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The Usefulness of Colchicine in the Prevention of Contrast-induced Acute Kidney Injury in Patients Undergoing Elective Percutaneous Coronary Intervention

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Contrast-induced acute kidney injury (CIAKI) it is an important complication of diagnostic and interventional procedures in cardiology. This work aimed to to investigate the preventive role of colchicine on CI-AKI in patients undergoing elective PCI considering to its anti-inflammatory and renoprotective effects.
Methods: This study was conducted on 400 patients who were admitted for elective PCI, and was carried out in CCU in Cardiology Department, Benha university hospital and National Heart Institute.

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The patients were equally divided into two equal groups: Group 1 (Case group): received colchicine plus standard anti ischemic treatment and croup 2 (Control group): received placebo plus standard anti ischemic treatment. All patients were subjected to standard 12-lead ECG, laboratory investigations encompassing kidney function tests 24 hours before the procedure and 48 hours after, random blood sugar, coagulation profile, liver function tests. and serum electrolytes and transthoracic echocardiography.

Results: There was a significant positive correlation between Serum creatinine and diabetes mellitus (r=0.245, P=0.001) and eGFR (r= 0.174, P<0.001). The multivariate regression analysis revealed that age, Colchicine use, diabetes mellitus, post CAG creatinine and post CAG eGFR, were the only significant predictors of incidence of CI-AKI.

Conclusions: Colchicine, when administered in conjunction with standard anti-ischemic treatment in patients undergoing elective PCI, significantly reduces the incidence of CI-AKI. The reduction in CI-AKI incidence not only has implications for patient outcomes but also reveals an important strategy in optimizing renal protection during PCI.

Keywords: Usefulness; colchicine; prevention; contrast-induced acute kidney injury, elective; PCI.

1. INTRODUCTION

"Contrast induced acute kidney injury (CI-AKI) is a serious complication of percutaneous coronary interventions (PCI) and is associated with increased morbidity and mortality" [1]. "Colchicine has long been used in the treatment of many inflammatory diseases such as acute flares of gouty arthritis, Behçet's disease, familial mediterranean fever (FMF), and pericarditis. Its main anti-inflammatory properties are likely associated with downregulation of cell surface adhesion molecules and decreased neutrophil adhesion and migration, through binding to tubulins and blocking the polymerization of microtubules" [2].

"CI-AKI is an important complication of diagnostic and interventional procedures in cardiology practice" [3]. "The incidence of CI-AKI varies between 2% and 30% depending on the definition of kidney injury, type of procedure and the presence of risk factors. CI-AKI is the third most common cause of hospital-acquired renal insufficiency and is also associated with a prolonged hospital stay, mortality, and increased costs" [4]. "The exact pathophysiological mechanism of CI-AKI is not well-defined and includes complex cascades of events. The medullary vasoconstriction and hypoxia, direct renal tubular cytoxicity of contrast media (CM), increased oxidative stress, free radical damage via leukocytes and impairment of tubuloglomerular feedback are the proposed pathophysiological mechanisms of CI-AKI" [5].

"In the literature, various strategies and drugs have been tested in attempts to prevent CI-AKI. To identify patients at increased risk of CI-AKI is the initial step in the prevention of CI-AKI. Patients with pre-existing chronic kidney disease (CKD), T2DM, female gender and impaired left ventricular functions are more susceptible to CI-AKI. According to the current knowledge, minimizing the contrast volume, using low or iso-osmolar contrast agents, periprocedural intravenous hydration and high dose statin treatment are the most effective preventive measures. Currently, multiple interventions to prevent CI-AKI are still under investigation" [6].

The aim of this study was to investigate the preventive role of colchicine on CI-AKI in patients undergoing elective PCI considering to its anti-inflammatory and renoprotective effects.

2. METHODS

This multicenter prospective interventional study was conducted on 400 patients who were admitted for elective PCI, and was carried out in CCU in Cardiology Department, Benha university hospital and National Heart Institute from July 2023 to January 2024.

The inclusion criteria were patients admitted for elective percutaneous coronary intervention (PCI), including both sexes, and those with an estimated glomerular filtration rate (eGFR) of 45 mL/min/1.73 m^2 or higher, ensuring relatively preserved renal function at baseline.

The exclusion criteria were patients with an eGFR below 45 mL/min/1.73 m^2, with history of contrast media exposure in the last 10 days, with known contrast allergy, or those with acute coronary syndrome.

