



GUIDELINES AND AUDIT
IMPLEMENTATION NETWORK

Northern Ireland Guidelines for Acute Kidney Injury

**This document is a revision of the original Northern Ireland Acute Kidney Injury
Guidelines developed by GAIN in 2010**

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Introduction

Acute kidney injury (AKI) is a common clinical problem, expensive to manage and associated with a high mortality. Early recognition of individuals at risk of AKI, accurate clinical assessment of patients with kidney injury and avoidance of further nephrotoxic insults can help to prevent or reverse AKI.

Normal kidney function depends on having an adequate blood pressure and fluid volume to perfuse the kidneys. The kidney itself must have a sufficient number of intact functional nephrons (glomeruli and tubules) to achieve normal glomerular filtration and electrolyte balance. Finally there must be free drainage of the urinary tract.

The majority of AKI developing in the community is due a pre-renal state (90% cases), typically hypotension associated with sepsis and/or fluid depletion (e.g. vomiting or diarrhoea) or sepsis. Hypovolaemia and hypotension compromise the normal perfusion of the kidney. This problem can be further exacerbated by commonly prescribed drugs e.g. ACE inhibitors or NSAIDs that impair how the kidneys respond to hypotension. AKI, developing in this community setting will often respond to fluid replacement and temporary withdrawal of drugs that adversely affect kidney function. It is important to avoid further nephrotoxins, such as radiocontrast and high dose aminoglycoside, where possible.

In hospitalized patients this “volume sensitive” or “pre-renal” AKI often precedes the development of AKI characterized by pathological damage to kidney tissues. Once established, this more severe form of AKI takes much longer to resolve and will not respond to vigorous intravenous fluid volume replacement. This “renal” AKI (also known as acute tubular necrosis or ATN) is associated with prolonged hospitalization and higher mortality both during the hospital stay and following discharge. Over 75% of all AKI in hospitalized patients is due to the volume sensitive and ATN forms of AKI. Of the remaining causes of AKI, urinary tract obstruction is the most important to rapidly exclude, as early drainage of obstructed kidneys improves longer term renal function.

Clinically, AKI is easily recognised by decreasing urine volume (oliguria or anuria) and a rise in serum creatinine. AKI, if unrecognised and allowed to worsen, will result in progressive uraemia (toxic waste accumulation), metabolic acidosis, hyperkalaemia and pulmonary oedema if fluid balance is not carefully monitored. These complications prolong hospitalization and are associated with increased mortality.

Published studies of AKI suggest a large percentage of episodes are preventable, or potentially reversible through simple interventions such as prompt fluid replacement, discontinuing and/or avoiding potentially nephrotoxic agents, relief of urinary tract obstruction and earlier recognition of conditions causing rapid progression of AKI.

Although AKI may be reversible some long-term follow up studies indicate that AKI is also associated with an increased risk of developing chronic kidney disease (CKD). Furthermore, episodes of AKI may exacerbate pre-existing CKD.

This GAIN guideline provides information relevant to the assessment and management of AKI in adults. For additional information on AKI in children and young person's up to 16 years of age refer to NICE clinical guideline 169.

Methodology

Who is the guideline intended for?

The guideline is relevant to all healthcare professionals who come into contact with patients with acute kidney injury.

It is also expected that the guideline will be of value to those involved in clinical governance in both primary and secondary care to help ensure that arrangements are in place to deliver appropriate care to this group of patients.

The remit of the guideline is to develop a guideline for professionals caring for patients who develop acute kidney injury.

The Terms of Reference for the Guideline

The Terms of Reference were developed by the Guideline Development Group (GDG).

These would be:

- To write pragmatic clinical guidelines to help practitioners prevent, recognise, investigate and manage acute kidney injury
- To design a one page clinical algorithm to help practitioners recall key management steps in the initial management of persons at risk of AKI

Involvement of Stakeholders

Key to the development of GAIN guidelines is the involvement of relevant professional and patient/carer organisations. A list of the GDG for the Northern Ireland Guidelines for Acute Kidney Injury can be found in Appendix 2.

