Preventing Renal Complications from the Use of Contrast Agents

Focus on at-risk patients.

By Tracey L. King, MSN, RN, CCRN, and Mary Lou Sole, PhD, RN, CCRN, FAAN

J\n\n\nue Martinez, a 46-year-old, is admitted to the critical care unit from the ED with a four-day history of intermittent pain in the chest, left arm, and back associated with exertion, especially walking. The chest pain is across the precordium, moderate in intensity (4 on a scale of 0 to 10), and relieved by rest.

On admission, Mr. Martinez reports radiation of pain into his back and arms, as well as some diaphoresis. His blood pressure was 255/156 mmHg with a regular pulse of 120 beats per minute. Electrocardiography reveals ST-segment depressions consistent with myocardial ischemia. Initial testing of electrolytes shows that they’re within normal limits. The blood urea nitrogen level is 23 mg/dL; creatinine, 3.7 mg/dL; and glucose, 232 mg/dL. Serum enzyme levels are normal as well, except for a mildly elevated troponin-T level, at 0.21 ng/mL. The complete blood count is normal.

Mr. Martinez reports a history of type 2 diabetes and hypertension. He has been previously diagnosed with renal insufficiency and was told a year ago that he should never undergo diagnostic tests requiring administration of IV contrast agents.

Mr. Martinez takes atenolol (Tenormin) and an unspecified diuretic for management of hypertension.

Mr. Martinez’s chest pain was relieved by IV nitroglycerin and β-blocker therapy (IV metoprolol [Lopressor] 5 mg every five minutes for three doses). His atenolol is discontinued, the IV metoprolol is replaced by an oral dose, and he is started on aspirin, clopidogrel (Plavix), IV heparin (Calciparine), oral β-blocker therapy, and amlodipine (Norvasc) for hypertension. Cardiac catheterization is scheduled after a blood test establishes a baseline creatinine level of 3.7 mg/dL.

One day after admission, Mr. Martinez is taken to the cardiac catheterization laboratory. To prevent radiocontrast-induced acute renal failure, several therapies are initiated. On the day before the scheduled procedure, N-acetylcysteine 600 mg had been started twice daily and hydration initiated with IV 0.45% saline to keep urine output greater than 150 mL/hr. During the procedure a low-osmolality contrast agent is used, and the amount of contrast volume is closely monitored and minimized. Serum electrolytes, blood pressure, and heart rate are closely monitored before, during, and after cardic catheterization.

Juan Martinez, a 46-year-old, is admitted to the critical care unit from the ED with a four-day history of intermittent pain in the chest, left arm, and back associated with exertion, especially walking. The chest pain is across the precordium, moderate in intensity (4 on a scale of 0 to 10), and relieved by rest.

On admission, Mr. Martinez reports radiation of pain into his back and arms, as well as some diaphoresis. His blood pressure was 255/156 mmHg with a regular pulse of 120 beats per minute. Electrocardiography reveals ST-segment depressions consistent with myocardial ischemia. Initial testing of electrolytes shows that they’re within normal limits. The blood urea nitrogen level is 23 mg/dL; creatinine, 3.7 mg/dL; and glucose, 232 mg/dL. Serum enzyme levels are normal as well, except for a mildly elevated troponin-T level, at 0.21 ng/mL. The complete blood count is normal.

Mr. Martinez reports a history of type 2 diabetes and hypertension. He has been previously diagnosed with renal insufficiency and was told a year ago that he should never undergo diagnostic tests requiring administration of IV contrast agents.

Mr. Martinez takes atenolol (Tenormin) and an unspecified diuretic for management of hypertension.

Mr. Martinez’s chest pain was relieved by IV nitroglycerin and β-blocker therapy (IV metoprolol [Lopressor] 5 mg every five minutes for three doses). His atenolol is discontinued, the IV metoprolol is replaced by an oral dose, and he is started on aspirin, clopidogrel (Plavix), IV heparin (Calciparine), oral β-blocker therapy, and amlodipine (Norvasc) for hypertension. Cardiac catheterization is scheduled after a blood test establishes a baseline creatinine level of 3.7 mg/dL.

