Update in Contrast Induced Nephropathy

Yves Pirson

Service de Néphrologie, Clin. Univ. St-Luc - UCL
A 76-year-old man with
- type 2 diabetes
- CKD (ser. creat.: 1.8 mg/dl; GFR: 32)

presents with angina pectoris

→ indication for elective coronaryography
Course of kidney function

Definition of CIN:
- either $\uparrow$ in ser. creat. $> 0.5$ mg/dl
- or $> 25\%$ increase in ser. creat.
Risk score for prediction of CIN after percutaneous coronary intervention

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 75</td>
<td>4</td>
</tr>
<tr>
<td>Diabetes</td>
<td>3</td>
</tr>
<tr>
<td>Systolic BP &lt; 80 mmHg durant &gt; 1h</td>
<td>5</td>
</tr>
<tr>
<td>GFR 40 – 60</td>
<td>2</td>
</tr>
<tr>
<td>20 – 39</td>
<td>4</td>
</tr>
<tr>
<td>Contrast volume</td>
<td>1/100 ml</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk score</th>
<th>Risk of CIN (%)</th>
<th>Risk of dialysis (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 – 5</td>
<td>7.5</td>
<td>0.04</td>
</tr>
<tr>
<td>6 – 10</td>
<td>14</td>
<td>0.12</td>
</tr>
<tr>
<td>11 – 15</td>
<td>26</td>
<td>1.09</td>
</tr>
<tr>
<td>≥ 16</td>
<td>57</td>
<td>12.6</td>
</tr>
</tbody>
</table>

*(Mehran R et al., J Am Coll Cardiol 2004; 44: 1393)*
### Prevalence of CKD in the US

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
<th>1988-1994</th>
<th>1999-2004</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>(persistent albuminuria + normal GFR)</td>
<td>1.7%</td>
<td>1.8%</td>
</tr>
<tr>
<td>Stage 2</td>
<td>(persistent albuminuria + GFR 60-89)</td>
<td>2.7%</td>
<td>3.2%</td>
</tr>
<tr>
<td>Stage 3</td>
<td>(GFR 30-59)</td>
<td>5.4%</td>
<td>7.7%</td>
</tr>
<tr>
<td>Stage 4</td>
<td>(GFR 15-29)</td>
<td>0.2%</td>
<td>0.4%</td>
</tr>
<tr>
<td>Stage 5</td>
<td>(kidney failure)</td>
<td>0.2%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>10.0%</td>
<td>13.1%</td>
</tr>
</tbody>
</table>

*(Andersen PE, World J Radiol 2012; 4: 253)*
Is it important to avoid CIN?

Pathophysiology of CIN

Risk factors for developing CIN

How to prevent CIN?
Yes, AKI Truly Leads to CKD

Chi-yuan Hsu
Division of Nephrology, University of California–San Francisco, San Francisco, California

Persistent Renal Damage After Contrast-Induced Acute Kidney Injury
Incidence, Evolution, Risk Factors, and Prognosis

Mauro Maioli, MD; Anna Toso, MD; Mario Leoncini, MD; Michela Gallopin, MD; Nicola Musilli, MD; Francesco Bellandi, MD

Background—The temporal evolution of renal function in patients with acute kidney injury after contrast medium (CI-AKI) is not well known. The aim of this observational study was to evaluate the incidence, risk factors, and prognostic implications of persistent renal damage (RD) in patients with preexistent moderate-to-severe renal dysfunction.

