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Contrast-induced nephropathy

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Interventional radiological procedures involving anaesthesia are generally increasing. Contrast-induced nephropathy (CIN), usually defined as an increase in serum creatinine of 44 μmol litre^{-1} (0.5 mg dl^{-1}) or a 25% increase from the baseline value 48 h after intravascular injection of contrast media, is a common and potentially serious complication of the use of iodinated contrast media in patients at risk of acute renal injury. It is an important cause of hospital-acquired renal failure, may be a difficult differential diagnosis and the incidence does not appear to have changed over the last few decades. In the general population, the incidence of CIN is estimated to be 1–2%. However, the risk for developing CIN may be as high as 50% in some patient subgroups, such as those with diabetes mellitus and pre-existing renal impairment. The impact of CIN on clinical outcomes has been evaluated most extensively in patients undergoing percutaneous coronary intervention where it is associated with increased mortality both in hospital and at 1 yr. As treatment is limited to supportive measures while awaiting the resolution of the renal impairment, emphasis needs to be directed at prevention.

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Contrast-induced nephropathy (CIN) is a well-documented phenomenon and now ranks third in the causes of hospital-acquired acute renal failure in the USA.[19 77] Diagnostic and therapeutic procedures requiring the use of i.v. contrast are increasing with many of these procedures being developed as alternatives to open surgery in elderly patients and those with significant co-morbidities. An estimated 75 million doses of contrast are administered yearly.[18] Many of these procedures such as endovascular stenting and peripheral angioplasty are performed in operating theatres and may require general/regional anaesthesia or sedation and analgesia for successful completion. Patients may also present to the operating theatre for their definitive surgical treatment shortly after exposure to high doses of i.v. contrast, for example, semi-emergency coronary artery surgery. Inevitably, anaesthetists are becoming more involved with the care of these individuals. Although CIN is a reasonably well-recognized and reviewed topic within the radiology and cardiology literature, the focus has been on patients in the ambulatory setting. The purpose of this article is primarily to alert the anaesthetist to the possibility of perioperative CIN in surgical patients. An overview of the current understanding of this condition, its pathophysiology, and some of the preventative measures that have been investigated will be provided. Some perioperative strategies will be proposed which may benefit these patients. Considering that the stress of surgery, disturbances of fluid balance, perioperative fasting, and administration of anaesthesia can adversely affect renal function, as perioperative physicians, anaesthetists could play a pivotal role in minimizing renal damage in this group of patients.

Definition

CIN is implied when there is a temporal link between deterioration of renal function and the administration of i.v. contrast, in the absence of any other aetiology. The widely quoted definition requires an absolute increase in serum creatinine of 44 μmol litre^{-1} or a relative increase of 25% from baseline.[10 71]

Incidence of CIN

Quoted figures are likely to underestimate the true extent of the problem given that serum creatinine inherently has limited sensitivity in detecting renal dysfunction and earlier studies used a more conservative criterion. However, for those without pre-existing renal impairment, figures range from 3.3% to 8%,[10 94] and increases to 12–26% for those with renal disease or diabetes mellitus.[33] The impairment tends to be non-oliguric and transient, with the peak serum creatinine usually occurring around day 3 and returning to normal in the majority of cases

within 2 weeks.[122] Some patients may develop more severe renal failure, with approximately 1% of this group requiring dialysis. Although small in number, the morbidity and mortality rates in this group are alarmingly high, with an in-hospital mortality rate of up to 30% and an 80% 2 yr mortality rate.[34 67]

The large discrepancy in the documented incidence of CIN is probably related to the variation in definitions, type and volume of contrast used, employment of any preventative measures, diverse combinations of patient risk factors, and, most importantly, what actually constitutes pre-existing renal impairment. Although small in relative terms, the absolute number of patients affected is not when considering the number of doses of contrast administered per annum.

