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## Association Between Acute Kidney Disease and IV Dye Administration in Acute Stroke Patients: A Population-Based Study

Stacie L Demel, DO, PhD<sup>1,2</sup>, Aaron W Grossman, MD, PhD<sup>1,2</sup>, Jane C Khoury, PhD<sup>1,3</sup>, Charles J Moomaw, PhD<sup>1</sup>, Kathleen Alwell, RN BSN<sup>1</sup>, Brett M Kissela, MD MS<sup>1,2</sup>, Daniel Woo, MD, MS<sup>1,2</sup>, Matthew L Flaherty, MD<sup>1,2</sup>, Simona Ferioli, MD<sup>1,2</sup>, Jason Mackey, MD MS<sup>4</sup>, Felipe De Los Rios la Rosa, MD<sup>1,5</sup>, Sharyl Martini, MD, PhD<sup>6</sup>, Opeolu Adeoye, MD<sup>1,7</sup>, and Dawn Kleindorfer, MD<sup>1,2</sup>

<sup>1</sup>Neuroscience Institute, University of Cincinnati College of Medicine, Cincinnati, OH

<sup>2</sup>University of Cincinnati Medical Center Department of Neurology and Rehabilitation, Cincinnati, OH

<sup>3</sup>Cincinnati Children's Hospital Medical Center Division of Biostatistics and Epidemiology, Cincinnati, OH

<sup>4</sup>University of Indiana School of Medicine Department of Neurology, Indianapolis, IN

<sup>5</sup>Sanna Healthcare Network, Lima, Peru

<sup>6</sup>Baylor College of Medicine Department of Neurology, Houston, TX

<sup>7</sup>Department of Emergency Medicine, University of Cincinnati College of Medicine, Cincinnati, OH

### Abstract

**Background and Purpose**—CT angiography (CTA) and conventional angiography provide timely vascular anatomical information in stroke patients. However, iodinated contrast dye may cause acute kidney injury (AKI). Within a large, biracial population, we examined in-hospital incidence of new or worsening kidney disease in stroke patients and its association with administration of intravenous (IV) dye.

**Methods**—All adult residents of the Greater Cincinnati/Northern Kentucky (GCNK) region with acute ischemic stroke (IS) or intracerebral hemorrhage (ICH) who presented to an emergency department in 2010 were included. Prevalence of unsuspected kidney disease at the time of ED presentation was determined as well as incidence of AKI after admission in two groups of patients – those who did and those who did not receive IV dye.

**Results**—In 2010, 2299 patients met inclusion criteria (89% IS, 11% ICH); mean age 69 years (SD 15), 22% black, 54% women. Among these patients, 37% had kidney disease at baseline, including 22% (516/2299) in whom this was unsuspected. Two percent (2%; 15/853) of patients

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Corresponding author: Stacie L. Demel 260 Stetson Street, Suite 2300 Cincinnati, OH 45219, fax: 513-558-4887; phone: 513-558-5478; Stacie.demel@gmail.com.

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with baseline kidney disease developed AKI during the hospital stay. Of those with no baseline kidney disease, 1% (14/14467) developed AKI. There was no association between dye administration and new or worsening kidney disease.

**Conclusions**—Although 22% of patients in the GCNK stroke population had unsuspected kidney disease, the incidence of new or worsening kidney disease was low, and AKI was not associated with dye administration. These findings confirm single-center reports that the risk of severe renal complications after contrast dye is small.

### Keywords

Diagnostic Imaging; Stroke; Acute Kidney Injury; Contrast Media; Epidemiology

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## INTRODUCTION

In patients with acute ischemic or hemorrhagic stroke, early vascular imaging is important for medical and interventional management. However, iodinated radiographic contrast media carries a risk of renal toxicity and decreased renal function<sup>1, 2</sup>.

