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ORIGINAL ARTICLE

Uric Acid may predict post cardiac surgery renal function in patients with or without chronic kidney disease

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ABSTRACT

BACKGROUND: Acute kidney injury (AKI) is one of the most severe complications of cardiac surgery. Early detection of AKI is of great significance as the development of AKI is a heavy burden on both patients and the health care system. Many biomarkers have been shown to predict and diagnose AKI in recent years. The elevated serum uric acid level was associated with an increased probability of acute kidney injury through multiple and interactive mechanisms that lead to the development of chronic kidney disease. The aim of the present study was to obtain the role of serum level of Uric Acid (SUA)preoperative in prediction of acute renal injury (AKI)in post cardiac surgery. **METHODS**:This is a prospective observational study of patients undergoing cardiac surgery. Blood tests for preoperative assessment of uric acid and postoperative follow-up of AKI. Patients were split into two classes, AKI and non-AKI.**RESULTS**: Sixty five patients (44 Males, 21 females) were included. After cardiac surgery, 24 (36.92%) patients developed AKI and serum uric acid was significantly increased in AKI patients. A higher predictive ability of SUA for AKI (AUC; 0.74, P=0.016) is observed by using receiver operator characteristics (ROC) curve analysis **CONCLUSIONS**: Preoperative uric acid may be an independent risk factor for postoperative AKI after cardiac surgery and may significantly predict AKI.

Keywords:
Acute kidney injury, Uric Acid, postcardiac surgery

INTRODUCTION

Acute kidney injury (AKI) following cardiac surgery is known to lead to increased mortality and morbidity in these cases. The development of AKI after cardiovascular surgery has been well recognized in the past **[1]** and was included as a contributing factor in the poor outcomes and elevated mortality of these patients**[2]**.

For diagnosis and prediction of AKI, new application of several biomarkers are discovered recently**[3]**.Uric acid was found as a pathogenic factor in AKI development. Key mechanisms include increased inflammation, compromised renal self-regulation, renal vasoconstriction and apoptosis**[4]**. Previous studies suggested serum uric acid may be risk factor for AKI postcardiac surgery **[5]**.

The patients' outcomes are significantly affected by AKI incidence. Prevention of AKI is essential (if applicable) because no definite measures are effective in treating AKI post operatively. So, if high-risk patients are

diagnosed earlier, there is still the chance for prevention and further improvement of the outcomes of acute kidney injury**[6**].

The end-product of purine metabolism is uric acid which is excreted by kidney. Several epidemiologic studies that suggested an elevation of uric acid is related with diabetes mellitus, the progression of CKD, hypertension and cardiovascular diseases[**7**].

The aim of this study was to study the role of preoperative serum Uric Acid level in prediction of AKI after heart surgery in patients with or without history of chronic kidney disease (CKD).

PATIENTS AND METHODS

This is a prospective, observational study .All Patients who underwent cardiac surgery (elective coronary artery bypass graft and/or cardiac valve surgery) between 2015 and 2017 in Zagazig university hospitals were selected in this study . Both research participants signed informed consent documents. AKI was described by the KDIGO AKI guideline: increase in serum creatinine to 1.5 times the baseline level or increase in serum creatinine to 0.3 mg / dL within 48 h. AKI was graded as: stage I AKI when serum creatinine rose to 1.5–1.9 times baseline, stage II – serum creatinine elevation to 2.0–2.9 times baseline and stage III – rise in serum creatinine to 3.0 times baseline (including renal replacement therapy initiation) **[8]**.

Written informed consent was obtained from all participants, the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University. This research was carried out in compliance with the Code of Ethics of the World Scientific Association (Declaration of Helsinki) for studies involving humans.

