British Journal of Hospital Medicine

Br. J. Hosp. Med. (Lond) **2026**; 87(2): 50919
https://doi.org/10.31083/BJHM50919

Review

Practical Acute Kidney Injury Care: Embedding the UK Kidney Association Summit Recommendations Across Hospital Settings

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Academic Editor: John Alcolado
Submitted: 26 June 2025 Revised: 3 September 2025 Accepted: 28 September 2025 Published: 11 February 2026

Abstract

Acute kidney injury (AKI) affects up to 20% of hospitalised patients and is associated with significant morbidity, mortality, and healthcare burden. Despite national guidelines, variability in recognition and management persists. This review bridges the UK Kidney Association (UKKA) AKI Summit recommendations with real-world National Health Service (NHS) clinical practice, summarising 24 key recommendations into core principles and translating them into practical guidance for clinicians across emergency, ward-based, critical care, and geriatric settings. Emphasis is placed on early identification, fluid and medication management, escalation to specialist teams, and discharge planning. We highlight implementation tools, including e-alert systems, care bundles, and standardised referral pathways. Finally, the article discusses barriers to consistent AKI care and proposes system-wide strategies to support education, coordination, and long-term follow-up. This practical review offers a setting-specific roadmap to improve patient outcomes and promote consistent, proactive care across the AKI continuum.

Keywords: acute kidney injury; patient safety; clinical decision-making; interdisciplinary communication; hospital units; health services; continuity of patient care; clinical decision support systems; patient discharge

1. Introduction

Acute kidney injury (AKI) is a common and serious complication among hospitalised patients, affecting an estimated 15–20% of admissions [1–3]. It is strongly associated with increased in-hospital mortality, prolonged length of stay, higher readmission rates, and significant healthcare costs. Despite improvements in awareness and recognition over the past decade, AKI continues to impose a heavy burden on patients and services across the National Health Service (NHS) [4].

In clinical practice, AKI often begins insidiously and may not cause symptoms until kidney function is significantly compromised. Early identification, therefore, relies heavily on biochemical markers and careful clinical observations. According to the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 clinical practice guideline for AKI, a diagnosis can be made based on an acute rise in serum creatinine ($\geq$26 µmol/L within 48 hours or $\geq$50% within 7 days) or a sustained reduction in urine output ($<$0.5 mL/kg/h for more than 6 hours) [5]. Common early indicators include oliguria, fluid imbalance, and biochemical abnormalities, for instance, hyperkalaemia or metabolic acidosis, although these may be subtle or absent in many cases. Clinical responses remain highly variable across institutions and departments, and the implementation of national guidelines into everyday practice is inconsistent [1]. This variation can lead to delays in diagnosis, missed opportunities for prevention, and fragmented post-discharge care. As Hsu and Hsu [6] note, the optimal approach to managing AKI survivors is yet to be defined, and more actionable tools are needed to support consistent and timely care delivery.

In 2024, the UK Kidney Association (UKKA) published the AKI National Summit Report, outlining 24 key recommendations for the detection, management, and follow-up of AKI across hospital and community settings. The National AKI Summit was held in September 2023, bringing together 120 multidisciplinary professionals, including representation from the Royal College of Physicians (RCP), the Society for Acute Medicine (SAM), the British Society for Heart Failure (BSH), the Intensive Care Society (ICS), British Geriatric Society (BGS) and others, ensuring cross-speciality and multidisciplinary representation.

The report calls for improved clinical engagement, integration of e-alert systems, multidisciplinary education, and structured care pathways to reduce avoidable harm and long-term renal complications [4]. Importantly, it urges a system-wide cultural shift towards earlier intervention, better continuity, and more patient-centred care.

This article aims to bridge the gap between these national recommendations and real-world clinical settings. While international guidelines such as KDIGO 2012 AKI guideline and the National Institute for Health and Care Excellence (NICE) AKI guideline issued in 2019, provide essential diagnostic and management frameworks, they of-

fer limited setting-specific operational guidance. In contrast, this review includes multidisciplinary, scenario-based strategies that address the complexities of implementation in different hospital environments. It first summarises the report's twenty-four recommendations into thematic core principles and then presents them as practical guidance tailored to specific inpatient settings, from the emergency department to intensive care units.

These core principles were derived through thematic grouping determined by the authors, based on the recurrence of topics across the recommendations, their relevance to frontline clinical decision-making, and their applicability in different care settings. Rather than reflecting a strict prioritisation, they serve as a pragmatic framework to support implementation and guide practice. Drawing on both recent literature and the UKKA AKI Summit Report, the article also highlights common barriers and proposes tools for improving consistency, communication, and patient outcomes throughout the AKI care continuum [3,7–10].

2. Core Principles of AKI Management

2.1 Early Recognition and Risk Stratification

Early recognition of AKI is crucial to prevent further deterioration of renal function and the patient's overall condition. Identifying individuals at risk allows clinicians to implement preventative measures before injury is established. AKI can progress rapidly, making early detection a decisive factor in patient outcomes. Delays in recognising AKI have been associated with increased morbidity, prolonged hospitalisation, higher mortality rates and worse clinical outcomes, especially in critically ill patients [3,8,11]. Patients with comorbidities such as heart failure, liver disease, diabetes, or chronic kidney disease (CKD) are particularly vulnerable and may deteriorate rapidly if early signs of AKI are missed [2].