Contrast-induced nephropathy (CIN) is defined as acute renal failure occurring within 24–72 hours after the administration of iodinated radiographic contrast media that cannot be attributed to other causes<sup>3</sup>. CIN is uncommon in patients with normal preexisting renal function, but it occurs more frequently in patients with underlying renal disease, particularly diabetic nephropathy<sup>4</sup>. CIN accounts for 12% of all cases of hospital-acquired acute renal failure<sup>2</sup>, and its occurrence can be associated with worse outcomes and increased health-care costs<sup>5</sup>. The safety of administering contrast without regard to renal function in an acute stroke patient is controversial and much of the evidence that exists is limited to single center retrospective studies<sup>6, 7</sup>. Given the advanced age and vascular risk factors of stroke patients, it is possible that the incidence of CIN may be significantly higher in these patients and that CIN might lead to higher rates of poor outcomes. The risk of CIN is a common rationale for delaying vascular imaging until a serum creatinine has been obtained, thus delaying identification of large vessel occlusions and interventional therapy, in the case of ischemic stroke. Yet within the stroke population, the rates of unsuspected kidney disease (patients with elevated creatinine despite no known history of CKD) and the risk of new or worsened kidney disease after contrast-based imaging remain unknown.

We therefore used data from the Greater Cincinnati/Northern Kentucky Stroke Study (GSNKSS), a large population-based stroke incidence study to pose the following questions: 1) what is the prevalence of unsuspected kidney disease in stroke patients at the time of presentation, and 2) how often was administration of iodinated contrast during cerebrovascular imaging, associated with AKI or renal failure requiring dialysis during their hospitalization? These data may help practitioners assess the risk of first-line vascular imaging during acute evaluation of stroke patients.

## METHODS

Details of the design and conduct of the GCNKSS have been described elsewhere<sup>8</sup>. Briefly, in calendar year 2010, all acute ischemic strokes (AIS) and intracranial hemorrhages (ICH) among the approximately 1.3 million residents of the GCNK region were ascertained by identifying inpatient discharge International Classification of Diseases-Ninth Revision codes 430 to 436 at fifteen area adult hospitals. We have previously found that residents in this population exclusively seek care at these hospitals<sup>9</sup>. All residents of the region 20 years of age with AIS or ICH who presented to an emergency department and survived at least two days were included in this analysis. Medical records from the acute hospitalization were retrospectively reviewed by study nurses, and designation of stroke and stroke type were verified by study-physician review. Age, race, sex, stroke risk factors, admission serum creatinine (Cr), and vascular imaging were among the items included in the abstraction. Either history of chronic kidney disease (CKD) and/or elevated Cr at the time of presentation represented “baseline renal insufficiency” in this study. “Unsuspected kidney disease” was defined as a Cr  $\geq 1.2$  on presentation, without prior history of CKD or end-stage renal disease (ESRD) as provided by chart review or patient history. The decision of whether or not IV dye was administered hyper-acutely was made by individual ER physician and/or individual physicians who are part of the University of Cincinnati Stroke Team based upon their clinical judgment.

Our study methodology systematically reviews each patient’s record for medical comorbidities, as detailed previously<sup>10</sup>. Development of an AKI during the hospital stay was defined as chart documentation of “acute renal failure,” “new dialysis,” or “renal insufficiency” documented in a patient with no prior history of such. Worsening kidney disease was determined by physician documentation of “acute on chronic renal failure,” “new dialysis,” or “tunnel dialysis catheter insertion” in patients with history of CKD or ESRD. Incidence of new or worsening kidney function was assessed and stratified by use of IV dye.

### Data analysis

SAS, version 9.4 (SAS institute, Cary NC) was used for analysis. Univariate analysis was used to examine distributions of continuous variables and frequency of categorical variables. T-test and Chi-square analysis was used as appropriate to compare groups (those with versus without renal insufficiency, and those with and without use of dye). Results were used to determine potential predictors of unsuspected renal insufficiency; variables associated with unsuspected renal insufficiency at  $p < .20$  were included in the initial multiple logistic regression model. Fisher’s exact test was used to test the association of use of dye with new or worsening kidney function individually as numbers were too small for using multiple logistic regression analysis to examine multiple predictors of this outcome. Multiple logistic regression was used to examine the association of dye administration with the combined outcome of new or worsening kidney function, adjusting for the a priori chosen covariates of renal function at baseline, history of renal disease and age.

