Original
ISSN 2413-0516

The Risk of Acute Kidney Injury in Patients Undergoing Coronary Angiography

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(Submitted: 06 February 2024 – Revised version received: 27 February 2024 – Accepted: 12 March 2024 – Published online: 26 June 2024)

Abstract

Objective: This study was conducted to estimate the risk of contrast-induced AKI (CI-AKI), among Saudi patients who underwent CA or PCI at a tertiary care hospital, to understand the incidence and the underlying contributory factors of CI-AKI.

Methods: We conducted a retrospective review of patients who underwent CA or PCI from 1st January 2018 to 31st December 2020, at the King Abdulaziz University Hospital, Jeddah, Saudi Arabia. All authors had no access to information that could identify individual participants during or after data collection. The exclusion criteria comprised age <18 years, preexisting chronic kidney disease (stage III–V), prior renal transplantation and records with missing key clinical information. Occurrence of AKI was defined using the "Kidney Disease: Improving Global Outcomes" (KDIGO) consensus definition. Statistical Package for the Social Sciences (SPSS) software, version 21 was used for statistical analysis. The prevalence is presented as a percentage with a 95% confidence level. *P*-value <0.05 was considered statistically significant.

Results: We reviewed 825 patient files, of which 754 met the inclusion criteria. According to the KDIGO classification, the mean overall incidence of Stage I CI-AKI in our study cohort was 8.1%, while no patients developed stage II and III AKI. The incidence of CI-AKI was 6.4% in patients <55 years of age (n = 264) and 7.8% in the age group of 55–70 years. A significantly higher incidence of 13.3% was seen in patients above the age of 70 years. The increase in the incidence of CI-AKI in patients above 70 years, versus their younger counterparts was statistically significant ($P = 0.075$).

Conclusions: Based on the results of this study and past literature, the overall incidence of CI-AKI is around ≤10%, among patients undergoing CA or PCI; which seems lower than the high risk perception among cardiologists. While CI-AKI is a known post-procedural complication of CA or PCI, the apprehension of the potential risk of CI-AKI, should not defer or obstruct the decision to perform CA or PCI for deserving and needy candidates. Exercising caution among high-risk patients, individual risk-benefit assessment and employing well-established pre and peri-procedural prevention protocols can significantly mitigate the risk of CI-AKI; even among high-risk patients.

Keywords: Contrast-induced acute kidney injury (CI-AKI), acute kidney injury (AKI), coronary angiography (CA), percutaneous coronary intervention (PCI)

Introduction

The rise in morbidity and mortality associated with acute kidney injury (AKI) has been a matter of growing medical and public concern worldwide.[1] Approximately 13.3 million patients are affected annually, of which 1.7 million succumb to AKI every year.[1] AKI is defined as a rapid decline in renal function, reflected by a change in serum creatinine levels and/or urinary output; it also has been linked to an imbalance of body fluids, electrolytes, and acid-base homeostasis. The occurrence of AKI has been attributed to several etiological factors like dehydration or volume depletion, sepsis, chronic systemic diseases and nephrotoxic drugs[2].

During the last few decades, AKI has been reported as a frequent complication of coronary angiography (CA) or percutaneous coronary intervention (PCI). These procedures are frequently employed for confirmatory diagnosis and interventional management of coronary stenosis and other cardiovascular disorders, by cardiologists all over the world.[3,4] Intravenous or intra-arterial contrast media or dye administration, employed during CA or PCI, has emerged as one of the key causes of hospital-acquired acute kidney injury (AKI), as the dyes used are known to have nephrotoxic adverse effects. This type of AKI is referred to as contrast-induced AKI (CI-AKI). While contrast media have been implicated as a cause of AKI, it is well known that several systemic factors might also contribute to the development of AKI; other than the contrast medium *per se*.[5] These factors include internal bleeding, cardiogenic shock, sepsis, and respiratory failure among others.[2]

From an epidemiological standpoint, past research has put forth varying figures of incidence of CI-AKI. A study by Helgason et al. showed that the incidence of CI-AKI following CA was 2% in a total of 13,561 patients.[6] Another randomized clinical trial performed in 2018 showed that after PCI, the prevalence of CI-AKI was approximately 10–12%. This incidence has been shown to rise to 20–30% in patients with prior chronic renal disease.[7] Several past studies have also demonstrated the steep rise in hospital related mortality, due to CI-AKI.[8-14]

