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ACUTE KIDNEY INJURY GUIDELINES FOR PRIMARY CARE

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COMMUNITY ACUTE KIDNEY (AKI) GUIDELINE

AKI Alert

Check previous U&Es to differentiate AKI from stable CKD

AKI is diagnosed on comparison of recent versus previous U&E or drop in urine output as defined below:

Stage 1: Increase in serum creatinine to 1.5-1.9 x baseline or
Increase in serum creatinine by ≥ 27 µmol/L in 48 hours
Stage 2: Increase in serum creatinine to2.0-2.9 x baseline
Stage 3: Increase in serum creatinine to 3.0 x baseline or
Increase in serum creatinine to≥354 µmol/L or
Initiation of renal replacement therapy or
In patients < 18yrs, decrease in eGFR to <35mL/min/1.73m^2

Stages 2&3 will be alerted to GPs by telephone from the labs

Is this AKI?

No → Is the patient acutely unwell?

No → Repeat Creatinine after 48 to 72 hours

Yes → Assume AKI until proven otherwise

Yes ← Can the patient be managed in the Community?

Essential steps to be initiated by GP/Community Team

Think FLUiDS

Fluid balance: Check for signs of dehydration and treat and encourage oral fluids *(Could we start subcuts via DN/IRS team?)*

Low Blood Pressure: Check BP and if low (SBP<110) withhold anti hypertensive, diuretics. If H/O angina/arrhythmia, reduce βBlocker

Urine: Dip test and microscopy

Bladder palpable: Catheterise

Drugs and Toxins: Stop NSAID (Ibuprofen COX 2 inhibitors, Trimethoprim, ACEi, AGBs. Any new drugs with temporal relationship to onset of AKI. Avoid any nephrotoxic medications)

Sepsis: Look for signs of sepsis and treat accordingly

Early review with repeat U&Es: If getting worse discuss

If patient is well

- **Continue with essential steps listed above**
- **Repeat U&Es in 24 to 48 hours**
- **Renal screen (**ANCA, AntiGBM Ab, Autonatibodies, serum immunoglobulin and paraprotein, urine Bence Jones protein, urine albumin creatinine ratio

REFER IF:

- **Serum K >5.7 mmol/L** – refer to AED unless advance directive states otherwise
- **If acute vasculitis or acute nephritis is suspected**
- **Worsening U&Es or persistent oliguria despite treatment**(unless advance directive states otherwise)
- **If stage 3 AKI**
- **If Stage 2 AKI and acutely unwell**
- **Refer to Urology if suspicion of urinary tract obstruction**
- **Refer to Obs/Gynae where woman is pregnant**
- **Urinalysis of ≥2+ blood AND protein**
- **Systemic symptoms (**e.g. arthralgia, rash, epistaxis, haemoptysis)
- **Prior CKD stage 4 or 5 and added AKI**
- **Renal transplant with any AKI**

OOH Alerts will be referred by the Labs to the CCC who will co-ordinate review of the pt

Where pts require repeat bloods and monitoring over the weekend/BH, a referral should be sent to CCC who will co-ordinate care with the Community Matron on call

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POST AKI MANAGEMENT IN PRIMARY CARE

1. Has Renal Function recovered?	**Yes:** No further action on this point. Move to point 2
	No: 1. Repeat creatinine after 2-4 weeks to assess for further recovery 2. New onset CKD: check proteinuria and repeat U&Es in 3 months, manage as per CKD guidelines 3. Significant decline in renal function: consider nephrology referral *(≥25% decline in eGFR or change in CKD stage)*

2. Review medications: have medications been stopped that now need re starting?	**Yes:** 1. BP tablets are often stopped but need re starting post discharge 2. ACEi/ARB can be restarted once the renal function has stabilised – U&Es should be checked one week after reintroduction 3. If Aspirin (75mg) and statins were stopped, these should be re started unless specific reason not to. 4. If a drug has been specifically implicated in causing AKI update practice records to prevent re-prescribing.
	No: no further action on this point, move to point 3

3. Give advice to prevent further AKI episodes	**Consider:** 1. AKI sick day rules (see appendix for a patient leaflet) 2. Avoiding prescription of long term NSAIDs where possible, particularly in high risk patients and those with CKD 3. Avoiding prescribing triple combination of spironolactone, NSAID and ACEi/ARB 4. Monitor renal function after introducing certain medications – ACEi/ARB/spironolactone, and in patients with CKD trimethoprim and loop diuretics.

