

Contrast nephropathy: can we see the whole picture

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Introduction

Renal protection has remained a highly topical subject for years. The focus of any prophylactic strategy remains *adequate hydration*. It has become clear that the *liberal hydration policy* practised for years by most anaesthetists has been based on flawed logic and has been, to a large extent, inappropriate and even harmful to patients.¹

We see an increased usage of invasive radiology techniques for both diagnostic and therapeutic procedures. The radiocontrast media used in these procedures may cause a reversible form of acute renal failure.

Frequency

Peri-operative renal failure has an incidence of 0,1 - 50%, and a mortality rate of ~ 50%, a statistic which has not changed in 50 years. Local figures are not available, but approximately 10 million contrast studies take place each year in the United States. The incidence of contrast nephropathy (CN) averages 3% in patients with a serum creatinine of less than 1,5 mg/dl, or a creatinine clearance of greater than 50 ml/minute. This incidence rises to 13% when the creatinine is greater than 1,5 mg/dl or the clearance is less than 50 ml/min. This translates to between 300 000 and 1,3 million cases of CN per year. It would appear that CN is the third leading cause of acute renal failure and accounts for 10% of hospital-acquired acute renal failure.

Why does administration of contrast lead to CN?

The kidney has a unique anatomical and physiological make-up, which renders it particularly vulnerable to insult:

- The corticomedullary gradient. 90% of the blood flows through the cortex, but is responsible for only 10% of the metabolic work. Conversely, the medulla gets only 10% of the renal blood flow (RBF), but is responsible for 90% of the metabolic work. (Na⁺/K⁺ ATPase-dependent solute resorption; i.e. high O₂ requirements of active Na⁺ transport and counter-current mechanism.)
- This metabolically active medulla, operates in a chronically "hypoxic environment" with medullary PaO₂ ~ 10 - 20mmHg compared with cortical PaO₂ ~ 50 mmHg. Medullary blood supply is also viscous due to osmotic removal of water.

- The renal medullary vascular bed, the vasa recta, is composed of long vessels of small diameter. Blood flow is facilitated by maintenance of low viscosity. The medullary thick ascending loop (mTAL) cells are therefore particularly at risk in the face of hypotension and the concomitant reduction in RBF.

Definition of CN

This definition is problematic, due to the fact that there are multiple definitions in the literature. A working definition states that chronic nephropathy occurs when there is a rise of 25% in the serum creatinine when the baseline is less than 1,5 mg/dl, or an absolute rise of 1 mg when the baseline is greater than 1,5 mg/dl. This definition is relevant when contrast has been administered and where there is no alternative aetiology for the renal impairment.

Classification of radiocontrast agents^{2,3}

Iodinated radiocontrast agents are typically described in terms of structure (ionic and non-ionic monomers or dimers), and osmolality.

First generation agents

First generation agents are ionic monomers, i.e. they consist of a benzene ring and 3 I₂ atoms. These agents have an osmolality of ~ 1400 - 1800 mosmol/kg and are, therefore, hyperosmolar with respect to plasma (~ 290 mosmol/kg). ? Urografin

Second generation agents

Second generation agents (e.g. Iohexol (Omnipaque®); Iopamidol (Jopamiron®)) are non-ionic monomers with an osmolality less than that of the first generation agents (500 - 850 mosmol/kg). Although these agents are termed "low osmolality" agents, it must be noted that they are still hyperosmolar with respect to plasma.

Until recently, all available non-ionic agents were such "low osmolality" agents. In addition, there is an ionic low osmolar contrast agent (ioxaglate (Hexabrix®)). Although ionic, this intermediate agent has a low osmolality. This is because the I₂-containing organic molecule, being a dimer, is twice as large as the other molecules in the ionic group. There are therefore half the number of osmotically active particles per gram of I₂ in solution.²⁷

The newest non-ionic contrast agents are dimers (two benzene rings joined together as a single molecule) with an even lower osmolality. Iodixanol (Visipaque®), the first such agent, is iso-osmolal (~ 290 mosmol/kg) with respect to plasma.

The “iso-osmolal agents” have a lower osmolality than so called “low osmolal” second generation drugs. The nephrotoxic properties of these agents appear to vary, with low- and iso-osmolal agents being associated with a relatively decreased incidence of renal injury among high-risk patients.²

Pathogenesis

The mechanism of contrast induced acute tubular necrosis is not fully understood. Animal research supports two major theories:

- Renal vasoconstriction: Possibly mediated by decreased nitric oxide, increased or decreased endothelin, and/or increased adenosine; resulting in medullary hypoxaemia.
- Direct contrast-mediated cytotoxicity.

