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Role of Urine Output as Index to Acute Kidney Injury after Coronary Angiography

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Abstract

Background

Contrast-induced acute kidney injury (CI-AKI) is a significant cause of iatrogenic renal dysfunction, contributing to morbidity, prolonged hospitalization, mortality and increased costs of health care. In our study we sought to investigate the relationship between urine volume after exposure to intravenous iodine-based contrast and the occurrence of contrast-induced acute kidney injury (CI-AKI; defined as an increase in serum creatinine of ≥0.5 mg/dL or a 25% relative rise within 48 h after contrast exposure) in patients who underwent coronary angiography.

Methods

We identified consecutive patients who were admitted for coronary angiography between January and June 2016 at King Abdulaziz University Hospital. We included patients who were admitted with acute coronary syndrome or admitted electively for an angiogram. Urine output was calculated at 12 & 24 hours and serum creatinine was measured at 24 hours following coronary angiography.

Results

The incidence of CI-AKI in was found to be 35%. One hundred patients were included, mean age 58±10 years and 66% were males. . BMI was 29.1±5.6, ejection fraction EF 45±11.6% and baseline creatinine was 90.9±26.6 μmol/L. Mean creatinine, 24-hours following the procedure, was 111.5±58.8 ml with overall 21.8% increment in the mean creatinine compared to the baseline creatinine. Mean measured urine output at 12 hours = 1200±443.9 ml and at 24 hours = 1938.5±699.6 ml.

CI-AKI occurred in 35 patients. There was a significant correlation between age of the

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patient and development of contrast induced acute kidney injury (r=0.2, p-value 0.04). There was no significant correlation between urine output at 12 hours or 24 hours and the development of CI-AKI (r=0.04, 0.01 and p-value=0.6, 0.8), respectively.

Conclusion

Contrast-induced acute kidney injury is common following intravenous contrast exposure and the incidence is higher in older patients. Urine output in the first 12 and 24 hours following exposure to contrast was not found to be correlated to contrast induced acute kidney injury.

Keywords: Coronary angiography, Urine output, kidney injury.

Introduction

Contrast-induced acute kidney injury (CI-AKI) is a significant cause of iatrogenic renal dysfunction, contributing to morbidity, prolonged hospitalization, mortality, and increased costs of health care. Contrast-induced acute kidney injury (CI-AKI) is a well-established complication of the use of intravenous iodine contrast media representing the third most common cause of acute kidney injury in hospitalized patients (1). It is associated with high in-hospital and long-term morbidity and mortality rates. The CI-AKI has commonly been referred to as contrast induced nephropathy (CIN) defined as an increase in serum creatinine ≥ 25% or ≥ 0.5 mg/dl (44 μmol/L) from baseline within 48–72 h after contrast exposure (2). Patients at high risk for CI-AKI includes those with baseline chronic kidney disease, diabetes mellitus, congestive heart failure (CHF), hypotension, and anemia; Procedural variables includes the use of intra-aortic balloon pump (IABP) and contrast volume which is a major contributor to CI-AKI. Efforts to modify the risk of CI-AKI include were investigated previously, which included various hydration strategies,

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N-acetylcysteine use, different types of contrast agents, haemofiltration protocols, and most recently statin therapy which showed mixed and conflicting results (3).

The reported incidence ranges from 5% in unselected populations to 50% in high-risk populations. The development of CI-AKI is associated with increased morbidity, length of hospitalization, chronic renal impairment, and higher mortality (4). Contrast-induced acute kidney injury (CI-AKI) is a common complication in patients undergoing coronary angiography and percutaneous coronary intervention (PCI).

Early detection of CI-AKI and identifying methods of prevention are very important for reducing morbidity and mortality related to CI-AKI. We sought to identify the relationship between urine volume and the occurrence of CI-AKI after exposure to iodine-based dye.

Methods

We identified consecutive patients who were admitted for coronary angiography between January and June 2016 at King Abdulaziz University Hospital. Inclusion criteria were any patients above the age of 18 who was admitted to the cardiology ward or CCU and required coronary angiography. We included patients who were admitted with acute coronary syndrome or admitted electively for an angiogram. Patient's exclusion criteria included pre-existing renal impairment, end stage renal disease (ESRD), prior episode of CI-AKI and use of diuretics.

For patients included in this study, urine output was calculated at 12 & 24 hours following coronary angiogram and urine output was indexed to the patient's body weight. Serum creatinine levels were measured prior to the procedure and 24 hours after.