Needs Assessment

As part of the guideline development process, a meeting was held with key stakeholders to identify pragmatic questions related to the prevention, recognition, investigation and management of Acute Kidney Injury. Revision of the 2010 GAIN guidance on Acute Kidney Injury (AKI) was necessary because of several changes in practice within the last three years. These included the widespread adoption of National Early Warning Scores (NEWS) charts, the introduction of clinical chemistry electronic alerts for AKI and new evidence indicating the potential risk of developing AKI from prescribing hydroxyethyl starch based colloid solutions.

The literature review for this guideline retrieved articles and guidelines published within the previous ten years. (2004-13). This guideline development was restricted to adults (deemed to be those over the age of 16 years of age). Relevant publications were obtained by searching Medline, Pubmed and Cochrane databases and were further supplemented by trawling of specified websites including those for NICE, NCEPOD and Renal Association. Departmental Strategic documents 'standards documents' were also reviewed to inform the development of these guidelines. This evidence table is available on the GAIN website – www.gain-ni.org

Who Developed the Guideline?

Overview

Based on methods outlined in the 'Advice for Guideline Development in Northern Ireland' document a team of health professionals, lay representatives, technical experts and GAIN known as the GDG (see Appendix 2) undertook the development of this clinical guideline. The basic steps in the process of developing a guideline were also taken from Appendix 5 of the 'Advice for Guideline Development in Northern Ireland' document.

The Guideline Development Group (GDG)

The Acute Kidney Injury GDG was recruited by requests for nominations being sent to the main stakeholder organisations and patient organisations/charities.

At the start of the guideline development process all GDG members' interests were recorded on a standard declaration form that covered consultancies, fee-paid work, share-holdings, fellowships and support from the healthcare industry. At all subsequent GDG meetings, members declared new, arising conflicts of interest which were recorded. The development of the guideline was supported by GAIN staff.

Guideline Development Group Meetings

Three meetings were held between October 2013 and February 2014. During each meeting clinical questions and clinical evidence were reviewed and assessed and recommendations formulated. At each meeting patient/carer and service-user concerns were routinely discussed as part of a standing agenda item. In addition the guideline documents were reviewed many times during email discussions between GDG members.

The Co-Chairs divided the GDG workload by allocating specific topics, relevant to their area of clinical practice to small sub-groups of the GDG in order to simplify and speed up the guideline development process. These groups considered the evidence, as reviewed by the systematic reviewer, and synthesised it into draft recommendations prior to presenting it to the GDG as a whole.

Patient/carer Representatives

A user representative participated on the GDG providing valuable perspective on patient experience of Acute Kidney Injury.

Expert Advisers

During the development phase of the guideline the GDG identified areas where there was a requirement for expert input on particular specialist topic areas. The topics were addressed by either the production of a position paper or a formal presentation by a recognised expert who had been identified via the relevant registered stakeholder organisation. All relevant position papers are presented as part of the evidence review.

Updating the Guideline

In keeping with GAIN requirements these guidelines will be reviewed in 2017 or sooner in light of any emerging evidence.

Funding

The GDG was commissioned by GAIN to develop this guideline.

Recognition of Acute Kidney Injury

Acute Kidney Injury (AKI) has traditionally been defined as the abrupt loss of kidney function resulting in the retention of urea and other nitrogenous waste products and in the dysregulation of extracellular volume and electrolytes. This can occur in the setting of previously normal renal function or in patients with pre-existing renal disease (acute on chronic kidney disease).

More recently it has been recognised that even very small increases in serum creatinine are associated with adverse patient outcomes.

How to diagnose AKI in Hospitalised Patients

AKI can be recognised by accurate interpretation of changes in urine output coupled with regular measurement of kidney function (U&E) and recognition of significant changes in serum creatinine.

