NHS
Greater Glasgow and Clyde

CLINICAL GUIDELINE

eGFRsupport Acute Kidney Injury - Investigations and Management

A guideline is intended to assist healthcare professionals in the choice of disease-specific treatments.

Clinical judgement should be exercised on the applicability of any guideline, influenced by individual patient characteristics. Clinicians should be mindful of the potential for harmful polypharmacy and increased susceptibility to adverse drug reactions in patients with multiple morbidities or frailty.

If, after discussion with the patient or carer, there are good reasons for not following a guideline, it is good practice to record these and communicate them to others involved in the care of the patient.

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Important Note:

The Intranet version of this document is the only version that is maintained.
Any printed copies should therefore be viewed as 'Uncontrolled' and as such, may not necessarily contain the latest updates and amendments.

Acute Kidney Injury (AKI)

Introduction

The definition of AKI is an abrupt and sustained decline in glomerular filtration rate (GFR). If creatinine is rising, GFR may be close to zero and therefore eGFR is of no value in the assessment of AKI. AKI is classed into three stages (table 1). Common risk factors for AKI are listed in table 2. This list is not exhaustive.

Box 1 – Stages of AKI

AKI stage	Definition
I	Rise in creatinine of ≥1.5-1.9 from baseline
II	Rise in creatinine of ≥2.0-2.9 from baseline
III	Rise in creatinine of ≥3 from baseline

Box 2 – AKI risk factors

Patients at risk of developing AKI

- Age
- Diabetes
- Chronic kidney disease (CKD)
- Chronic liver disease (CLD)
- Congestive cardiac failure (CCF)
- Those on polypharmacy

Other risk factors for AKI

- Hypotension
- Hypovolaemia

  Drugs including ACEi/ARB/NSAIDs and contrast*
- Sepsis

*If contrast is necessary and a patient has an AKI or CKD with an eGFR ≤30ml/min, the radiologist should be informed and consider administering 1 litre of saline in the 12 hours prior to and following the scan. Urgent and essential scans should not be delayed to allow fluid administration.

Aetiology of AKI

This can be separated into pre-renal, intrinsic renal and post-renal causes as highlighted in figure 1.

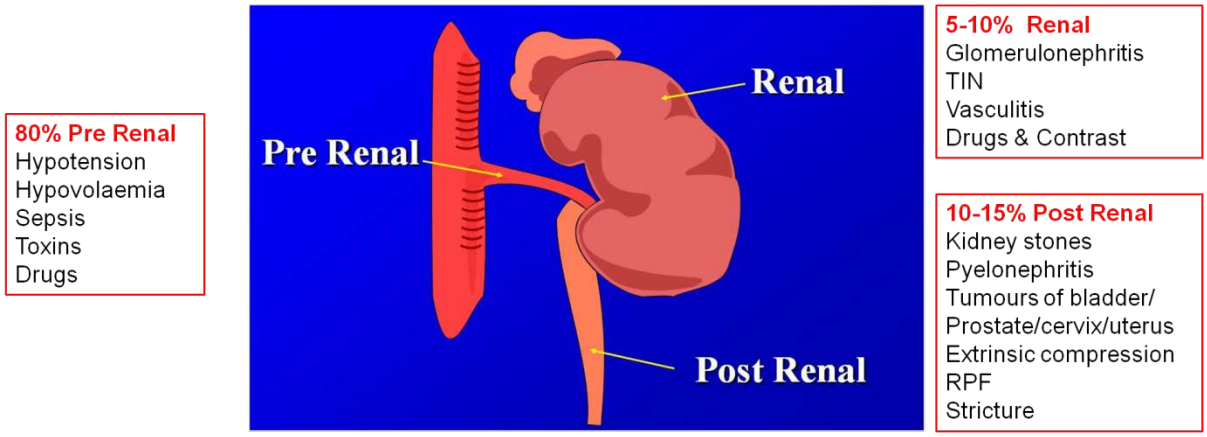
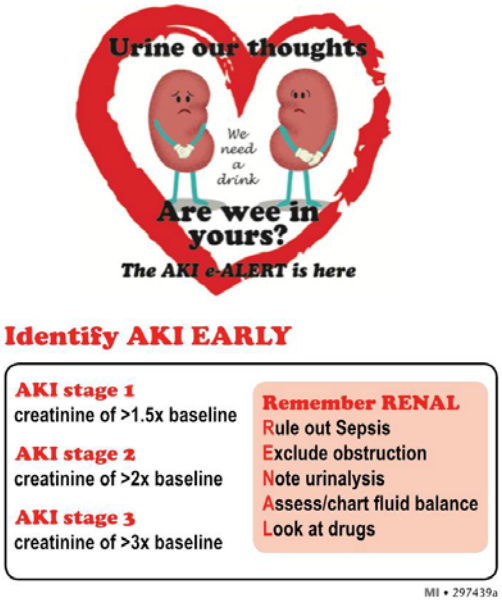


