Acute Kidney Injury (AKI) Guidelines for Primary Care Barnsley CCG (January 2023 Update)

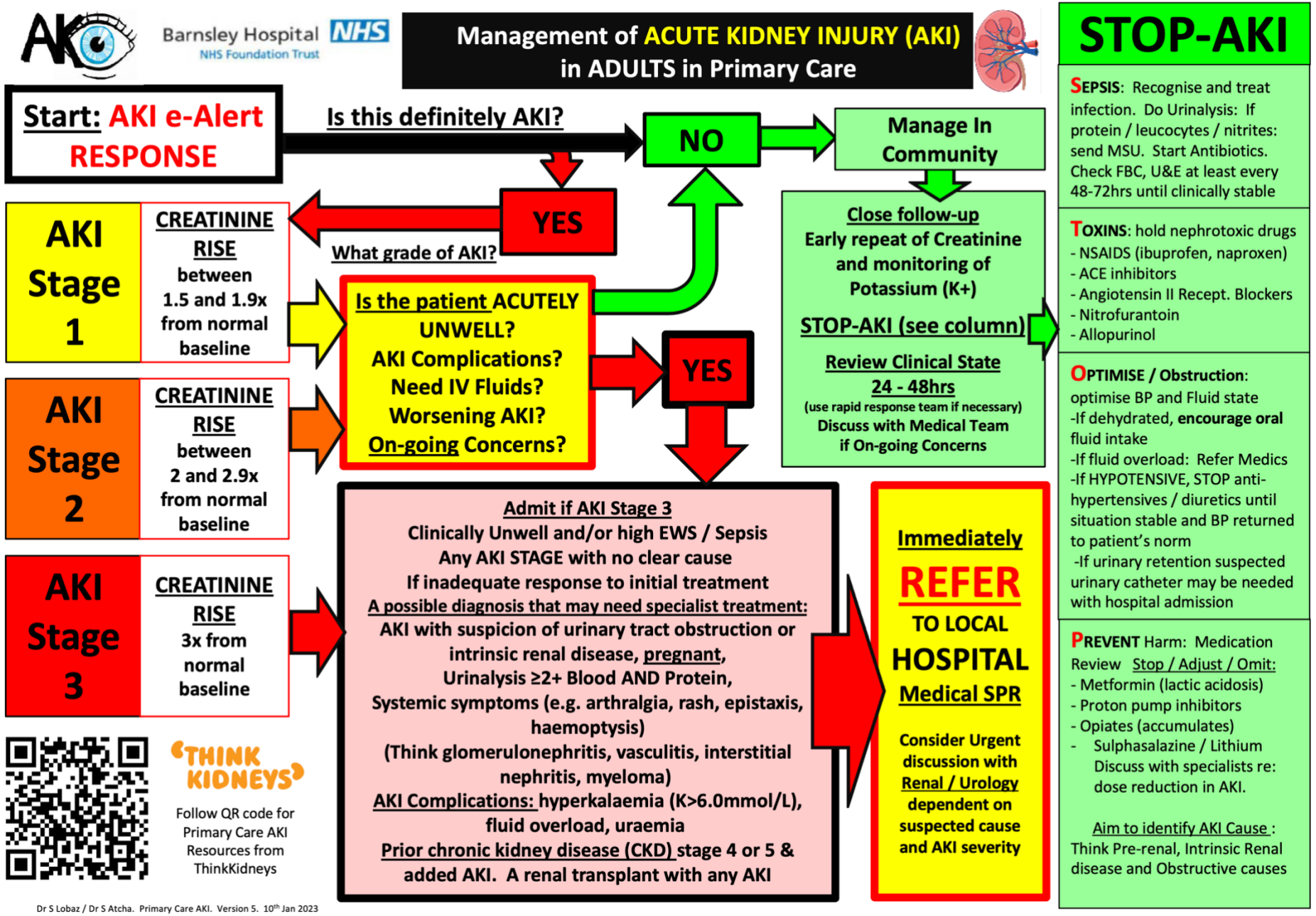
(adapted with permission from Derby AKI Primary Care Guidelines, Prof Selby)