WHAT IS AKI?

Acute Kidney Injury (AKI) was previously known as acute renal failure and simply means a sudden reduction in renal function. It is **not** a traumatic injury to the kidney as the name may imply, nor is it a diagnosis in its own right, rather a syndrome with many different underlying causes

WHY IS AKI IMPORTANT?

AKI is extremely common in hospitalised patients, occurring in 10-20% of emergency hospital admissions and is associated with extremely poor outcomes. However, AKI is not just a secondary care problem – primary care has a crucial role to play, particularly in prevention and post-AKI care (see below).

Poor outcomes associated with AKI:

- Extremely high mortality rates (more than **20% of patients with AKI will die** during hospital admission, rising to >35% in those with AKI stage 3).
- Increased length of hospital stay and higher healthcare resource utilisation
- Failure of renal recovery – episodes of AKI cause and increase progression of Chronic Kidney Disease (CKD)
- Increased risk of poor long term outcomes: life expectancy, cardiovascular risk, quality of life

In part, these poor outcomes reflect the fact that AKI acts as a 'force multiplier' and increases severity of any co-existing acute illness. **As such, AKI is a marker of the 'sick patient' who requires prompt recognition and management.**

It is also important that there are significant opportunities to improve AKI care. This is recognised in a major, national programme from NHS England called 'Think Kidneys' (https://www.thinkkidneys.nhs.uk/) This programme aims to reduce avoidable harm associated with AKI across all healthcare settings.

So why is AKI important for Primary Care?

- Up to two-thirds of patients who sustain AKI have already developed this by the time they are admitted to hospital, so preventative strategies have to include pre-hospital care.
- Increased recognition of AKI based on outpatient bloods: from January 2016 results from electronic detection systems situated in biochemistry labs will be sent to primary care, aiming to make changes in serum creatinine concentration easier to spot. This means that guidance for the outpatient management of AKI should be in place. Further information available at: https://www.thinkkidneys.nhs.uk/aki/resources/
- Finally, **improvements are required at discharge from hospital** so that patients who have recovered from AKI have clear plans for follow up and for reintroduction of long term medications that may have been stopped during admission.

HOW TO RECOGNISE AKI

The presence of AKI is determined using internationally recognised criteria that are based on individualised changes in serum creatinine concentration with respect to that person's usual (or baseline) value, and reduction in urine volume. **In practice, the urine output criteria can only be applied to hospitalised patients who are catheterised.**

AKI is defined by any of the following:

- Increase in serum creatinine by ≥27µmol/L within 48 hours; or
- Increase in serum creatinine by ≥1.5 times baseline, which is known **or presumed to have occurred*** within the prior seven days; **or**
- **Urine volume <0.5 mL/kg/h for six hours**

*** This is crucial, because it is common to see patients with an increase in serum creatinine and longer gap between current value and baseline. In this situation, there are two things to consider:**

1. **Is the patient acutely unwell?** If so, AKI is more likely.
2. **Repeating the creatinine within 48-72hrs.** A repeat creatinine will help to determine whether the changes are dynamic or are stable (i.e. more consistent with CKD).