Such past clinical evidence has cast a shadow of doubt and concern, over the risk-benefit ratio of performing a contrast-guided CA or PCI, especially among patients with pre-existing kidney disease and other comorbidities known to increase the risk of CI-AKI. This apprehension concerning CI-AKI has perhaps caused clinicians to become over-cautious and hesitant in performing contrast-based CA

or PCI, even among patients who are otherwise suitable and needy candidates for these procedures.[14-25]

The incidence of coronary artery disease in the Saudi population has been estimated to be 5.5%.[26,27] Based on this, it can be logically inferred that a large subset of these patients would be candidates for either CA or PCI. Yet, there exists a dearth of evidence regarding the regional incidence of post-procedural CI-AKI in Saudi Arabia. Hence, we conducted this retrospective review, among Saudi patients who have undergone CA or PCI; to estimate the incidence of CI-AKI and understand its underlying contributory and prognostic factors.

Methods

Ethical Approval

The research ethics committee of the King Abdulaziz University Hospital (KAUH) approved this study.

Study design and sample size: This was a retrospective study conducted at KAUH, Jeddah, Saudi Arabia; among patients who underwent CA or PCI from 1st January 2018 to 31st December 2020.

Inclusion criteria: Patients >18 years who underwent CA or PCI at KAUH from 2018 to 2020 were included. Only the first CA was considered for patients who underwent >1 such angiography.

Exclusion criteria: The exclusion criteria comprised age <18 years, preexisting chronic kidney disease (stage III–V), prior renal transplantation and records with missing key clinical information.

Data Collection

Data collection was performed from June to August 2021 and data were extracted from the Phoenix system using data collection sheets that included the following: medical record number; demographic data including age, height, weight, sex and nationality; systolic and diastolic blood pressure (BP); hemoglobin levels; pre- and post-procedural serum creatinine and albumin levels; glycated hemoglobin (HbA1c); and the type of procedure performed (whether it was solely CA, PCI or coronary artery bypass surgery). We included patients who had the following final diagnoses: ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), unstable angina, stable angina or unspecified chest pain. The following comorbidities were included: diabetes mellitus, hypertension, dyslipidemia, liver disease, malignancy, heart failure, ischemic heart disease, peripheral vascular disease, cerebrovascular disease, chronic kidney disease, thyroid disease, respiratory disease, autoimmune disease and previous COVID-19 infection.

For normal reference ranges, we used the following: systolic BP < 140 mm Hg and diastolic BP < 90 mm Hg; creatinine, 115 µmol/L; albumin: 3.4–5.4 g/dL (40.2–47.6 g/L); HbA1C, 4.2–6.4 %; random glucose, 3.9–6.7 mmol/L; hemoglobin, women = 12–15 g/dL and men= 14–18 g/dL; proteinuria: not present in urine (negative).

Definition of AKI Cases in the Retrospective Review

Patients with AKI were defined using the kidney disease: Improving Global Outcomes (KDIGO) general agreement. In accordance to this, AKI was defined as either a rise in serum creatinine level ≥0.3 mg/dL (≥26.5 mmol/L) within 48 hours, a rise to ≥1.5 times from the baseline serum creatinine levels, or a drop in the volume of urine to ≤0.5 mL/kg/hour. Baseline serum creatinine levels were defined as the last value within the six months preceding CA.

The KDIGO classification includes three stages depending on the magnitude of changes in serum creatinine level and/or levels of urinary output. Stage I is a rise of serum creatinine to ≥ 0.3 mg/dL (≥ 26.5 mmol/L) or by 1.5–1.9 times from baseline and/or a drop in urinary output to < 0.5 mL/kg/h for 6–12 hours. Stage II is a rise of serum creatinine by 2–2.9 times from baseline and/or a drop in urinary output to less than 0.5 mL/kg/h for >12 hours. Finally, stage III is a rise of serum creatinine to ≥353.6 mmol/L, a rise of more than three times from baseline, a decrease in glomerular filtration rate (GFR) to less than 35 mL/ min/1.73 m^2, and/or a drop in urinary output to <0.5 mL/kg/h for >24 hours or anuria for >12 hours. Patients with proteinuria were classified as micro-proteinuria (less than 30 mg/g), albuminuria (30–300 mg/g) or macr-oalbuminuria (more than 300 mg/g). Pre-diabetes was defined as HbA1C between 5.7-6.4%. A patient was considered diabetic if the HbA1C was ≥ 6.5%.

