Acute renal failure induced by contrast medium: steps towards prevention

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BMJ 2006;333;539-540
doi:10.1136/bmj.38943.401852.80

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(Accepted 3 August 2006)

doi 10.1136/bmj.38946.616551.BE1

Lesson of the week

Acute renal failure induced by contrast medium: steps towards prevention

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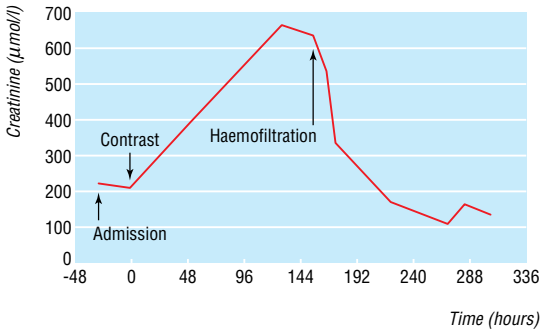
Acute renal failure is a well known complication of procedures that involve iodinated contrast media.[1] Despite this, contrast medium induced nephropathy accounts for about 12% of all cases of hospital acquired renal failure.[2] Prevention of this type of nephropathy is crucial as it is associated with prolonged hospital stay, risk of permanent renal impairment, and a more than fivefold increase in mortality.[3 4] We report a case of acute renal failure in a woman with chronic renal disease who was investigated for metastatic breast cancer with contrast enhanced computed tomography. This case shows the importance of carrying out a risk assessment for contrast medium induced nephropathy before using procedures that involve iodinated contrast media.

Case report

An 81 year old woman with type II diabetes was admitted after a hypoglycaemic episode. She had a four week history of non-productive cough, which failed to resolve with antibiotics. She had been treated with surgery and radiotherapy for breast cancer 13 years previously. She also had stage 3 kidney disease and hypertension.

Breast examination was normal but there were signs of a left sided pleural effusion. Chest x ray showed a possible left sided coin lesion and pleural effusion. Her white blood cell count was 5.6×10^9/litre, C reactive protein was 15 mg/litre, and the pleural aspirate was an exudate containing 38 g/litre protein and 0.5×10^9/litre white blood cells. Plasma creatinine was 224 µmol/litre, which was not very different from her preadmission value. In view of the history of breast cancer, contrast enhanced computed tomography of the chest and abdomen was performed. No abnormalities were detected in the lungs, but a lesion, suggestive of metastasis, was detected in the left adrenal.



Serum creatinine values during admission in a woman with contrast medium induced acute renal failure

Over the next two days, the patient became increasingly nauseous and anorexic. Creatinine rose to 392 µmol/litre (figure). Despite supportive treatment for acute renal failure she became anuric, her blood pressure began to drop, and creatinine rose to 664 µmol/litre. She was transferred to the intensive care unit, where haemofiltration was started. Her renal function started to recover over the next few days and her condition began to improve.

Discussion

Nephropathy induced by contrast medium is defined as an impairment in renal function that occurs within 72 hours of giving contrast medium.[5] This impairment is characterised by an increase in serum creatinine of at least 44 µmol/litre or 25% above the baseline. Creatinine typically peaks three to five days after contrast administration and returns to baseline values within two weeks. Renal replacement therapy is needed in a minority of patients, and in-hospital mortality may be as high as 62% in these cases.[4] No specific treatment is available for contrast medium induced nephropathy and management is supportive.

Risk assessment for renal failure must be performed before procedures involving the administration of contrast media

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BMJ 2006;333:539–40

Risk factors for contrast medium induced nephropathy

- Pre-existing renal insufficiency
- Diabetes mellitus
- Age >75 years
- Concurrent use of nephrotoxic drugs (non-steroidal anti-inflammatory drugs, aminoglycosides)
- Dehydration
- Hypotension
- Heart failure
- Cirrhosis
- Nephrotic syndrome
- Hypertension

The box shows the key risk factors for developing contrast medium induced nephropathy.[6] Underlying impairment of renal function, particularly if secondary to diabetes, is the most important risk factor, and the associated incidence of contrast nephropathy is 12-27%. This compares with an incidence of 0-5% in patients with normal renal function.[5] Serum creatinine should ideally be measured in all patients before giving contrast medium, but particularly if a history of renal impairment or diabetes is present. Serum creatinine, however, is a poor indicator of renal function. The best measure of renal function—and therefore risk of contrast nephropathy—is the glomerular filtration rate. The rate can be estimated from serum creatinine using the Cockroft-Gault formula or the modification of diet in renal disease formula.[7] It is especially useful to estimate the glomerular filtration rate in patients with borderline serum creatinine—a value of less than 60 ml/min/1.73 m^2 predicts increased risk of contrast nephropathy.[8]

If risk factors are present then an alternative imaging technique should be used. If however, administration of contrast medium is deemed necessary, then several steps can be taken to reduce risk. The most important of these is adequate hydration. The most effective regimen has not been defined, but one study showed that intravenous hydration with normal saline at a rate of 1 ml/kg/hour for 24 hours, beginning 12 hours before administration of the contrast medium, was associated with a significantly lower incidence of contrast nephropathy (3.7%) compared with unrestricted oral hydration (34.6%).[9] Isotonic sodium bicarbonate has been shown to be superior to normal saline in preventing contrast nephropathy, but further evidence is needed to support this intervention.[10] Nephrotoxic drugs increase the risk of developing contrast nephropathy, and it is recommended that these drugs be stopped at least 24 hours before investigation.[11] In addition, use of the minimum volume of low osmolar or iso-osmolar contrast media is associated with a lower incidence of contrast nephropathy compared with high osmolar media.[12–14] Oral N-acetylcysteine has been used as a prophylactic treatment for contrast nephropathy, but there is currently insufficient evidence to advocate its use.[15 16] Daily monitoring of renal function after contrast administration in high risk patients enables early diagnosis and treatment of renal failure.

We did not perform risk assessment for the development of contrast nephropathy in our patient. We did not provide the relevant clinical details when we ordered computed tomography and the necessary precautions were not taken. In retrospect, this was a serious oversight. Contrast nephropathy is highly underestimated. A retrospective study of 89 patients undergoing contrast enhanced computed tomography showed that none had creatinine concentrations documented on the request card. Consequently, steps to reduce risk were taken in only two of 10 patients at risk.[17]

This case emphasises the role of the clinician in preventing contrast medium induced nephropathy. A risk assessment must be performed and details of this should be provided to the radiology department. If it is deemed necessary to give contrast medium to patients with risk factors, measures must be taken to prevent renal failure from developing.

Contributors: RM and WW had the original idea. RM wrote the paper. RM and KH searched the literature. KH and WW cared for the patient. WW revised the final manuscript and is guarantor.

Funding: None.

Competing interests: None declared.

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(Accepted 21 July 2006)

doi 10.1136/bmj.38943.401852.80