

Acute kidney injury for the non specialist

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Outline

- 3 brief clinical cases
- Overview of acute kidney injury

Case 1

History

- 62 year old lady, type 2 diabetes
- Unwell for 2 weeks
- Nausea, poor appetite, shivery
- Poor urine output for 2 days
- DH - metformin, losartan, atenolol, furosemide, insulin, simvastatin
- BP 100/50, obese

Investigations

- Hb 12.1, WCC 14, Plt 366
- Na 130, K 6.2, Urea 30.4, Creat 600
- LFTs normal, Ca 2.32
- CRP 21, glucose 4.2

- How would you assess her further?

Key assessments

- Urine dip - 1+ blood, no protein, nitrites and leukocytes
- Fluid status - JVP not visible, dry+++
- Urine output - minimal, 5-10mls/hour
- Renal ultrasound - normal

What's the diagnosis?

- Acute on chronic renal failure
- Pre renal / Acute tubular injury
- Urinary tract infection
- Dehydration
- Drugs - ACE / diuretics

What would you do next?

- Admit to Medical Admissions Unit
- Fluids to correct hypotension
- Frequent review of fluid status
- Monitor urine output
- Antibiotics for UTI
- Review / stop drugs

- Renal referral if not improving

How could this admission be avoided?

- Earlier consultation
- Advise to stop metformin, ACE/ARB and diuretics during intercurrent illness
- Frequent review at home

Case 2

History at surgery

- 59 year old man
- Previously fit and well
- No regular medication
- Swollen feet following a flu-like illness 4 weeks previously

