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RESEARCH ARTICLE

The Impact of Low-osmolar Iodinated Contrast on Renal Recovery in Critically Ill Patients with Severely Impaired Renal Function

John Ferguson[1] · Will DuSablon[2]

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Abstract

Background Contrast-enhanced diagnostic testing is often withheld in critically ill patients with acute kidney injury (AKI) due to safety concerns associated with iodinated contrast media.

Purpose The impact of iodinated intravenous contrast agents on the rate of renal recovery is unknown when administered to patients who present to the intensive care unit with severe acute kidney injury.

Materials and methods A retrospective cohort study of cases admitted to the intensive care unit with severe acute kidney injury who received a contrast-enhanced CT (CECT), propensity-matched to controls who received an unenhanced CT (UECT). Primary endpoints were the incidence of subjects with progressive AKI that met criteria for contrast-induced nephropathy, the incidence of renal replacement therapy (RRT) initiation, and the rate of renal function recovery over the first 120 h of hospitalization.

Results The incidence of progressive acute kidney injury was similar between patients who received a CECT and those who received a UECT in both unmatched and propensity-matched patients with eGFR less than 30 mL/min/1.73 m^2 at the time of imaging. There was no increased utilization of RRT after 24 h in those who received a CECT than those who received a UECT. Renal recovery rate was similar between cases and controls, and unaffected by the provision of iodinated contrast.

Conclusion The use of low-osmolar intravenous iodinated contrast does not impede the recovery of eGFR in critically ill patients with severe acute kidney injury.

Keywords Contrast induced nephropathy · Acute renal failure · Iodinated contrast · CT imaging

1 Background

Radiocontrast media is essential for enhancing the diagnostic accuracy of CT imaging but are often withheld from patients with impaired renal function at the time of imaging due to the potential risk of nephrotoxicity [1–4] although evidence regarding its contribution to progressive renal impairment is conflicting. Kidney injury, however, is common among patients who require hospitalization [5] even in those who lack exposure to contrast media. The use of low-osmolar and iso-osmolar formulations has not demonstrated a clear association with kidney injury in patients who require hospitalization, including those admitted to the intensive care unit [6–8] or those with reduced renal function [9–12]. Nevertheless, The American College of Radiology (ACR) advises considering withholding radiocontrast media in patients with an eGFR less than 30 mL/min/1.73 m^2.

2 Materials and Methods

2.1 Study Design

A single-center retrospective observational study of subjects who were admitted to two intensive care units from the emergency department, with an eGFR less than 30 mL/

✉ John Ferguson
jferguson@rockymtnpulmonary.com

Will DuSablon
willdusablon.do@gmail.com

[1] Department of Internal Medicine, Rocky Mountain Pulmonary and Critical Care Medicine, 730 Ward Road, #201, Arvada, CO, USA

[2] Department of Internal Medicine, Rocky Vista University, Parker, CO, USA

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min/1.73 m^2, who underwent CT imaging of the chest, or abdomen and pelvis. IRB certificate of approval was obtained through the local health research institute having met criteria for exempt research. Data collected was retrospective and in accordance with ethical standards and the Helsinki Declaration of 1975.

2.2 Patients

Subjects aged at least 18 years were included who had an eGFR less than 30 mL/min/1.73 m^2 at the time of imaging, regardless of their pre-hospitalization baseline renal function. Subgroup analysis was performed on subjects who had a diagnosis of chronic kidney disease (CKD) stage 3 prior to admission, but whose eGFR at the time of admission had declined to less than 30 mL/min/1.73 m^2. eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. All subjects were admitted to either a mixed medical/cardiac intensive care unit or to a neurocritical care unit over a five-year period between July 1, 2018, and July 1, 2023. Exclusion criteria included: those treated with chronic intermittent dialysis, arterial contrast administration within 72 h of admission, initiation of renal replacement therapy (RRT) within 24 h of admission, renal transplant recipients, death or lack of data within 48 h, or an obstructed chronic urinary catheter. All subjects received either Isovue-370 (lopamidol) or Omnipaque-350 (iohexol) contrast in a 100 mL volume.

