Acute Kidney Injury

Best Practice Guidance: Responding to AKI Warning Stage Test Results for Adults in Primary Care

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Think Kidneys is a national programme led by NHS England in partnership with UK Renal Registry

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1. An Overview of Acute Kidney Injury

What is AKI?

Acute kidney injury (AKI) simply means a sudden reduction in renal function that makes maintaining fluid, electrolyte and acid-base balance difficult. The term has replaced 'acute renal failure' and includes earlier stages of kidney damage other than just 'failure'.[1] The diagnosis of AKI and its staging is based on acute changes in serum creatinine and/or a reduction in urine output (see Box 1 on page 6).[1 2] It is **not** a traumatic injury to the kidney as the name may imply, rather a clinical syndrome with various causes and variable outcomes.[1]

What causes AKI?

There are many causes of AKI. Most cases occur in conjunction with co-existing acute illness and are a result of infection, hypovolaemia, hypotension or medication effects; these causes, often in combination, account for up to 80% of cases, on a background of increased risk.[1-4] Patients at risk are often frail with co-morbidities including diabetes, chronic kidney disease (CKD), chronic liver disease and heart failure. Post-renal causes (e.g. bladder outflow obstruction) accounts for between 5 to 10% of cases of AKI.[3] Intrinsic kidney diseases are less common, but it is important they are not missed because early access to specialised management in these cases is crucial. This category includes a variety of less common conditions such as: systemic vasculitis, rapidly progressive glomerulonephritis, drug induced tubulo-interstitial nephritis, and myeloma-related kidney disease.

Any drug that reduces blood pressure, circulating volume, or renal blood flow will increase the ***risk*** of AKI. NSAIDs reduce renal blood flow by reducing intrarenal vasodilator prostaglandins. Diuretics may worsen hypovolaemia. All blood-pressure-lowering drugs should be reviewed in acute illness. In addition to their effect on blood pressure, ACE inhibitors (ACEi) and angiotensin II receptor blockers (ARB) also 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 hypovolaemia/hypotension. ACEi and ARBs also increase the risk of hyperkalaemia by inhibiting aldosterone production.

Why is early recognition of AKI Important?

AKI is extremely common in hospitalised patients, occurring in 10-20% of emergency hospital admissions, and is associated with extremely poor outcomes.[5] However, AKI is not just a secondary care problem – primary care has a crucial role to play, particularly in prevention, early detection and management as well as post-AKI care.

Poor outcomes associated with AKI include:

- 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)[5]
- Increased length of hospital stay and higher healthcare resource utilisation[1 6]
- Incomplete recovery of kidney function – many patients will be left with chronic kidney disease (CKD) and/or are at increased risk of progressive loss of GFR over time[1 7]

- Increased risk of poor long term outcomes: reduced life expectancy, increased cardiovascular risk, and poorer quality of life[1]

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

Why does Primary Care have an important role?

Think Prevention: Up to two-thirds of patients with AKI have already developed it by the time they are admitted to hospital, so preventative strategies need to include a focus on primary care.[5]

Think Early Detection and Management: From April 2016, AKI Warning Stage Test Results generated 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. There is a need to ensure that these test results are considered in a clinical context, with an imperative of treating the patient, not the test result.

At present, data on the detection of AKI in primary care is limited. However, early findings provided to the NHS England Think Kidneys Programme from six regions across England suggest that a FTE GP may expect to receive **one** AKI Warning Stage Test Result **every 1 to 2 months**. Of these, over half are likely to be AKI Warning Stage 1 Results.

Think Post-AKI Care: Improvements are required at discharge from hospital for patients who have had an episode of care complicated by AKI.[8 9] Patients who have recovered from AKI need clear plans for follow up. This includes 1) early review to assess the extent of renal recovery as well as review of long term medications that may have been stopped during admission (see When to restart drugs stopped during an episode of AKI); and 2) longer-term monitoring to assess for the development or progression of CKD.[7] Review appointments provide an opportunity to communicate the diagnosis of AKI and raise awareness of associated risks.

2. Responding to an AKI Warning Stage Test Result in Primary Care

AKI is a clinical syndrome, not merely a biochemical diagnosis. As such, there is a need to ensure that test results are considered with an understanding of the clinical context in which a blood test was taken. Communicating and providing access to salient clinical information when taking blood tests through use of laboratory forms, in medical records, and through hand over documents can help support a timely and appropriate response to a test result. This is particularly important when the alert is communicated out of hours to GPs with no knowledge of the patient.