Dr Steve Lobaz

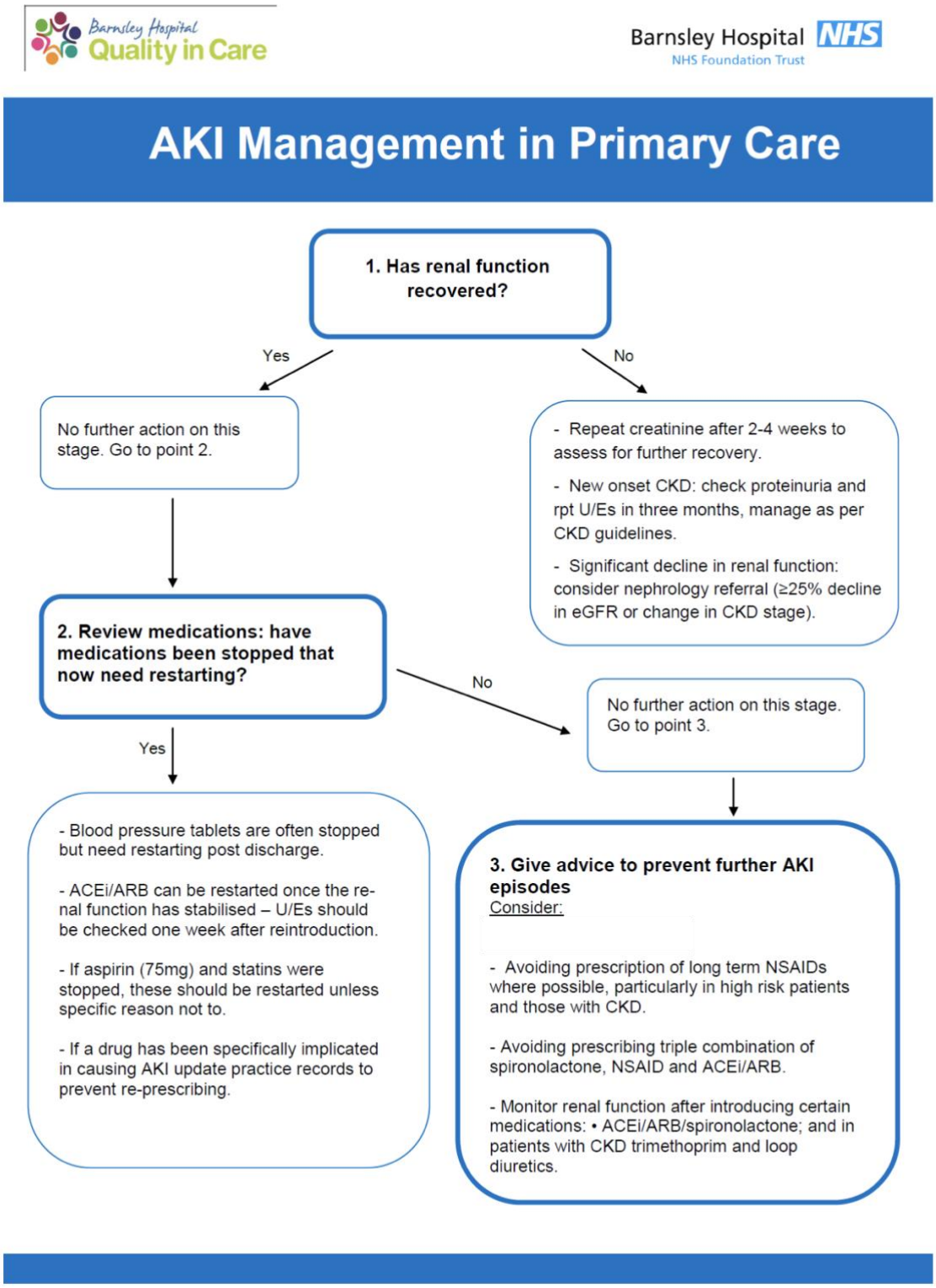
Consultant in Anaesthetics and Intensive Care Medicine, Fluid and AKI Lead.
Barnsley Hospital NHS Foundation Trust

Contents:

FLOW DIAGRAM 1: MANAGEMENT OF PATIENTS WITH AKI DETECTED IN PRIMARY CARE / RESPONSE TO AKI e-ALERTS



FLOW DIAGRAM 2: POST AKI MANAGEMENT IN PRIMARY CARE (Jan 2023 Update)



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 their 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 (AKI e-alerts), 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/primary-care/

- 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 KDIGO 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 ≥27micromol/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 the KDIGO group categorising into three stages (stage 1 being the least severe and stage 3 being the most severe) as follows:

Stage	Serum Creatinine (Cr) Increase	Urine Output (UO)
1	**1.5-1.9x** baseline OR **≥26.5µmol/L**	**<0.5ml/kg/hr** for 6-12hrs
2	**2.0-2.9x** baseline	**<0.5ml/kg/hr** for ≥12hrs
3	**3.0x** baseline OR Rise **≥353.6µmol/L** OR Initiation of Renal Replacement Therapy OR, in patients <18yrs old, decrease in eGFR to <35ml/min per 1.73m^3	**<0.3ml/kg/hr** for >12hrs OR **Anuria** for ≥12hrs

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 to nephrology (renal) early:

- AKI with blood and protein (≥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**
  - o Increasing age
  - o CKD
  - o Diabetes mellitus
  - o Heart failure
  - o Liver disease

- **Situation specific**
  - o Hypovolaemia, dehydration, reduced oral intake
  - o Hypotension
  - o Sepsis
  - o 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 (ARBs) 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/ARBs 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. **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.

