RESEARCH ARTICLE

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Coenzyme Q10 in Prevention of Contrast-induced Nephropathy in Patients with Acute Coronary Syndrome Undergoing Coronary Angiography

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Abstract

Background and Aim: Contrast-induced nephropathy (CIN) is a serious complication of coronary angiography (CA), associated with increased morbidity and mortality. Coenzyme Q10 (CoQ10), an endogenous antioxidant, has shown promise in mitigating oxidative renal injury. This study investigated CoQ10's protective effect against CIN in acute coronary syndrome (ACS) cases undergoing CA.

Materials and Methods: In a prospective randomized clinical trial (registration number: NCT06429345, date: 19.03.2024), 300 ACS cases were enrolled between March and September 2024. Cases were randomized into a CoQ10 group (n=200) receiving oral CoQ10 and a control group (n=100) receiving standard care. Serum creatinine, estimated glomerular filtration rate (eGFR), and urine output were monitored for three days post-procedure. CIN was defined as a ≥0.5 mg/dL or ≥25% increase in serum creatinine or a ≥25% decline in eGFR within 48 hours.

Results: CIN incidence was significantly lower in the CoQ10 group (9%) compared to controls (21%) ($P = 0.004$). Postoperative serum creatinine levels were markedly lower, and eGFR notably higher, in the CoQ10 group on days two and three ($P < 0.01$). Multivariate logistic regression identified high body mass index [odds ratio (OR) =6.976, $P < 0.001$], chronic kidney disease (OR =6.288, $P = 0.001$), and balloon dilatation (OR =3.116, $P = 0.012$) as independent predictors of CIN.

Conclusion: CoQ10 supplementation significantly reduced CIN incidence in ACS cases undergoing CA. CoQ10's antioxidative and anti-inflammatory properties support its potential as a safe adjunctive therapy for CIN prevention.

Keywords: Contrast induced nephropathy, acute coronary syndrome, coenzyme Q10

INTRODUCTION

Acute kidney injury (AKI) is a major complication following cardiac catheterization, associated with prolonged hospitalization and increased mortality. Beyond its immediate impact, AKI acts as an independent prognostic risk factor, contributing to the development of atrial fibrillation, progression of chronic kidney disease (CKD), and a higher risk of myocardial infarction or the need for dialysis after discharge. A specific subset, contrast-induced (CI)-AKI, is defined by at least a 25% increase in serum creatinine or a rise of 0.5 mg/dL within 48-72 hours of contrast exposure.[1]

The development of CI-AKI is multifactorial, with predisposing factors such as CKD, older age, inadequate hydration, and comorbidities like diabetes, heart failure, and peripheral vascular disease playing key roles. Procedural risks-including nephrotoxic contrast exposure, intraoperative hypotension,

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and blood loss-as well as postprocedural factors like anemia and prolonged intensive care unit stay, further contribute to the incidence of AKI.[2] Prevention strategies during percutaneous coronary intervention (PCI) include minimizing contrast volume, optimizing hydration, and using adjunctive pharmacotherapies such as N-acetylcysteine, statins, and renin-angiotensin-aldosterone inhibitors though with variable success.[3]

The pathophysiology of CI-AKI involves medullary ischemia, reduced nitric oxide availability, increased oxidative stress, and direct cytotoxicity to renal cells. Coenzyme Q10 (CoQ10), an endogenous antioxidant and anti-inflammatory molecule, has shown promise in mitigating oxidative and inflammatory renal injury. Emerging evidence suggests that CoQ10 supplementation offers organ-protective effects, potentially reducing the risk of CI-AKI, particularly when used alongside saline hydration in high-risk cardiac cases.[4]

This study aims to investigate the potential protective effect of CoQ10 against contrast-induced nephropathy (CIN) in acute coronary syndrome (ACS) cases undergoing coronary angiography (CA).

METHODS

This prospective randomized clinical trial (registration number: NCT06429345, date: 19.03.2024) was conducted at Ain Shams University Hospitals from March 1st to September 1st, 2024, and included 300 patients who presented with ACS, sample size calculation was performed based on a previous study involving 150 patients, which showed an approximate CIN incidence of 22% in the control group and 7% in the CoQ10 group.[5] Using these proportions, power analysis and sample size 15 program for sample size calculation was used setting power at 80% and an alpha level of 0.05 required a minimum of 168 patients to detect a statistically significant difference. To accommodate potential dropouts and missing data, we increased the total number to 300, using a 2:1 randomization ratio to obtain more experience and safety data for the CoQ10 group. Ethical approval was obtained from the Ethical Committee at Ain Shams University prior to initiating the research (approval no.: MS70/2024, date: 05.02.2025). Informed written consent was obtained from all participants, ensuring adequate privacy and confidentiality.

