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Acute kidney injury

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ABSTRACT Acute kidney injury (AKI) represents a medical emergency associated with poor clinical outcomes. The international guideline group Kidney Disease: Improving Global Outcomes (KDIGO) has defined AKI according to rises in serum creatinine and/or reductions in urine output. Any patient who meets the criteria for AKI should be reviewed to ascertain the cause of AKI and the severity of the injury should be staged. Patients with more severe AKI are at greater risk of progression to chronic kidney disease (CKD). The 2009 National Confidential Enquiry into Patient Outcomes and Death (NCEPOD) reported that only 50% of patients who died with a diagnosis with AKI received good care. The mortality from AKI has remained unchanged for the last four decades and there are currently no specific therapies for the majority of cases of AKI. Patients with rarer forms of AKI need urgent renal referral for specific therapy. At present, serum creatinine and urine output remain the best biomarkers for detecting AKI. However, significant kidney damage has usually occurred by the time changes in these biomarkers are manifest and newer biomarkers are under investigation. Management of AKI is based upon general supportive measures, which includes treatment of the underlying cause and the initiation of renal replacement therapy (RRT) in patients with complications refractory to medical management. The optimal choice of intravenous fluid therapy remains controversial. There is currently renewed interest in more specific therapies for AKI secondary to hypoperfusion and/or sepsis, which have been previously unsuccessful. A number of therapeutic strategies are presently being explored in clinical trials.

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INTRODUCTION

Acute kidney injury (AKI) is a syndrome with many different causes characterised by a rapid loss in renal function, resulting in a failure to maintain electrolyte, fluid, and acid-base balance. The previous lack of a universal definition has hampered the collection of accurate epidemiological data regarding its incidence and outcomes. In 2009, the National Confidential Enquiry into Patient Outcome and Death (NCEPOD) published a report, *Adding Insult to Injury*, which identified significant deficiencies in the care of patients dying with a diagnosis of AKI.[1] Only 50% of these patients received satisfactory or good care and 20% of hospital-acquired AKI was thought to have been avoidable.[1] Indeed the diagnosis of AKI was delayed in 43% of patients who developed it after admission to hospital.[1] In a significant number of patients the development of AKI, as signalled by rises in serum creatinine, occurs secondary to another disease process and may be the first indication that this is occurring. In these cases AKI is acting as an early biomarker of systemic illness. Acute kidney injury occurs across many different medical and surgical specialties, with frail older people being particularly vulnerable. Acute kidney injury commonly occurs secondary to a number of other insults and is often under-recognised due to pre-existing knowledge gaps in undergraduate and postgraduate training. Most patients who develop AKI are not under the care of a renal physician.[2] It is therefore important that non-specialists receive appropriate training to enable them to recognise and respond appropriately. There has recently been a concerted effort in the UK to raise awareness among healthcare professionals of the importance of AKI and to improve its management. Guidance on AKI was published in 2013 by the the National Institute for Health and Care Excellence (NICE).[3] The management of AKI with respect to the role of fluids, e-alerts and biomarkers was the subject of a Royal College of Physicians of Edinburgh UK Consensus Conference in 2012.[4]

Definitions

The international guideline group Kidney Disease: Improving Global Outcomes (KDIGO)[5] defines AKI as occurring when:

- Serum creatinine rises by 26 μmol/L within 48 hours; or

- Serum creatinine rises by 1.5-fold from the baseline value, either known or thought to have occurred within one week; or
- Urine output is less than 0.5 mL/kg/hour for more than six consecutive hours.
- Patients identified as having met the definition of AKI should have the cause of AKI ascertained and the stage determined (Table 1).

