Acute Kidney Injury (AKI) In Primary Care

Supporting early detection and consistent management

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

THINK KIDNEYS

THINK KIDNEYS NHS

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

AKI in Primary Care

AKI: Context and focus for primary care

- Definition, staging and association with acute illness
- Implications for patients, the NHS and primary care

AKI: Detection in primary care

- Identifying patients **at risk** of AKI
- Interpreting AKI warning stage test results **within clinical context**

AKI: Management in primary care

- Think Kidneys → Think **Cause**, Think **Drugs**, Think **Fluid Status**, Think **Review**
- When to consider admission and / or renal referral

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AKI: Context and focus for primary care

What is AKI?

- A **sudden reduction in kidney function** (► usually coincides with onset of acute illness)

Why is AKI important?

- Associated with **adverse outcomes for patients** (► consider AKI an **acute illness severity marker**)
- **Common** (► more than $^{1}/_{2}$ million people in England develop AKI every year)

Why has a national "Think Kidneys" campaign been established to raise AKI awareness?

- Public[1] **and** Healthcare Professional[2] awareness of AKI is poor
- UK Study (2009) found **deficiencies in AKI care were common -** including **delayed AKI recognition**[3]

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[1]Ipsos MORI Survey (2014). Understanding what the public know about their kidneys and what they do.
[2]Muniraju et al (2012). Diagnosis and management of acute kidney injury. Clinical Medicine 12(3): 216-221.
[3]NCEPOD Report (2009). Acute Kidney Injury: Adding Insult to Injury.

AKI: Context and focus for primary care

Why are primary care teams being alerted to AKI?

- **Many patients in community are at risk of AKI** (► require prompt review when acutely unwell)
- **Most AKI occurs in community** (► $^{2}/_{3}$ of hospital AKI cases begin pre-hospital admission)[1]

What can primary care teams do to reduce patient harm caused by AKI?

1. **Raise AKI awareness and limit AKI risk** (► AKI often asymptomatic → further delaying AKI detection)
2. **Promote prompt AKI detection** (► consider AKI **early** during acute illness episodes)
3. **Initiate simple interventions early** (► increase chance of recovery / reduce treatment costs)
4. **Perform post-AKI review** (► detect new or progressive CKD +/- restart drugs suspended during AKI)

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1 Selby et al. (2012). Defining the Cause of Death in Hospitalised Patients with AKI. PLoS ONE. 7 (11): e48580

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AKI: Definition, Staging and Association with Acute Illness

AKI definition

- AKI is a **clinical and biochemical syndrome** reflecting **abrupt kidney dysfunction**
- AKI **is not** a primary disease **nor** a “diagnosis”
- AKI **is** a **heterogeneous syndrome** with **various causes** and **variable outcomes**

AKI staging

- **AKI stage is determined by acute changes to serum creatinine and / or urine output**

AKI usually occurs secondary to acute illness (► commonly sepsis)

- **Identifying underlying acute illness causing AKI is key to establishing primary diagnosis**
- **Treating underlying acute illness key to treating most AKI**

AKI Definition (Kidney Disease Improving Global Outcomes, KDIGO criteria[1])

	Serum Creatinine[2]	Urine Output[4]
AKI Definition	Increase in serum creatinine by >26μmol/L **≤ 48 hrs**	Urine volume <0.5 mL/kg/hr **for ≥ 6 hrs**
	Increase in serum creatinine by ≥ 1.5 times baseline[3] **which is known or presumed to have occurred within previous 7 days**	

[2]Note **serum creatinine changes** and **not** *estimated* GFR (*e*GFR) define AKI (as *e*GFR is **not** a reliable indicator of *true* GFR during unsteady clinical states associated with AKI) ► **Drug dosing** should **not** be based upon *e*GFR during AKI episodes.

[2] Note **timescale** of creatinine change is central to AKI definition → if no recent preceding blood test then **incorporate clinical context to determine if creatinine change likely to have occurred during preceding week** (ie. 'acutely').

[3] **'Baseline' creatinine value** should be considered as the **patient's 'usual' creatinine when clinically well** → determine by reviewing patient's previous blood results within clinical context. Assume normal baseline if no previous blood tests.

[4] In practice **urine output criteria** can only be applied to hospitalised patients who are catheterised - **but a reliable history of low or absent urine output should alert the clinician to the possibility of AKI**.

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[1] Kidney Disease Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney International Supplement 2012;2(1):1–138.