All patients were admitted consecutively to the Cardiovascular Surgery Department during the periods listed above and evaluated for suitability for the inclusion requirements of the report. Inclusion requirements included age more than 18 years, elective open cardiac surgery (regardless of the particular requirement for operation) and preoperative glomerular filtration (eGFR) of more than 30 mL / min/1.73 m^2 and left ventricular ejection fraction (LVEF) of more than 30%. Exclusion criteria were patients who had their SUA and serum creatinine concentrations were missing; if they had undergone preoperative hemodialysis; if they had a prior history of nephrectomy or organ transplantation. Patients with emergency and/or evolving heart surgery, previous history of acute myocardial infarction 1 month prior to surgery and alleged contrast induced nephropathy were removed. As regard urine output data, were not used for diagnosis of AKI due to the effects of administered diuretics and insufficient recording in the studied patients.

Health history, current medicines and comorbid ities have been reported and/or compiled from medical charts. Other variables such as type of operation, cardiac functional parameters, cardiopulmonary bypass period (CPB), serum creatinine, eGFR and duration of hospital stay have also been reported. The preoperative eGFR values were calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation (eGFR=141×min (SCr/κ, 1)α×max (SCr/κ, 1)-1.209×0.993age×1.018 [if female]×1.159 [if Black] **[9].**

Enzymatic methods were used to measure serum uric acid by using an automatic biochemistry analyzer and the reference range for this uric acid assay is 3.0-7.0 mg/dL.

Measurement of serum urea in blood, HB, Albumin creatinine and serum uric acid (SUA) levels have been obtained prior to cardiac surgery. The blood samples were obtained after 12-h fasting and at some times after open cardiac surgery, centrifuged at 3800 g for 5 min, and the serums remained at less than 20^{o}C before review. All patients involved in the study were followed-up before hospital discharge. The follow-up included AKI clinical and laboratory follow-up.Sixty-five patients were included in the current study with creatinine mean ±SD 1.11±0.41 ; all patients were classified as acute kidney and non-acute kidney injury classes following cardiac surgery according to the above criteria. Patients with Chronic Kidney Disease (CKD) experience were sub-grouped into (AKI and non-AKI) patients.

Surgical protocol

Both patients were treated by the same cardiac surgery team in the Cardiothoracic Department of the University Hospital of Zagazig. Surgical procedures and methods of anesthesia (including on-pump and off-pump) have been implemented as per local practice.

Fluid regime pre- operative and post-operative

fluid protocol was used as the following: prior to surgery, patients were fasted for 12 h and injected with 100 mL / h of saline fluid during the 8 h pre-operative cycle. Patients with DM were treated with 500mL 10 per cent dextrose + 16 IU insulin + 10mL 5 per cent potassium chloride. In the post-operative cycle, 5 per cent of 1000 ml dextrose + 16 IU insulin was infused at a dose of 100 mL/ h.

Statistical analyses

All statistical analyzes were conducted using SPSS 23 (SPSS Inc., Chicago, IL). Classical variables were defined as numbers and percentages and continuous variables as mean ± standard deviation or median with range.

Univariate analysis between groups was performed using either the Student's t-test or the Mann-Whitney rank-sum test for continuous variables and the chi-square test for categorical variables. Logistic regression analyzes have been performed to test the hypothesis that preoperative SUA is linked to postoperative AKI. Multivariate logistic regression analysis was used to determine the predictors for AKI growth. Comparisons between groups were tested for categorical variables with the Chi-square test or Fisher's exact test, and for numerical variables with Kruskal – Wallis and/or Mann – Whitney U-test. An study of the receptor operator characteristics curve (ROC curve) was performed to determine the sensitivity and specificity of uric acid in acute renal injury predictions.