Risk factors

Factors that have been identified to be associated with the development of CIN include: pre-existing renal impairment, diabetes mellitus, advanced age, peri-procedural intravascular depletion, congestive heart failure, volume and type of contrast administered, and concomitant use of other nephrotoxic drugs.[67 68 94] The proportional contribution of any one factor to the development of CIN has not been specifically investigated. However, attempts have been made to further stratify patients by assigning a weighting to each of the risk factors, resulting in the development of risk scores for those undergoing coronary angiography.[68]

A recent prospective analysis of 6773 patients undergoing coronary angiography revealed that both a low baseline haematocrit and a peri-procedural drop in haematocrit regardless of baseline were independent risk factors. Patients were analysed in quintiles of haematocrit level, with those below a level of 36.8% having the greatest incidence of CIN. Each further 3% decrease in haematocrit increased the odds of CIN by 30% or 26%, respectively, in those with or without baseline renal impairment.[80]

Pharmacology of contrast

Commercially available media are all tri-iodinated benzene derivatives and are classified according to their ionization, osmolarity, and structure. Ionic contrast media dissociate in water whereas their non-ionic counterparts do not, despite being water-soluble. The ratio of iodine to dissolved particles describes an important relationship between opacification and osmotoxicity of the agent, with higher ratios being more desirable. Agents have been classified into high-osmolar (ratio=1.5), low osmolar (ratio=3.0), and isotonic agents (ratio 6.0). In the 1990s, agents with dimeric molecules containing two benzene rings were produced, which possessed the desirable qualities of no ionicity, low osmolarity combined with increased iodine atoms per molecule, and greater water solubility, but they were of a higher viscosity compared with their monomeric predecessors. The increased viscosity, in turn, could potentially affect renal blood flow and tubular urine flow.[51] However, initial studies point to a lower incidence of CIN with the use of isotonic dimeric agents in high-risk patients.[4 17]

The kinetics of distribution of all contrast media is similar, with low lipophilicity, low plasma protein binding, and minimal biotransformation. The contrast particles quickly equilibrate across capillary membranes after i.v. injection, except in the brain with an intact blood–brain barrier.[90] They tend to remain extracellular with the exception of proximal renal tubular cells.[47] Movement of particles back into the circulation occurs, resulting in a bi-exponential decay in plasma iodine levels,[73] with an elimination half-time of about 2 h. Nearly, all the injected substance is cleared by the kidneys and excreted in the urine unchanged in those with normal renal function. However, hepatic metabolism, enterohepatic circulation, and biliary elimination are increased in those with renal impairment.[14] Contrast molecules are easily removed by dialysis as there is little protein binding.

Renal handling of contrast media

Contrast medium molecules are freely filtered and the concentration in the ultrafiltrate initially approaches that of plasma. As the filtrate proceeds along the tubules, variable amounts of water are reabsorbed, resulting in concentrations that can be 50–100 times that of plasma.[113] There is a very small amount of contrast media uptake into the proximal tubular cells where it forms conspicuous vacuoles, which may be detectable up to 28 days after administration.[23] Like other osmotic particles, contrast molecules in the tubular lumen reduce overall water reabsorption, leading, therefore, to an increase in intraluminal pressure and a decrease in the gradient for filtration from glomerular capillaries. This increase in sodium and water delivery to the distal portion of the tubules activates the 'tubuloglomerular feedback' mechanism that reduces the glomerular filtration rate (GFR) of the whole kidney. The raised renal interstitial pressure may contribute to the reduced GFR[118 119] and probably to renal medullary hypoxia by local compression of vasa recta.[88]

Pathophysiology of CIN

Although the pathogenesis of this condition is not fully understood, it is most likely the result of the prolonged vasoconstriction and impaired autoregulation induced by contrast media predisposing to medullary hypoxia, in combination with direct cytotoxicity (Table 1).[117] This may be further influenced by contributions from several systemic factors. Therefore, an appreciation of the factors affecting renal microcirculatory haemodynamics is pivotal to understanding the pathogenesis of CIN and the expected response to preventive measures. The reader is referred to reviews in this area for a more thorough understanding of

Table 1 Factors implicated in the pathogenesis of CIN

Exaggerated vasoconstriction
Adenosine
Endothelin
Tubuloglomerular feedback
Direct action of contrast on vascular smooth muscle
Reperfusion and free radical
Intrarenal pressure changes
Aggregration of RBC in medullary circulation
Direct tubular cell injury
Systemic factors
Hypovolaemia
Anaemia
Changes in blood rheology
Other renal toxins

the intricacy of this complex circulatory system under normal circumstances.[25 84] A brief overview of some of the factors governing the intrarenal haemodynamic balance will be presented focusing on those factors germane to understanding CIN.