AKI Staging (Kidney Disease Improving Global Outcomes, KDIGO criteria[1])

AKI Stage	Serum Creatinine	Urine Output
Stage 1	Increase in serum creatinine by >26μmol/L **≤ 48 hrs** **OR** an increase in serum creatinine by **≥ 1.5** x baseline[2]	urine output <0.5mL/kg/hr for 6-12hrs
Stage 2	Increase in serum creatinine by **≥ 2** x baseline[2]	urine output <0.5mL/kg/h for ≥12hrs
Stage 3	Increase in serum creatinine by **≥ 3** x baseline[2] **OR** an increase in serum creatinine by ≥1.5 baseline **to > 354 μmol/L**	urine output <0.3mL/kg/h for ≥24hrs **OR** anuria for ≥12 h

[2] **When creatinine change** is known or presumed to have occurred **within previous 7 days**

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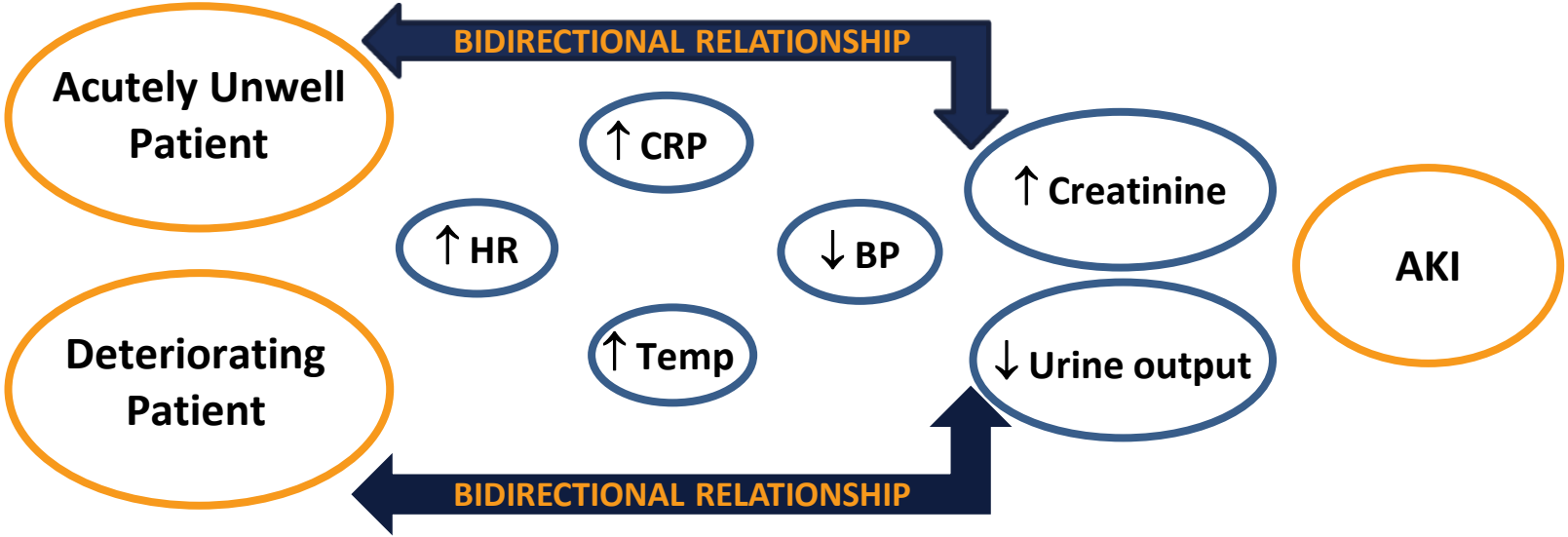
[1] Kidney Disease Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney International Supplement 2012;2(1):1–138.

AKI as a patient safety barometer associated with acute illness



Prompt recognition and good management of AKI

requires and often reflects

Prompt recognition and good management of acutely unwell patients

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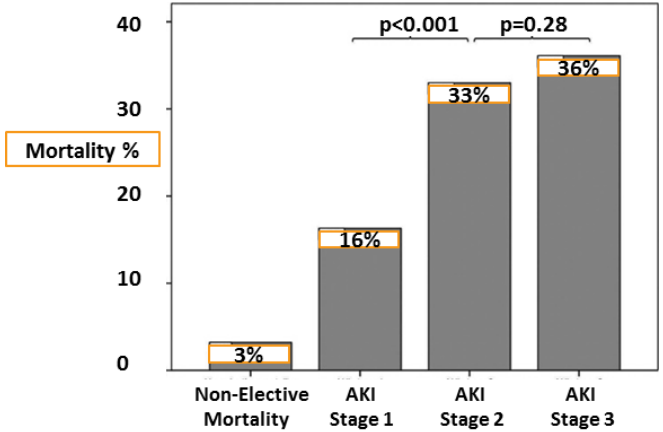
AKI Patient Implications: Independently associated with adverse acute and chronic outcomes

- **AKI associated with increased patient mortality**
- Odds of **death** ∞ AKI **severity** in UK Study[1]



- Other studies show association with **death persists** if
  - ➢ Acute and chronic **co-morbidities accounted** for[2]
  - ➢ Patients followed up **post discharge / longer term**[3]