AKI is an abrupt reduction in kidney function and is associated with:

- a rise in serum creatinine of 26 $\mu\text{mol/L}$ or greater within 48 hours
- or a 50% or greater increase in serum creatinine (1.5 fold from baseline) within the past 7 days
- or a fall in urine output to less than 0.5 mL/kg/hour for more than 6 hours)

Accurate measurement of urine output may not be routine outside of the intensive care unit (ICU). Urine output may be influenced by the use of diuretics or the presence of urinary tract obstruction. Nevertheless, a reduction in urine output is a sensitive indicator of renal dysfunction and often precedes a rise in serum creatinine in critically ill patients.

The regional introduction by clinical chemistry laboratories of electronic alerts (e-Alerts) to flag changes in creatinine indicative of AKI will also help identify patients with AKI at an earlier stage.

Persons at higher risk of Acute Kidney Injury

Individuals are more likely to develop AKI if they have any of the following risk factors.

- Pre-existing chronic kidney disease (CKD) - (eGFR < 60 mL/min/1.73m²)
- Older age (> 65 years old)
- Sepsis
- Deteriorating early warning scores
- Co-existing illness including: cardiac failure, liver disease and diabetes mellitus
- Use of NSAIDs including COX-II selective inhibitor drugs
- Use of ACE inhibitors or angiotensin receptor blocker (ARB) medication particularly in the setting of hypovolaemia
- Hypotension (systolic blood pressure < 110 mmHg)
- Symptoms and signs of hypovolaemia (vomiting, diarrhoea, tachycardia, hypotension)
- Urinary tract symptoms (especially reduced urine output or anuria associated with urinary tract obstruction)
- Those receiving intravenous contrast for diagnostic and therapeutic procedures
- Patients in the peri-operative period

Prevention of Acute Kidney Injury

Patients in the community with reduced kidney function (eGFR < 60 mL/min/1.73m²) and patients treated with an ACE inhibitor or ARB are at increased risk of AKI if they develop an illness associated with hypovolaemia and hypotension. Consideration should be given to the temporary cessation of medication which may in this setting induce, exacerbate or complicate AKI (e.g. diuretics, ACE inhibitors, ARBs, NSAIDs or metformin).

In **hospitalized patients** a strategy for prevention of AKI should include the following management principles.

- All patients, both on admission and during their hospital stay, should be assessed regularly for their risk for developing AKI.
- Patients admitted as an emergency should have their baseline renal function established on admission. Patients with an eGFR < 60 mL/min/1.73m² have a higher risk for AKI and need closer monitoring of their renal function (serum creatinine and record of daily urine output).
- Fluid volume status should be carefully assessed paying attention to symptoms and signs of both fluid depletion and fluid overload. Where patients are at risk of dehydration (poor oral intake, fasting for procedures) then prescription of maintenance IV fluids should be considered.
- Fluid balance, urine output and U&E should be carefully monitored especially during periods of antibiotic therapy, episodes of clinical deterioration and in the peri-operative period.
- Hypotension (systolic blood pressure < 110 mmHg; mean arterial pressure < 65 mmHg) needs urgent assessment and treatment with the use of appropriate IV fluid challenges and vasopressor agents where indicated.
- Care should be taken prescribing NSAIDs in patients at risk of developing AKI. Regular monitoring of renal function (serum creatinine and record of daily urine output) should occur in patients taking these medications during their inpatient stay.
- Temporary cessation of ACE inhibitors (and ARBs) is appropriate in patients with dehydration, hypotension (systolic blood pressure < 110 mmHg) and/or deteriorating renal function. Alteration of timing of drug prescription to 6 pm will allow adequate time during the working day to assess clinical state and renal function in case there is a need to temporarily hold these medications.
- Where clinically indicated aminoglycosides can continue to be used in AKI with dosing adjusted to GFR level. However, renal function and drug levels of potentially nephrotoxic antibiotics (e.g. gentamicin) must be carefully monitored during their administration.

Diagnosis

For patients with hospital-acquired AKI the cause is frequently associated with several risk factors. Assessment of the patient with AKI therefore starts with a careful history and examination, including a thorough review of the patient's notes and drug treatment records where available.

Any evidence of chronic kidney disease (CKD) should be sought from either previous hospital attendances/admissions or the patient's GP.