## RESULTS

In 2010, 2299 patients among residents of the GCNK region who presented to an emergency department with AIS (89%) or ICH (11%) met inclusion criteria for this analysis. The mean age was 69 years (standard deviation 15); 22% of the cohort was black, and 54% were female. Among all 2299 patients, 283 (12%) had IV dye-based vascular imaging, and 853 (37%) had either history of kidney disease (n=337) or elevated Cr (n=516) at the time of presentation (Table 1). Older age, black race, male sex, history of diabetes, and history of hypertension were associated with the finding of CKD at baseline. Current smoking and ICH were more prevalent in the group without CKD at baseline.

### Characteristics of patients stratified by baseline renal function and contrast exposure

We evaluated differences in baseline characteristics between those who received contrast for vascular imaging and those who did not and stratified by baseline renal function (Table 2). We found that in the 1447 patients without baseline kidney disease, 204 (14%) received dye. Patients without baseline kidney disease who received dye were younger, had less hypertension, were more commonly current smokers, and were more likely to be male compared with those without baseline kidney disease who did not receive IV dye. In the 853 patients with baseline kidney disease, 79 (9%) received contrast. Patients with baseline kidney disease who received contrast were younger, but no other characteristics were significantly different between those receiving dye versus not receiving dye.

### Unsuspected kidney disease

In this population of 2299 stroke patients, 1962 had no history of CKD. Using Cr of 1.2 upon presentation as a definition of unsuspected renal insufficiency, 516 of these patients (26%) presented with unsuspected kidney disease (Table 3). Multivariable logistic regression analysis showed that older age, black race, male sex, hypertension, and diabetes were all independently associated with unsuspected kidney disease (R-square=0.14). However, current smoking and stroke type was not statistically significant. Adjusted odds ratios are shown in Table 3.

Of the 516 patients with unsuspected kidney disease, 53 received IV contrast for vessel imaging. Comparison of characteristics, between those who did versus those who did not receive IV dye, revealed the only significant difference between groups was mean age (67.1 vs 72.8 years;  $p = .003$ ), with older patients less likely to receive contrast.

Of note, 40 patients with a history of CKD had normal Cr upon arrival, i.e., they had “unsuspected renal *sufficiency*.”

### Incidence of new or worsening renal insufficiency in acute stroke patients stratified by contrast exposure

Only 29 cases (1.3%) out of the total cohort (N = 2299) had either acute kidney injury or worsening kidney disease. After adjusting for renal function at baseline, history of renal disease and age, the administration of dye was not statistically significant for increasing the

odds of the outcome of acute or worsening kidney disease,  $p = 0.69$ ; aOR = 1.125 (95% CI 0.42, 3.70).

Those who developed new or worsening kidney disease were stratified by baseline renal function and by whether contrast was administered for cerebrovascular imaging (Table 4). There was no significant difference in the incidence of new or worsening kidney disease between patients who received IV contrast for acute stroke vessel imaging and those who did not receive contrast. Of the 79 patients with baseline kidney disease who received dye, only one (1.3%) developed worsening renal function, compared with 14 (1.8%) of the 774 who did not receive dye. Among the 53 patients with unsuspected kidney disease who received IV dye, 1 (1.9%) had worsening creatinine during their hospital stay compared with 4 (0.9%) of the 463 who did not receive dye ( $p=.42$ ). Independent predictors of new or worsening kidney disease among patients who received IV dye cannot be determined because of the small number of patients with the outcomes of interest. Similar proportions were seen in the group of patients with normal renal function at baseline, in which three (1.5%) of the 204 who received dye versus 11 (0.9%) of the 1242 who did not receive dye developed an AKI ( $p=.43$ ).