Results

Sixty five patients were involved in this study, their ages ranged from 38 to 78 years with a mean 54.85±7.63 years. 44 patients (67.7%) were males and 21 patients (32.3%) were females. Common comorbidities that were prevalent were diabetes, hypertension, history of chronic kidney disease (CKD) and dyslipidemia (table 1). There were 24 patients (36.92%) who developedAKI and 41 patients (63.08%) did not develop AKI (non-AKI). The basic characteristics of patients who developed AKI and did not develop AKI (non-AKI) have been shown in (Table 1). There was no difference between the groups in terms of gender and BMI, but the mean age in the AKI group was substantially higher than in the non-AKI group. There was little distinction between the groups with respect to underlying chronic conditions except that the history of CKD, chronic heart failure and COPD were more prominent in the Acute Kidney Injury community compared to the non-Acute Kidney Injury community.There was no difference between the heart function classes (SBP, DBP and EF percent). Baseline pre-operative serum urea, creatinine and uric acid levels were significantly higher in AKI patients relative to non-AKI patients. Hemoglobin levels were also slightly lower in the AKI community relative to non-AKI. There was a substantial gap between the groups as to the average duration of hospital stay. Type of surgery didn't differ except that CABG + Valves was significantly lower in AKI group. Nevertheless, CPB time >60Min was substantially more frequent in AKI-developed patients compared to non-AKI patients (Table 1).

Patients with history of CKD (34 patient) were grouped into two subgroups, there were 21 patients (61.76%) who developed AKI and 13 patients (38.24%) did not develop AKI (non-AKI). There was no statistical disparity between the groups with respect to demographic features, laboratory functions, cardiac function, comorbidity, overall duration of hospital stay and type of operation, except that heart failure and history of cardiac surgery were more prevalent in non-AKI patients, but CABG was substantially more prevalent in patients with Acute Kidney Injury compared with non-Acute Kidney Injury patients and valve were significantly more in other group also uric acid was significantly higher among AKI (Table 2).

Another subgrouping of patients as regard mean serum uric acid was created (more than and less than 5). There was no difference between groups in terms of demographic characteristics, cardiac function, type of surgery and laboratory except that creatinine level significantly high in patients with Uric acid≥5 (mg/dl) compared with Uric acid<5 (mg/dl) patients (Table 3). Patients with AKI were more common with Uric acid≥5 (mg/dl) but not statistically significant different (Table 3).

In table (4) Multiple logistic regression analysis including Age≥55 years, History of CKD, urea (mg/dl), Creatinine (mg/dl), Uric acid ≥5(mg/dl), HB(g/dl) and Cardiopulmonary bypass >60 minutes as independent predictors of AKI in the studied patients (P<0.05) as a dependent predictors only History of CKD, Creatinine and uric acid were significant independent predictors

In table 5 and figure 1, ROC curve was calculated for uric acid values as a predictor of AKI in all patients and CKD patients. The area under the curve (AUC) was 0.74 and confidence interval CI 95% (0.53-0.89). By using the mean value of uric acid as a cut-off value to predict AKI in all patients, sensitivity was 75.5%, specificity of 84.37%, and (P=0.016).

Table 1: General characteristics of the study patients

Parameters	All patients, n=65	AKI, n=24	non AKI, n=41	P-value
	Mean±SD	Mean±SD	Mean±SD	
Age	54.85±7.63	57.87±7.02	53.02±7.47	0.012*
Gender (male/female)	44/21 (67.7%/32.3%)	14/10(58.3%/41.7%)	30/41(73.2%/26.8%)	0.17
BMI	24.11±2.08	24.65±1.93	23.79±2.12	0.10
Dyslipidemia	25 (38.5%)	10 (41.7%)	15 (36.6%)	0.68
DM	42 (64.6%)	16 (66.7%)	26 (63.41%)	0.79
Hypertension	41 (63.1%)	18 (75%)	23 (56.1%)	0.09
Pervious cardiac surgery	5 (7.7%)	2 (8.3%)	3 (7.3%)	0.88
Past history of CAD	12 (18.5%)	4 (16.7%)	8(19.5%)	0.77
Chronic heart failure	7 (10.8%)	5 (20.8%)	2 (4.87%)	0.002*
Peripheral vascular disease	8 (12.3%)	4 (16.7%)	4 (9.8%)	0.22
COPD	4 (6.2%)	3 (12.5%)	1 (2.4%)	0.02*
History of CKD	34 (52.3%)	21 (87.5%)	13(31.7%)	<0.001*
Caradiac function				
SBP (mmHg)	137.70±16.73	136.54±16.63	138.39±16.95	0.67
DBP (mmHg)	83.63±9.03	10.28±9.27	83.12±8.96	0.55
EF %	51.66±7.51	51.41±8.32	52.21±7.07	0.54
Renal functions				
Urea (mg/dL)	54.69±17.22	60.75±15.17	51.14±17.53	0.019*
Creatinine (mg/dL)	1.11±0.41	1.27±0.36	1.016±0.41	0.002*
Uric acid (mg/dL)	4.84±1.86	5.57±1.64	4.42±1.87	0.012*
Albumin (g/Dl)	4.06±0.53	3.97±0.39	4.11±0.55	0.28
HB (g/dl)	11.63±1.64	10.72±1.29	12.16±1.60	< 0.001*
Type of surgery				
CABG	45(69.23%)	19(79.20%)	26 (63.4%)	0 .18
Valves	12(18.46%)	4(16.7%)	8(19.50%)	0.77
CABG+ Valves	8(12.31%)	1(4.2%)	7(17.1%)	0.004*
On-Pump (CPB)	50 (76.9%)	19 (79.2%)	31 (75.6%)	0.74
CPB time >60Min.	16(24.6%)	12(50%)	4(9.80%)	< 0.001*
Aortic-cross clamp time, min	59±8.41	75.26±9.11	74.93±13.41	0.19
Total length of stay (days)	11.34±2.48	13.70±3.34	11.86±3.01	0.02*