CI-AKI was defined as increase in serum creatinine $\geq 25\%$ or ≥ 0.5 mg/dl (44 μmol/L)

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from baseline within 48–72 h after contrast exposure. As part of hospital protocol for all patients undergoing coronary angiography, they receive hydration protocol of 1mg/kg of 0.9% normal saline intravenous infusion, which starts 8 hours prior the procedures and continues for 8 hours afterwards.

Statistical Analysis:

Continuous variables are presented as means and standard deviations or median and interquartile range and were analyzed using Student's t-test. Categorical variables are presented as frequencies with percentages. Chi square test or Fisher's exact test were used for categorical variables as appropriate.

A multivariate analysis using a logistic regression model was performed to control for differences in baseline characteristics between the two study groups. Variables with $p<0.10$ by univariate analysis were included in the model. A two sided p-value ≤ 0.05 was considered statistically significant. SPSS was used for all statistical analyses.

Results

During the specified period, 100 patients were included, mean age 58±10 years and 66% were males. Baseline characteristics are shown in table 1. BMI was 29.1±5.6, ejection fraction EF 45±11.6% and baseline creatinine was 90.9±26.6 µmol/L (minimum 40 & maximum 380 µmol/L). Total contrast volume administered during the procedure was 184.3±107.5 ml (minimum 40 & maximum 570).

Table 2 showed that mean creatinine, 24-hours following the procedure, was 111.5±58.8 ml (minimum 63 & maximum 582 ml) with overall 21.8% increment in the mean

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creatinine compared to the baseline creatinine. In addition, table 2 showed mean measured urine output at 12 hours = 1200±443.9 ml and at 24 hours = 1938.5±699.6 ml. Moreover, hourly urinary volume was 1.25±0.5 ml/kg/min.

According to the pre-specified definition of CI-AKI in the methods, CI-AKI occurred in 35 patients. Table 4 identified the relationship between different parameters and development of CI-AKI.

There was a significant correlation between age of the patient and development of contrast induced acute kidney injury (r=0.2, p-value 0.04). There was no significant correlation found between BMI and CI-AKI (r=0.1, p-value=0.25), as well as EF was no correlated with CI-AKI (r=0.06, p-value=0.54).

Most importantly, there was no correlation between urine output at 12 hours or 24 hours and the development of CI-AKI (r=0.04, 0.01 and p-value=0.6, 0.8), respectively. In addition, contrast volume was not found in our study to be related to CI-AKI (r=0.12, p-value 0.2).

Discussion

This study was designed to evaluate urine volume as a predictor for early detection of contrast induced acute kidney injury (CI-AKI). Although the number of patients was small in our study, the baseline characteristics were similar amongst patients who developed CI-AKI and those who did not. Our study showed no correlation between the immediate urine volume post procedure (12 and 24 hours) and the development of

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CI-AKI, (r=0.04, 0.01 and p-value=0.6, 0.8), respectively.

This result is in agreement with the previously reported results by Aldjia Hocine et al, which investigated urine output as index to contrast induced nephropathy and they showed that the predictive value of urine output for the development of CI-AKI was low, which limits its use for assessing the effect of therapeutic interventions on the development of AKI. (5)

We reported in our study the incidence of CI-AKI to be 35 %, which is in keeping with previously reported incidence between 5 - 53 % according to presence or absence of certain risk factors (6). In Aldajia et al study, the incidence of CI-AKI was 15.3%.

The findings of this study suggests that patients aged 60 years or older are more prone to develop renal impairment after exposure to intravenous contrast medium when compared to patients aged less than 50 years. In other studies, age was no found to be predictive of CIN (7). On the other hand, other studies showed similar result to ours. George Dangas et al reported old age as a predictor for contrast induced acute kidney injury (8).

One might argue that older patients have significant co-morbidities that predispose them to CI-AKI. Renal function is also known to decline with age and morphological changes, such as decrease of kidney weight, appearance of sclerotic glomeruli and intimal proliferation within the renal artery, are some of the causes of renal dysfunction (9). A plausible hypothesis for the difference in CI-AKI between young and elderly patients is

the high prevalence of pre-existing chronic kidney disease and the presence of multiple risk factors for the development of acute kidney injury such as hypertension, diabetes and smoking. Also, the prevalence of using nephrotoxic medications like nonsteroidal anti-inflammatory drugs is quiet high in the elderly population (10).

