- systematic consistently timed trend of Cr may be more informative
- large number of patients excluded
- the decision to obtain an enhanced study depends on many factors
- the value of defining AKI is debated
- would like to see a stricter definition of CIN than reliance on a Cr defined AKI (many etiologies: pre-renal, obstruction, nephrotoxic)
- no discussion regarding change in protocols and types of contrast materials used over 17 years.

Clinical Bottom Line: The current data seems to defend contrast administration when clinically indicated. Better markers / working definition for CIN and prospective blinded study would advance our understanding as would have randomized controlled studies.

Editorial Comment: I think their study was a compromise, a midway point towards a greater goal. They say they want to study a causal association between contrast and subsequent AKI but their ultimate goal is to investigate a causal association between contrast and CIN. The more rigorous they make the definition of AKI, I would argue the better data they obtain. As things are it would be impossible to prove a causal association for CIN with a study based on serum Cr values. This is a best first step, by design to dispute AKI is associated with contrast and thus to move forward with a RCT for deliberate study of CIN.

	subgroup with eGFR of 30–59 (n = 5510), OR was **0.94** (95% CI: 0.76, 1.18), P = .65; for the subgroup with eGFR of less than 30 mL/min/1.73 m2 (n = 1486), OR was **0.97** (95% CI: 0.72, 1.30), P = .89. Fairly precise considering the narrow range of the CI's
V. **Will the results help me in caring for my patients? (Applicable?)**	Yes, it offers some support for the use of contrast, particularly in the chronically ill who are set up for more pathology and more likely to have lower baseline eGFR. The patient population included some in-patients as well as outpatients.
1. Were the study patients similar to my patient?	They were primarily inpatient with available Cr data Probably similar to our admitted population.
2. Were all clinically important outcomes considered?	Probably not. AKI within 72 hours was the only measure of AKI. No patient-centered outcomes such as death, dialysis or renal impairment over a longer f/u period.
3. Are the likely treatment benefits worth the potential harm and costs?	For the diagnosis of emergent pathology that includes patient-centered decision making, I would say so. That stated, this is a retrospective study that is limited by both selection bias and performance bias of clinicians caring for these patients at the time of their assessments.

Study Limitations
- lab assay for serum Cr changed during the study
- Individuals with decreased GFR have greater variability of serum Cr
- no way to control for nephrotoxic drugs
- no way to control for ppx measures like additional fluid
- propensity scores cannot account for all variables
- potential to "cook" the data with a scoring system to alter results
- other confounders for AKI (e.g. pt hydration status)

	Propensity score generation and matching for each GFR group for both NC and Contrast groups Patients were similar in respect to age, race, medical comorbidities. They were not assessed by indication for CT scanning or surgical vs. medical management, or other AKI RF's such as nephrotoxic drugs.
III. **Did experimental and control groups retain a similar prognosis after the study started (answer the questions posed below)?**	
1. Were patients aware of group allocation?	Yes. The patients probably knew they were getting an IVC CT. This was retrospective so that knowledge would not predispose to performance bias on the part of the patient undergoing the test.
2. Were clinicians aware of group allocation?	Yes. Which is an area of performance bias where clinicians may have taken steps to try to mitigate IVC risks prior to or after IVC, which was not included in the data presented.
3. Were outcome assessors aware of group allocation?	Probably, no mention that outcome assessors were blinded to outcome measures. This is an area where blinding can be achieved in retrospective studies.
4. Was follow-up complete?	yes (for the potential 96 hour period for SCr data) or pt was excluded
IV. What were the results? Answer the questions posed below	
1. How large was the treatment effect? (Difference between treatment and control group).	After propensity matching, the incidence of AKI increased with decreasing eGFR from 1% in those with eGFR>90 to 14% in those with eGFR <30
2. What was the estimated treatment effect at a 95% confidence interval? (Precision)	For the subgroup with eGFR of 90 or greater (n = 1642), odds ratio (OR) was **0.91** (95% confidence interval [CI]: 0.38, 2.15), P = .82; for the subgroup with eGFR of 60–89 (n = 3870), OR was **1.03** (95% CI: 0.66, 1.60), P = .99; for the

	had demographic variables for Modification of Diet in Renal Disease (MDRD) eGFR equation
4. Exclusion Criteria	-preexisting dialysis requirements prior to or on the day of the scan -underwent additional contrast procedures within a 14-day period of scan -dx ARF within 14 days prior to scan (as determined by ICD9 coding) -inadequate labs for detection of AKI
5. Interventions Compared	Iodinated Contrast CT vs NC CT
6. Outcomes Evaluated	Incidence of AKI (>= 0.5 Cr increase) 24-72 hours after imaging. Propensity score matching of 41,249 patients was applied at each GFR group, using logistical regression that was derived from 13 clinical variables.
II. **Are the results of the study valid**	
1. Was the assignment of patients randomized?	no, retrospective study, however propensity scoring was applied to mitigate selection bias
2. Was randomization concealed (blinded)?	n/a
3. Were patients analyzed in the groups to which they were randomized?	Yes. All patients were analyzed in the groups they were assigned to namely those who underwent IVC study and those who did not.
4. Were patients in the treatment and control groups similar with respect to known prognostic factors?	Pts were stratified by eGFR to mirror guidelines for CKD.

Journal Club Eastern Virginia Medical School
Therapy Article

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CITATION: McDonald JS, McDonald RJ, Carter RE, Katzberg RW, Kallmes DF, Williamson EE. Risk of intravenous contrast material-mediated acute kidney injury: a propensity score-matched study stratified by baseline-estimated glomerular filtration rate. Radiology. 2014 Apr;27 1 (1):65-73

I. **WHAT IS BEING STUDIED?**	
1. Study Objective	To determine the effect of baseline eGFR on the causal association between IV iodinated contrast and development of AKI in propensity score matched groups of patients note that this is AKI of any cause, so called "Post-contrast" AKI (PC-AKI) which is a correlative diagnosis
2. Study Design	Retrospective single-center study. Patients were stratified by eGFR using KDOQ cutoffs for CKD (>= 90, 60-89, 30-59, less than 30). MDRD equation used for eGFR. Propensity score generation and 1:1 matching of patients were performed in each eGFR subgroup. Incidence of AKI (Cr defined >= 0.5) was compared in matched subgroups using Fisher exact test This study used a previous dataset in which patients were stratified by serum Cr
3. Inclusion Criteria	All patients who had a contrasted CT or unenhanced CT between 1/01/2000 & 12/31/2010 at the Mayo Clinic (Rochester, MN) had pre and post scan SCr (24h prior and 24-72 hr following)