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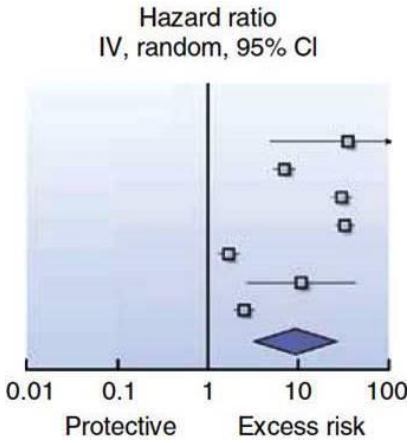
[1] Selby N. et al. (2012). Use of Electronic Results Reporting to Diagnose and Monitor AKI in Hospitalized Patients. CJASN. 7:533-540.
[2] Chertow et al. (2005). Acute Kidney Injury, Mortality, Length of Stay, and Costs in Hospitalized Patients. J Am Soc Nephrol 16: 3365–3370.
[3] Coca et al. (2012). Chronic kidney disease after acute kidney injury: a systematic review and meta-analysis. Kidney Int. 81, 442–448.

AKI Patient Implications: Independently associated with adverse acute and chronic outcomes

- **AKI associated with increased patient morbidity**
- Meta-analysis shows AKI is **risk factor for CKD** [1]



Pooled hazard adjusted ratios for CKD post-AKI[1]

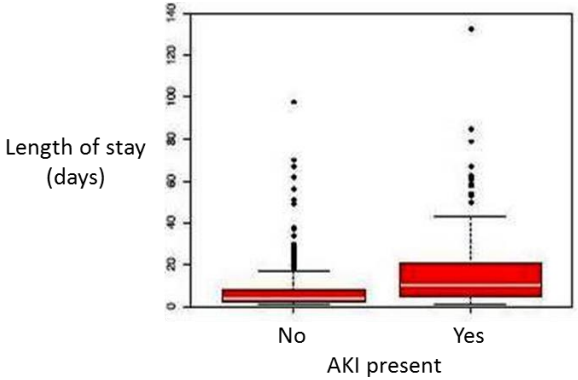
- CKD also associated with ↑ risk of **end-stage renal failure**[1], **cardiovascular disease** and **death**[2]

[1] Coca et al. (2012). Chronic kidney disease after acute kidney injury: a systematic review and meta-analysis. Kidney Int. 81, 442–448.

[2] Chronic Kidney disease Consortium (2010). Association of eGFR and albuminuria with all-cause & cardiovascular mortality. Lancet 375: 2073-2081.

AKI NHS Implications: Significant additional impact on Healthcare Resources

- AKI **commonly complicates acute illness** and hospital admissions
- AKI associated with **25.4% of unselected emergency admissions** to a large UK acute hospital Trust[1]
- AKI increases duration and complexity of acute illness ► AKI increases **length of hospital stay (LOS)**[1]:



  - ➢ AKI group LOS **almost 3x higher** than non AKI group (10 vs 4 days)[1]
  - ➢ AKI group more often **required critical care beds** (8.1% vs 1.7%)[1]

- AKI associated with **complex treatments** such as dialysis (► may be required permanently)
- **AKI significantly increases healthcare costs as a consequence of these complications**

[1] Challiner et al. (2014). Incidence and consequence of AKI in unselected emergency admissions to a large acute UK hospital trust. BMC Nephrology. 15:84

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AKI aspirations for primary care teams

- Primary care teams **well located** to:-

1. **Raise AKI awareness** and **limit AKI risk** in “at risk” patient groups
2. **Detect AKI** and deliver **simple interventions early** (► to limit AKI severity and duration)
3. **Undertake post AKI review** to
   a. **Detect new or worsening Chronic Kidney Disease** post AKI
   b. **Restart drugs suspended during AKI** (► especially if **prognostic** benefit)
   c. **Limit risk of further AKI** (► patient / carer advice where appropriate)

Ipsos MORI survey, July 2014

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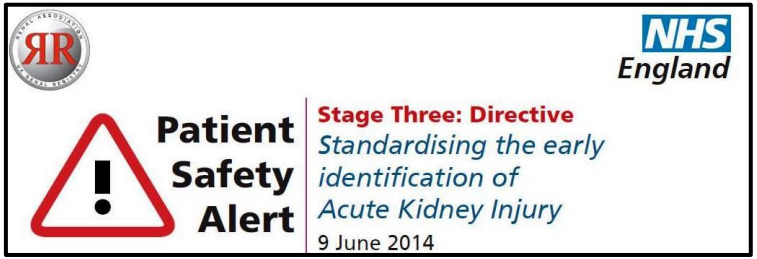
AKI and primary care: Prompt detection and management

- Two **National AKI Patient Safety Alerts** aim to promote **AKI care in the community**