Table 2: General characteristics of the CKD patients

Parameters	AKI in CKD n=21	non-AKI in CKD n=13	P-value
	Mean±SD	Mean±SD	
Age	57.38±7.15	56.38±8.18	0.71
Gender (male)	13/8 (61.9% / 38.1%)	11/2 (84.6% / 15.4%)	0.15
BMI	24.92±1.91	23.96±2.34	0.20
Dyslipidemia	9 (42.90%)	4 (30.80%)	0.18
DM	14 (66.70%)	6 (46.20%)	0.071
Hypertension	15 (71.4%)	9 (69.20%)	0.69
Pervious cardiac surgery	2 (9.50%)	3 (23.10%)	0.013*
Past history of CAD	3 (23.1%)	3(19.50%)	0.51
Chronic heart failure	2 (9.50%)	3 (23.10%)	0.013*
Peripheral vascular disease	2 (9.50%)	1 (7.70%)	0.85
COPD	3 (14.30%)	1 (7.70%)	0.12
Caradiac function			
SBP (mmHg)	136.04±16.62	137±18.18	0.69
DBP (mmHg)	84.19±9.73	85.69±10.28	0.67
EF %	52.61±7.71	51.92±7.87	0.80
Renal functions			
Urea (mg/dL)	61.85±15.50	63.38±19.25	0.80
Creat (mg/dL)	1.29±0.35	1.37±0.40	0.55
Uric acid (mg/dL)	5.89±1.66	4.72±1.65	0.035*
Albumin (g/Dl)	3.92±0.39	4.10±0.56	0.29
HB (g/dl)	10.60±1.27	11.28±1.47	0.21
Type of surgery			
CABG	17(81%)	6 (46.20%)	0.002*
Valves	3(14.3%)	4(30.8%)	0.015*
CABG+ Valves	1(4.8%)	3(23.10%)	0.0002**
On-Pump (CPB)	17 (81%)	9 (69.2%)	0.32
CPB time >60Min.	7(33.33%)	6(46.15%)	0.14
Aortic-cross clamp time, min	78.12±8.50	76.37±10.17	0.45
Total length of stay (days)	12.51±2.54	10.86±3.01	0.062

Table (3): Comparison between studied parameters in chronic kidney disease patients as regard mean serum uric acid levels