2.1 Grouping

The patients were equally divided into two equal groups; group 1 (Case group n=200): patients

received colchicine plus standard anti ischemic treatment and group 2 (Control group n=200): patients received placebo plus standard anti ischemic treatment.

All patients were subjected to data collection including full history taking, covering general characteristics such as age and gender; risk factors, which included a history of cigarette smoking, systemic hypertension, diabetes mellitus, hyperlipidemia, family history of premature CAD, known contrast allergy, and past history of CAD or previous PCI; and a standard 12-lead ECG was also performed to detect heart rate and the number of leads with pathological Q-waves and QRS complex width.

Laboratory investigations were conducted, encompassing kidney function tests (urea, creatinine, and eGFR) 24 hours before the procedure and 48 hours after, random blood sugar, coagulation profile (prothrombin time, prothrombin concentration, and partial thromboplastin time), liver function tests (serum total bilirubin, serum direct bilirubin, serum albumin, ALT, AST), and serum electrolytes (sodium and potassium).

Transthoracic echocardiography (TTE) was performed using a Philips Sonos 7500 device (Philips, Andover, MA, USA) and Philips EPIQ elite during the course of hospital admission to evaluate left ventricular systolic function. Image acquisitions and measurements followed the recommendations of the European Association of Echocardiography and the American Society of Echocardiography.

The details of PCI included the radial versus femoral approach, the number of vessels treated, the amount of dye used, and the calculation of Gensini and Syntax scores for all patients [7,8].

In course during hospital admission serum creatinine and eGFR, for CI-AKI were measured before the procedure and 48 hours post-procedure to assess the occurrence of CI-AKI. CI-AKI is defined as an increase in serum creatinine by more than 0.3 mg/dL or a 50% relative increase from baseline within 48 hours after contrast media exposure.

All adverse events occurring during the follow-up period were recorded systematically, including any clinical complications, symptoms, or abnormalities observed or reported by the patients. The adverse events were classified and analyzed to evaluate the safety profile of the treatments administered.

2.2 Statistical Analysis

Statistical analysis was done by SPSS v28 (IBM Inc., Armonk, NY, USA). Quantitative variables were presented as mean and standard deviation (SD) andcompared between the two groups utilizing unpaired Student's t- test. Qualitativevariables were presented as frequency and percentage (%) andwereanalysed utilizing the Chi-square test or Fisher's exact test when appropriate.Atwo tailed P value < 0.05 was considered statistically significant. Pearson or spearman correlation was done to estimate the degree of correlation between two quantitative variables. Multivariate logistic regression was also used to estimate the relationship between a dependent variable and more independent variables. Evaluation of diagnostic performance was performed by evaluation diagnostic sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV). Receiver Operating Characteristic curve (ROC-curve) analysis: The area under the curve (AUC) evaluates the overall test performance.

3. RESULTS

Table 1 show that There was an insignificant difference between the studied groups regarding the baseline characteristics, risk factors and the clinical examination of vital signs.

Table 2 show that There was an insignificant difference between both groups regarding clinical examination of vital signs, total cholesterol, triglycerides, HDL, LDL levels. and LVEF.

Table 3 show that Post CAG creatinine, was significantly lower in group 1 (case group) compared to group 2 (control group) ($P<0.001$), and the rate reduction of post CAG eGFR was significantly lower in group 1 (case group) compared to group 2 (control group) ($P<0.004$) with no significant difference regarding baseline creatinine and eGFR.

Table 4 show that There was an insignificant difference between the studied groups regarding the angiographic finding.

Table 1. Baseline characteristics, risk factors of the studied groups, of the studied groups

	Group 1 (Case group) (n=200)	Group 2 (Control group) (n=200)	P value
Age (years)	55.5 ± 8.87	54.3 ± 9.18	0.169
Sex Male	141 (70.5%)	126 (63%)	0.111
Female	59 (29.5%)	74 (37%)	
Weight (Kg)	76.2 ± 10.5	75.4 ± 9.57	0.468
Height (m)	1.69 ± 0.05	1.70 ± 0.05	0.113
BMI (Kg/m^2)	26.6 ± 3.84	26.1 ± 3.73	0.217
Current smoking	69 (34.5%)	62 (31%)	0.456
Hypertension	125 (62.5%)	118 (59%)	0.473
Diabetes mellitus	86 (43%)	97 (48.5%)	0.269
Hyperlipidemia	80 (40%)	74 (37%)	0.537
Past history of CAD	68 (34%)	57 (28.5%)	0.234

