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Contrast nephropathy

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Contrast Nephropathy

Elizabeth Messer, M.D.

Overview:

Contrast nephropathy is a form of acute kidney injury that occurs after the exposure to contrast media.

Pathogenesis:

The pathogenesis is not well understood. There are two main theories. The first theory is that contrast induces renal vasoconstriction causing medullary hypoxia leading to acute tubular necrosis (ATN). The other theory is that the ATN is a direct result of the cytotoxic effects of the contrast agent itself. Although contrast nephropathy is an ATN injury, it often can be clinically differentiated from ischemic ATN as it tends to be associated with a fractional excretion of sodium (FENa) $<1\%$ and less oliguria.

Epidemiology:

The overall risk for contrast nephropathy is low (negligible in patients with no risk factors but up to 10-20 percent in high risk patients). The greatest risk factors are in patients with CKD, diabetic nephropathy with renal insufficiency, and heart failure with reduced renal perfusion. High levels of contrast that are hyperosmolar also increase risk.

Clinical Features:

Although the renal injury seems to occur within minutes of the contrast agents, symptoms appear 24 to 48 hours later. Oliguria can occur but is uncommon. The main indication of injury is a rise in the serum creatinine. This will then decline around 3-7 days. You can also see other electrolyte disturbances associated with acute kidney injury such as hyperkalemia, hyperphosphatemia, and metabolic acidosis. A urinalysis can show muddy brown granular or epithelial casts (not red cell or white cell casts). Urine dipstick can also be positive for protein however this is commonly a false positive result from the contrast agent for the first 24 hours. There are no radiographic features specifically seen.

Diagnosis:

Diagnosis is made clinically after other causes of AKI are ruled out and injury is in the setting of recent contrast exposure in the last 24-48 hours. Basic laboratory values including electrolytes and creatinine are drawn. Urine studies are performed including urine sodium and creatinine to allow the calculation of a FENa. Ultrasound and biopsy are not recommended unless there is question of the diagnosis or the classical clinical course is not followed.

Differential:

The differential diagnosis is broad and includes ischemic ATN, acute interstitial nephritis, renal atheroemboli, and prerenal disease.

Prognosis/Treatment:

Contrast nephropathy is usually a mild disease that resolves by three to seven days. During this time supportive care should be provided to maintain fluid and electrolyte balance. Dialysis is rarely required nor should it be routine practice to perform dialysis immediately after a contrast study unless hemodialysis access is already established (for another reason) prior to the study. Prophylactic hemofiltration and hemodialysis have been tested with the theory of removing the contrast compound. Overall studies have shown no benefit from this and it is also expensive with significant risks to the patient.

Prevention:

There is no good evidence for how best prevent contrast nephropathy. The first preventative method is to avoid contrast by performing an MRI, a CT without contrast, or an ultrasound. (Of note, gadolinium based MRI should be avoided if glomerular filtration rate is $<30\text{ml/min/1.73M}^2$) as it can lead to nephrogenic systemic fibrosis). If contrast must be used the contrasts that are least osmolar have the lowest incidents of injury and should be used in the smallest volumes possible. At the same time, repeated studies should be spaced greater than 48 to 72 hours apart. Other mechanisms include avoiding prerenal states such as volume depletion or renal vasoconstriction with NSAID. Mannitol and diuretics may worsen AKI through volume depletion. Hydration is important in patients who can tolerate it both prior to and post contrast exposure. The main fluid is intravenous isotonic saline though some evidence shows that sodium bicarbonate may be just as beneficial. Both are thought to provide benefit by volume expansion and urine pH control. Intravenous hydration has been shown to be more effective than oral hydration in the prevention of contrast nephropathy. Oral n-acetylcysteine has also been used with the hypothesis that it works based on antioxidant and vasodilator properties. IV acetylcysteine however should be avoided as it has the risk of an anaphylactic reaction changing the risk benefit equation. Dosing for n-acetylcysteine is not well established in children.

References:

Rudnick, Michael. Pathogenesis, clinical features, and diagnosis of contrast-induced nephropathy. In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. (Accessed on July 7, 2015.)

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