Parameters	Uric acid<5 (mg/dl) n=10	Uric acid≥5 (mg/dl) n=24	P-value
	Mean±SD	Mean±SD	
Age	55.10±6.27	57.79±7.87	0.34
Gender (male)	9/1 (90.0%/ 10.0%)	15/9 (62.5%/ 37.5%)	0.11
BMI	24.17±2.57	24.72±1.91	0.79
Renal functions			
Urea (mg/dL)	61.40±15.61	62.87±17.53	0.81
Creat (mg/dL)	1.014±0.49	1.45±0.20	0.001**
Albumin (g/Dl)	3.93±0.4	4.01±0.49	0.63
HB (g/dl)	11.21±1.41	10.78±1.35	0.41

Parameters	Uric acid<5 (mg/dl) n=10 Mean±SD	Uric acid≥5 (mg/dl) n=24 Mean±SD	P-value
Type of surgery			
CABG	8(80%)	15 (62.50%)	0.13
Valves	1(10%)	6(25%)	0.31
CABG+ Valves	1(10%)	3(12.5%)	0.83
On-Pump (CPB)	8 (80%)	18 (75%)	0.75
CPB time >60Min.	5 (50%)	8 (33.33%)	0.06
Aortic-cross clamp time, min	76.31±8.50	74.98±8.09	0.62
Acute kidney injury	6(60%)	15(62.5%)	0.89

Table (4): Multivariate analysis for predictors of AKI in the studied patients

Parameters	Odd's ratio	95% Confidence Interval	P-value
Age≥55 years	2.44	0.787- 14.89	0.245
History of CKD	18.89	1.45- 95.95	<0.001*
Urea (mg/dL)	4.778	0.951-8.041	0.087
Creatinine (mg/dL)	5.883	2. 011- 11.54	0.015*
Uric acid ≥5(mg/dL)	5.844	1.78 - 10.31	0.002*
HB(g/dl)	3.86	0.75-5.051	0.114
Cardiopulmonary bypass>60 min.	3.21	0.885-18.51	0.311

Table (5): Roc curve of uric acid as a predictor for AKI in all patients

Group	AUC	CI (95%)	Cut-off	Sensitivity	Specificity	Accuracy	P-value
All patients	0.74	0.534 - 0.899	5 mg/dl	75.5%	84.37%	77.58%	**0.016***
CKD patients	0.43	0.228 - 0.640	5.3 mg/dl	66.5%	50%	53.25%	**0.12**

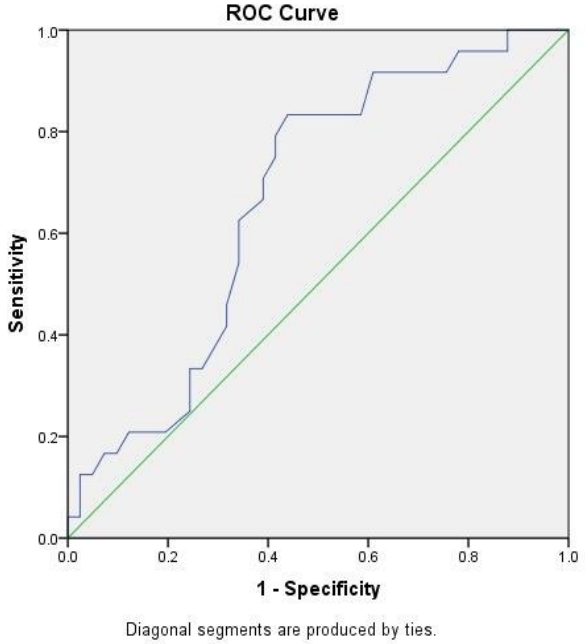

Figure (1): Roc curve of uric acid as a predictor for AKI in all patients

DISCUSSION

Cardiac surgery is a significant risk factor in the growth of AKI. Pre-existing renal disease and the difficulty of surgery are both high risk factors. The frequency of AKI after adult cardiac surgery is around 5-10% **[10]**. In a recent meta-analysis, the AKI incidence after heart surgery was 22.3% in the world (CI: 95%, 19.8-25.1) **[11]**.

Early identification of AKI is of great significance because the production of AKI is a significant burden on both patients and the health care system. In addition to a better understanding of certain risk factors linked to cardiac surgery-associated acute kidney injury**[12]**.