2.3 Data Collection

Patient demographics, principal diagnosis, eGFR prior to hospitalization, comorbid diagnoses, SOFA score, antibiotic use, and left ventricular ejection fraction were retrospectively collected through the electronic medical record. eGFR values were assigned to the corresponding time point if they were collected within six hours of that time.

2.4 Endpoints

Acute kidney injury due to contrast media (CIN) was defined as a rise in serum creatinine of 0.5 mg/dL or a greater than 25% increase in creatinine between 48 and 72 h from the time of CT imaging [13]. Acute kidney injury, utilizing a modified Kidney Disease Improving Global Outcomes (KDIGO) criteria included: stage one as either a 0.3 mg/dL increase or 1.5-to-1.9-fold increase in creatinine, stage two defined by a 2-2.9-fold increase in creatinine. Stage three, which includes the need for RRT, was evaluated separately.

Primary endpoints included: the incidence of acute kidney injury, with separate assessments using both CIN and KDIGO criteria, the need for RRT during hospitalization, and the rate of recovery of kidney function. Renal recovery rate was determined by the change in eGFR from the time of imaging through the subsequent 120 h. Additionally, the time to recovery to baseline renal function category as defined by CKD stages, was used to assess renal recovery rate. Secondary endpoints included hospital mortality, hospital length of stay, and persistent need of RRT after discharge.

3 Statistical Analysis

Data analysis was performed using Stata (StataCorp 2019. Stata Statistical Software: Release 16.1. College Station, TX: StataCorp LLC). Subjects were categorized by those who received either a CECT (cases) or a UECT (controls). Nearest neighbor matching was performed by propensity-matching the eGFR value at the time of imaging and is displayed in Table 1. Mean values for continuous variables were compared using an unpaired t-test, while binary outcome frequencies were assessed using a Fisher's exact test. After matched subjects were established as equal and data as normal, regression analysis utilizing variables of time and iodinated contrast administration was performed to determine the treatment-effect coefficient of the change in eGFR from admission to the subsequent 120 h. A log-rank test was used to determine the difference in renal recovery to the CKD stage prior to admission, as categorized by: eGFR less than 15, 15–29, 30–44, 45–59, 60–90, and greater than 90 mL/min/1.73 m^2. Dropout values for measurements of eGFR, including those receiving RRT or deceased, were handled as missing data and not included within data point means.

4 Results

4.1 Baseline Data

A total of 673 subjects were identified who were admitted and met the inclusion criteria. Out of the 673 subjects identified, 293 were excluded (105 with end-stage renal disease, 61 who underwent a repeat CT within 48 h of admission, 29 who lacked 48 h of data, 36 who received contrast within 48 h of a UECT, 51 who received RRT within 24 h of admission, 7 with anatomic considerations, and 4 whose eGFR at the time of imaging was greater than 30 mL/min/1.73 m^2). The final number of included subjects was 380, with 67 cases receiving a CECT and 313 controls receiving a UECT. Propensity matching to a nearest neighbor of the CECT cases resulted in 67 controls receiving a UECT with a 1:1 ratio to the CECT cases.

