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Perioperative acute kidney injury: risk factors, recognition, management, and outcomes

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Summary points

- Perioperative acute kidney injury (AKI) is common but poorly recognised and managed
- Perioperative AKI increases surgical mortality and morbidity and increases cost
- An apparently successful surgical outcome may not mean a successful renal outcome
- Careful and thoughtful preoperative assessment, including identifying patients with existing chronic kidney disease and stopping and avoiding nephrotoxic drugs, will reduce the incidence of perioperative AKI.
- Management of AKI centres on optimising fluid status and blood pressure, treating sepsis, and removing nephrotoxic agents where possible
- Patients with AKI are often complex to treat, and senior help should be sought at an early stage

Acute kidney injury (AKI), formerly known as "acute renal failure," is associated with increased morbidity, mortality, duration of hospital stay, and healthcare cost.[w1] Despite this, published data on perioperative acute kidney injury, occurring between the time of admission for surgery and the time of discharge, are scarce outside the cardiovascular surgery setting. Regardless of the clinical setting, the diagnosis of AKI is often delayed, and treatment is suboptimal in a large proportion of cases.[1] To improve diagnosis and treatment, clinicians need to understand the risks and triggers for perioperative AKI, the association of even small transient rises in creatinine concentration with risk of death,[2] and what actions they need to take promptly on diagnosis. The term acute kidney injury reflects the importance of thinking of the condition as a spectrum or continuum of disease that may be recognised at an early stage, rather than as an "all or nothing" phenomenon as implied by the term acute renal failure. Recognising earlier stages of renal impairment allows for early appropriate action that may interrupt a process of functional decline.

In this article we recommend the introduction of systems to ensure that changes in creatinine concentration from baseline are urgently highlighted to the clinical team. We outline the risk factors for perioperative AKI and discuss how to recognise the condition, manage it, and improve outcomes, focusing on the non-specialist surgery setting and using evidence from randomised trials, retrospective studies, meta-analyses, and expert reviews, as well as the recommendations of recent guidelines.

Why identify who is at risk for perioperative acute kidney injury?

Recognising patients who are particularly vulnerable to AKI allows for the potential manipulation of their clinical journey to minimise exposure to renal insults, and to optimise the potential for renal recovery should injury occur. The consequences of perioperative AKI are serious.

Increased risk of death

A retrospective cohort study of 10 518 patients has shown that AKI after major surgery worsens long term survival in patients with normal baseline renal function.[3] Death may occur as a direct result of kidney failure or as a result of complications in other organ systems. It is increasingly recognised that distant organ injury occurs after AKI, and it is often the failure of that organ that leads to death.[4] A retrospective study has shown that AKI after non-cardiac surgery is an independent predictor for hospital mortality (odds ratio 3.12, 95% confidence interval 1.41 to 6.93, P=0.005) with a hospital mortality of 26.4% compared with 2.5% of patients without AKI in a recent retrospective cohort study.[5] An observational study found that patients with AKI (class F according to the RIFLE criteria[6]—see table 1 for classification of AKI) were found to be 25 times more likely to die after cardiac valve surgery than those with no AKI.[7] Patients who completely recover after postoperative AKI still have an increased adjusted hazard ratio for death of 1.20 (95% confidence interval 1.10 to 1.31, P<0.001) over the longer term compared with patients without AKI according to a recent cohort study.[3]

Table 1 RIFLE staging for acute kidney injury[13]

Risk
Increased serum creatinine x1.5 or GFR decrease >25%
Urine output <0.5 ml/kg/h x6 hours
Injury
Increased serum creatinine x2 or GFR decrease >50%
Urine output <0.5 ml/kg/h x12 hours
Failure
Increased serum creatinine x3 or GFR decrease >75% or serum creatinine 4 mg/dl (353.6 μmol/l) (acute rise 0.5 mg/dl or 44.2 μmol/l)
Urine output <0.3 ml/kg/h x24 hours or anuria x12 hours
Loss
Persistent acute kidney injury=complete loss of kidney function >4 weeks (ie. dialysis dependence for 4 weeks)
End stage kidney disease
End stage kidney disease (ie. dialysis dependence for >3 months)

Increased length of hospital stay
Although good quality data are lacking, the findings of observational studies suggest that postoperative AKI increases the duration of both intensive care and hospital stay.[5] This would seem likely, given AKI's association with other organ dysfunction, electrolyte and acid-base disorders, and an increased risk of fluid overload, which may in turn contribute to postoperative immobility, infection, and poor wound healing.

