

# Guidance for mental health professionals on the management of acute kidney injury

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## Disclaimer

To the best of our knowledge, the contents of this publication are in line with National Institute for Health and Care Excellence guidance relating to the management and treatment of acute kidney injury.

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## 1. Background

The National Institute for Health and Care Excellence (NICE) Acute Kidney Injury (AKI) clinical practice guideline ([NICE CG169: Acute kidney injury: prevention, detection and management](#)) identified a number of key priorities for implementation, which included recognising patients at risk of AKI in different settings. NHS England in partnership with the UK Renal Registry has established the 'Think Kidneys' national programme with the main aim of ensuring avoidable harm related to AKI is prevented in all care settings. A working group comprising mental health and physical health professionals, clinical biochemists, pharmacists and colleagues have developed this guidance document, which provides support to health and care professionals who are managing the care of patients at risk of, or with AKI. Further information can be found on the Think Kidneys website: [www.thinkkidneys.nhs.uk](http://www.thinkkidneys.nhs.uk)

## 2. Introduction

Acute kidney injury (AKI), previously known as acute renal failure, is a global healthcare challenge (Lewington et al 2013, Mehta et al 2015). It is characterised by a sudden decline in kidney function and is rarely caused by trauma to the kidneys. AKI can occur without symptoms and is detected through a routine blood test (serum creatinine) and/or a decrease in urine output (KDIGO 2012). It has many different causes but most commonly occurs secondary to other serious illnesses such as sepsis, or conditions associated with hypovolaemia and a drop in blood pressure e.g. vomiting, diarrhoea or blood loss. In some cases, certain medications can also affect the kidneys adversely and this can cause AKI or increase its severity. Acute kidney injury is associated with an increased risk of death, length of hospital stay, risk of chronic kidney disease (CKD) and cost to the NHS.

Older patients with chronic (long-term) medical conditions e.g. chronic kidney disease, diabetes mellitus, heart failure, cancer, who are taking regular medications are at increased risk of AKI if they become acutely ill (NICE, 2013). It is estimated that one in five emergency admissions to hospital are associated with AKI (Wang et al, 2012). Up to 100,000 deaths in hospitals are associated with AKI and a quarter to a third of these could potentially be prevented as reported by the National Confidential Enquiry into Patient Outcome and Death (NCEPOD, 2009) Adding Insult to Injury 2009 – [see link](#). The financial burden of AKI upon the NHS is significant with estimates indicating the annual cost is £1.02 billion in England for the acute care and £179 million following the episode related to an increase in patients with CKD and end stage kidney disease (Kerr et al, 2014).

## 3. Psychiatric patients at risk groups

The identification of mental health patients at risk of AKI requires a multidisciplinary approach – from the health care assistant who spots the cup of tea that hasn't been drunk, to the consultant appraising the patient's prescriptions. Multidisciplinary training in the identification and management of AKI in mental health settings should be routine.

With respect to psychiatry, there are times when patients lack the drive or motivation to care for themselves such as in severe depressive illness, and in some cases they lack the ability to care for themselves, such as in dementia. This self-neglect may lead to dehydration and consequent AKI. Dehydration may also occur associated with laxative or diuretic abuse in anorexia nervosa. The logistical challenges of performing blood tests in mental health units and the relative rarity of the need to keep fluid charts on patients in psychiatric units, compared to those admitted to acute general wards, means that vigilance by the multidisciplinary team and escalation protocols in the instance of clinical concerns regarding hydration must be robust.

Use of drugs, both prescribed and non-prescribed, can cause AKI. Lithium, commonly prescribed for bipolar disorder, or as an adjunctive treatment in major depressive disorder, must be monitored closely because of potential harm to the patient's kidneys. AKI is also seen as a side effect of recreational drugs, such as ketamine, synthetic cannabinoids and 'legal highs'. Self-harm by way of poisoning can also cause AKI through overdose of medications or use of substances such as antifreeze. The Renal Pharmacy Group has developed the [AKI Medicines Optimisation](#) Toolkit for the Think Kidneys AKI programme which supports healthcare professionals in optimising medicines in AKI or at risk of AKI. A two page medicines optimisation briefing by Royal Pharmaceutical Society / Centre for Post-graduate Pharmacy Education is also available ([Click here for details](#)).

We have highlighted some areas for concern for patients with specific issues:

### **Patients with dementia**

Patients with dementia should be reviewed at least annually in primary care – or in line with local shared care arrangements, and this is an ideal opportunity to discuss with carers the risk of AKI due to inability to self-care and particularly to regulate their fluid intake when unwell. Patients with dementia should have a full medication review and medications for co-morbidities stopped if no longer in the patient's best interest, some of which may have the potential to increase the risk of AKI. Some dementia drugs such as galantamine and memantine may not be suitable in people with severe kidney problems, so renal function should be reassessed to ensure safety.