AKI is defined by an increase in serum creatinine or a reduction in urine volume, with thresholds universally agreed upon in KDIGO guidelines. Specifically, these are an increase in serum creatinine by $\geq$0.3 mg/dL ($\geq$26.5 µmol/L) within 48 hours; or an increase in serum creatinine to more than 1.5 times baseline, presumed or known to have occurred over the past 7 days; or a urine volume $<$0.5 mL/kg/h for at least 6 hours. The classification of AKI according to KDIGO guidelines is outlined in Table 1 (Ref. [5]).

To support sensible identification, many NHS hospitals utilise electronic AKI alert systems that flag biochemical changes suggestive of kidney injury. Whilst e-alerts can prompt faster clinical action, their effectiveness depends on clinician engagement and their integration into meaningful workflows [10]. Overreliance on automated notifications without contextual interpretation may reduce utility and contribute to alert fatigue.

A comprehensive risk stratification strategy also includes the proactive identification of high-risk populations. This involves close monitoring of patients exposed to nephrotoxic medications, those with sepsis or undergoing major surgery, and individuals with a history of CKD. Recognising these risk factors at admission and throughout hospitalisation enables robust monitoring and preventive actions, such as maintaining hydration [12].

Routine assessment of serum creatinine and urine output remains fundamental to AKI surveillance. Daily biochemical monitoring and regular documentation of fluid balance are especially important in unstable or elderly patients, where small changes may reflect significant renal decline. Embedding these practices into ward routines fosters a 'culture of vigilance', meaning a habitual state of heightened awareness, proactive monitoring, and prompt response to early signs of AKI, ultimately supporting early intervention [1,4].

2.2 Fluid & Haemodynamic Optimisation

Effective fluid management plays a central role in the prevention and treatment of AKI. Accurately assessing volume status at the bedside is often challenging but remains a requirement in guiding intervention. Clinical assessment should include a combination of vital signs, capillary refill time, jugular venous pressure, and peripheral oedema. In selected cases, and when available, bedside ultrasound to evaluate inferior vena cava variability and pulmonary congestion can further support fluid balance. No single parameter is sufficient on its own, reinforcing the need for a holistic clinical approach [13].

Importantly, not all AKI cases benefit from fluid administration. In patients with heart failure, AKI may be driven by volume overload and consequent renal venous congestion, rather than hypovolaemia. Venous congestion exacerbates renal dysfunction through elevated venous pressures and impaired renal perfusion [14]. In such cases, administering additional fluids may worsen congestion and contribute to further renal injury.

Instead, decongestive strategies using loop diuretics are often indicated in heart failure-associated AKI and may improve renal function by relieving venous congestion and restoring perfusion gradients [15]. Contrary to longstanding concerns, loop diuretics are not inherently nephrotoxic. Their suitable use in the context of fluid overload is safe and effective [16] and may be renal-protective in selected cases of AKI associated with decompensated heart failure. The misconception that diuretics routinely cause or worsen AKI in heart failure can lead to inappropriate under-treatment and delayed clinical improvement.

Fluid resuscitation should therefore be individualised based on the cause of AKI and volume status. In patients with sepsis or hypotension, adequate fluid administration will improve organ perfusion [13]. This includes scenarios where heart failure is a co-morbid factor and not the cause of an AKI. However, it is important to remember that the target is euvolaemia. Excessive or indiscriminate fluid use

Table 1. KDIGO classification of AKI.

Stage	Serum creatinine	Urine output
1	1.5–1.9 times baseline OR ≥0.3 mg/dL (≥26.5 µmol/L) increase	<0.5 mL/kg/h for 6–12 hours
2	2.0–2.9 times baseline	<0.5 mL/kg/h for ≥12 hours
3	3.0 times baseline OR Increase in serum creatinine to ≥4.0 mg/dL (≥353.6 µmol/L) OR Initiation of renal replacement therapy OR In patients <18 years, decrease in eGFR to <35 mL/min/1.73 m^2	<0.3 mg/kg/h for ≥24 hours OR Anuria for ≥12 hours

Note: Reproduced with permission from KDIGO [5]. Abbreviations: KDIGO, Kidney Disease: Improving Global Outcomes; AKI, acute kidney injury; eGFR, estimated glomerular filtration rate.

may worsen outcomes through iatrogenic volume overload [17].

The type of intravenous fluid also requires careful consideration. Balanced crystalloids are preferred over 0.9% saline for most patients at risk of AKI, due to their lower chloride content and reduced risk of hyperchloremic acidosis and renal vasoconstriction [18]. Colloid solutions, such as hydroxyethyl starch, are generally avoided because of their association with increased AKI risk in critically ill patients [19].

Clinicians should be mindful of both the type and volume of fluids administered, and the fluid strategy should be re-evaluated frequently during hospitalisation to ensure ongoing suitability.

2.3 Medication Management

2.3.1 Nephrotoxin Avoidance

Medication-related kidney injury is a well-recognised contributor to AKI, especially in hospitalised patients with complex comorbidities. Timely and thorough medication review is therefore essential to mitigate avoidable harm [20].

Commonly implicated nephrotoxic agents include aminoglycosides and non-steroidal anti-inflammatory drugs (NSAIDs), which should be reviewed and, when appropriate, temporarily discontinued in patients with suspected or confirmed AKI. It is also important to recognise medications that are not inherently nephrotoxic but may exacerbate renal dysfunction when renal perfusion is compromised. These include agents that affect renal haemodynamic or fluid balance, such as antihypertensives [9]. Renin-angiotensin-aldosterone system inhibitors (RAASi) are discussed separately below. Table 2 (Ref. [1,5,9]) shows medicines to be avoided or that need dose adjustment in reduced estimated glomerular filtration rate (eGFR).