One day after admission, Mr. Martinez is taken to the cardiac catheterization laboratory. To prevent radiocontrast-induced acute renal failure, several therapies are initiated. On the day before the scheduled procedure, N-acetylcysteine 600 mg had been started twice daily and hydration initiated with IV 0.45% saline to keep urine output greater than 150 mL/hr. During the procedure a low-osmolality contrast agent is used, and the amount of contrast volume is closely monitored and minimized. Serum electrolytes, blood pressure, and heart rate are closely monitored before, during, and after cardiac catheterization.

Figure 1. Pathophysiology of Radiocontrast-Induced Nephropathy

<table>
<thead>
<tr>
<th>Contrast agent</th>
<th>Shunting of blood from renal medulla to cortex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhibition of renal prostaglandins</td>
<td>vasoconstriction and renal blood flow</td>
</tr>
<tr>
<td>Red blood cell deformability</td>
<td>blood viscosity medullary ischemia</td>
</tr>
<tr>
<td>Altered glomerular permeability and selectivity</td>
<td>Direct toxicity of contrast media on renal tubular cells</td>
</tr>
<tr>
<td>Renal Tubular obstruction</td>
<td>Immunologic injury</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>free radicals oxidative stress</td>
</tr>
</tbody>
</table>

ACUTE RENAL FAILURE

Tracey L. King is director of medical care management at NCH Healthcare System in Naples, FL, and a doctoral student at University of Central Florida School of Nursing in Orlando, FL, where Mary Lou Sole is a professor. Contact author, Tracey L. King: tracey.king@nchmd.org.
The cardiac catheterization reveals that Mr. Martinez has diffuse disease of the right coronary artery and the left circumflex system. His left anterior descending coronary artery shows mild disease. He undergoes, with good results, percutaneous placement of multiple stents to the circumflex artery. His right coronary artery continues to have multiple areas of stenosis, for which a cardiothoracic surgeon recommends medical treatment. In light of Mr. Martinez’s baseline creatinine level of 3.7 mg/dL, the surgeon believes that if he were to undergo coronary artery bypass grafting (CABG) surgery, he would develop renal failure requiring hemodialysis. The surgeon notes that Mr. Martinez would be a high-risk candidate for CABG surgery or further percutaneous intervention if medical therapy fails.

After cardiac catheterization, Mr. Martinez’s creatinine level rises to 5.3 mg/dL. He is hydrated over the next 12 hours, and his creatinine level returns to baseline. He is discharged seven days after the cardiac catheterization, free of pain and normotensive.

**THE PROBLEM**

Radiocontrast-induced nephropathy (RCN) is an under-recognized adverse effect of cardiac catheterization or of any procedure that uses therapeutic or diagnostic dyes. Now the third leading cause of hospital-acquired acute renal failure, RCN is defined as a serum creatinine increase of more than 25% from baseline, or an absolute increase of greater than 0.5 mg/dL, within 48 hours of exposure to contrast agents (and when no other causes are apparent). RCN is associated with serious illness and death, increased hospital lengths of stay, and end-stage renal disease. Once a patient develops RCN, the risk of dying increases. A 1996 study of 16,248 patients undergoing percutaneous coronary interventions reported a hospital mortality rate of 34% among those who developed RCN requiring dialysis (the mortality rate was 7% among patients without renal failure). Identifying at-risk patients before exposure to any contrast media is the best option for prevention of RCN.

**Pathophysiology.** Blood flows through the kidneys at a rate of 1,200 mL/min, making the kidneys susceptible to injury if blood flow is diminished. Prevention of renal ischemia depends on increasing mean blood flow and balancing supply and demand. Under normal conditions, the medulla’s environment is hypoxic. Stressors such as dehydration or decreased renal blood flow cause an increase in hypoxia in the renal medullary cells.