The severity of AKI is described by categorising into three stages (Stage 1 being the least and stage 3 being the most severe) as follows:

AKI Stage	Serum creatinine criteria	Urine output criteria
1	**Increase in serum creatinine to 1.5-1.9 baseline** or Increase in serum creatinine by ≥27µmol/L within 48 hours	<0.5 ml/Kg per hour for 6-12 hours
2	**Increase in serum creatinine to 2.0-2.9 times baseline**	<0.5 mL/Kg per hour for≥12 hours
3	**Increase in serum creatinine to 3.0 times baseline;** **or** **Increase in serum creatinine to ≥354µmol/L:** or Initiation of renal replacement therapy; or In patients<18 years, decrease in eGFR to <35mL/min/1,73m^2	<0.3 mL/Kg per hour for ≥24 hours; or anuria for ≥ 12 hours

WHAT CAUSES AKI?

There are many causes of AKI. However, most cases occur in conjunction with co-existing acute illness and are a result of sepsis, hypovolaemia, hypotension or medication effects; these causes, often in combination, account for up to **80% of cases**. This scenario is commonly seen in patients with long term conditions or the frail/elderly.

5-10% of AKI is due to post renal obstruction, e.g. bladder outflow obstruction.

Intrinsic renal diseases are less common, but are important not to miss because it is crucial that specialised management of these cases is accessed early. This category includes a variety of less common conditions such as: drug induced tubulo-interstitial nephritis, vasculitis/rapidly progressive glomerulonephritis, myeloma. There are some 'red flag' signs that help to identify this group of AKI patients so they can be referred early:

- AKI with blood and protein ($\geq$2+) on urinalysis
- AKI with systemic symptoms of inflammatory process: vasculitic rash, arthralgia, epistaxis or haemoptysis
- AKI in relation to the introduction of a new drug (PPI, NSAID, antibiotic, diuretic, allopurinol) without any other explanations for AKI
- AKI and high calcium

WHO IS AT RISK OF DEVELOPING AKI?

The following are risk factors for the development of AKI:

Patient specific	Situation Specific
• Increasing age • CKD • Diabetes Mellitus • Heart Failure • Liver Disease	• Hypovolaemia, dehydration, reduced oral intake • Hypotension • Sepsis • Post-operative

In addition, medications also impact on this. Some, such as NSAIDs, are nephrotoxic and will directly increase the risk of AKI. Diuretics may worsen hypovolaemia. ACE inhibitors (ACEi) and angiotensin receptor blockers (ARB) reduce the ability of the kidney to adapt to changes in perfusion pressure. One of the actions of ACEi and ARBs that account for their reno-protective effects in diabetic nephropathy and proteinuric CKD is the reduction in efferent glomerular arteriolar tone. However this action also reduces the ability to maintain glomerular filtration pressure in the face of dehydration/hypoperfusion.

Initiation of ACEi/ARB

Current guidance is that creatinine should be checked one week after initiation of ACEi/ARB and that an increase of up to 20% is acceptable, as long as this rise is stable. This rise reflects the changes in glomerular haemodynamics as above and is **not** a sign of nephrotoxicity. AKI would only be diagnosed if this rise was greater than 50% (the increment of >27µmol/l does not apply because the gap between blood tests should be >48hrs)

HOW TO REDUCE THE RISK OF AKI

This can be approached in different ways. Some interventions may be undertaken on a systematic, practice wide basis. Others may be more appropriate for individual patient management, supported by the correct tools and information.

1. **Sick day rules for high risk patient groups**
   This strategy is endorsed by the NICE AKI clinical guideline. Patients at increased risk of AKI are advised to temporarily suspend certain medications during periods of acute illness, particularly when at risk of dehydration. An example leaflet, which has been formulated with patient and professional input, is included as an appendix. Sick day rules for AKI usually refer to ACEi/ARB, which can be stopped temporarily without risk (including in heart failure). Diuretics may sometimes be included in this advice depending on the clinical scenario (greater care needed not to precipitate decompensation in patients with moderate to severe heart failure).
   There are different options to disseminate sick day rule advice to patients. It is unlikely that one model will suit all GP practices. The following are some examples of possible models, none of these are meant to be prescriptive:
   - Giving out leaflets during consultations on an ad hoc basis
   - IT search to identify patients with chronic conditions and prescribed an ACEi/ARB. This resulting list may allow systematic distribution of leaflets (this doesn't specify who at the practice disseminates the leaflets, or how this is done) or be used to trigger automatic reminders
   - Adding leaflets to repeat prescriptions for patients on ACEi/ARB

