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## Relation of uric acid and contrast-induced nephropathy in patients undergoing percutaneous coronary intervention

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### Abstract

**Background:** As a straightforward laboratory test, serum uric acid (SUA) measurement could be easily carried out at low cost. Previously, it has been suggested as an independent CIN predictor among those having ST-elevation myocardial infarction (STEMI) who underwent treatment with primary percutaneous coronary intervention (PCI). This work was designed to investigate whether SUA levels could be a good predictor for CIN occurrence following PCI.

**Methods:** Our team designed a prospective observational study, involving 100 cases with ages ranging between 18 to 80 years, both genders, who underwent elective PCI procedure. All participants were equally categorized into two groups based on SUA level when admitted: Group A: SUA  $\leq$  7.0 mg/dl while group B: SUA above 7.0 mg/dl.

**Results:** Serum urea and SUA, baseline Serum creatinine level and serum creatinine clearance showed insignificant variance among both groups (CIN developed and not developed group). Nevertheless, a significant variance was noted in creatinine levels in addition to creatinine clearance at 24 hours, 48 hours and 72 hours among both groups. Angiographic and procedural characteristic (Number of vessels, Number of stents, Procedural duration and contrast volume exhibited insignificant variance among both groups. Medications along with the CIN incidence were insignificantly varied among both groups.

**Conclusion:** The CIN frequency of occurrence exhibited no variation among cases having normal and elevated uric acid, which is probably due to the presence of cases with reduced risk as well as interventions with sufficient preventive measures. Nevertheless, hyperuricemia was correlated with deteriorated kidney function both prior to and following the procedure.

**Keywords:** Uric acid, contrast-induced nephropathy, percutaneous coronary intervention, creatinine

### Introduction

Contrast-induced nephropathy (CIN) is a recognized early consequence, occurring after cardiac catheterization operations as a result of utilizing contrast agents containing iodine [1]. CIN incidence has been linked to higher rates of morbidity, mortality, prolonged hospital stays, in addition to the progression to end-stage renal disease among individuals with high susceptibility. Approximately 1% of cases developing CIN will need dialysis, while almost half of them will ultimately develop end-stage renal failure [2].

CIN is characterized by a rise in serum creatinine levels over 25% or more than 0.5 mg/dl (44  $\mu$ mol/l) of serum creatinine from baseline value in 48 – 72 hours after procedure in the absence of other etiologies for acute renal impairment [3].

The peak often occurs within three to five days, then recovers to its baseline levels within ten to fourteen days. The overall CIN occurrence among the general population has reached 1-6% [4]. The exact pathophysiological process behind the development of CIN yet to be completely understood. The possible causes of this condition include changes in the blood flow to the kidneys, oxygen free radicals inducing damage, direct harmful effect of contrast media on the cells of kidney tubules, or constriction of the small blood vessels in the kidneys [5]. As a straightforward laboratory test, serum uric acid (SUA) measurement could be easily carried out at low cost. Previously, it has been suggested as an independent CIN predictor among those having ST-elevation myocardial infarction (STEMI) who underwent treatment with primary percutaneous coronary intervention (PCI) [6].

SUA, a catabolic byproduct of purines, has been thorough investigation as a potential risk factor of cardiovascular disease. Hyperuricemia is linked to the suppression of nitric oxide production, the stimulation of the local renin-angiotensin system, the activation of pro-inflammatory and proliferative markers, along with the increased production of reactive oxygen species, resulting in oxidative stress as well as impaired kidney function [7]. This work was designed to investigate whether SUA levels could be a good predictor for CIN occurrence following PCI.

### Patients and Methods

Our team designed a prospective observational study, involving 100 cases with ages ranging between 18 to 80 years, both genders, who underwent elective PCI procedure. The utilized contrast agent was non-ionic, low osmolality "Iohexol" as convenient standard in catheterization laboratories. The study's timeframe fell between October 2020 till October 2022 after it got approved from the Ethical Committee Tanta University Hospitals, Tanta, Egypt. We asked all the participants to sign a written consent.