The renal circulation is subjected to autoregulation which typically refers to maintenance of a constant total renal flow within a range of arterial perfusion pressure. However, there may be regional cortico-medullary differences in the extent to which autoregulation occurs resulting from the net effect of the many neural, hormonal, paracrine, and autocrine influences. Medullary autoregulation may be, in part, dependent on extracellular hydration status, where volume expansion may impair autoregulatory mechanisms.[66] Evidence suggests nitric oxide (NO) has a key role in renal autoregulation through its vasodilatory effects and inhibition of NO production would interfere with this process.[48] Vasopressin can cause vasoconstriction via V_1 receptors predominantly in the medulla.[75] The renal medulla is predisposed to low oxygen tension, resulting in the release of adenosine. This product of adenosine triphosphate metabolism is produced, among other places, in various microvascular beds in response to ischaemia. It produces vasodilatation via low affinity A_2 receptor activation which may increase blood flow within the medulla.[101] However, it is a potent vasoconstrictor when it activates the high affinity A_1 receptors reducing cortical and net renal blood flow.[89] Angiotensin II provides constriction of the juxtamedullary microcirculation.[83] Prostaglandins cause redistribution of blood flow from the superficial to the juxtamedullary cortex and are found in abundance in the renal medulla.[46 59] Endothelins are endogenous peptides that are potent vasoconstrictors,[57] and can cause decreases in renal blood flow and GFR, and constrict outer medullary descending vasa recta, thus predisposing the renal medulla to ischaemia.[12]

Therefore, it would seem that microcirculatory tone is regulated by the interplay of various influences that mediate vasoconstriction and vasodilatation. As a primary role of the kidney is to maintain volume, the balance of the vasculature tends towards a constrictive state in the presence of hypovolaemia. Unrecognized hypovolaemia often exists in patients undergoing contrast studies for a variety of reasons, including debilitation from underlying medical disease, repeated fasting, and third space fluid losses from surgical procedures. Contrast media administration results in diminution of renal blood flow, contributing to the decrease in glomerular filtration and ischaemia in the medulla, an area with a high metabolic requirement and remote from its blood supply. This added vasoconstrictive response occurring in an already contracted renal vasculature may be responsible for the development of renal dysfunction in CIN.

Contrast media-induced vasoconstriction may be a result of a direct effect on vascular smooth muscle,[50] changes in calcium physiology,[9] or from a local increase in adenosine[89] and endothelin[6] production. In the canine model, contrast media initially cause vasodilatation, followed by prolonged vasoconstriction.[9] It has also been shown to cause dose-dependent, reversible contractions in human, rabbit, and dog, but not in pig renal arteries.[91] There may also be regional differences in the vascular response to contrast media, with a greater reduction in flow to the outer medulla.[81] In animals, the diminution of renal blood flow may also be a consequence of impaired vasodilation secondary to impaired NO production. NO production is reduced in rat renal artery smooth muscle cells exposed to radiographic contrast,[92] and a decreased NO level is associated with a concomitant reduction in medullary blood flow in the rat.[2] Finally, contrast media can increase intrarenal pressure and thus diminish renal blood flow from physical compression, an observation which correlates with the osmolarity of the agent.[52]

In addition to the disturbance in renal haemodynamics with resultant regional hypoxia, other mechanisms contribute to the development of this condition. These include direct toxic effects on renal tubules by the contrast,[40] changes in blood rheology, generation of reactive oxygen species (ROS),[88] and possibly a low haematocrit.[80]

CIN is primarily a tubular disorder,[13 15] and the direct toxic effects of contrast media on cells have mainly been performed in cell lines displaying tubular phenotypes.[36] Observed effects on cells included cellular energy failure, a disruption of calcium homeostasis,[44] a disturbance of tubular cell polarity,[37] and apoptosis.[11 43]

Viscosity of blood flowing through the vasa recta is usually kept low to counteract the high resistance in these vessels. Contrast media have complex effects on blood rheology, including an increase in whole blood viscosity, a decrease in haematocrit, and a reduction in red cell deformability, producing an increased resistance to flow.[88]

ROS are endogenously produced particles that can function as extracellular signalling molecules and may be involved in the actions of vasoconstrictors.[22 97 99] Oxidative stress occurs when the amount of ROS exceeds the levels of antioxidants. This is thought to be increased in chronic renal failure[65] and diabetes,[98] known risk

factors for CIN. Some studies examining the use of antioxidants in attenuating CIN have yielded positive results (discussed later). As such, ROS has been implicated in the pathogenesis of CIN.[87]