Development or progression of chronic kidney disease
Experts now understand AKI to have three possible outcomes (rather than just chronic kidney disease or recovery)[8] [w2]:

- Return to baseline function (recovery may be prolonged in elderly patients)
- Development of chronic kidney disease in previously normal kidneys
- Accelerated progression of disease in patients with pre-existing chronic kidney disease, and about a fivefold increased risk for end stage disease.[w3]

Whether the patients who return to baseline function are more prone to the subsequent development of chronic kidney disease is not clear.

Who is at increased risk of perioperative acute kidney injury?

Impaired clinical status
Comorbid illness, acute illness, or age related loss of physiological reserve may predispose a patient to AKI.

A risk index for developing AKI during general surgery has been produced using prospectively acquired data on 152 244 operations that took place in the United States in 2005-6.[9] The risk factors for AKI identified in this study were age 56 years, male sex, active congestive cardiac failure, presence of ascites, hypertension, emergency surgery, intraperitoneal surgery, preoperative creatinine >106 μmol/l, and diabetes mellitus (either tablet or insulin controlled). Patients with six or

more of these risk factors had a 10% incidence of AKI and a hazard ratio of 46.2 compared with patients with fewer than three risk factors.

Patients requiring postoperative critical care are an obvious at risk group. An analysis of prospective data on 17 363 critically ill patients from the United Kingdom Intensive Care National Audit and Research Centre Case Mix Programme found that 5.6% of patients with severe AKI during the first 24 hours of stay in an intensive care unit were elective surgical admissions and 10.8% were emergency surgical admissions.[10] Overall, more than 40% of patients had sepsis, but other predisposing factors are unclear from the paper. Unfortunately, the study used a creatinine concentration of 300 μmol/l and/or urea 40 mmol/l to define severe AKI, which means that a large number of patients with milder AKI were potentially excluded, limiting the usefulness of the study. A retrospective cohort study of 1166 postoperative (non-cardiac surgery) patients in an intensive care unit in Portugal[11] found AKI in 7.5% using the AKIN criteria[12] (see table 2). The following preoperative determinants for postoperative AKI were identified in this study on multivariate analysis:

- American Society of Anesthesiologists physical status score of IV or V (odds ratio 3.94, 95% confidence interval 2.07 to 7.51; P<0.001) for AKI
- Revised cardiac risk index score >2 (this comprises high risk surgery, congestive heart disease, ischaemic heart disease, cerebrovascular disease, and insulin dependent diabetes mellitus with one point allocated for each)—odds ratio 2.45 (95% confidence interval 1.52 to 3.96; P<0.001) for AKI
- High risk surgery (intraperitoneal, intrathoracic, or suprainguinal vascular)—odds ratio 3.34 (95% confidence interval 2.02 to 5.53; P<0.001) for AKI
- Congestive heart disease (odds ratio 2.34, 95% confidence interval 1.42 to 3.88; P=0.001) for AKI.

Table 2 AKIN staging for acute kidney injury[12]

Stage 1
Increase in serum creatinine of 0.3 mg/dl (26.4 μmol/l) or increase to 150% to 200% from baseline
Urine output <0.5 ml/kg/h for >6 hours

Stage 2
Increase in serum creatinine to >200% to 300% from baseline
Urine output <0.5 ml/kg/h for >12 hours

Stage 3
Increase in serum creatinine to >300% from baseline (or serum creatinine of 4.0 mg/dl (354 μmol/l) with an acute increase of 0.5 mg/dl (44 μmol/l); or receiving renal replacement therapy
Urine output <0.3 ml/kg/h for 24 hours or anuria for 12 hours

These risk factors are in broad agreement with the general surgery AKI risk index discussed earlier. We consider that duration of surgery, requirement for blood product administration, sepsis, exposure to hypovolaemia, and relative hypotension in the perioperative period are potential risk factors that should also be considered.