Regarding iodinated contrast, the UKKA Summit Report and professional guidelines such as those from NICE, the Royal College of Emergency Medicine (RCEM) and the Royal College of Radiologists (RCR) emphasise that the suggested risk of association to AKI is often overestimated, particularly with modern low- or iso-osmolar agents [1,21]. Studies consistently show that the occurrence of AKI in patients undergoing contrast imaging is more strongly associated with underlying illness than with the contrast media itself [22]. Decisions regarding contrast use should be guided by clinical urgency and risk–benefit analysis; delays in critical imaging due to concerns over potential AKI may result in poorer outcomes. Routine use of preventive strategies such as hydration may be reasonable for elective imaging, particularly in high-risk patients, but agents like N-acetylcysteine and sodium bicarbonate have not demonstrated benefit in large meta-analyses and are no longer recommended [23].

2.3.2 Dose Adjustments

In addition to the avoidance or temporary suspension of nephrotoxic agents, special attention must be paid to medications that may accumulate during AKI due to reduced renal clearance. These include insulin, gabapentin, opioids (e.g., morphine), digoxin, metformin, and renally cleared antibiotics such as aminoglycosides or vancomycin. Without proper dose adjustment, these agents may lead to serious complications such as hypoglycaemia, neurotoxicity, respiratory depression, lactic acidosis, or arrhythmias. Daily medication review, preferably by pharmacists, should be embedded in AKI care, particularly in older patients or those with polypharmacy. Recognising the altered pharmacokinetics and pharmacodynamics in AKI is crucial for patient safety and quality of care.

2.3.3 Renin-Angiotensin-Aldosterone System Inhibitors

RAASi, such as angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs), play a central role in the long-term management of patients with CKD, diabetes, and heart failure. They

Table 2. Medications to avoid or use with caution in AKI.

Medication/Class	Mechanism/Risk in AKI	Recommendation
NSAIDs (e.g., ibuprofen, diclofenac)	Inhibit prostaglandin synthesis → afferent arteriolar constriction → reduced renal perfusion	Avoid; reassess analgesic strategy
Aminoglycosides (e.g., gentamicin)	Tubular toxicity; risk of accumulation	Avoid if possible; monitor levels closely. If resumed, ensure renal function is stable and monitor trough level carefully; adjust dose for underlying eGFR
Amphotericin B (conventional)	Direct tubular toxicity; induces hypokalaemia	Prefer lipid or liposomal formulations; ensure close monitoring
Iodinated contrast agents	Risk of contrast-associated nephropathy (higher risk of AKI in patients with underlying CKD)	Use low-osmolar/non-ionic agents; pre-hydration is advised if the patient is hypovolaemic
Vancomycin	Acute tubular injury; nephrotoxic at high levels; additive risk with other agents	Monitor trough levels and renal function; adjust dose for underlying eGFR
Acyclovir (IV)/Valganciclovir	Crystalline nephropathy; risk of tubular obstruction	Ensure adequate hydration; adjust dose
Trimethoprim/Co-trimoxazole	Hyperkalaemia; potential for AKI via AIN or crystalluria	Avoid or adjust dose; monitor electrolytes
Methotrexate	Crystalline nephropathy; risk of accumulation in renal impairment	Avoid in AKI. Reinitiate only after renal function returns to baseline
Bisphosphonates (IV)	Risk of acute tubular necrosis if infused rapidly	Administer slowly; avoid in established AKI
Digoxin	Narrow therapeutic window; risk of toxicity in renal impairment	Monitor plasma levels; dose adjustment required
Lithium	Risk of accumulation; chronic tubulointerstitial nephropathy	Suspend during AKI; reintroduce once eGFR returns to baseline and lithium levels are within therapeutic range. Monitor levels closely
Calcineurin inhibitors (e.g., tacrolimus, ciclosporin)	Vasoconstriction of afferent arteriole; risk of AKI	Monitor trough levels and renal function closely

Note: Based on information from Perazella and Rosner [9], KDIGO [5] and NICE [1].

Abbreviations: AIN, acute interstitial nephritis; AKI, acute kidney injury; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; IV, intravenous; NSAIDs, non-steroidal anti-inflammatory drugs.

provide substantial long-term benefits in terms of morbidity and mortality [24]. However, they are frequently withheld inappropriately during episodes of AKI due to concerns over nephrotoxicity. The UKKA AKI Summit Report specifically discourages the routine labelling of RAASi as nephrotoxic, noting that they do not cause intrinsic kidney injury. However, it is acknowledged that they may lead to transient rises in creatinine due to changes in glomerular haemodynamic [4].

Temporary suspension of RAASi may be applicable in specific clinical contexts such as sepsis, hypotension, or hyperkalaemia [25]. When suspended, the indication, rationale, and plan for reinitiation should be clearly documented and communicated during transitions of care, including at discharge. Reinitiation should occur as soon as clinically safe, particularly in patients with heart failure with reduced ejection fraction (HFrEF), where premature or prolonged suspension is associated with increased risk of mortality and hospital readmission [26].

