NEFROPATIA DA MEZZO DI CONTRASTO: ANCORA UNA VECCHIA NEMICA?

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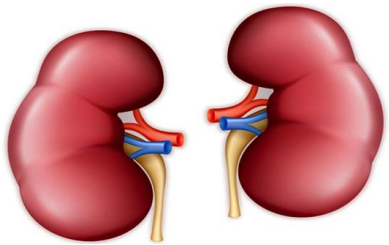
Chi di voi non ha mai discusso con un radiologo per eseguire una Angio-TC con MDC?

" ***Contrast-associated acute kidney injury*** *signifies a severe and usually reversible decline in kidney function that may develop within 72 hours after intravascular administration of iodinated contrast material ."*

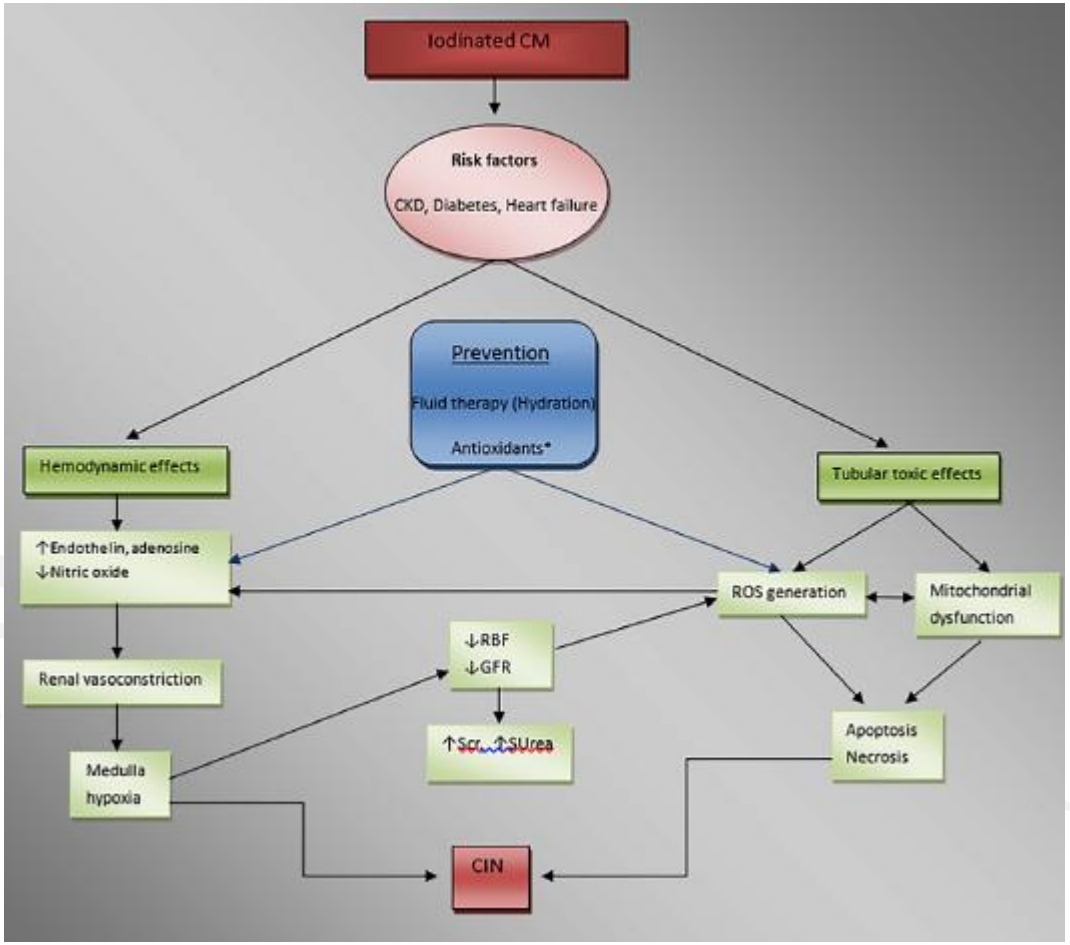
Disease state defined	Author or title of definition	Definition criteria
AKI	KDIGO[1]	Stage 1: Increase in SCr ≥ 0.3 mg/dL within 48 h OR increase in SCr by 1.5-2 times baseline within 7 d OR UOP < 0.5 mL kg^{-1} h^{-1} for 6-12 h Stage 2: Increase in SCr by 2.0-2.9 times baseline OR UOP < 0.5 mL kg^{-1} h^{-1} for ≥12 h Stage 3: Increase in SCr by ≥3 times baseline OR increase in SCr to ≥4.0 mg/dL (given they meet changes in SCr criteria of stage 1 AKI) OR initiation of renal replacement therapy OR UOP < 0.3 mL kg^{-1} h^{-1} for ≥24 h OR anuria for ≥12 h
	RIFLE[7]	Risk: Increase in SCr by 1.5 times baseline within 7 d and sustained for more than 24 h OR eGFR decrease >25% within 7 d and sustained for >24 h OR UOP < 0.5 mL kg^{-1} h^{-1} for > 6 h Injury: Increase in SCr by 2 times baseline OR eGFR decreased by >50% OR UOP < 0.5 mL kg^{-1} h^{-1} for >12 h Failure: Increase in Scr by 3 times baseline OR Increase in SCr >4 mg/dL with an acute rise >0.5 mg/dL OR eGFR decrease by >75% OR UOP < 0.3 mL kg^{-1} h^{-1} for >24 h OR Anuria for > 12 h Loss: Persistent acute renal failure (complete loss of kidney function >4 wk) End-stage kidney disease: ESRD >3 mo
	AKIN[8]	Stage 1: Increase in SCr ≥ 0.3 mg/dL within 48 h OR Increase in SCr by 1.5-2 times OR UOP < 0.5 mL kg^{-1} h^{-1} for >6 h Stage 2: Increase in SCr by 2.0-3.0 times baseline OR UOP < 0.5 mL kg^{-1} h^{-1} for >12 h Stage 3: Increase in SCr by >3.0 times baseline OR Increase in SCr ≥ 4.0 (with acute increase of ≥0.5 mg/dL) OR Initiation of renal replacement therapy OR UOP < 0.3 mL kg^{-1} h^{-1} for >24 h OR Anuria for >12 h
CI-AKI	Slocum et al[12] Harjai et al[11]	An absolute increase in SCr ≥ 0.5 mg/dL Increase in SCr by >0.5 mg/dL OR Increase in SCr by >25% from baseline



Rudnick M et al. Clin J Am Soc Nephrol 2008.

Pathophysiology of CIN has centered on its multifactorial pathogenesis, which includes:

- renal vasoconstriction
- tissue hypoxia
- direct cytotoxicity
- increased oxidative stress
- increased blood viscosity



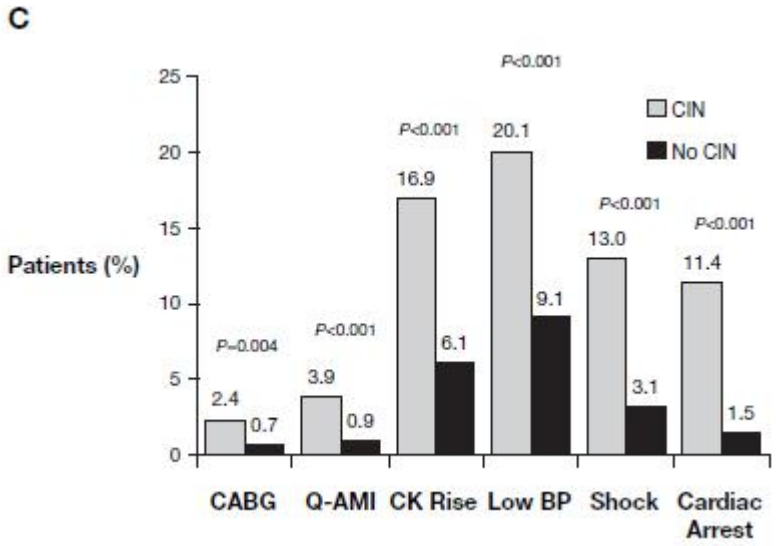
Mamoulakis C et al. Pharmacology & Therapeutics 2017.

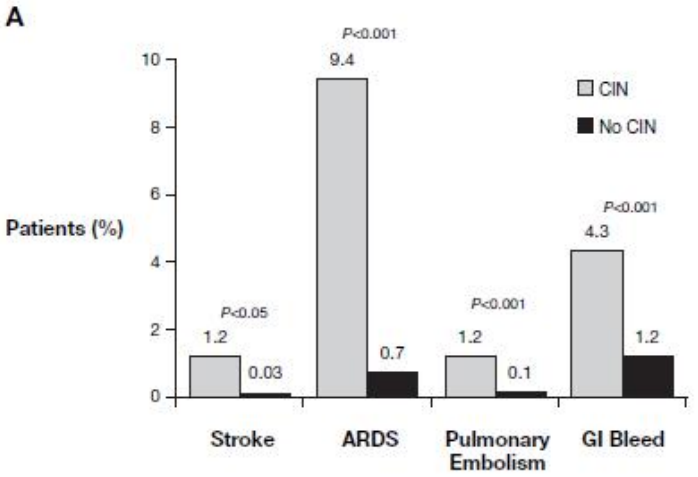
PROCEDURES RELATED TO CI-AKI

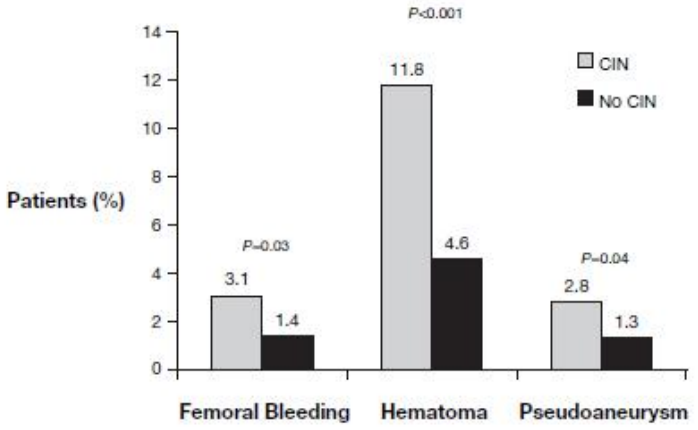
	CIN	Non-CIN	Incidence
Intravenous contrast-enhanced CT	734	4600	13.8%
Percutaneous coronary intervention	195	1464	11.8%
CT angiography	77	408	15.9%
Noncoronary angiography	32	176	15.4%
Other CM procedures	135	979	12.1%
Total	1173	7627	13.3%

Yin W et al. J Am Heart Assoc 2017.

Procedural complications in patients with and without CIN from an analysis of data from 7586 patients in the Mayo Clinic PCI Registry







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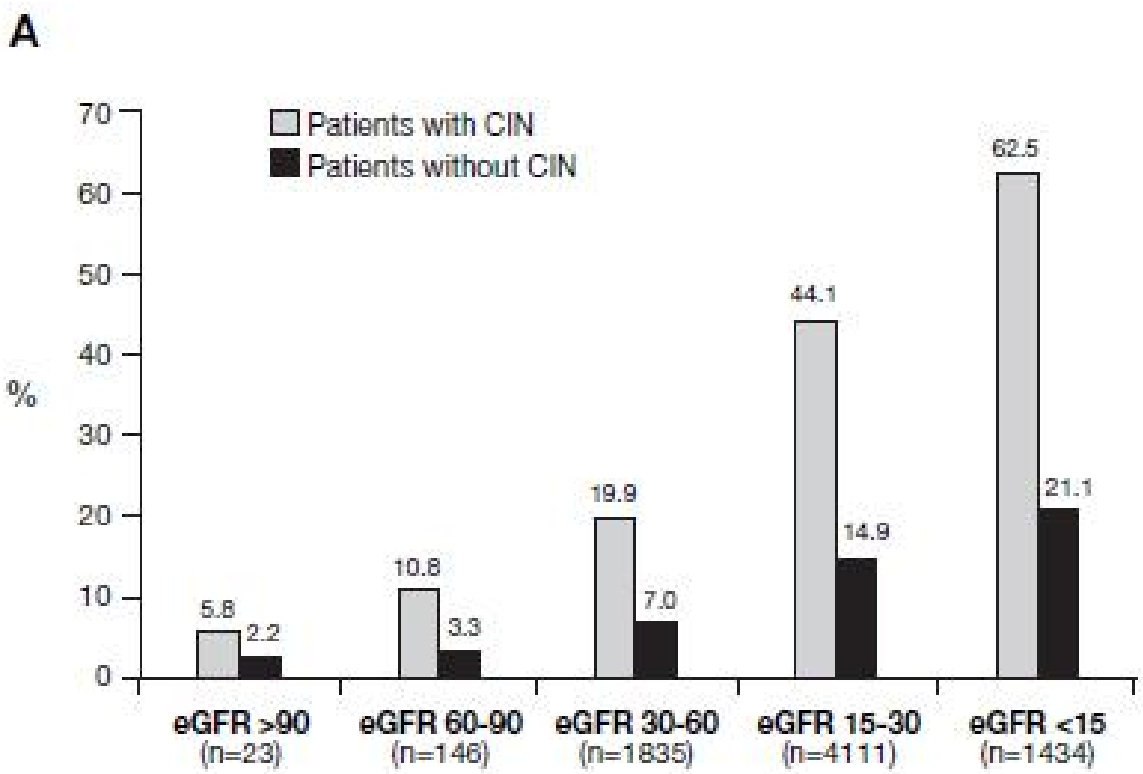
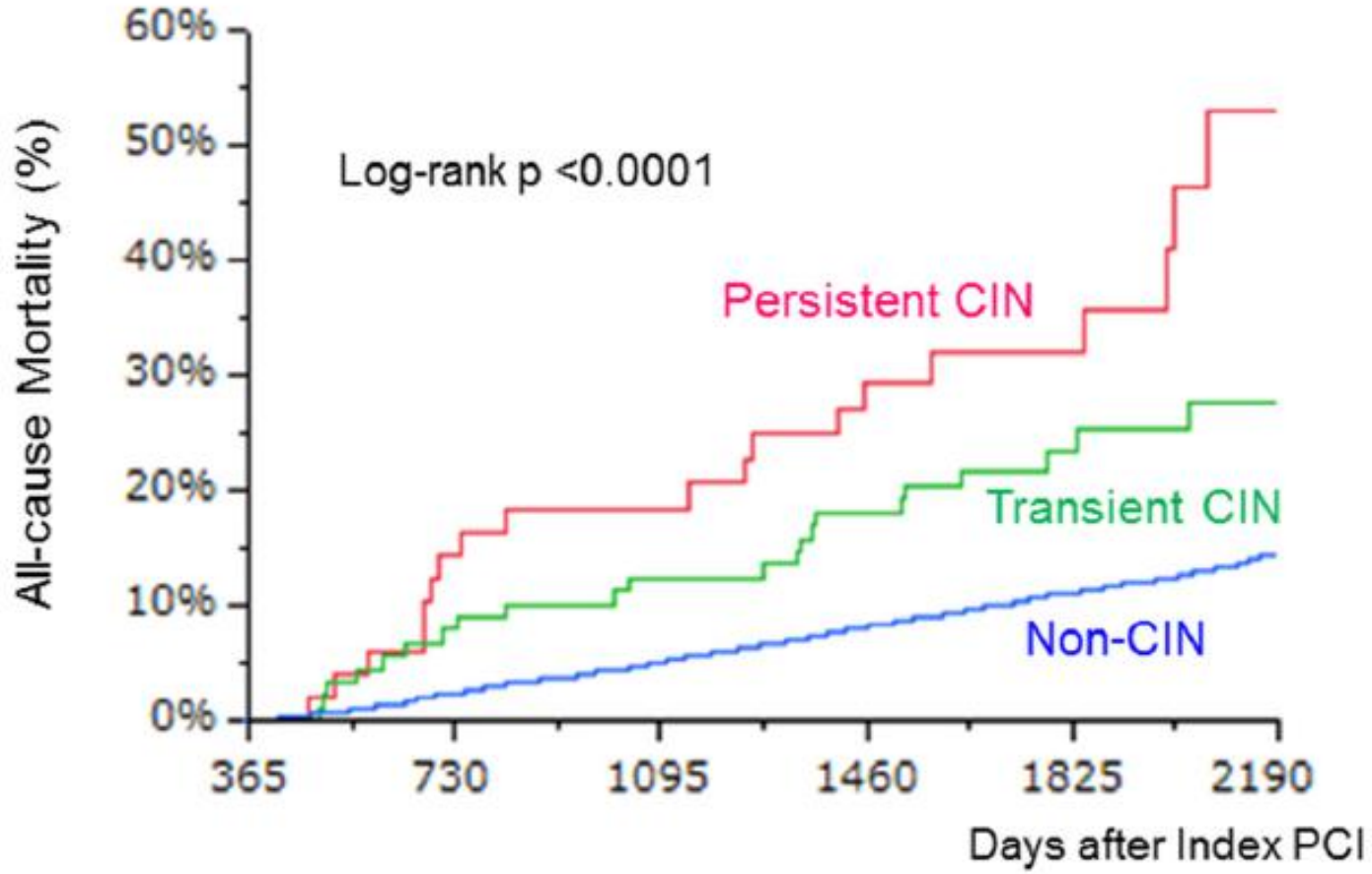
Rihal CS et al. Circulation 2002.