Medication that may impair renal function, and/or exposure to intravenous contrast

Many medications that are commonly used in the perioperative period have the potential to adversely affect renal function. Non-steroidal anti-inflammatory drugs (NSAIDs), including cyclo-oxygenase-2 (COX 2) inhibitors, impair renal autoregulation by inhibiting prostaglandin mediated dilatation in the afferent arteriole of the glomerulus and may lead to reduced glomerular perfusion in the face of a prerenal insult.[w4] NSAIDs may also cause acute interstitial nephritis.

Angiotensin converting enzyme inhibitors and angiotensin receptor blocking drugs diminish the ability of the efferent arteriole to constrict, another key component of renal autoregulation.[w5]

Antimicrobial drugs can cause tubular toxicity if their concentration in the renal tubule is too high (for example, aminoglycosides) or may be associated with acute interstitial nephritis (for example, penicillins, cephalosporins, and fluoroquinolones such as ciprofloxacin, ofloxacin, and levofloxacin). Tubular toxicity is detected in 2-3% of renal biopsies in patients with AKI and is responsible for 6.5-27% of cases of unexplained AKI.[w6] Antibiotics account for about a third of drug induced acute interstitial nephritis.

Finally, intravenous contrast agents are capable of inducing pathological vasoconstriction in a vulnerable kidney.

Specific operative interventions or perioperative care

Compared with the general surgical population, in which the incidence of AKI is about 1%,[12] some types of specialist surgery (such as on-pump cardiothoracic surgery and cardiac or liver transplantation) may be associated with a substantially increased risk of renal impairment. For cardiac surgery, the incidence of AKI has been reported as 10-30%. [12] [w7] This risk may relate to exposure to a preoperative contrast agent, a fall in relative perfusion pressure while on bypass, reduction of pulsatile blood flow on bypass, and activation of the inflammatory cascade when blood is exposed to the foreign surface of the extracorporal circuit. Longer bypass times increase the risk, and patients may develop impaired ventricular function immediately after surgery.[w8]

Liver transplantation carries a reported AKI incidence of anywhere from 17% to 95% of patients.[w9] Patients may already have impaired renal function, and there is a risk of haemodynamic instability, bleeding, and substantial exposure to blood product intraoperatively. In addition, immunosuppressive treatment such as ciclosporin, tacrolimus, or sirolimus can be nephrotoxic, and patients receiving these drugs not infrequently experience sepsis.[w9] The general principles of risk recognition and management of AKI that are outlined in this article also apply to these specialist areas.

Abdominal aortic aneurysm surgery carries a risk of impaired renal blood flow resulting from aortic clamping, perioperative hypotension, and atheroemboli, as well as that of exposure to blood products. A Brazilian single centre study of 77 patients after open repair of abdominal aortic aneurysm found that 57% of patients had an increase in serum creatinine concentration of 25% from baseline at 48 hours after surgery.[14]

How is acute kidney injury recognised and diagnosed?

Recognition of AKI requires (*a*) consistent diagnostic criteria that work in everyday practice and (*b*) an awareness from doctors so that they keep these criteria in mind when interacting with patients and reviewing their test results.

Multiple definitions for renal injury exist. However, no matter which definition is used, the clinician will currently recognise and grade the severity of AKI on the basis of the occurrence over a period of up to 48 hours of either a change in serum creatinine concentration or a decline in urine output, or both, from baseline. Tables 1 and 2 outline the two major classifications of AKI. In 2004, a consensus definition and classification system, informally referred to as the RIFLE criteria, was proposed.[6] This acronym refers to three grades of increasing severity of AKI based on serum creatinine concentration or urine output (Risk, Injury, Failure) and two outcome variables (Loss, and End stage renal disease).