- Mandates **national automated AKI detection system** to generate **AKI warning alerts** alongside blood tests
- Pilot studies indicate Full Time Equivalent GP expects about **one AKI e-alert every 1-2 months** (>$^{1}/_{2}$ likely AKI Stage 1)

- **Health care staff should be signposted to Think Kidneys AKI resources** (hyperlinks to relevant resources at foot of slides)
- Resources include AKI guidelines **to support appropriate response to AKI warning alerts by Primary Care Teams**

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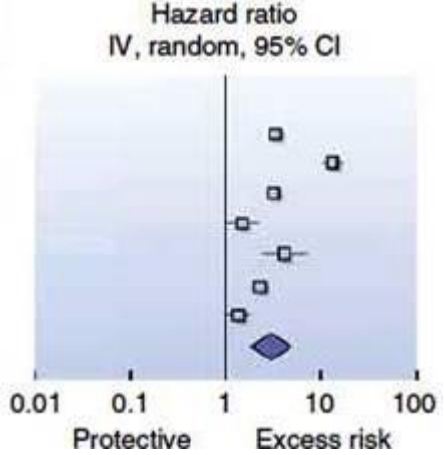
- **THINK KIDNEYS Resource: Full Primary Care AKI Guidelines** LINK

AKI and Primary Care: Post AKI review

- AKI also associated with **adverse long term outcomes**

Pooled hazard adjusted ratios for End-Stage Renal Failure (ESRF) post-AKI[1]



➢ **Renal Health**: AKI is associated with **new** or **worsening CKD, including ESRF**

► especially if **severe** or **multi-hit** AKI in

1. **Elderly patients**
2. Patients with **diabetes**
3. Patients with **pre-existing CKD**

➢ **General Health:** Drugs with **prognostic** long term benefit (eg ACE-I for heart failure) may be suspended in **clinical context** of acute illness and AKI

► **long term prognostic benefit** of such drugs **lost** if not restarted post AKI

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[1]Coca et al. (2012). Chronic kidney disease after acute kidney injury: a systematic review and meta-analysis. Kidney Int. 81, 442–448.

AKI and Primary Care: Post AKI review

- **Post AKI** reviews consider**:-**

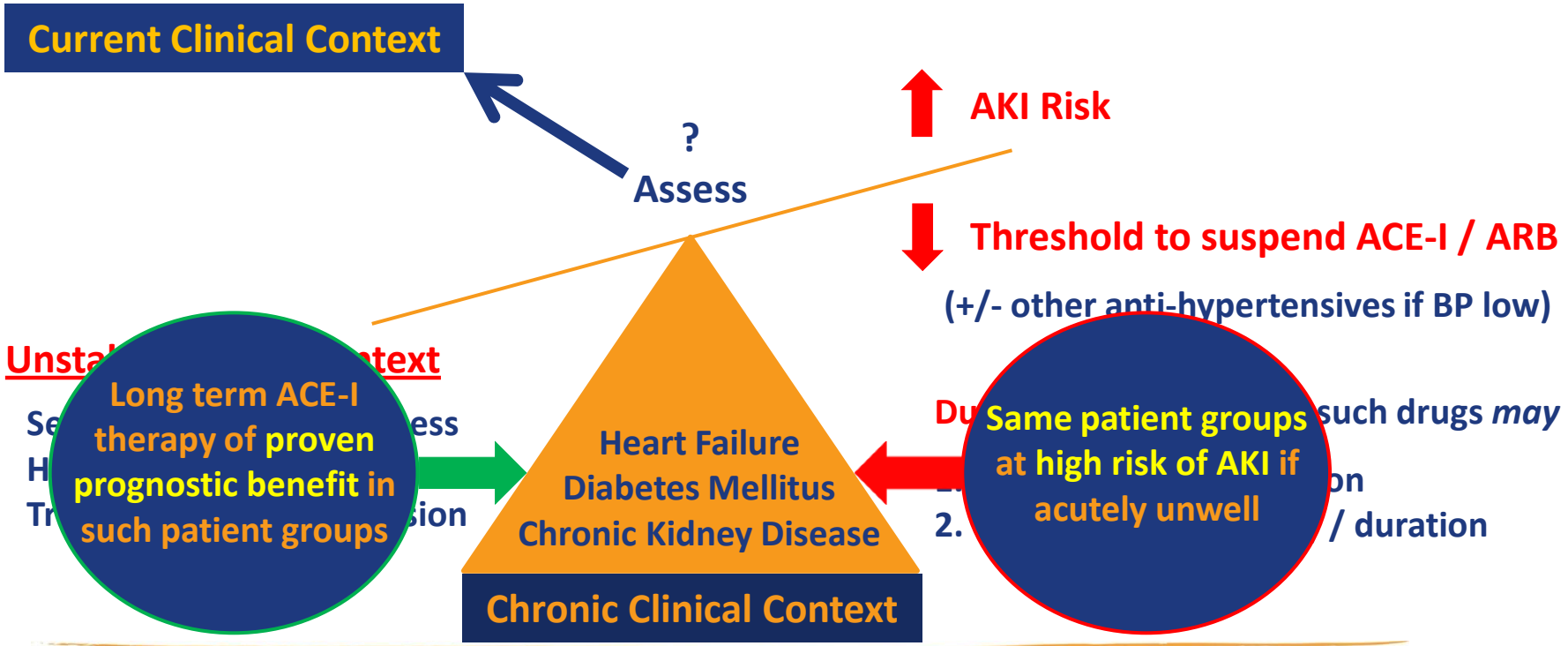
1. **Monitoring for new or worsening CKD especially if creatinine has not returned to baseline or other CKD risk factors.** NICE guidelines advocate monitoring renal function for 3 years post AKI.
2. **Restarting drugs** with **prognostic** benefit **once clinical context improves and stabilises / acute illness resolved** ► unless compelling contraindication to drug remains (or ongoing AKI risk > drug benefit).
3. **Onward drug adjustments tailored** to chronic disease **and** acute clinical context (►see 2 next slides)
4. **Patient / carer advice** to limit further AKI episodes (►utilise AKI Patient Leaflets, links below)