None of the patients who had contrast-based cerebrovascular imaging developed AKI requiring dialysis during their hospitalization. Only one patient required new dialysis during stroke admission; this patient did not receive IV contrast.

## DISCUSSION

In this population-based study, we found that although one in four patients presented with unsuspected kidney disease, the overall incidence of an AKI after IV contrast administration in stroke patients was extremely low. These findings are consistent with prior single-center studies of contrast induced nephropathy after cerebrovascular imaging in acute stroke patients<sup>11</sup>, but have not been previously reported in a population-based analysis.

Prior studies estimate that the incidence of CIN from IV dye given in an emergency department for all diagnoses is approximately 7%–11%<sup>12, 13</sup>. In a large, retrospective case-control study of 10,121 patients who received low osmolar iodinated contrast media and 10,121 control patients, a difference in nephrotoxicity was observed only in patients with  $eGFR < 30 \text{ mL/min/1.73m}^2$ . No difference was seen in the incidence of acute renal failure, dialysis, or death between groups in this study<sup>14</sup>. Furthermore, many studies have shown that kidney function usually returns to preexisting levels within 7 days<sup>15</sup>, and AKI after radiocontrast administration rarely requires acute dialysis treatment<sup>16</sup>. Finally, a single-center study found that after adjusting for differences in presumed risk factors of CIN, non-contrast CT scans were at equivalent risk of serum creatinine-defined acute kidney injury (AKI) compared with recipients of contrast-enhanced CT scans, regardless of baseline renal function<sup>17</sup> suggesting that baseline kidney function alone may not be the best predictor for who will go onto to develop an AKI after receiving IV dye. Although these studies have similar conclusions, they were not limited to stroke patients or to neurovascular imaging.

Because of the potential side effects, the American College of Radiology, the Canadian Association of radiologists, and the European Society of Urogenital Radiology have each published guidelines for IV contrast medium administration based on various and conflicting serum creatinine values. This practice translates into fewer IV contrast-based studies being completed at the expense of diagnostic accuracy.

With the recent publication of positive endovascular trials<sup>18–21</sup> the need for hyper-acute vessel evaluation and endovascular treatment is now stand of care. The recent Focused Update of the American Heart Association Guidelines for the Early Management of Patients with Acute Ischemic Stroke Regarding Endovascular Treatment updated their imaging recommendations:

- “If endovascular therapy is contemplated, a noninvasive intracranial vascular study is strongly recommended during the initial imaging evaluation of the acute stroke patient (Class I; Level of Evidence A).”<sup>22</sup>

Waiting for a creatinine on acute stroke patients before administering contrast may significantly delay treatment and decrease the chance for a good outcome, given that a 45-minute delay in reperfusion translates to a 10% decreased likelihood of a good outcome.<sup>23</sup> Our data suggest that it is generally safe to administer contrast for cerebrovascular imaging because the incidence of CIN nephropathy is extremely low, even in patients with known CKD and the ~27% of stroke patients with unsuspected renal insufficiency.

There are limitations in the interpretation of our results due to their retrospective nature. Serial creatinine lab values were not collected and therefore clinical chart review and subjective data were used to define both CKD and AKI in this study. Despite this drawback, we are confident that the risk of missed dialysis patients is low. In addition, the creatinine threshold used to define CKD in this study is lower than most definitions; yet we still found extremely low incidence of new AKI. Another important limitation is selection bias. Physicians were likely selecting healthier stroke patients to receive IV contrast dye, which was reflected in the multiple differences in baseline characteristics between those who received versus did not receive IV contrast. Furthermore, patients who died within two days, presumably the sickest patients, were not included, thus creating further selection bias. We are therefore unable to determine risk factors for CIN in all patients, and we can comment only on the complications among those selected to receive dye by the treating physicians. A prospective trial to answer this question is unlikely to be completed and ultimately the risk of renal failure must be weighed against the benefits of rapid assessment of the cerebrovasculature in patient’s suspected of having an ischemic stroke.