The AKIN criteria (published 2007) defined AKI as an abrupt (occurring within a 48 hour period) reduction in kidney function, with the diagnosis made on the basis of the specific changes from baseline in patients who have achieved an optimal state of hydration.[15]

The AKIN criteria recognise the clinical significance of even small increases in serum creatinine concentration given their reported association with adverse outcomes.[16] [17] Small increases in serum creatinine concentration are critically important and should be regarded as a red flag that prompts urgent assessment and corrective interventions. A change in creatinine concentration from baseline is what is important, not simply the value itself. Recognition of AKI is still delayed in a substantial number of patients, especially those who develop it after hospital admission.[1]

Although both AKIN and RIFLE use serum creatinine concentration and urine output as key markers, this approach is not without problems. It is especially important to recognise that patients with similar creatinine concentrations do not necessarily have similar renal function. Creatinine concentration is related to body muscle mass, and there are several patient groups in whom this mass can be markedly depleted, not least elderly patients. Such patients may have a lower baseline creatinine concentration and can have AKI despite their creatinine concentration being within the upper limits for the laboratory range of "normal." In addition, drugs, diet, body mass index, impairment of other organs, and ethnicity may alter the creatinine concentration.

Given that as much as 50% of functional nephron mass may be lost before changes in creatinine concentration become apparent, the ability to detect renal impairment before an increase in creatinine is noted would be an important advance. Several biomarkers for AKI have been studied but are not yet used in routine practice. Neutrophil gelatinase associated lipocalin (NGAL) is a protein found in excess in the plasma and urine of patients with AKI as many as 48 hours before serum creatinine concentration starts to increase.[18] It shows promise as both a predictive and prognostic marker (although plasma concentrations also rise in severe sepsis without AKI, making urinary NGAL more attractive).[19] [20] [21] Serum cystatin C (a protease inhibitor less affected than creatinine by age, sex, muscle mass, and diet) also allows earlier detection of AKI.[22]

In addition, the development of metabolic acidosis or electrolyte abnormalities, including hyperkalaemia, may indicate the onset of AKI, and so prompt careful assessment of the patient is needed.

What could be done to decrease the incidence of perioperative acute kidney injury?

Incorporate formal evaluation of renal risk into preoperative assessment

Many hospitals now run formal preoperative assessment clinics (models include physician led, nurse led, or hybrid), and even for major surgery they admit patients on the day of operation. As part of this process, we suggest that renal risk stratification is done using the factors discussed above. Staff working in these services must be aware of local policies and mechanisms for communicating

an increased risk of perioperative AKI to the patient's managing team. The preoperative phase is the ideal time to recognise previously undetected chronic kidney disease (table 3), optimise medications, and liaise with the anaesthesia team or the nephrology department (if necessary). However, no clear evidence exists that this approach can alter outcome.

Table 3 Chronic kidney disease stages

Stage 1
Chronic kidney disease with normal glomerular filtration rate but other kidney damage (eg, haematuria and proteinuria) **eGFR (ml/min/1.73m^2)* = 90**

Stage 2
Mild chronic kidney disease and other kidney damage **eGFR (ml/min/1.73m^2)* = 60-89**

Stage 3a
Mild to moderate chronic kidney disease **eGFR (ml/min/1.73m^2)* = 45-59**

Stage 3b
Moderate to severe chronic kidney disease **eGFR (ml/min/1.73m^2)* = 30-44**

Stage 4
Severe chronic kidney disease **eGFR (ml/min/1.73m^2)* = 15-29**

Stage 5
Established end stage renal disease **eGFR (ml/min/1.73m^2)* = <15 or on dialysis**

*eGFR is an estimate of kidney function, based on serum creatinine but taking into account age, race, and sex. In the UK, whenever a creatinine test is requested, laboratories will report a corresponding eGFR and the eGFR can be thought of as an approximate percentage of normal kidney function that the individual has. For some individuals, a preoperative assessment might be the first time they have had a creatinine check and therefore may be the first opportunity for chronic kidney disease to be diagnosed.