Randomization and Blinding of Patients

Methods of randomization: Patients were randomly assigned to either the CoQ10-treated group or the control group in a 2:1 ratio using the Research Randomizer software (https://www.randomizer.org/). To ensure allocation concealment and minimize selection bias, sequentially numbered, opaque, sealed envelopes were used. The envelopes were prepared in advance by an independent researcher not involved in patient recruitment, intervention, or outcome assessment. Study group (n=200): CoQ10 was administered orally at a dose of 400 mg before catheterization, followed by 200 mg twice daily for three consecutive days post-procedure, This dosing regimen follows the protocol used in a recent randomized clinical trial by Ahmadimoghaddam et al.[6] which reported a significant reduction in the incidence of CIN in ST elevation myocardial infarction (STEMI) patients undergoing primary PCI.

Additionally, intravenous normal saline hydration was provided prior to angiography for patients who did not require immediate intervention. Control group (n=100): cases received standard care, including intravenous saline hydration, and were administered an oral placebo capsule identical in appearance to the CoQ10 capsule to maintain blinding. A 2:1 allocation ratio was intentionally chosen to allow for more extensive evaluation of the safety and potential efficacy of CoQ10. Also, it allowed us to gain greater clinical experience with the active intervention and to improve the estimation of potential adverse events and variability within the treatment group.

1.Pre-catheterization phase:

Inclusion criteria specified patients aged ≥18 years with characteristics of ACS. Exclusion criteria included cases of renal transplants, end-stage renal disease requiring dialysis, peri-procedural bleeding, cardiogenic shock, or patients taking nephrotoxic medications such as aminoglycosides, amphotericin B, vancomycin, non-steroidal anti-inflammatory drugs (NSAIDs), cyclosporine, and tacrolimus. Patients with baseline renal impairment, including CKD stages 1-4, were included. This approach was intended to reflect the real-world clinical population at risk of CIN.

Detailed patient histories were recorded, including demographics, relevant risk factors, current pharmacologic therapies, and results of systemic and localized clinical assessments, with particular emphasis on chest pain characteristics. Electrocardiogram records, cardiac enzyme levels, random blood sugar, complete blood count, serum creatinine, urine output, estimated glomerular filtration rate (eGFR), and transthoracic echocardiography were evaluated. Left ventricular ejection fraction was assessed using the 2D Simpson method. Serum creatinine was measured using a standardized enzymatic method in the hospital's central laboratory, which undergoes regular internal quality control and external calibration procedures. eGFR was calculated using the CKD epidemiology collaboration equation. All laboratory personnel conducting the biochemical analyses were blinded to patient group assignments to minimize measurement bias.

Treatment was initiated immediately with appropriate medications, including antiplatelets, anticoagulants, nitrates, and beta-blockers. Emergency angiography was performed without delay in cases with ongoing ST-segment elevation (STE)

or non-STE (NSTE)-ACS accompanied by very high-risk criteria. High-risk NSTE-ACS cases were scheduled for early invasive intervention within 24 hours, while those with unstable angina received inpatient invasive assessment during hospitalization.

Additionally, intravenous normal saline hydration was provided prior to angiography in cases for whom immediate intervention was not required.

2.Intervention phase (catheterization):

CA was performed by an expert interventional cardiologist using the same type of contrast media, with the volume limited to 1-2 mL/kg to account for the iodine dose.

3.Post-catheterization:

All patients were admitted to the coronary care unit for monitoring and follow-up. Serum creatinine, eGFR, hemoglobin, and urine output (mL/kg/h) were assessed for three days post-catheterization. CIN was defined as either a ≥25% increase in serum creatinine from baseline, or a ≥25% decline in eGFR within 48 hours post-procedure. After catheterization, participants in the study group continued receiving 200 mg of CoQ10 orally twice daily for three days. Any side effects of CoQ10, such as nausea or skin rashes, were managed with appropriate antiemetic or antiallergic medications.