Three overarching principles guiding the communication of patient diagnostic tests have been published by NHS England and include[10]:

The first is that the clinician who orders the test is responsible for reviewing, acting and communicating the result and actions taken to the General Practitioner and patient even if the patient has been discharged.

The second is that every test result received by a GP practice for a patient should be reviewed and where necessary acted on by a responsible clinician even if this clinician did not order the test.

The third is that patient autonomy should be respected, consideration given to reasonable adjustments for people with learning disabilities and mental health problems and, where appropriate, families, carers, care coordinators and key workers should be given the opportunity to participate in the handover process and in all decisions about the patient at discharge.

This section is not exclusive but highlights **key factors** to consider when responding to an AKI Warning Stage Test Result for an adult in primary care:

- What is an AKI Warning Stage Test Result?
- Is it AKI?
- What is the stage of AKI?
- Is there a history of acute illness?
  - Think sepsis
  - Think hypotension
  - Think hypovolaemia
- Is there evidence of hyperkalaemia?
- Does the patient have existing significant co-morbidities and risk factors? ○ AKI Warning Stage Test Results in the context of Chronic Heart Failure ○ AKI Warning Stage Test Results in the context of Chronic Kidney Disease
- Has there been a recent increase in the dose of pharmacological therapy?
- Is intrinsic kidney disease suspected? Is
- urinary tract obstruction suspected?

What is an AKI Warning Stage Test Result?

Generation of an alert for AKI is best regarded as a two-step process. The first stage is the detection of creatinine changes consistent with AKI. This will be delivered by the NHS England detection algorithm running in the laboratory information management system (LIMS). This algorithm automatically identifies ***potential*** cases of acute kidney injury from laboratory data in real time and produces a test result (i.e. AKI stage 1, 2 or 3), reported alongside the serum creatinine result. The test result is named an 'AKI Warning Stage'.

The second stage of the process is the communication of the AKI result to clinicians – the alerting phase of the process. Positive AKI Warning Stage results will be sent from the laboratory system to General Practice Clinical Systems either through interruptive and/or non-interruptive methods of communication. Systems need to be established to ensure timely communication of test results to both in and out of hours primary care services. [11]

Once a test result is communicated, the primary care team need to decide how quickly (if at all) to act on the test result, and what action to take. Table 1 provides guidance to support a timely and appropriate initial response to AKI Warning Stage Test Results in Primary Care. With recognition that computerised algorithmic interpretation of serum creatinine tests may generate both false positives ('Pseudo-AKI') and false negatives ('Atypical AKI'),[1] a key question to consider is **'Why was the blood test taken?** Was the blood test taken in the context of:

- Routine chronic disease monitoring?
- Drug monitoring?
- Assessment of acute illness?

Is it 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/or reduction in urine volume (see Box 1).[1] In practice, the urine output criteria can only be applied to hospitalised patients who are catheterised. However, a reliable history of low or absent urine output should alert the clinician to the possibility of AKI.

Box 1. Staging of Acute Kidney Injury[1]

Adults:

AKI stage 1 is a rise of ≥1.5x baseline level, which is known or presumed to have occurred within the prior 7 days; or of >26 micromol/L within 48h, or a urine output <0.5mL/kg/h for 6-12h
AKI stage 2 is a rise of ≥2x baseline or a urine output <0.5mL/kg/h for ≥12h
AKI stage 3 is a rise of ≥3x baseline or a rise of ≥1.5 baseline to >354 micromol/L, a urine output <0.3mL/kg/h for ≥24h or anuria for ≥12 h

For age <18 years, AKI stage 3 is also defined as a rise in serum creatinine to >3 x the upper limit of the age-related reference range. The urine output criteria also differ for age <18 years: stage 1 is <0.5mL/kg/h for >8h; stage 2 is <0.5mL/kg/h for more than 16h; stage 3 is <0.3mL/kg/h for 24h or anuria for 12h.