Prophylactic measures

Hydration

Solomon and colleagues[102] demonstrated the benefits of hypotonic (0.45%) saline pre-hydration in reducing the risk of CIN in a group of patients with pre-existing renal insufficiency (Table 2). In that trial, the use of fluids alone was superior to fluids plus mannitol or furosemide. The observation that forced diuresis with furosemide and mannitol were no better than hydration alone was also demonstrated in the PRINCE study.[107] Indeed, the use of loop diuretics may actually exacerbate post-procedural renal function.[120] Normal saline has also been shown to be better than 0.45% saline, especially in women, diabetics, and patients receiving 250 ml or more of contrast.[74] The authors proposed that the sodium load is crucial for the protective effect as it enables more effective volume expansion and inhibition of rennin–angiotensin activity. A recent small prospective trial demonstrated that infusion of isotonic sodium bicarbonate was associated with a smaller increase in serum creatinine compared with isotonic saline, suggesting a benefit from alkalinization of the urine.[69] Evidence comparing i.v. and oral hydration is inconclusive.[112 116] However, hydration appears more beneficial if the fluid load is given before the procedure.[5] There are currently no studies of the optimal rate of pre-hydration, but most of the studies used a rate of 1–2 ml kg^{-1} h^{-1}, for 6–12 h before the procedure. There have been no reported complications from pre-hydration.

Table 2 Summary of prophylactic measures

Intervention	Level of evidence	Comments
Beneficial		
Hydration[102 107]	RCT	Beneficial, isotonic saline and sodium bicarbonate may be preferable
NAC[7 24 58 62 76 85]	Meta-analyses	Suggest overall reduction in incidence
Haemofiltration[64]	RCT	Beneficial in single study of chronic renal impaired patients
Equivocal		
Adenosine antagonist[8]	Meta-analyses	Meta-analysis showed a trend but no overall benefit
Calcium channel antagonists[55 78 96 103]	RCT's	Mixed results in small RCT's
Statins[54]	Retrospective analysis	
Not beneficial		
Diuretics[102 107 123]	RCT	Furosemide and mannitol lack beneficial effects and may even be harmful
Dopamine[31 38 49 124] and fenoldopam[108]	RCT	
Haemodialysis[61 106 121]	Small RCT's	No reduction in incidence

Diuretics

As mentioned earlier, diuretics and mannitol have no beneficial effects and may be harmful[102 107 123] and their use cannot be recommended for CIN prophylaxis.[28]

Antioxidants

N-Acetylcysteine is the acetylated form of the amino acid L-cysteine and has long been established clinically as a mucolytic and as a treatment for acetaminophen overdose. Being an abundant source of sulfhydryl groups, its metabolites are able to act as free radical scavengers.[53] This antioxidant property combined with a vasodilatory effect on the renal medullary circulation[29 41] has led to studies evaluating its effects on CIN. Owing to differences in patient selection, radiological procedures, dosages, type and volume of contrasts used, these trials have yielded inconsistent results. Although recent meta-analyses[7 24 58 62 76 85] have shown that *N*-acetylcysteine significantly reduced the incidence of CIN, particularly when used with pre-hydration, a number of the authors have cautioned against recommending *N*-acetylcysteine as universal prophylaxis before results from larger quality trials are available. Endovascular aortic aneurysm repair (EVAR) causes significant acute renal injury in most patients and a recent trial suggests that this is not attenuated by *N*-acetylcysteine.[70]

A single trial has demonstrated significant beneficial effects from the use of another antioxidant, ascorbic acid, in patients with renal insufficiency undergoing coronary angiography.[104] Although promising, further trials are required to validate these results.

Drugs acting on the renal circulation

Dopamine and fenoldopam

Studies of dopamine in the prevention of CIN are conflicting and most fail to show any benefit and undesirable side-effects such as tachycardia.[31 38 49 124] Fenoldopam mesylate is a selective dopamine$_1$ receptor agonist with no α_1 or β_1 effects and only mild α_2 antagonism.[79] Despite having favourable effects on renal haemodynamics, it failed to reduce the incidence of CIN.[108]

Calcium channel antagonists

Calcium channel antagonists have potential to prevent the adverse renal haemodynamic changes from hyperosmolar contrast media.[96] However, only one small trial demonstrated any value with their use,[78] whereas other studies showed no beneficial effects.[55 103]

Adenosine antagonists

Adenosine has a prominent role in the tubuloglomerular feedback mechanism[16 82 86 111] and has been implicated in the pathogenesis of CIN.[89] However, meta-analysis of clinical studies involving theophylline again showed heterogeneity of results, overall indicating a trend but no significant benefit of CIN.[8] Aminophylline did not demonstrate any benefit.[1 100]

Statins

Statins (hydroxymethylglutaryl co-enzyme A reductase inhibitors) have 'pleiotropic effects'[45] which encompass modification of endothelial function, plaque stability, thrombus formation, and inflammatory pathways. Preoperative statin therapy has been reported to be associated with improved postoperative outcomes.[42] Retrospective analysis demonstrated an association with a reduction in CIN in patients undergoing percutaneous coronary interventions.[54] Although side-effects are rare, larger prospective trials are required to delineate its role as prophylaxis for CIN. However, it is worth considering for patients who may gain from other beneficial effects of statins.

