024.
13. Haase M, Bellomo R, Devarajan P, Schlattmann P, Haase-Fielitz A, Group NM-aI: **Accuracy of neutrophil gelatinase-associated lipocalin (NGAL) in diagnosis and prognosis in acute kidney injury: a systematic review and meta-analysis**. *American journal of kidney diseases : the official journal of the National Kidney Foundation* 2009, **54**(6):1012-1024.
14. Khwaja A: **KDIGO clinical practice guidelines for acute kidney injury**. *Nephron Clinical practice* 2012, **120**(4):c179-184.
15. Evans RG, Goddard D, Eppel GA, O'Connor PM: **Factors that render the kidney susceptible to tissue hypoxia in hypoxemia**. *American journal of physiology Regulatory, integrative and comparative physiology* 2011, **300**(4):R931-940.
16. Liaño F, Pascual J: **Epidemiology of acute renal failure: a prospective, multicenter, community-based study. Madrid Acute Renal Failure Study Group**. *Kidney international* 1996, **50**(3):811-818.

17. McDonald ML, Lane BR, Jimenez J, Lee HJ, Yim K, Bindayi A, Hamilton ZA, Field CA, Bloch AS, Dey S *et al*: **Renal Functional Outcome of Partial Nephrectomy for Complex R.E.N.A.L. Score Tumors With or Without Neoadjuvant Sunitinib: A Multicenter Analysis**. *Clinical genitourinary cancer* 2018, **16**(2):e289-e295.
18. Bravi CA, Vertosick E, Benfante N, Tin A, Sjoberg D, Hakimi AA, Touijer K, Montorsi F, Eastham J, Russo P *et al*: **Impact of Acute Kidney Injury and Its Duration on Long-term Renal Function After Partial Nephrectomy**. *Eur Urol* 2019, **76**(3):398-403.
19. Hales M, Solez K, Kjellstrand C: **The anemia of acute renal failure: association with oliguria and elevated blood urea**. *Renal failure* 1994, **16**(1):125-131.
20. Choi YJ, Kim SO, Sim JH, Hahm KD: **Postoperative Anemia Is Associated with Acute Kidney Injury in Patients Undergoing Total Hip Replacement Arthroplasty: A Retrospective Study**. *Anesthesia and analgesia* 2016, **122**(6):1923-1928.
21. Han SS, Baek SH, Ahn SY, Chin HJ, Na KY, Chae DW, Kim S: **Anemia Is a Risk Factor for Acute Kidney Injury and Long-Term Mortality in Critically Ill Patients**. *The Tohoku journal of experimental medicine* 2015, **237**(4):287-295.
22. Nangaku M: **Chronic hypoxia and tubulointerstitial injury: a final common pathway to end-stage renal failure**. *Journal of the American Society of Nephrology : JASN* 2006, **17**(1):17-25.
23. Russell JA, Phang PT: **The oxygen delivery/consumption controversy. Approaches to management of the critically ill**. *American journal of respiratory and critical care medicine* 1994, **149**(2 Pt 1):533-537.
24. Napolitano LM, Corwin HL: **Efficacy of red blood cell transfusion in the critically ill**. *Critical care clinics* 2004, **20**(2):255-268.
25. Hébert PC, Yetisir E, Martin C, Blajchman MA, Wells G, Marshall J, Tweeddale M, Pagliarello G, Schweitzer I: **Is a low transfusion threshold safe in critically ill patients with cardiovascular diseases?** *Critical care medicine* 2001, **29**(2):227-234.
26. Karkouti K, Wijeysundera DN, Yau TM, Callum JL, Cheng DC, Crowther M, Dupuis JY, Fremes SE, Kent B, Laflamme C *et al*: **Acute kidney injury after cardiac surgery: focus on modifiable risk factors**. *Circulation* 2009, **119**(4):495-502.
27. Leaf DE, Rajapurkar M, Lele SS, Mukhopadhyay B, Rawn JD, Frendl G, Waikar SS: **Increased plasma catalytic iron in patients may mediate acute kidney injury and death following cardiac surgery**. *Kidney international* 2015, **87**(5):1046-1054.
28. Karkouti K, Grocott HP, Hall R, Jessen ME, Kruger C, Lerner AB, MacAdams C, Mazer CD, de Medicis É, Myles P *et al*: **Interrelationship of preoperative anemia, intraoperative anemia, and red blood cell transfusion as potentially modifiable risk factors for acute kidney injury in cardiac surgery: a historical multicentre cohort study**. *Canadian journal of anaesthesia = Journal canadien d'anesthesie* 2015, **62**(4):377-384.
29. Karkouti K: **Transfusion and risk of acute kidney injury in cardiac surgery**. *British journal of anaesthesia* 2012, **109 Suppl 1**:i29-i38.

