



Interventional Procedures: Best Practice to Avoid Complications

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Technological advances have allowed treatment of patients using interventional radiological imaging including the performance of multiple procedures in almost any vessel, eg, angioplasty, stenting, embolization, and coilings. Patients undergoing any procedure are at risk for complications because of contrast media, radiation exposure, vessel injury, and prolonged time in one position during the procedure. Diagnostic-only procedures minimize use of contrast medium and radiation time, and generally take about one hour to complete. On the other hand, interventional procedures can take several hours and require larger volumes of contrast medium and radiation, as well as increased time lying supine on a procedure table. This article will discuss several potential and known risks associated with interventional procedures, how to monitor for these risks, and evidence-based measures to prevent or minimize their occurrence.

Keywords: interventional procedures, contrast induced nephropathy, radiation injury, closure devices.

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ADVANCES IN TECHNOLOGY have allowed us to treat patients with vascular disease using interventional and minimally invasive procedures versus open surgical procedures. Physicians are better able to visualize arterial and venous bloodflow and measure vascular pressures, enabling optimal treatment of these patients. Treatment options include interventional procedures such as thrombectomy, angioplasty, stenting, embolization, or placement of coils. The angiogram, however, may identify that surgery is the best approach for managing the patient's vascular disease. This practice change has drastically improved the ability to provide quick and competent care to patients requiring elective or emergent procedures to improve blood flow to blocked arteries. The quick restoration of perfusion minimizes ischemic time, which can prevent tissue damage and cell death. Any major artery or vein in the human body can be visualized and evaluated. The physician can intervene via percutaneous approach by femoral, radial, and brachial arteries for arterial procedures and subclavian or femoral veins for venous procedures.

There are also known risks and complications associated with this advanced and increasing use of percutaneous procedures. Although the technology is constantly improving, physicians are required to complete intensive training for invasive procedures. Although every member of the team exercises precautions to minimize risks, complications can still occur. An awareness of the risks before the procedure and use of evidence-based initiatives to prevent complications during the procedure and post-procedural period can minimize and/or prevent the occurrence of complications.

Contrast-induced nephropathy (CIN), radiation injury, and bleeding from arterial access sites are known risks associated with interventional procedures, whether diagnostic or interventional. Diagnostic-only procedures minimize use of contrast medium and radiation time, and generally take about one hour to complete. Interventional procedures can take several hours and require larger volumes of contrast medium and radiation, as well as increased time lying supine on a procedure table. This article will discuss several potential and known risks associated with interventional procedures, how to monitor for these risks, and evidence-based measures to prevent or minimize their occurrence.

Procedure Statistics

It is difficult to gain accurate information related to the number of diagnostic or interventional procedures

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conducted annually because of the expanding number of specialties performing these procedures. Interventional procedures can be performed by specially trained and certified neurologists, cardiologists, radiologists, and surgeons in a variety of settings. The number of cardiac procedures alone performed in 2000 included 1.3 million diagnostic cardiac catheterizations and 561,000 cardiac interventions.¹

Contrast-Induced Nephropathy

Contrast-induced nephropathy (CIN) is defined as an increase in baseline serum creatinine of 0.5 mg/dL or greater than 25% from baseline 48 hours after exposure to contrast in the absence of other medical causes. Serum creatinine can increase for 3 to 5 days after exposure to contrast, and may not be identified if patients develop renal insufficiency or failure 3 to 7 days post exposure. Serum creatinine levels may rise and peak for 48 to 72 hours post procedure, but generally return to baseline in two weeks.²⁻⁴

CIN occurs in less than 2% of the population undergoing cardiac or peripheral procedures requiring injection of contrast medium.⁵ It can occur in 12% to 26% of patients with preexisting renal disease or diabetes who have not received adequate pre and postprocedure hydration.² CIN can also occur in 20% to 30% of patients with preexisting renal or cardiac disease, congestive heart failure (CHF), or diabetes. It is the third leading cause of hospital-acquired acute renal failure and is associated with increased morbidity and mortality,² contributing to 10% to 12% of hospital-acquired acute renal failure.^{2,6} Fewer than 1% of patients who develop CIN require dialysis; however, developing CIN can increase mortality during hospital admissions by 22.9%.⁷

