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Coronary Angiography Within 48 Hours Before Cardiac Surgery Increases the Risk of Postoperative Acute Kidney Injury

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ABSTRACT

Background: Cardiac surgery and coronary examination, such as invasive coronary angiography (CAG), are both possibly associated with acute kidney injury (AKI). Preoperative CAG examination and cardiac surgery within a short interval may increase the incidence of AKI.

Methods: We retrospectively reviewed 1112 patients who underwent CAG examination within 30 days prior to the cardiac operation in this study. Postoperative AKI was defined, according to Kidney Disease Improving Global Outcomes Definition and Staging (KDIGO) criteria.

Results: The total incidence of AKI was 40.8% and cystatin C level was 1.260 (1.028, 1.672) mg/L. For patients who received CAG, age, body mass index, cardiopulmonary time, and the time interval between preoperative CAG examination and cardiac operation within 48h was shown to be independent predictors of postoperative AKI. The incidence of AKI in patients undergoing preoperative CAG within 48h was 11.2% higher than in those more than 48h ($P < 0.001$). Patients undergoing valve surgery with or without coronary artery bypass grafting (CABG) exhibited a higher AKI risk than those only accepting CABG. The in-hospital stay of patients who developed AKI was 2 days longer than those without AKI. However, undergoing CAG within 48h prior to cardiac operation did not prolong ICU length of stay or hospital length of stay, nor did it increase the risk of death or renal failure after an operation.

Conclusion: Undergoing CAG within 48 hours before cardiac surgery increases the risk of postoperative AKI.

INTRODUCTION

Acute kidney injury (AKI) is one of the most frequent and serious complications after cardiac surgery, and it has been demonstrated to be associated with increased mortality and morbidity, thus contributing to longer hospitalization and more costs [Ko 2012]. For patients who are elderly or have high risks for coronary artery disease, coronary artery examination routinely should be performed before cardiac surgery, which usually includes less invasive coronary computed tomographic angiography (CTA) and invasive coronary angiography (CAG). However, contrast-induced vasoconstriction can increase ischemia-mediated oxidative stress, which leads to cytotoxicity and tubular cell death [Nunag 2009]. For CAG, the incidence of contrast-induced nephropathy can be as high as 10.5% [Tsai 2014; Inohara 2016], and for coronary CTA, it can be about 7% [Balemans 2012]. And it can reach as high as 50% in the presence of certain comorbid conditions [Mehran 2006; Dangas 2005]. Whether undergoing coronary examination, especially CAG just before cardiac surgery in a short time, increases the risk of postoperative AKI remains unclear. This study aims to preliminarily analyze the effects of preoperative CAG on the incidence of AKI and further investigate the relationship between time interval (between CAG and cardiac surgery) and the incidence of postoperative AKI, according to Kidney Disease Improving Global Outcomes Definition and Staging (KDIGO) criteria and cystatin C level.

METHODS

Study population: From January 2015 to September 2019, 1112 patients undergoing CAG examination within 30 days before surgery were included in this retrospective study. CAG was performed under standard procedure. The contrast agent iohexol 300 or iodixanol 270 (50-100 mL) was used for CAG (the volume can reach 150 mL for chronic total occlusion lesions). This study was approved by the Ethics Committee of the First Affiliated Hospital of Xi'an Jiaotong University.

Data collection: The primary outcome variable was the development of AKI after cardiac surgery. Postoperative AKI was staged, according to KDIGO criteria. Briefly, stage 1: increase in serum creatinine (sCr) ≥ 0.3 mg/dL or to 1.5 to 1.9 times more than baseline; stage 2: increase in sCr to 2.0 to 2.9 times the baseline; stage 3: increase in sCr to 3.0 or more times the baseline; increase in sCr ≥ 4.0 mg/dL; or the initiation of renal replacement therapy. The maximal value of cystatin C was recorded within seven days postoperative as an additional index for evaluating renal function. Additional

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data were included as follows: age, gender, body mass index (BMI), history of smoking, diabetes mellitus, prior myocardial infarction, unstable angina, hypertension, New York Heart Association (NYHA) functional classification, left ventricular ejection fraction (LVEF), preoperative hemoglobin level (g/L), chronic obstructive pulmonary disease, peripheral arterial disease, previous stroke, serum creatinine level (μmol/L), and preoperative estimated glomerular filtration rate (eGFR, mL/min). Intra-operative data included time interval between preoperative CAG and subsequent cardiac surgery (≤48h, 48-72h, and >72h), type of surgery (off-pump coronary artery bypass grafting, OPCAB, on-pump coronary artery bypass grafting, ONCAB, valve surgery, valve surgery + CABG, and other surgeries), cardiopulmonary bypass (CPB) time, cross-clamp time, and nadir hematocrit level during CPB. In-hospital outcome variables included in-hospital mortality, ICU and postoperative hospital stay, postoperative intra-aortic balloon pump (IABP) support, and postoperative continuous renal replacement therapy (CRRT) use.