Statistical Analysis

The collected data were coded and entered into IBM SPSS Statistics for Windows, version 23.0 (IBM Corp., Armonk, NY, USA), for statistical analysis. Shapiro-Wilk test was used to assess the distribution of the quantitative variables. Quantitative variables were expressed as mean ± standard deviation and range, when parametric, and median with interquartile range when non-parametric, whereas categorical variables were summarized as frequencies and percentages. Group comparisons for categorical variables were performed using the chi-square test or Fisher's exact test when applicable. Depending on the distribution pattern, continuous variables were analyzed using either the independent samples t-test (for normal distribution) or the Mann-Whitney U test (for non-normal distribution). Comparisons between more than two groups with non-parametric data were made using the Kruskal-Wallis test. The 2:1 randomization ratio was taken into account by using statistical tests that are robust to unequal group sizes, ensuring valid intergroup comparisons. A 95% confidence interval (CI) was calculated for key outcomes to reflect estimate precision. A *P*-value <0.05 was considered statistically significant. Also, Bonferroni correction was applied for multiple comparisons to control the family-wise error rate. Adjusted *P*-values were reported where applicable. For logistic regression analysis, variables included in the multivariate model were selected based on statistical significance in univariate analysis, using a Bonferroni-adjusted *P*-value threshold of less than 0.01 to account for multiple testing. This conservative approach was adopted to minimize false-positive findings. Multicollinearity was assessed using variance inflation factors (VIF), with all included predictors demonstrating acceptable VIF values (<2).

RESULTS

Baseline Demographic Data Characteristics and Risk Factors

There was no statistically significant difference between the groups in terms of age, body mass index (BMI), sex, smoking status, and ACS type [non-STEMI (NSTEMI) vs. STEMI]. In addition, the prevalence of baseline comorbidities and cardiovascular risk factors was comparable between the two groups (Table 1).

Laboratory Investigations and Vital Data

No statistically significant variation was observed between the two groups with regard to baseline laboratory parameters prior to catheterization (including serum creatinine, eGFR). Additionally, intraoperative contrast volume and access route (femoral or radial, stenting, balloon dilatation, stent length and size, or procedure time) were comparable between the two groups (Table 2).

Description of Postoperative Serum Creatinine and eGFR

The serum creatinine levels on the three consecutive postoperative days were markedly lower in the CoQ10-treated group compared to controls, with a *P*-value of 0.0002 on day 1 and <0.0001 on both days 2 and 3. Alternatively, the eGFR on the first postoperative day did not differ notably between the two groups. However, on days two and three, eGFR was notably higher in the CoQ10-treated group compared to the placebo controls, with *P*-values of 0.006 and 0.002, respectively (Table 3).

Postoperative Incidence of CIN

The CoQ10-treated group had a significantly lower incidence of CIN compared to the control (placebo) group ($P = 0.004$) (Figure 1).

Incidence of (CIN) in Relation to Demographic Data, Patient Characteristics and Comorbidities

No statistically significant differences were found between the CIN and no CIN groups regarding age, gender, smoking status, or comorbidities. In contrast, the CIN group showed a significantly higher BMI, with a mean difference of 2.42 kg/m² (95% CI: 1.12-3.72; $P = 0.0002$; Bonferroni-adjusted $P = 0.008$), as well as a significantly greater prevalence of CKD ($P = 0.0057$; Bonferroni-adjusted $P = 0.008$) (Table 4).

Table 1: Comparison of baseline demographics, clinical characteristics and comorbidities between the studied groups

		Study G	Control G	Difference	Test value	*P*-value
		No =200	No =100	Mean (95% CI)		
Age	Mean ± SD	58.42±9.92	56.99±11.82	1.43 (1.12-3.98)	1.099•	0.273
	Range	30-84	31-92			
Sex	Female	52 (26%)	20 (20%)	-	1.316#	0.251
	Male	148 (74%)	80 (80%)			
BMI	Mean ± SD	28.47±2.46	29.19±4.91	0.72 (0.12-1.56)	-1.704•	0.089
	Range	23.39-36.51	19.69-47.88			
Smoking	No	83 (41.5%)	41 (41%)	-	0.007#	0.934
	Yes	117 (58.5%)	59 (59%)			
Type of ACS	NSTEMI	53 (26.5%)	31 (31.0%)	-	0.670*	0.413
	STEMI	147 (73.5%)	69 (69.0%)			
HTN	No	88 (44%)	48 (48%)	-	0.430*	0.512
	Yes	112 (56%)	52 (52%)			
Diabetes	No	106 (53%)	48 (48%)	-	0.667*	0.414
	Yes	94 (47%)	52 (52%)			
CKD	No	190 (95%)	91 (91%)	-	1.798*	0.180
	Yes	10 (5%)	9 (9%)			

