The Incidence of Contrast Induced Nephropathy in Trauma Patients

Cordeiro SK, Fernandez H, Hill D, Petersen S

Department of Surgery, St. Joseph's Hospital and Medical Center, Phoenix, Arizona

Abstract

This study was aimed to evaluate the incidence and factors associated with contrast induced nephropathy (CIN) in the trauma patient population. A retrospective institutional review of 563 patients admitted to the trauma service at St. Joseph's Hospital and Medical Center was completed. Data was recorded for each patient including demographics, injury severity score (ISS), clinical prediction score (CPS), laboratory values and contrast doses. Contrast induced nephropathy was considered to be present if the patient received contrast material for CT scan and 24-72 hour creatinine increased by an absolute value of 0.5mg/dl or by 25% when compared to admission creatinine. We found the incidence of CIN to be 6.4% in this population and was significantly correlated with clinical prediction score, lowest systolic blood pressure (SBP), and the volume of IVF fluids given to patients.

Introduction

CIN has long been recognized as a potential adverse outcome in patients receiving intravenous contrast infusion. Though this was first recognized some 50 years ago, CIN is still the third largest cause of hospital-acquired renal insufficiency (HARI) in the United States. In the past there has been much research done looking at the incidence of hospital acquired nephropathy in the setting of contrast administration, however, trauma patients provide a novel population because of the acute need for radiologic evaluation with contrast. Unfortunately we have yet to develop a definitive preventative strategy to combat CIN. As the body of evidence regarding contrast induced nephropathy continues to grow, this study hopes to build on previous findings with a retrospective study investigating the incidence of contrast induced nephropathy as well as associated risk factors in this trauma patient population.

Methods

A single institution, retrospective chart review of trauma patients undergoing contrast enhanced CT scans during evaluation from 2005 to 2010 was conducted. This study was done in conjunction with the Department of Radiology, who provided all contrast dosages for each individual scan. 815 patients were initially identified to participate in the study, 252 met the exclusion criteria leaving 563 patients eligible for the study. Subjects were considered to have contrast-induced nephropathy if they received contrast material for a CT scan and 24-72 hour creatinine increased from admission creatinine by an absolute value of greater than or equal to 0.5 mg/dl or if 24-72 hour creatinine increased from admission creatinine by greater than or equal to 25%. In addition to demographic data and contrast doses, additional data including ISS, CPS laboratory values was collected. CPS was recorded as the the sum of points given to each patient for factors such as hypotension, age >75 years, anemia, volume of contrast administered and creatinine/eGFR. Analysis was done using univariate and multivariate regression.

Results

Figure 1 demonstrates the demographic data of the subjects enrolled in this study with the majority of the subjects being Caucasian (55%). Results of univariate analysis can be seen in *table 1* and demonstrate the following significant data: CIN vs age (p 0.004), CIN vs ISS (p <0.000), CIN vs CPS (p <0.000), CIN vs ICU length of stay (p 0.006), CIN vs total length of stay (p 0.002), CIN vs SBP (p <0.000), CIN vs IVF volume given in the 2nd 24 hours (p <0.000) and CIN vs IVF volume given in the first 48hrs (p <0.000). Also of note CIN vs contrast volume given acutely had a p value of 0.067. Data from multivariate regression analysis are shown in *table 1*. The relationship between CIN and CPS remains significant (p <0.000, CI $1.92E^{-2}$ – $3.93E^{-2}$), as did the relationship between CIN and SBP (p 0.003 CI $8.61E^{-4}$ – $4.41E^{-3}$) and CIN vs IVF vol 2nd 24 hours (p 0.001, CI $1.47E^{-5}$ – $5.91E^{-5}$). The mean CPS of patients who did and did not develop CIN were 9.09 and 3.12 respectively. The mean lowest SBP of a patients who did and did not develop CIN were 84 and 99mmHg respectively.. The mean IVF vol 2nd 24 hrs of patients who did and did not CIN were 2504ml and 5931ml respectively. Though not significantly different, patients who developed contrast-induced nephropathy, on average, were older, had a higher ISS, had a longer ICU and total hospital stay, received less IVF and more contrast. The difference in these means can be seen graphically in figure 2.