TABLE 1 KDIGO staging classification for AKI ([5]KDIGO 2012)

Stage	Serum creatinine (SCr) criteria	Urine output criteria
1	Increase 26 μmol/L within 48 hours or Increase 1.5 to 1.9 x baseline SCr	<0.5 mL/kg/hr for >6 consecutive hours
2	Increase 2 to 2.9 x baseline SCr	<0.5 mL/kg/hr for >12 hours
3	Increase 3x baseline SCr or increase ≥354 μmol/L or commenced on renal replacement therapy (RRT) irrespective of stage	<0.3 mL/kg/hr for >24 hours or anuria for 12 hours

It is intended that the KDIGO system for defining and staging AKI harmonises earlier systems such as the RIFLE (Risk, Injury, Failure, Loss, End-Stage) criteria proposed by the Acute Dialysis Quality Initiative (ADQI) and the subsequent modification of these criteria by the Acute Kidney Injury Network (AKIN). [6,7]

There continues to be considerable debate with respect to the definition of baseline creatinine. If a baseline serum creatinine is not available within one week, it is suggested that the lowest serum creatinine value recorded within three months of the episode of AKI can be used. If a baseline serum creatinine value is not available within three months and AKI is suspected, serum creatinine should be repeated within 24 hours. A baseline serum creatinine value can then be estimated from the subsequent lowest serum creatinine value if the patient recovers from AKI.

Epidemiology

Acute kidney injury is common, although previous disparities in definitions make it difficult to determine an accurate incidence rate. The National Institute for Health and Care Excellence estimates that AKI is seen in approximately 13–18% of people admitted to hospital, with the elderly population being particularly at risk.[3] Acute kidney injury can be considered as community-acquired or hospital-acquired, although the distinction in some cases may be challenging. The prevalence of each is currently difficult to quantify although a recent study investigating community-acquired AKI found it to be present in 17.7% of acute medical admissions.[8] Depending on the population studied, AKI has a mortality of 10–80% and is an important cause of chronic kidney disease (CKD), with 14% of those with AKI progressing to stage 4, and those with more severe AKI being at higher risk of progression.[9,10] Quantifying mortality is difficult and varies with severity and setting; furthermore most death certificates do not include AKI and usually record the cause of death as being the primary condition which led to AKI. It was reported in 2009–10 that patients who experienced an episode of AKI in the UK stayed in hospital an average of 4.7 days longer than patients without AKI, with an associated cost estimated to be between £434 and £620 million.[11] Acute kidney injury represents an important patient safety issue as recognised by NHS England and is a significant financial burden on healthcare services.

Globally, the prevalence of AKI may be even higher than current estimates with mortality from AKI under recognised. Most data on the prevalence and outcomes of AKI are derived from high income countries where the aetiology of hospital-acquired AKI is similar to that in low income countries. However the aetiology of community-acquired AKI is very different between these countries with malaria, diarrhoeal illnesses, haemolytic uraemic syndrome, obstetric complications and leptospirosis being the main causes in low income countries.[12] The patient population is younger and poor access to supportive treatment to dialysis will result in higher mortality rates.[12] The International Society of Nephrology (ISN) has therefore launched the 0 by 25 initiative with one clear and concise aim: that no one should die of untreated acute kidney failure in the poorest parts of Africa, Asia and South America by 2025.

CLINICAL ASSESSMENT OF ACUTE KIDNEY INJURY

Clinical assessment includes a comprehensive history and examination, including an evaluation of the patient's volume status. It is essential that the cause of AKI should be identified and documented in the patient's clinical record. Clinical signs suggesting the underlying cause include a palpable bladder, enlarged prostate gland, a vasculitic rash, uveitis, fevers or painful swollen joints.

The causes of AKI have traditionally been divided into pre-renal, intrinsic renal, and post-renal (Table 2), although AKI is often multifactorial. Pre-renal and post-renal AKI commence as functional processes, which, if reversed promptly, limit any residual renal damage, whereas intrinsic AKI represents structural damage.