Data presented as mean ± SD or frequency (%), BMI: body mass index. CAD: coronary artery disease, HF: heart failure. HR: heart rate

Table 2. Clinical examination of vital signs, lipid profile and Echocardiography of the studied groups

	Group 1 (Case group) (n=200)	Group 2 (Control group) (n=200)	P value
HR (beats/min)	84.9 ± 6.25	84.3 ± 5.38	0.296
SBP (mmHg)	139.5 ± 11.89	140.7 ± 12.58	0.328
DBP (mmHg)	81.80 ± 7	80.95 ± 7.41	0.239
Total cholesterol (mg/dL)	143.2 ± 27.26	147.5 ± 31.43	0.142
Triglycerides (mg/dL)	175.6 ± 44.17	177 ± 45.48	0.764
HDL (mg/dL)	52.9 ± 10.69	54.2 ± 10.49	0.240
LDL (mg/dL)	102.2 ± 18.16	100.2 ± 16.56	0.260
LVEF (%)	52.03 ± 4.65	51.25 ± 4.21	0.077

Data presented as mean ± SD, SBP: systolic blood pressure, DBP: diastolic blood pressure HDL: high density lipoprotein, LDL: low density lipoprotein. LVEF: left ventricular ejection fraction

Table 3. Creatinine and eGFR of Group 1, Creatinine and eGFR of the studied groups

	Group 1 (Case group) (n=200)		
	Baseline	**Post CAG**	**P value**
Creatinine (mg/dL)	0.85 ± 0.08	0.95 ± 0.14	<0.001*
eGFR (mL/min)	70.12 ± 15.30	67.1 ± 14.51	0.022*
	Group 2 (control group) (n=200)		
	Baseline	Post CAG	P value
Creatinine (mg/dL)	0.85 ± 0.09	1.11± 0.16	<0.0001*
eGFR (mL/min)	0.70-1	0.8-1.4	0.004*
	Group 1 (Case group) (n=200)	Group 2 (Control group) (n=200)	P value
Baseline creatinine (mg/dL)	0.85 ± 0.08	0.85 ± 0.09	0.339
Post CAG creatinine (mg/dL)	0.95 ± 0.14	1.11 ± 0.16	<0.001*
Baseline eGFR (mL/min)	70.12 ± 15.30	72.6 ± 13.14	0.274
Post CAG eGFR (mL/min)	67.1 ± 14.51	63.1 ± 12.79	0.004*

*Data presented as mean ± SD, CAG: coronary angiography, eGFR: estimated glomerular filtration rate, *: statistically significant as p value <0.05. CAG: coronary angiography, eGFR: estimated glomerular filtration rate, *: statistically significant as p value <0.05*

Table 5 show that There was an insignificant difference between both groups regarding the other adverse events (chest pain, hypersensitivity reaction, access site discomfort, fever, Ischemic stroke, Fluid overload, Bleeding and Palpitations). Concerning the outcome, CI-

AKI occurred in 5 (2.5%) patients in group 1 (case group) and 15 (7.5%) patients in group 2 (control group). The incidence of CI-AKI was significantly lower in group 1 (case group) compared to group 2 (control group) (P= 0.038).

Table 6 show that There was a significant positive correlation between Serum creatinine and diabetes mellitus (r=0.245, P=0.001) and eGFR (r= 0.174, P<0.001). There was an insignificant correlation between Serum creatinine and the other parameters.

Table 7 show that the multivariate regression analysis revealed that age, Colchicine use, diabetes mellitus, post CAG creatinine and post CAG eGFR, were the only significant predictors of incidence of CI-AKI.