One-year mortality stratified by baseline estimated GFR in patients with or without CIN



Dangas G et al. Am J Cardiol 2005.



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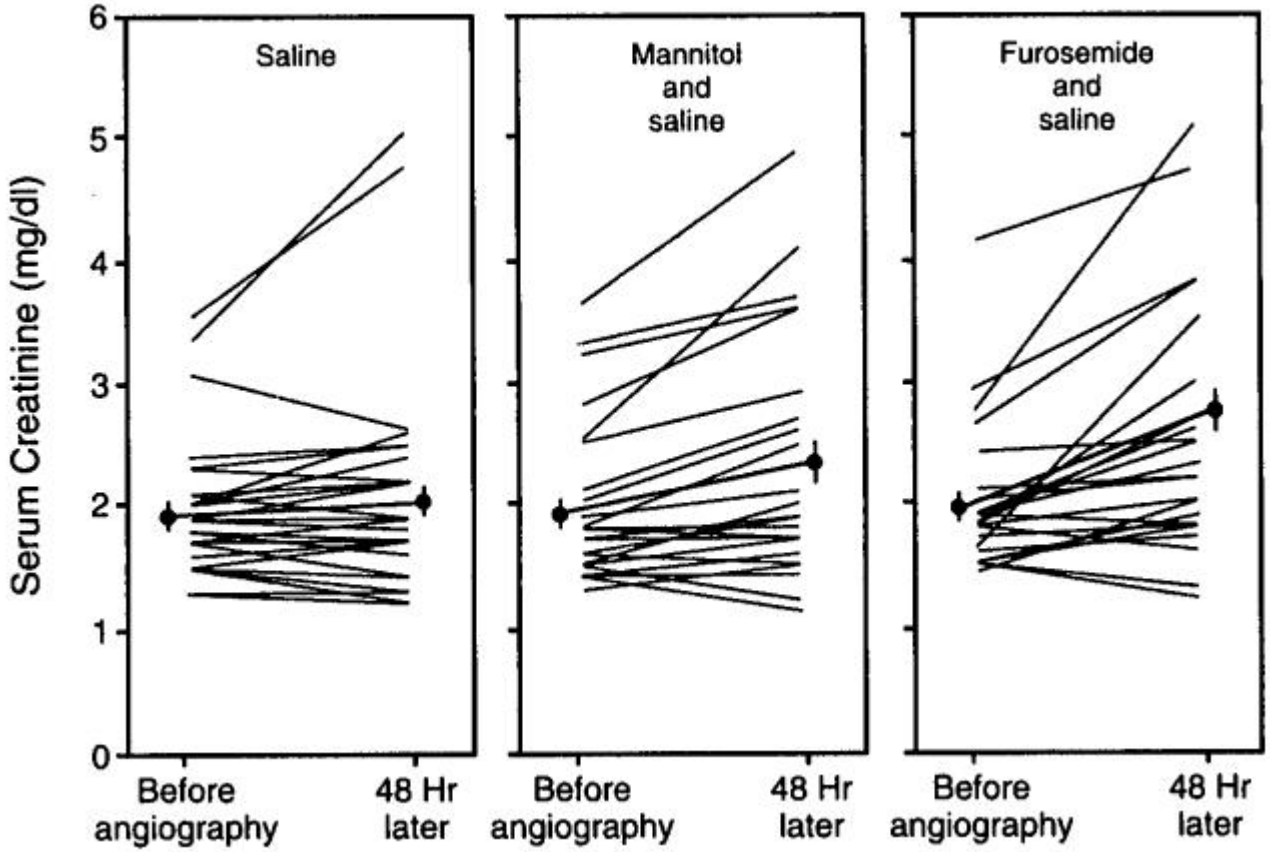
Abe M et al. Am J Cardiol 2017.

Various preventive strategies have been developed, with recent interest centering on the peri-procedural administration of intravenous sodium bicarbonate or oral acetylcysteine rather than on intravenous saline hydration, which has been the standard of care since the 1990s.

EFFECTS OF SALINE, MANNITOL, AND FUROSEMIDE ON ACUTE DECREASES IN RENAL FUNCTION INDUCED BY RADIOCONTRAST AGENTS

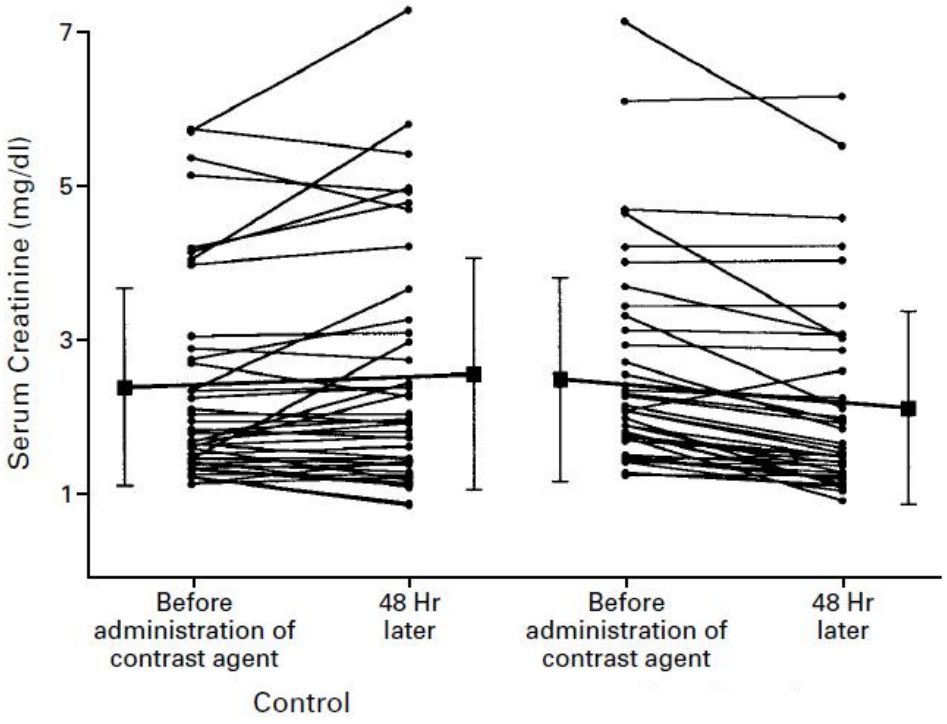
RICHARD SOLOMON, M.D., CRAIG WERNER, M.D., DENISE MANN, R.N., JOHN D'ELIA, M.D., AND PATRICIO SILVA, M.D.

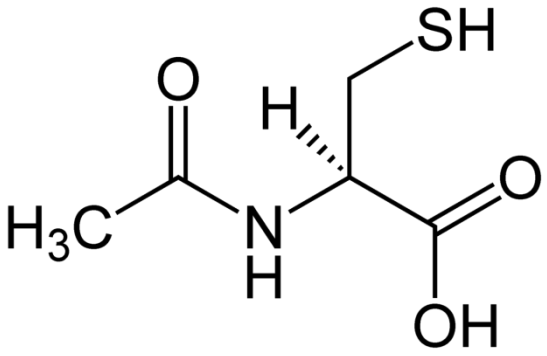


Variable	P Value	Saline (N = 28)	Mannitol and Saline (N = 25)	P Value	Furosemide and Saline (N = 25)	P Value
Change in serum creatinine — mg/dl						
24 Hr after radiocontrast agent	0.003†	0.0±0.2	0.2±0.2	0.01‡	0.3±0.4	0.002‡
48 Hr after radiocontrast agent	0.021†	0.1±0.5	0.3±0.4	0.10‡	0.5±0.6	0.01‡
Incidence of acute renal dysfunction — no. of patients (%)	0.05§	3 (11)	7 (28)	0.16¶	10 (40)	0.02¶

In patients with chronic renal insufficiency who are undergoing cardiac angiography, hydration with 0.45 percent saline provides better protection against acute decreases in renal function induced by radiocontrast agents than does hydration with 0.45 percent saline plus mannitol or furosemide.

ACETILCISTEINA





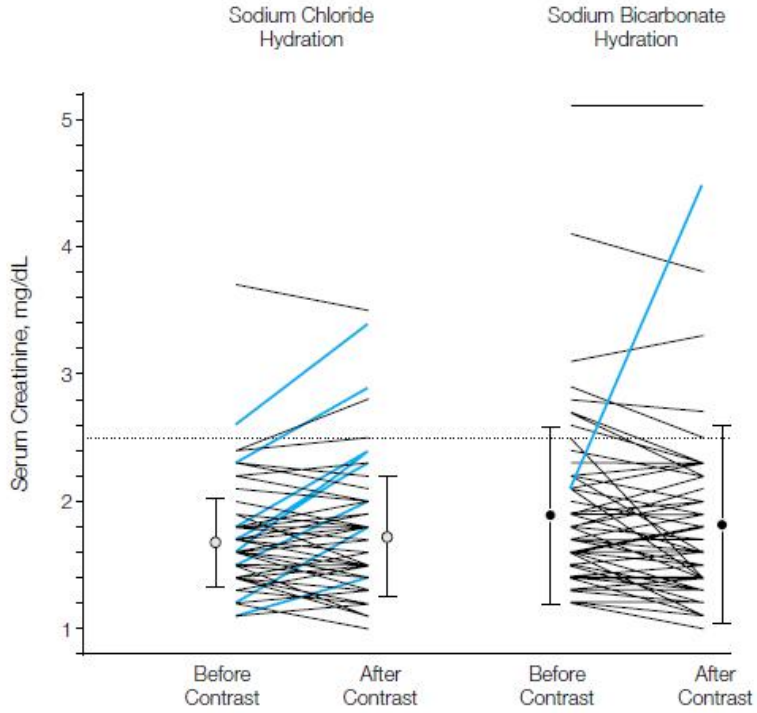
VARIABLE	ACETYL-CYSTEINE GROUP (N=41)	CONTROL GROUP (N=42)	P VALUE
Serum creatinine concentration — mg/dl			
Base line	2.5±1.3	2.4±1.3	0.55
Change 48 hr after administration of contrast agent	−0.4±0.4	+0.2±0.6	<0.001†
Incidence of acute reductions in renal function — no. (%)	1 (2)	9 (21)	0.01‡

Marenzi G et al. N Engl J Med 2006.

Tepel M et al. N Engl J Med. 2000.

Prevention of Contrast-Induced Nephropathy With Sodium Bicarbonate

A Randomized Controlled Trial

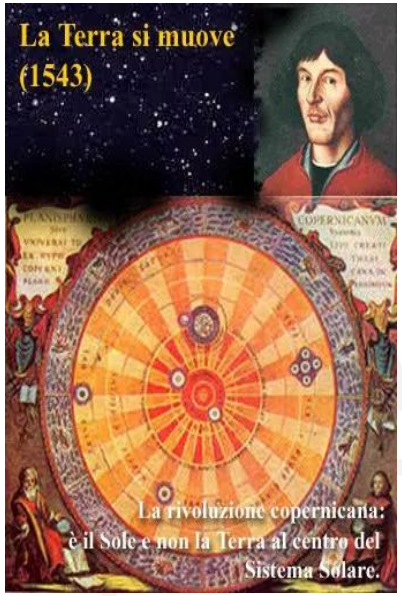


Hydration with sodium bicarbonate before contrast exposure is more effective than hydration with sodium chloride for prophylaxis of contrast-induced renal failure.

Merten GJ et al. JAMA 2004.

The NEW ENGLAND JOURNAL of MEDICINE

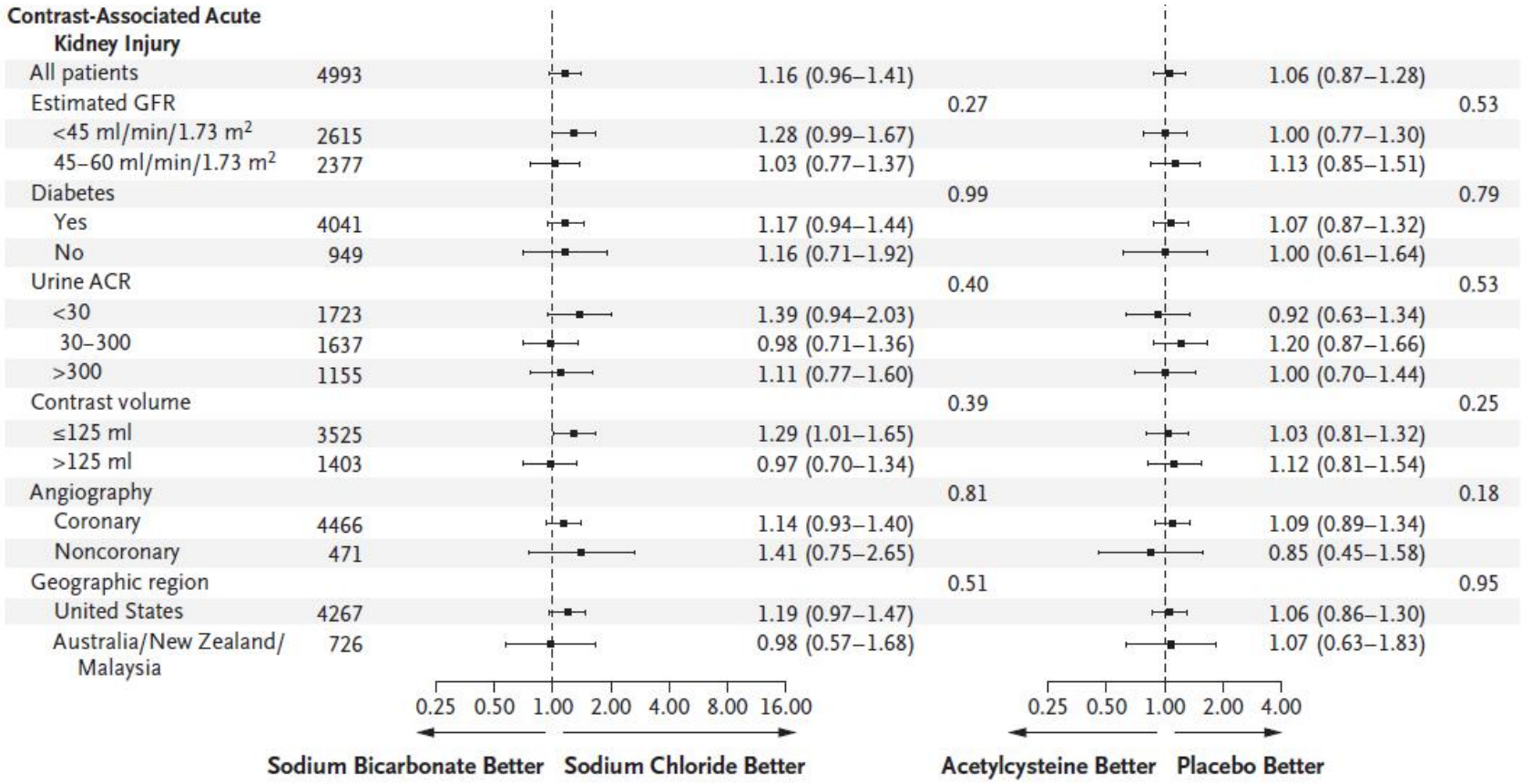
ESTABLISHED IN 1812 FEBRUARY 15, 2018 VOL. 378 NO. 7