Baseline laboratory investigations should include urea, electrolytes, serum creatinine, serum bicarbonate, full blood count, calcium, phosphate, inflammatory markers such as C-reactive protein, urinalysis, and in cases of suspected infection, blood and urine cultures. Other

J R Coll Physicians Edinb 2013; 43:323–9

TABLE 2 Causes of AKI

Pre-renal	Intrinsic renal	Post-renal
Hypotension due to: • Hypovolaemia, e.g. bleeding, gastrointestinal losses • Sepsis • Cardiac arrhythmias • Myocardial infarction • Antihypertensive medications Renal artery stenosis	Acute tubular injury Infiltrative disease, e.g. myeloma Nephrotoxins Glomerulonephritis Interstitial nephritis	Renal stone disease Pelvic masses e.g. cervical cancer Prostatic hypertrophy/cancer Urethral stricture Retroperitoneal fibrosis

investigations to be considered include blood film, creatine kinase, chest radiograph, and renal tract ultrasound within 24 hours in suspected obstruction (six hours in suspected pyonephrosis). In cases where there is no obvious precipitating cause for the AKI a renal immunology screen (including serum electrophoresis, antinuclear antibodies [ANA], complement, immunoglobulins, anti-neutrophil cytoplasmic antibodies [ANCA]), anti-glomerular basement membrane antibodies and Bence Jones urinary protein) and renal biopsy may be required.

The recognition of AKI currently relies on a rise in serum creatinine and/or a decrease in urine output, both of which are considered relatively poor biomarkers. Serum creatinine remains a non-specific marker of AKI and does not indicate the site or severity of injury. The rise in serum creatinine is delayed in relation to the onset of the injury. Outside of the critical care setting urine output measurements are not routinely recorded. Therefore more specific serum and urinary biomarkers of AKI are being sought.[13,14] A number of different biomarkers have been investigated in small heterogeneous studies including neutrophil gelatinase-associated lipocalin (NGAL), interleukin 18 (IL-18), kidney injury molecule-1 (KIM-1), liver fatty acid binding protein (L-FABP) and more recently the cell cycle arrest markers, Insulin-Like Growth Factor Binding Factor 7 (IGF BP-7) and Tissue Inhibitor of Metalloproteinases 2 (TIMP 2). More data is required in specific clinical context before their routine use can be recommended. It is unlikely that one biomarker will fit all and a panel of biomarkers will be required because of the heterogeneity of causes of AKI. In the meantime it is important to stress that if a patient experiences a rise in serum creatinine an underlying cause should be sought.

MANAGEMENT OF ACUTE KIDNEY INJURY

The cornerstone of AKI management remains supportive, with specific therapy reserved for the rarer causes. Therefore the prevention of AKI and minimisation of its severity and duration are essential aspects of its management.

Prevention

1. Identification of patients at risk of AKI

Both primary and secondary care physicians must be aware of patient groups who are at risk of AKI. Risk factors identified by NICE include:[3]

- Age over 65 years
- Chronic kidney disease (estimated glomerular filtration rate <60 mL/min/1.73 m^2)
- Heart failure
- Liver disease
- Diabetes mellitus
- History of acute kidney injury
- Neurological or cognitive impairment, or disability, which may result in decreased access to fluids due to reliance on carers
- Symptoms or history of urological obstruction, or conditions that may lead to obstruction
- Sepsis
- Deteriorating early warning scores
- Oliguria (urine output less than 0.5ml/kg/hour)
- Hypovolaemia
- Use of iodinated contrast agents within the previous week
- Use of nephrotoxins within the previous week, such as aminoglycosides, non-steroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor antagonists (ARBs) and diuretics, especially if hypovolemic