Statistical analyses: Continuous variables in this study were summarized as mean ± standard deviation or medians with interquartile range, and categorical variables were presented as numbers and percentages. Continuous data were compared with t-test when normally distributed and non-parametric Mann-Whitney U test when shewed. Categorical data were compared with the chi-square test. First, the incidence of AKI and cystatin C levels in all patients were counted. Moreover, the incidence of AKI and cystatin C levels in patients undergoing preoperative coronary examination within 48 hours also were analyzed. Univariable logistic regression analysis was performed to identify possible factors significantly associated with AKI from demographic, preoperative laboratory tests, and operative data. Time interval between CAG and cardiac surgery and variables with $P < 0.1$ in univariable logistic regression analysis were included in the multivariable logistic regression analysis. The variables with $P < 0.05$ were considered independent factors of AKI. According to the different time intervals between CAG and surgery, we divided the patients into three subgroups (≤48h, 48-72h, and >72h) and analyzed the incidence of AKI grade 1, AKI grade 2-3, total AKI, and the level of cystatin C in each subgroup, respectively. In-hospital outcomes of each subgroup, such as postoperative ICU stay, postoperative hospital stay, in-hospital mortality, postoperative IABP support, and postoperative CRRT support, also were analyzed. Finally, we compared the incidence of AKI and the level of cystatin C in patients undergoing different types of operation, including OPCAB, ONCAB, valve surgery, valve surgery + CABG, and others.

All analyses were two-sided, and $P < 0.05$ was considered statistically significant. Analyses were performed using SPSS 19.0.

RESULTS

The influence of preoperative CAG examination on the incidence of postoperative AKI: A total of 1112 patients who underwent preoperative CAG were included in this study. The total incidence of postoperative AKI was 40.8%, while cystatin C levels were 1.260 (1.028, 1.672) mg/L. (Table 1) Patients undergoing preoperative CAG within 48h had a higher percentage of urgent cases than those undergoing preoperative CAG more than 48h (27.6% vs. 20.8%, $P = .01$). Patients undergoing preoperative CAG within 48h had a much higher incidence of AKI than patients undergoing preoperative CAG more than 48h ($P < .001$) and had a higher cystatin C level ($P = .017$).

Predictors of AKI: The baseline characteristics of the 1112 patients who underwent preoperative CAG are shown in Table 2. (Table 2) Patients were 60 (55, 65) years old, and 63% were male. A total of 454 (40.8%) patients had AKI after cardiac surgery. Patients who were elder, male, had high BMI, concomitant prior myocardial infarction, unstable angina, or chronic obstructive lung disease were more prone to have AKI (Table 2). AKI patients also had a poor cardiac function, low preoperative eGFR, and low preoperative hemoglobin. Moreover, a longer CPB time and cross-clamp time during surgery were more likely to initiate postoperative AKI. In multivariable analysis, age, BMI, CPB time, and the time interval between CAG examination and subsequently cardiac surgery within 48h (OR: 1.516; 95% CI: 1.121-2.050; $P = .007$) were independent predictors of postoperative AKI. (Table 3)

The type of cardiac surgery also influenced postoperative AKI. Significant differences in the rate of AKI and cystatin C levels were observed. (Figure 1) (Figure 2) Overall, valve surgery with or without CABG had a higher postoperative risk of AKI than that in CABG. Patients undergoing OPCAB had a relatively low incidence of total AKI or AKI stage 1 and stage 2-3. Conversely, patients undergoing valve and CABG surgery simultaneously had a higher incidence of AKI.