*: Statically significant, #: Chi-square test, •: Independent t-test, BMI: Body mass index, ACS: Acute coronary syndrome, HTN: Hypertension, CKD: Chronic kidney disease, CI: Confidence interval, SD: Standard deviation, STEMI: ST elevation myocardial infarction, NSTEMI: Non-ST elevation myocardial infarction

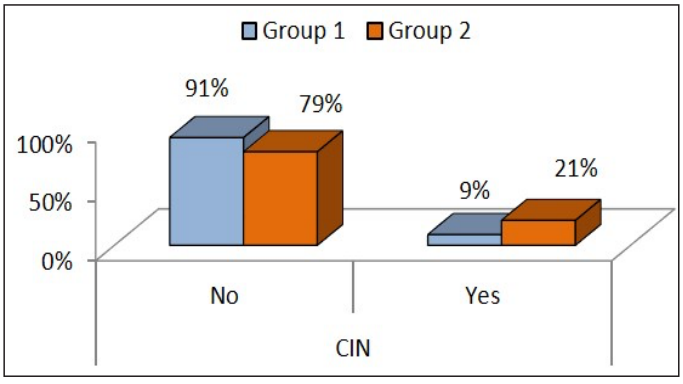

Figure 1: Comparison between study coenzyme treated group 1 and control placebo group 2 regarding postoperative CIN of the studied patients.

CIN: Contrast-induced nephropath

Univariate and Multivariate Logistic Regression Analysis of Factors Associated with the Incidence of CIN Among the Studied Cases

The univariate logistic regression analysis revealed a substantial increase in the occurrence of CIN among cases with high BMI and CKD, with *P*-values of <0.001 and 0.003, respectively. In the multivariate logistic regression analysis, BMI >31.2 kg/m² emerged as the most significant independent predictor of CIN, with an adjusted OR of 4.831 (95% CI: 2.249-10.375, $P < 0.001$). CKD was also significantly associated with CIN (adjusted OR: 2.700, 95% CI: 1.171-6.225, $P = 0.020$) (Table 5).

DISCUSSION

In this randomized clinical trial involving 300 CA procedures, CIN developed in 39 patients, representing 13% of the cohort. CIN was defined by an absolute increase in serum creatinine of ≥0.5 mg/dL, a relative increase of ≥25% from baseline, or a decrease of ≥25% in eGFR over 48 hours. The controls had a CIN incidence of 21% (21 out of 100 cases), whereas the CoQ10-treated group had a significantly lower incidence of 9% (18 out of 200 cases) ($P = 0.004$).

Among populations with minimal predisposing risk factors, the incidence of CIN is estimated to vary between 0.6% and 2.3%. Among cases requiring PCI during an acute myocardial infarction, the incidence has been reported to rise to 19% and to vary between 4.4% and 28% in other studies.[7,8] Previous clinical trials have documented a postoperative CIN incidence ranging from 13.1% to 30.3%.[9,10]

The variation in CIN incidence across studies can be attributed to differences in risk factors, contrast media type and volume, and CIN definitions. A recent study using a similar CIN definition reported an incidence of 14%,[6] while another study that defined CIN solely based on postoperative increases in serum creatinine reported a lower incidence of 10.7%.[9] High CIN rates following PCI, particularly primary PCI, are often linked to hemodynamic instability and inadequate prophylaxis.[11]

Our study found no notable variations in preoperative basal creatinine levels, demographic data, Killip classification, or contrast media between the control and CoQ10-treated

groups. The observed intergroup variation in postoperative creatinine and eGFR suggests a potential beneficial impact of CoQ10 administration. Specifically, the treated group showed a significantly lower incidence of CIN ($P = 0.004$), as evidenced by lower postoperative creatinine levels and higher eGFR. These findings are consistent with previous research,[12] which also identified changes in serum creatinine ≥0.1 mg/dL and decreases in eGFR ≤1.1 mL/min/1.73 m² as strong independent predictors of CIN. Our results align with these observations and further support the potential of CoQ10 in mitigating contrast-induced renal dysfunction.