Methods and Results—From June 2003 to March 2008, 3986 patients underwent coronary angiography at our institution; 1490 of 3986 had an estimated creatinine clearance of <60 mL/min and were enrolled. CI-AKI was defined as an absolute increase $\geq 0.5 \text{ mg/dL}$ over baseline serum creatinine within 3 days after the administration of contrast medium (iodixanol). In patients who developed CI-AKI, persistent RD was defined as a relative decrease of creatinine clearance $\geq 25\%$ over baseline at 3 months. Patients whose creatinine clearance returned to baseline (or nearly) were classified as transient RD. The overall incidence of CI-AKI was 12.1%, and persistent RD occurred in 18.6% of CI-AKI patients. At Cox regression analysis, nephropathy risk score $\geq 17$, left ventricular ejection fraction $\leq 30\%$, and increased value of serum creatinine $\geq 1.5$-fold from baseline within 5 days were found to be significant risk factors for persistent RD. At 5 years, the incidence of death was significantly higher in patients with persistent RD than in both patients with transient RD ($P=0.015$) and those without CI-AKI ($P=0.0001$). A similar trend was observed for the combined end point of death, dialysis and cardiovascular events.

Conclusions—These results suggest that CI-AKI is not always a transient, benign creatininohipathy, but rather a direct cause of worsening renal function. The occurrence of CI-AKI can identify patients at increased risk of cardiovascular events. (Circulation. 2012;125:3099-3107.)
(Maioli M, Circulation 2012;125:3099)
June 2003 – March 2008
3986 patients with coronary angiography

1490 patients with moderate to severe renal dysfunction

72 hours post angiography or PCI

NO CI-AKI
1310 patients

Deaths
3 patients (0.2%)

Hospital discharge
1307 patients

Deaths
15 patients (1.2%)

Lost to 3 month creatinine analysis
362 patients (27.6%)

3-month creatinine analysis
930 patients (71.0%)

Confirmed No renal damage
921 patients 99.1%

Persistent renal damage
9 patients 0.9%

CI-AKI
180 patients

Deaths
10 patients (5.5%)

Hospital discharge
170 patients

Deaths
3 patients (1.7%)

3-month creatinine analysis
167 patients (92.8%)

Transient renal damage
136 patients 81.4%

Persistent renal damage
31 patients 18.6%

12%

2%
Effect of acute kidney injury frequency on survival to stage 4 CKD
(Chawla LS, Kidney Int 2012; 82: 516)
Is it important to avoid CIN?

Pathophysiology of CIN

Risk factors for developing CIN

How to prevent CIN?
Administration of contrast media

↑ Tubular load
↑ Tubular transport

↑ Hydrolysis of ATP

↑ Adenosine receptor signaling

↑ Blood viscosity

↑ Endothelin release
↓ Nitric oxide release

↑ ROS generation

↑ Vasoconstriction of afferent arterioles

↓ Renal blood flow
↓ GFR

Renal ischemia

AKI and/or acute renal tubular injury

CIAKI
Progression of diabetic nephropathy

(Calvin AD, Nat Rev Nephrol 2010; 6: 679)
Is it important to avoid CIN?

Pathophysiology of CIN

Risk factors for developing CIN

How to prevent CIN?
Patient-related risk factors for CIN

Classical
- Age > 70
- CKD
- Diabetes mellitus
- Congestive heart failure
- Dehydration (! diuretics)
- Use of nephrotoxic drugs (NSAID, anticalcineurins, some antivirals)

Recently recognized risk factors for AKI in general
- Albuminuria with preserved eGFR
Adjusted hazard ratio for acute kidney injury according to eGFR and albuminuria

Albumin-to-creatinine ratio

> 300 mg/g
30 – 299 mg/g
< 30 mg/g

(Gansevoort R, Kidney Int 2011; 80:93)
Is it important to avoid CIN?

Pathophysiology of CIN

Risk factors for developing CIN

How to prevent CIN?
How to prevent CIN?

Assessment of the individual risk for CIN

Minimisation of this risk

Volume expansion

Pharmacological prevention?

Non-pharmacological preconditioning?

Clinical practice
Screening of the population at risk for CIN

- Ser. creat. in all patients
  ➔ at risk: eGFR < 45

- Simple risk-factor questionnaire:
  - known CKD?
  - diabetes?
  - c-v disease?
  - list of current medications
  - contrast media within the last 3 days?

- Urinary-protein screening advisable
In patients with increased risk:

is contrast injection absolutely needed?
How to prevent CIN?

Assessment of the individual risk for CIN

Minimisation of this risk

Volume expansion

Pharmacological prevention?