Table 1. The influence of preoperative CAG on the incidence of postoperative AKI

	Total ($N = 1112$)	≤48h ($N = 362$)	>48h ($N = 750$)	P
AKI incidence	454 (40.8%)	175 (48.3%)	279 (37.1%)	<0.001
Cystatin C(mg/L)*	1.260 (1.028, 1.672)	1.459 (1.125, 1.942)	1.250 (0.970, 1.487)	0.017

Patients were divided into two parts: those undergoing preoperative coronary examination within 48h marked as ≤48h group, and those more than 48h marked as >48h group. CAG, coronary angiography; AKI, acute kidney injury. *Variables are presented as median (25th, 75th percentiles)

Relationship of different time intervals from preoperative CAG with AKI and in-hospital outcomes: In this study, patients had a median 4 days (interquartile range 2-7 days) interval between preoperative CAG and cardiac surgery. Patients who had AKI exhibited a shorter time interval than those who did not (median 4 days, interquartile range 2-6 days vs. median 5 days, interquartile range 2-7 days, $P < .001$). The patients were divided into three groups, depending on the different time intervals. (Table 4) Patients undergoing CAG within 48h before surgery had a higher incidence of AKI and cystatin C levels than other groups. Overall, the incidence of AKI decreased with the prolongation of the time interval.

For in-hospital outcomes, the ratio of postoperative CRRT use in patients who developed AKI was 0.9% vs. those who did not was 0.3% ($P = .197$), and in-hospital mortality of patients who developed AKI was 2.4% vs. those who did not was 1.4% ($P = .193$). But patients who developed AKI had a longer postoperative hospital stay than those who did not (median 12 days, interquartile range 10-15 days vs. median 14 days, interquartile range 10-18 days, $P < .001$).

The in-hospital outcomes of different time interval groups were shown in Table 4. There was no difference in in-hospital mortality or postoperative IABP use or postoperative CRRT use for each group. There was a significant difference in ICU stay between patients who underwent preoperative CAG within 48h and more than 72h ($P = .024$). But patients who underwent preoperative CAG for more than 72h had a longer postoperative hospital stay than those within 48h ($P = .001$).

DISCUSSION

Table 3. Multivariable predictors of acute kidney injury after cardiac surgery

Variables	Odds ratio (95% CI)	*P*
Age (years)	1.051 (1.029-1.073)	<0.001
BMI (kg/m^2)	1.077(1.028-1.129)	0.002
CPB time (min)	1.005 (1.000-1.009)	0.035
Time interval (≤48h)	1.516 (1.121-2.050)	0.007

BMI, body mass index; time interval (≤48h), time between coronary angiogram and cardiac surgery

Table 2. Baseline characteristics of the patients undergoing CAG

Variables	All patients (*N* = 1112)	AKI (*N* = 454)	No AKI (*N* = 658)	*P*
Age (years)*	60 (55, 65)	61 (56, 65)	59 (53, 63)	0.001
Male (%)	63	60	65	0.095
BMI (kg/m^2) *	23 (21, 25)	23 (21, 25)	22 (20, 24)	0.023
Ever smoked (%)	45	42	47	0.226
Hypertension (%)	39	42	37	0.148
Diabetes mellitus (%)	15	14	16	0.370
Prior myocardial infarction (%)	11	8	14	0.004
Unstable angina (%)	41	37	44	0.019
Chronic obstructive lung disease (%)	6	8	5	0.046
Peripheral arterial disease (%)	4	5	3	0.261
Cerebrovascular disease (%)	7	7	8	0.765
NYHA III/IV (%)	63	66	62	0.040
LVEF (%)*	61 (52, 67)	63 (53, 67)	60 (51, 67)	0.108
Preoperative creatinine (μmol/L)*	64 (57, 76)	63 (55, 74)	65 (57, 75)	0.505
Preoperative eGFR (mL/min)*	98 (88, 104)	97 (85, 103)	98 (90, 104)	0.041
Preoperative hemoglobin (g/L)*	136 (127, 146)	133 (124, 144)	136 (127, 148)	0.035
CPB time (min)*	106 (81, 136)	109 (79, 139)	102 (79, 134)	0.073
Cross-clamp time (min)*	72 (51, 97)	74 (51, 98)	71 (50, 96)	0.042
Nadir hematocrit on CPB (%)*	24 (22, 27)	24 (22, 27)	25 (22, 27)	0.500
Time interval (days)	4 (2, 7)	4 (2, 6)	5 (2, 7)	<0.001

BMI, body mass index; NYHA, New York Heart Association; LVEF, left ventricular ejection fraction; eGFR, estimated glomerular filtration rate; CPB, cardiopulmonary bypass. *Variables are presented as median (25th, 75th percentiles).