Table 4. Angiography data and findings of the studied groups

		Group 1 (Case group) (n=200)	Group 2 (Control group) (n=200)	P value
Contrast volume (ml)		150.4 ± 27.78	154.6 ± 32.12	0.158
Number of stents		2.01 ± 0.78	2.07 ± 0.82	0.493
Total stent length (mm)		28.3 ± 5.99	27.7 ± 6.65	0.390
Stent diameter (mm)		3.30 ± 0.47	3.26 ± 0.45	0.437
Initial TIMI flow grade	TIMI 2	141 (70.5%)	129 (64.5%)	0.200
	TIMI 3	59 (29.5%)	71 (35.5%)	
Final TIMI flow grade	TIMI 2	27 (13.5%)	40 (20%)	0.082
	TIMI 3	173 (86.5%)	160 (80%)	
Syntax score		21.9 ± 5.44	21.2 ± 4.88	0.203
Gensini score		35.7 ± 8.25	35.9 ± 8.96	0.821
Number of coronary arteries revascularized	1	85 (42.5%)	76 (38%)	0.640
	2	61 (30.5%)	64 (32%)	
	3	54 (27%)	60 (30%)	
Access	Radial	162 (81%)	152 (76%)	0.223
	Femoral	38 (19%)	48 (24%)	

*Data presented as mean ± SD or frequency (%), TIMI: thrombolysis in myocardial infarction, *: statistically significant as p value <0.05*

Table 5. Adverse events of the studied groups during the course of the hospital admission and Outcome of the studied groups

	Group 1 (Case group) (n=200)	Group 2 (Control group) (n=200)	P value
Chest pain	29 (14.5%)	34 (17%)	0.492
Hypersensitivity reaction	3 (1.5%)	3 (1.5%)	1.0
Access site discomfort	2 (1%)	1 (0.5%)	1.0
Fever	0 (0%)	0 (0%)	--
Ischemic stroke	0 (0%)	0 (0%)	--
Fluid overload	0 (0%)	0 (0%)	--
Bleeding	1 (0.5%)	2 (1%)	1.0
Palpitations	0 (0%)	0 (0%)	--
CI-AKI	5 (2.5%)	15 (7.5%)	0.038*

Data presented as frequency (%) CI-AKI: contrast-induced acute kidney injury,: statistically significant as p value <0.05*

Table 6. Correlation between serum creatinine and different parameters

	Serum creatinine (mg/dL)	
	r	P
Age (years)	0.001	0.982
Sex	0.018	0.714
BMI (Kg/m^2)	0.015	0.767
Diabetes mellitus	0.245	0.001*
HR (beats/min)	0.011	0.829
SBP (mmHg)	0.095	0.056

	Serum creatinine (mg/dL)	
	r	P
DBP (mmHg)	-0.089	0.076
Total cholesterol (mg/dL)	0.077	0.125
Triglycerides (mg/dL)	-0.003	0.950
HDL (mg/dL)	0.072	0.151
LDL (mg/dL)	-0.001	0.980
eGFR (mL/min)	0.174	<0.001*
LVEF (%)	0.055	0.271
Contrast volume (ml)	0.076	0.127
Number of stents	-0.098	0.051
Total stent length (mm)	0.001	0.984
Stent diameter (mm)	-0.036	0.475
TIMI flow grade	-0.095	0.058
Syntax score	-0.026	0.605
Gensini score	-0.024	0.626

*Data presented as mean ± SD or frequency (%), BMI: body mass index, HR: heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, HDL: high density lipoprotein, LDL: low density lipoprotein, eGFR: estimated glomerular filtration rate, LVEF: left ventricular ejection fraction, TIMI: thrombolysis in myocardial infarction, r: correlation coefficient, *: statistically significant as p value <0.05*

Table 7. Multivariate logistic regression analysis for prediction of the incidence of CI-AKI

	OR	95% CI	P value
Age (years)	1.0593	1.0145 to 1.1062	0.009*
Colchicine use	0.2443	0.0893 to 0.6678	0.006*
Diabetes mellitus	0.1519	0.0338 to 0.6839	0.014*
Post CAG creatinine (mg/dL)	17.3799	1.3476 to 224.145	0.028*
Post CAG eGFR (mL/min)	1.0768	1.0177 to 1.1392	0.010*
LVEF (%)	1.0428	0.9533 to 1.1407	0.360
Contrast volume (ml)	1.0489	0.9593 to 1.1467	0.294
Number of stents	1.0305	0.4493 to 2.3631	0.944
Total stent length (mm)	1.0543	0.9810 to 1.1332	0.150
Stent diameter (mm)	1.2748	0.2413 to 6.7342	0.775
Final TIMI flow grade	0.7261	0.2596 to 2.0312	0.542
Syntax score	1.0144	0.9408 to 1.0937	0.710
Gensini score	0.9944	0.9660 to 1.0237	0.705
Number of coronary arteries revascularized	0.8762	0.5222 to 1.4703	0.617