Outcomes after Angiography with Sodium Bicarbonate and Acetylcysteine

Table 2. Serious Adverse Events, with the Exclusion of Kidney-Related Events.*

Type of Event	Sodium Bicarbonate (N=2511)	Sodium Chloride (N=2482)	Acetylcysteine (N=2495)	Placebo (N=2498)
	number of patients with event (percent)			
Cardiac	961 (38.3)	955 (38.5)	951 (38.1)	965 (38.6)
Heart failure	201 (8.0)	166 (6.7)	170 (6.8)	193 (7.7)
Arrhythmia	97 (3.9)	114 (4.6)	103 (4.1)	108 (4.3)
Coronary event	477 (19.0)	496 (20.0)	498 (20.0)	475 (19.0)
Gastrointestinal	81 (3.2)	60 (2.4)	81 (3.2)	60 (2.4)
Infectious	183 (7.3)	205 (8.3)	175 (7.0)	213 (8.5)
Neurologic	73 (2.9)	77 (3.1)	77 (3.1)	73 (2.9)
Pulmonary	116 (4.6)	126 (5.1)	119 (4.8)	123 (4.9)
Vascular	130 (5.2)	120 (4.8)	118 (4.7)	132 (5.3)



Among patients at high risk for renal complications who were undergoing angiography, there was no benefit of intravenous sodium bicarbonate over intravenous sodium chloride or of oral acetylcysteine over placebo for the prevention of death, need for dialysis, or persistent decline in kidney function at 90 days or for the prevention of contrast-associated acute kidney injury.

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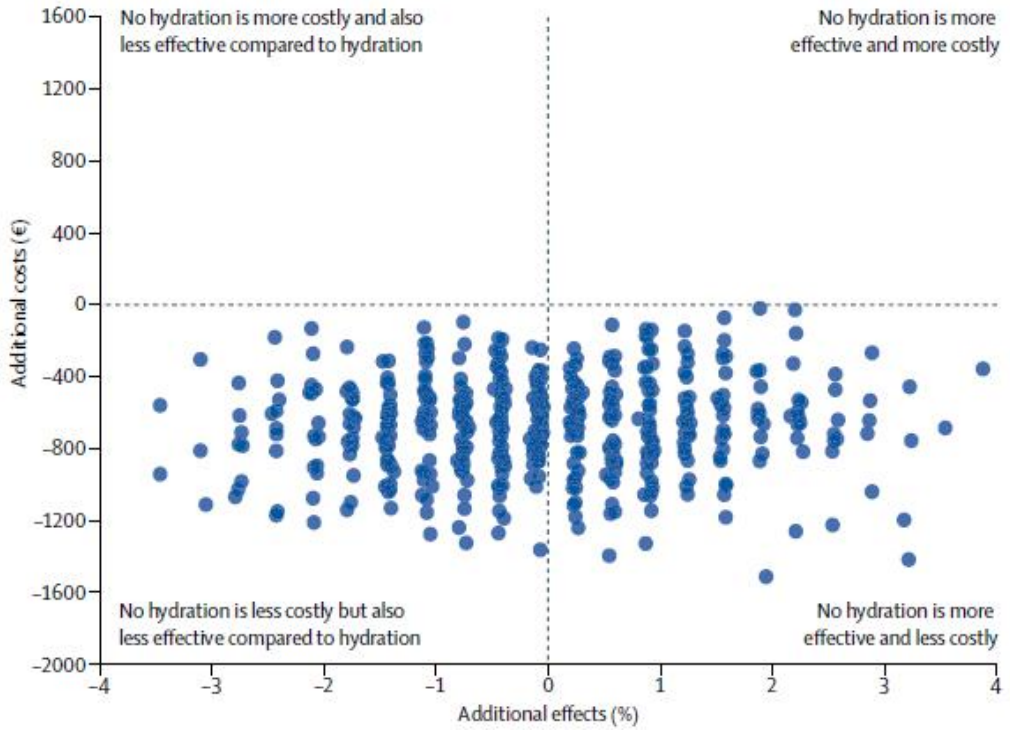
Prophylactic hydration to protect renal function from intravascular iodinated contrast material in patients at high risk of contrast-induced nephropathy (AMACING): a prospective, randomised, phase 3, controlled, open-label, non-inferiority trial

	H+ group	H– group	Absolute difference: H–group minus H+ group (95% CI)	p value
Renal events within 26–35 days post-contrast				
Renal failure (eGFR <15 mL per min/1·73 m²)	0	0	0	1·0000
>10 eGFR unit renal function decline from baseline	7/260 (2·7%)	11/260 (4·2%)	1·5 (–1·60 to 4·68)	0·3512
Renal function decline to eGFR <30 mL per min/1·73 m²	7/260 (2·7%)	6/260 (2·3%)	–0·4 (–3·07 to 2·30)	0·7881
Both >10 eGFR unit decline from baseline and a decline to eGFR <30 mL per min/1·73 m²	2/260 (0·8%)	2/260 (0·8%)	0·0 (–1·50 to 1·50)	>0·9999
Mortality, dialysis, and intensive care admission within 35 days post-contrast				
All-cause mortality	0/328	3/332 (0.9%)	0·9 (–0·11 to 1·92)	0·1267
Dialysis	0/328	0/332	0	1·0000
Intensive care admission	0/328	0/332	0	1·0000
Sequelae of intravenous hydration in the standard prophylactic treatment group				
Symptomatic heart failure	13/328 (4·0%)	0/332	–4·0 (–6·08 to –1·85)	0·0001
Hypernatraemia	0/328	0/332	0	1·0000
Hyponatraemia	1/328 (0·3%)	0/332	–0·3 (–0·90 to 0·29)	0·4970
Arrhythmia	4/328 (1·2%)	0/332	–1·2 (–2·41 to –0·03)	0·0604

Nijssen EC et al. Lancet 2017.

The AMACING study found no prophylaxis to be non inferior to prophylactic intravenous hydration in the prevention of contrast-induced nephropathy, as well as cost-saving. Additionally, we noted that hydration by itself sometimes leads to complications.



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Nijssen EC et al. Lancet 2017.

Risk of Acute Kidney Injury After Intravenous Contrast Media Administration

Methods: This single-center retrospective cohort analysis was performed in a large, urban, academic emergency department with an average census of 62,179 visits per year; 17,934 ED visits for patients who underwent contrast-enhanced, unenhanced, or no CT during a 5-year period (2009 to 2014) were included. The intervention was CT scan with or without intravenous contrast administration. The primary outcome was incidence of acute kidney injury. Secondary outcomes included new chronic kidney disease, dialysis, and renal transplantation at 6 months. Logistic regression modeling and between-groups odds ratios with and without propensity-score matching were used to test for an independent association between contrast administration and primary and secondary outcomes. Treatment decisions, including administration of contrast and intravenous fluids, were examined.

Results: Rates of acute kidney injury were similar among all groups. Contrast administration was not associated with increased incidence of acute kidney injury (contrast-induced nephropathy criteria odds ratio=0.96, 95% confidence interval 0.85 to 1.08; and Acute Kidney Injury Network/Kidney Disease Improving Global Outcomes criteria odds ratio=1.00, 95% confidence interval 0.87 to 1.16). This was true in all subgroup analyses regardless of baseline renal function and whether comparisons were made directly or after propensity matching. Contrast administration was not associated with increased incidence of chronic kidney disease, dialysis, or renal transplant at 6 months. Clinicians were less likely to prescribe contrast to patients with decreased renal function and more likely to prescribe intravenous fluids if contrast was administered.

Conclusion: In the largest well-controlled study of acute kidney injury following contrast administration in the ED to date, intravenous contrast was not associated with an increased frequency of acute kidney injury. [Ann Emerg Med. 2017;69:577-586.]

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Annals *of* Emergency Medicine
An International Journal

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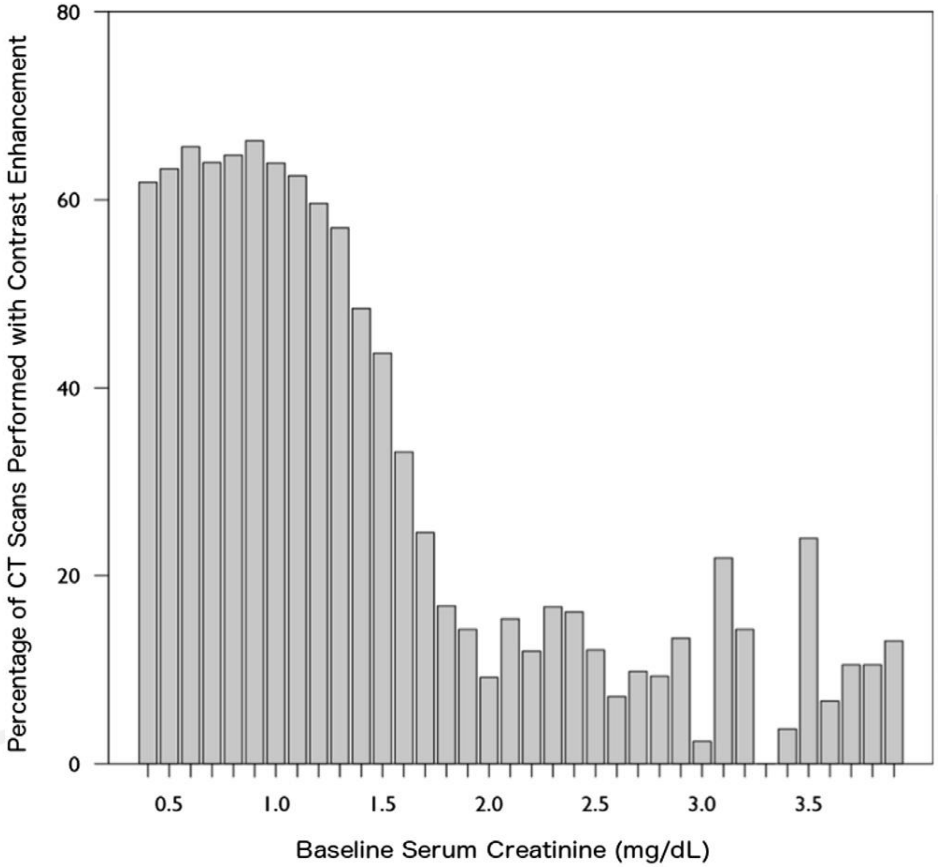
Intravenous contrast was not associated with CI-AKI!

Annals of Emergency Medicine
An International Journal



"Indeed, our findings, along with those of several other retrospective studies performed in other contexts, support the notion that randomization of patients to receive intravenous contrast, once considered ethically infeasible, is very likely safe (at least in patients with serum creatinine level <4.0 mg/dL) and will be necessary to fully understand the role of contrast media in precipitation of renal dysfunction."

Hinson JS. Ann Emerg Med 2017.

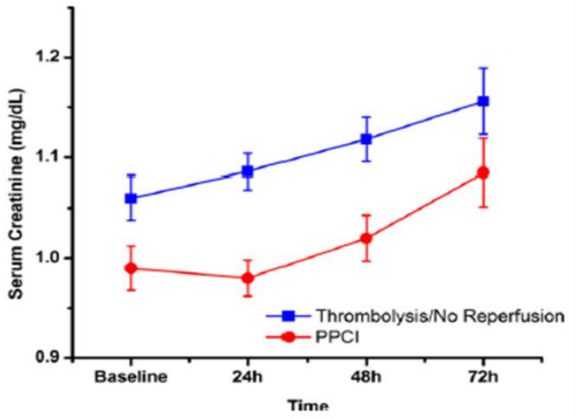
American Heart Association | American Stroke Association.

Acute Kidney Injury After Primary Angioplasty: Is Contrast-Induced Nephropathy the Culprit?

Oren Caspi, MD, PhD; Manhal Habib, MD, PhD; Yuval Cohen, MD; Arthur Kerner, MD; Ariel Roguin, MD, PhD; Eitan Abergel, MD; Monther Boulos, MD; Michael R. Kapeliovich, MD, PhD; Rafael Beyar, MD, DSc; Eugenia Nikolsky, MD, PhD; Doron Aronson, MD

Methods and Results—We studied 2025 patients with ST-segment–elevation myocardial infarction who underwent pPCI and 1025 patients receiving fibrinolysis or no reperfusion who were not exposed to contrast material during the first 72 hours of hospital stay (control group). AKI was defined as creatinine of ≥0.5 mg/dL or >25% rise within 72 hours. AKI rates were similar in the pPCI and control groups (10.3% versus 12.1%, respectively; *P*=0.38). Propensity score matching resulted in 931 matched pairs with PCI and no PCI, with balanced baseline covariates (standardized difference <0.1). Among propensity score–matched patients, AKI rates were not significantly different with and without PCI (8.6% versus 10.9%, *P*=0.12). In the pPCI cohort, independent predictors of AKI included age ≥70 years, insulin-treated diabetes mellitus, diuretic therapy, anterior infarction, baseline estimated glomerular filtration rate, and variables related to the presence of pump failure (higher Killip class, intra-aortic balloon pump use) and reduced left ventricular ejection fraction but not contrast material dose. A risk score based on the PCI cohort had similar discriminatory capacity for AKI in the control group (C statistic 0.81±0.02 and 0.78±0.02, respectively; *P*=0.26).