A focused history can identify pre-existing risk factors and potential causes for AKI including reduced fluid intake and/or increased fluid losses, urinary tract symptoms and recent drug ingestion.

AKI secondary to systemic disease may be associated with other clinical features such as fever, rash, joint pains and non specific malaise.

Clinical examination includes assessment of volume status, which is guided by clinical signs including core temperature, peripheral perfusion, heart rate, blood pressure and jugular venous pressure. Postural changes in pulse and blood pressure should be recorded and are more sensitive indicators of hypovolaemia than supine observations. The abdomen must be examined carefully for the presence of a palpable bladder.

Urine output should be measured with consideration of urinary catheterisation in patients with no demonstrated urine output within 4 hrs, hypotension, sepsis and/or pulmonary oedema.

Reagent strip urinalysis should be performed on all emergency admissions. Positive protein and blood indicators of 2+ to 4+ on reagent strip testing of the urine suggests intrinsic glomerular disease and warrants prompt action. A high specific gravity (>1.020) may suggest volume depletion responsive to a fluid challenge.

The laboratory evaluation should include a U&E (with particular reference to concentrations of urea, creatinine, sodium, potassium and bicarbonate), full blood count, CRP, liver function tests, glucose and bone profile. Where there is clinical suspicion of muscle injury or pancreatitis, creatinine kinase and amylase levels should be checked.

Measure of urine sodium concentration may be helpful in identifying volume sensitive pre-renal AKI (urine sodium concentration < 20 mmol/L).

Where there is evidence of a significant metabolic acidosis (serum bicarbonate < 20 mmol/L) an arterial blood gas should be undertaken to check the pH and pCO₂.

Where there is evidence of hypotension (systolic BP < 110 mmHg) a serum lactate should be measured to determine if there is evidence of tissue hypoperfusion and shock.

Ultrasound of kidneys is ideally undertaken within 6 hrs when upper tract renal obstruction is suspected (i.e. oliguric/anuric but normotensive) and within 24 hrs where there is failure of improvement in renal function following treatment of the underlying condition.

A less common cause for AKI may be considered if

- clinical features such as fever, rash, joint pains or pulmonary infiltrates are present
- there is no obvious cause for AKI (e.g. absence of hypotension, dehydration, sepsis, nephrotoxins, obstruction)
- abnormal urinalysis (proteinuria +/- haematuria) is found
- thrombocytopenia and haemolytic anaemia are present (suggestive of haemolytic uraemic syndrome)

Such features should prompt investigation for rarer causes for AKI such as systemic vasculitis (ANCA, C3, C4), anti-GBM nephritis (anti-GBM antibody), lupus nephritis (auto-antibody screen) and myeloma (serum free light chains, serum immunoglobulins, plasma protein electrophoresis).

NB: It should be stressed that “volume responsive” AKI and acute tubular necrosis are much more common forms of AKI in community (90%) and hospital settings (>70%) than AKI due to intrinsic glomerular disease (3%).

Treatment Principles

There is no specific pharmacological treatment for AKI. There is no evidence to support the use of loop diuretics or dopamine as treatments for AKI.

Loop diuretics may have a limited role in managing severe fluid overload in selected patients who are not hypotensive or anuric.

The focus of therapy for AKI should be:

- To correct those conditions causing or contributing to the acute kidney injury
- Where indicated to support kidney function by means of renal replacement therapy (dialysis) until recovery of independent kidney function has occurred

It is critical that the volume status of patients with AKI is accurately assessed. This is usually accomplished by clinical assessment and dynamic response to appropriate fluid replacement. Where there is clinical uncertainty of fluid and volume status then patients should be considered for invasive haemodynamic monitoring in a high dependency unit (HDU) setting.

