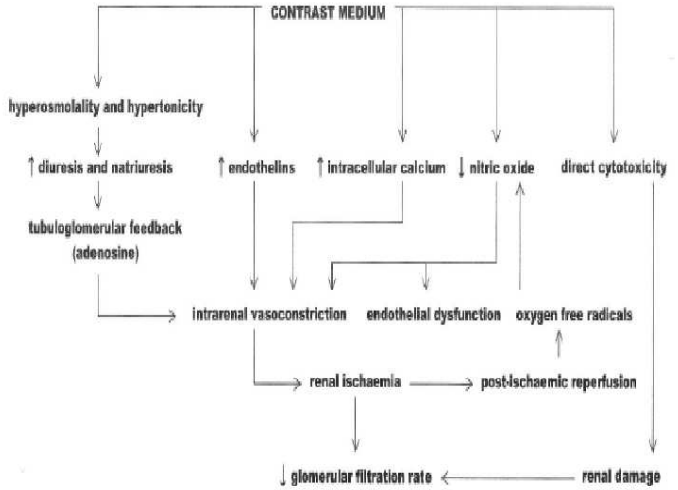
Contrast-induced nephropathy [created by Paul Young 02/10/07]

general

defined as increase of 44umol/L from baseline or 25% rise in creatinine after exposure to contrast

Nash et al found it to be 3rd most common cause of renal failure in hospitalised patients

pathogenesis



risk factors

1. pre-existing renal disease
- risk only 8% if less than 135 umol/L in one study and none had a rise of greater than 89umol/L
2. diabetes mellitus
3. age >75 years
4. periprocedural volume depletion
5. heart failure
6. nephrosis
7. cirrhosis
8. hypertension
9. proteinuria
10. NSAIDS and other nephrotoxins
11. high doses of contrast

Mehran et al have devised a score which can be used to predict the risk of an acute decline in kidney function after percutaneous coronary intervention

strategies for prevention

if GFR is less than 50, particularly if there are risk factors then consideration should be given to alternative imaging approaches

1. fluid administration
- generally recommended to give prehydration
- optimal duration and type of fluid not well defined
- several small trials exist comparing iv saline with oral fluid, shorter regimens and 0.45% saline
- iv 0.9% saline a 1ml/kg/hr for 24 hours beginning 2-12 hours before administration of contrast medium

2. contrast medium
- metanalysis of several RCTs show that low osmolality contrast media are lower risk
- use lowest dose possible to complete the procedure

3. N-acetylcysteine
- most commonly given 600mg orally every 12 hours for 4 doses beginning before administration of contrast medium
- multiple RCTs and meta-analyses performed with inconsistent trial results for unknown reasons
- may decrease release of creatinine from skeletal muscle than effecting renal function directly

4. intravenous sodium bicarbonate
- proposed that alkalinisation of renal tubular fluid might be beneficial by reducing the levels of pH-dependent free radicals
- dose in trial 154mEq HCO3 at 3ml/kg/hr before contrast & then 1ml/kg/hr for 6 hours afterwards
- one trial which showed benefit from bicarb; however, study was terminated early despite the fact the timing of interim analysis and the stopping values were not prespecified and the p value for the difference in event rates (P=0.02) was higher than is standard for stopping a trial early

6. other approaches
(i) diuretics
- lead to similar or higher rates of nephropathy
(ii) various vasodilators
- dopamine, fenoldopam, atrial natriuretic peptides, calcium blockers, PGE2 & a non-specific endothelin receptor antagonist are not beneficial
(iii) captopril
- small trial showed a benefit from captopril but confirmation is needed
(iv) ascorbic acid
- one trial showed apparent benefit from this; however, baseline renal function was worse in placebo group
(v) theophylline and aminophylline
- metanalysis shows lower risk but significant heterogeneity between studies
(vi) haemodialysis or haemofiltration
- appears to reduce mortality when used prophylactically; however, results need confirmation and intervention is very labour intensive

5. cease nephrotoxins prior
- NB: metformin should be ceased because of risk of lactic acidosis if nephrotoxicity develops