- BP 189/92, T 37, P 112
- No other abnormal examination findings

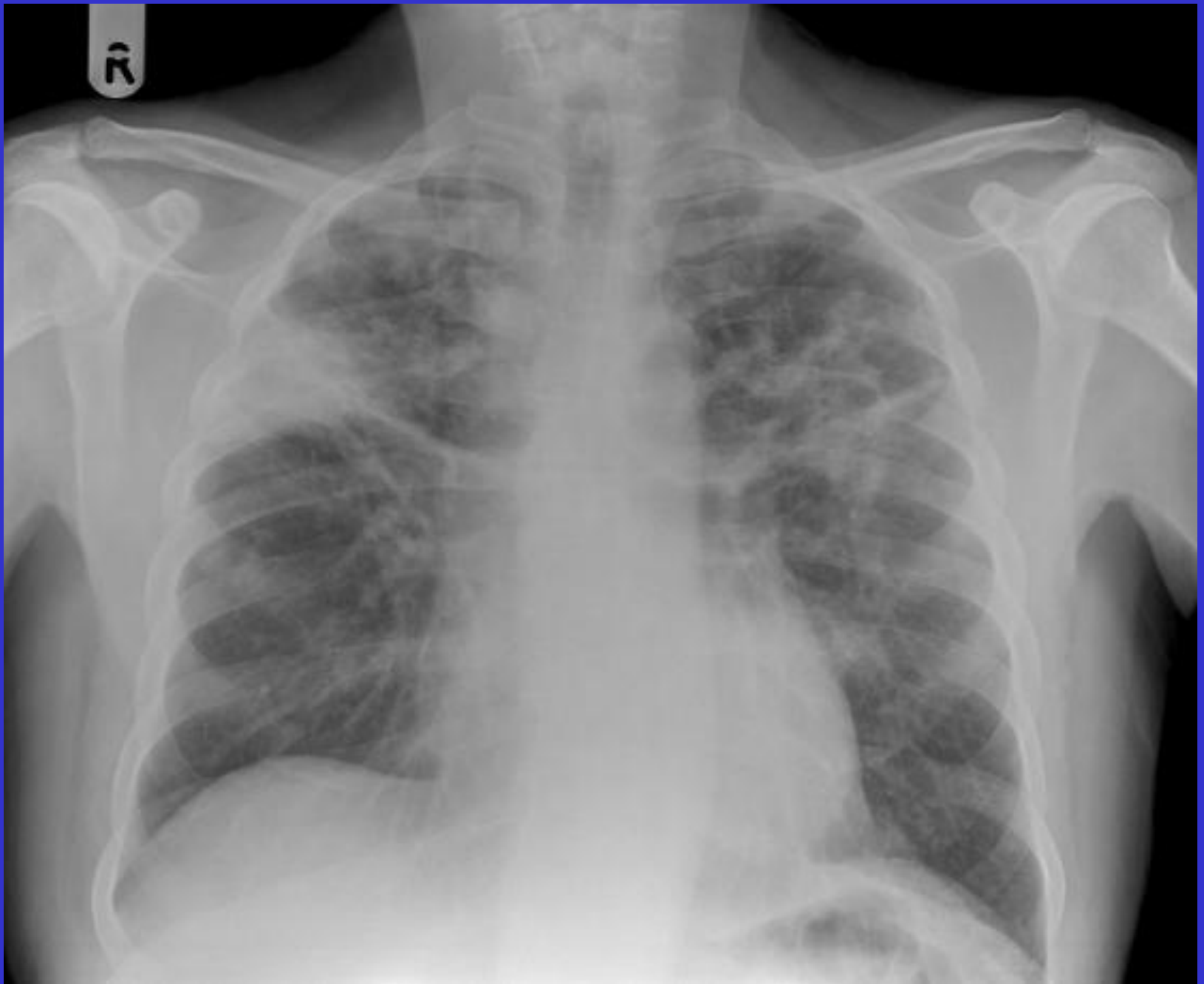
Investigations

- Hb 7.1, WCC 10.1, Platelets 476
- CRP 198, ESR 119
- Albumin 24, LFTs normal
- Na 133, K 5.6, Ur 22, Creat 314
- eGFR 18

- What vital piece of information is missing?

Key assessments

- Urine dip - 3+ blood, 3+ protein
- Fluid status - euvolaemic
- Urine output - normal
- Renal ultrasound - normal



What's the diagnosis?

- ANCA positive glomerulonephritis
- Wegeners granulomatosis
- Lung and renal involvement

Further investigations

- Positive cANCA and PR3
- Renal biopsy
 - crescentic glomerulonephritis
- Commenced cyclophosphamide and prednisolone
- After 2 months creatinine 130 and CXR improved



Case 3

History

- 52 year old lady
- Previously fit and well, no medication
- Generally unwell over 2 months
- Increasing tiredness
- 6 week history of PV bleeding
- BP 200/106, mild oedema

Investigations

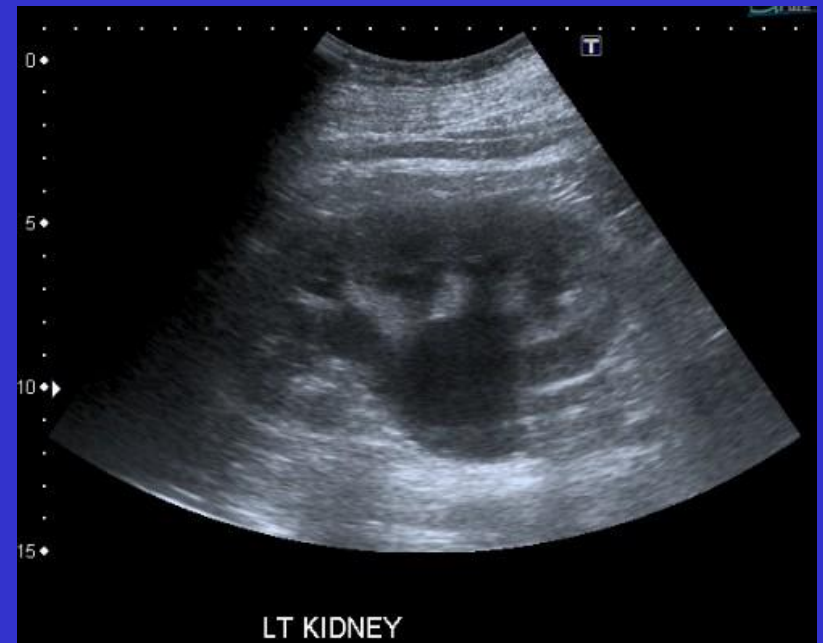
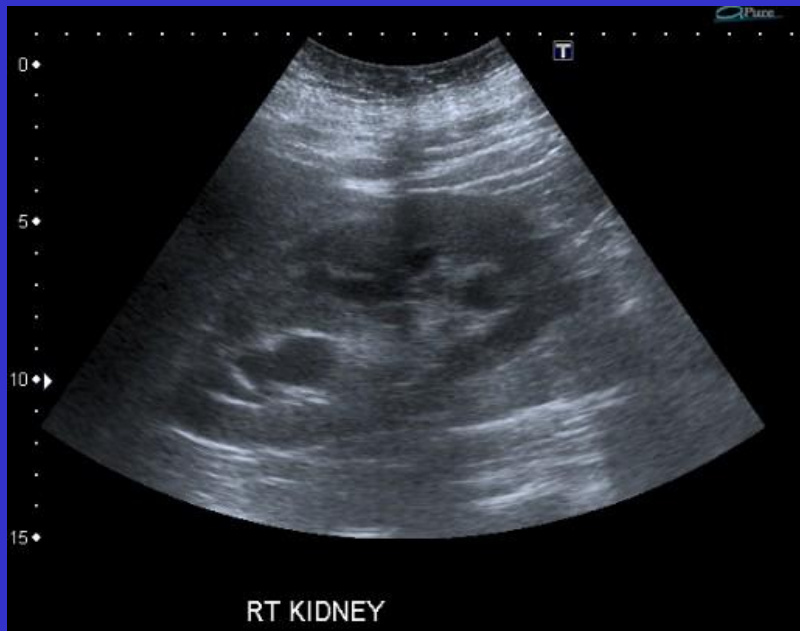
- Hb 6.4, WCC 8.9, Plt 355
- Na 134, K 5.9, Urea 31.3, Creat 1774
- CRP 80
- Alb 30
- Ca 2.26, PO4 2.71