Figures

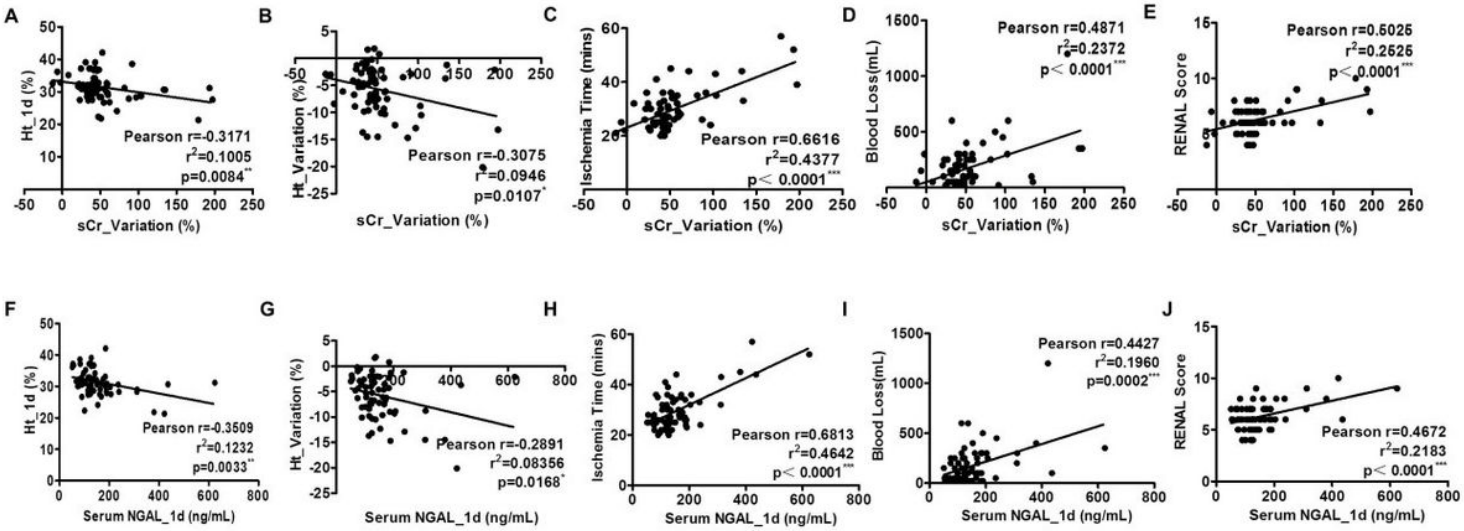

Figure 1

Pearson correlation analysis of variation of postoperative renal function and patients' demographic characteristics A to E represented the correlation of sCr variation and Ht 1 day post PN, Ht variation, ischemia time, blood loss and R.E.N.A.L. score, correspondingly; F to J represented the correlation of postoperative serum NGAL and Ht 1 day post PN, Ht variation, ischemia time, blood loss and R.E.N.A.L. score, correspondingly Abbreviations NGAL neutrophil gelatinase-associated lipocalin, Ht haematocrit, sCr serum creatinine *** meant p < 0.001, ** meant p < 0.01, * meant p < 0.05, were assumed as statistically significant

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Supplementtable1.docx