History of chronic kidney disease (CKD) ~~were~~ is ~~very~~ a common risk factor in development of postoperative AKI. These results have been consistent with other studies that have shown that pre-existing CKD raises the risk of acute kidney injury.

Ishani et al. reported that the incidence of AKI was 8.8% in CKD patients compared to 2.3% in non-CKD patients.**[13]**.

Pannu et al. stated that 18-fold increase in the risk of AKI patients with an eGFR< 30 ml/min/1.73 m^2 rather than with an eGFR> 60 ml / min/1.73 m^2 **[14]**.

In addition, an elevated SUA levels is found to be connected with a number of diseases such as atherosclerosis **Zhao et al. [17]**, hypertension **Susic and Frohlich[15]**, diabetes mellitus **Lytvyn et al. [7]**, metabolic syndrome **Billiet et al. [16]**, myocardial infarction **Yan et al. [18]**, and stroke**Kanbay et al. [19]**.

Preoperative rise in serum uric acid levels was associated with an increased risk of postoperative AKI in the current research. Patients with preoperative elevation of serum uric acid≥5 mg/dl had a significantly higher risk of postoperative AKI even after important preoperative and intraoperative maneuvers adjustment.Uric acid concentration preoperatively was connected with postoperative acute kidney injury significantly (odds ratio, 5.84; 95% confidence interval, 1.78 - 10.31; *P*=0.002). Recent laboratory and clinical findings show that elevated uric serum acid could be an independent risk factor for CKD AKI [**20]**.

Li et al stated that uric acid was considered a simple marker for chronic kidney disease. There is also evidence that uric acid may be an independent risk factor for developing chronic kidney disease.**[21]**.

Despite the strong associations between renal disease and elevated serum uric acid in several clinical conditions **Feig et al. [22]**, to date, some few studies have assessed the preoperative increased uric acid concentration

effect on postoperative AKI in patients undergoing heart surgery **[23]**. In **Ejaz et al** study, a complicated heart surgery was done for 58 patients; the preoperative serum uric acid concentration was found to be more than 6.0 mg/dL and connected with increased length of stay in hospital than preoperative SUA less than 6.0 mg/dL and a nearly fourfold increased risk of acute kidney injury **[24]**. In an another study, 190 patients undergoing heart surgery, multivariate analysis showed that a 35-fold risk was connected with patients of acute kidney injury whose SUA concentrations more than 7.0 mg/dL after adjustment for other variables, and also, increased the length of hospital stay and the duration of mechanical ventilation support **[23]**. Our study findings are in consistent with those studies and there were a similar relationship between preoperative serum uric acid and postoperative AKI but with levels slightly closer to previous studies. Also, **Lee et al, [25]**reported that serum uric acid levels preoperatively was significantly connected with acute kidney injury postoperatively (odds ratio, 1.18; 95% CI, 1.10-1.26; $P < 0.001$). There were a significant relation between postoperative acute kidney injury and preoperative serum uric acid which improves the prediction of AKI incidence.

Kanda et al,[26] reported that the association with kidney function loss and low serum uric acid (male <5 mg/dl; female <3.6 mg/dl) has a U-shaped relationship and also possible predictor for CKD.

In an other study, development of AKI and chronic kidney disease in a long period could be predicted by elevated levels of serum uric acid **[27]**. **Ejaz and coworkers** reported that the level uric acid concentration is an important marker for development of AKI in the future independent of major confounders, especially levels of baseline GFR**[28]**. Also, the same authors reported that in a retrospective study; Preoperative SUA was associated with increased occurrence and risk of AKI, higher postoperative serum creatinine levels, and increased hospital stay in patients undergoing cardiac surgery**[29]**.