The renal impairment in CIN cases is explained by the direct toxic effect of the contrast media, the associated pro-inflammatory effects, the greater reactive oxygen species (ROS) generation and the frequent hemodynamic instability.[13,14]

Table 2: Comparison of pre-operative laboratory investigations and operative data between the studied groups

		Study G	Control G	Difference	Test value	*P*-value
		No =200	No =100	Mean (95% CI)		
Preoperative serum creatinine	Mean ± SD	0.87±0.28	0.89±0.29	0.02 (0.05-0.09)	-0.611•	0.542
	Range	0.32-2.22	0.5-1.8			
Preoperative GFR	Mean ± SD	93.62±27.63	99.12±34.46	5.5 (-1.75-12.748)	-1.491•	0.137
	Range	29.5-163.2	39.8-194			
Contrast volume	Median (IQR)	200 (150-210)	200 (10-255)	3.65 (-14.515-21.82)	-0.094≠	0.925
	Range	50-350	50-350			
Access	Femoral	195 (97.5%)	97 (97%)	-	0.064#	0.800
	Radial	5 (2.5%)	3 (3%)			
Stenting	No	11 (5.5%)	7 (7%)	-	0.266#	0.606
	Yes	189 (94.5%)	93 (93%)			
Balloon dilatation	No	83 (41.5%)	35 (35%)	-	1.180#	0.277
	Yes	117 (58.5%)	65 (65%)			
Stent size	Mean ± SD	2.97±0.41	3.06±0.29	0.09 (0.00-0.18)	-1.955•	0.052
	Range	1.5-3.5	2.5-3.5			
Stent length	Mean ± SD	25.93±6.9	26.71±7.06	0.78 (-0.89-2.45)	-0.913•	0.362
	Range	12-48	18-48			
Procedure time	Median (IQR)	28 (22-37.5)	28 (22-30)	0.475 (-2.894-3.844)	-0.347≠	0.729
	Range	10-70	10-70			

#: Chi-square test; •: Independent t-test; ≠: Mann-Whitney test, GFR: Glomerular filtration rate, SD: Standard deviation, IQR: Interquartile range, CI: Confidence interval

Table 3: Comparison of postoperative serum creatinine and eGFR between the studied groups

			Study G	Control G	Difference	Test value	*P*-value
			No =200	No =100	Mean (95% CI)		
Serum creatinine	**1 day**	**Mean ± SD**	0.88±0.31	1.04±0.37	0.16 (-0.08-0.24)	-3.833•	0.0002*
		Range	0.33-2.15	0.6-2.5			
	2 days	**Mean ± SD**	0.91±0.36	1.11±0.47	0.2 (-0.10-0.29)	-4.134•	<0.0001*
		Range	0.4-2.49	0.5-2.9			
	3 days	**Mean ± SD**	0.93±0.38	1.16±0.47	0.23 (-0.13-0.32)	-4.552•	<0.0001*
		Range	0.3-2.3	0.6-2.6			
eGFR	**1 day**	**Mean ± SD**	89.83±23.26	84.64±27.46	5.19 (0.77-11.15)	1.714•	0.088
		Range	32-136	29-147.6			
	2 days	**Mean ± SD**	90.35±25.53	81.38±28.35	8.97 (-2.58-15.36)	2.766•	0.006*
		Range	28-180.72	26.5-148			
	3 days	**Mean ± SD**	88.51±23.83	78.91±29.01	9.6 (-3.41-15.78)	3.055•	0.002*
		Range	27-129	25.5-160.2			

*: Statically significant. •: Independent t-test, CI: Confidence interval, eGFR: Estimated glomerular filtration rate, SD: Standard deviation

Table 4: Relation of CIN Incidence to demographic data and patient characteristics and comorbidities