Table 1 Summarizes unmatched and propensity-matched baseline characteristics

Characteristic	CECT	Unmatched UECT		Propensity-matched UECT	
	CECT ($n=67$)	UECT ($n=313$)	p	UECT ($n=67$)	p
Age (years)	66.97 (15.25)	66.97 (13.26)	0.99	69.10 (14.17)	0.40
Men, n (%)	27 (40.30)	159 (50.80)	0.14	22 (32.8)	0.79
Race, n (%)					
White	56 (83.58)	265 (84.66)	0.85	55 (82.09)	1.00
Black	1 (1.49)	3 (0.96)	0.54	0 (0)	1.00
Hispanic	9 (13.43)	36 (11.50)	0.68	10 (14.93)	1.00
Other	1 (1.49)	10 (3.19)	1.00	2 (2.99)	1.00
Weight (kg)	79.57 (24.48)	84.94 (51.59)	0.40	81.22 (29.20)	0.72
BMI (kg/m^2)	28.65 (8.09)	29.70 (16.11)	0.61	28.93 (8.49)	0.85
Pre-hospital CKD stage 3	25/55 (45.45)	88/273 (28.11)	0.06	28/58 (48.38)	0.85
Pre-hospital CKD stage 4	0/55 (0)	19/273 (6.96)	0.05	1/58 (1.72)	1.00
Diabetes mellitus, n (%)	10 (14.92)	78 (24.92)	0.08	16 (23.88)	0.28
Primary Diagnosis, n (%)					
Medical	60 (89.55)	262 (83.71)	0.27	58 (86.57)	0.72
Neurologic	4 (5.97)	25 (7.99)	1.00	4 (5.97)	1.00
Surgical	3 (4.48)	30 (9.58)	0.23	5 (7.46)	0.72
SOFA score	6.00 (3.41)	6.79 (3.56)	0.10	6.09 (3.03)	0.87
Invasive mechanical ventilation, n (%)	27 (40.30)	85 (27.16)	0.04	18 (26.87)	0.14
Vasopressor use, n (%)	25 (37.31)	161 (51.44)	0.04	33 (49.25)	0.22
Vancomycin, n (%)	24 (35.82)	110 (35.15)	1.00	24 (35.82)	1.00
Piperacillin-tazobactam, n (%)	11 (16.42)	59 (18.85)	0.73	9 (13.43)	0.81
Nephrology consultation, n (%)	17 (25.4)	107 (34.19)	0.20	10 (14.92)	0.20
Left ventricular ejection fraction (%)	57.49 (13.97)	57.34 (13.71)	0.94	56.45 (13.08)	0.71

Patient demographics. All values are described as mean (standard deviation), unless otherwise noted by (n, % of subjects). BMI = body mass index. SOFA = sequential organ failure assessment. CNS = central nervous system disease. DKA = diabetic ketoacidosis. CHF = congestive heart failure. GIB = gastrointestinal bleed. ACS = acute coronary syndrome

Table 2 Primary and secondary outcomes

Outcome	CECT	Unmatched UECT		Propensity-matched UECT	
Primary outcomes	CECT ($n=67$)	UECT ($n=313$)	OR (95% CI)	UECT ($n=67$)	OR (95% CI)
CIN, n (%)	7 (10.45)	38/310 (12.26)	0.84 (0.30, 2.02)	10 (14.92)	0.67 (0.20, 2.09
KDIGO stage 1–2, n (%)	7 (10.44)	41/310 (13.23)	0.77 (0.28, 1.86)	13 (19.40)	0.48 (0.15, 1.43)
Inpatient RRT, n (%)	4 (5.97)	32 (10.22)	0.56 (0.14, 1.66)	4 (5.97)	1.00 (0.18, 5.62)
Secondary outcomes					
Discharged on RRT, n (%)	0 (0.00)	6 (1.92)	0.00 (0.00, 2.95)	2 (2.98)	0.00 (0.00, 1.91)
Death in hospital, n (%)	13 (19.40)	63 (20.13)	0.96 (0.45, 1.91)	13 (19.40)	1.00 (0.39, 2.58)
Length of stay (days)	14.10 +/- 13.73	9.77 +/- 7.82	0.0005	8.85 +/- 13.73	0.05

CIN = contrast-induced nephropathy. KDIGO = kidney disease improving global outcomes. RRT = renal replacement therapy

4.2 Primary Endpoints

4.2.1 Incidence of Progressive AKI

Table 2 summarizes the primary and secondary endpoints. The incidence of progressive AKI, assessed separately using CIN and KDIGO criteria, was similar between unmatched cases and controls, with odds ratios (ORs) of 0.84, 95% CI [0.30, 2.02] and 0.77, 95% CI [0.28, 1.86], respectively. Following propensity-matching, there was no difference in the incidence of progressive AKI by these criteria with ORs of 0.67, 95% CI [0.20, 2.09] and 0.48, 95% CI [0.15, 1.43].