Non-pharmacological preconditioning?

Clinical practice
Minimisation of the risk

- Discontinue concurrent nephrotoxic medications (NSAID, diuretics) whenever possible for > 3 days
- No compelling reason to discontinue ACE-I/ARB
- Avoid repeat contrast injection within 72 hours
- Use the lowest volume of contrast
- Apply a prevention protocol
Which prevention strategy?
How to prevent CIN?

Assessment of the individual risk for CIN

Minimisation of this risk

Volume expansion

Pharmacological prevention?

Non-pharmacological preconditioning?

Clinical practice
« Despite the recognition of volume depletion as an important risk factor for AKI, there are no RCT that have directly evaluated the role of fluids vs placebo in the prevention of AKI » (KDIGO)

There is however a large consensus as to recommend volume expansion in at-risk patients

But:

• Oral or i-v ?
• Na chloride or bicarbonate ?
A randomized trial of saline hydration to prevent contrast nephropathy in chronic renal failure patients

Bertrand Dussol¹,², Sophie Morange², Anderson Loundoun³, Pascal Auquier³ and Yvon Berland¹,²

<table>
<thead>
<tr>
<th>Arm</th>
<th>Treatment Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>(n = 79) NaCl 1g/10kg/d per os for 2 days</td>
</tr>
<tr>
<td>B</td>
<td>(n = 77) 0.9% NaCl i.v. 15 ml/kg for 6 h before</td>
</tr>
<tr>
<td>C</td>
<td>(n = 80) idem arm B + theophylline 5mg/kg 1 h before</td>
</tr>
<tr>
<td>D</td>
<td>(n = 79) idem arm B + furosemide 3mg/kg i.v. after</td>
</tr>
</tbody>
</table>

Oral Hydration and Alkalization is Noninferior to Intravenous Therapy for Prevention of Contrast-Induced Nephropathy in Patients with Chronic Kidney Disease

ROY CHO, M.D., NOSHEEN JAVED, M.D., DARREN TRAUB, M.D., SOBAN KODALI, M.D., FOLEFAC ATEM, Ph.D., and VENKATRAMAN SRINIVASAN, M.D.
Oral route vs i-v route

« We recommend not using oral fluids alone in patients at increased risk of CIN »

KDIGO. Kidney Int 2012; Suppl 2: 69

« We suggest using the oral route for hydration, on the premise that adequate intake of fluid and salt are assured (2C).

We suggest that, when oral intake of fluid and salt is deemed cumbersome in patients at increased risk of CIN, hydration should be performed by intravenous route (2C) »

ERBP. Nephrol Dial Transplant 2012; 27: 4263
Prevention of Contrast-Induced AKI: A Review of Published Trials and the Design of the Prevention of Serious Adverse Events following Angiography (PRESERVE) Trial

