INTRODUCTION

Acute kidney injury (AKI) is a common, life-threatening condition associated with poor outcomes. Current NHS expenditure on AKI and its consequences is greater than for prostate, bowel and lung cancer combined. There is evidence that many patients are not well managed and 20–30% of cases are potentially avoidable. Optimal care could save up to 12,000 lives a year and produce substantial financial savings. Clear clinical guidelines on the early identification and management of patients with AKI will help to inform the effective commissioning of care for these patients.

AKI may present in primary care or as acute admissions to hospital, but may also develop during hospital admission. Only a minority of AKI cases will reach specialist nephrology care. Many people with AKI are frail and elderly with complex co-morbidity, and present with acute illness.

Care of patients can be improved by doing the basics well. This includes:

- Early recognition of those at risk of AKI.
- Informing patients at risk of AKI and their carers when to temporarily discontinue ACE inhibitors (ACEi) angiotensin receptor blockers (ARB), diuretics and non-steroidal anti-inflammatory drugs (NSAID) during acute illness.
- Improved training and education of clinical teams responsible for their care.
- Hospitals must provide adequate systems and staffing to deliver high quality care, ensuring continuity of care and appropriate escalation to senior medical staff for assessment of complex cases.
- Agreed referral criteria for specialist nephrology input.
- Assessment of risk factors for AKI in all acutely ill patients. Risk scores, already in use in some patient groups at risk of AKI, need to be developed and validated for wider use.
- All patients admitted non-electively into hospital and all acutely ill patients in primary care will require assessment of their volume status, urinalysis and a medicines review. ACEi/ARB, NSAID should be withheld pending senior review within 12 hours.
- All patients admitted non-electively into hospital should also have baseline measurement of serum creatinine and electrolytes (including chloride), repeated within 24 hours. Urinalysis will help to identify the minority with intrinsic kidney disease that require early specialist assessment.

An international clinical practice guideline on AKI has recently been published (Kidney Disease Improving Global Outcomes – KDIGO). In the UK, NICE guidance on AKI is in preparation. This consensus statement makes recommendations on three aspects of AKI care which are not a major focus of the KDIGO and NICE work.

What is the role of fluid therapy in AKI?

Summary: Fluid therapy should be guided by repeated evaluation of volume status. A balanced salt solution should be the usual fluid for volume replacement.

- All hospitals must have fluid therapy guidelines for resuscitation, replacement and maintenance which will inform the timeliness of intervention, choice of fluid, and frequency of reassessment.
- Patients with AKI receiving intravenous (IV) fluid therapy require regular re-evaluation of volume status, daily weights, and regular monitoring of creatinine and electrolytes (including chloride and bicarbonate).
- Evaluation of volume status should be based on history, cumulative fluid balance and clinical examination (including pulse, blood pressure (BP), jugular venous pressure, capillary refilling, weight and postural change in pulse and BP).
- Clinical assessment of volume status is difficult and should be a focus for education and training of clinical staff.
- Central venous pressure (CVP) measurement does not have a role in the routine assessment of volume status in the ill patient at risk of AKI.
- Choice and prescription of maintenance IV fluids must be guided by a daily assessment of the patient’s water and electrolyte requirements.
- Crystalloid solutions are preferred to colloid.
• Balanced salt solutions should be the standard IV fluids for the correction of hypovolaemia.
• Research is required to evaluate and validate new techniques to assist with the clinical assessment of volume status.

**What is the role of e-alerts in AKI?**

**Summary:** Identification of AKI in both primary and secondary care should be facilitated through introduction of e-alert systems

• If early identification of AKI, defined by changes in serum creatinine concentration, is to be achieved and early treatment facilitated, e-alerts should prove valuable tools in primary and secondary care.
• E-alert systems should be introduced in the context of e-guidance on management of AKI, continuing education of clinical teams, and agreed care bundles.
• At present systems are being developed ad hoc. A national group should be established to develop agreed standards for e-alert systems recognising the need for some system-dependent local flexibility. Components of the system should include an agreed definition of AKI based on the KDIGO classification and a standardised methodology for derivation of baseline serum creatinine. We recommend use of an enzymatic serum creatinine assay with an IDMS-traceable calibration to enable standardisation.
• Healthcare providers employing e-alert systems must have robust arrangements to ensure appropriate and prompt responses with clear lines of accountability.
• We recommend audit and research to confirm that in addition to identification of AKI the use of e-alert systems improves outcomes.

**What is the role of biomarkers in AKI?**

**Summary:** It is premature to recommend the use of novel biomarkers of AKI in current clinical practice.

• Identification of accurate and early biomarkers may be a key to improving outcomes in AKI.
• Serum creatinine is an index of glomerular filtration and is therefore not an ideal biomarker for AKI.
• The ideal biomarker for AKI should provide timely diagnosis, have high diagnostic and prognostic accuracy, and allow assessment of response to treatment. Measurement of biomarkers should be affordable and reproducible.
• Novel markers of early kidney damage are being developed and evaluated, but their exact role in diagnosis and management in AKI remains unproven.
• Further research should evaluate the role of novel biomarkers in multicentre clinical intervention studies throughout the patient pathway. Future studies require better integration between laboratory scientists and clinicians.

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