   ➢ Encourage **early** medical contact to assess blood pressure, renal function and medications if

   a. Acutely unwell
   b. Unable to maintain good fluid intake
   c. Reduced urine output noted

- **THINK KIDNEYS Resources: Restarting drugs LINK, Sick Day Guidance for Drugs LINK, Patient Advice Leaflets for those who have sustained AKI LINK and those at persistent risk of AKI LINK**

Clinical Context and ACE-Inhibitors



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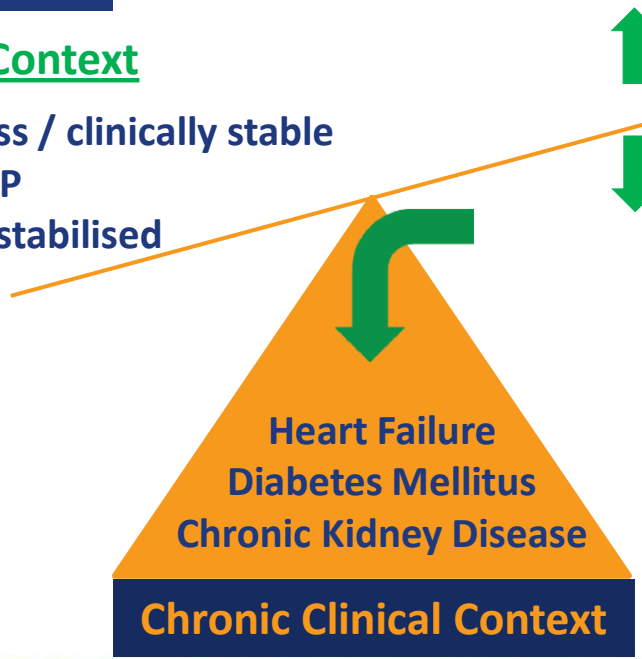
Clinical Context and ACE-Inhibitors

Current Clinical Context

Stable Clinical Context

- Recovery from acute illness / clinically stable
- Restoration of volume / BP
- Post AKI / Renal function stabilised



Prognostic benefit of ACE-I / ARB

Threshold to resume ACE-I / ARB

- Try to restart drug if strong indication
- Review patient & bloods ≤ 1-2 weeks
- See resource below but generally:
  - Initial creatinine rise ≤ 30% often OK
  - If creatinine rise ≥ 30% / progressive:

1. Suspend drug and review patient
2. Consider renal opinion
3. If indication for ACE-I is heart failure
   ►consider cardiology opinion

THINK KIDNEYS RESOURCE: ACE-Inhibitor and diuretic use in Primary Care LINK

ACE-I / ARB initiation or dose up-titration in Primary Care

Initial Assessment

1. Ensure clinical context is stable ► consider patient 'sick day' advice.
2. Use Immediate pre-treatment creatinine as baseline creatinine.
3. Arrange repeat blood tests within 1-2 weeks.

Serum creatinine rise > 15% but < 30% from baseline

1. Continue drug but arrange to re-assess clinical status, BP and bloods within 1-2 weeks.
2. Consider ↓ other BP-lowering drugs if sBP <120, including diuretics if evidence of hypovolaemia.
3. Continue drug if creatinine stabilises on repeat testing (< 30% above pre-treatment baseline).