Our team excluded all cases having an Estimated Glomerular Filtration Rate (eGFR) below sixty ml/min/1.73m<sup>2</sup>, cardiogenic shock, ejection fraction below forty percent, history of end-stage renal failure in addition to those being on dialysis. We also excluded those who were administered with IV contrast in the last fourteen days or developed previous contrast-induced adverse reactions.

All participants were equally categorized into two groups based on SUA level when admitted: Group A: SUA ≤ 7.0 mg/dl while group B: SUA above 7.0 mg/dl.

Firstly, we took a detailed medical history from all cases, then clinical examination was performed. All laboratory investigations were obtained, including [complete blood count (CBC), random blood sugar as well as kidney functions (SUA, serum urea, serum creatinine, creatinine clearance in addition to eGFR. Measuring serum creatinine was done at 24, 48 and 72 hr. after the procedure] and radiological investigations [Standard 12-lead ECG and resting transthoracic echocardiography (TTE)].

### Resting Transthoracic Echocardiography (TTE)

A detailed transthoracic ECG was obtained from all

participants utilizing GE Vivid 9.

**These measurements were as follows:** LV Wall Thickness, internal dimensions, LV Ejection Fraction (EF) by biplane 2D Echo and fractional shortening (FS).

### Percutaneous coronary intervention

All patients who are candidates for elective PCI received the loading dose of aspirin and clopidogrel before the procedure. The interventional cardiologist had the choice to choose the access site for PCI (either femoral or radial) and choose the number of vessels to be accessed. After the decision of coronary intervention. The utilized contrast medium was-ionic, low osmolality. The volume of contrast used during the procedure for every patient was estimated. After the procedure, every case was admitted to the coronary care unit.

### Measurements

Serum creatinine at baseline and after 24.48 and 72hrs. CrCl at baseline and after 24.48 and 72hrs. Baseline Uric acid level. Angiographic and procedural characteristics (Procedural duration, contrast volume, hydration volume, and number of vessels and stents). Medication (angiotensin-converting enzyme (ACE) or angiotensin receptor blockers (ARB), Beta blocker (BB), Calcium channel blockers (CCB) as well as Antihyperglycemic).

### Statistical analysis

Our team analyzed the data statistically utilizing SPSS v26 (IBM Inc., Chicago, IL, USA). Quantitative variables were illustrated through mean and standard deviation (SD). Comparisons among the two groups were made utilizing unpaired Student's t-test. Qualitative variables were showcased through frequency and percentage (%). Then, analysis was conducted utilizing the Chi-square or Fisher's exact test when appropriate. A two-tailed P value of less than 0.05 was deemed statistically significant.

### Results

Demographic data, anthropometric measurement in addition to risk factors were insignificantly different among both groups. Table 1.

**Table 1:** Comparison among both groups based on demographic data, anthropometric measurement along with risk factors

		Group A (n = 50)	Group B (n = 50)	P
Age (years)		56.18±9.70	56.28±7.45	0.954
Sex	Male	33(66.0%)	38(76.0%)	0.271
	Female	17(34.0%)	12(24.0%)	
Height (m)		1.72±0.09	1.74±0.07	0.103
Weight (Kg)		89.72±10.28	92.98±9.70	0.106
BMI (kg/m <sup>2</sup> )		30.37±2.33	30.58±2.75	0.676
Risk factor	Smoking	18(36.0%)	20(40.0%)	0.680
	DM	21(42.0%)	25(50.0%)	0.422
	HTN	34(68.0%)	31(62.0%)	0.529
	IHD	20(40.0%)	24(48.0%)	0.420
	Family history	15(30.0%)	13(26.0%)	0.656
	Hyperlipidemia	19(38.0%)	21(42.0%)	0.683

Data are illustrated through mean ± SD or frequency (%). BMI: Body mass index, DM: diabetes mellitus, HTN: hypertension, IDH: ischemic heart disease

Hematological data (Hb, WBCs, PLT and RBS), serum urea, baseline creatinine and creatinine levels and creatinine clearance and creatinine clearance at 24 hours, 48 hours and

72 hours were insignificantly different between the two studied groups. SUA showed significant variation among both groups ( $p < 0.05$ ). Table 2.