Additional contributors may include rheologic alterations, activation of the tubuloglomerular feedback mechanism, regional hypoxia, and production of reactive oxygen species.

Renal vasoconstriction

This occurs commonly and is believed to be due to a combination of contrast-induced release of vasoconstrictors, like endothelin and adenosine on the one hand, and a decrease in endogenous vasodilators like nitric oxide and prostaglandins on the other. Preliminary human studies suggest, however, that the role played by endothelin may not be significant. Wang et al showed that endothelin receptor antagonists failed to prevent contrast-induced renal failure.⁴

Weisberg et al showed that renal vasoconstriction alone correlates poorly with subsequent rise in plasma creatinine.⁵ Reduced medullary flow may, however, still be of primary importance. The vasoconstrictor ioholamate decreased medullary blood flow in rats, but did not produce renal failure until it was administered with blockers of nitric oxide and prostaglandin.⁶

Patients with diabetes mellitus and heart failure have an increased risk of developing contrast-induced renal failure. This is possibly due to impaired nitric oxide generation.

This reduction in medullary blood flow may be compounded by increased blood viscosity, as is seen with contrast media, in particular high- and low-osmolal preparations. As mentioned previously, the medullary vasa recta are long, thin blood vessels, and flow through them depends on maintenance of a low viscosity. Increased viscosity may also enhance tubular interstitial pressure, which further reduces medullary blood flow.

Direct cytotoxicity

The second theory implicates the ability of the contrast to cause tubular injury, either directly, or via release of oxygen-free

radicals. Antioxidant activity may explain the apparent benefit of acetylcysteine, and some animal models suggest that decreased activity of protective antioxidant enzymes may explain the enhanced risk with hypovolaemia.

Both of these mechanisms probably play a role. In a study by Katholi et al, for example, administration of a non-ionic, low-osmolality contrast agent led to an 18% decrease in creatinine clearance and increased adenosine excretion.⁷ Concurrent use of theophylline, an adenosine receptor antagonist, prevented the fall in creatinine clearance. In comparison, an ionic high-osmolality contrast agent produced a 42% decrease in creatinine clearance that was only partially corrected by theophylline and was associated with a more prolonged increase in adenosine excretion, suggesting concurrent tubular injury.

Risk factors

- Pre-existing renal insufficiency with [plasma creatinine] > 1,5 mg/dl (132 µmol/l) or glomerular filtration rate (G)FR < 60 ml/min per 1,73 m². (This is not usually measured clinically).
- Diabetic nephropathy.
- Any cause of reduced renal perfusion, for example advanced heart failure or hypovolaemia.
- High total dose of contrast agent, or multiple contrast studies within a 72 hour period. Some studies show that low doses of contrast variably defined as less than 70 ml, less than 125 ml, or less than 5 ml/kg (to a maximum of 300 ml), divided by the plasma [creatinine], are less likely to cause renal dysfunction. Manske et al showed, however, that diabetics with [plasma creatinine] > 5 mg/dl (440 µmol/l) may be at risk from as little as 20 - 30 ml of contrast.⁸
- Percutaneous coronary intervention: contrast, atheroemboli, myocardial ischaemia-related hypoperfusion.
- Multiple myeloma is associated with < 1,5 % incidence of renal failure if a modern contrast agent is used. Contributing factors include volume depletion (promotes the intratubular precipitation of filtered light chains) and a possible interaction between light chains and the contrast agent.

Clinical presentation

Radiocontrast-induced renal failure is usually mild, transient and nonoliguric. It begins within 12 – 24 hours of contrast administration, and usually recovers within 3 - 5 days. Occasionally, patients may develop more significant renal dysfunction with creatinine peaks > 5 mg/dl (440 µmol/l). This usually occurs if baseline plasma creatinine > 4 mg/dl (352 µmol/l), and may require dialysis. Persistent renal failure has been described and occurs primarily in patients with pre-existing advanced underlying disease, particularly in diabetics.

McCullough et al reviewed more than 1 800 consecutive patients who underwent coronary intervention with contrast.⁹ The overall incidence of acute renal failure was 14,4%, and 0,8 % of these required dialysis. The need for dialysis significantly increased in-hospital mortality, and 2 year survival was only 19%.