Minimise exposure to perioperative nephrotoxins
It may be appropriate to avoid using angiotensin converting enzyme inhibitors and angiotensin receptor blocking drugs on the day of surgery, both to protect the kidney and to minimise the risk of hypotension when anaesthesia is delivered in the presence of these drugs. If withheld, we recommend not restarting these drugs until the patient has recovered from surgery, the risk of hypoperfusion is minimal, and kidney function has been shown to be at baseline. If no hospital protocol exists for the management of perioperative medications then specific advice can be obtained from the anaesthetist responsible for the case.

Although NSAIDs are useful as part of perioperative pain relief strategies, their benefits must be balanced against their adverse effects, which include renal impairment. Anaesthetists will usually weigh up the risk-benefit balance when deciding whether to give NSAIDs in the operating theatre, but postoperative use needs to be considered too. The risk of AKI with NSAIDs has led the Medicines and Healthcare Products Regulatory Agency to issue drug safety advice, which recommends that NSAIDs are best avoided in hypovolaemia or in patients who have sepsis, even if their serum creatinine concentration is normal (see www.mhra.gov.uk/Publications/Safetyguidance/DrugSafetyUpdate/CON046451).

Patients who are at risk of renal impairment and who require the administration of intravenous contrast in the perioperative period require very careful management. Perioperative contrast induced AKI has not been studied in detail, but data from interventional cardiology settings show that it occurs more commonly in patients with certain features (see the box outlining the risks of contrast induced AKI).[23] Risk reduction involves the use of the lowest volume of contrast agent compatible with examination success, together with consideration of non-ionic iso-osmolar contrast or low osmolar contrast.[24] We recommend using an isotonic intravenous fluid solution such as 0.9% sodium chloride or isotonic sodium bicarbonate to ensure adequate prehydration. Controversy also surrounds the use of oral N-acetylcysteine (an antioxidant that is most commonly used as a cysteine donor to enhance formation of glutathione after paracetamol overdose), although positive results have been reported with doses of 1200 mg orally twice daily on the day before use of a contrast agent and on the day of exposure.[25]

Patient features associated with increased risk of contrast induced acute kidney injury

- Advanced age
- Chronic kidney disease
- Diabetes mellitus
- High doses of contrast agent
- Intra-arterial (rather than intravenous) injection of contrast agent
- Hypertension
- Congestive heart failure
- Hypotension
- Anaemia
- Non-steroidal anti-inflammatory drugs
- Hypovolaemia
- Cardiac surgery after contrast agent

Much of the literature in this area centres on the administration of contrast agents during angiography and not, for example, on perioperative abdominal computed tomography. We recommend that clinicians follow their institution's protocol or in the absence of such a protocol ensure adequate intravascular volume using 0.9% sodium chloride solution.

Intraoperative management and haemodynamic optimisation

Renal protection during surgery relies primarily on maintaining adequate renal perfusion, which in turn relies on appropriate intravascular volume replacement and mean arterial pressure. The aim is to maintain a systemic arterial perfusion pressure that is appropriate for the patient (taking into account preoperative blood pressure and surgical requirements for relative hypotension). Aggressive fluid replacement alone may not overcome the hypotensive effects of anaesthesia in some patients and may in turn lead to postoperative complications. The management of blood pressure during anaesthesia is outside the scope of this article and will not be discussed here.

Unfortunately no intraoperative "magic bullets" exist that will consistently reduce the risk or severity of renal impairment. In particular, "renal dose" dopamine is of no benefit,[26] and mannitol, although aiding diuresis and reducing compartment pressures in cases of rhabdomyolysis, is generally ineffective in a non-cardiac surgery setting.[w10] Early indications are that newer agents such as fenoldopam (a selective dopamine agonist) and atrial natriuretic peptide may help to prevent AKI in selected patient groups (such as those having cardiac surgery).[27] [28]