Clinical trials comparing i-v bicarbonate with i-v saline for CIN

<table>
<thead>
<tr>
<th>Authors</th>
<th>Sample Size (N)</th>
<th>Diabetes (%)</th>
<th>Baseline SCr (mg/dl)</th>
<th>Primary Outcome</th>
<th>Cl-AKI (%)</th>
<th>Prespecified Effect Size (%)</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Bicarbonate Arm</td>
<td>Saline Arm</td>
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<tr>
<td>Positive studies</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Briguori et al. (5)</td>
<td>219</td>
<td>52</td>
<td>2.0</td>
<td>↑ SCr ≥25%</td>
<td>1.9</td>
<td>9.9</td>
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<tr>
<td>Masuda et al. (6)</td>
<td>59</td>
<td>31</td>
<td>1.3</td>
<td>↑ SCr ≥0.5 mg/dl or ≥25%</td>
<td>6.6</td>
<td>34.5</td>
</tr>
<tr>
<td>Merten et al. (7)</td>
<td>119</td>
<td>48</td>
<td>1.7–1.9</td>
<td>↑ SCr ≥25%</td>
<td>1.7</td>
<td>13.6</td>
</tr>
<tr>
<td>Ozcan et al. (9)</td>
<td>176</td>
<td>45</td>
<td>1.4</td>
<td>↑ SCr ≥0.5 mg/dl or ≥25%</td>
<td>4.2</td>
<td>16.6</td>
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<tr>
<td>Pakfet et al. (10)</td>
<td>192</td>
<td>30</td>
<td>1.1</td>
<td>↑ SCr ≥0.5 mg/dl</td>
<td>4.2</td>
<td>12.5</td>
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<tr>
<td>Recio-Mayoral et al. (11)</td>
<td>111</td>
<td>30</td>
<td>1.0</td>
<td>↑ SCr ≥0.5 mg/dl</td>
<td>1.8</td>
<td>21.8</td>
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<tr>
<td>Maioli et al. (18)</td>
<td>450</td>
<td>21</td>
<td>1.1</td>
<td>↑ SCr ≥0.5 mg/dl or ≥25%</td>
<td>12</td>
<td>22.7</td>
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<tr>
<td>Motohiro et al. (8)</td>
<td>155</td>
<td>58</td>
<td>1.5</td>
<td>↑ SCr ≥0.5 mg/dl or ≥25%</td>
<td>2.6</td>
<td>13</td>
</tr>
<tr>
<td>Negative studies</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adolph et al. (13)</td>
<td>145</td>
<td>34</td>
<td>1.5–1.6</td>
<td>↑ SCr ≥0.5 mg/dl or ≥25%</td>
<td>4.2</td>
<td>2.7</td>
</tr>
<tr>
<td>Brar et al. (14)</td>
<td>353</td>
<td>44</td>
<td>1.5</td>
<td>↑ eGFR ≥25%</td>
<td>13.3</td>
<td>14.6</td>
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<tr>
<td>Maioli et al. (12)</td>
<td>502</td>
<td>24</td>
<td>1.2</td>
<td>↑ SCr ≥0.5 mg/dl</td>
<td>10</td>
<td>11.5</td>
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<tr>
<td>Vasheghani et al. (20)</td>
<td>265</td>
<td>22</td>
<td>1.6–1.6</td>
<td>↑ SCr ≥0.5 mg/dl or ≥25%</td>
<td>7.4</td>
<td>5.9</td>
</tr>
<tr>
<td>Lee et al. (17)</td>
<td>382</td>
<td>65</td>
<td>1.5</td>
<td>↑ SCr ≥0.5 mg/dl or ≥25%</td>
<td>9.0</td>
<td>5.3</td>
</tr>
<tr>
<td>Briguori et al. (15)</td>
<td>292</td>
<td>70</td>
<td>1.8</td>
<td>↑ SCr ≥0.3 mg/dl</td>
<td>20.5</td>
<td>11</td>
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<tr>
<td>Shavit et al. (19)</td>
<td>87</td>
<td>44</td>
<td>1.8–1.9</td>
<td>↑ SCr ≥0.3 mg/dl or ≥25%</td>
<td>9.8</td>
<td>8.4</td>
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<tr>
<td>Rosenstock et al. (21)</td>
<td>142</td>
<td>51</td>
<td>1.7</td>
<td>↑ SCr ≥0.5 mg/dl or ≥25%</td>
<td>NR</td>
<td>NR</td>
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<tr>
<td>Castini et al. (16)</td>
<td>156</td>
<td>27</td>
<td>1.5–1.6</td>
<td>↑ SCr ≥25%</td>
<td>14</td>
<td>15</td>
</tr>
</tbody>
</table>