Continuing ACE-I in chronic heart failure sometimes may be overall beneficial even if creatinine rise > 30%

Serum creatinine rise > 30% from baseline

1. Promptly re-assess clinical, fluid and BP status.
2. Consider ↓ other BP-lowering drugs if sBP <120, including diuretics if evidence of hypovolaemia.
3. Repeat bloods ≤ 5-7 days ► if renal function remains > 30% despite above measures:
    1. Stop drug and consider local renal opinion
    2. If indication for drug is heart failure also obtain advice from local heart failure team

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THINK KIDNEYS RESOURCE: ACE-Inhibitor and diuretic use in Primary Care LINK

THINK KIDNEYS RESOURCE: Patient Sick Day Guidance for Drugs LINK

AKI in primary care

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AKI in Primary Care: Patients at risk of AKI in Community

Situation Specific - Exposure

- **Any acutely unwell patient** is at **acute risk of AKI**
- ↑ AKI vigilance in **clinically unstable**, especially if

- Hypovolaemia, dehydration, reduced oral intake
- Absolute hypotension (sBP < 90 mmHg)
- Relative hypotension (↓40 mmHg from baseline BP)
- Sepsis
- Recent operation or iodinated contrast scan
- NSAIDs, BP-lowering ± diuretic drug use ≤ 1week

Patient Specific - Susceptibility

- Many patients **remain at persistent ↑ AKI risk**
- ↑ AKI vigilance in **'at risk' communities**

- Older age patients (especially with polypharmacy)
- Co-morbidities (eg. CKD, DM & Heart Failure)
- Psycho-social setting (eg. In care home, ↓ mobility / dementia → unable to self regulate fluid intake)

- Consider **risk reduction strategies** in such groups
- **promote self care** (or carers) to **avoid dehydration**
- similar advice / resources as for "Post AKI review"

THINK KIDNEYS Resource: **Advice on communities at risk of AKI** LINK

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Interpreting AKI warning stage test results in Primary Care: Clinical context is Key

- A national **automated AKI detection system** aims to **improve early recognition** of AKI
- Presentation of **AKI warning alerts** depends upon Pathology System used, examples below:

Sodium	*125*	*mmol/L*	*(133 to 146)*
Potassium	4.2	mmol/L	(3.5 to 5.3)
Bicarbonate	28	mmol/L	(22 to 29)
Chloride	87	mmol/L	(95 to 108)
Urea	25.6	mmol/L	(2.5 to 7.8)
Creatinine	611	umol/L	(50 to 120)
eGFR/ 1.73M^2	6	ml/min	
AKI Warning stage	3		

Sample BC4806218 (BLOOD WHOLE) Collected 06 May 2015 06:50 Received 06 May 2015 11:13

Urea & Electrolytes

Sodium		145	mmol/L	133 - 146
Potassium		3.7	mmol/L	3.5 - 5.3
Bicarbonate		26	mmol/L	22 - 29
Urea	*	22.8	mmol/L	2.5 - 7.8
Creatinine	*	182	umol/L	50 - 120
eGFR/ 1.73M^2	*	31	ml/min	
Chloride		107	mmol/L	95 - 108
AKI Warning stage	*	2		

AKI Stage 2 Alert - Review Nephrotoxic Medication.
Refer to Trust AKI guidance under 'Policies, procedures and guidelines'.

- Alert system relies upon computerised interpretation of blood results **in isolation from clinical context**
- AKI is **not merely** a 'biochemical finding' ►do **not rely upon alert system** to detect all AKI cases
- Always review current and previous blood results **within clinical context** in order to **validate** AKI alert

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Interpreting AKI warning stage test results in Primary Care: Clinical context is Key

- A **positive** AKI alert simply alerts clinician to **possibility** of AKI ► **false positives** can occur (see table below)
- A **negative** AKI alert **does not always rule out** AKI ► **false negatives** can occur (see table below)

False positive examples	False negatives example
Recent Pregnancy: Creatinine falls during pregnancy ►**creatinine rise expected** / normal post delivery.	**Previous AKI within last year:** Algorithm may calculate **spuriously high baseline creatinine** for patient.
Drugs (eg. trimethoprim) inhibiting tubular creatinine secretion: Can cause creatinine rise **whilst GFR stable**.	
Recent IV fluid: ►**spuriously low baseline creatinine.**	

- **If alert unexpected and stable clinical context** ►consider repeating bloods within 48-72hrs to determine whether any creatinine changes are truly dynamic (AKI) or relatively stable / false positive.
- **If no alert issued though high clinical suspicion of AKI / acute illness** ► it especially important for clinician to review current and previous blood results **before ruling out** AKI.