In time, resources that will help support these strategies are likely to be developed locally and nationally that will be shared

2. **When a patient at increased risk of AKI presents with an intercurrent illness, check renal function**
   There is no point in checking blood tests if the patient you are seeing requires immediate admission to hospital on clinical grounds. However, checking renal function in unwell patients with risk factors for AKI may allow earlier detection and intervention.

3. **Avoid prescription of long term NSAIDs where possible, particularly in high risk patients and those with CKD**

4. **Avoid prescribing triple combination of spironolactone, NSAID and ACEi/ARB**
   Evidence exists that this triple combination confers an increased risk of AKI in its own right. Equally, joint prescription of ACEi and ARBs is now generally not recommended (MHRA safety notice) and the combination of spironolactone and ACEi/ARB in CKD patients should also be carefully considered.

5. **Monitor renal function one week after the introduction of the following medications:**
   - ACEi/ARB
   - Spironolactone
   - Trimethoprim in patients with CKD
   - Loop diuretics in patients with CKD

HOW TO MANAGE A PATIENT WITH AKI DETECTED IN PRIMARY CARE

Not all patients with a rise in creatinine will need admission to hospital. The following is a guide but **clinical judgement must always prevail.**

1. **Is this definitely AKI?**
   As detailed above, not every patient with a rise in creatinine will have a recent baseline to compare. The result has to be taken in a clinical context, and a repeat creatinine (after 48-72hrs) if the situation allows may help distinguish dynamic changes in serum creatinine from a more stable CKD picture.

2. **Is the patient acutely unwell?**
   A blood test has to be interpreted within the overall clinical scenario, and this is especially important for AKI. Blood tests suggesting AKI in an unwell patient (especially if there are signs of sepsis) should increase clinical concern.

3. **How severe is the AKI?**
   Increasing severity of AKI correlates with higher risk of worse outcomes. AKI stage 3 should be managed in secondary care.

4. **Think about cause of AKI: does the patient have any red flag signs for urinary obstruction or intrinsic renal disease?**
   DIP THE URINE: this is an important diagnostic step.
   - AKI and negative urinalysis: usually pre-renal causes (also consider drug causes)
   - AKI with ≥2+ blood and protein: wider differential diagnosis that includes intrinsic renal disease

   If there are clinical pointers to urinary obstruction or intrinsic renal disease, these patients will need specialist referral (obstruction to urology, intrinsic renal disease to nephrology)

5. **Approach to outpatient management of AKI**
   - Avoid or correct dehydration
   - Medication review
     - Consider temporary suspension of ACEi/ARB +/- diuretics
     - Consider temporary suspension of metformin (to avoid risk of lactic acidosis)
     - Stop nephrotoxic medications such as NSAIDs
     - In the absence of an obvious cause of AKI, consider if any new drugs have been introduced that have a temporal relationship to the change in renal function: especially antibiotics and PPIs
   - Early review and repeat U/Es: seek advice from Urology SpR (available 8am-4pm daily via TRFT switchboard)or Surgical SpR after 4pm, for patients who are getting worse despite the above

6. **If in doubt, discuss with**
   - Contact Medical SpR on call (available 24 hours via TRFT switchboard)
   - If pregnant discuss with Obsetrician.

POST AKI CARE: WHAT TO DO WHEN A PATIENT HAS BEEN DISCHARGED AFTER AN EPISODE OF AKI

In the near future Hospital discharge summaries from the Rotherham NHS Foundation Trust will have a mandatory section that will detail information about AKI episodes that have occurred during a patient's hospital stay

In addition, the following is recommended to assess a patient following an episode of AKI:

1. **Assess degree of renal recovery**
    - Use creatinine at discharge and consider whether a repeat measure of renal function is required for those patients who have not returned to their normal baseline renal function.
    - If a patient has new onset CKD following an episode of AKI, assess and follow up as per CKD guidelines, which includes an assessment of proteinuria and a repeat creatinine at three months.
    - If you are concerned about a significant reduction in renal function following an episode of AKI, then contact nephrology for advice.