Access to clinical information is important in order to ascertain whether an AKI Warning Stage Test Result represents true AKI. As indicated in Box 1, AKI is defined by any of the following:

- Increase in serum creatinine by >26micromol/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 creatinine changes that occur over a longer time period may reflect progression of chronic kidney disease, for example, rather than acute kidney injury. If the blood tests used to assimilate the patient's usual ('baseline') creatinine have not been taken recently, then clinical context should be incorporated to help decide if the creatinine rise is likely to be 'acute' (and thus consistent with 'acute kidney injury'). A further repeat blood test may be helpful in such circumstances. Factors to consider include:

- Is the patient acutely unwell? If so, AKI is more likely.
- Check if the patient has had a previous creatinine result
- Is there at least a 50% rise in
- creatinine? Is this a false positive alert?
    - Is the patient known to have chronic kidney disease (CKD) and is the change in serum creatinine due to progression of CKD rather than an acute change?
      Particularly consider this if the baseline creatinine values are from nearly 12 months ago
      or if their real 'baseline' creatinine values are different from the one being used by the AKI Algorithm. Look at all the serum creatinine values over a longer period of time to see the pattern.
    - Has the patient been treated with Trimethoprim? This drug can cause an increase in serum creatinine without changing Glomerular Filtration Rate, by inhibiting tubular secretion of creatinine – and can thus cause a 'false positive' test result.
    - Has the patient recently completed a pregnancy? Serum creatinine naturally falls during pregnancy, so a rise in creatinine after delivery may cause a false positive warning stage test result.
    - Depending on the clinical history, consider 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).

There is also a need to consider the possibility of a false negative alert. Patients with a history of recurrent AKI may not always trigger an AKI alert if their median creatinine (days -7 to -365) is elevated by previous episodes of AKI (leading to a spuriously high baseline creatinine being generated within the current AKI algorithm).

In summary, AKI Warning Stage Test Results are only an aid to prompt recognition of AKI. AKI alerts should not be relied upon to detect all AKI cases in a timely fashion, nor replace close inspection and comparison of patient serum creatinine measurements. To reiterate, AKI is a clinical diagnosis – The gold standard for AKI diagnosis is clinician review of current and previous blood results – taking clinical context into account – and comparing against AKI diagnostic and staging criteria.

What is the stage of AKI?

The severity of AKI is described by categorising into three stages, with stage 1 being the least severe and stage 3 being the most severe (see Box 1).[1] Table 1 provides guidance on the timeliness of clinical response according to stage, with consideration of a more prompt response required with increasing severity irrespective of other clinical factors.

Increasing severity of AKI correlates with higher risk of worse outcomes. Depending on clinical context, AKI stage 1 can usually be managed in primary care whereas AKI stage 3 should usually be managed in secondary care. Table 1 also highlights risk factors and clinical features prompting earlier clinical review.

Is there a history of acute illness?

If a blood test has been taken in the context of an episode of acute illness, then consider AKI likely until proven otherwise irrespective of stage.

What was the reason for the blood test? Have kidney function blood tests been taken in the context of a patient presenting with an episode of acute illness but which was deemed not to require immediate admission at the point of initial assessment? – see Tables 1 & 2 to support timely assessment and management. Examples for consideration include patients who have had blood tests taken in the context of an episode of acute illness such as diarrhoea or vomiting caused by gastroenteritis, urinary tract infection, or respiratory infection. Reviewing the patient to assess for evidence of sepsis, hypotension and hypovolaemia will help determine appropriate management.

Think Sepsis:

Clinical evaluation of acute illness requires an assessment for infection and particularly for sepsis, which demands urgent attention.[12] Sepsis is defined as 'life-threatening organ dysfunction caused by a dysregulated host response to infection.'[12] In lay-terms, it is a 'life-threatening condition that arises when the body's response to infection injures its own tissues.'[12] In order to aid recognition, the National Confidential Enquiry into Patient Outcome and Death (NCEPOD) report into sepsis recommends that 'an early warning score' should be used in both primary and secondary care.[13] Patients suspected of having sepsis require immediate admission, whether or not they have AKI.[12]

Think Hypotension:

The development of absolute hypotension (systolic blood pressure less than 90 mmHg) or relative hypotension (an unexpected fall of 40 mmHg from a previous baseline even if blood pressure remains within the normal range) is a clinical red flag. In this setting, consider hypovolaemia, sepsis, review all anti-hypertensive drugs and consider the need for hospital admission.[14] [15]

Think Hypovolaemia:

Hypovolaemia associated with any type of insult including dehydration or over-diuresis is probably the most modifiable risk factor for acute kidney injury. Management in the community includes ensuring maintenance of fluid intake and correction of hypovolaemia. Patients who are particularly at risk of dehydration in the community include those who have neurological or cognitive impairment or disability, which may mean limited access to fluids because of reliance on a carer.[2]

Assessment of volume status is essential. Evaluation of volume status should be based on history (particularly of fluid intake and all fluid losses) and clinical examination (including pulse, blood pressure (BP), jugular venous pressure, capillary refilling, dry axillae; recent change in weight and postural change in pulse and BP; and absence of signs of fluid overload including peripheral oedema).[4 16]

Note that patients may develop non-oliguric or polyuric AKI as well as oliguric AKI. If there is evidence of hypovolaemia, admission should be considered for appropriate intravenous fluid replacement and monitoring.