Approximately 65% of patients admitted to hospital are over the age of 65, presenting challenges for healthcare services and their design.[15] Given the co-morbidities of this population, polypharmacy is common and significant numbers of patients take combinations of ACE-inhibitors or ARBs and NSAIDs.[16] An observational ecological study recently made an association between increased prescribing of ACE-inhibitors and ARBs and increased admissions with AKI, attributing up to 15% of AKI admissions to these drugs.[17] However this study did not demonstrate causation and other factors, such as an ageing population and increased recognition of AKI, may be involved. The NICE guidance recommends the use of ACE-inhibitors and ARBs for conditions such as heart failure, hypertension and CKD with proteinuria.[16] Patients at risk of AKI should be advised of 'sick day rules' such as withholding antihypertensive medications

EDUCATION

and diuretics when acutely ill with conditions such as vomiting and diarrhoea, and contacting their GP early for assessment and check of renal function. This may help reduce the number of episodes of community-acquired AKI.

2. Optimisation of volume status

The NCEPOD report highlighted poor management of sepsis and hypovolaemia as being important contributors to AKI.[1] A careful volume status assessment should be made in all patients, including assessment of peripheral perfusion, pulse rate, blood pressure, jugular venous pressure (JVP), skin turgor (over the clavicle), mucous membranes, presence of pulmonary and peripheral oedema, changes in blood pressure and heart rate with posture, urine output, and trends in weights. Post-operative AKI is frequently encountered and may be secondary to poor peri-operative volume status assessment and inadequate fluid therapy. The insertion of a urinary catheter may be necessary to aid with monitoring in the critically ill. Routine insertion of a urinary catheter in all patients with AKI is not recommended due to the associated infection risk, although is clearly indicated if urethral obstruction is considered to be the cause of AKI.

The correction of hypovolaemia is best achieved by repeated boluses of 500 ml (250 ml if there is a history of cardiac failure or the patient is elderly) crystalloid or colloid followed by immediate assessment of clinical response. Balanced electrolyte solutions (potassium containing) such as Hartmann's solution and PlasmaLyte are preferred unless the patient has hyperkalaemia (potassium >5.5 mmol/L), oliguric AKI, or rhabdomyolysis, when 0.9% sodium chloride solution should be used initially. There is ongoing debate regarding the various merits of crystalloids versus colloids as resuscitation fluids. The sole prescription of colloids is not recommended due to an association with a hyperosmolar state and should therefore be prescribed together with crystalloids. The Colloids Versus Crystalloids for the Resuscitation of the Critically Ill (CRISTAL) trial demonstrated no difference in 28 day mortality between its crystalloid (0.9% saline, hypertonic saline or Ringer's lactate) group and colloid (albumin, hydroxyethyl starch, gelatins or dextrans) group but suggested an improvement in 90 day mortality in the colloid group, as well as reductions in the need for mechanical ventilation, vasopressor support and fluid volumes.[18] However, the Crystalloid versus Hydroxyethyl Starch Trial (CHEST), a recent large randomised controlled trial in a population of critically ill patients, suggested that HES-based colloids were associated with increased adverse events and an increased need for RRT, although did not demonstrate a difference in mortality between the normal saline group and hydroxyethyl starch (HES) group.[19] The findings from CHEST were supported by the earlier Scandinavian Starch for Severe Sepsis/Septic Shock (6S) trial, which demonstrated that HES-based colloids were associated with an increased risk of death at 90 days and RRT in patients with severe sepsis when compared with Ringer's Lactate.[20] The Medicines and Healthcare products Regulatory Agency (MHRA) has since suspended licences for all HES products, such as Tetraspan, Volulyte and Voluven.[21]

It is important that clinicians recognise that the daily sodium intake in healthy people is between 70 and 100 mmol/day. This has implications for the prescription of maintenance fluid regimens as the excessive prescription of 5% dextrose may precipitate hyponatraemia, while the sole prescription of 0.9% sodium chloride may result in hyperchloraemic metabolic acidosis, sodium and water overload. The prescription of maintenance fluid regimens needs to be tailored to the requirements of each individual patient. At the time of writing, NICE guidelines on IV fluid therapy are in development and due to be published in December 2013.