Within the subgroup of subjects with pre-hospitalization CKD stage 3, whose eGFR had declined to less than 30 mL/min/1.73 m^2 at the time of imaging, there was no difference in the incidence of AKI between cases and controls, using either CIN criteria or KDIGO criteria, with ORs in unmatched subjects of 1.07, 95% CI [0.23, 3.96] and 0.66 95% CI [0.11, 2.68], and in propensity-matched subjects of 0.88, 95% CI [0.15, 4.70] and 0.41, 95% CI [0.06, 2.13].

Among the most severely impaired subjects with eGFR < 15 mL/min/1.73 m^2 at the time of imaging, there was no difference in the incidence of progressive AKI by either CIN or KDIGO criteria between cases and controls

in unmatched (OR 0.00, 95% CI [0, 6.56]) subjects. There were no instances of progressive AKI in all 12 matched subjects.

4.2.2 Incidence of RRT

There was no difference in the RRT utilization in either unmatched or propensity-matched subjects, with ORs of 0.56, 95% CI [0.14, 1.66] and 1.00, 95% CI [0.18, 5.62], respectively. Of the 36 unmatched subjects who required inpatient RRT, 13 (33.3%) had pre-hospitalization CKD stage 3 or greater. Of the eight propensity-matched subjects who required inpatient RRT, 6 (75%) had pre-existing CKD stage 3 or greater.

4.2.3 Rate of Renal Recovery

The progression of eGFR over the hospital duration is displayed in Fig. 1. Among propensity-matched subjects, eGFR was similar between cases and controls through the first 72 h, however, subjects who received a CECT experienced a greater eGFR than those who underwent UECT at both 96 h (64.33 vs. 49.49 mL/min/1.73 m^2, $p=0.01$) and at 120 h (70.16 vs. 50.48 mL/min/1.73 m^2, $p=0.003$). A linear regression model demonstrated a more rapid recovery of eGFR in subjects who received a CECT than those who received a UECT. The coefficient difference was 2.15 mL/min/1.73 m^2 per 24 h, 95% CI [1.64, 2.66], $p<0.05$) in unmatched subjects, and 3.71 mL/min/1.73 m^2 per 24 h, 95% CI [2.62, 4.80], $p<0.05$) in propensity-matched subjects.

Of unmatched subjects who had available pre-hospital kidney function, more subjects who received a CECT experienced resolution back to their baseline CKD stage or better by 120 h than those who received a UECT (69.49% vs. 49.27%, $p=0.0003$). Among propensity-matched subjects, there was no difference in recovery to baseline renal function between subjects who received a CECT or a UECT (69.49% vs. 57.38%, $p=0.25$) (Fig. 2).

4.3 Secondary Endpoints

4.3.1 Hospital Mortality

Hospital mortality was similar between cases and controls, with ORs of 0.96, 95% CI [0.45, 1.91] and 1.00, 95% CI [0.39, 2.58] both before and after propensity-matching, respectively.

4.3.2 Persistent RRT at Discharge

No cases who received a CECT required RRT after discharge, although death occurred in two of the four subjects who received RRT during their hospitalization. Of