Conclusions—The development of AKI in patients with ST-segment–elevation myocardial infarction undergoing pPCI is mainly related to older age, baseline estimated glomerular filtration rate, heart failure, and hemodynamic instability. Risk for AKI is similar among ST-segment–elevation myocardial infarction patients with and without contrast material exposure. (***J Am Heart Assoc.* 2017;6:e005715. DOI: 10.1161/JAHA.117.005715.**)



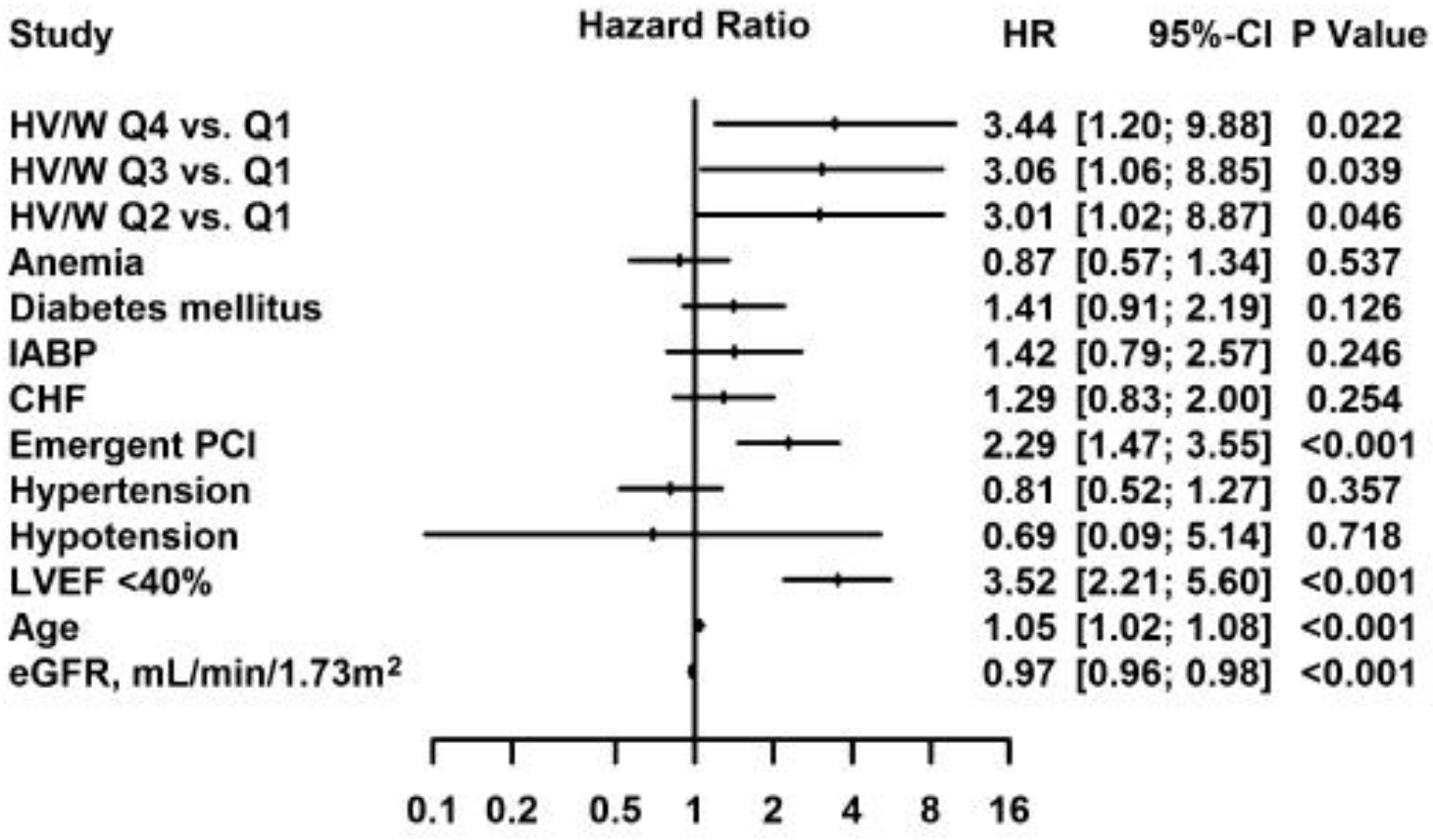
Excessively High Hydration Volume May Not Be Associated With Decreased Risk of Contrast-Induced Acute Kidney Injury After Percutaneous Coronary Intervention in Patients With Renal Insufficiency

Yong Liu, MD;* Hualong Li, MD;* Shiqun Chen, MS;* Jiyan Chen, MD, FACC, FESC; Ning Tan, MD, FACC, FESC; Yingling Zhou, MD; Yuanhui Liu, MD; Piao Ye, MD; Peng Ran, MD; Chongyang Duan, MS; Pingyan Chen, MS

***Background*—**No well-defined protocols currently exist regarding the optimal rate and duration of normal saline administration to prevent contrast-induced acute kidney injury (CI-AKI) in patients with renal insufficiency.

***Methods and Results*—**Hydration volume ratios (hydration volume/weight; HV/W) were calculated in 1406 patients with renal insufficiency (estimated glomerular filtration rate [eGFR], <90 mL/min per 1.73 m^2) undergoing percutaneous coronary intervention (PCI) with routine speed hydration (1 or 0.5 mL/kg per hour). We investigated the relationship between hydration volume, risk of CI-AKI (increase in serum creatinine ≥0.5 mg/dL or 25% within 48–72 hours), and prognosis. Mean follow-up duration was 2.85±0.88 years. Individuals with higher HV/W were more likely to develop CI-AKI (quartiles: Q1, Q2, Q3, and Q4: 4.3%, 6.6%, 10.9%, and 15.0%, respectively; $P<0.001$). After adjusting 12 confounders, including age, sex, eGFR, anemia, emergent PCI, diabetes mellitus, chronic heart failure, diuretics, contrast volume, lesions, smoking status, and number of stents, multivariate analysis showed that a higher HV/W ratio was not associated with a decreased CI-AKI risk (Q2 vs Q1: adjusted odds ratio [OR], 1.13; Q3 vs Q1: adjusted OR, 1.51; Q4 vs Q1: adjusted OR, 1.87; all $P>0.05$) and even increased CI-AKI risk (HV/W >25 mL/kg: adjusted OR, 2.11; 95% CI, 1.24–3.59; $P=0.006$). Additionally, higher HV/W was significantly associated with an increased risk of death (Q4 vs Q1: adjusted hazard ratio, 3.44; 95% CI, 1.20–9.88; $P=0.022$).

***Conclusions*—**Excessively high hydration volume at routine speed might be associated with increased risk of CI-AKI and death post-PCI in patients with renal insufficiency. **(*J Am Heart Assoc.* 2016;5:e003171 doi: 10.1161/JAHA.115.003171)**



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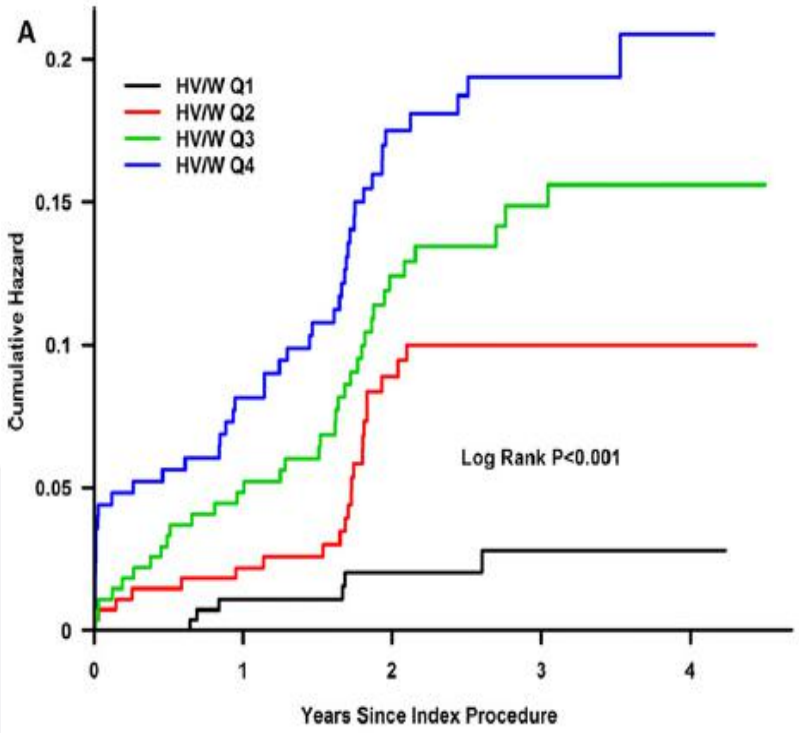
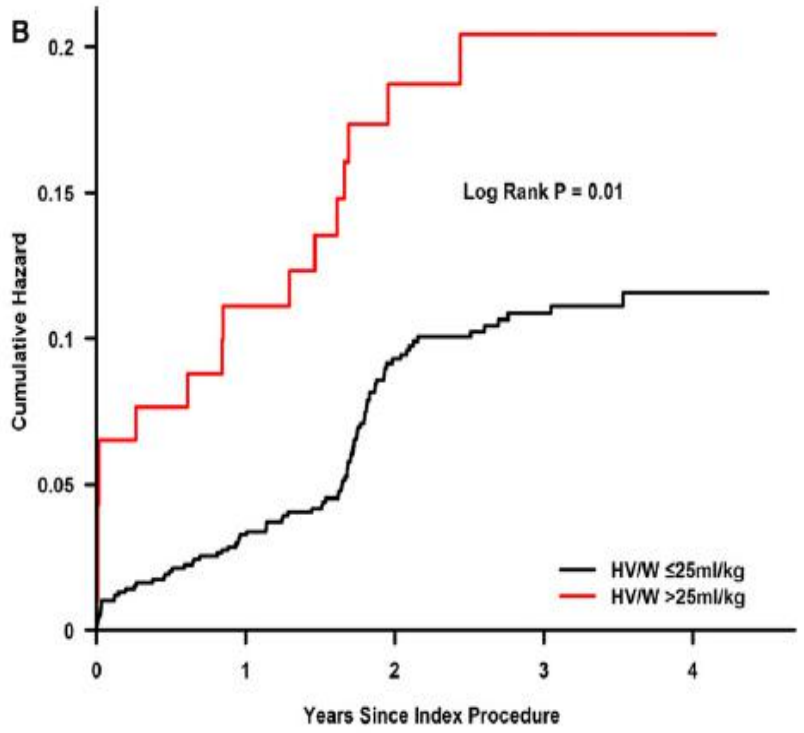
J Am Heart Assoc 2016.



Number of patients at risk				
HV/W Q1	279	277	211	130
HV/W Q2	277	272	189	177
HV/W Q3	276	264	198	140
HV/W Q4	256	236	195	150
Years Since Index Procedure	0	1	2	3

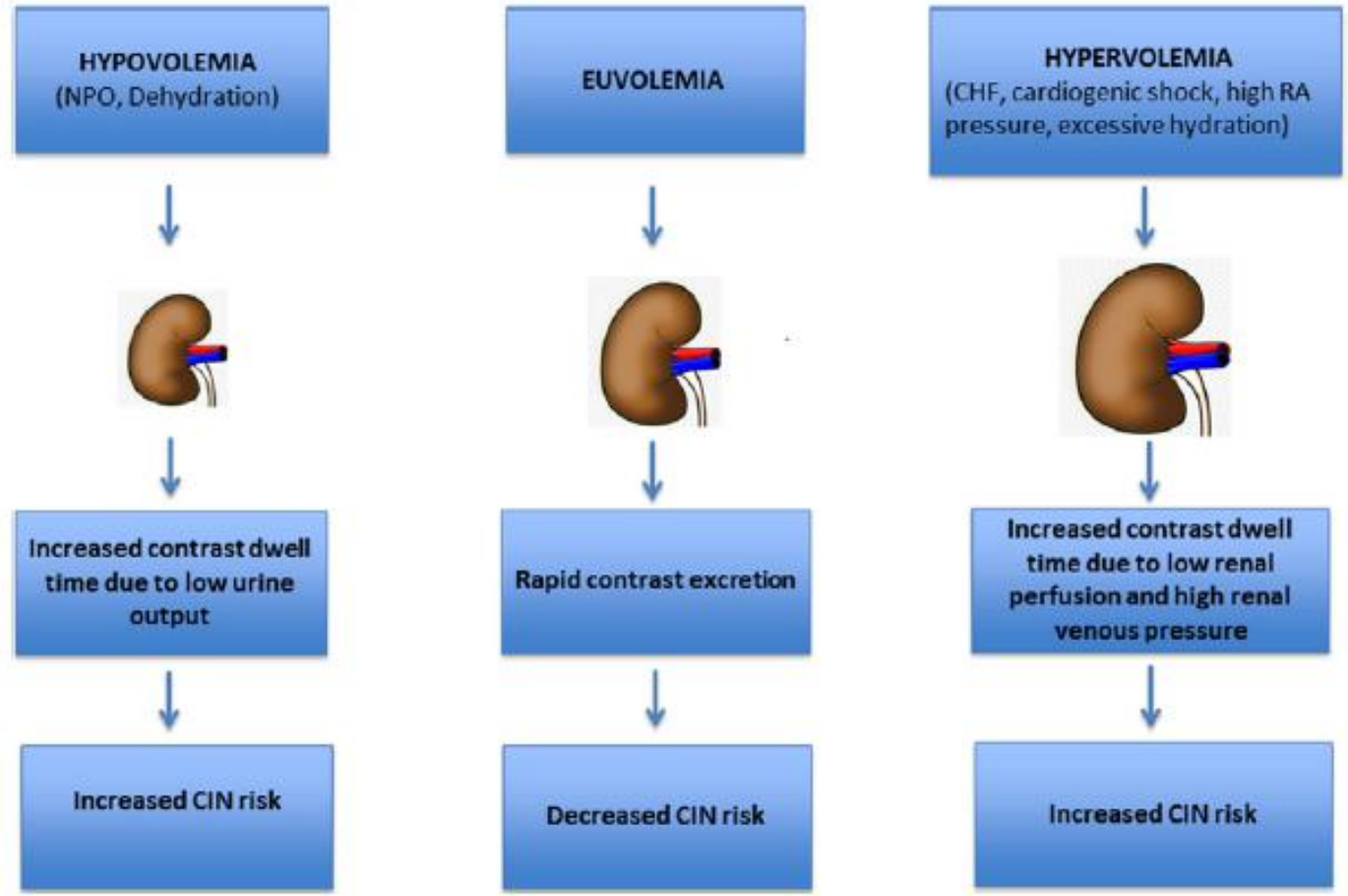


Number of patients at risk					
HV/W ≤25ml/kg	993	954	679	424	35
HV/W >25ml/kg	95	85	71	43	6
Years Since Index Procedure	0	1	2	3	4

The finding that high HV/W ratio is associated with CI-AKI may be related to an adverse effect of the excessive hydration itself or to residual confounding from the measured and unmeasured factors that were more frequently present in the group with high HV/W.

J Am Heart Assoc 2016.

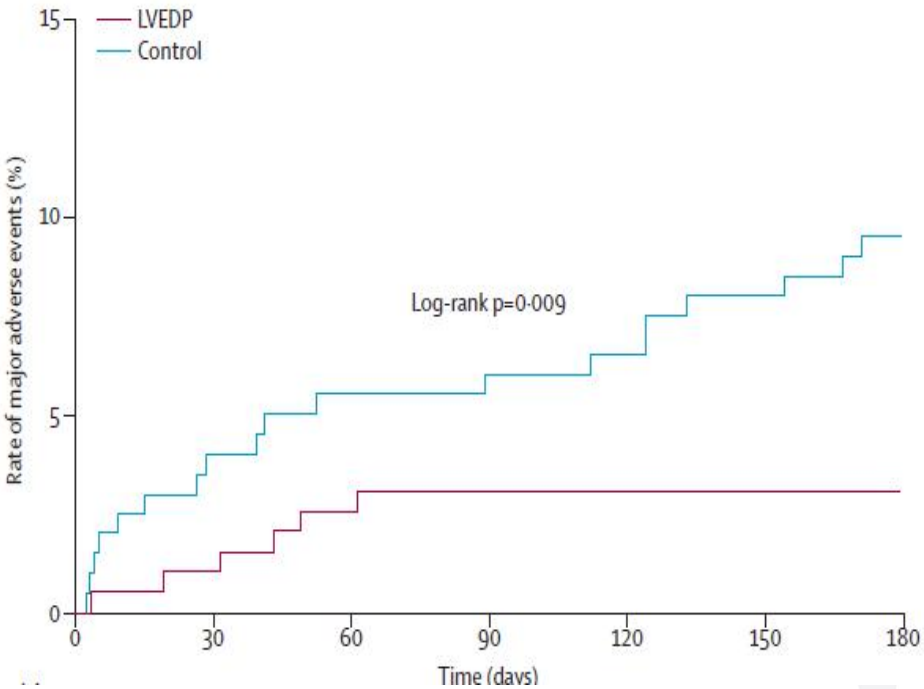
VOLUME STATUS AND RISK OF CI-AKI



J Am Heart Assoc. 2016

Haemodynamic-guided fluid administration for the prevention of contrast-induced acute kidney injury: the POSEIDON randomised controlled trial

Somjot S Brar, Vicken Aharonian, Prakash Mansukhani, Naing Moore, Albert Y-J Shen, Michael Jorgensen, Aman Dua, Lindsay Short, Kevin Kane



Left ventricular end-diastolic pressure-guided fluid administration seems to be safe and effective in preventing contrast-induced acute kidney injury in patients undergoing cardiac catheterisation.