Physical measures

Prophylactic haemofiltration

Prophylactic haemofiltration in an ICU setting reduced the incidence of CIN, in hospital and cumulative 1 yr mortality in chronic renal failure patients undergoing coronary angiography when compared with hydration alone.[64] However it is invasive, inconvenient, labour, and equipment intensive, may only be cost-effective under very limited circumstances[56] and cannot be universally applied to all high-risk patients.

Haemodialysis

A number of studies have examined the effect of immediate post-procedural haemodialysis on the incidence of CIN in patients with chronic renal failure.[61 106 121] Although small in patient numbers, none of the studies has shown any benefits.

Given the number of proposed mechanisms for the genesis of CIN, it is not surprising that the multiple prophylactic measures that have been tried have produced equivocal or negative results. This may also reflect the quality of the studies, which are frequently small and, thus, underpowered and poorly designed. It is also not surprising that hydration is efficacious and diuresis is not. Normovolaemia can probably best influence the overall balance of renal haemodynamics by, among others, preventing the activation of tubuloglomerular feedback and of the rennin–angiotensin system. In contrast, the various other drugs target only a component of the control of renal blood flow and, therefore, are not sufficiently effective in swinging the balance.

Peri-procedural management

Identifying those at risk

The anaesthetist should be aware of patients who will receive contrast media as part of their operative procedure and those who have received contrast within the previous 10 days. Identifying those with risk factors for CIN should be helpful. Certain patient subgroups may be prone to develop perioperative renal dysfunction independent of the use of contrast agents, for example, those with sepsis. Even small decrements in renal function in these susceptible individuals could potentially increase morbidity and length of hospital stay. Recognition of CIN, along with identifying the patients at risk, is the first step towards minimizing the harm from contrast.

Serum creatinine has been the traditional marker to screen for baseline renal dysfunction but other factors such as age, gender, body mass, and ethnicity can also influence its level. Determination of GFR provides more accurate information, but it is cumbersome and expensive to measure and is not feasible for all patients undergoing radiological procedures. Estimation of GFR using equations such as the Cockcroft and Gault formula or the Modification of Diet in Renal Disease Study equation may be more useful clinically as they may identify mild to moderate decreases in GFR in patients with 'normal' creatinine levels. It would also help to stage the degree of renal impairment. Serum Cystatin C is a protein which is secreted by virtually all nucleated cells. It is not subject to the same confounding factors as creatinine and may be more sensitive in detecting reductions in GFR. There is evidence that Cystatin C increases earlier after contrast administration compared with serum creatinine, and therefore is potentially useful in early detection of CIN, especially in patients with short periods of hospitalization.[93]

Evaluation of fluid status

It is vitally important that hypovolaemia be avoided in those undergoing contrast studies. Review of the patient's history may reveal events such as repeated nil by mouth orders or reduced fluid intake that would predispose the patient to develop dehydration. It is difficult to establish clear, objective, clinically useful end-points for optimal hydration that are universally applicable. Short of invasive monitoring, clinical indicators such as normal heart rate, normotension with warm peripheries, and adequate urine output must be used. Right heart catherization should be considered for haemodynamic monitoring in patients with uncompensated heart failure.[105]

Drug–contrast media interactions

The patient's preoperative drugs should be checked to avoid potentially nephrotoxic drugs if possible. It is more difficult to determine which drug may be potentially harmful in patients awaiting contrast administration. An illustrative example is the use of angiotensin-converting enzyme inhibitors (ACEI). Contrast agents are known to inhibit ACE.[60] ACEI and diabetes mellitus have been shown to be associated with CIN in a trial where patients were infused with a specially prepared solution containing half-normal sodium chloride, mannitol, $NaHCO_3$, and furosemide at 100 ml h^{-1} from 1 h before to 2 h after the procedure.[63] The increased risk of developing CIN with the use of captopril was also demonstrated in a small randomized controlled trial (RCT) of patients undergoing coronary angiography.[115] In contrast, a small-randomized trial comparing the use of *N*-acetylcysteine, fenoldopam, and normal saline, the risk of developing CIN was not associated with concomitant use of an ACEI.[3] However, the same trial also failed to show any association with diabetes mellitus, a known risk factor for CIN. Another RCT demonstrated some protective effect with ACEI in diabetic patients.[35] In this trial, there was a rather high rate of rise in post-procedural creatinine of 29%. On balance, there is insufficient evidence to support ACEI use as prophylaxis in CIN. However, there is little evidence supporting its omission in those who are stable on therapy.