Is there evidence of hyperkalaemia?

The presence of hyperkalaemia is a complicating factor and its presence needs to be considered when responding to AKI Warning Stage Test Results.

The Renal Association guidelines recommend that all patients with severe hyperkalaemia (≥ 6.5 mmol/l irrespective of kidney function) are referred to secondary care for immediate assessment and treatment.[17]

The urgency in assessment of patients with mild (K+ ≥ 5.5-5.9 mmol/L) or moderate (K+ 6.0-6.4 mmol/L) hyperkalaemia depends on clinical context. Findings from a Think Kidneys consensus process, using RAND methodology, indicated a need to consider earlier review for patients with moderate hyperkalaemia associated with an AKI Warning Stage Test Result irrespective of AKI severity (see Table 1)[17-19].

http://bit.ly/hyperkalaemia-guidelines

Does the patient have existing significant co-morbidities and risk factors?

Box 2. Risk Factors associated with Acute Kidney Injury[1 2]

Patient specific – Susceptibility	Situation specific – Exposure
• Older age • Immunosupressed or deficient immunity e.g. malnutrition, patients with cancer • CKD (eGFR <60) • Diabetes mellitus • Heart failure • Liver disease • Past history of AKI • Neurological or cognitive impairment or disability, which may mean limited access to fluids because of reliance on a carer • Symptoms or history of urinary tract obstruction, or conditions that may lead to obstruction	• Hypovolaemia, dehydration, reduced oral intake • Hypotension • Sepsis • Post-operative • Use of iodinated contrast agents within the past week • Use of drugs such as non-steroidal anti-inflammatory drugs [NSAIDs], angiotensin-converting enzyme [ACE] inhibitors, angiotensin II receptor antagonists [ARBs] and diuretics) within the past week, especially if hypovolaemic

Box 2 highlights factors to consider when responding to an AKI Warning Stage Test Result. Responding to test results in patients with Chronic Heart Failure and/or Chronic Kidney Disease requires particular attention.

AKI Warning Stage Test Results in the context of Chronic Heart Failure

Patients with chronic heart failure represent a population with increased morbidity and mortality, and account for 5% of all emergency medical admissions to hospital.[20] Responding to AKI Warning Stage Test Results generated for patients with known chronic heart failure requires particular attention:

- Patients are at increased risk of acute kidney injury during episodes of acute illness.[2] Patients with chronic heart failure require increased monitoring of their renal function:
- Pharmacological treatment of chronic heart failure can include use of angiotensin-converting enzyme (ACE) inhibitors, angiotensin 2 receptor antagonists (ARB), aldosterone antagonists and diuretics, all of which have a renal effect.[20 21]

Better evidence on how to respond to changes in serum creatinine in the context of chronic heart failure is needed. Trade-offs exist and whilst awaiting an evidence base, an important rule of thumb is to treat the patient and not the blood result. In order to put the blood test in a clinical context, key questions to consider include:

- What is the patient's clinical status and stability? What is the patient's volume status: is the patient fluid overloaded? About right? Or is it possible that they have become over-diuresed? In which case, hypovolaemia may be causing genuine AKI.

- Does the patient have an inter-current acute illness?
- Has there been a recent increase in the dose of drug therapy (diuretics, ACE Inhibitors, ARBs, aldosterone antagonists – see below)?
- Is there any abnormality of serum potassium or serum sodium? – hyponatraemia and hypo- or hyper-kalaemia may justify a change in treatment or specialist referral even if the rise in serum creatinine does not.