### **Older People**

Older people in general and older people with mental health problems in particular, are vulnerable and at risk of AKI, due to:

- Normal ageing process (physiological changes in the body organs)
- Polypharmacy
- Comorbidity with mental and physical disorders

The mechanisms could be interactions between ageing physiological changes (e.g. renal handling) and drugs, drug-drug interactions, drug-disease/illness interactions, and complex interactions among all factors, compounded by mental illness/disorder.

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As for management, awareness of above factors by all professionals in prevention and timely treatment of AKI is key.

### **Patients taking lithium**

Lithium is commonly used for the treatment of affective disorder. It frequently causes diabetes insipidus, which is frequently irreversible after long-term use. Lithium toxicity causes Acute Kidney Injury. A minority of patients also develop progressive chronic kidney disease. Patients should be informed of these risks. There are specific requirements for regular monitoring of lithium levels, renal function and thyroid function for patients on lithium and the patient should be provided with a purple "[Lithium Therapy Booklet](#)" provided by the former National Patient Safety Association and downloaded from the link above.

Non-steroidal anti-inflammatory drugs, thiazide diuretics, angiotensin converting enzyme inhibitors and angiotensin receptor blockers can all raise lithium levels and trigger AKI. Lithium is renally excreted and poor hydration whether due to physical problems (e.g. vomiting or diarrhoea – which can also be signs of lithium toxicity) or mental ill-health (e.g. depressive psychomotor retardation) can cause a toxic rise in lithium levels that further increases the risk of AKI. Lithium levels should be monitored every three months for the first year and then six monthly or three monthly in at-risk patients; renal and thyroid function and serum calcium ( ) should be monitored six monthly ([NICE CG 185](#)). If the patient's renal condition is a potential concern, a lithium dose can be withheld pending the results of the blood tests.

### **Patients with delirium**

Delirium is often the pathway by which physiological crisis such as AKI is manifested, particularly in the elderly and those with pre-existing cognitive impairment. It leads to a vicious circle of greater difficulty in ensuring hydration and worsening delirium. Dementia raises the risk of delirium by fivefold (Fong et al, 2015). In those with frailty or dementia, even minor physiological disturbance can trigger delirium. Delirium frequently presents with drowsiness and retardation rather than agitation. It is important that in people with a history of mental health problems, acute changes in mental state are not automatically attributed to underlying mental ill-health rather than acute conditions like AKI.

### **Patients who misuse substances**

Patients who misuse drugs and alcohol are at risk of AKI via a number of mechanisms including: dehydration due to sedation, muscle breakdown from immobilisation, thrombosis (especially for intravenous drug misuse), amyloidosis from 'skin popping' and the direct effects of stimulant drugs. Ketamine, a dissociative anaesthetic, has direct toxic effects on the urinary tract and has been associated with AKI. Similarly heroin, cocaine, ecstasy, hallucinogens, inhalants etc. are also known to cause kidney damage. Contaminants in street drugs can also cause AKI (e.g. Levamisole-induced Vasculitis). Caution may be required when treating substance misusers with methadone or

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buprenorphine, common drugs used in opioid dependence / addiction, if they have significant renal dysfunction.

### **Patients with eating disorders**

Acute kidney injury can develop in eating disorders through a number of mechanisms. These can include stone formation, hypokalaemia and other electrolyte imbalance, as well as volume loss through induced vomiting, abuse of diuretics and laxatives, and low dietary intake. As eating disorders are more commonly seen in a relatively young population, the risk of longer term kidney damage is a concern. It is recommended that healthcare professionals should be vigilant of developing AKI in patients with eating disorders.

### **Perinatal mental health services**

As perinatal mental health services are relatively new and still in the developmental stages across the England, it is important for mental health professionals to be aware of the relevance of acute kidney injury in their services.

As outlined in the NHS England Five Year Forward Review for Mental Health, perinatal mental health services include specialist community perinatal services and inpatient mother and baby units (MBUs).

Perinatal period is an important time for overall health of mother and child because of natural physiological changes in both mother and baby. There are specific physical and mental health conditions relevant to perinatal period which service users may have both at the same time. Moreover, psychotropic drugs used may interact with mother and baby through various mechanisms e.g. through milk via breast feeding.

Through learning, training and CPD (Continuing Professional Development), mental health professionals will have knowledge, experience and skills on managing AKI in perinatal mental health services.