		CIN		Difference (95% CI)	Test value	*P*-value	Adjusted *P*-value (Bonferroni)
		No CIN (No =261)	CIN (No =39)				
Age	**Mean ± SD**	57.54±10.34	60.64±11.92	3.1 (-0.47-6.67)	-1.713•	0.088	0.704
	Range	30-84	36-92				
Sex	**Female**	58 (22.2%)	14 (35.9%)	-	3.479#	0.062	0.496
	Male	203 (77.8%)	25 (64.1%)				
BMI	**Mean ± SD**	28.39±2.81	30.81±6.02	2.42 (1.27-3.57)	-4.152•	0.0002*	**0.008***
	Range	19.69-42.97	21.5-47.88				
Type of ACS	**NSTEMI** **STEMI**	75 (28.7%) 186 (71.3%)	9 (23.1%) 30 (76.9%)	-	0.539*	0.463	1.000
HTN	**No**	117 (44.8%)	19 (48.7%)	-	0.207*	0.649	1.000
	Yes	144 (55.2%)	20 (51.3%)				
Diabetes	**No**	136 (52.1%)	18 (46.2%)	-	0.481*	0.488	1.000
	Yes	125 (47.9%)	21 (53.8%)				
CKD	**No**	249 (95.4%)	32 (82.1%)	-	10.195*	0.0057*	**0.008***
	Yes	12 (4.6%)	7 (17.9%)				
Balloon dilatation	**No**	110 (42.1%)	8 (20.8%)	-	6.654	0.010*	0.080
	Yes	151 (57.9%)	31 (79.5%)				

*: Statically significant, #: Chi-square test, •: Independent t-test, ACS: Acute coronary syndrome, HTN: Hypertension, CKD: Chronic kidney disease, STEMI: ST elevation myocardial infarction, NSTEMI: Non-ST elevation myocardial infarction, CIN: Contrast-induced nephropath, CI: Confidence interval

Table 5: Predictors of CIN by logistic regression

	Univariate				Multivariate				VIF
	P-value	OR	95% CI for OR		*P*-value	OR	95% CI for OR		
			Lower	Upper			Lower	Upper	
BMI >31.2	<0.001*	5.000	2.358	10.604	<0.001*	4.831	2.249	10.375	1.001
CKD	0.003	4.539	1.666	12.365	0.020	2.700	1.171	6.225	1.001

*: Statically significant, OR: Odds ratio, CI: Confidence interval, BMI: Body mass index, VIF: Variable inflation factor, CKD: Chronic kidney disease

The protective effects of CoQ10 have been well-documented in several studies. In one trial involving 150 cases with coronary heart disease undergoing elective cardiac catheterization, the combination of CoQ10 and trimetazidine markedly reduced the incidence of CIN to 6.67% compared to 21.3% in the placebo group.[5] Similarly, a 2023 study involving 153 cases with STEMI found that CoQ10, used alongside saline hydration, decreased CIN incidence to 8% compared to 20% in the controls.[6] CoQ10's nephroprotective effect is primarily ascribed to its antioxidative and anti-inflammatory effect.[4]

CoQ10 counteracts the adverse effects of contrast media by mitigating direct cytotoxicity and abnormal energy metabolism caused by impaired mitochondrial enzyme activity.[15] As a crucial component of the mitochondrial respiratory chain, CoQ10 facilitates adenosine triphosphate synthesis through oxidative phosphorylation. Furthermore, contrast media generates ROS, leading to oxidative stress and decreased antioxidant enzyme activity.[15] CoQ10 helps maintain redox balance and regulates ROS generation, thereby protecting cells from oxidative damage.[16] Additionally, CoQ10 elevates the total antioxidant capacity in kidney tissue.[17]

Contrast media also induce renal interstitial inflammation by increasing immune cell migration and cytokine accumulation, which triggers systemic inflammation.[14] CoQ10 has demonstrated anti-inflammatory effects and regulates lysosomal function.[18] Furthermore, contrast media alter renal hemodynamics by increasing vasoconstrictors (e.g., renin, angiotensin, aldosterone, endothelin) and decreasing vasodilators (e.g., nitric oxide, prostacyclin), leading to medullary hypoxia. CoQ10 improves vasoactive hormone balance.[13,19]

Our findings align with previous research suggesting CoQ10's renal protective role against various forms of AKI, including those induced by drugs (e.g., NSAIDs),[20] contrast media,[5] sepsis,[21] and ischemia-reperfusion injury.[22]

Given the association between CIN and adverse outcomes in hospitalized cases, several predictive scoring systems have been developed to identify individuals at heightened risk. In the present study, the majority of variables included in the Mehran risk score-such as age, hypotension, cardiovascular comorbidities, diabetes mellitus, anemia, contrast volume, baseline serum creatinine, and a history of renal impairment-were assessed. No substantial differences were found between the two groups with respect to any of these parameters.[23]