*Data presented as mean ± SD or frequency (%), OR: odds ratio, CI: confidence interval, eGFR: estimated glomerular filtration rate, *: statistically significant as p value <0.05*

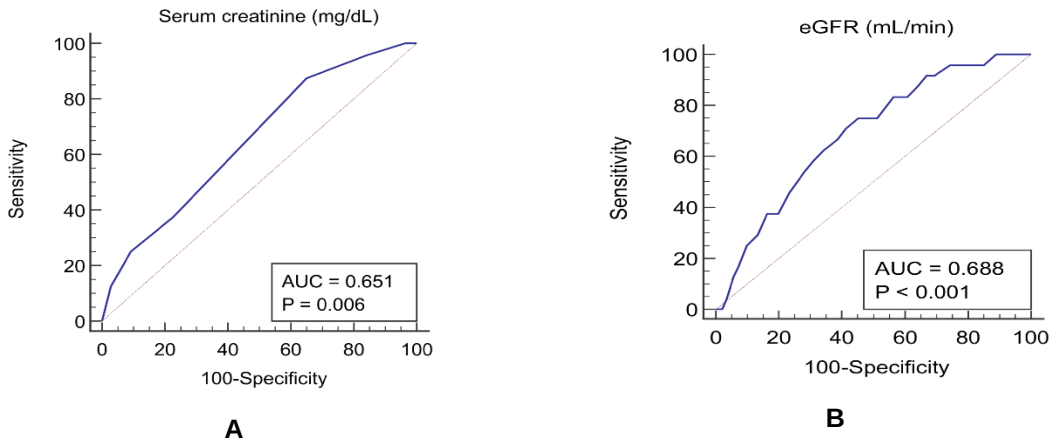

Fig. 1. ROC curve analysis of A) serum creatinine and B) eGFR for prediction of CI-AKI

Serum creatinine can significantly predict the incidence of CI-AKI at AUC of 0.651 and P value of 0.006, and at cutoff >0.9 mg/dL with 87.50% sensitivity, 34.84% specificity, 7.9% PPV and 97.8% NPV. eGFR can significantly predict the incidence of CI-AKI at AUC of 0.688 and P value of <0.001, and at cutoff >73 mL/min with 83.33% sensitivity, 39.10% specificity, 8.0% PPV and 97.4% NPV Fig. 1.

4. DISCUSSION

Contrast-induced acute kidney injury (CI-AKI) is a significant complication arising from diagnostic and interventional procedures that involve contrast media, particularly among patients undergoing percutaneous coronary intervention (PCI) [9]. CI-AKI not only complicates the immediate recovery of patients' post-procedure but also contributes to increased long-term morbidity and mortality [10].

Regarding demographic and clinical data, the baseline characteristics were insignificantly different between both groups. There was an insignificant difference between the studied groups regarding the risk factors. The clinical examination of vital signs revealed an insignificant difference between both groups regarding the HR, SBP and DBP.

Confirming our findings, Elhodhod et al. [11] carried out a study to demonstrate the effect of colchicine on contrast-induced nephropathy (CIN) in patients undergoing primary PCI. They enrolled 100 STEMI patients planned for primary PCI and randomized them into two groups: a control group receiving standard guideline-based medical treatment and a study group receiving the same treatment plus colchicine. CIN was defined as an absolute rise in serum creatinine of 0.5 mg/dL or a relative rise of 25% or more from baseline over three days. They reported similar demographics and clinical data as our study.

In contrast, a Turkish investigation was conducted to explore the preventive effects of colchicine on CI-AKI among patients undergoing elective PCI, taking into account its anti-inflammatory and renoprotective properties. This study encompassed 280 patients undergoing elective PCI with an eGFR of >45 mL/min/1.73 m². One hundred and forty patients were randomly assigned to the colchicine treatment group (mean age, 60±9 years), while 140 patients were assigned to the control group (mean age, 61±7 years). CI-AKI was defined as either a 50% relative increase in serum creatinine levels from baseline or a 0.3 mg/dL increase in the absolute value measured 48 hours post-PCI. Colchicine treatment commenced 24 hours prior to PCI and persisted for 48 hours post-PCI. The study revealed that in the control group, the proportion of male individuals was higher compared to the colchicine treatment group (N.=104, 74% vs. N.=80, 57%; P=0.045) [1].