Data Analysis

The programs used in this report included Microsoft Office Excel 2016 for entering data and the Statistical Package for the Social Sciences (SPSS) software, version 21, for statistical analysis. Categorical variables, including primary variables, were described using frequencies. The continuous variables were described using mean ± standard deviation (SD) for normally distributed data. Univariate analysis was used for the categorical variables and the chi-square test was performed to check for all the risk factors. Logistic regression was used to evaluate the relations mentioned in the study. Odds ratios, confidence interval of odds ratios and *P*-values were generated for side effects. The prevalence is presented as a percentage with a 95% confidence level. *P*-value <0.05 was considered statistically significant.

Results

In this retrospective review, we analyzed 823 patient files, of which 754 met the pre-defined criteria and were included in our study. A comparable number of patients underwent either a CA (n = 363, 48%) or PCI (348, 46%). The final study sample had 78.2% males and 21.8% females, with a mean age of 58 years. Body mass index analysis showed that 98.5% of the patients were underweight. The baseline characteristics of study patients have been summarized in Table 1.

According to the KDIGO classification, the mean overall incidence of Stage I CI-AKI in our study cohort was 8.1%, while no patients developed stage II and III AKI. The incidence of CI-AKI was 6.4% in patients <55 years of age (n = 264) and 7.8% in the age group of 55–70 years. A statistically significantly higher incidence of 13.3% was seen in patients above the age of 70 years (*P* = 0.075).

The most common baseline comorbidity was hypertension, reported in 64.8% patients; followed by ischemic heart disease in 57.9% and diabetes mellitus in 57.4% of patients. A diagnosis of COVID-19 was reported in 4.2% of the patients during the study period. A summary of baseline comorbidities has been presented in Table 2. Most comorbidities were

Table 1. **Baseline characteristics of study patients**

Descriptive statistics		
	Mean (SD)	**Median (IQR)**
Age	58.24 (11.46)	58 (51–66)
SBP	132.34 (20.81)	130 (118–146)
DBP	76.00 (13.43)	75 (66–85)
Pre-CA serum creatinine	93.20 (61.85)	83 (69–100)
Pre-CA albumin	34.76 (6.14)	34 (31–38.55)
HbA1C	7.79 (2.31)	7 (6–9)
Blood glucose level	8.94 (4.10)	7.70 (5.7–11.3)

CA: coronary angiography; DBP: diastolic blood pressure; HbA1C: hemoglobin A1c; IQR: interquartile range; SBP: systolic blood pressure SD: standard deviation.

Table 2. **Summary of baseline comorbidities**

Comorbidity	Percent of the patients
DM	57.4%
HTN	64.8%
Dyslipidemia	24.5%
Liver disease	1.6%
Malignancy	4.8%
Heart failure	12.6%
IHD	57.9%
Peripheral vascular disease	4%
Cerebrovascular disease	6%
Chronic kidney disease	3.6%
Thyroid disease	6.1%
Respiratory disease	11.8%
Autoimmune disease	0.8%
Covid 19	4.2%

DM: diabetes mellitus; HTN: hypertension; IHD: ischemic heart disease.

not statistically significantly associated with the occurrence of CI-AKI, except for respiratory diseases ($P = 0.005$).

The correlation between baseline comorbidities and CI-AKI has been summarized in Table 3.

The most frequent diagnosis upon admission was STEMI, reported in 25.1% patients, followed by unstable angina, with a frequency of 20.2%. A summary of patients' diagnoses upon admission has been displayed in Figure 1. A statistically significant correlation was found between death and patients' diagnoses; most patients who died during the follow-up period were diagnosed with STEMI ($P = 0.002$). Mean follow up duration was around 2 years.