Special consideration should be given to

- Urgent relief of urinary tract obstruction
- Prompt fluid resuscitation to restore an effective circulating blood volume
- Prompt restoration of an effective blood pressure
- Avoidance of fluid overload
- Stopping potentially nephrotoxic drugs where possible
- Adjustment of drug dosing appropriate for GFR where indicated

Restoration of kidney perfusion

Correct dehydration

- In ill patients, fluid replacement is best achieved through the rapid infusion of repeated small volumes (e.g. 250 - 500 mL of intravenous crystalloid) to achieve clinical evidence of adequate perfusion (systolic blood pressure of > 110 mmHg, pulse rate < 100/min, increased urine volume/hour, capillary refill time < 3 seconds). Hartmann's solution is preferable to normal saline provided serum K⁺ < 5.5 mmol/L.
- Where there are clinical uncertainties consider more invasive haemodynamic monitoring in an HDU setting.
- Once volume state has been corrected prescribe maintenance fluids at an hourly rate according to total losses + 30 mL/hour.
- Avoid potassium containing fluids (Hartmann's) where the serum potassium is above 5.5 mmol/L.
- Care should be taken to avoid fluid overload. Once the patient is haemodynamically stable, if the urine output remains poor (< 50 mL/hr) further fluid boluses should not be administered in an attempt to improve urine output. This practice often results in the patient developing fluid overload with a worse outcome in AKI.

STOP drugs that interfere with kidney function

- In hypotensive and/or hypovolaemic patients stop ACE inhibitors, angiotensin receptor blockers, diuretics, other anti-hypertensive agents and NSAIDs.
- These drugs will exacerbate the hypoperfusion of kidneys and should be withheld until the patient is stabilized and the AKI has resolved.
- When aminoglycosides are indicated prescribe at reduced dose according to GFR.

Restore an effective blood pressure

- Sepsis is a common cause of AKI. Prompt treatment of sepsis is an important principle in reducing risk of acute kidney injury
- Where the patient is hypotensive (systolic blood pressure < 110 mmHg; mean arterial pressure < 65 mmHg) repeat 500 mL boluses of intravenous crystalloid (250

mL if history of cardiac failure) up to total dose of 1.5 – 2.0 L within a 2 hour time frame. Assess for fluid overload after each fluid bolus.

- Where the systolic blood pressure remains labile (systolic blood pressure < 110 mmHg, mean arterial pressure < 65 mmHg) despite 1.5- 2.0L of intravenous fluid then consideration should be given to HDU/ICU referral for invasive haemodynamic monitoring and administration of vasopressor therapy (e.g. noradrenaline) in an HDU - ICU setting.

Medication Review

Medications commonly contribute to the development of AKI. In addition, with a sudden reduction in kidney function many drugs require dose modification to avoid hazardous side effects e.g. oral hypoglycaemic drugs have a much longer duration of action in kidney failure.

Patients with AKI are by definition in a clinically unstable condition and all medication including “usual” prescriptions should be reviewed as soon as AKI is identified.

The following list is not exhaustive but will provide a useful start.

Drugs interfering with renal perfusion

- ACE inhibitors and angiotensin receptor blockers
- NSAIDs
- All antihypertensives
- Diuretics (loop and thiazide)
- Nitrates
- Nicorandil

Common drugs requiring dose reduction or cessation

- Low molecular weight heparins
- Opiates
- Penicillin based antibiotics
- Metformin (increased risk of lactic acidosis)
- Sulphonylurea-based hypoglycaemic agents
- Aciclovir

Drugs requiring close monitoring

- Warfarin
- Aminoglycosides

Drugs aggravating hyperkalaemia

- Digoxin
- Beta blockers
- Trimethoprim
- Potassium sparing diuretics e.g. spironolactone, amiloride

Prevention of Radiocontrast Nephropathy

Acute kidney injury secondary to radiological contrast media classically occurs within 72 hours of receiving such agents. There is no specific treatment.

Prevention involves identifying those patients at risk and avoiding dehydration before, during and after the radiological procedure.

Who is at risk?