3. Reducing the risk of contrast-induced AKI

The increased availability of radiological iodinated contrast studies has seen contrast-induced AKI (CI-AKI) become an important hospital-acquired cause of AKI. It develops due to a combination of cytotoxicity of contrast media and afferent arteriolar vasoconstriction. Acute kidney injury following exposure to iodinated contrast is uncommon in the general population but NICE suggest that increased risk is associated with:[3]

- Chronic kidney disease (particularly if eGFR is less than 40ml/min/1.73m^2)
- Diabetes in the context of CKD
- Heart failure
- Renal transplant
- Age 75 years or over
- Hypovolemia
- Increasing volume of contrast agent
- Intra-arterial administration of contrast

Contrast-induced AKI occurs up to 72 hours following iodinated contrast and is non-oliguric in nature. There is no specific therapy and prevention through adequate volume expansion remains the most important measure. Nephrotoxic medications such as gentamicin and NSAIDs should be avoided. There is currently no evidence at present to recommend routinely withholding angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers in haemodynamically stable patients although clearly these should be reviewed in hypotensive patients.[22] Metformin is not directly nephrotoxic but is exclusively excreted by the kidneys. The Royal College of Radiologists state that if serum creatinine is above the reference range, or estimated glomerular filtration rate (eGFR) is <60 ml/min/1.73 m^2, then a decision to stop metformin for 48 hours should be at the discretion of the patient's clinician.[23] Patients at risk of CI-AKI should receive appropriate volume expansion, with joint guidelines from the Renal

J R Coll Physicians Edinb 2013; 43:323–9

Association, British Cardiovascular Intervention Society and Royal College of Radiologists recommending the use of isotonic sodium bicarbonate or intravenous 0.9% sodium chloride at a rate of 1 ml/kg/hour for 12 hours pre- and post-procedure.[22] There is no compelling evidence supporting the routine use of N-acetylcysteine to prevent CI-AKI.[22]

MANAGEMENT OF ESTABLISHED AKI

Earlier detection of AKI should facilitate earlier intervention and therefore reduce the severity and duration of AKI and limit the risk of progressing to CKD. In its early stages, AKI is often reversible. It is essential to determine the cause of AKI as this dictates whether any specific therapies, besides supportive strategies, are required. Most cases of AKI are multifactorial. However rarer causes need to be identified early and in these cases early referral to renal services is recommended. The mainstays of supportive management include optimising volume replacement, prompt treatment of sepsis with appropriate antibiotics and the use of sepsis bundles,[24] avoidance of nephrotoxic medications, and the use of RRT where indicated. Obstruction, if present, must be relieved and NICE have included guidance on when to refer urgently to urology.[3] There is no evidence to support the use of any particular pharmacological treatment in AKI secondary to hypoperfusion and/or sepsis. Dopamine is still used in some clinical contexts to improve renal blood flow and urine output. However this is not recommended by NICE as this approach is not evidence-based and there is no demonstrable clinical benefit. In fact there is an increased risk of cardiac arrhythmias and myocardial and intestinal ischaemia.

Medicines management

Nephrotoxic medications such as gentamicin and NSAIDs should be avoided in established AKI. Anti-hypertensives should be withheld in hypotensive patients as these will exacerbate renal hypoperfusion. Medications that are metabolised and excreted by the kidneys should be identified and doses adjusted accordingly to limit accumulation and toxicity. Common examples include opiates, fractionated heparins, antibiotics such as penicillins and intravenous vancomycin, and antiviral agents such as aciclovir. A renal pharmacy working group recently created an 'AKI medications optimisation toolkit'.[25] It is important to work closely with ward pharmacists and consult the *British National Formulary* and *Renal Drug Handbook* as invaluable sources of information. Electronic prescribing will hopefully provide improved patient safety with respect to medicines in patients with AKI.