Hyperkalaemia, common in AKI and often a consequence of RAASi use [24], must be recognised and managed promptly as a medical emergency. While temporary cessation of potassium-retaining agents may be required, including mineralocorticoid receptor antagonists (MRAs), clinicians should not forgo opportunities to optimise long-term care. The development of the novel potassium binders patiromer and sodium zirconium cyclosilicate (SZC) has transformed the management of moderate, asymptomatic hyperkalaemia. These agents enable continuation or reinitiation of RAASi in certain patients [26]. Their use should be incorporated into local protocols, and accessible prescribing pathways established to facilitate suitable intervention.

Clinical reinitiation of RAASi should be guided by patient-specific factors, including stable blood pressure, improving renal function, and controlled potassium levels, typically <5.5 mmol/L. If potassium levels are above these figures, a potassium binder can be started on a maintenance dose to allow for RAASi use. Close follow-up is essential after restarting therapy, ideally in coordination with nephrology or heart failure services. Evidence from trials such as the Patiromer for the Management of Hyperkalaemia in Subjects Receiving RAASi for Heart Failure (DIAMOND) trial reinforces the prognostic benefit of reintroducing RAASi in eligible patients with HFrEF, particularly when potassium-lowering strategies are employed concurrently [27].

2.4 Escalation & Specialist Input

Escalation of care is a critical component in the effective management of AKI. Involvement of nephrology and intensive care specialist teams can significantly influence patient outcomes [28]. However, delays in referral remain a recurrent issue, often contributing to avoidable deterioration [4].

Clinicians should be encouraged to initiate early discussions with nephrology when faced with diagnostic uncertainty, worsening renal function despite appropriate management, persistent electrolyte disturbances such as hyperkalaemia or metabolic acidosis, or suspected intrinsic renal disease [29]. AKI stage progression or persistent oliguria despite fluid resuscitation may also warrant nephrology input. Local protocols, as recommended by the UKKA Summit Report, should clearly define these referral triggers, including failure of renal recovery after several days, AKI stage ≥ 2 persisting beyond 48 hours, or clinical concern for underlying glomerular or systemic disease.

Escalation to critical care services is equally important in patients with haemodynamic instability, severe metabolic derangements, progressive multi-organ dysfunction, or anticipated need for renal replacement therapy (RRT). These decisions should not be delayed until irreversible decline has occurred. Structured multidisciplinary discussions involving renal, critical care, and general medicine or surgical speciality teams can support joint decision-making and improve consistency of care.

The concept of proactive escalation is also emphasised in the Summit Report, where deteriorating patients are identified early, even before conventional biochemical or clinical thresholds are met. Promoting open communication across care levels and embedding escalation plans into electronic patient records visible to all team members can facilitate a rapid response. AKI specialist nurses, where available, play a key role in reinforcing escalation protocols and ensuring ongoing reviews [4].

3. AKI in Different Clinical Settings: Practical Guidance for Clinicians

3.1 Emergency Department and Acute Medical Unit Perspective

More than half of AKI cases are diagnosed in the emergency department (ED) or Acute Medical Unit, making early recognition and prompt management in this setting crucial. In these settings, clinicians often make rapid decisions with limited background information [4]. Recognising patients at risk of AKI early is crucial to prevent clinical deterioration and progression to severe AKI. In older or acutely unwell patients, even modest changes in serum creatinine or urine output can be significant and should not be dismissed. Early documentation of urine output and a focus on dynamic trends, rather than isolated values, is key [1,4].

The ED environment presents unique challenges to AKI management. Time pressure, diagnostic uncertainty, and clinical instability demand fast yet thoughtful decisions. Volume assessment can be particularly complex in patients with coexisting heart failure or sepsis. Balanced crystalloids are recommended for fluid resuscitation in hypovolaemia [18], but fluid overload must be avoided. Medication reconciliation, including temporary suspension of nephrotoxins, should be prioritised early. Bedside tools,