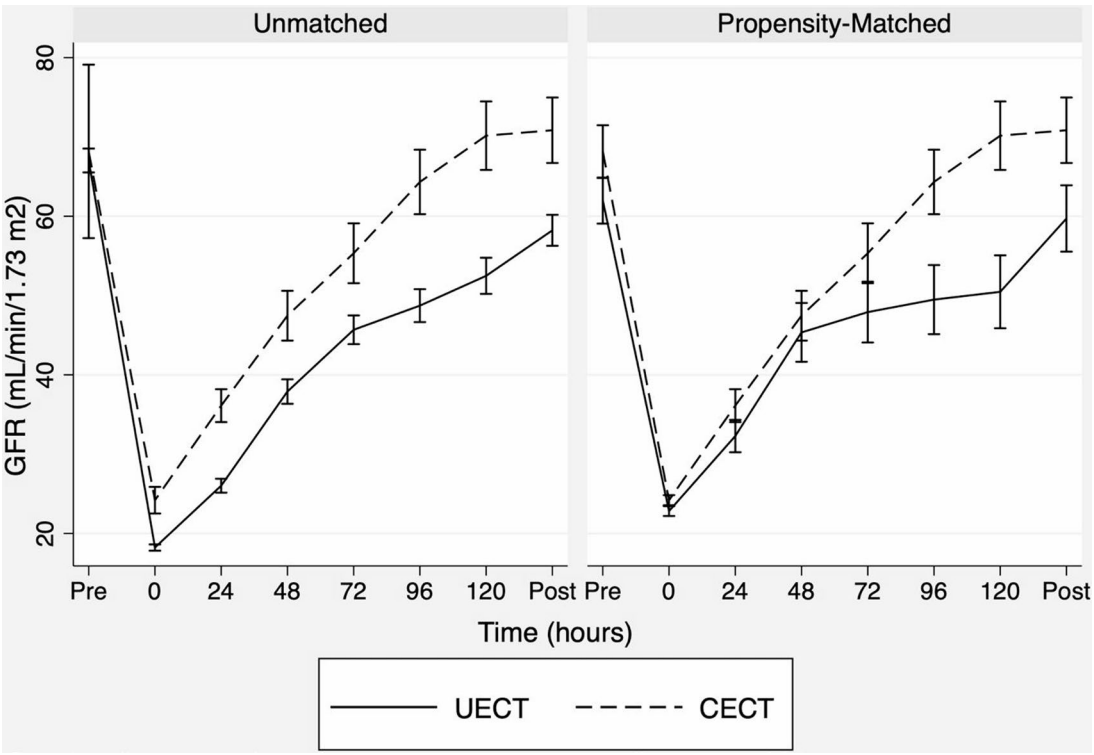

Fig. 1 eGFR over time of hospitalization Data points are displayed as mean values with standard error of the mean bars. Pre = Baseline eGFR prior to hospitalization. Post = eGFR at time of discharge