Some examples of pregnancy-related causes of AKI (PR-AKI) are preeclampsia, acute fatty liver of pregnancy, HELLP (Haemolysis, Elevated Liver function tests, Low Platelets) syndrome, and the thrombotic microangiopathies (thrombotic thrombocytopenic purpura, atypical haemolytic-uremic syndrome, HUS (Jim and Garovic, 2017).

## **4. What do blood test results mean?**

Taken from the Think Kidneys publication "[Acute Kidney Injury Warning Algorithm Best Practice Guidance](#)"

A rise in creatinine may indicate acute kidney injury stage 1, 2 or 3. Please note that creatinine may still appear to be within the normal range but it should be viewed against a patient's baseline creatinine result to determine whether there is an increased of a diagnosis of AKI.

<b>AKI stage 1</b>	is a rise of >1.5x baseline level, or of >26 $\mu$ mol/L within 48h, or a urine output <0.5mL/kg/h for 6-12h
<b>AKI stage 2</b>	is a rise of >2x baseline or a urine output <0.5mL/kg/h for $\geq$ 12h
<b>AKI stage 3</b>	is a rise of >3x baseline or a rise of >1.5 baseline to >354 $\mu$ mol/L, a urine output <0.3mL/kg/h for $\geq$ 24h or anuria for $\geq$ 12 h

## 5. What steps should be taken

This guidance emphasises the importance of having a shared care arrangement, established locally, between primary care and mental healthcare trusts in prevention and management of AKI.

It is also important that mental healthcare teams have a protocol agreed, at the individual team level in each mental healthcare trust, on the mechanism and process of managing AKI.



**Table1. Acute Kidney Injury: Recommended response times to AKI Warning Stage Test Results for Adults in Mental Health facilities**

AKI Warning Stage Test Result Confirm <b>or</b> refute automated AKI Test Result by comparing patient’s current creatinine <b>within clinical context</b> 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
<b>AKI Warning Stage 1</b> Current creatinine $\geq 1.5$ x baseline level (or creatinine rise $>26$ mol/L 48 hrs)	<b>Consider clinical review <math>\leq 72</math> hours of e-alert*</b> <b>If AKI confirmed</b> — manage as per table 2	<b>Consider clinical review <math>\leq 24</math> hours of e-alert*</b> <b>Likely Stage 1 AKI</b> — manage as per table 2
<b>AKI Warning Stage 2</b> Current creatinine $\geq 2$ x baseline level	<b>Consider clinical review <math>\leq 24</math> hours of e-alert*</b> <b>If AKI confirmed</b> — manage as per table 2	<b>Consider clinical review <math>\leq 6</math> hours of e-alert*</b> <b>Likely Stage 2 AKI</b> — manage as per table 2
<b>AKI Warning Stage 3</b> Current creatinine $\geq 3$ x baseline level (or creatinine $1.5$ x baseline and $>354$ mol/L)	<b>Consider clinical review <math>\leq 6</math> hours of e-alert*</b> <b>If AKI confirmed</b> — consider transfer	<b>Consider Immediate Transfer*</b> <b>Likely Stage 3 AKI</b>

**#Clinical Context**

Why was the blood test taken?

- Were they unwell? – high probability of AKI
- A ‘routine’ test in a stable person – low probability

Check their pulse & blood pressure!  
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 ( $K^+ \geq 6.5$ )<sup>‡</sup>
- Known history of CKD stages 4 & 5 or history of kidney transplant
- New drugs started
- 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

<sup>‡</sup> UK Renal Association Clinical Practice Guidelines (2014) recommends emergency assessment and treatment of severe hyperkalaemia ( $K^+ \geq 6.5$  mmol/l) – click here Refer to main guidance document – Guidance for mental health professionals on the management of acute kidney injury