Diagnosis

Diagnosis is made by exclusion of other causes, and is based on the characteristic rise in [plasma creatinine] beginning within the first 12 - 24 hours. In the differential diagnosis, consider conditions like ischaemic acute tubular necrosis and acute interstitial nephritis, particularly if an additional insult like sepsis, hypotension, or medication exposure was present, and renal atheroemboli.

Any diffusely atherosclerotic patient undergoing arteriography is at risk of developing renal atheroemboli, and the following features help distinguish this event from CN:

- The presence of other embolic lesions (e.g. on the toes) or livedo reticularis;
- Transient eosinophilia and hypocomplementaemia;
- Delayed onset renal failure (days to weeks post-procedure);
- Protracted course with frequently little or no recovery of renal function.

Prevention of radiocontrast media induced acute renal failure³

There is no specific treatment for contrast-induced acute renal failure. If it develops, it should be managed as you would any cause of acute tubular necrosis, focusing on fluid maintenance and electrolyte balance. The best management of the nephropathy is, therefore, prevention.

Suggested methods for reducing the incidence of this disease entity include:

- When clinically possible, it may be worth considering alternative diagnostic modalities such as ultrasonography, magnetic resonance (MR) imaging with gadolinium, or computerised tomography (CT) without radiocontrast agents, particularly in high risk patients.
- The use of low or iso-osmolal, nonionic contrast agents, whenever possible.
- Limit both the total dose and the number of doses administered in a 48 - 72 hour period.
- CO₂ can be used as an alternative contrast agent in certain high risk patients.
- Limit contributory risk factors like hypovolaemia, NSAIDs and certain antibiotics.
- Many specific prevention strategies have been investigated. These include intravenous saline, sodium bicarbonate, acetylcysteine (antioxidant), prophylactic haemofiltration/haemodialysis, aminophylline, vasodilators, diuretics, statins and ascorbic acid.

Type of contrast agent

The risk of contrast nephropathy appears to be a function of both the agent structure (ionic vs. non-ionic compounds) and the agent osmolality relative to plasma (hyperosmolal [1400 - 1 800 mosmol/kg]; low osmolal [500 - 850 mosmol/kg] or iso-osmolal [~290 mosmol/kg]). **The incidence appears to be**

lower with agents that are non-ionic and agents that are non-hyperosmolar.

There are few direct comparisons between various agents, however, and many questions remain unanswered. The data suggests the following:

- The primary benefit of nonionic contrast agents, whether low or iso-osmolal, is seen in high-risk patients (e.g. plasma [creatinine] \geq 1.5 mg/dl (132 mmol/l) or GFR < 60 ml/min per 1.73m²), particularly if the patient is diabetic.
- Iodixanol is, at present, the only non-ionic, iso-osmolal agent available. It appears to be superior to some of the low osmolal agents in its ability to reduce the risk of contrast nephropathy in high-risk patients (e.g. diabetics with renal insufficiency). It is, however, expensive, and further investigation is required to establish the extent and consistency of this perceived benefit.

Carbon dioxide has been used successfully as an alternative contrast agent (alone or in combination with small doses of iodinated contrast) in high risk patients. It is, however, neurotoxic and should not be used for cerebrovascular imaging. All access to the cerebral circulation should be limited (e.g. right-to-left intracardiac shunts), and its use should be limited to imaging below the diaphragm.

Contrast-enhanced

Unlike the iodinated contrast agents used in CT imaging and angiography, those used in MR imaging are chelates of gadolinium, which have been found to be less nephrotoxic if used in small doses. It is the opinion of some that these paramagnetic contrast agents are an alternative in digital subtraction angiography or interventional procedures, particularly in patients with renal insufficiency or iodinated contrast allergy. Emerging data suggest that if given in doses greater than 0,3 mmol/kg, nephrotoxicity may occur. These agents do, however, appear to be safer and may be preferable in high risk patients requiring vascular imaging. At the recommended dose of < 0,3 mmol/kg, however, diagnostic image quality is diminished and the modality is not supported by the radiological fraternity.

Specific strategies

Hydration

Optimal hydration is a vital component of any renal protection strategy. It would appear that intravenous hydration is superior to oral hydration. The optimal fluid choice, infusion rate & volume are unclear. Solutions that have been investigated include isotonic normal N-saline; ½ N-saline, and isotonic sodium bicarbonate.

The following must be taken into consideration with fluid selection and rate of administration:

- Recent changes in overall peri-operative hydration policies;
- The patient's ability to tolerate a fluid load (e.g. may precipitate failure in individuals with reduced left ventricular function)

- The ability to tolerate alkalinisation; and
- The degree of underlying risk for nephropathy.