2. **Review medications**
    - Restart appropriate medications that may have been stopped during an AKI episode:
        - Blood pressure tablets are often stopped but need restarting when BP rises during recovery.
        - ACEi/ARB can be restarted (unless specific advice to the contrary) once the renal function has stabilised – U/Es should be checked one week after reintroduction.
        - Cardiovascular risk: if aspirin (75mg once daily) and statins were stopped, these should be restarted unless specific reason not to. Aspirin 75mg is not nephrotoxic.
        - If a drug has been specifically implicated in causing AKI (e.g. PPI leading to interstitial nephritis or NSAIDs), practice records should be updated to prevent the patient receiving these in future.

3. **Reduce risk of further AKI episodes in the future**
   As per points listed on page 7

4. **Code the occurrence of an AKI episode using the specific Read codes that currently exist (AKI 1, AKI 2, AKI 3)**

AKI SICK DAY RULES LEAFLET

MEDICINES AND DEHYDRATION

Patient Information

Need more Information?

Please contact your pharmacist, doctor, or nurse

This leaflet is about what actions you should take if you develop an illness that causes dehydration. These actions are called "medicine sick day rules"

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Who is this leaflet for?

You have been given this leaflet as you are taking the following long term medicine(s). Your pharmacist, doctor or nurse can circle your medicine(s) on this list or on the right hand side of your repeat prescription

- **ACE inhibitors: a medicine for high blood pressure and heart conditions.**
  Examples: names ending in "pril" such as lisinopril, perindopril, ramipril
- **ARBS: a medicine for high blood pressure and heart conditions**
  Examples: names ending in "sartan"such as losartan, candesartan, valsartan
- **NSAIDs: anti-inflammatory pain killers**
  Examples: ibuprofen, naproxen, diclofenac
- **Diuretics: sometimes called "water tablets" for excess fluid and high blood pressure**
  Examples: furosemide, bendroflumethiazide, indapamide, spironolactone
- **Metformin: a medicine for Diabetes**

Which illnesses cause dehydration?

Dehydration is the loss of fluid from your body. Vomiting, diarrhoea and fever (high temperature, sweats, shaking) can make you dehydrated. If you are sick once or have diarrhoea once, then you are unlikely to become dehydrated. Having two or more episodes of vomiting or diarrhoea can lead to dehydration: in these cases, you should follow the advice on this leaflet

What is the problem?

Taking certain medicines when you are dehydrated can result in you developing a more serious illness. These are:

- **ACE inhibitors, ARBs and NSAIDs:** if you are dehydrated, these medicines can stop your kidneys working properly
- **Diuretics:** these medicines can make dehydration more likely
- **Metformin:** dehydration can make it more likely that you will develop a serious side effect called lactic acidosis

What action should I take?

If you develop a dehydrating illness, you should temporarily stop taking the medicines listed on this leaflet. It is very important that you re-start your medicine(s) once you have recovered from the illness. This would normally be after 24-48 hours of eating and drinking normally. When you re-start your medicines, just take them as normal: do not take extra for the doses you have missed. If you have not recovered within five to seven days seek further advice from your health care professional.
If you take any medicines which require regular blood level monitoring (e.g. Lithium) you should contact the health care professional who is responsible for this medicine for further advice.

This leaflet can be opened and printed on the following link: *to be inserted when put on the intranet*

OTHER USEFUL RESOURCES:

NHS England AKI Programme (Think Kidneys)	www.thinkkidneys.nhs.uk
Open access e-learning package for primary care	http://www.uhl-library.nhs.uk/aki_gp/index.html
NICE AKI guidelines (CG169)	https://www.nice.org.uk/guidance/cg169