In our study, we investigated multiple parameters in relation to CI-AKI, such as smoking, gender, EF, and diastolic dysfunction. We found no statistically significant correlation between these factors and the development of CI-AKI. This is in contrast to the results of Chong et al who studied the risk factors of CIN and found that age, female gender, insulin dependent diabetes mellitus, presence of hypotension, anemia and low LVEF are predictors of CIN (11). The contradiction between our results and Chong et al's study can be explained by our good routine hydration protocol of 1 ml/kg normal saline 8 hours prior and 8 hours following the procedure.

Limitations

There are several limitations to our study. First, our study was not powered to detect clinical differences in outcomes in patients who develops CI-AKI versus the ones who does not, thus these results can be considered hypothesis generating.

Secondly, our definition of CI-AKI relied solely on measurements of serum creatinine, which is one measurement of renal function without the use of direct urinary markers of tubular dysfunction.

Thirdly, the turnover of our patients in the cardiology units is rapid and sometimes the

follow up of patients who are referred to our centre from outside the city is very challenging. That limited our ability to measure serum creatinine at 48 and 72 hours in most patients, which may detect more patients with CI-AKI.

Conclusion

Contrast-induced acute kidney injury is common following intravenous contrast exposure and the incidence is higher in older patients. Urine output in the first 12 and 24 hours following exposure to contrast was not found to be correlated to contrast induced acute kidney injury.

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Table 1: Baseline characteristics of the patient population

	Min	Max	Mean	STD
Age	29	87	58	10
Weight (kg)	40	120	78	16.2
Height (cm)	145	188	166.5	8.5
BMI (kg/m2)	17	45	29.1	5.7
Cholesterol (mmol/L)	1.3	7.8	4.4	1.2
LDL	0.7	5.1	2.9	0.9
HDL	0.3	101	2	10
EF%	13	70	45	11.6
Diastolic dysfunction	0	3	1.2	0.9
Contrast volume (ml)	40	570	184.3	107.5

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Table 2. Creatinine and urinary volume results:

Creatinine (Baseline)	40	380	90.9	26.6
Creatinine (24hr)	63	582	111.5	58.8
%Change	0	106.5	21.8	22.2
UOP 12h	400	2500	1200.0	443.9
UOP 24h	200	3900	1938.5	699.6
Hourly UOP (ml/kg/h)	0.4	3	1.3	0.5

Table 3: Correlation between different parameters and development of CI-AKI

Parameter	r	P-value
Age	0.20	0.04
Weight	0.07	0.4
Height	-0.18	0.07
BMI	0.12	0.2
Gender	-0.06	0.5
Smoking	-0.14	0.2
Cholesterol	0.09	0.4
LDL	0.07	0.5
HDL	-0.10	0.3

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EF%	-0.06	0.5
Diastolic dysfunction	0.11	0.2
Contrast volume (ml)	0.13	0.2
Creatinine (Baseline)	0.08	0.4
Creatinine (24hr)	0.39	0.0
UOP 12h	0.04	0.6
UOP 24h	-0.02	0.8
Hourly UOP (ml/kg/h)	0.03	0.7

Table 4: Comparison between patients who developed CI-AKI and non CI-AKI

Parameter	CI-AKI GROUP		NON CI-AKI GROUP		P-value
	MEAN	SD	MEAN	SD	
Age	60.17	8.68	56.79	10.69	0.1
Weight	80.80	17.74	77.33	15.53	0.3
Height	164.94	8.46	167.44	8.59	0.2
BMI	30.44	6.57	28.38	5.08	0.08
Cholesterol	4.37	1.19	4.41	1.17	0.8
LDL	2.89	0.99	2.93	0.96	0.8
HDL	0.97	0.24	2.59	12.62	0.4
EF	44.97	11.37	45.05	11.95	0.9

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Diastolic dysfunction	1.28	0.94	1.19	0.95	0.7
Contrast volume (ml)	201.94	118.20	174.21	101.43	0.2
Creatinine (Baseline)	94.42	56.44	88.98	18.26	0.5
Creatinine (24hr)	138.14	88.10	96.25	22.18	0.0005
% Change	46.24	18.16	7.91	7.72	0.0
UOP 12h	1220.83	494.39	1188.10	419.99	0.7
UOP 24h	1926.39	746.39	1945.40	683.33	0.8
Hourly UOP (ml/kg/h)	1.24	0.53	1.27	0.52	0.7