Mueller et al enrolled 1 620 patients in a prospective randomised controlled trial and compared the effects of a 1 ml/kg/hr infusion of either isotonic N-saline or ½ N-saline, from the morning of the procedure.¹¹ The incidence of "contrast nephropathy" (defined as an increased [creatinine] of ≥ 0.5 mg/dl (44 mmol/l) within 48 hours) was 0,7% in the isotonic saline group, and 2% in the ½ N-Saline group.

Alkalinisation may protect against free radical injury. For this reason Merten et al compared isotonic N-saline (154 meq/l) versus sodium bicarbonate.¹² This study showed marked benefit in those given sodium bicarbonate, but study weaknesses necessitate the need for further investigation to fully define its role.

Acetylcysteine

Solmuco[®] is a thiol compound with antioxidant and vasodilatory properties. A theoretical mechanism of benefit in the prevention of CN, therefore, would include minimising contrast-induced vasoconstriction and oxygen-free radical generation. The results from clinical trials have been inconsistent, but the trend is suggestive of benefit.

Nallamotheu et al looked at data from twenty randomised trials (n = 2 195 patients).¹³ Acetylcysteine was associated with a 27% reduction in the risk of developing CN. The most commonly studied dose regimen for acetylcysteine prophylaxis is 600 mg orally twice daily. Two studies (Briguri et al and Marenzi et al) compared 600 mg and 1200 mg twice daily, and suggested slightly better outcomes with the higher dose.^{14,15}

Webb et al looked at patients with a mean baseline plasma creatinine of 1.6 mg/dl (140 μ mol/l).¹⁶ They found no benefit in therapy with 500 mg of intravenous acetylcysteine just prior to the procedure.

A study by Baker et al did demonstrate benefit.¹⁷ Intravenous acetylcysteine (150 mg/kg pre-procedure, and 50 mg/kg post-procedure, administered over four hours) was compared with isotonic saline (1 ml/kg/hr for 12 hours, pre- and post-contrast). Fewer patients (5%) in the acetylcysteine group developed acute renal failure, as compared with 20% in the control group. However, at the high doses used, 7% developed anaphylactoid reactions.

On the basis of conflicting data and a documented risk of anaphylactoid reactions, Rudnick et al **do not advocate the routine use of intravenous acetylcysteine for the prevention of contrast nephropathy.**³

Prophylactic haemofiltration and haemodialysis

This is based on the theory that removal of the inciting compound from the circulation might prevent contrast-induced acute renal failure.

Marenzi et al compared haemofiltration with intravenous saline in 114 high risk patients.¹⁸ These patients had chronic renal failure (mean [creatinine] 3 mg/dl [265 mmol/l]). The haemofiltration group showed less chance of [creatinine] rising > 25% above baseline, less chance of requiring dialysis and, lower in-hospital and 1-year mortalities. Study flaws make interpretation of these results difficult. **The routine clinical application of this technique is not warranted.**

Prophylactic haemodialysis has been advocated for both the removal of contrast and the prevention of volume overload. **This cannot be recommended in routine clinical practice.**

Aminophylline

Adenosine is a well known coronary and peripheral vasodilator. It has, however, been shown to vasodilate only isolated rings of renal vasculature, and generally to result in renal vasoconstriction. Aminophylline inhibits the adenosine receptor, thereby preventing vasoconstriction.

Many trials have looked at its potential renoprotective benefit, but results are conflicting. Bagshaw and Ghali conducted a meta-analysis.¹⁹ The theophylline group showed a marginal benefit. In contrast, concurrent administration of an adenosine agonist like the antiplatelet agent dipyridamole, may increase contrast toxicity.⁷

Katholi et al showed that theophylline prevented a fall in measured creatinine clearance post administration of a nonionic, low-osmolality contrast agent.⁷ The protective effect was only partial when an ionic high-osmolality contrast agent was used.

Vasodilators

It is unclear if pharmacologic inhibition of renal vasoconstriction will protect high risk patients from developing renal failure. The acute reduction in GFR induced by contrast agents may be theoretically minimised or prevented in some patients by the use of vasodilators. Various agents have been proposed as renal vasodilators. There is little evidence to support their routine use. Some examples are discussed below:

Dopamine

Numerous studies attest to the potential harmful effects of dopamine: arrhythmias; myocardial, peripheral vascular and gastrointestinal ischaemia; pulmonary hypertension; impaired hypoxic ventilatory response; reduced gastric motility; increased metabolic rate and weight loss; and endocrine and immune dysfunction. Its routine use is not warranted.