In the present work, there was an insignificant difference between both groups regarding total cholesterol, triglycerides HDL and LDL levels.

Confirming our findings, Elhodhod et al. [11] reported insignificant variations in the studied groups regarding TC, TG, LDL-c and HDL-c.

In disagreement with our findings, when comparing pre-procedural laboratory values, Oktay et al. [1] reported that low-density lipoprotein cholesterol (LDL-C) levels were lower in the control group (117±39 vs. 134±38; P=0.009). This finding contrasts with our study, where LDL-C levels were not significantly different between the treatment and control groups. The discrepancy in LDL-C levels between the two studies may reflect variations in patient demographics, treatment protocols, or other factors influencing lipid metabolism and cardiovascular risk profilesIn the present work, post CAG creatinine, baseline eGFR were significantly lower in group 1 (case group) compared to group 2 (control group) (P<0.001). Post CAG eGFR was significantly higher in the case group than the controls (68.98 ± 7.44 vs. 66.88 ± 6.73, p-value = 0.003). There was no significant difference regarding baseline creatinine between both groups.

Supporting our findings, Oktay et al. [1] in patients under colchicine treatment, the rate of eGFr reduction after CAG was significantly lower than in those not under colchicine treatment (69±13 vs. 66±15, P=0.227; 72±11 vs. 63±13 P<0.001) [1].

Regarding the adverse events, there was an insignificant difference between both groups regarding the other adverse events (chest pain, hypersensitivity reaction, access site discomfort, fever, Ischemic stroke, Fluid overload, Bleeding and Palpitations). Concerning the outcome, CI-AKI occurred in 5 (2.5%) patients in group 1 (case group) and 15 (7.5%) patients in group 2 (control group), PCI-related myocardial injury

occurred in 7 (3.5%) patients in group 1 (case group) and 10 (5%) patients in group 2 (control group). The incidence of CI-AKI was significantly lower in group 1 (case group) compared to group 2 (control group) (P= 0.038), with no significant difference between both groups regarding PCI-related myocardial injury.The lower incidence of CI-AKI in the case group (group 1) compared to the control group (group 2) could be attributed to the potential renoprotective effects of colchicine, as suggested by the study design. Colchicine, a medication with anti-inflammatory properties, may mitigate the inflammatory response triggered by the contrast media used during PCI. Contrast media can induce renal vasoconstriction, oxidative stress, and inflammation, contributing to the development of CI-AKI. By inhibiting inflammatory pathways, colchicine could attenuate the renal injury cascade initiated by contrast media exposure, thereby reducing the incidence of CI-AKI. Additionally, colchicine may exert direct protective effects on renal function. Studies have suggested that colchicine can modulate various cellular processes involved in kidney injury, such as apoptosis, fibrosis, and endothelial dysfunction.

These protective mechanisms could help preserve renal function and reduce the risk of CI-AKI in patients undergoing PCI.Moreover, the study's rigorous protocol, including pre- and post-procedural monitoring of renal function and standardized treatment regimens, may have contributed to the observed differences in CI-AKI incidence between the two groups. Strict adherence to protocol guidelines, coupled with vigilant management of patient risk factors and timely intervention, could enhance renal outcomes and reduce the incidence of CI-AKI in the case group compared to the control group.

Similarly, carried out a randomized trial to assess the effect of an anti-inflammatory medication colchicine on periprocedural myocardial injury. They reported that colchicine given 6 to 24 hours before PCI reduced the rate of periprocedural myocardial injury compared with placebo, in select patients. Also, there were no adverse effects reported by patients in relation to study medications [12].

There was a significant positive correlation between Serum creatinine and SBP (r= 0.099, P=0.047) and eGFR (r= 0.174, P<0.001). There was an insignificant correlation between Serum creatinine and the other parameters.

The multivariate regression analysis revealed in the current study revealed that age, colchicine use, diabetes mellitus, post CAG creatinine and post CAG eGFR, were the only significant predictors of incidence of CI-AKI.

Comparably, multiple regression analysis in Elhodhod et al.'s [11] study identified colchicine use as the most significant CIN risk-lowering factor (p = 0.039).