Lancet 2014.

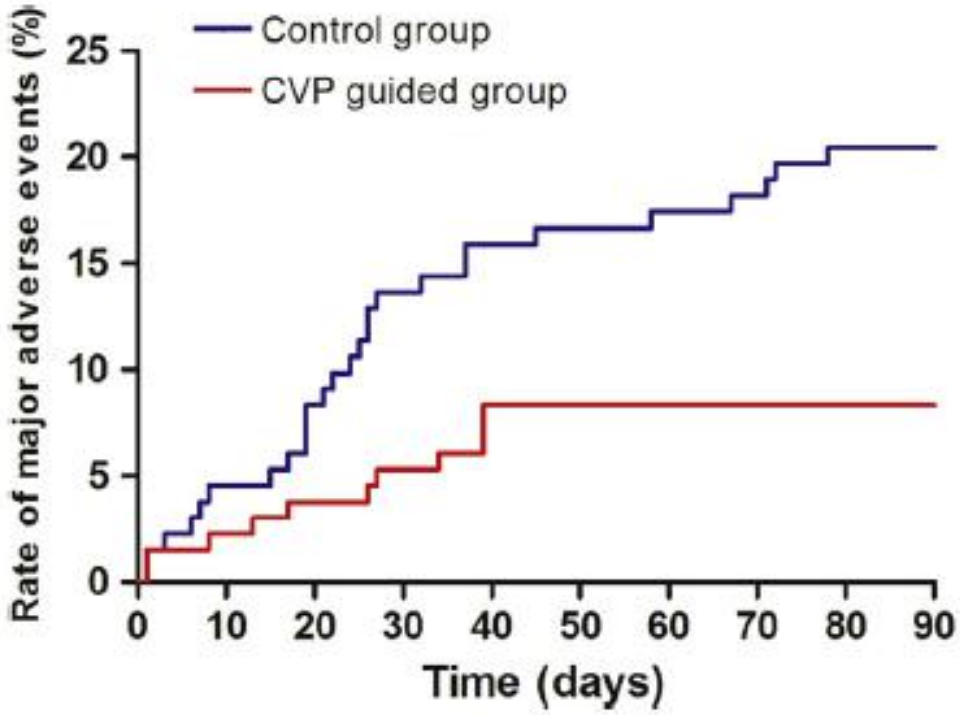
	LVEDP hydration-guided group	Control group	Relative risk (95% CI)	Risk difference (95% CI)	p value
Primary endpoint					
>25% or 0·5 mg/dL increase in serum creatinine	12/178 (6·7%)	28/172 (16·3%)	0·41 (0·22–0·79)	−9·5 (−2·9 to −16·2)	0·005
Secondary endpoints					
>25% increase in serum creatinine	12/178 (6·7%)	27/172 (15·7%)	0·43 (0·22–0·82)	−9·0 (−2·5 to −15·5)	0·008
>0·5 mg/dL increase in serum creatinine	5/178 (2·8%)	11/172 (6·4%)	0·44 (0·16–1·24)	−3·6 (−8·0 to 0·8)	0·11
Sensitivity analyses					
≥0·3 mg/dL increase in serum creatinine	24/178 (13·5%)	43/172 (25·0%)	0·54 (0·34–0·85)	−11·5 (−3·3 to −19·7)	0·006
>25% or 0·5 mg/dL increase in serum creatinine in participants with ≥1 serum creatinine value available	12/190 (6·3%)	28/196 (14·3%)	0·44 (0·23–0·84)	−8·0 (−2·0 to −14·0)	0·01

	LVEDP-guided group (n=196)	Control group (n=200)	Relative risk (95% CI)	Risk difference (95% CI)	p value
At 30 days					
All-cause mortality	0	3 (1·5%)	..	..	0·25
Myocardial infarction	1 (0·5%)	4 (2·0%)	..	..	0·37
Renal replacement therapy	1 (0·5%)	3 (1·5%)	..	..	0·62
Cumulative major adverse events	2 (1·0%)	8 (4·0%)	0·26 (0·05–1·19)	−3·0 (−6·0 to 0·1)	0·11
At 6 months					
All-cause mortality	1 (0·5%)	8 (4·0%)	..	..	0·037
Myocardial infarction	4 (2·0%)	13 (6·5%)	..	..	0·029
Renal replacement therapy	1 (0·5%)	4 (2·0%)	..	..	0·37
Cumulative major adverse events	6 (3·1%)	19 (9·5%)	0·32 (0·13–0·79)	−6·4 (−11·2 to −1·7)	0·008

Lancet 2014.

Prevention of Contrast-Induced Nephropathy by Central Venous Pressure–Guided Fluid Administration in Chronic Kidney Disease and Congestive Heart Failure Patients

Geng Qian, MD, Zhenhong Fu, MD, Jun Guo, MD, Feng Cao, MD, Yundai Chen, MD



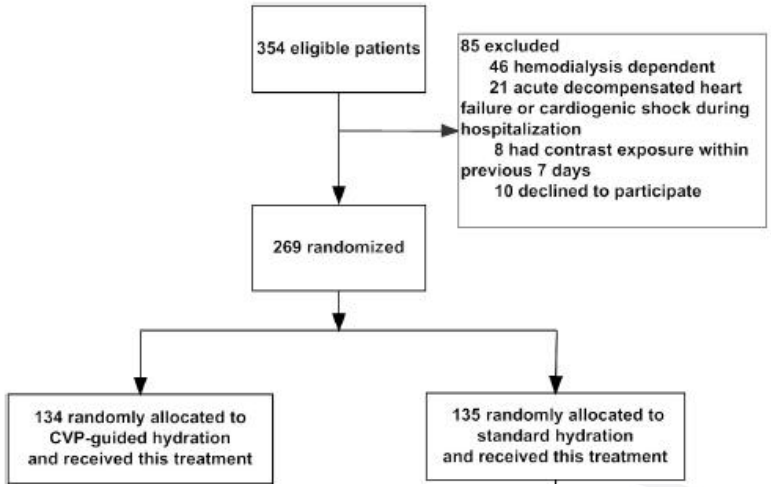


TABLE 2 Incidence of CIN in Study Patients

Definition of CIN	CVP-Guided Hydration Group (n = 132)	Control Group (n = 132)	Absolute Difference (95% CI)	p Value
SCr >50% ↑	5 (3.79)	13 (9.85)	3.1 (0.0-12.1)	0.042
SCr >25% ↑	19 (14.4)	32 (24.2)	9.8 (0.3-19.4)	0.030
SCr >0.5 mg/dl ↑	18 (13.6)	35 (26.5)	4.9 (3.3-22.5)	0.007
SCr >0.3 mg/dl ↑	26 (19.7)	46 (34.8)	15.2 (4.5-25.8)	0.004
Incidence of CIN	21 (15.9)	39 (29.5)	13.6 (3.6-23.7)	0.006

TABLE 4 Ninety-Day Main Adverse Events in Study Patients

	CVP-Guided Hydration Group (n = 132)	Control Group (n = 132)	Absolute Difference, % (95% CI)	p Value
All-cause mortality	3 (2.3)	5 (3.8)	1.5 (−2.7 to 5.7)	0.722
Myocardial infarction	4 (3.0)	13 (9.8)	6.8 (0.9 to 12.7)	0.019
Renal replacement therapy	4 (3.0)	13 (9.8)	6.8 (0.9 to 12.7)	0.019
Cumulative major adverse events	11 (8.3)	27 (20.5)	12.1 (3.7 to 20.5)	0.004

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J Am Coll Cardiol Intv 2016

EDITORIAL

American Heart Association | American Stroke Association

Intravenous Hydration and Contrast-Induced Acute Kidney Injury: Too Much of a Good Thing?

Rajesh Gupta, MD; Ankush Moza, MD; Christopher J. Cooper, MD

- *It is possible that the current findings are largely attributable to residual confounding because participants in Q4 for HV/W ratio had many baseline variables associated with CI-AKI risk.*
- *Too much hydration increases the risk of CI-AKI in some people; it is well known that congestive heart failure is associated with CI-AKI risk, and renal venous hypertension (or congestion) may contribute to the pathogenesis of AKI.*
- *It is not that intravenous fluid hydration is automatically good but rather that the right amount of fluid for each patient is needed to optimize outcomes and reduce the risk of CI-AKI.*

2. Risk Assessment for Contrast Induced Acute Kidney Injury (CI-AKI)

Guideline 2.1 – Risk Assessment for CI-AKI

We suggest that prior to any imaging using iodinated contrast media baseline kidney function and presence of other risk factors for CI-AKI should be identified. The exception to this is when the benefit of very early imaging outweighs the risk of delaying the procedure. (Not Graded)

Guideline 2.2 – Risk Assessment for CI-AKI

We suggest that estimated glomerular filtration rate (eGFR) should only be used to assess kidney function in stable outpatients. (Not Graded)

We suggest that serum creatinine is used to assess kidney function in acutely ill patients or patients with acute kidney injury. All such patients should be considered as at increased risk of CI-AKI. (Not Graded)

Guideline 2.4 – Risk Assessment for CI-AKI

We suggest that patients identified to be at high risk of CI-AKI may be discussed with a renal physician to assess whether the potential benefit from the iodinated constrast study outweighs the increased risk of CI-AKI. (Not Graded)

Future directions

1) Biomarkers

Use of Both Serum Cystatin C and Creatinine as Diagnostic Criteria for Contrast-Induced Acute Kidney Injury and Its Clinical Implications

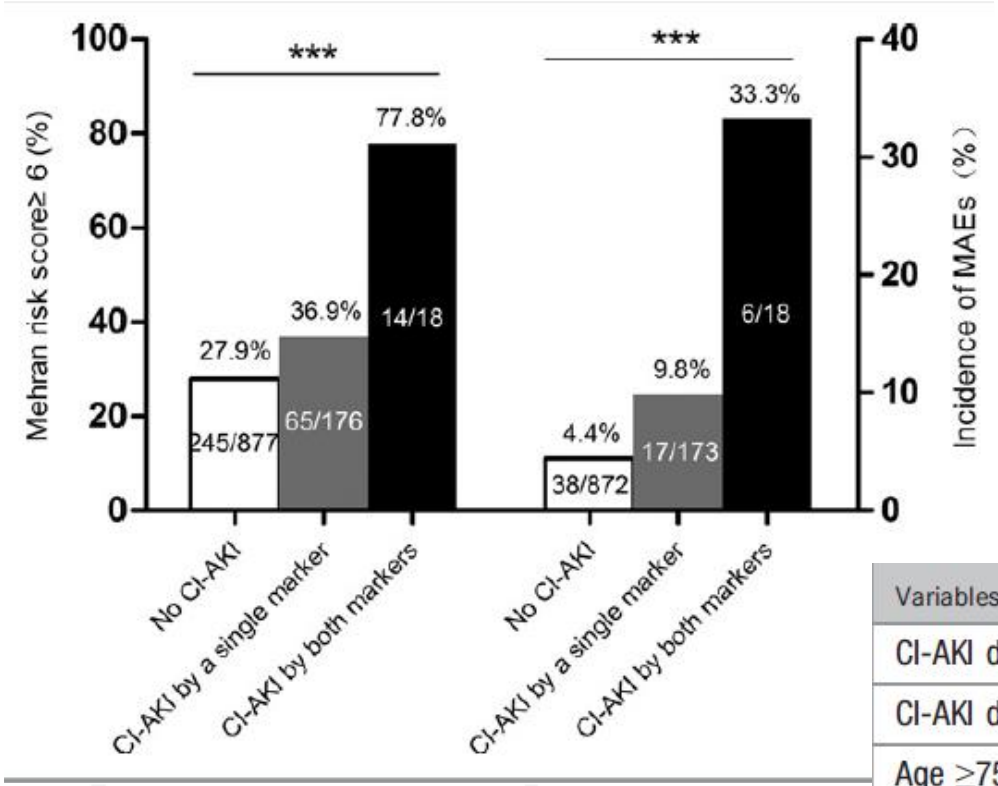
Wei-feng Zhang, MD;* Tuo Zhang, MD;* Ding Ding, MD; Shi-qun Sun, MD; Xiao-lei Wang, MD; Shi-chun Chu, MD; Ling-hong Shen, MD, PhD; Ben He, MD, PhD, FACC, FESC

Background—Contrast-induced acute kidney injury (CI-AKI) was traditionally defined as an increase in serum creatinine (sCr) after contrast media exposure. Recently, serum cystatin C (sCyC) has been proposed as an alternative to detect acute changes in renal function. The clinical implications of combining sCyC and sCr to diagnose CI-AKI remain unknown.

Methods and Results—One thousand seventy-one consecutive patients undergoing coronary angiography/intervention were prospectively enrolled. SCyC and sCr were assessed at baseline and 24 to 48 hours after contrast media exposure. CI-AKI determined by sCr (CI-AKI$_{sCr}$) was defined as an sCr increase greater than 0.3 mg/dL or 50% from baseline. Major adverse events at 12 months were assessed. CI-AKI$_{sCr}$ developed in 25 patients (2.3%). Twelve-month follow-up was available for 1063 patients; major adverse events occurred in 61 patients (5.7%). By receiver operating characteristic curve analysis, an sCyC increase of greater than 15% was the optimal cutoff for CI-AKI$_{sCr}$ detection, which occurred in 187 patients (17.4%). To evaluate the use of both sCyC and sCr as CI-AKI diagnostic criteria, we stratified patients into 3 groups: no CI-AKI, CI-AKI detected by a single marker, and CI-AKI detected by both markers. Multivariable logistic regression revealed that the predictability of major adverse events increased in a stepwise fashion in the 3 groups (no-CI-AKI group as the reference, CI-AKI detected by a single marker: odds ratio=2.25, 95% CI: 1.24–4.10, P<0.01; CI-AKI detected by both markers: odds ratio=10.00, 95% CI: 3.13–31.91, P<0.001).

Conclusions—Combining sCyC and sCr to diagnose CI-AKI would be beneficial for risk stratification and prognosis in patients after contrast media exposure. (***J Am Heart Assoc*. 2017;6:e004747. DOI: 10.1161/JAHA.116.004747.)**

Group	Definition	Risk Stratification
Group 1	No CI-AKI: sCr increase <0.3 mg/dL and 50% from baseline; and sCyC increase <15% from baseline.	No risk
Group 2	CI-AKI detected by a single marker: fulfill only 1 of criteria as below: (1) sCr increase ≥0.3 mg/dL or 50% from baseline; (2) sCyC increase ≥15% from baseline.	Potential risk
Group 3	CI-AKI detected by both markers: sCr increase ≥0.3 mg/dL or 50% from baseline; and sCyC increase ≥15% from baseline.	High risk



Variables	OR (95% CI)	*P* Value
CI-AKI detected by a single marker	2.25 (1.24–4.10)	<0.010
CI-AKI detected by both markers	10.00 (3.13–31.91)	<0.001
Age ≥75 years	0.54 (0.19–1.49)	0.234
Diabetes mellitus	0.94 (0.43–2.07)	0.887
Prior or new-onset MI	2.26 (1.37–3.73)	0.001
NYHA Grade III–IV	0.77 (0.16–3.11)	0.709
Baseline eGFR	1.00 (0.99–1.01)	0.909
Mehran risk score	1.03 (0.88–1.20)	0.730

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Zhang W et al. J Am Heart Assoc 2017.