- What would you do next?

Key assessments

- Urine dip - 2+ blood, no protein
- Fluid status - euvolaemic
- Urine output - normal
- Renal ultrasound

Ultrasound



Bilateral hydronephrosis

What's the diagnosis?

- Bilateral hydronephrosis
- Dilated ureters
- Likely gynaecological
 - Cervical or uterine cancer
- Bilateral nephrostomies and urology / gynaecology review

Overview of acute kidney injury in medical patients

Why is it important?

- Common - AKI complicates
 - 5% of hospital admissions
 - 30% of admissions to critical care
- High mortality rate
- Increases length of stay / cost
- Often iatrogenic and may be preventable
- Patients may need long term dialysis

What is Acute Kidney Injury?

Acute, usually reversible decline in GFR

- Abrupt (within 48 hours)
- Increase in serum creatinine of either $>25 \mu\text{mol/l}$ or $>50\%$ increase from baseline
- Reduction in urine output (oliguria $<0.5\text{ml/kg/hr} > 6$ hours)

Not necessarily from normal baseline

Relationship between GFR and serum creatinine in AKI

Notis905

Run Date: 13-SEP-2007 18:33

Nottingham University Hospitals 

Run By: SROE2

Results Report

NHS Trust
City Hospital Campus

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CLINICAL CHEMISTRY - GLOMERULAR FILTRATION RATE

Patient Id NB02556

Date Reported	Status	Ward	Consultant	Taken At Campus
12-SEP-2007 18:34	Final	BRAM	1SR	City

Taken: 12-SEP-2007 00:00 Laboratory No: 00070130504

GFR (MDRD CALCULATION) F	17
GFR (MDRD CALCULATION) F	GFR calculated by MDRD formula (mL/min/1.73m ²) -
GFR (MDRD CALCULATION) F	for Afro-Caribbeans multiply by 1.212. See UK CKD
GFR (MDRD CALCULATION) F	eGuide on the Renal Association website. <u>Invalid</u>
GFR (MDRD CALCULATION) F	<u>in acute renal failure</u> , dialysis and pregnancy.
GFR (MDRD CALCULATION) F	Please use Cockcroft-Gault estimate of creatinine
GFR (MDRD CALCULATION) F	clearance for drug dosing (see NUH Antibiotic
GFR (MDRD CALCULATION) F	Guide Homepage).