## Meta-analyses of bicarbonate vs saline for CIN prevention

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year Published</th>
<th>Included Trials (N)</th>
<th>Patients (N)</th>
<th>RR/OR(^a)</th>
<th>95% CI</th>
<th>Statistical Heterogeneity</th>
<th>Publication Bias</th>
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</thead>
<tbody>
<tr>
<td>Brar et al. (60)</td>
<td>2009</td>
<td>14</td>
<td>2290</td>
<td>0.85(^b)</td>
<td>0.63 to 1.16</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Brown et al. (61)</td>
<td>2009</td>
<td>10</td>
<td>1594</td>
<td>0.65</td>
<td>0.4 to 1.05</td>
<td>Yes</td>
<td>NR</td>
</tr>
<tr>
<td>Ho et al. (62)</td>
<td>2008</td>
<td>4</td>
<td>573</td>
<td>0.22</td>
<td>0.11 to 0.44</td>
<td>No</td>
<td>NR</td>
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<tr>
<td>Hogan et al. (63)</td>
<td>2008</td>
<td>7</td>
<td>1307</td>
<td>0.37</td>
<td>0.18 to 0.71</td>
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<tr>
<td>Hoste et al. (64)</td>
<td>2009</td>
<td>18</td>
<td>3055</td>
<td>0.66</td>
<td>0.45 to 0.95</td>
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<tr>
<td>Joannidis et al. (65)</td>
<td>2008</td>
<td>9</td>
<td>2043</td>
<td>0.45</td>
<td>0.26 to 0.79</td>
<td>Yes</td>
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<tr>
<td>Kanbay et al. (66)</td>
<td>2009</td>
<td>17</td>
<td>2448</td>
<td>0.54</td>
<td>0.36 to 0.83</td>
<td>Yes</td>
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<tr>
<td>Kuanadian et al. (70)</td>
<td>2011</td>
<td>7</td>
<td>1734</td>
<td>0.33</td>
<td>0.16 to 0.69</td>
<td>Yes</td>
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<tr>
<td>Meier et al. (67)</td>
<td>2009</td>
<td>17</td>
<td>2633</td>
<td>0.52</td>
<td>0.34 to 0.80</td>
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<tr>
<td>Navaneethan et al. (68)</td>
<td>2009</td>
<td>12</td>
<td>1652</td>
<td>0.46</td>
<td>0.26 to 0.82</td>
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<td>Yes</td>
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<tr>
<td>Trivedi et al. (71)</td>
<td>2010</td>
<td>10</td>
<td>1090</td>
<td>0.57</td>
<td>0.38 to 0.85</td>
<td>No</td>
<td>No</td>
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<tr>
<td>Zoungas et al. (69)</td>
<td>2009</td>
<td>23</td>
<td>3563</td>
<td>0.62</td>
<td>0.45 to 0.86</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

\(^{a}\) Relative Risk (RR) or Odds Ratio (OR)

\(^{b}\) Adjusted for baseline differences

We recommend volume expansion with either isotonic sodium chloride or sodium bicarbonate solutions, rather than no volume expansion, in patients at increased risk for CIN (1A).

KDIGO and ERBP 2012
How to prevent CIN?

Assessment of the individual risk for CIN

Minimisation of this risk

Volume expansion

Pharmacological prevention?

Non-pharmacological preconditioning?

Clinical practice
PREVENTION OF RADIOGRAPHIC-CONTRAST-AGENT-INDUCED REDUCTIONS IN RENAL FUNCTION BY ACETYLCYSTEINE

MARTIN TEPEL, M.D., MARCUS VAN DER GIET, M.D., CAROLA SCHWARZFELD, ULF LAUFER, M.D., DIETER LIERMANN, M.D., AND WALTER ZIDEK, M.D.

- **83 patients**
  ser.creat ~ 2,4 mg/dl
  enhanced CT

- **NaCl 0.45 % i-v**
  12h before → 12h after
  ± NAC 600 mg 2x/j
  J-1 and J0

Clinical trials comparing NAC with placebo for CIN prevention

15 positive vs 21 negative studies

… 17 meta-analyses with conflicting conclusions!