## CONCLUSION STATEMENT

In summary, data from a population-based epidemiology study do not show any increase in AKI in those receiving dye for imaging studies. While further study is needed, our results support previous work showing low risk. Given the potential benefit of acute vascular imaging, available data suggest that concern the risk of contrast induced nephropathy should not delay first-line cerebrovascular imaging that may be useful for acute stroke therapy.

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## REFERENCES

1. McCullough PA, Soman SS. Contrast-induced nephropathy. *Critical care clinics*. 2005; 21:261–280. [PubMed: 15781162]
2. Gleeson TG, Bulugahapitiya S. Contrast-induced nephropathy. *AJR. American journal of roentgenology*. 2004; 183:1673–1689. [PubMed: 15547209]
3. Andreucci M, Faga T, Pisani A, Sabbatini M, Michael A. Acute kidney injury by radiographic contrast media: Pathogenesis and prevention. *BioMed research international*. 2014; 2014:362725. [PubMed: 25197639]
4. Katzberg RW, Newhouse JH. Intravenous contrast medium-induced nephrotoxicity: Is the medical risk really as great as we have come to believe? *Radiology*. 2010; 256:21–28. [PubMed: 20574082]
5. Levy EM, Viscoli CM, Horwitz RI. The effect of acute renal failure on mortality. A cohort analysis. *JAMA : the journal of the American Medical Association*. 1996; 275:1489–1494. [PubMed: 8622223]
6. Mehdiratta M, Schlaug G, Kumar S, Caplan LR, Selim M. Reducing the delay in thrombolysis: Is it necessary to await the results of renal function tests before computed tomography perfusion and angiography in patients with code stroke? *Journal of stroke and cerebrovascular diseases : the official journal of National Stroke Association*. 2008; 17:273–275. [PubMed: 18755406]
7. Oleinik A, Romero JM, Schwab K, Lev MH, Jhavar N, Delgado Almandoz JE, et al. Ct angiography for intracerebral hemorrhage does not increase risk of acute nephropathy. *Stroke; a journal of cerebral circulation*. 2009; 40:2393–2397.
8. Kleindorfer DO, Khoury J, Moomaw CJ, Alwell K, Woo D, Flaherty ML, et al. Stroke incidence is decreasing in whites but not in blacks: A population-based estimate of temporal trends in stroke incidence from the greater cincinnati/northern kentucky stroke study. *Stroke; a journal of cerebral circulation*. 2010; 41:1326–1331.
9. Broderick J, Brott T, Kothari R, Miller R, Khoury J, Pancioli A, et al. The greater cincinnati/northern kentucky stroke study: Preliminary first-ever and total incidence rates of stroke among blacks. *Stroke; a journal of cerebral circulation*. 1998; 29:415–421.
10. Kissela B, Lindsell CJ, Kleindorfer D, Alwell K, Moomaw CJ, Woo D, et al. Clinical prediction of functional outcome after ischemic stroke: The surprising importance of periventricular white matter disease and race. *Stroke; a journal of cerebral circulation*. 2009; 40:530–536.
11. Krol AL, Dzialowski I, Roy J, Puetz V, Subramaniam S, Coutts SB, et al. Incidence of radiocontrast nephropathy in patients undergoing acute stroke computed tomography angiography. *Stroke; a journal of cerebral circulation*. 2007; 38:2364–2366.
12. Mitchell AM, Jones AE, Tumlin JA, Kline JA. Incidence of contrast-induced nephropathy after contrast-enhanced computed tomography in the outpatient setting. *Clinical journal of the American Society of Nephrology : CJASN*. 2010; 5:4–9. [PubMed: 19965528]
13. Traub SJ, Kellum JA, Tang A, Cataldo L, Kancharla A, Shapiro NI. Risk factors for radiocontrast nephropathy after emergency department contrast-enhanced computerized tomography. *Academic emergency medicine : official journal of the Society for Academic Emergency Medicine*. 2013; 20:40–45. [PubMed: 23570477]
14. Davenport MS, Khalatbari S, Dillman JR, Cohan RH, Caoili EM, Ellis JH. Contrast material-induced nephrotoxicity and intravenous low-osmolality iodinated contrast material. *Radiology*. 2013; 267:94–105. [PubMed: 23360737]
15. Guierrez NV, Diaz A, Timmis GC, O'Neill WW, Stevens MA, Sandberg KR, et al. Determinants of serum creatinine trajectory in acute contrast nephropathy. *Journal of interventional cardiology*. 2002; 15:349–354. [PubMed: 12440177]
16. Barrett BJ, Parfrey PS. Clinical practice. Preventing nephropathy induced by contrast medium. *The New England journal of medicine*. 2006; 354:379–386. [PubMed: 16436769]