Fenoldopam

Fenoldopam is a selective DA₁ agonist with vasodilating properties in renal, mesenteric, coronary and cerebral beds. Its role in peri-operative renal protection is as yet undefined.

A prospective randomised trial, CONTRAST, assessed the effectiveness of fenoldopam in 315 chronic renal failure patients (half of which were diabetic) undergoing a cardiovascular procedure.²⁰ There was no reduction in the incidence of CN in the fenoldopam group (34% versus 30% with placebo). It has been proposed that direct intrarenal administration may be more beneficial.²¹

Nitroglycerine

Nitroglycerine has no specific renoprotective action.

Endothelin antagonists

The possible importance of endothelin-induced renal vasoconstriction led to the evaluation of a nonselective endothelin receptor.⁴ Compared with those assigned to placebo, a significantly higher percentage of patients who received active therapy developed CN (56% versus 29 %); this observation raises the possibility that endothelin may actually provide an intrinsic protective effect rather than contributing to the development of acute renal failure. Alternatively, selective endothelin receptor antagonists may be required to demonstrate prophylactic value in this setting.

ACE inhibitors

Benefits remain unproven.

α-agonists

Clonidine showed animal benefits in the 80s.

β-blockers

Benefits remain unproven.

Calcium channel blockers

Both ischaemic and toxic acute renal failure (ARF) show an accumulation of intracellular Ca⁺⁺ which blocks ATP production and cellular regeneration. These drugs have the potential to worsen renal function in unstable patients, as cardiac depression and vasodilation may further decrease RBF and GFR.

PGE2

PGE2 is a renal vasodilator (opposes action of TXA₂) and therefore improves RBF.

Diuretics

The theoretical renoprotective properties of diuretics include:

- Free radical scavenging;
- Decreased O₂ consumption of mTAL cells;
- tubular effects: diuresis, high tubular flow, prevention of obstruction, prevention of tubular swelling, inhibition of tubulo-

glomerulo feedback and, therefore, increased GFR.

- Haemodynamic effects: decreased renin release; direct arterial smooth muscle relaxation and increased PG synthesis. Leads to increased RBF, decreased renal vascular resistance (RVR) and re-establishment of the cortico-medullary gradient.

Mannitol

Has no benefit and has a significant complication rate.

Furosemide

Clinical evidence shows increased urine output but no difference in mortality or in the need for dialysis.

Diuretics may increase urine output in some cases of renal impairment. Brown et al (1981 and 1991) and numerous other studies dispute the proposal that maintenance of a high urine output has any influence on outcome.

Atrial natriuretic peptide (ANP)

ANP (anaritide) has shown benefit in animal models of contrast nephropathy.²⁴ Kurnik et al, however, observed no benefit.³⁰

Weisberg et al randomly assigned patients to receive either saline or one of three renal vasodilator/diuretic drugs: dopamine (at 2 µg/kg/min); mannitol (15 g/dl in ½ isotonic saline @ 100ml/hr); or ANP.²³ They showed benefit in the nondiabetic group, while there was an increased incidence in the diabetic group

Statins

Statins have the ability to improve endothelial function, reduce arterial stiffness (via improved endothelin-mediated vasodilatation), and reduce inflammation and oxidative stress. Khanal et al examined their potential and **they do not recommend routine statin use.**²⁵

Ascorbic acid

Ascorbic acid has been seen to improve renal damage in experimental models of ischaemic or toxic injury. Spargias et al examined its potential benefit.²⁶ Patients received 3 g of ascorbic acid at least 2 hours pre-procedure, and 2 g on the night and morning following the procedure. Further work is required to define the role of ascorbic acid.

Growth factors

Insulin-like growth factor and epidermal growth factor have shown a promising ability, in animal studies, to enhance renal cell regeneration and growth and, therefore, hasten recovery from acute renal failure.

In the future, modalities may include agents like iNOS (inducible Nitric Oxide Synthetase) and anti-ICAM (cellular adhesion molecules).

Fluid considerations

Fluid volume¹

Fluid therapy is clearly a mainstay of renal protection and yet the topic itself is highly controversial. Brandstrup et al, in 2006, reviewed the evidence guiding "standard" peri-operative fluid therapy and came to the following conclusions:

- The evaporative loss from the abdominal cavity is highly overestimated;
- The non-anatomical "third space" loss is based on flawed methodology and is probably non-existent;
- The fluid volume accumulated in traumatised tissue is probably very small.
- The volume preloading of neuraxial blockade is not effective and may cause postoperative fluid overload.