"We suggest using oral NAC, together with i.v. isotonic crystalloids, in patients at increased risk of CIN (2D)"

KDIGO

"We suggest using oral N-acytely-cysteine (NAC) only in patients who receive appropriate fluid and salt loading (2D). We recommend not using oral NAC as the only method for prevention of CIN (1D)"

ERBP
Prevention of Contrast-Induced AKI: A Review of Published Trials and the Design of the Prevention of Serious Adverse Events following Angiography (PRESERVE) Trial

2 x 2 factorial design
- bicarbonate vs saline
- NAC vs placebo

90-day composite end-point (death or dialysis or persistant decline in kidney function)

> 8 000 participants, USA and Australia

Impact of a High Loading Dose of Atorvastatin on Contrast-Induced Acute Kidney Injury

Atorvastatin 80 mg within 24 h before contrast vs placebo + Bicarbonate and NAC in all patients

\[\text{CI AKI} (\%)\]

\[
\begin{align*}
\text{Control group} & : 17.8\% \\
\text{Atorvastatin group} & : 4.5\% \\
37/208 & \quad 9/202 \\
\end{align*}
\]

OR = 0.22; 95% CI = 0.07-0.69
\[p = 0.005\]
Does ascorbic acid protect against contrast induced acute kidney injury in patients undergoing coronary angiography – a systematic review with meta-analysis of randomized controlled trials

Umar Sadat, MD, PhD1, Ammara Usman, MB BS, MBA2, Jonathan H. Gillard, MD, FRCR3, Jonathan R. Boyle, MD, FRCS1

1 Department of Surgery, Cambridge University Hospitals NHS Foundation Trust, Cambridge, United Kingdom
2 Department of Internal Medicine, Cardiovascular Medicine Division, Cavalry Hospital, Lahore, Pakistan
3 University Department of Radiology, University of Cambridge, Cambridge, United Kingdom

9 RCTs, 1536 patients

33% less risk of CIN among patients receiving ascorbic acid
How to prevent CIN?

Assessment of the individual risk for CIN

Minimisation of this risk

Volume expansion

Pharmacological prevention?

Non-pharmacological preconditioning?

Clinical practice
Preconditioning: 4 cycles of alternating 5' inflation and 5' deflation of a standard BP cuff to the individual SBP + 50 mmHg, within 1 before coronaryography

50 pts vs 50 controls

S creat > 1.4 mg/dl or eGFR < 60
Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium

CHARLES E. MURRY, B.S., ROBERT B. JENNINGS, M.D., AND KEITH A. REIMER, M.D., PH.D.

Circulation 74, No. 5, 1124–1136, 1986.

Regional Ischemic ‘Preconditioning’ Protects Remote Virgin Myocardium From Subsequent Sustained Coronary Occlusion

Przyklenk K, Circulation 1993; 87: 893
Ischemic Preconditioning in the Animal Kidney, a Systematic Review and Meta-Analysis

Effect of IPC on serum creatinine

(Weever KE, PlosOne 2012;7: e32296)
What is the molecular mechanism(s) behind ischemic preconditioning?

- inducible NO *(Park KM, 2003)*?
- adenosine *(Wever KE, 2011)*?
- hypoxia inducible factor 1 α *(Malfoud, 2012)*?
- ....
Ultrasound Prevents Renal Ischemia-Reperfusion Injury by Stimulating the Splenic Cholinergic Anti-Inflammatory Pathway

ABSTRACT

AKI affects both quality of life and health care costs and is an independent risk factor for mortality. At present, there are few effective treatment options for AKI. Here, we describe a nonpharmacologic, noninvasive, ultrasound-based method to prevent renal ischemia-reperfusion injury in mice, which is a model for human AKI. We exposed anesthetized mice to an ultrasound protocol 24 hours before renal ischemia. After 24 hours of reperfusion, ultrasound-treated mice exhibited preserved kidney morphology and function compared with sham-treated mice. Ultrasound exposure before renal ischemia reduced the accumulation of CD11b+Ly6G^{high} neutrophils and CD11b+F4/80^{high} myeloid cells in kidney tissue. Furthermore, splenectomy and adoptive transfer studies revealed that the spleen and CD4^{+} T cells mediated the protective effects of ultrasound. Last, blockade or genetic deficiency of the α7 nicotinic acetylcholine receptor abrogated the protective effect of ultrasound, suggesting the involvement of the cholinergic anti-inflammatory pathway. Taken together, these results suggest that an ultrasound-based treatment could have therapeutic potential for the prevention of AKI, possibly by stimulating a splenic anti-inflammatory pathway.