Post-CA serum creatinine was significantly increased compared to pre-CA serum creatinine ($P < 0.001$), while the difference between pre-and post-CA serum albumin was not significant (Table 4). Of note in this regard, proteinuria was not measured and reported in the majority of cases, as this was not available in the original records.

Discussion

In patients undergoing CA or PCI, CI-AKI represents a daunting clinical challenge, attributed to the administration of radio-opaque contrast media. The first case report of CI-AKI dates back to the 1950s, wherein fatal acute renal failure occurred following intravenous pyelography, in patients with CKD and multiple myeloma. Over the years, as the application of contrast-based diagnostics grew sharply in interventional cardiology, the related occurrence of CI-AKI also rose by leaps and bounds.

In spite of this, there exists a scarcity of epidemiological data regarding CI-AKI, from the region of Saudi Arabia. Hence, we found it necessary and prudent to conduct this retrospective review to generate evidence from Saudi Arabia, on this pertinent medical issue. We believe this retrospective analysis, will encourage the medical fraternity in Saudi Arabia, to conduct similar and larger studies across the region.

In our retrospective analysis, while we screened 823 records of patients who underwent CA or PCI at our hospital, only 754 met our inclusion criteria. Of these, 8.1% developed Stage 1 CI-AKI, as defined by the KDIGO classification. A comparable incidence of 7.4% Stage 1 CI-AKI was seen in a large study, comprising 14782 Canadian patients who underwent contrast-based CA.[28] None of the patients in our study developed Stage II or III CI-AKI perhaps due to the small sample size of this analysis. However, in the large Canadian cohort too, the incidence of Stage II and III cases was merely 2.2%, despite a much larger sample size than ours.[28]

Large clinical studies conducted by the National Cardiovascular Data Registry of the American College of Cardiology have estimated the incidence of CI-AKI following CA or PCI, to be in the range of 7.1% – 10.5%.[29,30] The incidence of 8.1% reported in our study, also lies within the same range. Previous studies conducted in China and Austria have reported an incidence of 7.2% and 10.2% respectively, which again seem to be comparable to the incidence of CI-AKI in our study.[31,32]

On the other hand, some isolated studies have also reported a lower incidence of CI-AKI. The cohort study conducted by Morabito et al showed an overall incidence of 5.1%, much lower than our study as well as most recently published studies, in our knowledge.[33] In sharp contrast, Abdalla et al, reported a whopping 31% incidence of CI-AKI, among a cohort of 163 Sudanese patients, who underwent contrast guided CA.[34] An incidence of as high as 50% has also been reported among vulnerable and high-risk patients. In spite of technological advances in contrast-based diagnostics, CI-AKI continues to account for one-third of all hospital-acquired AKI and is associated with a high morbidity and mortality in the long run, especially in the high-risk subgroup.[35]

Past research has identified these high-risk patient subgroups, who are more susceptible to CI-AKI. These include patients with pre-existing kidney disease, diabetes mellitus, obesity and hypertension, chronic heart failure, respiratory disorders, ischemia and anemia among other systemic conditions.[36] Our study sample too included patients from different high-risk groups, having baseline comorbidities like hypertension, diabetes, ischemic heart disease, dyslipidemia, respiratory disorders and coagulopathies. However, in our study, none of these baseline comorbidities, had a statistically significant correlation with the incidence of CI-AKI, except