Future directions

1) Biomarkers
2) Predicting scores

Ho and Harahsheh Journal of Intensive Care (2018) 6:3
DOI 10.1186/s40560-018-0274-z

Journal of Intensive Care

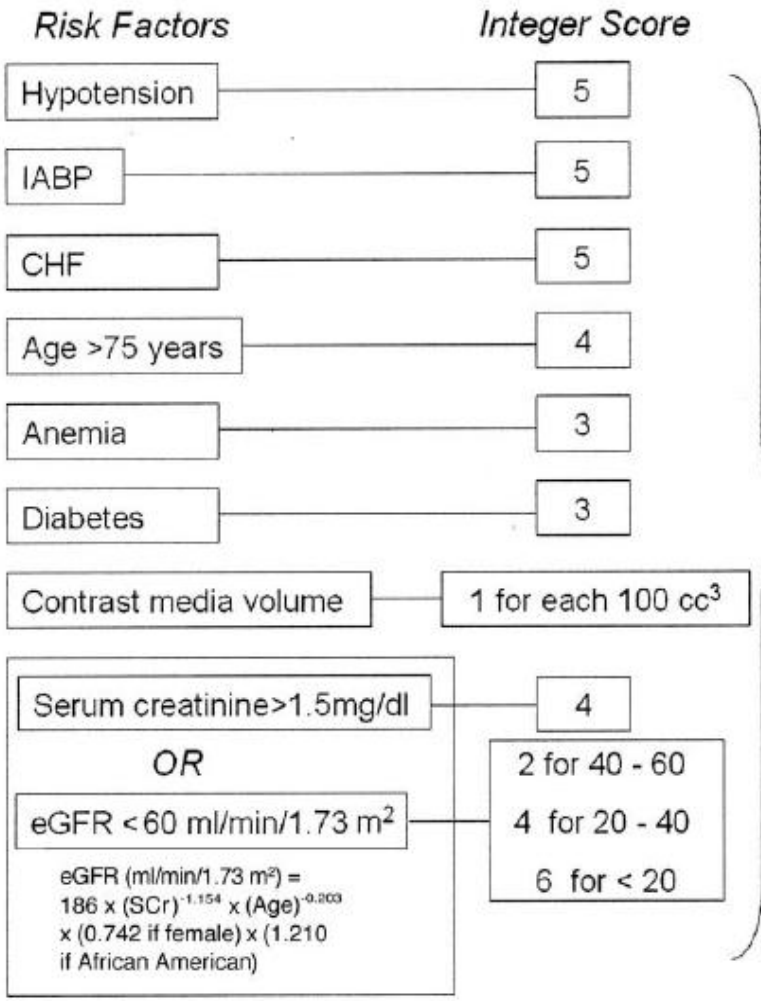
RESEARCH Open Access

Predicting contrast-induced nephropathy after CT pulmonary angiography in the critically ill: a retrospective cohort study

The decision to proceed with a CTPA to exclude a life-threatening acute PE in patients with multiple risk factors for CIN is difficult. Theoretically, a CTPA will be justifiable provided its benefits outweigh its harms. In practice, to balance the benefits and risks of a radiocontrast study for critically ill patients is challenging.

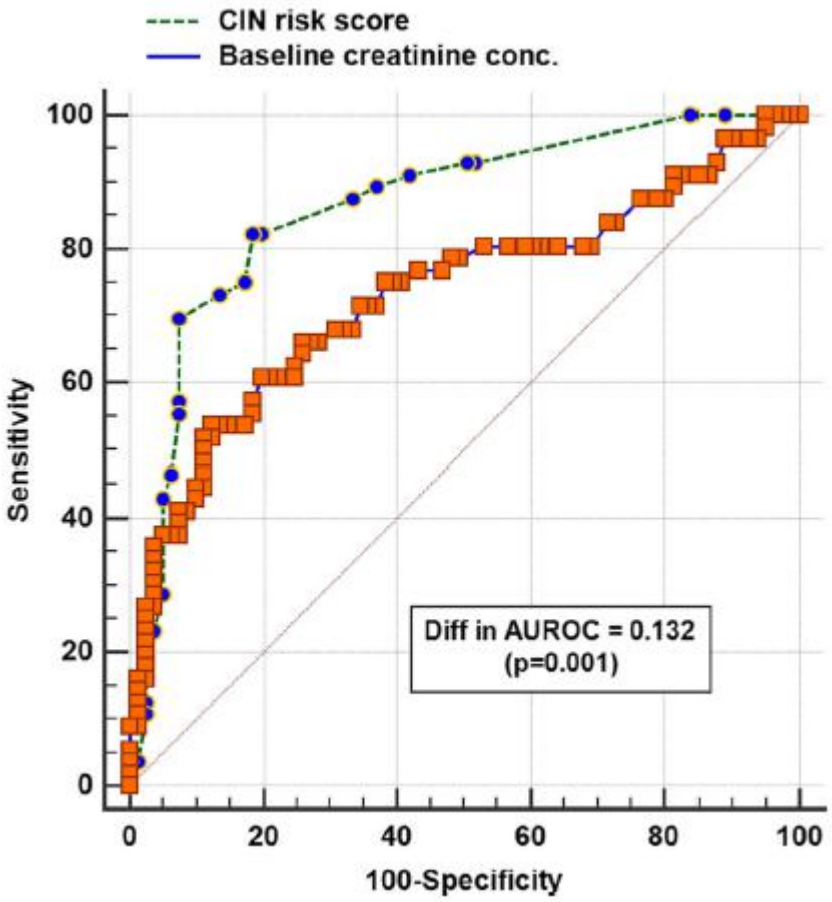
In critically ill patients presenting with symptoms and signs of a life-threatening PE, opportunities to use prophylactic measures against CIN, including aggressive intravenous hydration, are limited. Secondly, many risk factors for CIN, including heart failure, hypotension, and increasing age, are also risk factors for mortality in acute PE, suggesting that patients who are most at risk of dying from acute PE are, perhaps, also most at risk of developing CIN after a CTPA scan.

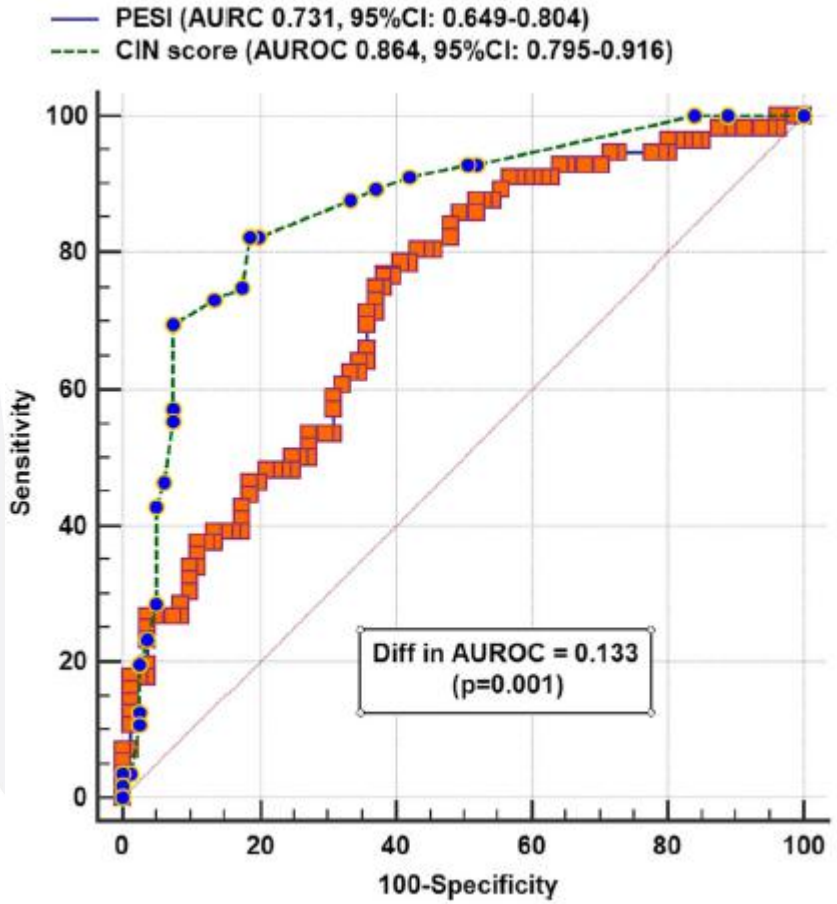
Mehran contrast nephropathy risk score



Risk Score	Risk of CIN	Risk of Dialysis
≤ 5	7.5%	0.04%
6 to 10	14.0%	0.12%
11 to 16	26.1%	1.09%
≥ 16	57.3%	12.6%

J Am Coll Cardiol 2004.





The CIN prediction score had a good ability to discriminate between critically ill patients with and without developing CIN after CTPA.
Used together for critically ill patients with suspected acute PE, the CIN prediction score and PESI may be useful to inform clinicians when the benefits of a CTPA scan will outweigh its potential harms.

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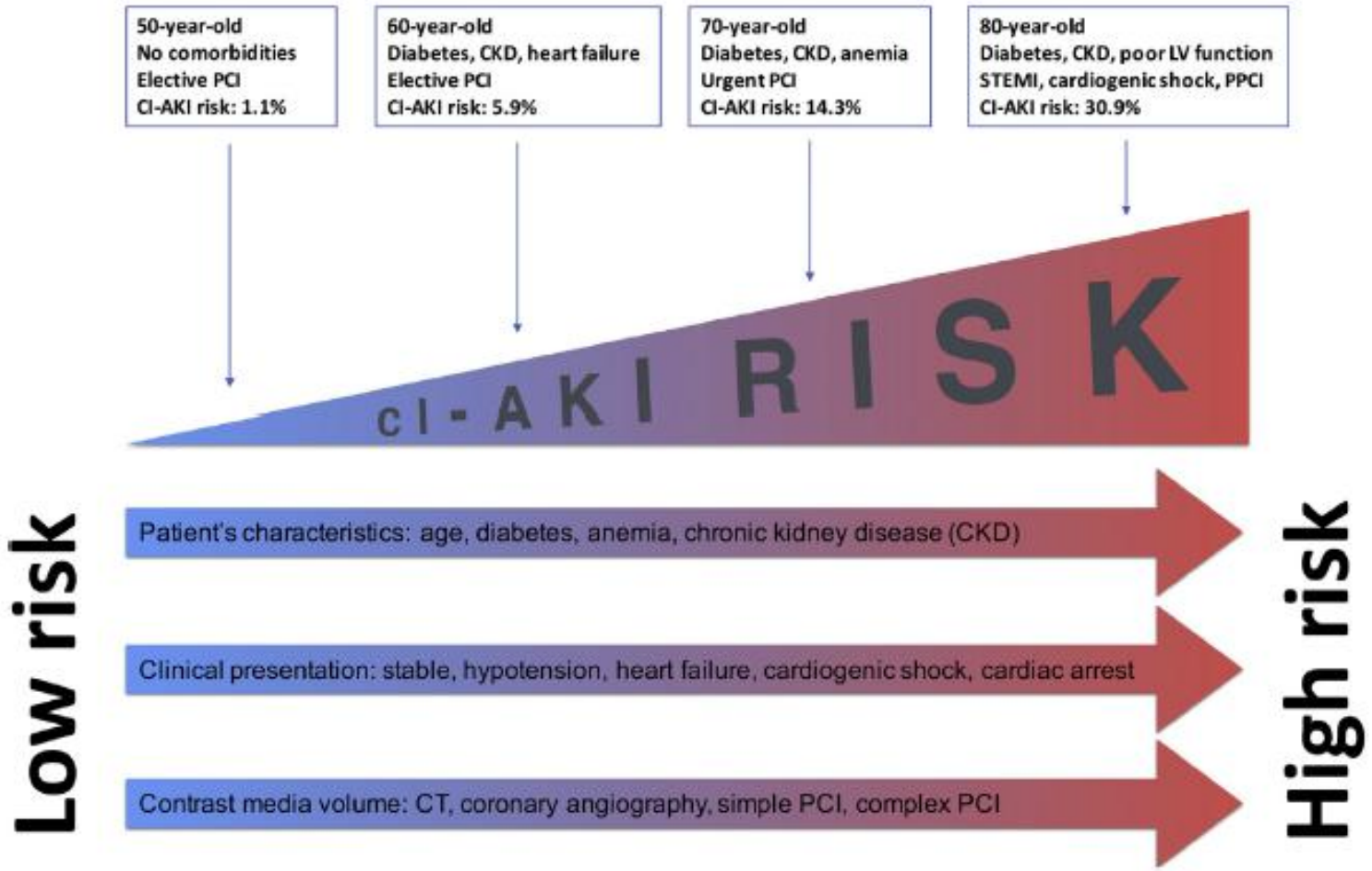
Ho et al. J Intens Care 2018.



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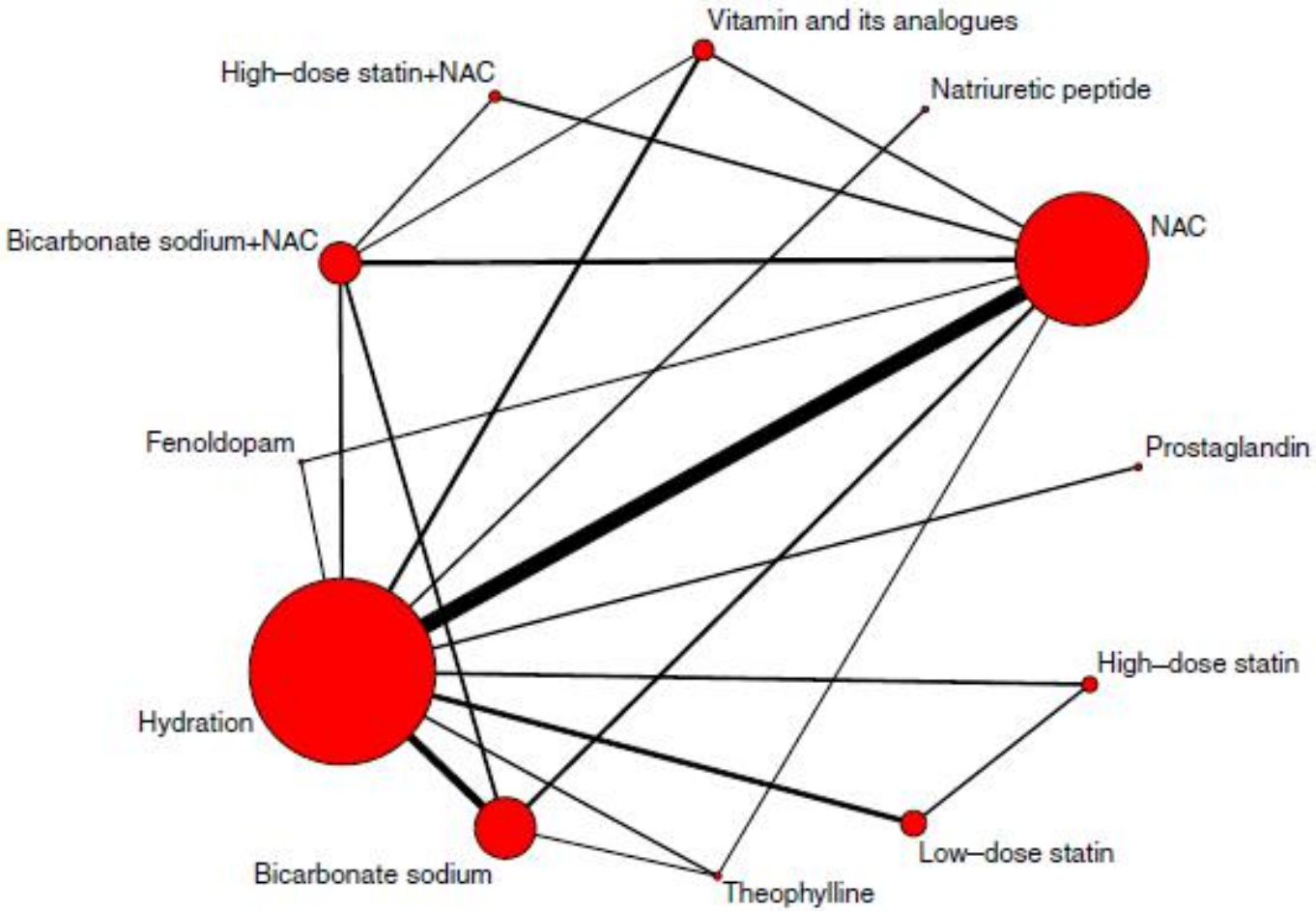
Azzalini L et al. Can J Card 2017.