- Estimated GFR ≤ 30 mL/min/1.73m²
- Estimated GFR between 31 and 60 mL/min/1.73m² and “AT RISK” patients (see page 9)

Management: Five point plan

- Recommended for all patients with an eGFR of ≤ 30 mL/min/1.73m²
 - Consider for all patients with an eGFR of ≤ 60 mL/min/1.73m² and risk factors listed in page 9
1. Adequate hydration is crucial for the patient's management prior to the procedure. If at risk of developing contrast nephropathy the patient should receive either intravenous 0.9% sodium chloride or 1.4% sodium bicarbonate at a rate of 3mL/kg/hour for 1 hour pre-procedure and 1 mL/kg/hour for 6 hours post-procedure.
 2. Potentially nephrotoxic medications should be avoided.
 - Omit ACE inhibitors or ARBs for on the day of procedure pre and do not restart post procedure until U&E stable (back to baseline at 48 – 72hrs).
 - Omit NSAIDs on the day of procedure and do not restart post procedure until U&E stable (back to baseline at 48 and 72 hrs).
 - Metformin should be stopped on or prior to the day of study and not restarted until renal function has been demonstrated to be stable because of the risk of lactic acidosis.
 3. Low osmolar agents are associated with a decreased risk of nephrotoxicity as compared to the high osmolar agents, particularly in those at risk from contrast media-associated AKI. Patients with CKD should receive the iso-osmolar non-ionic contrast agent iodixanol which has been shown to reduce the risk of contrast induced nephropathy in this patient group.
 4. The dose of contrast media should be minimised and further exposure to contrast media should be delayed until full recovery of renal function unless absolutely necessary.
 5. Renal function should be checked up to 48-72 hours following the procedure if in a high risk group to ensure stable renal function.

There is no compelling evidence for the routine use of N-acetylcysteine.

Indications for AKI Referral to a Nephrology Team

Early contact with a nephrology team is advisable for those patients who are likely to need renal replacement therapy (dialysis) or for whom intrinsic renal disease is considered to be a cause of their AKI. For patients in whom AKI is as part of multi organ failure it will be more appropriate to refer to the local Critical Care Team.

In particular referral to a nephrology team is appropriate if a patient with AKI has:

- Hyperkalaemia (> 6.5 mmol/L) refractory to medical management
- Pulmonary oedema refractory to medical management
- Severe metabolic acidaemia $\text{pH} \leq 7.2$ due to kidney failure
- Progressive renal failure (creatinine ≥ 300 $\mu\text{mol/L}$ and/or rise in serum creatinine of >100 $\mu\text{mol/L/day}$)
- Uraemic complications (pericarditis or uraemic encephalopathy)
- Renal transplant
- Chronic kidney disease (stage 4 or 5)
- Patients suspected of intrinsic renal disease (vasculitis, primary glomerulonephritis, interstitial nephritis)

Nephrology - Critical Care Interface

Many patients who develop AKI do so in the context of multiple organ dysfunction. This is often manifest by hypotension, lactic acidosis and severe respiratory compromise.

Such patients require invasive monitoring and multi-organ support which may include mechanical ventilation, vasopressor support to facilitate their optimal clinical care. There are several modes of renal replacement therapy (dialysis) that are appropriate for critically ill patients with AKI in the setting of multiple organ failure. The critical care team will decide on the choice of dialysis modality in ICU and HDU environments with input as required from the nephrology service. Liaison between nephrologist and critical care staff is essential to help plan for AKI management during and after ICU admission.

Early involvement with the ICU team in assessment of ill and clinically deteriorating patients with AKI is to be encouraged.

Nephrology – Conservative Care Interface

It should be recognised that AKI may be just part of a terminal illness in a hospitalized patient. The terminal nature may reflect the severity of the clinical event or the progression of advanced untreatable co-morbidity.

In such patients provision of renal replacement therapy (dialysis) is futile and may prolong suffering of a dying patient and lead to false hopes in relatives for the expected survival of a family member.

Such patients should be identified by their senior medical staff early in the course of their deterioration to decide on ceilings of care for both resuscitation (invasive monitoring, vasopressor therapy) and appropriateness of a referral to a nephrology team.