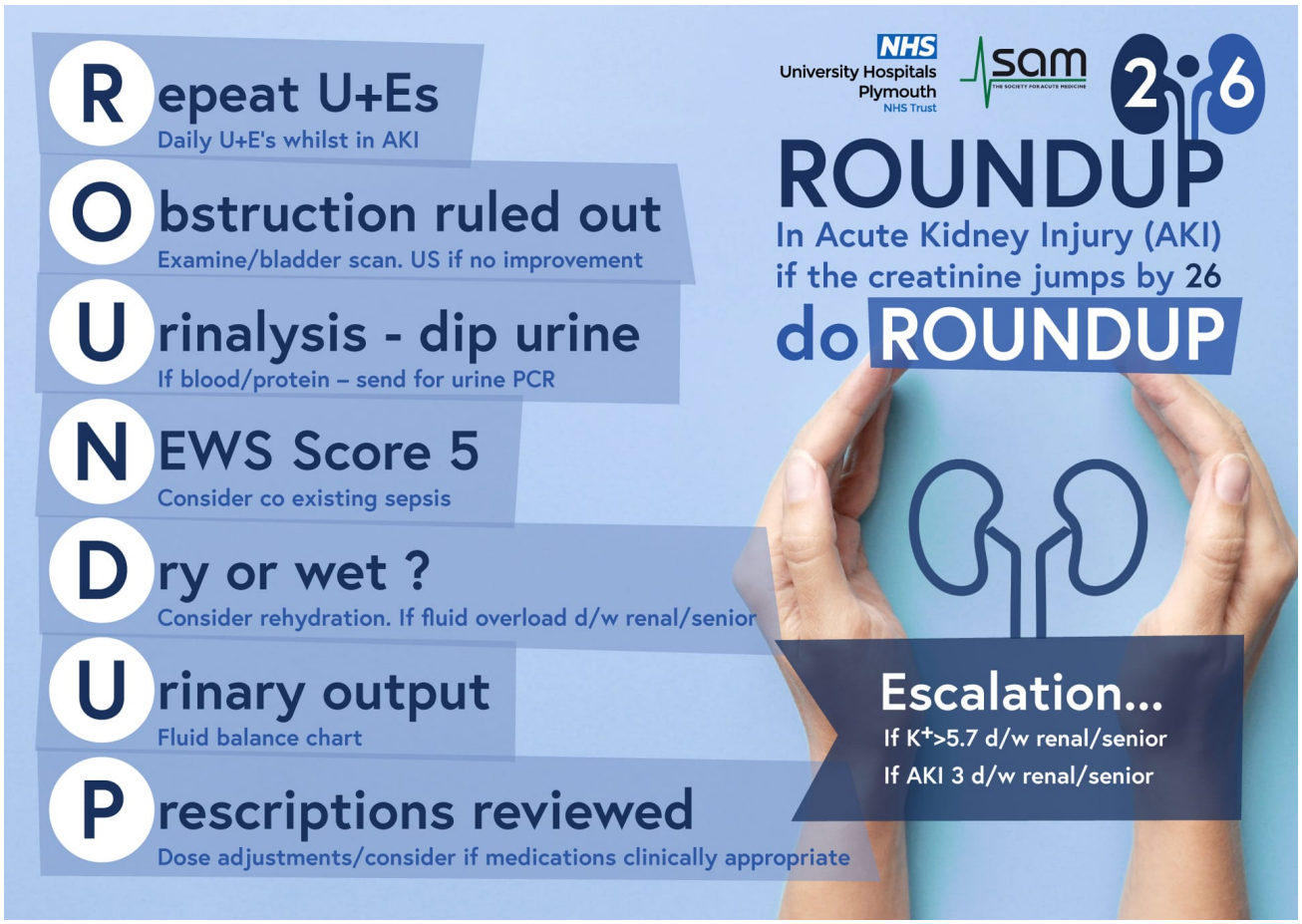

Fig. 1. ROUND-UP 26 bundle framework. Note: Reproduced with permission from The Society for Acute Medicine (SAM) Acute Kidney Injury SIG [30]. Abbreviations: d/w, discuss with; K^+, potassium; NEWS, national early warning score; PCR, protein-to-creatinine ratio; U+Es, urea and electrolytes; US, ultrasound; NHS, National Health Service.

such as bladder scanners and urine output charts, are valuable but often underutilised due to workflow constraints [1].

To improve consistency and reliability in the early management of AKI, several hospitals have adopted structured clinical tools such as acute care bundles. These aim to standardise best practices during the initial hours of care and promote interventions across multidisciplinary teams. One example is the ROUND-UP 26 bundle (Fig. 1 Ref. [30]), which supports clinicians in reviewing potential causes, optimising volume status, undertaking investigations, reviewing nephrotoxins, documenting findings, monitoring urine output, and planning escalation. The UKKA AKI Summit Report supports the use of such structured approaches to reduce variability and enhance care delivery.

The ROUND-UP 26 bundle is typically initiated upon detection of an AKI e-alert, a confirmed rise in serum creatinine by $\geq$26 µmol/L within 48 hours, or when clinical suspicion arises based on signs such as oliguria, hypotension, or fluid imbalance. Each step is designed to be implemented within the early hours of recognition. Reviewing medications and assessing volume status should occur immediately, alongside early actions such as repeat serum urea and electrolytes (U+Es), excluding obstruction via bladder scanning or catheterisation, and performing a urine dipstick. If blood or protein is detected on urinalysis, a sample should be sent for urinary protein-to-creatinine ratio (uPCR) to support further diagnostic assessment. Escalation of care should be considered in parallel, particularly in patients with AKI stage 3, potassium $\geq$5.7 mmol/L, or clinical instability. Monitoring of urine output is an ongoing task, and documentation of AKI stage and investigations should follow promptly. The bundle functions as a dynamic checklist that reinforces shared responsibility across teams during the initial phase of AKI management.

Disposition decisions from the ED should be guided by AKI severity, the presumed aetiology, the patient's response to initial treatment, and the presence of physiological instability or multi-organ involvement. Referral to nephrology is indicated for unclear diagnoses, progression despite supportive care, or significant electrolyte abnormalities. Early consultation with critical care should be considered for patients with haemodynamic instability, severe metabolic derangement, or suspected need for renal replacement therapy [1].

3.2 General Medicine & Surgery Wards

AKI is common and associated with poor outcomes across all clinical settings. Awareness of risk and effective management are crucial for healthcare professionals across both medical and surgical specialities. On general wards, patients may develop AKI either as a progression of illness presenting in the emergency department or as a new complication during hospitalisation. Whilst the risk of AKI in medical patients is well known, surgical patients face a higher risk of AKI than is perhaps acknowledged demonstrated an AKI prevalence exceeding 10% for inpatient episodes with a primary diagnosis of pancreatitis, cholecystitis, trauma, or neck of femur fracture, with associated high inpatient mortality [31]. It is therefore fundamental that hospital teams, including surgeons, ward staff, and theatre colleagues, receive structured education in AKI risk recognition and response.

Daily monitoring of renal function is vital for early identification of AKI, especially in patients receiving nephrotoxic medications or those undergoing procedures associated with haemodynamic shifts. Individuals who have recently had surgery, are on diuretics, or present with risk factors such as sepsis or dehydration require close observation. Deterioration in renal function may be subtle, and even small increases in serum creatinine or minor reductions in urine output must be recognised and reviewed.