R.I.F.L.E

GFR

Urine

RISK

↑ [creat] × 1.5

<0.5 ml/kg/hr × 6 hrs

INJURY

↑ [creat] × 2

<0.5 ml/kg/hr × 12 hrs

FAILURE

↑ [creat] × 3

<0.3 ml/kg/hr × 24 hrs

[creat] > 350 $\mu\text{mol/l}$ Anuria × 12 hrs
rise >50 $\mu\text{mol/l}$

LOSS

Complete loss of function > 4 weeks

ERF

Established kidney disease > 13 weeks

Most important causes of community-acquired ARF

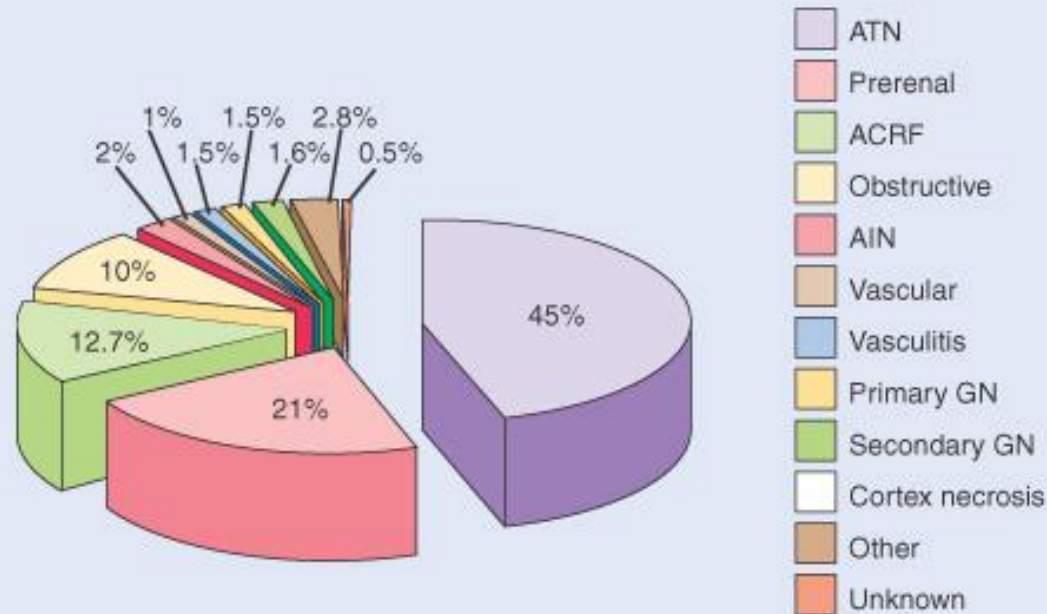


Figure 64.2 Most important causes of community-acquired acute renal failure (ARF). ACRF, acute-on-chronic renal failure; AIN, acute interstitial nephritis; ATN, acute tubular necrosis; GN, glomerulonephritis.

(Redrawn from Liano F, Pascual J: Epidemiology of acute renal failure: A prospective, multicenter, community-based study. Madrid Acute Renal Failure Study Group. *Kidney Int* 1996; 50:811–818.)

Incidence ARF 209 per million population

Outcome in patients with AKI requiring dialysis

	ARF	A/CRF
Died within 10 days of RRT	30%	20%
Died within 90 days of RRT	50%	43%
On dialysis at 90 days	13%	53%

Scottish study of ARF treated with RRT. Prescott et al, NDT (2007) 22: 2513

Causes of AKI

Acute Renal Failure

Pre-Renal

Renal

Post-renal

ATN

Vascular

TIN

Glomerular

Ischaemia

Toxins

Pigments

50%

35%

10%

5%

Clinical evaluation of ARF

- Is this acute, acute-on-chronic or chronic?
- Is there evidence of hypovolaemia or reduced arterial blood volume?
- Has there been a major vascular occlusion?
- Is there evidence of parenchymal renal disease (not ATN)?
- Is there renal tract obstruction?

Distinguishing between AKI and CKD

- Old results
- Renal bone disease
 - ↓Ca ↑PO₄ occur in both ARF and CRF
 - Radiological changes &/or ↑↑PTH suggest CRF
- Renal size on ultrasound
 - >10cm Bipolar diameter cortical thickness >1 cm suggest ARF
 - Except in DM, Amyloid, Myeloma, Cystic disease
- Anaemia
 - Can occur very rapidly after the onset of ARF

Pre-Renal

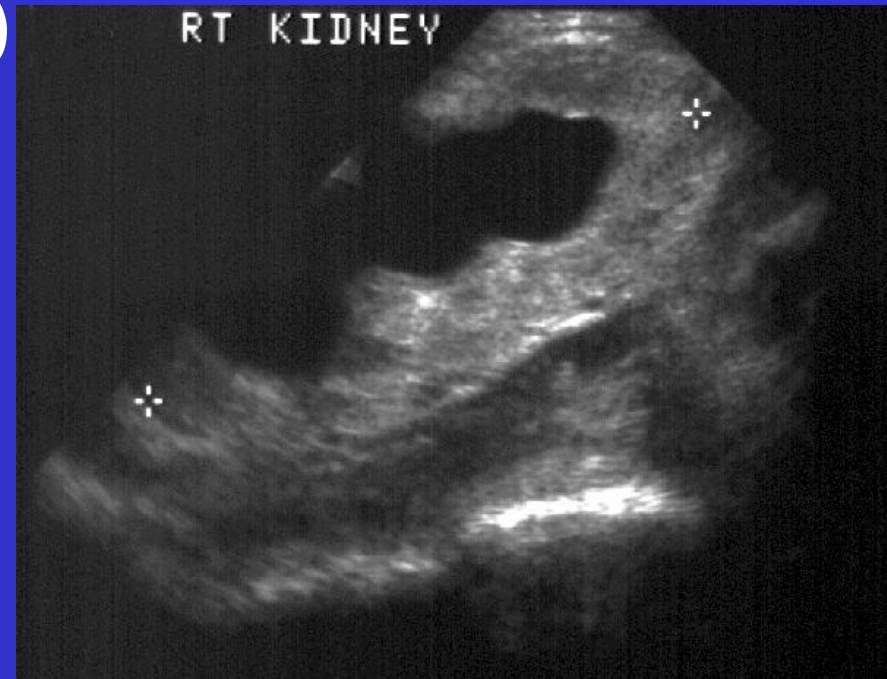
- Intravascular volume depletion
 - Haemorrhage
 - Fluid losses
 - GI tract: Fistulas, pancreatitis, bowel obstruction, D&V
 - Urine: Diabetes, diuretic use
 - Shock: burns, trauma
- Reduced cardiac output
 - Acute MI, arrhythmias, LVF
- Severe hypotension
 - Sepsis, drugs