Nephrology – Primary Care Interface

As highlighted throughout this GAIN guidance, GPs play a key role in identifying and managing patients with AKI, particularly those who develop AKI in the community (community-AKI). Some patients are at increased risk of AKI when they develop an acute illness associated with sepsis, volume depletion or hypotension.

The risk of community-AKI is also higher in patients predisposed by chronic conditions such as CKD, heart failure, diabetes or urinary tract obstruction. Older persons are at increased risk of AKI as they have lower baseline kidney function (natural age related nephron loss) and may in certain circumstances have limited access to adequate oral fluids.

GPs prescribing decisions can have a significant impact on risk of community-AKI. For instance, the co-prescription of NSAIDs with ACEI or ARBs is a potentially nephrotoxic combination. These drugs impair the normal physiological response to hypovolaemia and hypotension (glomerular auto-regulation maintaining GFR) and can contribute to the development of AKI. Temporarily withholding these medications in acute illness, e.g. vomiting and diarrhoea, can help to reduce the risk of AKI in community settings.

It is not necessary to refer all persons with AKI to hospital if they are responding to timely community management of their underlying medical condition. Outpatient referral to a nephrologist should be considered for individuals who have appear to have recovered from AKI but have a persisting eGFR < 30 mL/min/1.73m², or significant proteinuria (>1.0g/24hr).

Patients discharged from hospital still recovering from AKI require follow-up biochemical tests (serum creatinine; eGFR) to document recovery of baseline renal function or help with information needed for an outpatient nephrology referral (as above). Medications that potentially contributed to AKI may have been withdrawn during hospital admission. Care should be exercised on re-introducing drugs, such as antihypertensive medication, that may impact on renal function recovery. A patient with a history of AKI is at higher risk for further episodes of AKI or later development of CKD. Patient education on hospital discharge and at community follow up should reinforce the importance of temporarily withholding medications predisposing to community-AKI when indicated.

AKI RISK ASSESSMENT

Background Factors

- CKD (eGFR <60mL/min)
- Age >65 yr
- Co-morbidity (IHD, CCF, DM)

Acute Context

- Sepsis
- Peri-operative period

Illness Severity

- Hypovolaemia
- Systolic BP <110mmHg
- Deteriorating NEWS

Medication Use

- NSAID, COX II, ACEi, ARB
- Aminoglycoside
- Iodinated Radiocontrast

≥2 Risk factors
'AKI AT RISK'

Daily U&E
Accurate Urine Output record
Close attention to fluid balance

ESTABLISHED AKI

DEFN: sCreatinine rise >26umol/L in 48hrs, >50% from baseline value within 7 days, UO < 30mL/hr for 6hrs

INVESTIGATE URGENTLY

LABS – U&E, FBP, CRP, LFT, Glucose, Bone profile, Coagulation Screen
Urine dipstix analysis, *Urine Na*

RENAL ULTRASOUND

- Within 6hrs if upper tract obstruction considered
- Within 24hrs if AKI not responding to treatment

SELECTED CASES -

- Nephritis - ANCA, Anti-GBM
- HCO₃ <19mmol/L - ABG
- Rhabdomyolysis – CK
- SBP <110mmHg - Lactate

RESTORE KIDNEY PERFUSION

OPTIMISE VOLUME

- Bolus 250-500mL crystalloid targeting SBP >110mmHg/clinical evidence of euvoalaemia
- 2L max IV fluids within 2hr
- **After each bolus** check for signs of fluid overload
- Seek senior help before repeating 2L fluid challenge

OPTIMISE BLOOD PRESSURE

If despite adequate volume challenge hypotension persists (SBP<110 +/-or MAP <65mmHg) obtain a urgent senior review

Consideration should be given to HDU/ICU referral

PRESCRIBE SAFELY

STOP NSAIDs, COX II, ACEi, ARBs
Metformin
AVOID WHEN SBP <120mmHg
Antihypertensives, Diuretics

CORRECT dosing to GFR level (e.g. aminoglycosides, metformin and sulphonylureas, LMWH and many antibiotics)