Future directions

1) Biomarkers
2) Predicting scores
3) Preventive therapies

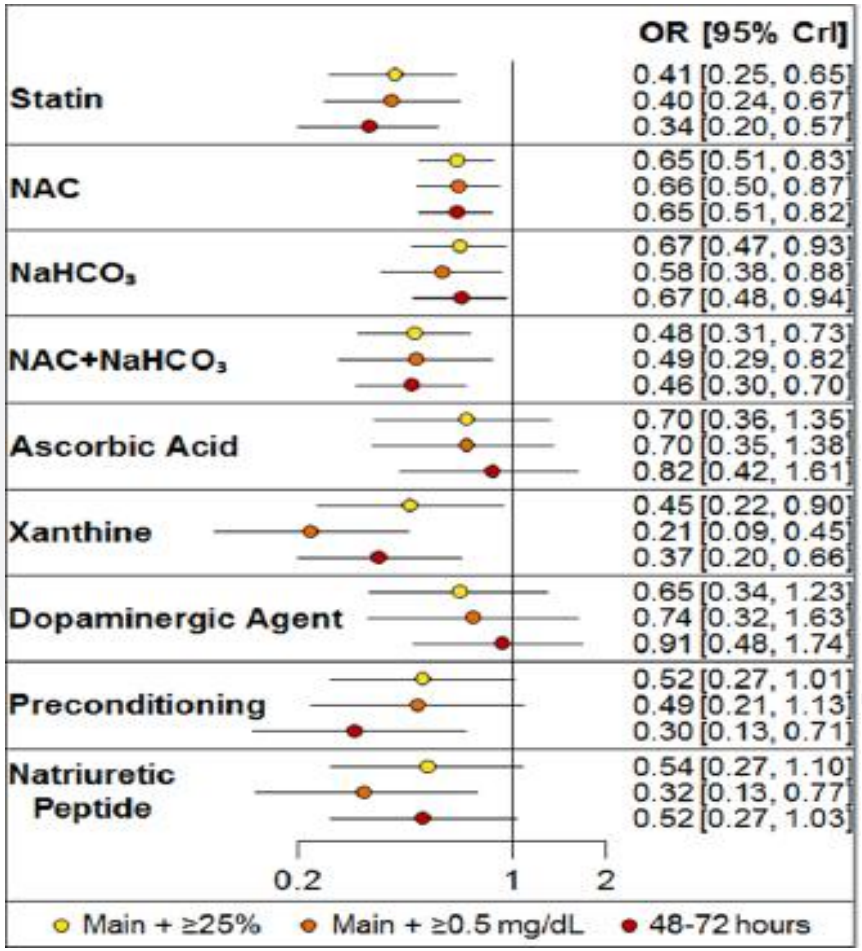


Su X. Am J Kidney Dis 2016.

Preventive Strategies for Contrast-Induced Acute Kidney Injury in Patients Undergoing Percutaneous Coronary Procedures

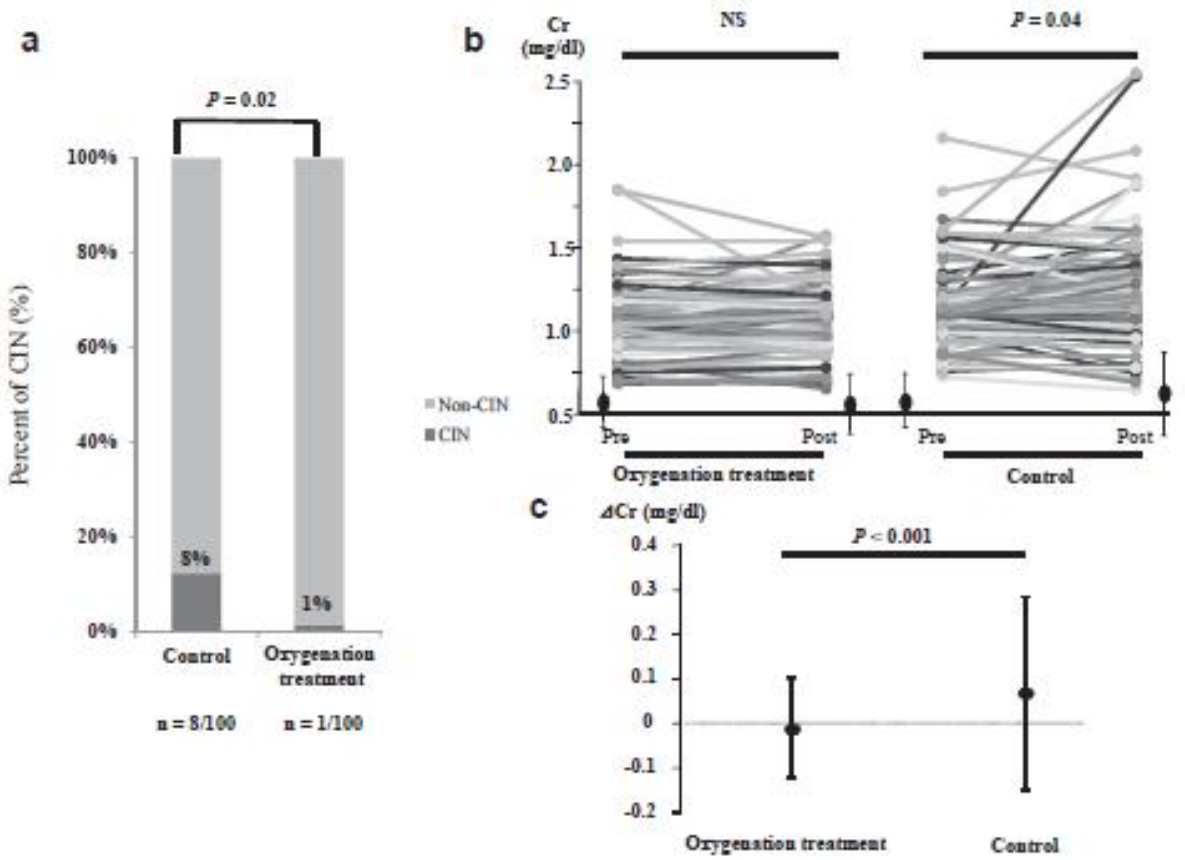
Evidence From a Hierarchical Bayesian Network Meta-Analysis of 124 Trials and 28 240 Patients

In patients undergoing percutaneous coronary procedures, **statin** administration is associated with a marked and consistent reduction in the risk of CI-AKI compared with saline.
Although xanthine, NAC, NaHCO3, NAC+NaHCO3, ischemic preconditioning, and natriuretic peptide may have nephro-protective effects, these results were not consistent across multiple sensitivity analyses.



Giacoppo D et al. Circ Cardiovasc Interv 2017.

Contrast-Induced Nephropathy and Oxygen Pretreatment in Patients With Impaired Renal Function



Oxygenation, a simple, nonpharmacological strategy, may be beneficial when using contrast media in patients with impaired renal function from noninvasive angiography to emergency catheterization.

Sekiguchi H et al. Kidney Int Rep 2018.

CONCLUSIONS

- CI-AKI is a real complication, although until recently its risk has been overestimated, particularly in cohorts of stable patients who undergo radiological imaging.
- Subjects who undergo emergency interventions have a higher prevalence of CI-AKI risk factors and are subject to a greater risk of CI-AKI, and therefore greatly benefit from targeted preventative measures to expand intravascular volume and augment renal perfusion.
- The right amount of fluid for each patient is needed to optimize outcomes and reduce the risk of CI-AKI.
- We look forward to solid randomized clinical trials that will identify optimal strategies to reduce the incidence, severity, and sequelae of CI-AKI, to optimize the outcomes of patients who undergo invasive management.

Memories from SIMEU Summer School 2017…

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American Journal of Emergency Medicine

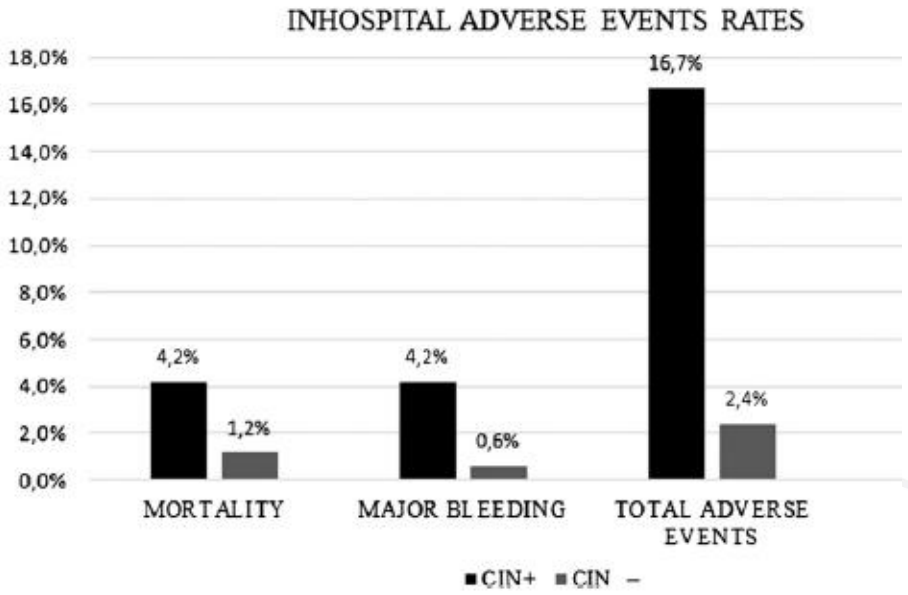
journal homepage: www.elsevier.com/locate/ajem

Original Contribution

Relation of contrast nephropathy to adverse events in pulmonary emboli patients diagnosed with contrast CT☆,☆☆

Multivariate analysis prediction of CIN

	OR	95% CI		p value
Age > 75 years	4.2	1.5	11.9	0.006
Baseline eGFR <60 ml/min/1.73m^2	3.2	1.1	9.8	0.033
History of DM	1.7	0.6	4.7	0.3
History of HT	2.0	0.5	8.3	0.1
Hemoglobin gr/dl	1.0	0.7	1.2	0.9
History of CHF	2.0	0.5	8.3	0.3



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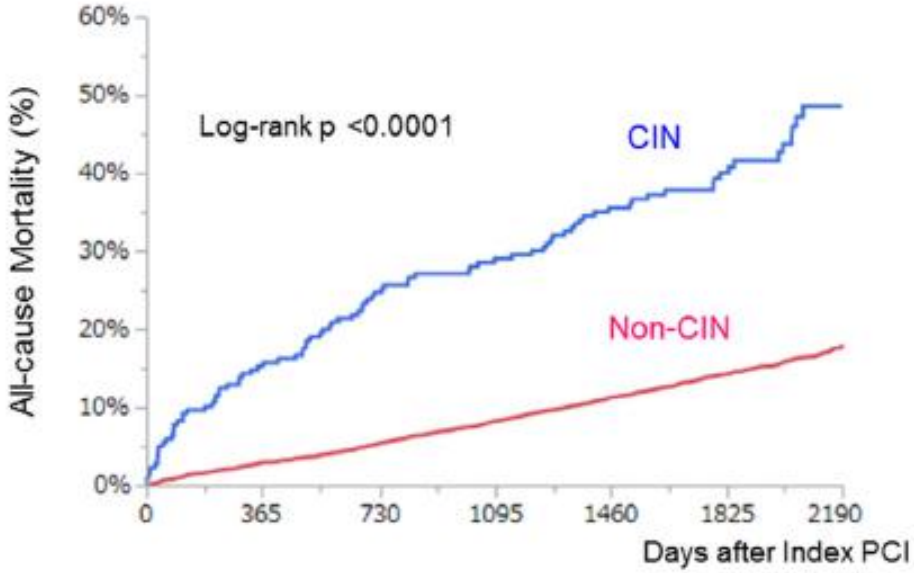
S. Yazıcı et al. American Journal of Emergency Medicine 2016.

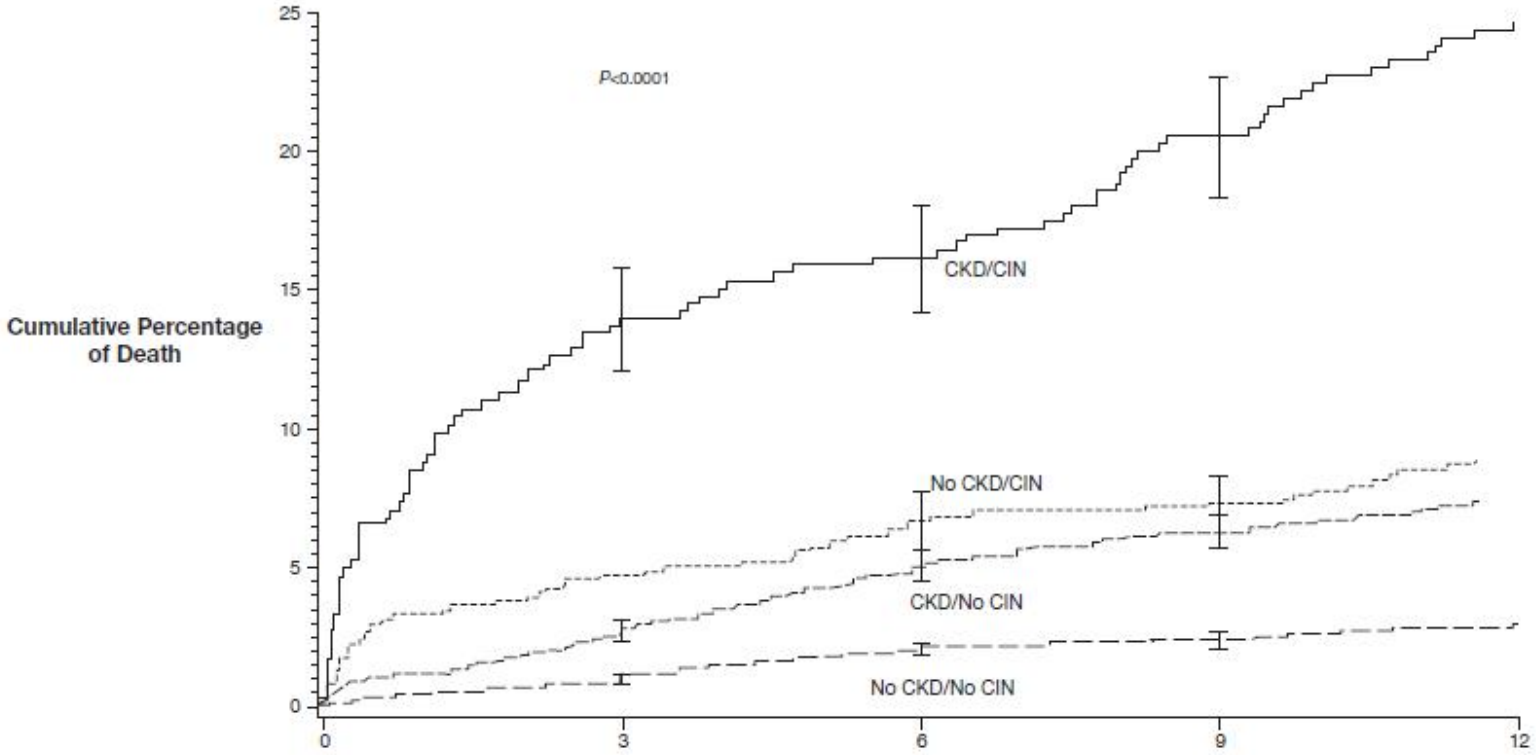


Time courses of serum creatinine values in patients with persistent CIN, transient CIN, and non-CIN

Definition of CIN	Pre-PCI	Post-PCI	1-year follow-up
Serum creatinine, mg/dL			
Persistent CIN	1.60 (1.08–3.03)	2.60 (1.87–4.28)	2.78 (1.80–5.04)*
Transient CIN	1.00 (0.90–1.53)	1.80 (1.50–2.33)	1.20 (1.00–1.70)*,†
Non-CIN	0.85 (0.70–1.00)	0.90 (0.77–1.09)	0.90 (0.75–1.08)†

Abe M et al. Am J Cardiol 2017.