- **THINK KIDNEYS Resource: Further guidance on page 7 of Primary Care AKI Guidelines** LINK

Interpreting AKI warning stage test results in Primary Care: Infrastructure & Process

- Any automated detection system is **only effective** if leads to **timely** and **appropriate intervention**

- Detection system does **not issue interruptive alert** and **in isolation** does **not ensure timely intervention**
- **Correct effective utilisation** of automated AKI system thus requires clinicians:-

1. **Actively review alerts within clinical context** in **timely fashion**
   - Practices should ensure clinicians reviewing alerts know **reason why** blood tests were taken
   - **Particular challenge** if results reviewed by '**out of hours**' services
2. **Respond to alerts and clinical context** with **timely intervention**
   - Think Kidneys Primary Care Resource includes **recommended alert response times** (►next slide)

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- **THINK KIDNEYS Resource: Full Primary Care AKI Guidelines** LINK

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

Also consider if other features present to prompt earlier review / hospital admission

Confirm **or** refute automated AKI Test Result by

LOW Pre-test Probability of AKI

HIGH Pre-test Probability of AKI

1. What was clinical context when the blood test was taken?

Stable Clinical Context	Unstable Clinical Context
Chronic disease / drug monitoring	Assessment of acute illness

(Assume unstable clinical context if clinical context unknown)

2. Are risk factors for AKI present?

Chronic AKI Risk Factors	Acute AKI Risk Factors
Chronic Kidney Disease	Acute illness
Chronic Heart Failure / Liver Disease	New drug started
Diabetes Mellitus	Poor oral fluid intake
Cognitive / Neurological Disease	Recent previous AKI

3. Are factors present to suggest acute kidney dysfunction?

Clinical Features	Biochemical Features
Reduced urine output	Creatinine rise from recent baseline
Patient unwell	Further creatinine rise on repeat test

4. Are additional factors present to prompt early review?

Patient Factors	Clinical / Biochemical factors
Stage 4 or 5 CKD	Patient unwell
Kidney transplant recipient	Serum K+ ≥ 6.0 mmol/l
Frail / co-morbidities	Likely intrinsic kidney disease
Urinary tract obstruction	

Providing access to salient clinical data when taking blood tests via laboratory forms, medical records or handover will support timely appropriate response → especially when alert reviewed by out of hours GP unfamiliar with patient

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Common causes of AKI in Primary Care: Pre-renal and Post Renal AKI

AKI: Pre-renal Renal Insults

- **Kidney function** requires **adequate renal perfusion**
- **80-90% of all AKI** due to **acute illness** causing a significant / sustained reduction in renal perfusion:-
  - **Vasodilatation** and **hypotension** due to **sepsis**
  - **ECV loss** due to **diarrhoea & vomiting**, or **bleeding**
  - **Hypotension** due to **acute heart failure**
- Some **drugs** may magnify AKI during such states

AKI: Post-renal Renal Insults

- **Kidney function** requires **adequate urine drainage**
- ↓ threshold for early renal USS in patients reporting ↓ urine output, especially if unwell +/- history of :-
  - Males with enlarged prostates
  - Renal calculi
  - Pelvic or abdominal masses
- **Delays** relieving obstruction may magnify AKI

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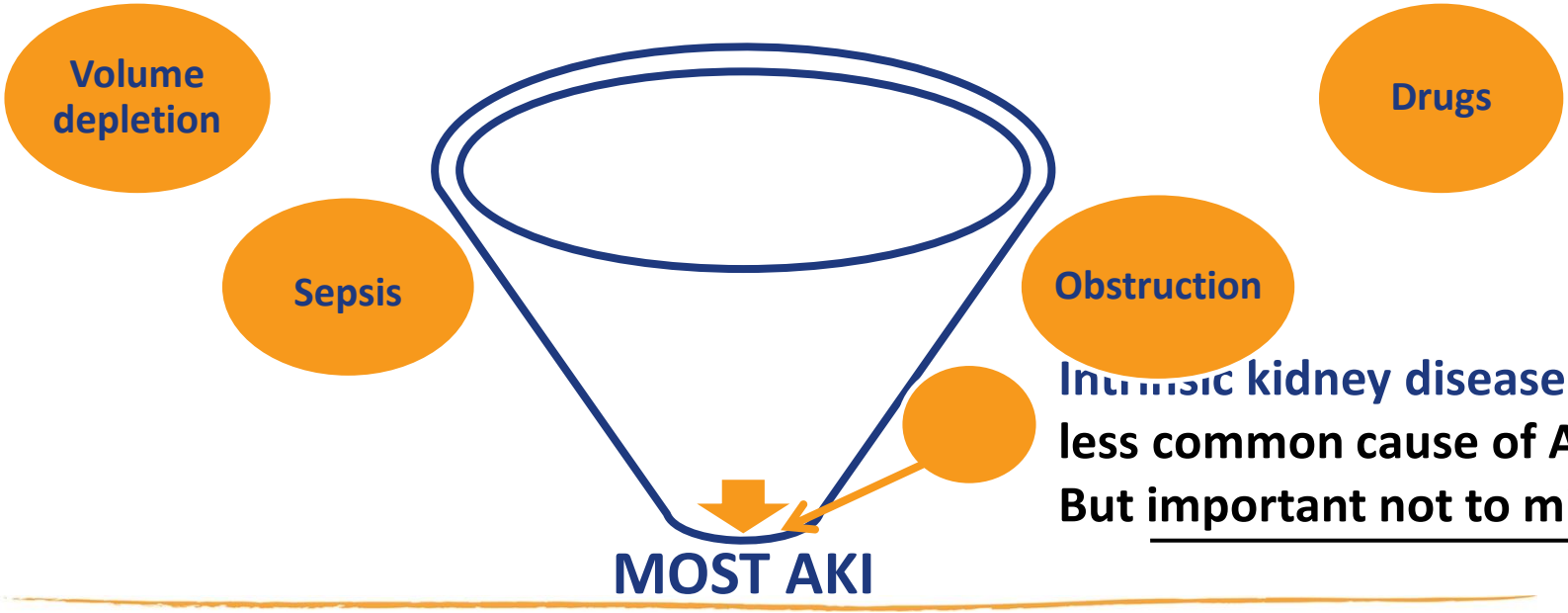
- THINK KIDNEYS Resource: **AKI & Drugs Guideline** LINK