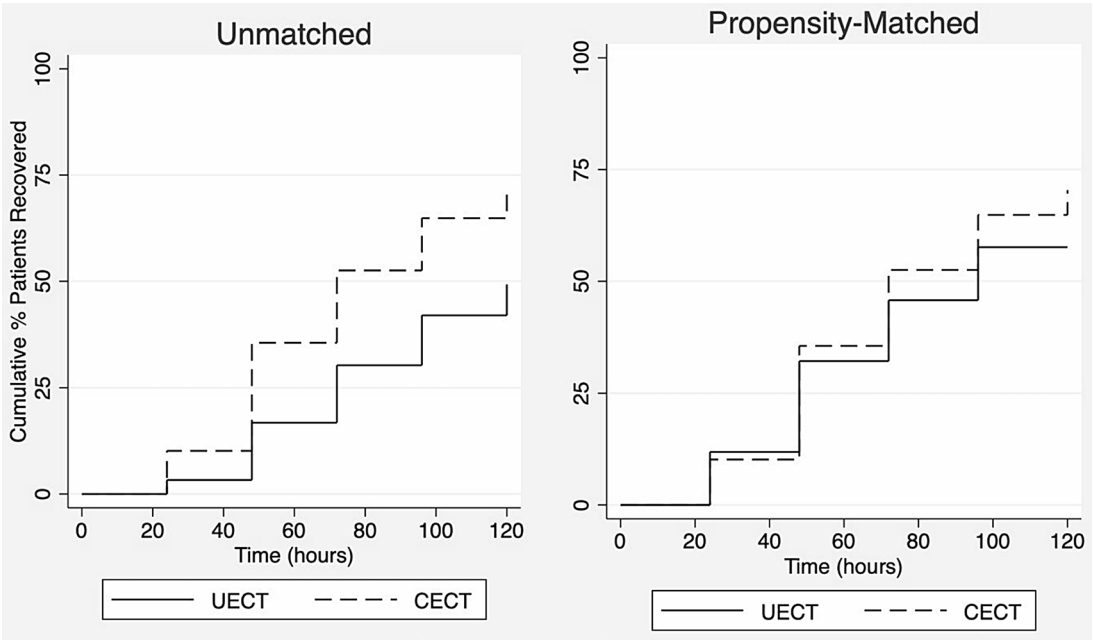

Fig. 2 Time until recovery to prior CKD stage, alive, and without RRT

the six controls who received a UECT and required RRT after discharge, one had pre-existing CKD stage 3, one had CKD stage 4, two had normal renal function, and two had unknown baseline renal function.

4.3.3 Length of Stay

Length of stay was greater in subjects who received a CECT compared to those who received a UECT, both before (14.10 +/- 13.73 vs. 9.89 +/- 7.86, $p=0.00005$), and after propensity matching (14.10 +/- 13.73 vs. 8.85 +/- 13.73, $p=0.05$).

5 Discussion

In conclusion, among critically ill patients admitted to the ICU with an eGFR less than 30 mL/min/1.73 m^2, those who received a CECT faced no greater risk of progressive kidney injury than those who received a UECT. These findings are consistent with the ORs of CIN or KDIGO criteria in patients with similarly impaired renal function as reported by Hinson 0.86 and 0.68) [10], Tao (0.46 and 0.31) [9], and McDonald (0.97 and Unavailable) [12], but differ with those of Gorelik (Unavailable and 1.52) [4], Davenport (Unavailable and 2.96) [1], Su (1.38 and 1.36) [3], and Ellis (Unavailable and 3.96) [2].

The explanation for the discordant findings may be related to the chronicity of renal impairment at the time of contrast administration. Most subjects in our cohort had documented pre-hospital renal function with an eGFR greater than 30 mL/min/1.73 m^2. This population who presented with acute renal failure is similar with the patients admitted through the emergency department as reported by Hinson [10]. The population studied by Ehmann, which showed no increased risk of persistent AKI at discharge (OR 0.88), likewise required acute kidney injury for enrollment, although patients with CKD stage 4 or higher were not explicitly excluded [11]. Similarly, on account of patient enrollment methods, the cohort studied by McDonald tended to be hospitalized patients with less chronic kidney disease (greater than 90% of the study population was inpatient, and 50% were classified as acute renal failure) [12]. Iodinated contrast media may pose a greater risk to patients with stable chronic renal disease than those with acutely impaired renal function. For example, among patients with a stable eGFR less than 30 mL/min/1.73 m^2, iodinated contrast has been found to be an independent risk factor for progression of AKI as described by Ellis, who concluded that these results might only be applicable to patients with stable CKD [2]. Although not explicitly stated, Su hypothesized that their large proportion of patients with chronically impaired kidney disease in a Taiwanese population might explain the increased incidence of AKI observed [3]. Likewise, Davenport included only patients with impaired, but stable, renal function and excluded those with acute injury [1]. In contrast, Tao did not observe any increased incidence of AKI in patients with stable CKD stage 4–5, although these were in small numbers and all patients enrolled carried a diagnosis of nephrotic syndrome [9].