AKI Warning Stage Test Results in the context of CKD

CKD is the most consistently reported condition associated with acute kidney injury and it is advised that measuring serum creatinine should be considered in adult patients who have CKD and present with an episode of acute illness.[2 7]

As indicated above, it is important to determine whether the AKI Warning Stage Test Result represents true AKI (see pages 6 and 7). In terms of timeliness in response, consider earlier clinical review for patients with a known history of CKD stages 4 or 5, or in a patient who has had a history of a kidney transplant (see Table 1). This reflects NICE guidance, which recommends discussing the management of these patients with a nephrologist as soon as possible.[2]

Has there been a recent increase in the dose of pharmacological therapy?

As indicated within NICE Clinical Guidelines for Acute Kidney Injury (cg169), it is important to assess whether the introduction, or change in dose, of a diuretic, ACE Inhibitor, ARB, or aldosterone antagonist may have contributed to a significant rise in serum creatinine.[2 7 20 21] Understanding clinical context is central to interpreting these changes.

Current guidance is that serum creatinine should be checked between one to two weeks after initiation of an ACEi/ARB and that an increase of up to 30% from baseline is acceptable (and up to 50% in patients with chronic heart failure), as long as the patient is asymptomatic and the rise is stable.[7 21] 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 >26µmol/l does not apply because the gap between blood tests should be >48hrs).[1]

Is Intrinsic Kidney Disease suspected?

Think about acute nephritis based on history or examination including evidence of proteinuria and haematuria on urinalysis without evidence of urinary tract infection, or trauma due to catheterisation. Consider systemic symptoms associated with intrinsic renal disease: arthralgia, arthritis, mononeuritis multiplex, rash, uveitis, epistaxis or haemoptysis. There are some 'red flag' signs that help to identify this group of AKI patients so they can be referred to nephrology early.[1] [2] Key questions to consider:

- Has urinalysis been carried out and what did it show?
  - Dip the urine: this is an important diagnostic step.
  - AKI and negative urinalysis: usually pre-renal causes (also consider drug causes).
  - AKI with blood and protein only (without evidence of UTI, or trauma due to catheterisation): consider wider differential diagnoses including intrinsic kidney disease.

- 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.
    - AKI in relation to the introduction of a new drug (Proton Pump Inhibitor, NSAID, antibiotic, diuretic, allopurinol) without any other explanations for AKI may indicate drug-induced interstitial nephritis (NB eosinophilia should increase suspicion of drug-induced interstitial nephritis, but many patients with this do not have eosinophilia).
- AKI with systemic symptoms of inflammatory process: vasculitic rash, arthralgia, epistaxis or haemoptysis.
- AKI in context of high calcium (hypercalcaemia can cause AKI; may also be an indicator of myeloma).

Consider early clinical review if intrinsic kidney disease is suspected (see Table 1). This reflects NICE guidance, which recommends discussing the management of AKI with a nephrologist as soon as possible when the differential diagnosis includes tubulointerstitial nephritis, glomerulonephritis (indicated by haematuria/proteinuria), systemic vasculitis, or myeloma.[2]

Is urinary tract obstruction suspected?

Consider urinary tract obstruction when history or examination suggests the patient may have renal stones, pyonephrosis, blocked catheter, pelvic mass, enlarged prostate, known prostate or bladder disease, abdominal or pelvic carcinoma, retroperitoneal fibrosis, known previous hydronephrosis, recurrent UTI; or other conditions consistent with possible obstruction.[1 2]

N.B. Think about concomitant pathologies (e.g. pre-renal and post-renal) contributing to the development of AKI. Think about the cause of AKI and if clinical assessment points to evidence of urinary tract obstruction then the patient needs urgent specialist urology referral.

Table 1. Acute Kidney Injury: Recommended response times to AKI Warning Stage Test Results for Adults in Primary Care

AKI Warning Stage Test Result Confirm **or** refute automated AKI Test Result by comparing patient's current creatinine **within clinical context** against baseline creatinine	**Clinical Context Within Which Blood Test Taken**[#] If clinical context is unknown, then assume high pre-test probability until proven otherwise	
	LOW Pre-test Probability of AKI Stable Clinical Context	**HIGH Pre-test Probability of AKI** Context of Acute Illness
AKI Warning Stage 1 Current creatinine ≥1.5 x baseline level (*or* creatinine rise >26 µmol/L ≤48 hrs)	**Consider clinical review ≤ 72 hours of e-alert*** **If AKI confirmed**→ manage as per table 2	**Consider clinical review ≤ 24 hours of e-alert*** **Likely Stage 1 AKI**→ manage as per table 2
AKI Warning Stage 2 Current creatinine ≥2 x baseline level	**Consider clinical review ≤ 24 hours of e-alert*** **If AKI confirmed**→ manage as per table 2	**Consider clinical review ≤ 6 hours of e-alert*** **Likely Stage 2 AKI**→ manage as per table 2
AKI Warning Stage 3 Current creatinine ≥3 x baseline level (*or* creatinine 1.5 x baseline and >354 µmol/L)	**Consider clinical review ≤ 6 hours of e-alert*** **If AKI confirmed**→ consider admission	**Consider Immediate Admission*** Likely Stage 3 AKI

[#]Clinical Context

Why was the blood test taken?