17. McDonald RJ, McDonald JS, Bida JP, Carter RE, Fleming CJ, Misra S, et al. Intravenous contrast material-induced nephropathy: Causal or coincident phenomenon? *Radiology*. 2013; 267:106–118. [PubMed: 23360742]
18. Berkhemer OA, Fransen PS, Beumer D, van den Berg LA, Lingsma HF, Yoo AJ, et al. A randomized trial of intraarterial treatment for acute ischemic stroke. *The New England journal of medicine*. 2015; 372:11–20. [PubMed: 25517348]
19. Saver JL, Goyal M, Bonafe A, Diener HC, Levy EI, Pereira VM, et al. Stent-retriever thrombectomy after intravenous t-pa vs. T-pa alone in stroke. *The New England journal of medicine*. 2015; 372:2285–2295. [PubMed: 25882376]
20. Jovin TG, Chamorro A, Cobo E, de Miquel MA, Molina CA, Rovira A, et al. Thrombectomy within 8 hours after symptom onset in ischemic stroke. *The New England journal of medicine*. 2015; 372:2296–2306. [PubMed: 25882510]
21. Goyal M, Demchuk AM, Menon BK, Eesa M, Rempel JL, Thornton J, et al. Randomized assessment of rapid endovascular treatment of ischemic stroke. *The New England journal of medicine*. 2015; 372:1019–1030. [PubMed: 25671798]
22. Powers WJ, Derdeyn CP, Biller J, Coffey CS, Hoh BL, Jauch EC, et al. 2015 american heart association/american stroke association focused update of the 2013 guidelines for the early management of patients with acute ischemic stroke regarding endovascular treatment: A guideline for healthcare professionals from the american heart association/american stroke association. *Stroke; a journal of cerebral circulation*. 2015; 46:3020–3035.
23. Khatri P, Yeatts SD, Mazighi M, Broderick JP, Liebeskind DS, Demchuk AM, et al. Time to angiographic reperfusion and clinical outcome after acute ischaemic stroke: An analysis of data from the interventional management of stroke (ims iii) phase 3 trial. *The Lancet. Neurology*. 2014; 13:567–574. [PubMed: 24784550]



**Table 1**

Characteristics of Ischemic and Hemorrhagic Stroke Patients in the Greater Cincinnati/Northern Kentucky Stroke Study of 2010, Stratified by Baseline Renal Function

	<b>Total population (n = 2299)</b>	<b>Without kidney disease at baseline (n = 1446; 62.9%)</b>	<b>With kidney disease at baseline* (n = 853; 37.1%)</b>	<b>p-value **</b>
Age; mean (SD)	69.3 (15.0)	67.3 (15.3)	72.7 (14.0)	<.0001
Race (black)	513 (22.3%)	281 (19.4%)	232 (27.2%)	<.0001
Sex (female)	1241 (54.0%)	855 (59.1%)	386 (45.2%)	<.0001
HTN	1853 (80.6%)	1085 (75.0%)	768 (90.0%)	<.0001
DM	775 (33.7%)	410 (28.4%)	365 (42.8%)	<.0001
Current smoking	629 (27.4%)	429 (29.7%)	200 (23.4%)	.001
ICH	258 (11.2%)	181 (12.5%)	77 (9.0%)	.01

\* Defined as either elevated Cr (  $\geq 1.2$ ) upon arrival or history of kidney disease

\*\* Comparison of patients with and without kidney disease at baseline

Data presented as mean (standard deviation) or n (%)

Characteristics of Ischemic and Hemorrhagic Stroke Patients in the Greater Cincinnati Northern Kentucky Stroke Study, Stratified by Presence or Absence of Contrast Administration for Cerebrovascular Imaging.