Causes of Intrinsic AKI

- Acute tubular nephritis (ATN)
 - Ischaemic (extreme end of pre-renal failure, pancreatitis, burns, sepsis)
 - Exogenous toxins (radiocontrast, nephrotoxic drugs)
 - Endogenous toxins (rhabdomyolysis, massive haemolysis, tumour lysis syndrome)
- Acute Glomerulonephritis / RPGN (5%)
- Interstitial nephritis (10%)
- Vascular
 - Cholesterol emboli, renal artery compromise
- Hepatorenal syndrome

Post renal / obstruction

- Anuria is uncommon (consider vascular event)
- Can be obstructed and not dilated (tumours, RPF)
- USS can miss obstruction if done too early or when patient volume deplete



Key assessments

- Assess fluid status - pre-renal
 - JVP
 - Blood pressure
 - Urine output
- Urine dipstick - renal
 - Blood and protein
- Renal ultrasound - post-renal
 - Renal size (>10cm), cortical thickness
 - Renal asymmetry

Management of AKI

- Optimise volume status
- Stop contributing drugs
 - ACE / ARB
 - NSAIDS - NB over the counter
 - Aminoglycosides
- Look for "renal" causes
- Consider and treat sepsis
- Monitor urine output
- Treat complications - high K, acidosis
- Refer if not responding

Indications for RRT in critically ill patients

- Persistent anuria /oliguria
- Severe hyperkalaemia (>6.5)
- Refractory fluid overload
- Metabolic acidosis (pH < 7.1)
- Uraemic complications
 - Pericarditis, encephalopathy, neuropathy
- Severe uraemia
- Severe dysnatraemia (Na >160, <115)

Management of AKI often sub-optimal

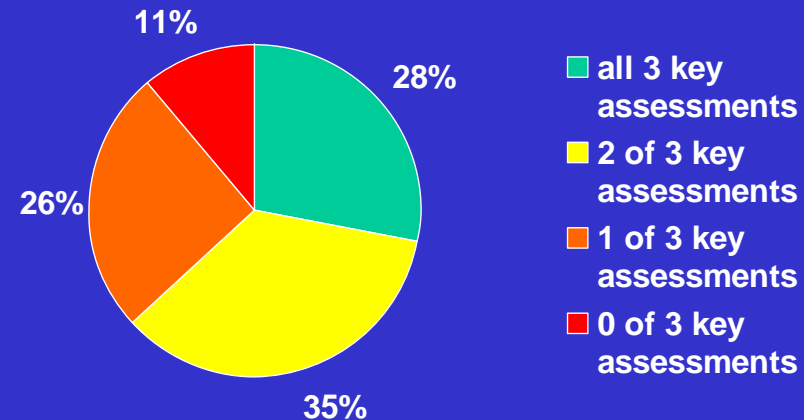
- Kent & Canterbury hospital
- 12 month prospective study
- 288 pts developed AKI (486 pmp/yr)

Key assessments in patient with AKI

- Assessment of fluid status
- Renal tract imaging (ultrasound)
- Urinalysis
- Respiratory rate / O₂ saturation
- Acid/base status
- Calcium / phosphate levels

Key assessments often not carried out

Physiological variable	AKI - all causes (%)	AKI - 2° sepsis (%)
Record of urine output	73	79
Record of respiratory rate	29	38
Renal tract imaging	48	45
CVP measurement	32	47
Acid-base status assessed	60	78
Urinalysis	57	73
Blood cultures	42	67
C-Reactive Protein	18	32
Clotting studies	42	56
Calcium and phosphate studies	66	75
Record of O2 status	30	49



Summary

- Pre-renal, renal and post-renal
- Key assessments:
 - Fluid status
 - Urinalysis
 - Ultrasound
- Review and stop drugs
- Monitor urine output
- Refer for advice of if not improving