Optimising intravascular volume and cardiac output may have a positive effect on perioperative renal function according to a recent meta-analysis in this area that identified 20 randomised controlled trials (most of which used pulmonary artery catheters) with sufficient data to determine renal outcomes.[29] This analysis found that in high risk patients (those having emergency surgery; those with a higher revised cardiac risk index or a higher risk according to criteria of the American Society of Anesthesiologists; and those aged >60 years) optimisation of cardiac output or oxygen delivery resulted in a decreased risk of perioperative AKI both in the subgroup in which this was started preoperatively (odds ratio 0.70, 95% confidence interval 0.53 to 0.94; P=0.02) and in the combined subgroup in which it was started either intraoperatively or in the early postoperative phase (0.47, 0.27 to 0.81; P=0.006). Both normal and supranormal target achievement were effective in reducing AKI. No comment could be made on the impact on mortality because of heterogeneity.

How to manage a patient with acute kidney injury?

A full review of the management of AKI is beyond the scope of this article. On the basis of evidence from trials and expert reviews we suggest the following strategies as an optimal management approach for patients identified as having AKI perioperatively, and we recommend that all hospitals have in place "renal rescue" guidance based on these principles.

Careful clinical examination

Conduct a careful clinical examination to search for the cause (consider prerenal, renal, and postrenal causes) and look specifically for renal obstruction by organising ultrasonography of renal tracts within 24 hours of identification of AKI.

Volume status

Assess the patient's volume status—that is, have they had adequate preoperative and intraoperative fluid or are they overloaded?

Complications

Look for complications of AKI, which may be acid-base disturbances; electrolyte abnormalities, such as hyperkalaemia; uraemia; or volume overload. In volume replete oliguric patients consider reducing fluid intake to keep up with output, but remember an allowance for insensible losses. In patients with fluid overload, loop diuretics may have a limited role, but a decision to start diuretics should ideally be made after consultation with a nephrologist. Although loop diuretics may be helpful in responsive patients in preventing fluid overload, they will not prevent AKI and may cause tinnitus, and their use in high doses is associated with increased risk of death or non-recovery of renal function (this association may be related to increased severity of renal impairment and so resistance to the effects of the drugs).[w10]

Minimise ongoing renal damage

Start taking therapeutic measures to minimise ongoing renal damage and promote recovery.

Administering fluids to restore effective circulating volume and blood pressure

This can be achieved by rapid administration of small to moderate fluid challenges, such as 500 ml crystalloid or 250 ml colloid, repeated as necessary with careful monitoring for fluid overload. A review of the potential effects of various colloids on renal function is beyond the scope of this article, but note that high molecular weight hydroxy-ethyl starch solutions have been associated with increased risk of renal impairment in patients with sepsis. A useful predictor of fluid responsiveness is an increase in the patient's blood pressure or a fall in heart rate in response to head down tilt or leg elevation. In cases where infusion of 1.5-3 litres of crystalloid or 1 litre of colloid (or lower volumes

in elderly patients or those with cardiac impairment) have not produced improvement, consider requesting advice from the intensive care unit as more invasive monitoring may be indicated. End points for fluid resuscitation include capillary refill time <3 seconds, restoration of blood pressure, and fall in arterial lactate concentration. Relentless administration of fluids to achieve blood pressure targets or in an attempt to reverse oliguria will lead to fluid overload and pulmonary oedema.

Administering vasopressors or inotropes
These may be used to maintain adequate perfusion pressure (such as mean arterial pressure >65 mm Hg, systolic pressure >100 mm Hg) where fluids are ineffective or only transiently effective. In addition, inotropes may be given concurrently with fluids in patients with moderate to severe ventricular dysfunction. However, patients with a complex condition are better managed in a high dependency environment that allows for optimal monitoring.

Aggressive treatment of sepsis
The treatment of sepsis includes not only antimicrobials but also urgent interventions to control the source of an infection, such as draining abscesses and collections.