- Routine chronic disease monitoring
- Drug monitoring
- Assessment of acute illness

Creatinine rise within stable clinical context **may** reflect unstable CKD instead of AKI, especially if **longer time period** between current and baseline creatinine.

***AKI Risk Factors/Clinical Features Prompting Earlier Review**

- Poor oral intake/urine output
- Evidence of hyperkalaemia, especially if moderate(K^+ 6.0-6.4) or severe ($K^+ \geq 6.5$)[¥]
- Known history of CKD stages 4 & 5 or history of kidney transplant
- Deficient Immunity
- Frail with co-morbidities (CKD, diabetes, heart failure, liver disease, neurological or cognitive impairment)
- Past history of AKI
- Suspected intrinsic kidney disease
- Suspected urinary tract obstruction

¥ UK Renal Association Clinical Practice Guidelines (2014) recommends emergency assessment and treatment of severe hyperkalaemia ($K^+ \geq 6.5$mmol/l) – http://bit.ly/hyperkalaemia-guidelines Refer to main guidance document. *The table is a guide to support an initial response to an AKI Warning Stage Test Result but clinical judgement must prevail.*

The table does not apply to children and young people (<18 years) or patients receiving end of life care.

Table 2: Recognising and Responding to Acute Kidney Injury for Adults in Primary Care*

"Think" Cause	"Think" Medication#	"Think" Fluids	"Think" Review¥
History of acute Illness? • Think Sepsis • Think Hypotension Intrinsic kidney disease? (E.g. vasculitis) • Think Urinalysis Urinary tract obstruction?	Any medication which could exacerbate AKI? Consider withholding: • NSAIDs • Diuretics • Antihypertensive medication Any medication which may accumulate and cause harm during AKI? Any new medication that may cause AKI?(E.g. drug induced tubulo-interstitial nephritis)	What is the patient's volume status? If hypovolemia present: • When did patient last pass urine? • Can the patient increase fluid intake? • Is admission for IV fluid replacement and monitoring required? Does the patient have and/or need carer support?	Does the patient need acute admission? If not, when will you review? Have you ensured handover?¥

*Refer to main guidance document – Responding to AKI Warning Stage Test Results in Primary Care

Refer to medicines optimisation toolkit for primary care http://www.thinkkidneys.nhs.uk/aki/medicines-optimisation-for-aki

¥ Refer to overarching principles in communication of diagnostic test results https://www.england.nhs.uk/patientsafety/discharge

The table is a guide to support recognition and response to AKI in primary care
The table does not apply to children and young people (<18 years) or patients receiving end of life care

Appendix A: Other Useful Resources

- **Guidelines for Medicines Optimisation in Patients with AKI**

  Points to note and factors to consider in the medicines management of patients either with, or at risk of AKI. For example, which medications should or should not be suspended, which may be used with caution and alternative therapeutic options.

  Quick Guide to Potentially Problematic Drugs and Actions to Take in Primary Care

- **When or if to re-start drugs after an episode of AKI**

- **Patient Leaflets** – for 1) patients at risk of AKI, and 2) a patient who has had an episode of AKI

Other resources to help your practice include:

- **A short film on AKI and primary care**
- **Statement on 'Sick Day Guidance' from Think Kidneys**
- **Communities at Risk of Developing AKI – publication detailing those most at risk of AKI**
- **Understanding what the public know about their kidneys** – report of low awareness and understanding of kidneys, their function and how to keep them healthy
- **Why measure AKI data?** Background to the patient safety alert for AKI and prevalence
- The RCGP e-learning renal module is in the final stages of development and includes AKI– should be live during June 2016.

For more information on AKI and resources on its prevention, detection, treatment and management, visit www.thinkkidneys.nhs.uk/aki

Appendix B: Acknowledgements

The following organisations, working groups and individuals contributed to the development of this guidance document:

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