**Table 2**

	Stroke patients without kidney disease at baseline (n= 1446; 62.9%)		Stroke patients with CKD or elevated Cr at baseline (n=853; 37.1%)		p-value
	Received Dye n=204	No Dye n=1242	Received Dye n=79	No Dye n=774	
Age	58.2 (14.6)	68.8 (14.9)	68.5 (13.4)	73.1 (14.0)	.005
Race (black)	45 (22.1%)	236 (19.0%)	16 (20.2%)	216 (27.9%)	.15
Gender (female)	100 (49.0%)	755 (60.8%)	33 (41.8%)	353 (45.6%)	.51
HTN	132 (64.7%)	953 (76.7%)	72 (91.1%)	696 (89.9%)	.73
DM	49 (24.0%)	361 (29.1%)	33 (41.8%)	332 (42.9%)	.85
Smoking (current)	77 (37.8%)	352 (28.3%)	24 (30.4%)	176 (22.7%)	.13
ICH	26 (12.8%)	155 (12.5%)	3 (3.8%)	74 (9.6%)	.10

Data presented as mean (standard deviation) or n (%)

**Table 3**

Characteristics of Patients with Unsuspected Kidney Disease\* within the GCNK Stroke Study.

	Stroke patients with no history of kidney disease at baseline (n=1915 <sup>***</sup> )		p-value	Adjusted odds ratio (95% CI)
	Serum Creatinine < 1.2 (n=1399)	Serum Creatinine 1.2 (n=516)		
Age	67.3 (15.3)	72.2 (14.2)	<.0001	1.20 <sup>**</sup> (1.15, 1.26)
Race (black)	268 (19.2%)	130 (25.2%)	.004	1.82 (1.40, 2.37)
Gender (female)	847 (60.5%)	204 (39.5%)	<.0001	0.32 (0.26, 0.41)
HTN	1045 (74.7%)	444 (86.0%)	<.0001	1.70 (1.26, 2.29)
DM	391 (28.0%)	177 (34.3%)	.007	1.23 (0.98, 1.56)
Smoking (current)	410 (29.3%)	142 (27.5%)	.44	1.19 (0.91, 1.56)
ICH	176 (12.6%)	49 (9.5%)	.06	0.70 (0.49, 1.00)

\* Unsuspected renal insufficiency was defined as a patient who had no prior history of renal insufficiency, but who had a creatinine 1.2 on initial serum testing on presentation for acute stroke.

Data presented as mean (standard deviation) or n (%).

<sup>\*\*</sup> per 5-year increment

<sup>\*\*\*</sup> 47 patients with no baseline serum creatinine were excluded from this analysis.

**Table 4**

Kidney Disease Related Outcomes in Patients with Normal Renal Function, Known Chronic Kidney Disease and Unsuspected Renal Insufficiency Stratified by IV dye administration

	Normal renal function at baseline (n=1446)			Baseline kidney disease* (n=853)			Unsuspected renal insufficiency (n=516 of the 853)		
	Received IV dye N = 204	No IV dye N = 1242	p-value **	Received IV dye N = 79	No IV dye N = 774	p-value **	Received IV dye N = 53	No IV dye N = 463	p-value **
Acute kidney injury	3 (1.5%)	11 (0.9%)	0.43						
Worsening kidney disease				1 (1.3%)	14 (1.8%)	1.0	1 (1.9%)	4 (0.9%)	0.42

\* Defined as either elevated Cr upon arrival or history of chronic kidney disease

\*\* Fisher's exact test