Relieving urinary obstruction urgently if present
Modify prescribed drugs if they are likely to be contributing to the AKI. This includes stopping angiotensin converting enzyme inhibitors, angiotensin receptor blocking drugs, and other antihypertensives (in the presence of hypotension), nicorandil, and NSAIDs. Ensure that antibiotic levels are not in the toxic range. Adjust doses of opioid analgesics, metformin, and low molecular weight heparin. Consider stopping drugs that may aggravate hyperkalaemia, such as β blockers, digoxin, trimethoprim, and potassium sparing diuretics. A clinical pharmacist can advise on how best to alter drug doses.

Seek help from a senior colleague, working up your own "chain of command" in the first instance. Advice from specialists may be needed. Consult a nephrologist if there is single organ failure and a critical care specialist team if there is multiorgan failure. Have relevant information to hand when you call (recent surgical history, medication history, laboratory results, clinical observations, fluid balance, overall plan for patient).

Conclusion

AKI is a life threatening clinical problem. The impact of even mild degrees of AKI is both important and all too often ignored. Most cases of AKI that occur in the perioperative period are associated with relative hypoperfusion of the kidney and/or exposure of the kidney to nephrotoxins, and not with primary intrinsic renal disease. The likelihood of serious long term consequences for the patient will be reduced by following a logical strategy that includes a prompt reaction to AKI, with early appropriate fluid and medication management, the involvement of nephrology services to help with the recognition of important intrinsic renal causes for AKI, and the arrangement of prompt access to renal replacement therapy if needed.

Sources and selection criteria

We searched PubMed databases for acute kidney injury, acute renal failure, prerenal azotemia, perioperative, surgery, postoperative, and complications. We reviewed the reference lists of retrieved articles. We also looked at available guidelines from the Renal Association, the National

Confidential Enquiry into Patient Outcome and Death, and the National Institute for Health and Clinical Excellence and used our own clinical experiences

Tips for non-specialists

- Perioperative AKI increases surgical mortality and morbidity and increases hospital costs
- Careful preoperative assessment can identify patients at particular risk of AKI and could allow for additional monitoring and planning
- Perioperative AKI rarely indicates an isolated renal problem but rather a physiologically unstable patient who may deteriorate further and must not be ignored
- The successful prevention and management of AKI involves timely recognition of perhaps subtle abnormalities, basic clinical assessments and observations, and quick and appropriate reaction to information, including getting senior and specialist help
- Ensure that the patient with a diagnosis of AKI is normovolaemic, has an adequate mean arterial pressure, and preferably is not exposed to nephrotoxins
- Many surgical patients have a history of ventricular dysfunction, and optimisation of cardiac function may require inotropic support
- Renal tract obstruction must be excluded radiologically within 24 hours of a diagnosis of AKI

Additional educational resources

- National Confidential Enquiry into Patient Outcome and Death. Acute kidney injury: adding insult to injury. 2009. (A report highlighting the process of care of patients who died in hospital with a primary diagnosis of acute kidney injury.) www.ncepod.org.uk/2009aki.htm
- Renal Association (the professional body for UK nephrologists) guidelines on the treatment of AKI (mainly focused on dialysis therapies). www.renal.org/Clinical/GuidelinesSection/AcuteKidneyInjury.aspx
- Kidney Disease: Improving Global Outcomes (www.kdigo.org/)—A global non-profit foundation dedicated to improving the care and outcomes of patients with kidney disease
- Northern Ireland guidelines for the management of chronic kidney disease. 2010. www.gain-ni.org/Library/Guidelines/Chronic%20Kidney%20Disease.pdf
- Website of the Edinburgh Renal Unit (www.edren.org/)—Has freely accessible information for patients, for doctors who are not renal specialists, and for anyone who is interested in kidney diseases. For AKI see www.edren.org/)pages/edreninfo/acute-renal-failure-aki.php

Areas for future research

- Clarification of AKI epidemiology in common surgical groups. We hope the recent developments in defining AKI (RIFLE and AKIN) will make this more achievable
- Further investigation of the extent to which new biomarkers can impact on the course of AKI —can they detect AKI in time to improve outcomes?
- Exploration of the patient, timing, and procedural factors that alter the effectiveness of drugs such as fenoldopam and atrial natriuretic peptide

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