Figure 1: Aetiology of AKI

Assessment

GG&C introduced the eAlert for AKI in 2017 (figure 2). This is being rolled out across all wards and hospitals. The eAlert flags on both Portal and Trakcare when a patient has an AKI. Figure 2 is a representation of how the eAlert appears. If a patient has an AKI, consider **RENAL:**



R ule out sepsis

E xclude obstruction

N ote urinalysis

A ssess fluid balance

L ook at drugs

Figure 2: The eAlert and RENAL

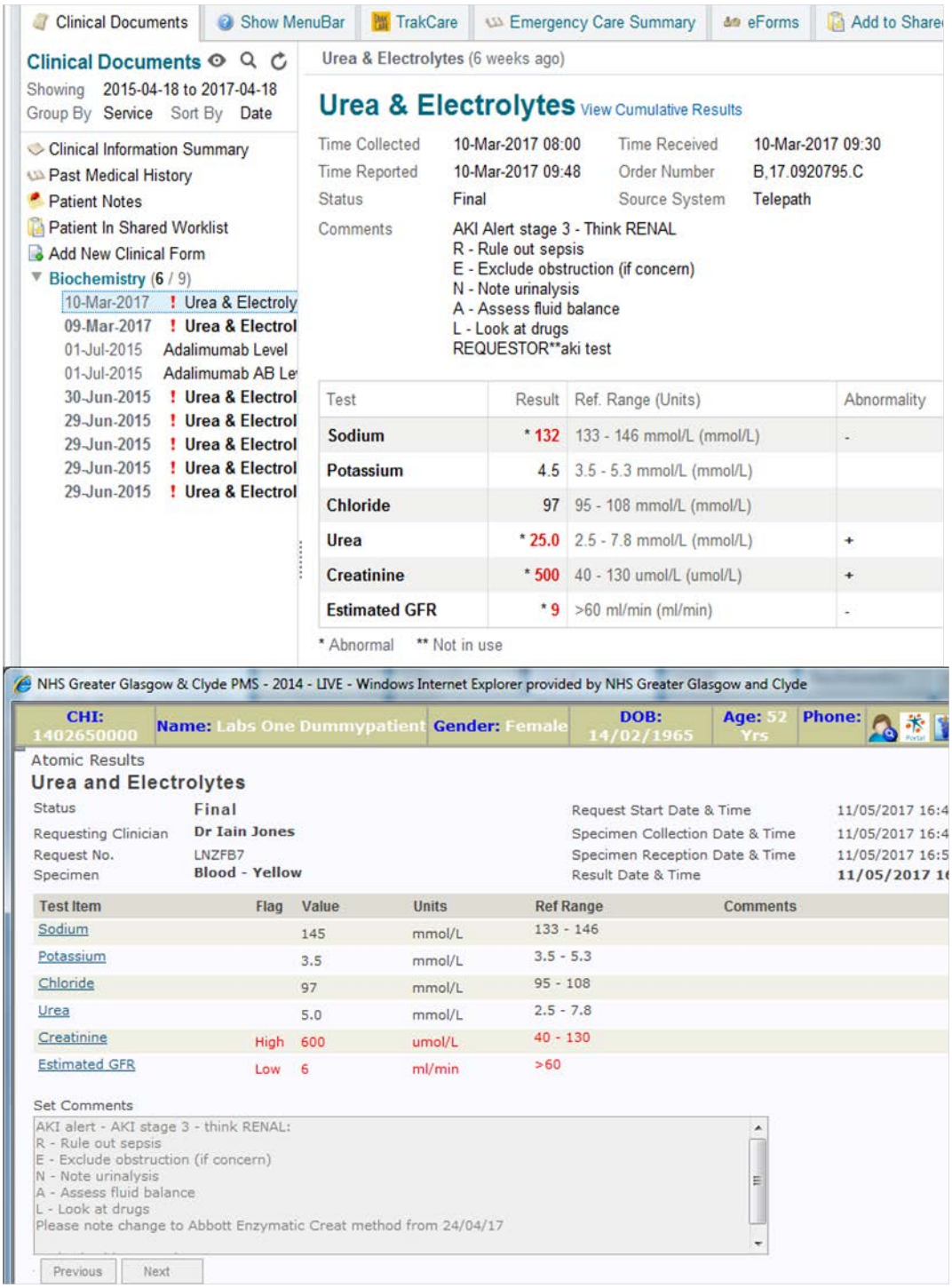


Figure 3: The eAlert as it appears on portal (top) and trakcare (bottom)

Assessment of AKI is reliant on a detailed history and examination, including urinalysis. History should include a thorough drug history including new or recent medications/supplements, illicit substances and specific questioning about oral intake, gastrointestinal symptoms, urine output, the presence of rash or joint symptoms and propensity to urinary tract infections.

The urine should be dipped and the result recorded in the notes. A urine specimen should be sent in a white top universal container for protein:creatinine ratio. In patients with symptoms and a positive urinalysis consider if it is appropriate to send a urine culture and/or a glomerulonephritis (GN) screen. A GN screen comprises rheumatoid factor, ANA, ANCA, anti-GBM antibody, complement, immunoglobulins and electrophoresis and a urinary bence jones protein. It is important to rule out obstruction; consideration should be given to renal tract ultrasound (USS), in all patients with an AKI. In some cases, an USS may not be necessary, for example, a patient with significant volume depletion who responds rapidly to fluid resuscitation. If there is any doubt, USS should be performed.

A crucial part of assessment is fluid balance. Is the patient ‘wet’ or ‘dry’ or euvolaemic? Clues from the history include asking about thirst and fluid losses. Examination includes looking at blood pressure (and comparing to the normal blood pressure of an individual patient), skin turgor, JVP and assessing for peripheral oedema, ascites and pulmonary oedema. Urine output needs to be monitored. Catheter is not a necessity but can be considered.