One-year survival after percutaneous coronary intervention in patients with or without CKD and with or without CIN

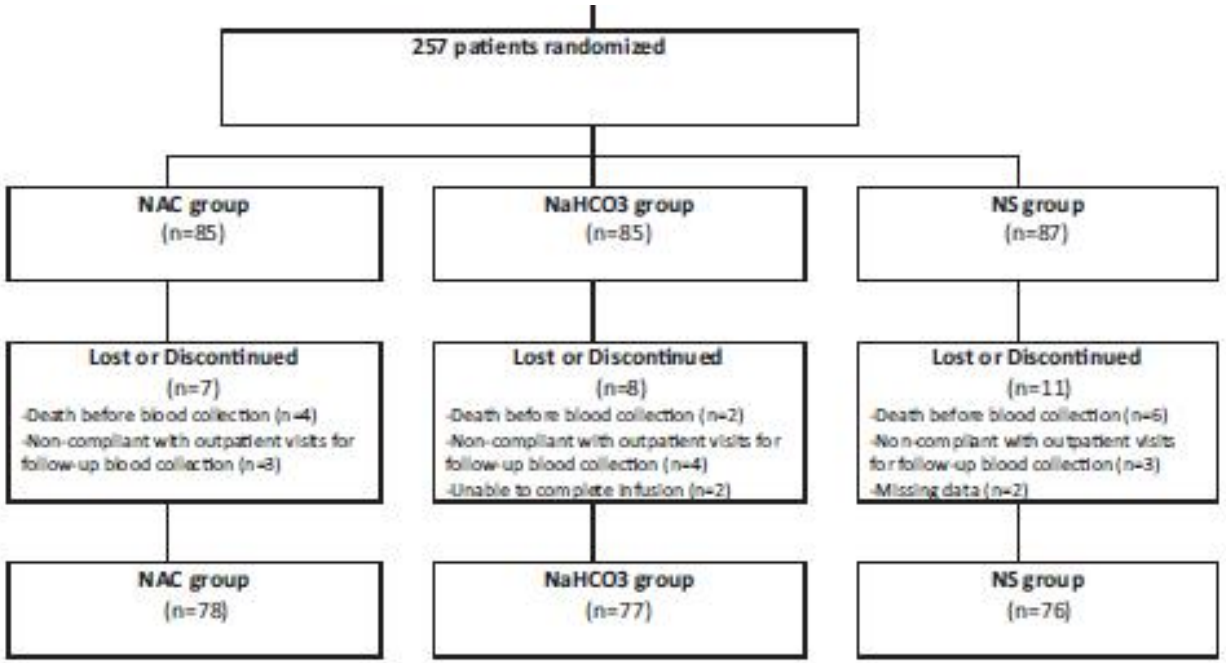


Dangas G et al. Am J Cardiol 2005.

Academic Emergency Medicine
Official Journal of the Society for Academic Emergency Medicine

ORIGINAL CONTRIBUTION

The High Risk of Contrast-induced Nephropathy in Patients with Suspected Pulmonary Embolism Despite Three Different Prophylaxis: A Randomized Controlled Trial



Association Between Acute Kidney Disease and Intravenous Dye Administration in Patients With Acute Stroke

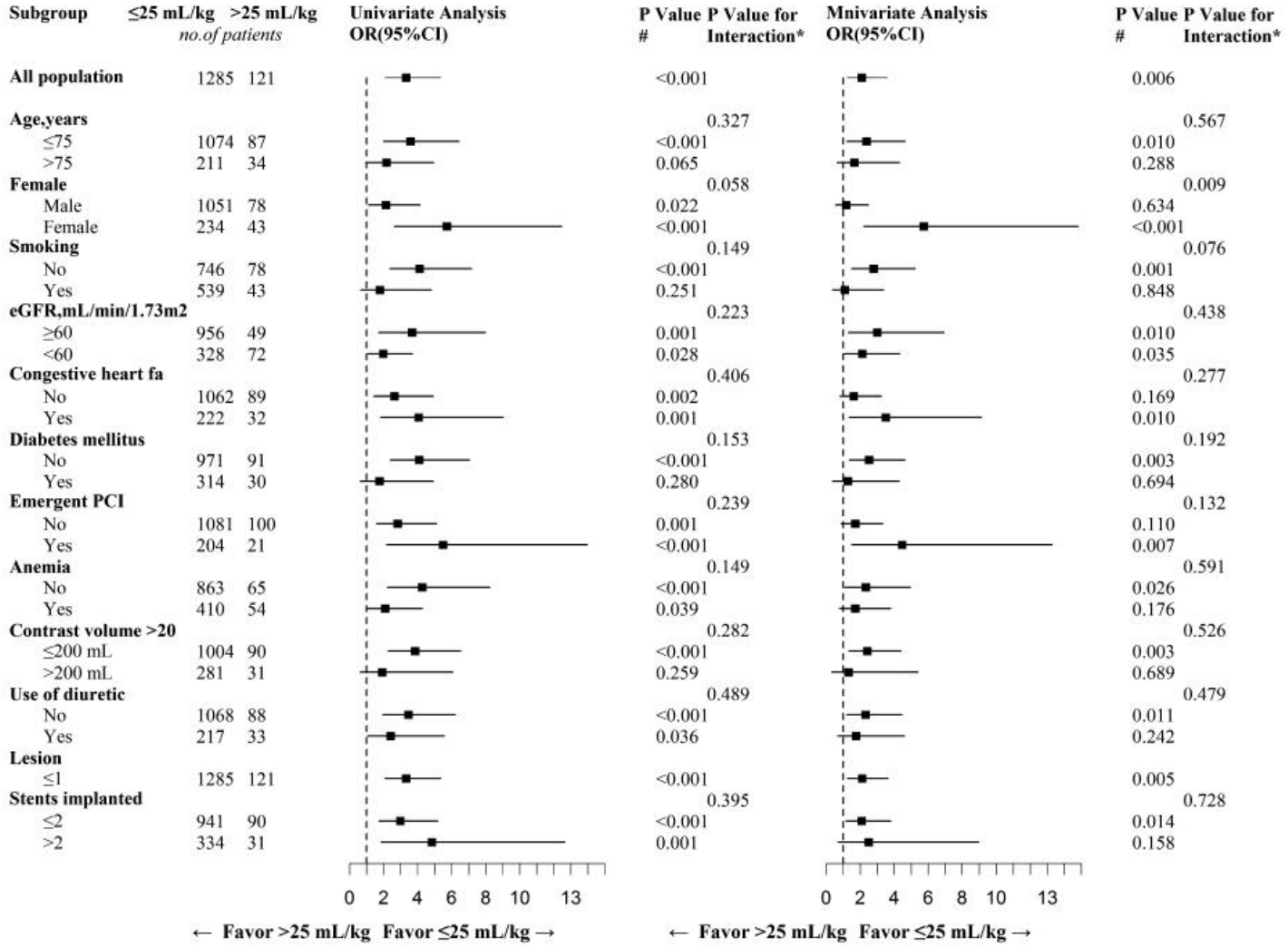
A Population-Based Study

Methods—All adult residents of the Greater Cincinnati/Northern Kentucky region with acute ischemic stroke or intracerebral hemorrhage who presented to an emergency department in 2010 were included. Prevalence of unsuspected kidney disease at the time of emergency department presentation and the incidence of AKI after admission in 2 groups of patients—those who did and those who did not receive intravenous dye—were determined.

Results—In 2010, 2299 patients met inclusion criteria (89% ischemic stroke and 11% intracerebral hemorrhage); mean age 69 years (SD 15), 22% black, and 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 with baseline kidney disease developed AKI during the hospital stay. Of those with no baseline kidney disease, 1% (14/14 467) developed AKI. There was no association between dye administration and new or worsening kidney disease.

Conclusions—Although 22% of patients in the Greater Cincinnati/Northern Kentucky 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. **(*Stroke*. 2017;48:835-839. DOI: 10.1161/STROKEAHA.116.014603.)**

	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



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J Am Heart Assoc. 2016

JACC: CARDIOVASCULAR INTERVENTIONS
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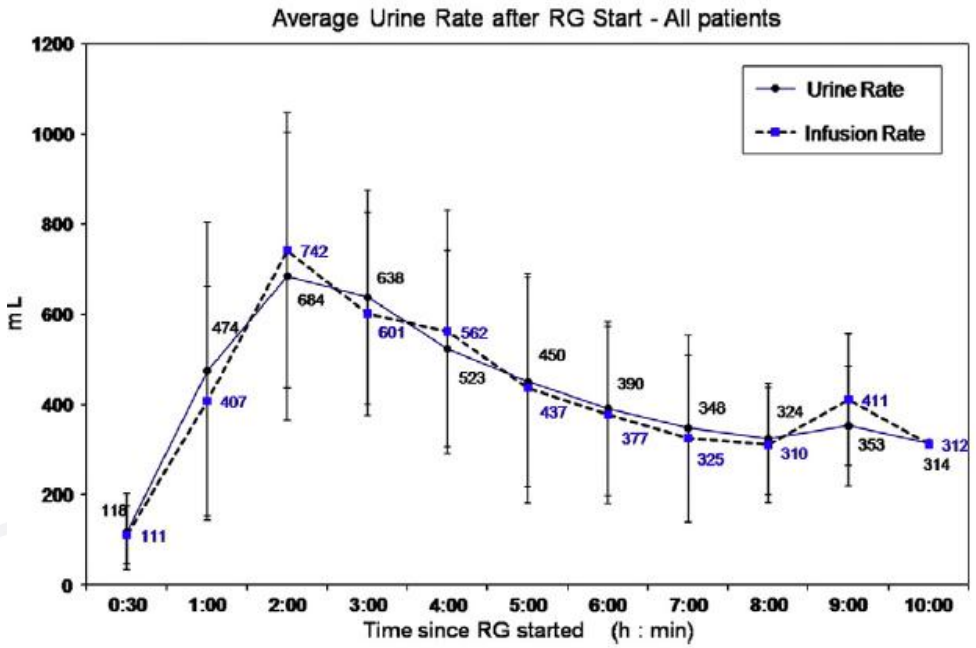
http://dx.doi.org/10.1016/j.jcin.2016.11.006

Prevention of Contrast-Induced Acute Kidney Injury by Furosemide With Matched Hydration in Patients Undergoing Interventional Procedures

A Systematic Review and Meta-Analysis of Randomized Trials



The RenalGuard System delivers intravenous fluids matched to urine output with a combination of hydration with normal saline at an initial dose bolus plus a low dose of furosemide and continuous monitoring for a urine output flow of >300 ml/h sustained for 6 h.

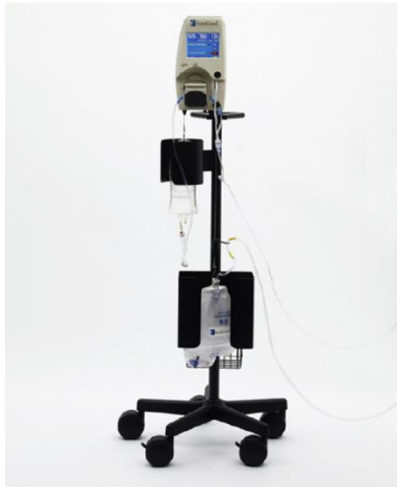


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Table 1. Overall and GFR stratified analysis for CIAKI prevention, with NNT.

Treatment	Overall analysis		GFR 59–30 ml/min		GFR <30 ml/min	
	Probability to be the best [%]	NNT	Probability to be the best [%]	NNT	Probability to be the best [%]	NNT
Saline	-	-	-	-	-	-
Saline plus N-acetylcysteine	0.0	30.79	0.0	33.90	16.99	5.35
Sodium bicarbonate	0.0	32.61	0.0	35.74	na	na
Sodium bicarbonate plus N-acetyl cysteine	0.006	20.22	0.001	24.89	na	na
Ascorbic acid	0.03	19.97	0.004	25.63	na	na
Statin	1.53	13.07	1.14	14.57	na	na
Furosemide	0.0	-14.29	0.13	-5.35	0.73	-3.51
Probucol	5.24	13.48	na	na	na	na
Methylxanthines	2.17	13.48	3.66	14.10	na	na
Fenoldopam	0.01	14.81	0.02	-9.52	1.64	-2.31
Device-guided matched hydration	12.45	12.15	11.74	11.62	na	na
Renal replacement therapy	0.14	19.74	0.002	-5.61	62.40	4.72
Nebivolol	0.94	14.81	1.93	13.97	na	na
Natriuretic peptides	3.22	14.84	0.78	-3.93	18.24	5.49
Mannitol	1.13	13.52	8.22	10.71	na	na
Prostaglandins	31.03	10.87	35.63	10.24	na	na
Trimetazidine	27.38	10.97	33.24	10.43	na	na
LVEDP-guided hydration	14.37	12.21	3.51	13.43	na	na

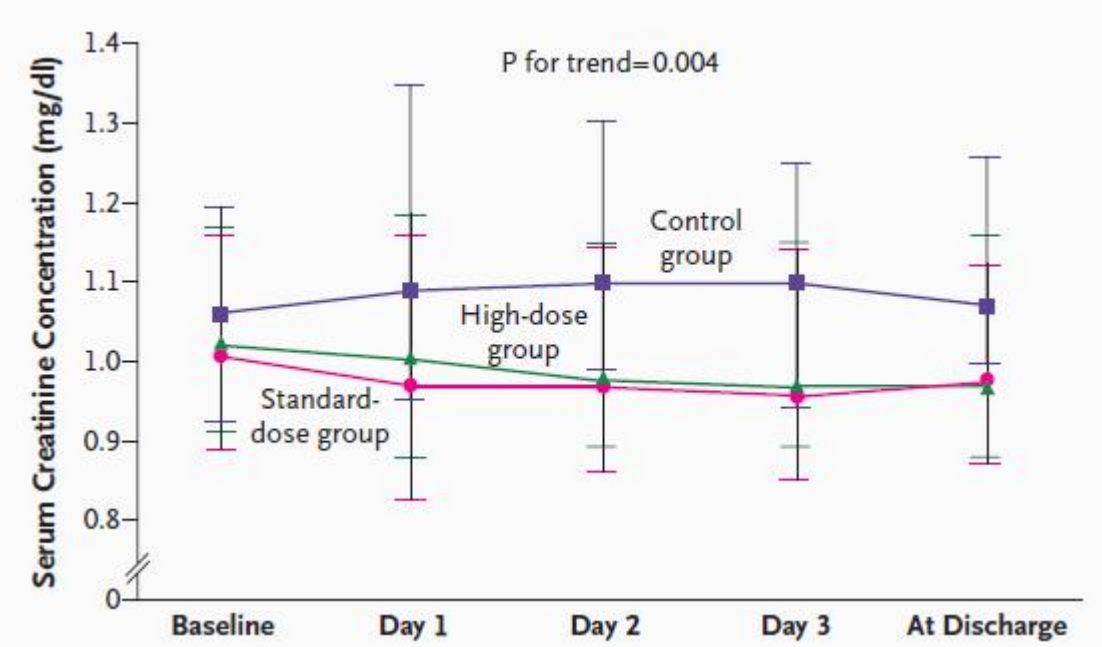
Currently recommended treatment with saline as the only measure to prevent CIAKI during cardiovascular procedures may not represent the optimal strategy. Vasodilators, when added to saline, may significantly reduce the odds of CIAKI following cardiovascular procedures.

Navarese EP et al. PLoS ONE 2017.

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

N-Acetylcysteine and Contrast-Induced Nephropathy in Primary Angioplasty



Marenzi G et al. N Engl J Med 2006.