IV FLUID PRESCRIBING -

MAINTENANCE FLUIDS

RATE = Urine Output + 30mL/hr

BOLUS FLUIDS

AVOID Hartmann's if K >5.5mmol/L

INDICATIONS FOR REFERRAL TO NEPHROLOGY

Suspected intrinsic renal disease

- Blood and Protein on urinalysis with suspicion of glomerulonephritis
- Unclear aetiology of AKI (no pre-renal or obstructive cause identified)

Potential need for renal replacement therapy (dialysis)

- Refractory hyperkalaemia (>6.5mmol/L) or pulmonary oedema
- Severe metabolic acidosis (HCO₃ < 15mmol/L)
- Progressive AKI (creatinine >300umol/L or rise >100umol/L in 24hr)

AKI occurring in

- Renal transplant patients
- Patients with baseline GFR <30mL/min

Glossary of Terms

ABG	arterial blood gas
ACEi	angiotensin converting enzyme inhibitor
AKI	acute kidney injury
ANCA	anti-neutrophil cytoplasmic antibody
Anti-GBM	anti-glomerular basement membrane
ARB	angiotensin receptor blocker
ATN	acute tubular necrosis
CCF	congestive cardiac failure
CK	creatinine kinase
CKD	chronic kidney disease
COX-II	cyclooxygenase II
CRP	C-reactive protein
DM	diabetes mellitus
eGFR	estimated glomerular filtration rate
GFR	glomerular filtration rate
HDU	high dependency unit
ICU	intensive care unit
IHD	ischaemic heart disease
IV	intravenous
LFTs	liver function tests
LMWH	low molecular weight heparin
MAP	mean arterial pressure
NEWS	National Early Warning Score
NICE	National Institute for Health and Care Excellence
NINF	Northern Ireland Nephrology Forum
NSAID	non-steroidal anti-inflammatory drug
SBP	systolic blood pressure
U&E	urea and electrolytes

Membership of GAIN AKI Guideline Development Group

The Northern Ireland Guideline for Acute Kidney Injury Guidelines were revised in 2014 by:

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Additional Sources of Guidance

- NICE Acute Kidney Injury clinical guidance (CG169) (2013)
<http://guidance.nice.org.uk/CG169>
- National Confidential Enquiry into Patient Outcome and Death (NCEPOD)
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<http://www.ncepod.org.uk/2009aki.htm>
- National Confidential Enquiry into Patient Outcome and Death (NCEPOD)
“Elective and Emergency Surgery in the Elderly: An Age Old Problem (2010)
<http://www.ncepod.org.uk/reports2.htm>
- Renal Association Clinical Practice Guidelines: Acute Kidney Injury (2011)
<http://www.renal.org/Clinical/GuidelinesSection/AcuteKidneyInjury.aspx>
- British Consensus Guidelines on Intravenous Fluid Therapy for Adult Surgical Patients (2011) http://www.bapen.org.uk/pdfs/bapen_pubs/giftasup.pdf
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<http://guidance.nice.org.uk/CG/Wave25/5>
- Perioperative acute kidney injury: risk factors, recognition, management, and outcomes. Clinical Review. Borthwick E, Ferguson A. BMJ 2010 Jul 5;341:c3365. doi: 10.1136/bmj.c3365.
- Northern Ireland Guidelines and Audit Implementation Network (GAIN) guidelines
www.gain-ni.org/Guidelines/index.asp
- GAIN Northern Ireland Guidelines for Management of Chronic Kidney Disease (2010) <http://www.gain-ni.org/images/Uploads/Guidelines/Chronic%20Kidney%20Disease.pdf>
- GAIN Hyponatraemia in Adults on or after 16th birthday (2010)
http://www.gain-ni.org/images/Uploads/Guidelines/Hyponatraemia_guideline.pdf
- GAIN Guidelines for the Treatment of Hyperkalaemia in Adults (2009)
http://www.gain-ni.org/images/Uploads/Guidelines/hyperkalaemia_guidelines.pdf
- Surviving Sepsis Campaign
<http://www.survivingsepsis.org/bundles/Pages/default.aspx>