Management

Management of pre-renal AKI is largely supportive and the best evidence is for optimisation of fluid balance and cardiovascular support. Guidance on fluid prescription is outwith the scope of this guide but it is worth noting that if a patient is extracellularly fluid deplete, 5% dextrose is not a suitable replacement fluid on its own. If a patient is well-filled and hypotensive, consideration must be given to the use of inotropic support. Hyperkalaemia is a medical emergency and must be treated promptly.

Close attention should be paid to medication. Involve the ward pharmacist. Regular medication may need to be withheld or be prescribed at an altered dose. When prescribing new medication, consider if dose reduction is necessary. Take care with simultaneous prescriptions of medication which can affect the kidneys, for example, diuretics, analgesia and antibiotics. We are not advocating avoiding these but it is essential to consider any new prescription alongside current medication. Opiate

based analgesia can accumulate in AKI (and in CKD) and thus the risk of side effects is higher. A change in preparation/dose/both might be needed.

Certain types of GN can be treated. If there is a suspicion of GN or vasculitis then this should be discussed with the renal unit (#82417). Obstruction should be discussed with urology.

Indications for dialysis (and early discussion with the renal unit) are not absolute but would include resistant hyperkalaemia, hyperkalaemia in the setting of oliguria and fluid overload which is unresponsive to diuretics. The following are also reasons to contact the Renal Unit:

Indications to contact The Renal Unit

- Stage 3 AKI
- Stage 2 AKI that is not resolving after 24/48hrs
- Suspicion of vasculitis
- Any transplant or dialysis patient
- New nephrotic syndrome