Cardiovascular disease is the number one cause of death in patients with chronic renal insufficiency (CRI).⁸ Diabetes, ischemic heart disease, hypertension, age, and elevated LDL levels all contribute to renal insufficiency. Other risk factors include hypovolemia, heart failure (ejection fraction [EF] < 35%), liver disease, multiple myeloma, nephrotoxic medication administration, NSAIDs, and total volume of contrast used during the procedure.⁸⁻¹⁰ Patients with end-stage renal failure (ESRF) have a 5 to 20 times higher chance of developing coronary artery disease (CAD). Chances of developing CAD can occur in as many as 38% of patients who have mild renal disease, and as many as 67% of patients with end-stage renal disease (ESRD) who are receiving dialysis therapy.^{1,2,5}

The contrast medium injected to visualize blood vessels during procedures is based on ionic or nonionic molecular structure and osmolality. Early ionic, high osmolar formulas required two osmotically active particles to deliver three iodine molecules, and had higher osmolality at 2,000 mOsm/L.¹ Formulas used since the 1980s

tend to be nonionic, requiring one osmotically active particle to deliver three iodine molecules, with an osmolality of 600 to 900 mOsm/L. A more recent nonionic formula requiring one osmotically charged particle to deliver six iodine molecules is more iso-osmolar at 300 mOsm/L.^{1,3,9,11-13} The higher the ratio of iodine molecules to particle charges, the better the x-ray image.⁵ Osmolality refers to the viscosity or thickness of liquid, and normal blood viscosity is 275 to 300 mOsm/L. The thinner viscosity of the low or iso-osmolar contrasts makes it easier for them to pass through the kidneys, possibly preventing and/or minimizing damage from contrast exposure.

Although the exact mechanism of CIN is not fully understood, the majority of literature points to inflammation and cell destruction in the renal vasculature. CIN occurs because of contact of injected contrast medium with the epithelial cells lining the renal nephrons and tubules. This contact leads to inflammation, cell dysfunction, and death. The development of CIN can be associated with risk factors such as preexisting renal function, amount of contrast used during a procedure, and intravenous (IV) fluid volumes infused pre and post procedure. Exposure of renal vasculature to contrast initially causes vasodilatation, then vasoconstriction. The vasoconstriction phase lasts longer, leading to hypoperfusion and ischemia to renal nephrons and tubules. Ischemic and dysfunctional endothelial cells lining the tubules release cell debris as they die, clogging the tubules and preventing tubular function. Cell dysfunction and death activates mediators such as endothelin, adenosine, and oxygen-free radicals, which are vasoconstrictive agents that further perpetuate cellular ischemia.^{1,5,14}

Several scales can be used to identify patients at risk for developing CIN. Most of the scales include measures of age, hypotension, CHF, preexisting renal disease, diabetes, EF < 35%, anemia, contrast volume, and glomerular filtration rate (GFR). Glomerular filtration rate is a calculated value based on age, height, weight, race, and serum creatinine. If GFR is < 60, pre and posthydration strategies should be initiated.^{2,5,10} Hydration volume loading and maintenance doses are based on weight as well as other components of the scale. Giving the full loading volume, or initial bolus volume should be reconsidered if patients have a cardiac or heart failure history because they may not tolerate the fluid load.

Although we strive to use evidence-based research to guide our practice, the evidence is inconclusive regarding best practice for preventive strategies for CIN. The literature is in agreement that using hydration pre and post exposure helps to decrease the occurrence of CIN. However, there is no consensus on fluid type or volume to use. The most common fluids referenced are 0.9 normal saline and 5% dextrose in water with 3 amps of

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