Elevated baseline serum creatinine levels (defined as ≥1 mg/dL in females and ≥1.3 mg/dL in males) have been described as clinically notable risk factors for CIN.[24] However, the univariate and multivariate logistic regression analysis in our study did not detect basal preoperative creatinine as a risk factor. This contradiction may be attributed to the lower basal values in the cases selected for our study where the means were 0.87±0.28 in the study group versus 0.89±0.29 in controls, $P = 0.542$. Several studies have emphasized that serum creatinine alone is an inadequate and insensitive marker of renal function, and that CIN may still develop in cases without established CKD.[25]

In our analysis, both elevated BMI and CKD demonstrated significant associations with the development of CIN. These findings align with prior research highlighting obesity and renal dysfunction as key risk factors for CIN. High BMI, often reflective of underlying atherosclerosis, dyslipidemia, and metabolic syndrome, may exacerbate renal susceptibility to contrast-induced injury.[26] Similarly, pre-existing CKD, particularly when accompanied by elevated blood urea nitrogen, has been consistently linked to increased CIN risk.[10] Notably, in the multivariate model, BMI emerged as the most powerful independent predictor of CIN, underscoring the impact of metabolic factors in renal outcomes following contrast exposure.

Interestingly, contrast media and diabetes were not significant risk factors in our study, despite 53.8% of CIN cases being in diabetic individuals. This may be attributed to pre- and post-contrast media intravenous hydration, which mitigates the impact of contrast doses and prevents dehydration in diabetic cases.[26]

In the analysis of ACS types, differentiating between STEMI and NSTEMI, there were no notable variations between the control and treatment groups or between CIN and non-CIN cases.[27] This observation suggests that the risk of CIN is comparable in both STEMI and NSTEMI cases. This finding aligns with a study involving 1,041 ACS cases, which reported no notable variations in CIN incidence between STEMI and NSTEMI groups.[28]

These results contrast with some studies that propose STEMI is associated with a higher CIN risk due to factors such as larger contrast volumes and the urgency of immediate revascularization.[9,29]

Short-term follow-up in our study, showed no poor intrahospital outcomes, with no need for dialysis or reported mortality. However, CIN outcomes vary widely in the literature, with reports ranging from renal function recovery within 10 to 14 days to higher rates of dialysis and mortality. This variability highlights the critical need for ongoing research aimed at elucidating the underlying mechanisms of CIN and developing strategies to reduce its associated risks.[30,31]

Study Limitations

This study has several limitations. First, it was conducted at a single center, which may limit the generalizability of the findings to broader or more diverse patient populations. Second, the sample size, while statistically adequate for detecting differences in CIN incidence, remains relatively small. These factors restrict the external validity of the results. Therefore, multicenter randomized trials with larger cohorts are needed to confirm the reproducibility and broader applicability of our findings.

Consequently, stratification of cases across the entire spectrum of renal dysfunction was not feasible, as conservative treatment strategies were predominantly employed in individuals with severely reduced eGFR. In addition, the type and volume of contrast media administered were not standardized but varied according to availability and clinical urgency, introducing potential variability in exposure.

Although renal function was monitored for up to 72 hours post-procedure, aligning with standard CIN definitions, we acknowledge that this short-term follow-up may not fully capture persistent or delayed renal dysfunction. Future studies with extended follow-up are warranted to assess the long-term renal outcomes associated with contrast exposure and CoQ10 administration. Lastly, although blood and urinary CoQ10 levels could serve as valuable biomarkers for monitoring treatment efficacy and renal delivery, such analyses were not performed due to feasibility constraints.

CONCLUSION

CoQ10 therapy yielded a significant reduction in the incidence of CIN in patients with ACS who underwent CA. CoQ10 can be used as an agent to reduce the incidence of CIN, owing to its antioxidant and anti-inflammatory effects. BMI, a history of CKD, and intraoperative balloon dilation may be considered significant risk factors for CIN.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Ethical Committee at Ain Shams University prior to initiating the research (approval no.: MS70/2024, date: 05.02.2025).

Informed Consent: Informed written consent was obtained from all participants, ensuring adequate privacy and confidentiality.

Footnotes

Authorship Contributions

Surgical and Medical Practices: R.E.S., S.E.E., I.B., N.O., Design: I.B., Data Collection or Processing: S.E.E., N.O., Analysis or Interpretation: N.O., Literature Search: R.E.S., Writing: R.E.S.

Conflict of Interest: No conflict of interest was declared by the authors.

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