Table 3. **Correlation between comorbidities and CI-AKI incidence**

		AKI				*P*-value (chi-square test)
		No AKI		Stage 1		
		Count	Row *N* %	Count	Row *N* %	
Diabetes mellitus	No	302	93.8%	20	6.2%	*P* = 0.106
	Yes	393	90.6%	41	9.4%	
Hypertension	No	242	91.0%	24	9.0%	*P* = 0.478
	Yes	453	92.4%	37	7.6%	
Dyslipidemia	No	520	91.1%	51	8.9%	*P* = 0.126
	Yes	175	94.6%	10	5.4%	
Liver disease	No	684	91.9%	60	8.1%	*P* = 0.973
	Yes	11	91.7%	1	8.3%	
Malignancy	No	661	91.8%	59	8.2%	*P* = 0.570
	Yes	34	94.4%	2	5.6%	
Heart failure	No	610	92.3%	51	7.7%	*P* = 0.347
	Yes	85	89.5%	10	10.5%	
Ischemic heart disease	No	290	91.2%	28	8.8%	*P* = 0.527
	Yes	405	92.5%	33	7.5%	
Peripheral vascular disease	No	667	91.9%	59	8.1%	*P* = 0.774
	Yes	28	93.3%	2	6.7%	
Cerebrovascular disease	No	653	91.8%	58	8.2%	*P* = 0.722
	Yes	42	93.3%	3	6.7%	
Chronic kidney disease	No	672	92.2%	57	7.8%	*P* = 0.190
	Yes	23	85.2%	4	14.8%	
Thyroid disease	No	651	91.7%	59	8.3%	*P* = 0.339
	Yes	44	95.7%	2	4.3%	
Respiratory disease	No	620	93.0%	47	7.0%	P = 0.005
	Yes	75	84.3%	14	15.7%	
Autoimmune disease	No	690	92.0%	60	8.0%	*P* = 0.438
	Yes	5	83.3%	1	16.7%	
Covid-19	No	667	92.1%	57	7.9%	*P* = 0.347
	Yes	28	87.5%	4	12.5%	

CI-AKI: Contrast-induced acute kidney injury.

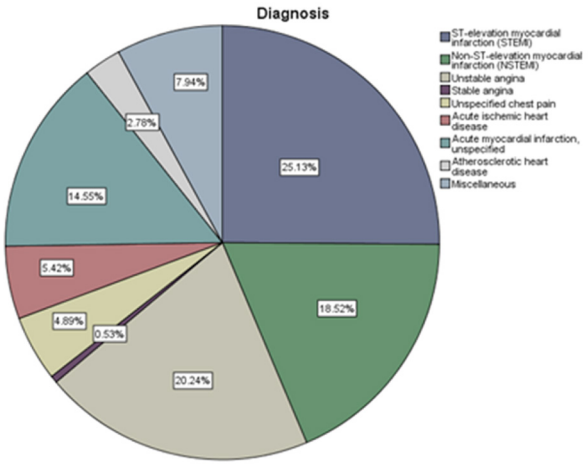

Fig.1 **Most frequent diagnoses among patients who underwent coronary angiography.**

Table 4. **Pre and post-procedural levels of creatinine and albumin**

N	Pre-CA		Post-CA		*P*-value
	Mean	SD	Mean	SD	
756	93.2	2.25	97.9	2.49	<0.001
499	34.6	0.27	35.2	0.38	0.75

CA: coronary angiography; SE: standard deviation.

respiratory disorders. This finding seems unusual compared to past evidence wherein a high incidence of CI-AKI has been frequently attributed to such comorbidities.[1,37] Diabetic nephropathy has also been identified as a key risk factor, with an estimated incidence of 9–40%, at serum creatinine level >124 μmol/l and 50–95% in those with a serum creatinine level >177 μmol/l. CI-AKI incidence rate of 20% among

diabetes patients with overt CKD and 11.4% among pre-diabetics with early CKD has been reported previously. With regards to diabetes patients without CKD; some researchers have shown this to increase the risk of CI-AKI while some others have not been able to demonstrate any such related escalation in risk.[38] Previous studies have also shown a higher incidence of CI-AKI among patients with dyslipidemia, coronary artery disease and acute coronary syndrome.[39-41] Perhaps, it is due to the small sample size of our study that we did not find a statistically significant correlation between the incidence of CI-AKI and the majority of baseline comorbidities. Larger studies from Saudi Arabia would perhaps be needed, to elucidate the effect of such comorbidities on the incidence of CI-AKI, among our population.

However, we did find a statistically significant correlation between respiratory disorders and the incidence of CI-AKI. Long standing respiratory disorders are often characterized by the presence of hypoxemia, hypercapnia and low bicarbonate levels; which can collectively lead to prolonged renal hypoperfusion, renal ischemia and oxidative stress. This can, in turn, trigger a series of nephrotoxic changes leading to AKI. The administration of a contrast medium can only make matters worse.[42] During our review of the literature, we did not come across any past studies assessing a possible correlation between pre-existing lung disorders and increased incidence of CI-AKI. We thus recommend that future studies should include and evaluate patients with pulmonary comorbidities, undergoing contrast-based CA or PCI, in order to assess the incidence of CI-AKI in this important cohort.