In line with our findings, Mo et al, [13]. showed an incidence of CIN of 3.2% and 1.2% under the old and new criteria, respectively. Three independent predictors were identified: baseline uric acid level, creatine kinase-MB level, and log (N-terminal pro-brain natriuretic peptide) level. They also identified significant predictors and developed a model for predicting CIN, highlighting the importance of identifying high-risk patients to minimize the risk of CI-AKI. Our study also demonstrated the efficacy of colchicine in reducing CI-AKI incidence in patients undergoing elective PCI, reinforcing the significance of preventive strategies in managing CIN risk. The consistency between these findings underscores the potential of targeted interventions and predictive modeling in improving patient outcomes during PCI procedures.

In the currents study, ROC curve analysis were performed for eGFR and creatinine for prediction of CI-AKI. Serum creatinine can significantly predict the incidence of CI-AKI at AUC of 0.651 and P value of 0.006, and at cutoff >0.9 mg/dL with 87.50% sensitivity, 34.84% specificity, 7.9% PPV and 97.8% NPV. eGFR can significantly predict the incidence of CI-AKI at AUC of 0.688 and P value of <0.001, and at cutoff >73 mL/min with 83.33% sensitivity, 39.10% specificity, 8.0% PPV and 97.4% NPV.

A study by Mo et al. [13] reported similar findings as we found that serum creatinine levels can predict CI-AKI occurrence with an AUC of 0.651, indicating a moderate prediction ability. The P value of 0.006 shows this relationship is statistically significant. With a cutoff point greater than 0.9 mg/dL, serum creatinine predicted CI-AKI with a high sensitivity (87.50%), meaning it correctly identified a large proportion of patients who developed CI-AKI. However, its specificity (34.84%)—the ability to correctly identify those who did not develop CI-AKI—was low. PPV and NPV indicate the likelihood that patients truly have or do not have CI-AKI based on the test

result, with values of 7.9% and 97.8%, respectively. A high NPV suggests that when the test predicts no CI-AKI, it is very likely to be correct.

In the current work, eGFR, a measure of the kidney's filtering capacity adjusted for body surface area, also significantly predicts CI-AKI risk, with an AUC of 0.688 indicating a slightly better prediction ability than serum creatinine. The P value of <0.001 denotes a very statistically significant relationship. With a cutoff value greater than 73 mL/min, eGFR predicted CI-AKI with a sensitivity of 83.33% and a specificity of 39.10%, similar to serum creatinine in its predictive pattern but slightly less sensitive and more specific. Its PPV and NPV are 8.0% and 97.4%, respectively, also indicating a high likelihood of accurately predicting the absence of CI-AKI. This was also supported by [14].

Both serum creatinine and eGFR have significant predictive value for CI-AKI, with high sensitivity and NPV, suggesting their utility in identifying patients at risk for CI-AKI. However, due to their low specificity and PPV, these markers are more effective at ruling out than confirming CI-AKI. These were also deemed by [13,15]

Limitations: The study's follow-up period extended only up to hospital discharge, limiting theabilityto assess long-term outcomes of colchicine use in preventing CI-AKI.The exclusion of patients with eGFR <45 mL/min/1.73 m2, recent contrast media exposure, known contrast allergy, and acute coronary syndrome may limit the applicability of the findings to a broader PCI patient population. These criteria exclude a segment of high-risk patients who might potentially benefit from or react differently to colchicine prophylaxis.

Therefore, further studies are needed with multicenter cooperation and larger sample sizes to validate our findings, Investigating the dose-response relationship of colchicine in the prevention of CI-AKI could help in identifying the optimal dosage, timing, and duration of treatment. Detailed studies focusing on pharmacokinetics and pharmacodynamics aspects of colchicine in the context of PCI could lead to more precise dosing guidelines

5. CONCLUSIONS

Colchicine, when administered in conjunction with standard anti-ischemic treatment in patients undergoing elective PCI, significantly reduces the incidence of CI-AKI. This renoprotective effect reveals the potential utility of colchicine as a preventive strategy against CI-AKI, particularly in high-risk patient populations. The reduction in CI-AKI incidence not only has implications for patient outcomes but also reveals an important strategy in optimizing renal protection during PCI.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

CONSENT AND ETHICAL APPROVAL

Informed written consent was obtained from all subjects. The study was done after approval from the Ethics Committee on research involving human subjects of Benha faculty of Medicine.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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