Radiocontrast agents induce vasoconstriction in the renal medulla and shunt blood flow from the medulla, thus increasing ischemia. This leads to inhibition of renal prostaglandins, causing vasoconstriction and a further decrease in renal blood flow. This in turn causes red blood cell deformity, increased blood viscosity, and further medullary hypoxia. At this point, glomerular selectivity...
and permeability are altered by renal tubular obstruction and an increase in renal interstitial pressure; this leaves the renal tubules vulnerable to the toxic effects of contrast agents. Figure 1, page 72AA, depicts the pathophysiological events contributing to radiocontrast-induced renal failure.

**Risk factors** for the development of RCN include chronic renal insufficiency, diabetes, large amounts of contrast agents administered, dehydration, advanced age, systemic hypertension, concomitant administration of nephrotoxic drugs, multiple myeloma, congestive heart failure, and liver disease. Of these, the three most significant are preexisting renal insufficiency, diabetes mellitus, and the amount of contrast used. Many patients who require percutaneous cardiac interventions have these risk factors, making them a higher risk for developing RCN. In-depth medical history and baseline blood tests assist in identifying those who may be at risk.

**PREVENTION**

Those determined to be at risk for RCN can be treated prophylactically; aggressive preprocedural hydration is the only therapy definitively shown to decrease the incidence of acute renal failure in at-risk patients. Important preventive measures beyond the avoidance of volume depletion prior to the procedure include using nonionic low-osmolality contrast agents and minimizing contrast volume, withholding nephrotoxic drugs, administering N-acetylcysteine 600 mg twice a day, and performing any procedures requiring the administration of contrast agents at least five days apart. A summary of preventive strategies is shown in Table 1, page 72CC.

**Hydrate aggressively.** One of the most important measures to prevent RCN is the maintenance of a high urinary output before, during, and after coronary intervention. A urinary flow rate of more than 150 mL/hr is believed to prevent the crystallization of dye in renal tubules. There has been, however, a debate as to whether hypotonic or isotonic solutions should be used to prevent RCN. To maintain a urinary output of greater than 150 mL/hr, an infusion of a saline solution is initiated 12 hours before a contrast agent is given and continued for 12 hours afterward. Use contrast agents judiciously. The amount and type of contrast agent administered is important in preventing RCN. The amount given to an at-risk patient should be minimal and the contrast load should be reduced. Many facilities have instituted the use of flow sheets that track a patient’s exposure to contrast agents, including the total amount administered. These flow sheets can be used to make sure procedures involving the administration of contrast agents are separated. Examples of the nonionic, low osmolality contrast agents include iohexol (Omnipaque), iopamidol (Isovue), and ioversol (Optiray).

**Avoid nephrotoxic drugs.** In the at-risk patient, nephrotoxic drugs should be withheld prior to any contrast-containing procedure. Common nephrotoxic agents include nonsteroidal antiinflammatory drugs and angiotensin-converting enzyme inhibitors.

**Administer N-acetylcysteine.** The administration of N-acetylcysteine 600 mg twice a day, starting the day of the procedure and continuing for 48 hours after the procedure, is another preventive measure, although no multicenter, randomized trial has been conducted to confirm this.
conducted that supports anecdotal evidence of its benefits. It’s believed that the antioxidant properties of acetylcysteine improve the survival of renal medullary cells under oxidative stress.8

**What doesn’t work?** Diuresis by administering a saline infusion with furosemide (Lasix) and mannitol (Osmirol) has been found to increase the incidence of acute renal failure (as compared with diuresis with a saline-only infusion).9 Renal-dose dopamine has also proven ineffective and even harmful as a prevention strategy.10 And despite promising initial studies pointing to fenoldopam as a potentially effective therapy, larger recent studies have failed to support these hopes.7 ▼

**REFERENCES**