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

3. **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.

4. **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 or e-alert will have a recent baseline to compare. The result has to be taken in 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 (Royal Hallamshire, Sheffield), intrinsic renal disease to Nephrology / Renal (Northern General, Sheffield)

5. Approach to outpatient management of AKI

Think '**STOP**-AKI' for immediate management
• Avoid or correct dehydration
• Medication review
i. Consider temporary suspension of ACEi/ARB +/- diuretics
ii. Consider temporary suspension of metformin (to avoid risk of lactic acidosis)
iii. Stop nephrotoxic medications such as NSAIDs
iv. 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 help from nephrology / renal SpR on call (via Northern General, Sheffield switchboard) for patients who are getting worse despite the above

6. If in doubt, discuss with nephrology (renal)
• Contact nephrology / renal SpR on call (via Sheffield switchboard)

STOP-AKI

SEPSIS: Recognise and treat infection. Do Urinalysis: If protein / leucocytes / nitrites: send MSU. Start Antibiotics. Check FBC, U&E at least every 48-72hrs until clinically stable

TOXINS: hold nephrotoxic drugs
- NSAIDS (ibuprofen, naproxen)
- ACE inhibitors
- Angiotensin II Recept. Blockers
- Nitrofurantoin
- Allopurinol

OPTIMISE / Obstruction:
optimise BP and Fluid state
-If dehydrated, **encourage oral** fluid intake
-If fluid overload: Refer Medics
-If HYPOTENSIVE, STOP anti-hypertensives / diuretics until situation stable and BP returned to patient's norm
-If urinary retention suspected urinary catheter may be needed with hospital admission

PREVENT Harm: Medication Review Stop / Adjust / Omit:
- Metformin (lactic acidosis)
- Proton pump inhibitors
- Opiates (accumulates)
- Sulphasalazine / Lithium

Discuss with specialists re: dose reduction in AKI.

Aim to identify AKI Cause :
Think Pre-renal, Intrinsic Renal disease and Obstructive causes

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

Since February 2016, hospital discharge summaries from Barnsley Hospital have had a specific section that should detail information about AKI episodes that have occurred during a patient's hospital stay.

The following information should be provided to Primary Care in the discharge letter (D1):

1. **AKI Severity (KDIGO Stage 1, 2 or 3):** The AKI Stage should be documented along with any risk factors, cause of AKI and any follow-up that has been arranged or required (e.g. with GP, Hospital physician or Renal / Urology Team) on discharge
2. **Medications Advice:** Any medications that have been stopped or held in hospital and those that are needing to be reviewed / reintroduced by the GP should be documented.
3. **Type blood tests (e.g. U&Es) and**
4. **Frequency of blood tests:** The Royal College of Physicians recommends that blood tests are only required on discharge for monitoring if new medications are to be introduced or previously held medications restarted and/or unresolved AKI issues remain.

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 (U&Es, eGFR) 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 (see links below), which includes an assessment of proteinuria and a repeat creatinine at three months.

- UK e-Chronic Kidney Disease (CKD) (http://www.renal.org/information-resources/the-uk-eckd-guide#sthash.4RreRG9r.dpbs)

- NICE guideline CG182 (https://www.nice.org.uk/Guidance/CG182)

For eGFR online calculation see: http://pathlabs.rlbuht.nhs.uk/eGFRcalculator.htm

• If you are concerned about a significant reduction in renal function following an episode of AKI, then contact nephrology/Renal (Sheffield) for advice.

2. **Review medications**

• Restart appropriate medications that may have been stopped during an AKI episode:

i. Blood pressure tablets are often stopped but need restarting when BP rises during recovery.

ii. ACEi/ARBs can be restarted (unless specific advice to the contrary) once the renal function has stabilised – U/Es should be checked one week after reintroduction.

iii. 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.
iv. 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 previously

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

USEFUL RESOURCES:
NHS England AKI Programme (Think Kidneys) www.thinkkidneys.nhs.uk

https://www.thinkkidneys.nhs.uk/aki/resources/primary-care/

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