AKI Causes: Pre-renal and Post-renal account for majority of AKI

Addressing these 4 common drivers of AKI will address majority (> 90%) of AKI



Intrinsic kidney disease is a less common cause of AKI…
But important not to miss

AKI Causes: When to suspect Intrinsic Renal Disease

- **Intrinsic Renal Disease** is a less common cause of **AKI** (≤ 5%)
- **Important not to miss** ► may benefit from **early** renal referral
- Group of disorders reflecting toxin and / or immune-mediated kidney damage
- **Urine dip often key to diagnosis** ► protein ± blood on dip should **raise suspicion** of intrinsic disease
- Myeloma and Tubulo-Interstitial Nephritis (TIN) exceptions ► can be present with normal urine dip
- Especially **consider intrinsic renal disease** as cause of AKI if:-

1. **No common / obvious cause for AKI** (ie. sepsis, volume depletion, drugs or obstruction) **and / or**
2. **Urine dip +ve for protein +/- blood and / or**
3. **Clinical features of nephritis or systemic disease causing AKI present** (► see next slide)

AKI Causes: When to suspect Intrinsic Renal Disease

Clinical Clues and Screening for intrinsic renal disease in AKI		
Clinical context	**Potential diagnosis**	**Example screening tests**
Rash +/- arthralgia	SLE, vasculitis, HSP, cryoglobulinaemia	ANA, ANCA, ↓ complement
Haemoptysis	Anti-GBM disease, vasculitis	Anti-GBM Ab, ANCA
Crush injury / long lie	Rhabdomyolysis	Creatine kinase
Haemolysis & ↓ platelets	Thrombotic microangiopathy (TTP, HUS)	Blood film, LDH
Severe hypertension	Malignant hypertension	Fundoscopy
Vascular intervention	Cholesterol embolisation	↓ Complement, ↑eosinophils
Recent chemotherapy	Tumour lysis syndrome	Uric acid level
↑ Ca^{2+} +/- bone pain	Multiple myeloma	Myeloma screen
Recently started new drug	Tubulo-Interstitial Nephritis (TIN)	↑Eosinophils (not always)

THINK KIDNEYS Resource:

AKI & Drugs Guidelines LINK

Table 2: Management of AKI in Primary Care

"Think" Cause	"Think" Medication	"Think" Fluids	"Think" Review
Review patient within clinical context **History of acute Illness?** ➢ Think Sepsis ➢ Think Hypotension **Positive Urinalysis?** UTI symptoms absent? Multisystem symptoms? ➢ Think intrinsic disease **Urinary Tract Symptoms?** Palpable bladder? Consider urgent USS ➢ Think obstruction	**Review drugs within clinical context** **Could drug be driving AKI?** ➢ Think suspend drug? eg • NSAIDs • BP drugs if low BP • Diuretics if dehydrated **Could drug accumulate?** ➢ Think change dose? eg • Diabetic medication • Digoxin • Opiates / gabapentin **Could new drug cause AKI?** ➢ Think causes of TIN ? eg • NSAIDs, antibiotics • Proton pump inhibitors	**Tailor fluid advice to clinical context** **If hypovolemic consider if** ➢ Urine output +/- BP low? ➢ Can patient drink more? ➢ Are IV fluids required? **If fluid overloaded consider** ➢ If risk of lung oedema? ➢ Is patient passing urine? ➢ Are diuretics indicated?	**Time next review to clinical & chemical context** **Consider early review (< 12 hours) +/- admission if** ➢ Patient unwell ➢ Stage 3 AKI ➢ K^+ >6.5 (not haemolysed) ➢ Risk of lung oedema **Consider repeating bloods:-** ≤ 72 hrs for stage 1 AKI ≤ 24 hrs for stage 2 AKI ≤ 12 hrs for stage 3 AKI **and** **Ensure clinical context for repeat bloods handed over to those reviewing results**

Contact Think Kidneys

How to find out more

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