In this study, we included 1112 patients who underwent preoperative CAG examination within 30 days. It was shown that the incidence of AKI in patients undergoing preoperative CAG within 48h was higher than that in patients undergoing preoperative CAG more than 48h (OR: 1.516; 95% CI: 1.121-2.050; *P* = .007). Undergoing preoperative CAG within 48h did not prolong the length of ICU stay or hospital stay, nor did it increase the risk of death or renal failure after the operation.

Traditional diagnostic criteria for AKI are based on changes in serum creatinine and urine output. However, these two indicators have many disturbing factors, and obvious changes in serum creatinine may not be seen until 48-72h after renal injury, potentially delaying the diagnosis of AKI [Al-Naimi 2019; Fan 2018]. Thus, cystatin C, a relatively more sensitive and highly specific biomarker, was introduced as an additional index to reflect postoperative renal injury [Moledina 2018; Zhang 2011]. Overall, there was a similar trend in the change of cystatin C levels and the occurrence of AKI.

The incidence of postoperative AKI in our patients who underwent preoperative CAG was 40.8%, a little higher compared with other studies [Ko 2012; Mehta 2011; Ranucci 2013]. This result may be related to the fact that 63.8% of our patients in this study underwent valve surgery, and many had severe valve disease. In this cohort, advanced age, high BMI, and the time interval between preoperative CAG examination and operation within 48h were identified to be the independent predictors of AKI after cardiac surgery. Notably, preoperative eGFR and preoperative cardiac function grading were not independent risk factors in multivariate analysis. This may be related to better perioperative volume management and better intraoperative myocardial protection in patients with renal insufficiency or heart failure. CPB time and cross-clamp time were considered to be related to the occurrence of AKI after cardiac surgery [Yue 2019; Ghincea 2019; Karim 2017]. In this study, we found that the incidence of postoperative AKI in the patients who underwent OPCAB was only 26.1%, which was significantly lower than that in patients with intraoperative cardiopulmonary bypass. Valve operation is demonstrated to be an independent risk factor for postoperative acute renal insufficiency, which could be further increased by a longer CPB duration [Grayson 2003]. We also found that patients undergoing valve operation, especially

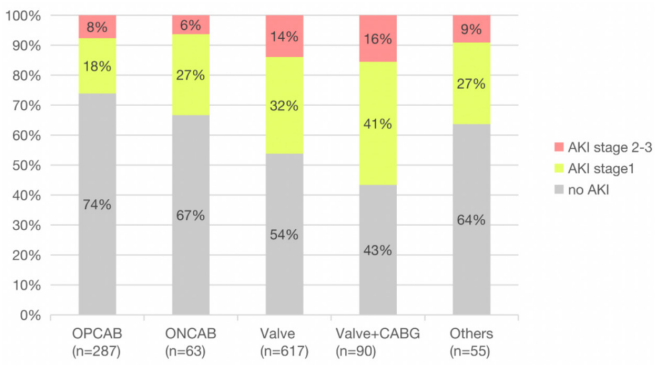

Figure 1. Incidence of postoperative AKI in relation to type of cardiac surgery

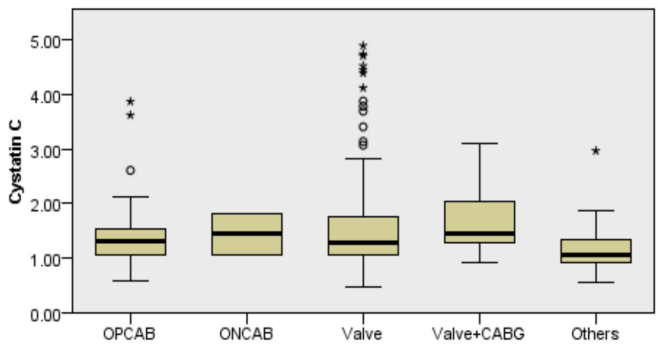

Figure 2. Level of Cystatin C in relation to type of cardiac surgery

Table 4. Incidence of postoperative AKI and in-hospital outcomes in relation to different time interval