Routine medication review should be embedded into ward practice as part of AKI care. The UKKA AKI Summit Report highlights the importance of systematically identifying and adjusting nephrotoxic agents or those with renal function-dependent dosing, with pharmacists playing an active role in multidisciplinary teams. Hospital guidelines should provide clear recommendations for pausing or modifying medications in response to renal function trends.

Managing fluid balance in ward-based AKI demands nuanced clinical judgement. Overhydration may lead to pulmonary oedema or worsening heart failure, whereas underhydration may perpetuate hypoperfusion and renal injury. Accurate documentation of fluid input and output, daily body weights, and frequent bedside assessments should underpin decisions regarding intravenous fluids or diuretics.

Persistent AKI or failure to respond to basic supportive measures should prompt escalation to senior clinicians and nephrology input when applicable. Vigilance is warranted when features suggest intrinsic renal disease, such as haematuria, proteinuria, or systemic signs like rash and arthralgia. Encouraging proactive AKI management through ongoing education of ward teams is essential to reduce variability in care and improve outcomes [1,4].

3.3 Geriatrics & Frailty Care

Older adults represent a high-risk population for the development and progression of AKI, often due to multiple long-term conditions, age-related physiological changes, polypharmacy, and frailty [32]. In this group, AKI is frequently under-recognised and may present atypically, with non-specific symptoms such as lethargy, reduced oral intake, or functional decline, rather than overt signs of volume depletion or oliguria [4].

Baseline renal function in older patients may be difficult to assess due to low muscle mass and, therefore, low serum creatinine levels, which can mask significant kidney dysfunction. Clinical teams must interpret laboratory results with caution and prioritise trend monitoring over isolated values. In selected cases, measurement of cystatin C should be considered. It is a validated alternative to creatinine for estimating glomerular filtration rate (eGFR), is less influenced by muscle mass, and has been shown to better predict outcomes in older or frail patients [2,8,33]. In practice, requesting cystatin C may be most useful when renal function appears disproportionately preserved in the context of multimorbidity, sarcopenia, metabolic abnormalities such as acidosis or unexplained deterioration, and the result can guide safer drug dosing or strengthen the case for nephrology referral. However, availability across NHS laboratories remains limited, so its use in the acute setting will differ between localities. In addition, urine output monitoring may be challenging in patients with incontinence or cognitive impairment, further complicating sensible diagnosis.

Polypharmacy is a major contributor to AKI in frail older adults. The regular use of medications such as NSAIDs, diuretics, RAASi, and certain antibiotics increases the risk of worsening renal function, particularly during intercurrent illness or following changes in hydration status. Medication reviews should be conducted proactively, with particular attention to interactions and dose adjustments in the context of renal function. The UKKA Summit strongly encourages active involvement of pharmacists in multidisciplinary geriatric care to support these reviews [4].

Fluid management in this population requires special care. Both hypovolaemia and fluid overload can be poorly tolerated in frail individuals, leading to increased risk of delirium, falls, heart failure exacerbation, or worsening AKI. Oral hydration should be prioritised where feasible, while intravenous fluid administration must be regularly reassessed to avoid harm.

Clinicians must also recognise that AKI may signal broader deterioration in frail individuals. This requires a shift from purely organ-focused care to holistic assessment, including advanced care planning and goals-of-care discussions where relevant. Close liaison with primary care, geriatricians, and nephrology services is recommended, especially in cases of recurrent AKI, uncertain prognosis, or incomplete renal recovery.

3.4 Intensive Care Unit Approach

Patients admitted to intensive care units (ICUs) represent the most severe spectrum of AKI, often in the context of multi-organ dysfunction, sepsis, or haemodynamic instability. AKI in critically ill patients is associated with high morbidity, prolonged ICU stays, and increased mortality, and often reflects a combination of systemic insults, including hypoperfusion, inflammation, and nephrotoxic exposure [4,8].

In the ICU setting, routine evaluation to support early recognition of AKI includes hourly urine output monitoring with continuous fluid balance evaluation, and frequent serum creatinine measurements [34].

Management should prioritise individualised optimisation of haemodynamic status using vasopressors when needed to maintain adequate renal perfusion, alongside careful fluid administration [35]. Over-resuscitation must be avoided, particularly in patients with capillary leak, acute respiratory distress syndrome (ARDS), or pre-existing heart failure [17].

Medication reconciliation is crucial in ICU AKI patients, especially during transitions of care and when renal function changes rapidly, such as during AKI recovery [4]. Dosing adjustments for renally-excreted medications should be made promptly, and nephrotoxic drugs should be avoided or minimised whenever possible [5].

RRT should be considered in patients with persistent or severe metabolic complications, including refractory acidosis, fluid overload unresponsive to diuretics, refractory hyperkalaemia, or overt uraemic complications. The decision to initiate RRT should be based on the overall clinical picture rather than any single biochemical threshold. Where available, early nephrology involvement is recommended to support modality selection, timing, and ongoing care coordination [8].

Finally, as noted in the UKKA Summit Report, recovery from AKI in ICU patients often extends into the post-critical care phase. Structured follow-up after ICU discharge is vital to detect persistent dysfunction, guide medication reintroduction, and support long-term renal and cardiovascular health.