**Table 2:** Comparison among both groups based on hematological data, renal function, creatinine level in addition to creatinine clearance

	Group A (n = 50)	Group B (n = 50)	p	
Hb (g/dl)	13.96±1.40	14.11±1.17	0.558	
WBCs (10 <sup>3</sup> /mm <sup>3</sup> )	8.46±1.59	9.03±1.72	0.092	
PLT (10 <sup>3</sup> /mm <sup>3</sup> )	202.22±49.51	214.92±38.76	0.156	
RBS (mg/dl)	164.88±22.75	171.4±15.55	0.098	
Renal function	Urea	37.28±5.35	38.92±5.71	0.142
	Uric acid	6.19±0.36	8.19±0.79	<0.001*
Creatinine level	Baseline	0.86±0.12	0.87±0.15	0.669
	24 hours	0.88±0.14	0.91±0.20	0.290
	48 hours	0.89±0.18	0.96±0.30	0.174
	72 hours	0.91±0.24	0.99±0.37	0.226
Creatinine clearance	Baseline	123.4±29.0	128.04±27.13	0.413
	24 hours	121.2±30.14	123.2±29.28	0.736
	48 hours	120.5±31.55	120.3±31.80	0.979
	72 hours	119.3±32.4	119.2±33.2	0.987

Data are illustrated through mean ± SD. \* Significant p value <0.05. Hb: hemoglobin, TLC: total leukocytic count, WBCs: white blood cells, RBS: random blood sugar

Angiographic and procedural characteristic (Number of vessels, Number of stents, Procedural duration and contrast volume exhibited insignificant variance among both groups. Table 3.

**Table 3:** Comparison among both groups based on Angiographic and procedural characteristic

	Group A (n = 50)	Group B (n = 50)	p
Number of vessels	1.40±0.57	1.38±0.49	0.961
1	32(64.0%)	31(62.0%)	MC <sub>p</sub> = 0.438
2	16(32.0%)	19(38.0%)	
3	2(4.0%)	0(0.0%)	
Number of stents	2.0±0.86	2.10±0.76	0.373
1	14(28.0%)	10(20.0%)	MC <sub>p</sub> = 0.432
2	26(52.0%)	27(54.0%)	
3	6(12.0%)	11(22.0%)	
4	4(8.0%)	2(4.0%)	
Procedural duration	43.20±9.41	44.10±9.30	0.631
Contrast Volume	175.0±48.71	190.0±44.03	0.109

Data are illustrated through mean ± SD or frequency (%). MC: Monte Carlo

Medications (ACE/ARB, BB, CCB and Anti-hyperglycemic) and incidence of CIN were insignificantly different among both groups. Table 4.

**Table 4:** Comparison among both groups based on medications and CIN

	Group A (n = 50)	Group B (n = 50)	p
ACE/ARB	22(44.0%)	30(60.0%)	0.109
BB	32(64.0%)	34(68.0%)	0.673
CCB	13(26.0%)	11(22.0%)	0.640
Anti-hyperglycemic	21(42.0%)	25(50.0%)	0.422
CIN	2(4.0%)	5(10.0%)	0.436

Data are illustrated through frequency (%). ACE: Angiotensin-converting enzymes, ARB: Angiotensin-receptor blocker, BB: Beta blocker, CCB: Calcium channel blocker, CIN: Contrast-induced nephropathy

Serum urea and SUA, baseline Serum creatinine level and serum creatinine clearance showed insignificant variance among both groups (CIN developed and not developed group). Nevertheless, a significant variance was noted in creatinine levels in addition to creatinine clearance at 24 hours, 48 hours and 72 hours among both groups. Table 5.