It is logical that a minor insult such as iodinated contrast would fail to alter the trajectory or prognosis of patients who present with acute severe impairment of renal function. The weight of the acute pathology that leads to impaired renal function will almost certainly exceed any contribution of iodinated contrast. Of the patients in our population who received a CECT, 55 possessed a pre-hospital baseline eGFR greater than 30 mL/min/1.73 m^2. The remaining 12 subjects had unknown baseline renal function, but presumably was within normal limits. Patients who received a CECT had a more rapid rate of recovery of renal function than those with a UECT by all measures, with curves diverging after 72 h in favor of those receiving a CECT. It is plausible that improved diagnostic testing hastened the improvement of renal function. It is equally plausible that this phenomenon can be explained by patient selection and more careful care, including early HD, of those with impaired function who received a CECT. Indeed, there was a trend towards increased nephrology consultation in those receiving a CECT in propensity-matched patients.

In addition to the incidence of AKI and time to recovery, other important endpoints should be considered, including persistent renal dysfunction at the time of discharge. Like the study by Ehmann [11], we found no increased risk of persistent AKI at the time of discharge in patients with an eGFR less than 30 mL/min/1.73 m^2. The use of RRT was more common in patients who received a UECT, and very few patients required RRT at discharge. Those who were initiated on early RRT within 24 h were excluded, and when including these subjects, the incidence of RRT was similar (19.3% vs. 23.2%). It is unclear as to why, despite a more rapid renal recovery, the length of stay was longer for those who received a CECT compared to those who received a UECT.

Limitations include the use of a calculated eGFR in patients with unstable renal function. Calculations of eGFR are validated for those with stable renal function, however, this is the value used in clinical practice for acutely ill patients. These findings cannot be generalized to patients with a steady stage eGFR and CKD stage 4 or 5. Second, our cohort consisted of a limited number of patients who received a CECT at 67 cases, although our number of patients with severely impaired renal function that was targeted was similar in number to that in Hinson [10] and Davenport [1], while the study by Gorelik [4] was approximately four times larger and Su [3] approximately ten times larger than our population. There were also few patients in our cohort with an eGFR less than 15 mL/min/1.73 m^2, although interestingly, none of the six subjects with this degree of impairment who received a CECT met either CIN criteria nor required RRT, whereas 9.48% of controls with this severity of impairment who received a UECT developed criteria for CIN. Our unmatched subjects had differing eGFR values at the time of CT imaging, which was expected due to ordering bias. Although our unmatched cohorts were similar in baseline characteristics, additional biases on the part of the prescribing physician, such as extremes of age, may influence the decision whether or not to administer iodinated contrast. Thus, propensity-matching was essential in determining the independent contribution of iodinated contrast, and following propensity matching, eGFR at the time of imaging was similar between cases and controls. Lastly, unlike prior studies, rather than extensive regression analysis, we used a descriptive linear regression of the change in eGFR over time. Given the sample size and similar baseline characteristics, it is unlikely that any other covariate would be expected to significantly change the outcome of the trend in eGFR.

Despite the conflicting literature regarding contrast-induced nephropathy, there remains no large randomized controlled trial between CECT and UECT in patients with an eGFR less than 30 mL/min/1.73 m^2. Ethically this would be difficult to perform and would be confounded by missed diagnoses and their consequences. It is not straightforward to claim that the use of iodinated contrast in isolation either does or does not contribute to progressive kidney injury. In the same manner that it is necessary to differentiate hyperosmolar from iso-osmolar or low-osmolar formulations, and intravenous from intra-arterial route of administration, baseline renal function must be considered in the clinical decision-making process. Similarly, the amount of iodinated contrast used may influence the risk of CIN; however, the volume of 100 mL administered at our institution appears to be safe. Iodinated contrast should not be withheld, nor solely held responsible, for AKI when needed to aid in a diagnosis of a critically ill patient, but alternative diagnostic tools may be considered in patients with known CKD stage 4 or 5. Among those with an eGFR acutely reduced to less than 30 mL/min/1.73 m^2, the use of low-osmolar iodinated intravenous contrast appears unlikely to impact the natural rate of kidney recovery.

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Data Availability Data may be available upon request from the corresponding author.

Code Availability Data analysis was performed using Stata (StataCorp 2019. Stata Statistical Software: Release 16.1. College Station, TX: StataCorp LLC.

Declarations

Competing Interests The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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