*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.*

*Adapted from primary care guidelines*



**Table 2: Recognising and Responding to Acute Kidney Injury for Adults in Mental Health facilities**

“Think” Cause	“Think” Medication#	“Think” Fluids	“Think” Review¥
<p>History of acute illness?</p> <ul style="list-style-type: none"> <li>• Think Sepsis – check temperature</li> <li>• Think Hypotension – check pulse and BP</li> </ul> <p>Intrinsic kidney disease? (E.g. vasculitis)</p> <ul style="list-style-type: none"> <li>• Think Urinalysis</li> </ul> <p>Urinary tract obstruction?</p>	<p>Any medication which could <b>exacerbate</b> AKI?</p> <p>Consider withholding:</p> <ul style="list-style-type: none"> <li>• NSAIDs</li> <li>• Diuretics</li> <li>• Antihypertensive medication</li> </ul> <p>Any medication which may <b>accumulate</b> and cause harm during AKI?</p> <p>Any <b>new</b> medication that may <b>cause</b> AKI?(E.g. drug induced tubulo-interstitial nephritis - <b>Lithium</b>)</p>	<p>What is the patient’s volume status?</p> <p>If hypovolemia present:</p> <ul style="list-style-type: none"> <li>• When did patient last pass urine?</li> <li>• Can the patient increase fluid intake?</li> <li>• Is transfer for IV fluid replacement and monitoring required?</li> </ul> <p>Does the patient have and/or need carer support?</p>	<p>Does the patient need transfer to medical unit?</p> <p>If not, when will you review?</p> <p>Have you ensured handover?¥</p>

\*Refer to main guidance document – Guidance for mental health professionals on the management of acute kidney injury

# 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 mental health facilities*

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

*Adapted from Primary Care guidelines*

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## 6. Next steps – discharge, transfer and communication

Local protocols for the drawing of blood, dispatch to the relevant laboratory and the receipt of results should be in place at all Mental Health Trusts. Daily access to phlebotomy is essential and ward doctor handover procedures must include a proactive approach to receiving potentially abnormal blood test results, especially those that have been requested urgently. Such an approach will require computer access to the local electronic results system by all mental health wards for efficient use of NHS resources. There should be a robust system in place where blood results are communicated effectively subject to local arrangements. The clinician who orders the blood test should be responsible for following up the results and acting on any abnormalities found.

Good communication is vital for the safe discharge of patients following an episode of AKI. The discharge letter needs to be clear about who needs to be the lead – GP or psychiatrist. A forward plan should be included showing when further investigations, follow up appointments etc. are planned. Please refer to the Think Kidneys publication: [“Guide for minimum contents in a discharge summary”](#) for guidance around the transfer and discharge of patients.

The timely communication of abnormal results is essential. Mental health facilities may not be familiar with AKI alerts used within the general hospital. Local protocols will need to be agreed between the mental health and local acute hospital to facilitate urgent communication of markedly abnormal results to the requesting clinician or his/her deputy (for example the duty doctor).

Many acute trusts have protocols and care bundles in place for the management of AKI and mental health providers should explore whether this is the case. If not more detailed guidance on the management of AKI, can be found in the Think Kidneys publication: [Recommended minimum requirements of a care bundle for patients with AKI in hospital](#). This provides a single page easy reference document which may be used as the basis for Trusts to develop a local care bundle for clinical use and which allows easy audit or subsequent quality improvement work. It also includes specific guidance on the themes of AKI management and essential components.

Also, please refer to the Think Kidneys publication [Sick Days Guidance Statement](#).

## 7. Conclusion

Patients with mental health problems are at heightened risk of AKI due to the manifestations of mental illness, psychiatric medication, lifestyle factors and physical problems being attributed to the psychiatric condition. There should be clear communication between acute and mental health services at a local level to help to ensure those at risk are identified and AKI is prevented.

## 8. References

Acute Kidney Injury in Pregnancy (Jim and Garovic, 2017)

<https://www.ncbi.nlm.nih.gov/pubmed/28711077>

Acute kidney injury in pregnancy: the thrombotic microangiopathies

<https://www.scribd.com/document/90180362/Acute-Kidney-Injury-in-Pregnancy>

Benjamin D. Robertson, MD; Timothy J. Robertson, MD. Postoperative Delirium After Hip Fracture  
J Bone Joint Surg Am, 2006 Sep; 88 (9): 2060 -2068.

<http://jbjis.org/content/88/9/2060>

Chad Cooper, Jorge E. Bilbao, Sarmad Said, Haider Alkhateeb, Jorge Bizet, Ahmed Elfar, Olinamyr Davalos, Ana T. Meza, German T. Hernandez Serum amyloid A renal amyloidosis in a chronic subcutaneous (“skin popping”) heroin user. Journal of Nephropathology. 2013; 2(3): 196-200

Fong TG, Davis D et al. The interface between delirium and dementia in elderly adults. Lancet Neurol 2015; 14: 823–32. Available at:

[http://www.thelancet.com/journals/laneur/article/PIIS1474-4422\(15\)00101-5/abstract](http://www.thelancet.com/journals/laneur/article/PIIS1474-4422(15)00101-5/abstract)

Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney inter., Suppl. 2012; 2: 1–138.

