CONCLUSIONS

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BACKGROUND

- Contrast-induced nephropathy (CIN) and its incidence are poorly characterized.
  - Thought to be a result of direct tubular toxicity of the contrast media and possibly due to vasoconstriction of the renal vasculature.
  - Iso-osmolar and low-osmolar CM are less likely to be associated with CIN.
- Only pre-hydration has been found to consistently reduce the risk of nephrotoxicity of CM.6
- CIN is thought to have multiple possible mechanisms: see below8

METHODS

- Conducted a retrospective review to determine the incidence of CIN in patients with elevated baseline Scr after undergoing (CT) using IVCM.
  - Queried the electronic medical record at a community hospital
    - Patients with Scr between 1.2 mg/dL and 2.5 mg/dL who underwent CT utilizing IVCM between January & July 2000.
    - Patients with Scr between 1.2 mg/dL and 2.5 mg/dL who underwent CT utilizing IVCM for CT imaging.

RESULTS

193 patients had 236 contrast studies

<table>
<thead>
<tr>
<th>9 of 193 patients had elevated creatinine within 1 month age 60-92</th>
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<tr>
<td>3 women, 6 men</td>
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<td>8 of 9 had oncologic diagnosis</td>
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<tr>
<td>1 of 9 had Crohn disease</td>
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<td>1 of 9 had a solitary kidney</td>
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<td>4 of 9 had HTN</td>
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<td>2 of 9 had DM2</td>
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<td>1 of 9 had more than 1 study in 48h</td>
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<td>7 of 9 died within 2 years</td>
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<td>5 of 9 hospitalized</td>
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<td>2 of 9 outpatients — 1 with chemotherapy</td>
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<tr>
<td>2 of 9 outpatients — 2 with chemo prior to Scr recheck</td>
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<tr>
<td>4 of 9 on chemotherapy</td>
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<td>3 of 9 got nephrotoxic chemotherapy</td>
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DISCUSSION

- The results show that CIN in patients with elevated baseline Scr given IVCM is uncommon.

- Incidence of CIN in our cohort was 4.7%, which is lower than the published incidence of CIN in patients at an even higher risk for CIN.
  - Newhouse et al’s study that examined hospitalized patients’ Scr changes in the absence of iodinated CM exposure published rates of elevation in Scr much higher than our patients. Based on this study, our study should have revealed at least a 7% to 26% prevalence of increase in Scr even without CM injection.

- Although CIN has been a named entity for years, its pathophysiology is not well understood and methods proven to prevent it have been lacking.
  - Incidence of CIN ranges widely—1% to 33%—suggesting that either CIN is not well defined or that it does not even exist.
  - Newhouse et al examined the frequency of Scr changes in the absence of CM and found that Scr changes of magnitudes specified in both absolute Scr in mg/dL and in percent change from baseline were similar to the published incidence of CIN.
  - Most prevention studies were done using intra-arterial administrations of CM, particularly for coronary catheterization procedures. There are few studies contrasting intra-venous and intra-arterial injections of CM.7
  - Few of these have utilized control groups not exposed to iodinated CM exposure.

- Patients with decreased renal function are treated as being at higher risk for CIN and preventive measures have been sought. Peri-procedural hydration is the standard of care — but is this necessary?
  - In a pooled analysis of two RCTs of patients with GFR <60ml/min utilizing IVCM for CT imaging, Thomas and Morcos1 found that patients needed only observation after CIN was diagnosed. All the patients recovered to their baseline.

- The role of IVCM in causing CIN and, thus, AKI, may be overestimated.
  - CIN is a clinically nebulous entity, and past published rates of CIN need to be evaluated within the context of the study populations, their baseline renal function, and the amount of change from this baseline in the individual studies. It is difficult to generalize the results from these studies.
  - Further controlled studies need to be done to determine whether CIN due to IVCM is, first, a true entity and, second, if patients with CKD and AKI are more at risk than people with normal renal function of developing CIN.
  - Would patients receiving IVCM for routine outpatient studies who have no other risk factors for kidney injury other than elevated Scr truly be at increased risk of developing CIN? Would the risk warrant the expense, risks, and inconvenience of peri-procedural IV hydration?
  - Furthermore, by expanding the population of patients who receive IVCM, we will obtain better imaging studies and, therefore, more accurate diagnoses.

CONCLUSIONS

- Stratta et al1 suggest that CIN is a marker for worsening renal and systemic prognosis.
  - Patients who develop CIN usually have multiple risk factors for AKI and all 9 of our patients who developed CIN had other risk factors for AKI.
  - Otherwise healthy people with elevated baseline Scr (1.2-2.5md/dL) should be at minimal risk.

- Limitations of our study:
  - Small sample size: question of enough power to detect CIN reliably.
  - Retrospective.
  - Did not control for hospitalized or non-hospitalized patients.
  - Hospitalized patients are more systemically ill and are more likely to be volume depleted. They have other complicating conditions: fluctuations in blood pressure and exposure to nephrotoxic medications.

- If most of our study patients were outpatients, the prevalence of CIN would likely be lower because these patients are less ill. Nonetheless, this is also a strength of our study because despite including more ill patients, the risk for CIN is minimal and should not require peri-procedural hydration.

REFERENCES