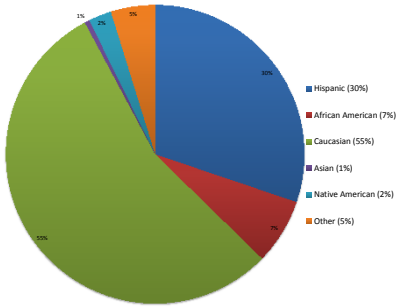

Figure 1. Demographic data of the patient's enrolled in the study.

	Univariate	Multivariate	95% Conf. Interval		Mean	
	p value	p value			(+) CIN	(-) CIN
CIN vs Age	0.004	0.914	-0.0013361	0.0015060	54	41
CIN vs ISS	<0.000	0.541	-0.0032229	0.0017886	23	13
CIN vs CPS	<0.000	<0.000	0.0192418	0.0393293	9.09	3.12
CIN vs ICU LoS	0.006	0.709	-0.0101594	0.0069159	7	3
CIN vs Total LoS	0.002	0.709	-0.0053490	0.0078563	12	6
CIN vs SBP	<0.000	0.003	0.0008613	0.0044137	84	99
CIN vs IVF Vol 2nd 24hr	<0.000	0.001	0.0000147	0.0000591	2504	5931
CIN vs IVF Vol 48h	<0.000	0.654	-0.0000125	0.0000208	3470	7033
CIN vs Contrast Vol 24h	0.235	-	-0.0002575	0.0010471	118	108
CIN vs Conrast Vol Acute	0.067	0.111	-0.0014413	0.0001685	115	102

Table 1. Univariate and Multivariate p values, confidence intervals and the mean values for subjects who developed contrast induced nephropathy vs those who did not develop contrast-induced nephropathy.

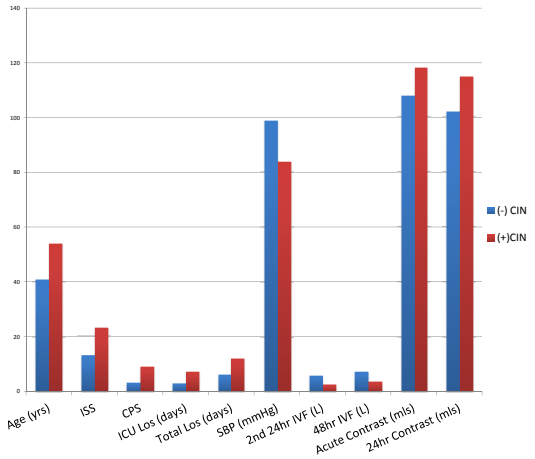

Figure 2. Mean data for variables of patients who developed contrast induced nephropathy compared to the mean data for variables of patients who did not develop contrast induced nephropathy.

Discussion and Conclusions

This study will help further delineate the risk of contrast induced nephropathy in trauma patients. A higher clinical prediction score, lower systolic blood pressure and fewer IV fluids administered in the second 24 hours seem to be significantly associated with increased risk of developing contrast induced nephropathy. This may not be surprising given that CPS looks at factors that can effect kidney function (ie. hypotension, contrast volume, creatinine) and IV fluids have been shown in other populations to be protective against the development of CIN. Hypotension may be somewhat of a confounder in this analysis as it can, independently of contrast administration, lead to kidney damage. ISS was shown in this analysis to not be significantly associated with CIN, most likely because it is focused more broadly on the anatomic severity of injury only. We hypothesized that the amount of contrast administered would be associated with the development of contrast induced nephropathy. However, these data demonstrate no significant correlation between volume of contrast given and the development of CIN. We believe that these negative findings probably reflect the insufficient power of this study rather than truly no correlation between these variables. Certain patient groups including those with higher clinical prediction scores, lower SBP and those receiving less IV fluids may benefit from aggressive mindfulness of the risk of contrast induced kidney injury. Continued investigation is needed to continue to identify risk factors and characterize methods to better identify trauma patients at increased risk for contrast induced nephropathy.

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