Many trials show that so-called "restricted fluid strategies" are associated with better outcome than the traditional liberal hydration protocols. Brandstrup et al state that, in view of their conclusions, these strategies are not evidence-based at all, and that restricted fluid administration is not restricted at all, but rather replaces only the small amount of fluid lost during surgery, thereby avoiding the overload resulting commonly from overestimation of perioperative losses.

Fluid type²⁸

The fluid most commonly used in the renoprotective trials showing benefit is 0.9% normal saline.

The following must be taken into consideration:

- "Normal" saline is not an ideal crystalloid solution. It is significantly *hypertonic* (osmolality = 308 mosmol/l) and has a very high chloride content (154 mmol/l).
- It has been shown that saline infusions of as little as 2 litres result in significant hyperchloraemic metabolic acidosis.
- There is also evidence that chloride loading may impair renal function (dose-dependent vasoconstriction and decreased GFR), and may interfere with coagulation.
- The significance of this acidosis is unclear, but volunteer studies display numerous adverse effects associated with chloride loading. No human data exist to show that this decreases survival.

These findings may mean that normal saline is not the ideal fluid for prevention of nephropathy. None of the available crystalloids, however, approximate the electrolyte content of plasma. This includes Ringer's lactate ($\text{Cl}^- = 115 \text{ mmol/l}$, $\text{Na}^+ = 131 \text{ mmol/l}$ and osmolality = 273 mosmol/l; measured osmolality due to incomplete ionisation of lactate salts = 255 mosmol/l). Perhaps the future will see better results with a more balanced salt solution being used to maintain hydration.

Summary and recommendations

Optimal prophylaxis for CN remains uncertain.

Patients with near normal renal function are at little risk and few precautions are necessary other than avoidance of volume depletion.

Patients at *increased risk* of nephropathy should adhere to the following:

- Alternative (non-contrast) diagnostic modalities where possible (e.g. ultrasonography, MRI, CT).
- Avoid high osmolal agents (1 400 to 1 800 mosmol/kg).
- Iso-osmolal agents (~ 290 mosmol/kg) are preferable to low osmolal agents (500 to 850 mosmol/kg).
- Use lower doses of contrast.
- Avoid repetitive, closely spaced studies (e.g. < 48 hrs apart).
- Avoid volume depletion and NSAIDs.
- If there are no contraindications to volume expansion, give isotonic intravenous fluids prior to, and for several hours after, contrast administration.
- The optimal type of fluid and timing of administration are not well established. However, since *isotonic saline* has been the hydration regimen used in the majority of studies showing benefit, it remains the fluid of choice

Suggested regimens

1. Isotonic saline administered at a rate of 1 ml/kg per hour, initiated at least 2 hours, and preferably 6 - 12 hours, prior to the procedure, and continuing for 6 - 12 hours after contrast administration. The duration of administration of fluid should be increased proportionately with more severe degrees of renal impairment

Data comparing *isotonic bicarbonate* to isotonic saline are limited.

2. The bicarbonate regimen may be preferable if there is not sufficient time for isotonic saline hydration prior to the procedure. A bolus of 3 ml/kg of isotonic bicarbonate is administered over the last hour before the procedure, and continued at a rate of 1 ml/kg for 6 hours post-procedure. This solution can be prepared by adding 150 meq of sodium bicarbonate (three 50 ml ampoules of 1 meq/ml sodium bicarbonate) to 850 ml of 5% dextrose in water.
3. Acetylcysteine, at a dose of 600 - 1 200 mg orally twice daily, administered the day before and the day of the procedure. Data are conflicting but it is worth using based on its potential for benefit, and low toxicity and cost.
4. Diuretics should be reserved for patients with volume overload. Mannitol should specifically be avoided.
5. Haemofiltration/haemodialysis are not recommended

Conclusion

Radiocontrast interventions are common practice. With the ageing population and their concomitant diseases, particularly type two diabetes mellitus, the possible occurrence of contrast-induced renal failure is a reality. As peri-operative physicians, we need to identify the patients at risk. The low risk patient may require little other than standard haemodynamic support, but the high risk patient will benefit from a knowledge of reno-protective strategies. With simple pharmacological manipulation, we will be able to alter the morbidity and mortality of an otherwise innocuous procedure.

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