**Table 5:** Relation between CIN along with renal function, serum creatinine and creatinine clearance in total sample

	CIN		P
	No (n = 93)	Yes (n = 7)	
Urea (mg/dl)	38.04±5.59	38.86±5.58	0.711
Uric acid(mg/dl)	7.15±1.15	7.81±1.49	0.148
<b>Serum Creatinine</b>			
Baseline	0.86±0.13	0.93±0.15	0.169
24 hours	0.87±0.14	1.28±0.14	<0.001*
48 hours	0.87±0.14	1.64±0.23	<0.001*
72 hours	0.88±0.15	1.92±0.33	<0.001*
<b>Creatinine clearance</b>			
Baseline	126.92±28.20	109.93±21.38	0.123
24 hours	125.2±28.45	82.72±8.87	<0.001*
48 hours	124.5±28.54	65.62±11.51	<0.001*
72 hours	123.9±28.59	57.13±14.17	<0.001*

Data are illustrated through mean ± SD. \* significant p value <0.05, CIN: Contrast-induced nephropathy

## Discussion

Coronary artery disease remains the leading cause of death in nations with advanced economies. Nevertheless, the expanded use of revascularization techniques, particularly in cases developing acute myocardial infarction, has significantly contributed to the notable decline in death rates seen within the recent decades. Thanks to advancements in stent technology, an increasing number of patients are undergoing percutaneous revascularization each year. This includes a bigger percentage of high-risk individuals, which involves cases of compromised renal function<sup>[8]</sup>.

In this research, seven percent of cases developed CIN (2% within group A while 5% within group B). Additionally, no significant variation was seen within the high uric acid group. Similarly, a prior research conducted by Abdollah *et al.*<sup>[9]</sup> highlighted that the increased uric acid were linked to a greater likelihood of CIN among cases having STEMI who exhibited normal blood creatinine levels following PCI. Our findings aligned with those of Mirbolouk *et al.*<sup>[10]</sup> who investigated the correlation between CIN and SUA levels. Approximately sixteen cases (7.5%) exhibited CIN. Out of the total of 16 individuals, 7 (8.04%) belonged to the high uric acid group, whereas 9 (7.2%) belonged to the normal uric acid group. No significant variation was noted among both groups. This prospective study involved 211 cases who were hospitalized for elective coronary angiography or

angioplasty. The researchers assessed serum creatinine and uric acid levels upon admission, and then reevaluated creatinine levels 48 hours and seven days postoperatively.

The disagreement may be addressed by the fact that the cases involved in the current research exhibited a usually decreased level of risk, in addition to having adequate renal function, since we specifically excluded those having an estimated glomerular filtration rate (eGFR) below 60 ml/min/1.73m<sup>2</sup>. This research did not include any cases of emergency PCI and specifically excluded individuals developing AMI. The interventions or treatments were voluntary. Additionally, sufficient hydration were provided to all cases for prophylactic purposes prior to coronary angiography.

Similarly, this research repeated measuring creatinine as well as GFR levels after 7 days then found that CIN cases continued to exhibit elevated creatinine and reduced GFR levels even one week postoperatively. The study demonstrated the prolonged contrast impact on renal function. In the case of hyperuricemia, this research addressed that this group exhibited a significant increase in creatinine in addition to reduced GFR after a period of seven days. Nevertheless, this research could not prove that increased uric acid can influence this finding. In fact, the hyperuricemia cases exhibited greater baseline creatinine along with reduced GFR. Additionally, such variations persisted 48 hours and seven days following the surgery. Our results came in consistence with the ones documented by Mirbolouk *et al.* [10].

Uric acid is the last byproduct of the breakdown of purines and is eliminated from the body via the kidneys. It induces nitric oxide inactivation, which is a relaxing agent generated from the endothelium. Consequently, increased uric acid levels may cause damage to the inner lining of blood vessels along with narrowing of the blood vessels in the kidneys. In addition, hyperuricemia speeds up the activation of proinflammatory pathways and the vascular smooth muscle cells proliferation, leading to endothelial dysfunction. Acute kidney injury may arise in cases of acute urate nephropathy with crystal-dependent pathways [11].

Limitations of the study included a modest sample size, and it was conducted at a single center. The study's timeframe was relatively short. It also involved cases with a reduced risk, so it is challenging to make generalizations based on the findings. The research did not perform complete renal function analysis.

### Conclusion

The CIN frequency of occurrence exhibited no variation among cases having normal and elevated uric acid, which is probably due to the presence of cases with reduced risk as well as interventions with sufficient preventive measures. Nevertheless, hyperuricemia was correlated with deteriorated kidney function both prior to and following the procedure.

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**Conflict of Interest:** Nil.

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