Members of the Contrast Media Safety Committee of the European Society of Urogenital Radiology have produced a summary document on interactions between contrast media and other drugs.[72] Of particular concern is the decreased renal clearance of drugs such as metformin, with the potential to cause lactic acidosis. Patients receiving beta-blockers are more likely to develop anaphylactoid reactions to iodinated contrast, and are potentially more resistant to treatment.

Concomitant consumption of nephrotoxic drugs such as aminoglycosides and non-steroidal anti-inflammatory drugs (NSAID) can potentiate CIN. The adverse effects of NSAIDs on renal function in susceptible individuals are well documented.[125] It appears that COX-2 selective inhibitors offer no advantage over traditional NSAID in terms of nephrotoxicity.[30] Although no specific trials have examined this issue, substitution of these medications with other forms of analgesics during the peri-procedural period is recommended.[28] Other nephrotoxic drugs must be used with caution, especially those requiring therapeutic level monitoring such as aminoglycosides and vancomycin. Concentrations of other drugs reliant on renal clearance, such as digoxin, may also be affected. Close monitoring of renal function, with drug level monitoring and possible dose adjustment, may be warranted.

Caution must also be exercised with the use of diuretics, as they may enhance the contrast-induced diuresis, which some claim contribute to renal toxicity.

Anaesthetic technique

At present, there are no data available suggesting that inhalation, i.v. or regional anaesthesia, significantly influence the development or severity of CIN. However, the anaesthetist should ensure that the patient is well hydrated before induction and avoid the indiscriminate use of vasopressors to support arterial pressure before fluid status is optimized. Both volatile agents and neuroaxial blockade cause systemic vasodilatation, which if left uncorrected, may affect renal perfusion. However, provided mean arterial pressure is adequate, epidural blockade does not significantly alter renal blood flow.[110] Although there appears to be no significant differences in the measured postoperative creatinine levels when the different volatile agents are compared,[20 32 109] there is some weak evidence that prolonged sevoflurane exposure is associated with transient glomerular and tubular injury.[26 39] It has to be emphasized that these studies involved comparing sevoflurane with desflurane and isoflurane, respectively, and were performed in patients not receiving contrast media. The EUROSTAR data indicate that patients appeared to benefit when a local anaesthetic technique was used for EVAR in treating infrarenal aneurysms of the abdominal aorta, although renal function was not a specific outcome measure.[95]

Alternative to iodinated contrast

Although gadolinium-based contrast agents in doses of up to 0.3 mmol kg^{-1} are considered to be non-nephrotoxic, renal dysfunction can occur after its administration in these doses, especially in patients with diabetic nephropathy or pre-existing renal impairment.[27 114] Currently, it is only approved for use in magnetic resonance imaging contrast studies, but it has been suggested and used as an alternative to iodinated agents for radiographic contrast studies. However, after review of current available evidence, both the CIN Consensus Working Panel and the Contrast Media Safety Committee of the European Society of Urogenital Radiology cannot recommend the use of this class of agents to avoid nephrotoxicity.[21 27]

Conclusions

CIN is a complex clinical disorder that may affect increasing number of patients in the perioperative period. It is unlikely that any single therapeutic intervention can prevent this problem apart from choosing a viable alternative imaging modality. The anaesthetist should be aware of the disorder, identify those patients at risk, and perform preoperative assessment with a focus on volume status, identifying and withholding potentially nephrotoxic agents and drugs that may accumulate with reduced renal function. The patient should be kept normovolaemic at the

time of contrast exposure and a low haematocrit avoided. For high-risk patients, prophylaxis with *N*-acetylcysteine should be considered and the operator should be reminded to use the minimal amount of contrast necessary. Sevoflurane may be avoided for general anaesthesia in prolonged procedures where there is a viable alternative and local anaesthesia used where appropriate. At risk patients should be adequately followed-up with renal function testing and maintenance of post-procedure hydration.

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