

Association for Radiologic & Imaging Nursing

CLINICAL PRACTICE GUIDELINE

Contrast Induced Nephrotoxicity

Overview

Renal failure due to contrast media has been reported to be the third most common cause of in hospital renal failure, after hypotension and surgery. Transient renal effects are seen after intravascular contrast and may be due to renal hemodynamic changes or direct tubular toxicity of the contrast media. Patients with normal renal function are at extremely low risk for contrast-induced nephrotoxicity (CIN).

The clinical course of contrast-induced nephrotoxicity depends on the patient's baseline renal function, co-existing risk factors, degree of hydration, and (perhaps) dose of contrast media. Serum creatinine begins to rise within the first 24 hours, peaks within 96 hours (4 days), and usually returns to baseline within 7-10 days. It is rare for patients to require temporary or permanent dialysis.

Target Audience

Radiology nurses, radiology technologists, radiologists, radiology administrators, radiology residents & fellows, medical students, other healthcare providers.

Risk Factors for CIN

Per the American College of Radiology Contrast Manual, the risk factors have been developed by "consensus" and include:

- Diabetes mellitus
- Dehydration
- Cardiovascular disease
- Diuretic use
- Advanced age
- Multiple myeloma
- Hypertension
- Hyperuricemia
- Multiple iodinated contrast medium doses

(ACR, 2013)

Nursing Implications

- Screen for risk factors in the patient population receiving intravenous contrast:
 - Pre-existing renal insufficiency (serum creatinine ≥ 1.5 mg/dl and serum glomerular filtration rate of < 60)
 - Low osmolality contrast media (LOCM) are generally less toxic than high osmolality contrast media (HOCM) in patients with underlying renal insufficiency. The exact mechanism for this is unclear.

- Patients with renal insufficiency who require intermittent or occasional dialysis are at substantial risk for contrast-associated nephrotoxicity, with further permanent worsening of their renal functioning. Alternate noncontrast imaging studies should be considered.
- Diabetes Mellitus (DM)
 - Patients with DM who are taking metformin or drugs containing metformin fall into a special category – see Clinical Practice Guideline on Metformin Therapy and Lactic Acidosis Risk.
- o Dehydration and/or use of diuretics
- o At highest risk: Patients with DM and renal insufficiency and dehydration
- Cardiovascular disease
- Advanced age (\geq 70 yrs)
 - Nonionic LOCM are less nephrotoxic in this population than ionic HOCM.
- Prevention of Contrast-Induced Nephotoxiticy:
 - Obtain a baseline serum creatinine and glomerular filtration rate in the following patients:
 - History of kidney disease including transplant and tumor
 - LOCM or Iso-osmolality contrast media (IOCM) are suggested for patients with renal insufficiency.
 - Family history of renal failure
 - DM treated with insulin or other prescribed hypoglycemic agent
 - Paraproteinemia syndromes (e.g., multiple myeloma)
 - Collagen vascular disease (e.g., lupus, scleroderma)
 - Medication use that includes one or more of the following:
 - Metformin or medications containing metformin
 - Nonsteroidal anti-inflammatory drugs
 - Antibiotics particularly the nephrotoxic aminoglycosides
 - BUN may help in determining hydration status but cannot reliably evaluate renal function.
 - If renal dysfunction is identified, the referring physician should be made aware of alternate imaging approaches. If no alternatives are available, a recommendation to extend the intervals between contrast exams and reduce contrast dose should be made, and renal function should be followed closely.
 - Hydration can be initiated to prevent contrast-associated nephrotoxicity. In the dehydrated state, renal blood flow and glomerular filtration rate (GFR) are decreased, and the effects of contrast media on these parameters are accentuated. There is a theoretical concern for prolonged tubular exposure to contrast media because of low tubular flow rates.
 - Patients who are not able to take oral hydration can receive intravenous hydration with 0.45% or 0.9% saline at 100 ml/hr for 6-12 hours prior to and 4-12 hours after administration of contrast.
 - The addition of pre- and post-treatment with parenteral or oral sodium bicarbonate can have a protective effect against contrast nephropathy.
 - The use of N-acetylcysteine for renal protection is based on the hypothesis that contrast nephropathy is due to renal vasoconstriction and free radical release (N-acetylcysteine has both vasodilating and antioxidant properties). Studies are mixed in their support of this method.
 - Chronic renal dialysis patients with end stage renal disease (ESRD) do not need immediate dialysis unless there is an underlying cardiac

dysfunction or very large volumes of contrast are used. Contrast can have a deleterious direct cardiotoxic effect on a damaged or diseased heart.

- Limit the dose of contrast used and choose LOCM or IOCM if there is a risk of adverse effects of hypertonicity.
- Contrast is not protein bound and has a low molecular weight so that it is readily cleared by dialysis.

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