4. Discharge Planning & Post-AKI Follow-Up

Recovery from AKI does not end at hospital discharge. Up to one-third of patients experience incomplete renal recovery, and many are at increased risk of recurrent AKI, CKD, cardiovascular complications, and hospital readmission in the months following the index event [4]. In a UK cohort study, 26.6% of 90-day readmissions following hospitalisation with AKI (in patients with baseline CKD) were due to acute pulmonary oedema, compared to just 4.0% in those without AKI and normal renal function [7].

Despite these risks, post-AKI care is often fragmented or overlooked entirely. The transition from hospital to community settings represents a critical opportunity to consolidate renal recovery and prevent long-term sequelae. Structured discharge planning and well-defined follow-up pathways are essential components of high-quality AKI care [4].

Before discharge, renal function should be reviewed to determine whether the patient's kidney function has returned to baseline or remains impaired. Persistent abnormalities may require further evaluation and will warrant early follow-up. The Royal College of General Practitioners (RCGP) AKI toolkit includes guidance on when follow-up should occur following discharge after AKI. This is stratified according to AKI severity, renal recovery, and comorbid risk factors. Renal recovery is defined by serum creatinine levels at discharge; good recovery corresponds to $\leq$25% above baseline, moderate to more than 25 to 50%, and poor to $>$50% [36]. A summary of the recommendations on post-discharge follow-up is found in Table 3 (Ref. [36]).

Medication plans must be carefully reviewed to ensure reinitiation of therapies withheld during the AKI episode, ideally before hospital discharge. If reinitiation is to occur post-discharge, the rationale for temporary suspension and clear criteria for reintroduction must be documented in the discharge summary and communicated to patients and primary care teams. Ensuring shared decision-making and well-defined clinical responsibilities during care transitions can significantly improve patient outcomes.

The decision to reintroduce RAASi or diuretics following AKI in patients with heart failure should be based on a careful clinical assessment. Important considerations include resolution of haemodynamic instability, serum potassium returning to acceptable levels (typically $<$5.5 mmol/L), and evidence of improving or stable renal function. Clinical signs of congestion should be reassessed, and the benefit of reintroducing guideline-directed medical therapy should be weighed against any residual risk. In one study, the long-term benefit of restarting versus remaining off RAASi was compared in 10,000 patients with AKI 2 or 3. Here, discontinuing RAASi was associated with a higher risk of death or cardiovascular events, but not associated with any greater risk of AKI recurrence [37]. Where uncertainty exists, input from cardiology or nephrology specialists is recommended [1].

Education for patients and caregivers plays a key role. Individuals should be counselled on the importance of hydration, avoidance of over-the-counter nephrotoxins (such as NSAIDs), and early recognition of symptoms that may indicate renal decline, including fatigue, swelling, or decreased urine output. In selected settings, "sick day rules" for temporarily holding medications during acute illness can be used after AKI, although the evidence base supporting them is limited [4].

The decision to refer patients for outpatient nephrology follow-up should be individualised. Those with persistent renal dysfunction, underlying CKD, proteinuria, re-

Table 3. Summary of recommendations on post-discharge follow-up.

Clinical context at point of discharge	Suggested review
Heart failure + poor kidney recovery	3 days
Heart failure + mod/good kidney recovery No other significant factors + poor kidney recovery Significant risk factor + moderate kidney recovery	1–2 weeks
No significant risk factor + moderate kidney recovery Significant risk factor + good kidney recovery	1 month
No significant risk factor + good kidney recovery	3 months

Note: Definitions adapted with permission from Royal College of General Practitioners [36]: Good kidney recovery: serum creatinine $\leq$25% above baseline; Moderate kidney recovery: serum creatinine $>$25% and $\leq$50% above baseline; Poor recovery: serum creatinine $>$50% above baseline. The colours indicated the differences in time to follow-up, from more urgent to less urgent.

current AKI episodes, or other indicators of high risk, such as stage 3 AKI or partial renal recovery at discharge, are likely to benefit from specialist follow-up within 1 to 3 months. Conversely, patients with full renal recovery following stage 1 AKI and no residual abnormalities may be safely followed in primary care, provided they receive proper guidance. This includes repeat renal function testing, medication review, re-initiation of RAASi where applicable, and documentation of "sick day rules" [7].

Clear documentation and effective discharge communication are essential to reduce fragmentation of care and support continuity. Structured discharge summaries should outline AKI stage and trajectory, key medication decisions, recovery status, and indications for re-referral. Embedding such plans into electronic records can reduce variation and promote safer long-term renal care. Tables 4,5 provide checklists of information to include in the discharge summary and of actions that should be taken prior to discharge, respectively.

5. Implementing the UKKA Recommendations in NHS Hospitals

While national guidance provides an essential foundation for AKI prevention and management, its true impact depends on effective local implementation. Translating the UKKA AKI Summit recommendations into routine clinical practice requires an organisational commitment to staff education, standardisation of care processes, and the reduction of system-level barriers [4].

Electronic AKI alert systems are widely used across NHS hospitals. While they can support earlier recognition and intervention, alerts that are poorly designed or lack clinical context can lead to alert fatigue, desensitisation, and missed opportunities for intervention. To be impactful, these systems must go beyond basic notifications and be paired with clear escalation protocols, decision-support tools, and access to real-time clinical input [38]. Practical adaptations include providing contextual data such as baseline renal function trends, filtering alerts by AKI stage or patient risk profile, and integrating alert reviews into multidisciplinary team meetings or safety huddles. These strategies help preserve clinician engagement, reduce unnecessary alerts, and ensure that automated systems contribute meaningfully to clinical care.