Referral criteria

Not all patients with AKI need to be referred to renal physicians. NICE guidelines recommend discussion with a renal unit within 24 hours in the following situations:[3]

- Stage 3 AKI
- Inadequate response to treatment
- A lack of a clear precipitating cause of AKI.
- Clinical suspicion for a diagnosis that may require specialist treatment (for example vasculitis, myeloma, glomerulonephritis, interstitial nephritis)
- A renal transplant
- Chronic kidney disease stage 4 or 5
- Complications of AKI requiring RRT:
  - Refractory hyperkalaemia.
  - Refractory pulmonary oedema.
  - Severe metabolic acidosis (pH <7.15).
  - Uraemic complications such as pericarditis and encephalopathy

It would also be prudent to refer patients suspected of having AKI secondary to poisoning. The presence of haemolysis and low platelets should raise suspicions for haemolytic uraemic syndrome and thrombotic thrombocytopaenic purpura; such patients also require discussion with a renal unit.

A subset of patients will benefit from management in a critical care setting and should therefore also be referred to the intensive care or high dependency unit. In particular, patients with AKI stage 3 are likely to have complications such as severe acidosis, hypotension and hypoxia secondary to pulmonary oedema. It can be difficult to manage such patients on a renal ward and they are likely to be unfit for inter-hospital transfers between district general hospitals and tertiary renal units.

Recovery of renal function and follow up

The earliest sign of renal recovery in patients with oliguric AKI is an increase in urine output. This is often accompanied by a decrease in the rise in serum creatinine, followed by a plateau period, and subsequently a fall in serum creatinine. Close monitoring of volume status and urea and electrolytes should be continued, as patients may develop polyuria and associated hypokalaemia and hypernatraemia in this period. Hypokalaemia must be treated early as it may cause life-threatening arrhythmias and ileus. Hypernatraemia occurs secondary to a free water deficit and if patients cannot drink an increased amount of water, intravenous 5% dextrose may be required. Polyuria occurs as injured proximal tubules are initially unable to concentrate urine. Careful volume assessment and replacement of these losses is required to enable continued recovery from AKI.

Patients who have had an episode of AKI are at risk of developing CKD. Those at highest risk are those whose renal function has not recovered to baseline at the time of discharge. These patients should be discharged with clear information on the cause of the AKI, its severity, the serum creatinine on discharge and advice on restarting any medicines that were stopped during admission. Failure to communicate clearly with general practitioners

following discharge can limit the ability of general practitioners to recognise patients at future risk of AKI. There must be advice on whether medications that were stopped during the admission may require restarting.

STRATEGIES FOR SERVICE IMPROVEMENT

Following on from the NCEPOD report, there has been an increasing interest in developing regional AKI networks to work towards improving the management of AKI (www.aki.org.uk). Networks should develop and harmonise local pathways, set referral criteria for specialist renal input, and standardise best practice. Nationally there is currently much work centering around developing electronic alert systems for both primary and secondary care based on the new definitions of AKI in order to facilitate earlier recognition.[26] The introduction of an electronic alert system requires a supportive educational package such that an appropriate response is triggered. Furthermore, hospitals are encouraged to develop local guidelines for intravenous fluid therapy.

CONCLUSIONS

- The recognition of AKI should be improved by adopting the newly proposed definitions, based on relatively small rises in serum creatinine, and developing electronic alert systems.
- The prevention of AKI is essential and includes identification of patients with risk factors, optimisation of fluid balance, prompt treatment of sepsis, and avoidance of nephrotoxins.
- The underlying cause of AKI must always be sought, as rarer forms require specialist therapy.
- The severity and duration of AKI predicts progression to chronic kidney disease.
- Regional AKI networks should be developed to improve the management of AKI.