	Overall (*N* = 1112)	≤48h (*N* = 362)	48-72h (*N* = 109)	>48h (*N* = 641)	*P*
Total AKI	454 (40.8%)	177 (48.9%)	51 (46.8%)	226 (35.3%)	<0.001†
AKI stage 1	321 (28.9%)	123 (34.0%)	33 (30.3%)	165 (25.7%)	0.001†
AKI stage 2-3	133 (12.0%)	54 (14.9%)	18 (16.5%)	61 (9.5%)	0.001†
Cystatin C (mg/L)*	1.260 (1.028, 1.672)	1.294 (1.042, 1.728)	1.316 (1.091, 1.71)	1.234 (1.01, 1.626)	0.153
ICU stay*	2 (2, 3)	2 (2, 3)	2 (2, 3)	3 (2, 3)	0.024
Postoperative hospital stay*	12.5 (10, 16)	12 (10, 15)	12 (10, 17)	13 (10, 17)	0.001
In-hospital mortality	20 (1.8%)	7 (0.6%)	1 (0.1%)	12 (1.1%)	0.891
Postoperative IABP use	19 (1.7%)	5 (0.4%)	1 (0.1%)	13 (1.2%)	0.454
Postoperative CRRT use	5 (0.4%)	1 (0.1%)	0 (0.0%)	4 (0.4%)	0.315

AKI, acute kidney injury; ICU, intensive care unit; IABP, intra-aortic balloon pump; CRRT, continuous renal replacement therapy. *Variables are presented as median (25th, 75th percentiles) and tested with the Kruskal-Wallis test. †Significantly different between the ≤48h and >72h subgroups.

valve operation combined with CABG, had a higher incidence of postoperative AKI and a higher level of cystatin C. However, different CPB times may have different influences on the occurrence of postoperative AKI. Karim's study indicated that the CPB duration of 71-140 min and >140 min increase the risk of cardiac surgery-associated kidney injury by an OR of 4.76 and 6.30, respectively [Karim 2017]. Patients in this study had a median CPB time and cross-clamp time of 106 min and 72 min, respectively, shorter than those in some research centers. In our center, we reduced the paralleled CPB time after the release of cross-clamp to reduce inflammation and kidney injury to a certain extent. Thus, it is acceptable for the result that CPB time and cross-clamp time were not independent risk factors in this study.

Although previous studies have reported the relationship of the time interval between CAG and cardiac surgery with postoperative AKI, there is no unified conclusion because of the different populations and heterogeneity of underlying diseases. More than 4000 patients undergoing preoperative CAG examination and cardiac surgery on the same day retrospectively were analyzed, and surgery on the day of CAG was shown to be an independent risk factor for acute kidney injury (OR 1.58, 95% CI 1.04-2.40) [Ranucci 2013]. But after the limitation of the practice of surgery on the same day of CAG performed by an institutional policy, the rate of AKI dramatically decreased, almost by 50%. Del Duca et al. included 649 patients who underwent cardiac surgery and found that CAG performed within 5 days before the operation was independently associated with postoperative AKI [Del Duca 2007]. However, Ko et al. included 2133 patients and drew a conclusion that the time interval between preoperative CAG examination and cardiac surgery does not affect the risk of postoperative AKI [Ko 2012]. Within 48 hours after injection, about 97% of iodinated contrast media is excreted unchanged by urine, and at 2-3 days basically excluded from the body [Svaland 1992; Azzalini 2017]. Therefore, theoretically, it is possible that AKI mostly will occur within 48 hours after the CAG examination. In this study, the incidence of AKI in patients who underwent preoperative CAG within 48h was 11.2% higher than in those more than 48h. In the subgroups, the incidence of total AKI and AKI stage 1 in <48h group were significantly higher than those in other groups. Notably, undergoing CAG within 48h before valve operation with CABG could significantly increase the risk of postoperative AKI. It was reported that AKI is associated with increased hospital stay and increased mortality after cardiothoracic surgery. In this study, most patients suffering from AKI after cardiac surgery can recover spontaneously in a few days. But the in-hospital stay of patients who developed AKI was 2 days longer than those without AKI. Undergoing CAG examination within 48h before cardiac operation did not prolong ICU length of stay or hospital stay, nor did it increase the risk of death or renal failure after an operation.

Several limitations exist. First, this study is a single-center retrospective study although the case number is relatively high. Second, the reasons for different times from angiography to surgery were complicated and not specified and could have influenced the development of AKI.

CONCLUSION

Postoperative AKI insult after cardiac surgery is multifactorial. According to our results, undergoing CAG within 48h before the cardiac operation is possibly an independent risk factor for postoperative acute kidney injury. The occurrence of AKI after cardiac surgery prolongs the in-hospital stay. Therefore, we recommend avoiding cardiac surgery within 48 hours after CAG, especially for valve surgery with CABG. In addition, coronary CTA can be used as an alternative for assessing coronary artery lesions before cardiac surgery.