Past evidence shows that CI-AKI has a definite female predilection.[43-47] Our retrospective study sample itself was highly skewed towards the male gender, with 78.2% study patients being male. This became a limiting factor that did not allow any gender-based analysis, in our sample. Past evidence also highlights a significantly higher incidence of CI-AKI in the geriatric population, especially among patients >70 years of age. We saw a similar trend in our study upon performing an age-stratified analysis of our sample. The incidence of CI-AKI was 6.4% in patients <55 years of age (n = 264) and 7.8% in the age group of 55–70 years. However, a statistically significantly higher incidence of 13.3% was seen in patients above the age of 70 years, versus their younger counterparts ($P = 0.075$).

As we discuss the spectrum of risk factors, it is also imperative to throw light on the pathophysiology of CI-AKI. The contrast media employed in CA or PCI are radio-opaque, iodinated compounds. They are cytotoxic due to their high ionic strength, viscosity and osmolality. This cytotoxicity, in turn, causes renal vasoconstriction, hypoperfusion and renal ischemia, particularly in the outer medullary area. As the contrast medium is filtered by the kidneys, it gets concentrated within the tubules. The subsequent rise in viscosity within the tubules leads to tubular obstruction. This coupled with oxidative stress, induces acute tubular injury.[35] Hence, in order to reduce such renal cytotoxicity, the use of contrast media with high osmolality was discouraged and has almost become obsolete all over the world. The high osmolality contrast media have now been replaced with safer and newer substitutes like the isoosmolar and low osmolar contrast media.

Several predictive risk scoring models for CI-AKI have been developed and employed, for pre-procedural risk stratification of patients. These risk scores typically factor in the known etiological factors and comorbidities that contribute to CI-AKI. Such risk prediction can become a tangible tool to ensure that CA and PCI are offered to as many deserving and needy patients as possible, while contraindicating them in the appropriate cases only. Discontinuing any ongoing known nephrotoxic drug around 48 hours prior to CA or PCI is another highly recommended preventive step. Several intra-procedural prevention strategies have been successfully adopted to lower the risk of CI-AKI. These include volume expansion through hydration with normal saline or isotonic sodium bicarbonate, use of N-Acetylcysteine, use of certain new-age nephroprotective device systems, lowering the contrast volume, minimizing the need for repeat use of contrast media, using iso-osmolar or low osmolar contrast media, taking a radical approach instead of femoral for injection of the dye and opting for intravascular ultrasound based, zero-contrast procedures, wherever feasible and available.[48]

Conclusion

Based on the results of this study and past literature, the overall incidence of CI-AKI is around ≤ 10%, among patients undergoing CA or PCI, which seems lower than the high-risk perception, among cardiologists and other clinicians. While CI-AKI is a known post-procedural complication, the apprehension of the potential risk, should not defer or obstruct the decision to perform CA or PCI for deserving and needy candidates. Caution needs to be exercised while performing these procedures in the elderly, female, diabetics, hypertensives, cardiac and respiratory patients. An individualized risk-benefit assessment is needed to ensure that the benefits of CA or PCI can be availed by the majority of needy patients, while also mitigating the risk of CI-AKI through well-established prevention strategies.

The current study was retrospective, single center and had a relatively smaller sample size. We recommend that to generate robust evidence on this subject; larger, multi-centric, prospective trials be conducted. We suggest that future studies on the same topic define a time frame for the pre-and post-CA creatinine measurements; unlike our patient records wherein this timeframe was undefined. Our study did not include the volume of contrast dye used during the CA, which may impact the risk of CI-AKI. Therefore, we suggest further studies to correlate the importance of contrast volume with the incidence of AKI. Also, we recommend that future studies should elucidate the risk of CI-AKI in patients with obesity and lung disorders; as evidence currently seems scarce, regarding the impact of these comorbidities on the risk of CI-AKI.

Conflicts of Interest

According to the authors, no conflicts of interest exist.

Authors' Contribution

All authors listed in the study have contributed equally towards the medical ideation, design, analysis, conduct and submission of this study.

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