AKI care bundles are recommended as structured tools to support early recognition and standardised management. However, unlike the nationally adopted "Sepsis Six" pathway [39], AKI bundles vary considerably across institutions. For doctors in training who frequently rotate between hospitals, this can impede learning, and for hospitals, this can make AKI teaching more onerous than would be necessary if a nationally agreed bundle were used. Prominent AKI bundles, which are used across multiple hospital sites in the UK, include ROUND-UP 26, STOP-AKI and national guidance on the content of these bundles, mirroring the approach of Sepsis Six, may be adopted in the future.

Education and training are pivotal. All staff who interact with acutely unwell patients must be equipped to identify AKI risk factors, interpret biochemical data, and initiate basic supportive interventions. This includes doctors in training, nurses, pharmacists and allied health professionals. The UKKA Summit Report highlights the value of cross-disciplinary education programmes that reinforce shared responsibility for renal safety across departments [4].

Table 4. Recommended details for inclusion in the discharge summary.

To include in the discharge summary
☐ AKI stage during admission and whether full, partial or no recovery occurred
☐ Baseline and discharge kidney function (include creatinine and eGFR trends)
☐ Cause of AKI, if identified (e.g., sepsis, hypovolaemia, nephrotoxin exposure)
☐ Relevant urinalysis finding
☐ Relevant scans performed (e.g., US/CT KUB)
☐ Medication changes (what was stopped, started, or dose-adjusted)
☐ RAASi/MRA/diuretic suspension and reinitiation plan, including rationale and criteria
☐ Need for follow-up blood tests (U+Es) and suggested timing
☐ Monitoring plan for comorbidities (e.g., heart failure, diabetes)
☐ Sick day rules explanation and documentation
☐ Patient education provided (hydration advice, NSAID avoidance, etc.)
☐ Follow-up plan (primary care vs nephrology referral), and timeline

Abbreviations: AKI, acute kidney injury; CT, computed tomography; eGFR, estimated glomerular filtration rate; KUB, kidneys, ureters and bladder; MRA, mineralocorticoid receptor antagonist; NSAID, non-steroidal anti-inflammatory drug; RAASi, renin-angiotensin-aldosterone system inhibitors; U+Es, urea and electrolytes; US, ultrasound.

Table 5. Pre-discharge actions.

To ensure before discharge
☐ Renal function reviewed within 48 hours of discharge
☐ Clear documentation of AKI episode and recovery status
☐ Decision about RAASi/other medications reinitiation documented and communicated
☐ Discharge summary sent to GP with action points clearly stated
☐ Patient understands the implications of AKI and signs/symptoms to monitor
☐ Plan for post-discharge blood tests arranged
☐ If appropriate, nephrology referral initiated
☐ Pharmacy review completed

Abbreviations: AKI, acute kidney injury; GP, general practitioner; RAASi, renin-angiotensin-aldosterone system inhibitors.

Standardised AKI care pathways can help reduce variability in clinical responses and streamline care escalation. These should include clear criteria for nephrology referral, guidance on fluid and medication management, and discharge planning protocols. Hospitals are encouraged to audit adherence to these pathways and to embed them into electronic health records wherever possible [1].

Finally, barriers to implementation must be recognised and addressed. These include limited nephrology coverage, inconsistent follow-up systems, or lack of dedicated AKI leadership. Appointing local AKI champions or specialist nurses can facilitate better coordination, bridge communication gaps, and support continuous quality improvement initiatives. These professionals are usually experienced clinicians, such as consultants, registrars or senior nurses, who take responsibility for promoting renal safety, ensuring adherence to AKI care pathways, supporting staff education, and acting as the main point of contact for complex or escalating cases. Their involvement helps strengthen local leadership and reinforce consistent renal safety practices across departments.

6. Conclusion

AKI remains a major challenge across hospital settings. Improving outcomes depends on early recognition, careful fluid and medication management, timely escalation, and coordinated multidisciplinary care. Structured discharge planning, follow-up, and patient education further reduce the risk of recurrence and long-term complications. Embedding these principles into everyday practice requires institutional commitment, standardised pathways, and sustained education to foster a culture of renal vigilance across the NHS.

Key Points

- Early recognition and prompt intervention are the foundation of effective AKI care.
- Individualised fluid management with frequent reviews avoids both dehydration and fluid overload.
- Regular medication reviews help prevent injury and support renal recovery.
- Escalate care promptly in patients with persistent, worsening, or severe AKI.

- Structured discharge planning and post-AKI follow-up are essential to reduce readmissions and progression to CKD, and to reinitiate long-term therapies stopped during AKI.
- Education and system-wide coordination remain pivotal to achieving better outcomes in AKI management and must be supported by institutional leadership.

Availability of Data and Materials

Not applicable.

Author Contributions

DG and BDJ contributed to the conceptualisation of the study. HMLeM was responsible for the first and final drafts, as well as data acquisition. DG and BDJ provided editorial input and manuscript revisions. All authors contributed to the important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

Acknowledgement

The authors would like to thank colleagues within the Renal Medicine Department at Salford Royal Hospital, as well as members of the UKKA AKI Special Interest Group, for their collaborative efforts in advancing AKI care across the UK.

Funding

This research received no external funding.

Conflict of Interest

DG and BDJ are members of the UKKA AKI SIG. DG has received consultancy fees and speaker fees from AstraZeneca, who produce sodium zirconium cyclosilicate, which is mentioned in this review. HMLeM declares no conflicts of interest.

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