Though DM, hypertension and dyslipidemia are known risk factor for kidney injury , but no statistically significant difference between Aki And non AKI group found in this study

In our study, multiple risk factors were independent predictors of AKI as determined by multivariate analysis and roc curve for uric acid including Age≥55 years, History of CKD, urea (mg/dl), Creatinine (mg/dl), Uric acid ≥5 (mg/dl), HB (g/dl) and Cardiopulmonary bypass bypass>60 minutes. The area under the curve (AUC) was 0.74 and confidence interval CI 95% (0.53-0.90). By using the mean value of uric acid as a cut-off value to predict AKI in all patients, sensitivity was 75.5%, specificity of 84.37%, and (P=0.016). Similarly, most of our findings were confirmed by study of **Mangos et al, [30]** and **Gaipov et al, [31]**they found that diabetes, COPD, peripheral vascular diseases, type of surgery, recurrent surgical interventions, cardiopulmonary bypass time, blood transfusions, decreased left ventricle function, advanced age, postoperative thrombocytopenia and leukocytosis, female sex, the use of medications that impair the self-regulation of renal blood flow (non-steroidal anti-inflammatory medications, diuretics, angiotensin-converting enzyme inhibitors are the most important risk factors for acute renal injury following cardiac surgery, etc.) as well as elevated preoperative uric acid and serum creatinine levels.

Lapsiaet al reported that in patients with SUA more than 5 mg/dL, the incidence of post-CABG acute kidney injury was elevated. These findings are in agreement with other study results that showing mildly elevated SUA concentrations within the normal range (≥5.5 mg/dL) have oxidant and inflammatory effects, and were connected with increased cardiovascular risk and AKI. This cut-off value is well lower than that usually used for hyperuricemia definition. **Lapsiaet al** results suggest that the incidence of postoperative acute kidney injury connected with preoperative increased SUA is not restricted to the normal range**[23]**.

The role of SUAin AKI is caused by multiple and interactive pathways. Uric acid

is known to cause increased IL-6 synthesis, diminishing of nitric oxide production, endothelial dysfunction, and vascular smooth muscle cell proliferation; this may contribute to the progress of CKD**[32]**.

Uric acid can cause AKI through a number of pathogenic mechanisms that can be direct tubular toxicity (crystal induced injury) **Roncal et al. [33]**or Indirect damage (inflammatory, due to oxidative stress, vasoconstriction, etc.). Uric acid has been developed in both human and animal models to limit the proliferation and migration of endothelial cells and to induce apoptosis and dysfunction of endothelial cells**[34]**. Vasoconstriction of renal vessels is one of the important risk factors for the development of acute renal injury to **Haase et al [35].** The expression of local chemokines is also induced by elevated uric acid, i.e. monocyte chemotactic protein 1 in the Kang **kidney[36**], and pro-inflammatory systemic cytokine i.e. TNF-α **[37]**. Oxidative damage of proximal tubule cells is induced by uric acid levels elevation through the nicotinamide adenine dinucleotide phosphate (NADPH) oxidase activation**[38]**.

There are no trials at present showed that decreasing levels of uric acid may be significant in acute kidney injury prevention. Reducing SUA by Allopurinol in patients with high SUA levels before cardiovascular surgery could improve postoperative cardiovascular outcomes through decreasing the oxidative stress **[39]**. However, the frequency of acute kidney injury after cardiac surgery in the previous study couldn't be prevented by allopurinol**[40]**.

Limitations; the sample size was relatively smalland it was a single-center study, the effects either hidden or unknown factors could not be excluded on the studied patients in spite of presence of several variables. Many medications used to treat our patients as angiotensin receptor blocker or angiotensin-converting enzyme inhibitors may also have an effect on preoperative SUA levels**[29]**.

Conclusions

Preoperative uric acid was significantly conntected to the postcatrdiac surgery AKI. Our study detect serum uric acid cut off level ≥ 5mg/dl as risk factor for AKI.though serum uric acid is within normal range ,possible underlying pathology which precipitate AKI.Further studies needed to detrmine cutoff level in both male and female and correlation with excretion of uric acid in urine to assess the benefit of preoperative lowering serum uric acid in patients with cardiac surgery to prevent postoperative AKI .

Declaration of interest

The authors report no conflicts of interest. The authors along are responsible for the content and writing of the paper.

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