[http://www.kdigo.org/clinical\\_practice\\_guidelines/pdf/KDIGO%20AKI%20Guideline.pdf](http://www.kdigo.org/clinical_practice_guidelines/pdf/KDIGO%20AKI%20Guideline.pdf)

Kerr M, Bedford M, Matthews B, O’Donoghue D. The economic impact of acute kidney injury in England. Nephrol Dial Transplant (2014) 29: 1362–1368.

<http://www.ncbi.nlm.nih.gov/pubmed/24753459>

Lewington AJ, Cerdá J, Mehta RL. Raising awareness of acute kidney injury: a global perspective of a silent killer. Kidney Int 2013; 84: 457–67.

<http://www.ncbi.nlm.nih.gov/pubmed/23636171>

Martina M. McGrath, Tamara Isakova, Helmut G. Rennke, Ann M. Mottola, Karen A. Laliberte, and John L. Nile, Contaminated Cocaine and Antineutrophil Cytoplasmic Antibody-Associated Disease.

[www.cjasn.org](http://www.cjasn.org) Vol 6 December, 2011

Mehta RL, Cerdá J, Burdmann E, Tonelli M, Garcia-Garcia G, Jha V, Susantitaphong P, Rocco M, Vanholder R, Sukra Sever M, Cruz D, Jaber B, Lameire NH, Lombardi R, Lewington AJ, Feehally J, Finkelstein F, Levin, N, Pannu N, Thomas B, Aronoff-Spencer E, Remuzzi G. International Society of Nephrology’s Oby25 initiative for acute kidney injury (zero preventable deaths by 2025): a human rights case for nephrology. Lancet Mar 2015; 385: 2616–43.

<http://www.sciencedirect.com/science/article/pii/S014067361560126X>

---

National Confidential Enquiry into Patient Outcome and Death (NCEPOD) 2009. Acute Kidney Injury: Adding Insult to Injury

<http://www.ncepod.org.uk/2009aki.htm>

National Institute for Health and Care Excellence (NICE) 2013, Clinical guideline 169, Acute Kidney Injury: prevention, detection and management

<https://www.nice.org.uk/guidance/cg169/resources/acute-kidney-injury-prevention-detection-and-management-35109700165573>

National Institute for Health and Care Excellence (NICE) 2014, Clinical guideline 182, Chronic Kidney Disease in adults: assessment and management.

<https://www.nice.org.uk/guidance/cg182/resources/chronic-kidney-disease-in-adults-assessment-and-management-35109809340613>

Perinatal Mental Health Toolkit

<http://www.rcgp.org.uk/clinical-and-research/toolkits/perinatal-mental-health-toolkit.aspx>

Perinatal

<https://www.england.nhs.uk/mental-health/perinatal/>

Pregnancy outcomes in patients with acute kidney injury during pregnancy: a systematic review and meta-analysis

<https://bmcpregnancychildbirth.biomedcentral.com/articles/10.1186/s12884-017-1402-9>

Pantelias, K and Grapsa, E. Drug abuse and kidney. Hippokratia. 2011; 15(suppl 2): 4-8.

<http://www.hippokratia.gr/images/PDF/15Sup2/831.pdf>

Selby NM, Crowley L, Fluck RJ, McIntyre CW, Monaghan J, Lawson N, Kolhe NV. Use of electronic results reporting to diagnose and monitor AKI in hospitalized patients. Clin J Am Soc Nephrol. 2012 Apr;7(4):533-40. doi: 10.2215/CJN.08970911. Epub 2012 Feb 23.

<http://www.ncbi.nlm.nih.gov/pubmed/22362062>

Selby NM, Anderson J, Peter Bungay, Chesterton LJ and Kolhe NV. Obstructive nephropathy and kidney injury associated with ketamine abuse. NDT Plus. 2008 Oct; 1(5): 310–312.

<http://ckj.oxfordjournals.org/content/1/5/310.full>

AU. Tan. A.H. Cohen, B.S. Levine, Department of Medicine, VAMC West Los Angeles, and Departments of Pathology and Medicine, Cedars-Sinai Medical Center and UCLA School of Medicine, Los Angeles, CA Renal Amyloidosis in a Drug Abuser (J. Am. Soc. Nephrol. 1995; 5:1653-1658)

Think Kidneys Recommended Response Times to AKI Warning Stage Test Results for Adults in Primary Care – Table 1

<https://www.thinkkidneys.nhs.uk/aki/resources/primary-care/recommended-response-times-aki-warning-stage-test-results-adults-primary-care-table-1/>

Think Kidneys Recognising and Responding to AKI in Primary Care – Table 2

<https://www.thinkkidneys.nhs.uk/aki/resources/primary-care/recognising-responding-aki-primary-care-table-2/>

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