**Mucomyst and Contrast nephropathy**

**Contrast nephropathy**: Mechanism not entirely understood: 2 theories - vasoconstriction induced by contrast-mediated alteration in nitric oxide or endothelin production vs. direct toxic effect of contrast on the renal tubules, caused by free radical generation. Small rise in plasma creatinine (averaging 0.2 mg/dl) is a common occurrence post contrast administration. Less common with newer nonionic contrast agents. MR agents have little to no renal toxicity.

**Risk factors** for more serious renal impairment post contrast include:
- Underlying renal insufficiency, w/ creatinine > 1.5
- DM nephropathy
- Advanced CHF or other causes of decreased renal perfusion
- High contrast dose (debatable) - dose dependence is debatable and poorly studied - evidence that diabetics may be at risk with as little as 20-30 ml of contrast.
- Multiple myelome (debatable) - dehydration promotes intratubular precipitation of light chains and increases risk of ARF in MM pts receiving contrast. May also be interaction between light chains and dye - unclear.

**Incidence** of contrast induced renal failure (rise of cr > 50% above baseline or more than 1 mg/dl) is negligible with normal renal function (even if pt has DM).
- 4-11% with cr 1.5 to 4.0
- 9-38% with cr 1.5 to 4.0 PLUS DM
- > 50% if cr > 4.0

**Clinical Course** - Renal failure starts immediately after administration. Almost always self limited. May require transient dialysis, especially if baseline cr > 4.0. Persistent renal failure rare and only described with severe CRI (cr usually 8 or greater at baseline)

**Prevention**
- Avoid contrast when possible
- Use lowest dose contrast
- Avoid dehydration and NSAIDS prior to contrast (both can cause vasoconstriction)
- Prehydrate if not contraindicated.
- NO support for peri-procedure mannitol, ANP, dopamine, prophylactic dialysis. Diuretics may make contrast induced renal failure worse. (2 randomized trials looking at pts with moderate CRI and the above interventions around the time of cardiac cath: KI 1994: 45(1), and NEJM 1994: 331(21))

**Mucomyst** (N-acetylcysteine). Thiol containing antioxidant, used prior to contrast administration in pts at risk. May act by scavenging free radicals, increasing the vasodilatory effect of nitric oxide, an dby increasing the expression of NO synthetase.
- Tepel, NEJM 2000: 343(3): Prospective trial of 83 patients with CRI who were going to undergo CT with contrast (nonionic). Randomized to receive 600 mg PO BID of mucomyst with 1/2NS (1 ml/kg/hr for 12 hrs prior to procedure) vs placebo and saline. 10 of 83 pts (12%) total had cr rise of 0.5 mg/dl, 1 of 41 (2%) in mucomyst group and 9 of 42 (21%) in control group (p<0.01). Only 30% of the patients in each group had diabetes. Unclear what the CT was for (elective, CT of chest or abdomen). More patients in the intervention group on an ACE (20 vs 14%)
- Briguori, JACC 2002; 40(2): 183 pts with mod CRI, undergoing cath or peripheral angiography, randomized to NAC+hydration vs hydration alone. NO protective effect of NAC. Lower dose contrast seemed to cause less nephrotoxicity.
- Diaz-Sandoval, Am J Card 2002 89(3) APART trial (Acetylcysteine to prevent angiography related renal tissue injury): 54 pts with CRI (1.6) randomly assinged to NAC or placebo prior to elective cardiac cath. At 48 hrs, ARF less likely in NAC tx’d group (8 vs 45%, p=0.005)

**Summary**: Paucity of data to support mucomyst. However, very benign treatment that MAY help prevent ARF in higher risk patients, in whom there are no other proven preventatives. Worth giving, but high quality data is needed.