Ultrasonic Stimulation of the Cholinergic Anti-Inflammatory Pathway for Renal Protection

Jean-Michel Hougardy,∗† Claude Sadis,†‡ and Alain Le Moine∗†
How to prevent CIN?

Assessment of the individual risk for CIN
Minimisation of this risk
Volume expansion
Pharmacological prevention?
Non-pharmacological preconditioning?
Clinical practice
Conclusions

1. Among at-risk patients (age >75; suspicion of CKD; diabetes, congestive heart failure etc ...) check eGFR

2. If eGFR < 40:
   - discontinuation diuretics, NSAID
   - i-v saline 1 ml/kg/h for 24 h
   - use the lowest dose of contrast
“Renalism”: Inappropriately Low Rates of Coronary Angiography in Elderly Individuals with Renal Insufficiency

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Abstract. Higher risk patients (including the elderly) receive more conservative therapy for cardiovascular diseases, even though the relative benefits of therapy tend to be greater. The perceived risk of radiocontrast-associated nephrotoxicity may influence the provision of coronary angiography and subsequent revascularization, especially among individuals with chronic kidney disease (CKD). The aim of this study was to determine whether there is excessive variation in the provision of coronary angiography after acute myocardial infarction on the basis of the presence of CKD and whether there is an association between angiography and mortality. Elderly (age 65 to 89 yr) individuals with acute myocardial infarction from the Cooperative Cardiovascular Project were classified by the presence or absence of CKD (defined as a baseline serum creatinine of 1.5 to 5.0 mg/dl). In CKD patients, the propensity to undergo coronary angiography was determined and the effect of coronary angiography on mortality was estimated using multivariable logistic regression and stratification. Mortality was significantly higher with CKD (52.6 versus 26.4%). Fewer patients with CKD underwent coronary angiography (25.2 versus 46.8%) despite the observation that a similar proportion of patients were deemed appropriate for angiography by standard, published criteria. When limiting the analysis to CKD patients who are considered appropriate, the multivariable estimate of the odds of death associated with coronary angiography was 0.58 (95% confidence interval, 0.50 to 0.67). With adjustment using propensity scores, the odds ratio averaged across propensity score quintiles was 0.62 (95% confidence interval, 0.54 to 0.70). Results were qualitatively similar when patients were stratified by CKD stage IV (estimated GFR <30 ml/min per 1.73 m²). There is a large relative decrease in utilization of coronary angiography among patients with CKD. Alteration in practice because of an aversion to the risk of radiocontrast-associated nephrotoxicity (“renalism”) is inappropriate, even if the true relative benefit of invasive strategies is a fraction of what is estimated here.
Impact of Iso-Osmolar Versus Low-Osmolar Contrast Agents on Contrast-Induced Nephropathy and Tissue Reperfusion in Unselected Patients With ST-Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention (From the Contrast Media and Nephrotoxicity Following Primary Angioplasty for Acute Myocardial Infarction [CONTRAST-AMI] Trial)

![Graph showing incidence of CI-AKI at 72h (%) for different groups of patients](image)

(Bolognese L, Am J Cardiol 2012; 190:67)
Renal Function-Based Contrast Dosing to Define Safe Limits of Radiographic Contrast Media in Patients Undergoing Percutaneous Coronary Interventions

A

Incidence of Contrast induced Nephropathy

- % of Patients with Contrast induced Nephropathy
- CV/CCC < 2: 1%
- CV/CCC 2-2.9: 2%
- CV/CCC ≥ 3: 7%
- P < 0.0001

B

Incidence of Nephropathy requiring Dialysis

- % of Patients with Nephropathy requiring Dialysis
- CV/CCC < 2: 0.1%
- CV/CCC 2-2.9: 0.3%
- CV/CCC ≥ 3: 0.7%
- P < 0.0001

(Gurm H, J Am Coll Cardiol 2011; 58:907)