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SELF-ASSESSMENT QUESTIONS

1. A 31-year-old man who is normally fit and well develops fever and profuse bloody diarrhoea. He does not initially seek medical attention but after three days presents to his GP, who refers him acutely to the emergency department. On arrival, he is pale and appears short of breath. Blood results are as follows: haemoglobin 7 mg/dL, white cell count 15 x 10^9/L, platelets 15 x 10^9/L, urea 30 mmol/L, creatinine 557 µmol/L, potassium 6 mmol/L, sodium 144 mmol/L. Urine dip demonstrates 2+ protein but no blood or leucocytes.
Which ONE of the following investigations would be most useful in establishing the diagnosis as soon as possible?

A. Peripheral blood film.
B. Blood cultures.
C. Liver function tests.
D. Renal tract ultrasound.
E. Stool cultures.

2. A 78-year-old gentleman with type 2 diabetes and stage 4 chronic kidney disease (estimated glomerular filtration rate 28 ml/min/1.73 m^2) develops central crushing chest pain. An electrocardiogram (ECG) shows deep T-wave inversion in the lateral leads and ST depression inferiorly. A 12-hour troponin is elevated and a serial troponin confirms increasing values. He continues to have intermittent chest pains at rest despite medical management for non-ST-elevation MI. It is decided that a coronary angiogram should be performed.
Which ONE of the following strategies is advised to reduce his risk of contrast-induced AKI (CI-AKI)?

A. Prescribe oral N-acetylcysteine pre- and post-procedure.
B. Stop metformin pre- and post-procedure.
C. Prescribe intravenous 0.9% sodium chloride pre- and post-procedure.
D. Stop angiotensin-converting-enzyme (ACE)-inhibitor pre- and post-procedure.
E. Prescribe oral high dose statins pre- and post-procedure.

3. A 45-year-old man, previously fit and well, presents to the emergency department with a three-month history of fatigue, joint pains, night sweats, and weight loss. Blood pressure is 138/90 mm Hg. Blood results demonstrate haemoglobin 8.1 mg/dL, white cell count 15 x 10^9/L, platelets 600 x 10^9/L, sodium 142 mmol/L, potassium 5 mmol/L, urea 15 mmol/L, creatinine 150 µmol/L. The creatinine was last measured two months ago, when it was 78 µmol/L. Urine dipstick demonstrates 3+ blood and 3+ protein.
What is the most likely diagnosis?

A. Pyelonephritis.
B. Acute tubular necrosis.
C. Vasculitis.
D. Rheumatoid arthritis.
E. Interstitial nephritis.

4. A 72-year-old female with a history of chronic kidney disease (CKD) and who takes ramipril for hypertension presents to the emergency department 24 hours post-discharge following a laparoscopic cholecystectomy. She has a fever and abdominal pain. On examination her temperature is 39 °C, pulse rate 110/min, blood pressure 80/50 mm Hg, jugular venous pressure (JVP) not visible, lungs clear, and abdomen tender.
Which fluid do you choose for volume resuscitation?

A. 5% glucose.
B. Hartmann's solution.
C. 0.9% sodium chloride.
D. 6% hydroxyethyl starch solution.
E. 5% human albumin solution.

5. A 65-year-old man with type 2 diabetes, hypertension, and ischaemic heart disease develops diarrhoea and vomiting. He continues to take his medications, which include ramipril, simvastatin, metformin, furosemide, aspirin, and bisoprolol. On examination he is unwell, clammy, and short of breath. His jugular venous pressure is elevated to his earlobe. Blood pressure is 90/50 mm Hg. Auscultation of his chest reveals crackles to both mid-zones. Blood results are phoned through from the laboratory and demonstrate sodium 145 mmol/L, potassium 8 mmol/L, urea 50 mmol/L, creatinine 843 µmol/L.
What is the first step in management?

A. Administer 100 mg furosemide IV.
B. Administer 10 ml 10% calcium gluconate intravenously (IV).
C. Administer 10 ml 8.4% sodium bicarbonate IV.
D. Administer 50 ml 50% dextrose with six units of actrapid insulin IV.
E. Administer 5 mg salbutamol nebuliser.

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EDUCATION