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REFERENCES

Al-Naimi MS, Rasheed HA, Hussien NR, et al. 2019. Nephrotoxicity: Role and significance of renal biomarkers in the early detection of acute renal injury. J Adv Pharm Technol Res. 10(3):95-99.

Azzalini L, Candilio L, McCullough PA, et al. 2017. Current Risk of Contrast-Induced Acute Kidney Injury After Coronary Angiography and Intervention: A Reappraisal of the Literature. Can J Cardiol. 33(10):1225-1228.

Balemans CE, Reichert LJ, van Schelven BI, et al. 2012. Epidemiology of contrast material-induced nephropathy in the era of hydration. Radiology. 263(3):706-713.

Dangas G, Iakovou I, Nikolsky E, et al. 2005. Contrast-induced nephropathy after percutaneous coronary interventions in relation to chronic kidney disease and hemodynamic variables. Am J Cardiol. 95(1):13-19.

Del Duca D, Iqbal S, Rahme E, et al. 2007. Renal failure after cardiac surgery: timing of cardiac catheterization and other perioperative risk factors. Ann Thorac Surg. 84(4):1264-1271.

Fan PC, Chang CH, Chen YC. 2018. Biomarkers for acute cardiorenal syndrome. Nephrology (Carlton). 23 Suppl 4:68-71.

Grayson AD, Khater M, Jackson M, et al. 2003. Valvular heart operation is an independent risk factor for acute renal failure. Ann Thorac Surg. 75(6):1829-1835.

Ghincea CV, Reece TB, Eldeiry M, et al. 2019. Predictors of Acute Kidney Injury Following Aortic Arch Surgery. J Surg Res. 242:40-46.

Inohara T, Kohsaka S, Miyata H, et al. 2016. Performance and Validation of the U.S. NCDR Acute Kidney Injury Prediction Model in Japan. J Am Coll Cardiol. 67(14):1715-1722.

Ko B, Garcia S, Mithani S, et al. 2012. Risk of acute kidney injury in patients who undergo coronary angiography and cardiac surgery in close succession. Eur Heart J. 33(16):2065-2070.

Karim HM, Yunus M, Saikia MK, et al. 2017. Incidence and progression of cardiac surgery-associated acute kidney injury and its relationship with bypass and cross clamp time. Ann Card Anaesth. 20(1):22-27.

Mehran R, Nikolsky E. 2006. Contrast-induced nephropathy: definition,

epidemiology, and patients at risk. Kidney Int Suppl. (100):S11-15.

Mehta RH, Honeycutt E, Patel UD, et al. 2011. Relationship of the time interval between cardiac catheterization and elective coronary artery bypass surgery with postprocedural acute kidney injury. Circulation. 124(11 Suppl):S149-155.

Moledina DG, Parikh CR. 2018. Phenotyping of Acute Kidney Injury: Beyond Serum Creatinine. Semin Nephrol. 38(1):3-11.

Nunag M, Brogan M, Garrick R. 2009. Mitigating contrast-induced acute kidney injury associated with cardiac catheterization. Cardiol Rev. 17(6):263-269.

Ranucci M, Ballotta A, Agnelli B, et al. 2013. Acute kidney injury in patients undergoing cardiac surgery and coronary angiography on the same day. Ann Thorac Surg. 95(2):513-519.

Svaland MG, Haider T, Langseth-Manrique K, et al. 1992. Human pharmacokinetics of iodixanol. Invest Radiol. 27(2):130-133.

Tsai TT, Patel UD, Chang TI, et al. 2014. Contemporary incidence, predictors, and outcomes of acute kidney injury in patients undergoing percutaneous coronary interventions: insights from the NCDR Cath-PCI registry. JACC Cardiovasc Interv. 7(1):1-9.

Yue Z, Yan-Meng G, Ji-Zhuang L. 2019. Prediction model for acute kidney injury after coronary artery bypass grafting: a retrospective study. Int Urol Nephrol. 51(9):1605-1611.

Zhang Z, Lu B, Sheng X, et al. 2011. Cystatin C in prediction of acute kidney injury: a systemic review and meta-analysis. Am J Kidney Dis. 58(3):356-365.