

LAKIN Academy 2014

AKI cases for acute physicians

Mark Devonald

Nottingham Renal and Transplant Unit



Nottingham University Hospitals **NHS**
NHS Trust

mark.devonald@nuh.nhs.uk

Case 1

A 54 year old woman, previously very fit and well, was transferred to the renal unit from A+E with SCr 807 $\mu\text{mol/L}$.

She had a 4 week history of malaise, nausea and then left loin pain which started 2 weeks prior to admission.

The GP treated her for presumed UTI with trimethoprim at that stage (2 weeks pre-admission). Symptoms persisted and after 4 days vomiting she presented to A+E.

On arrival at renal unit:

Malaise and continued left loin pain. Tender to palpation left loin.

Looked dehydrated. BP 121/68 T 36.9

Urinalysis revealed blood+++ prot+ nitrites+ leuc+

Creat 807 $\mu\text{mol/L}$, urea 22.5 mmol/L, K 5.9 mmol/L, Bicarb 16 mmol/L

Hb 10.5 WCC 8.6 Plts 323

CRP 178

U/S Right kidney slightly echogenic, otherwise unremarkable.

Case 1

A 54 year old woman, previously very fit and well, was transferred to the renal unit from A+E with SCr 807 $\mu\text{mol/L}$.

She had a 4 week history of malaise, nausea and then left loin pain which started 2 weeks prior to admission.

The GP treated her for presumed UTI with trimethoprim at that stage (2 weeks pre-admission). Symptoms persisted and after 4 days vomiting she presented to A+E.

On arrival at renal unit:

Malaise and continued left loin pain. Tender to palpation left loin.

Looked dehydrated. BP 121/68 T 36.9

Urinalysis revealed blood+++ prot+ nitrites+ leuc+

Creat 807 $\mu\text{mol/L}$, urea 22.5 mmol/L, K 5.9 mmol/L, Bicarb 16 mmol/L

Hb 10.5 WCC 8.6 Plts 323

CRP 178

U/S Right kidney slightly echogenic, otherwise unremarkable.

Q1. What are the possible causes of the AKI?

(Is trimethoprim 'nephrotoxic'?)

Q2. What further investigations would you arrange?

Q3. What would be your initial management?

Case 1

A 54 year old woman, previously very fit and well, was transferred to the renal unit from A+E with SCr 807 $\mu\text{mol/L}$.

She had a 4 week history of malaise, nausea and then left loin pain which started 2 weeks prior to admission.

The GP treated her for presumed UTI with trimethoprim at that stage (2 weeks pre-admission). Symptoms persisted and after 4 days vomiting she presented to A+E.

On arrival at renal unit:

Malaise and continued left loin pain. Tender to palpation left loin.

Looked dehydrated. BP 121/68 T 36.9

Urinalysis revealed blood+++ prot+ nitrites+ leuc+

Creat 807 $\mu\text{mol/L}$, urea 22.5 mmol/L, K 5.9 mmol/L, Bicarb 16 mmol/L

Hb 10.5 WCC 8.6 Plts 323

CRP 178

U/S Right kidney slightly echogenic, otherwise unremarkable.

She was treated with IV fluids and tazocin. Urine output satisfactory.

The following day: creat 680, urea 20.4, K 5.9. She felt a bit better. Same treatment continued.

One day later creat 736, urea 20.2, K 6.0

Q4. Would you continue with same management?

Q5. What is your diagnosis now? Which investigations would you request (if you hadn't already)?

Case 1 **subsequent** results:

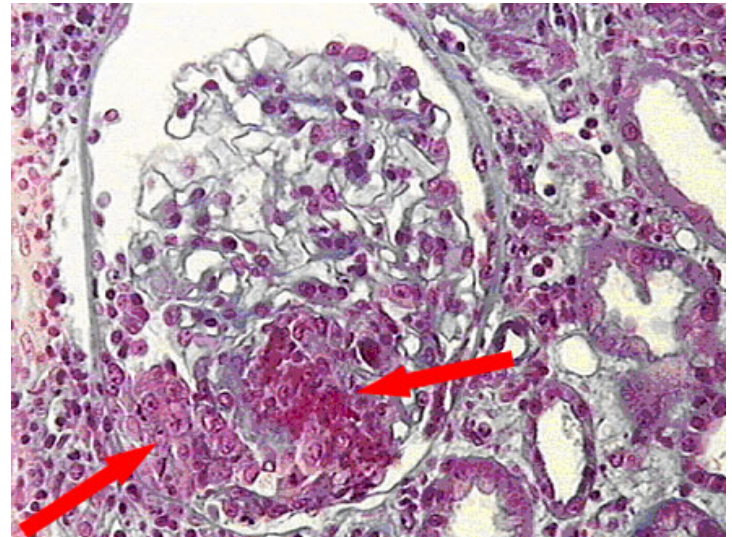
ANCA +ve (MPO >100 U/mL)

GBM +ve

Case 1 **subsequent** results:

ANCA +ve (MPO >100 U/mL)
GBM +ve

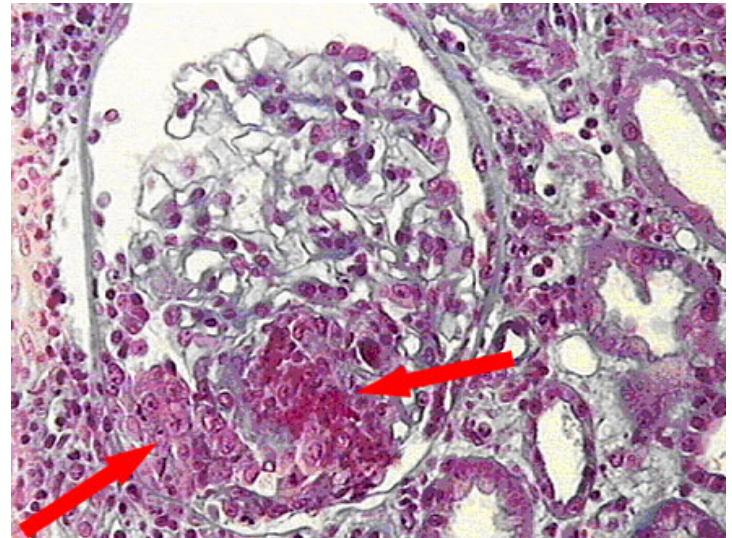
Right sided renal biopsy:



Case 1 **subsequent** results:

ANCA +ve (MPO >100 U/mL)
GBM +ve

Right sided renal biopsy:



Biopsy complicated by perinephric bleed

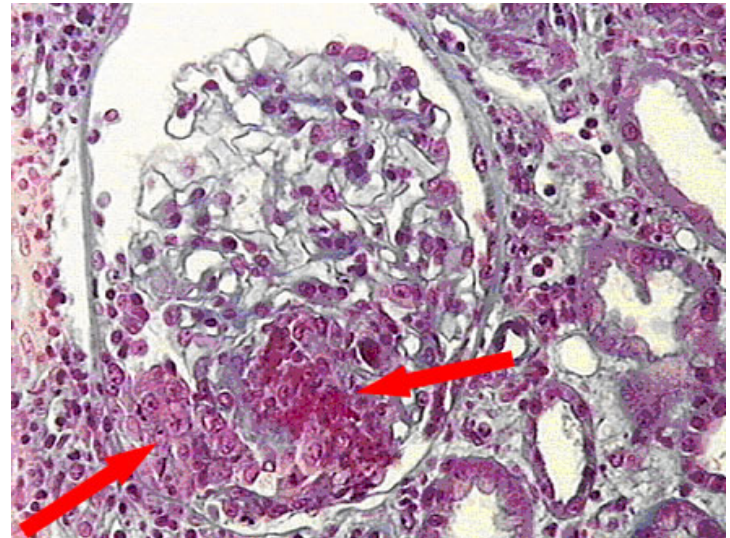


CT Scan

Case 1 **subsequent** results:

ANCA +ve (MPO >100 U/mL)
GBM +ve

Right sided renal biopsy:



Biopsy complicated by perinephric bleed



CT Scan

Right sided haematoma
+
Left renal vein thrombosis

Case 1 management?

- Plasma exchange + cyclophosphamide
- Anticoagulation – approx 6/12 then re-imaging of renal vein

Case 1 management?

- Plasma exchange + cyclophosphamide
- Anticoagulation – approx 6/12 then re-imaging of renal vein

Case 1 outcome:

- GFR approx 40 mL/min after 2 years
- Resolution of thrombus; perfusion of both kidneys

Case 1 learning points:

- Was the sepsis severe enough to account for severe stage 3 AKI?
- Why did renal function not continue to improve with IV fluid + Abs?
- SCr is not an accurate measure of renal function and can be affected by dilution

Case 2

A 69 year old man is referred by the cardiologists with cardiac failure attributed to chemotherapy to treat CLL.

He has increasing oedema and ascites despite increasing doses of diuretics:

IV furosemide 500mg (infusion) per day; metolozone 2.5mg alt days; spironolactone 25mg od; losartan 100mg od

Baseline GFR (3 months previously) approx 45-50 mL/min (SCr approx 140 umol/L).

Progressive worsening of biochemistry over 2/52:

K 6.0mmol/L, Urea 58 mmol/L, SCr 310 umol/L (GFR 19 mL/min), alb 29g/L, Hb 108g/L.

Recent echo: poor RV function, moderately impaired LV function.

Coronary angiogram – no significant CA disease.

O/E

BP 118/58, gross leg + abdominal wall oedema; ascites. chest clear.

Q1. What are the possible causes of the AKI?

**Q2. Would you make any changes to his medical management?
(what is 'diuretic resistance'?)**

Case 2

Losartan discontinued

Metolozone increased to 2.5mg od

Fluid restriction of 750 mL/day

Dietary salt restriction.

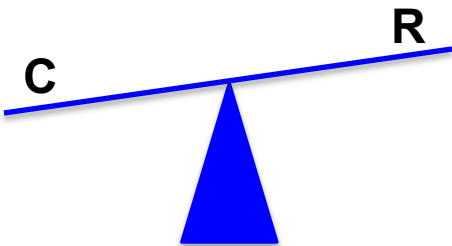
2 days later: K 5.6mmol/L, Urea 64 mmol/L, SCr 330 umol/L

Q3. What are the management options now?

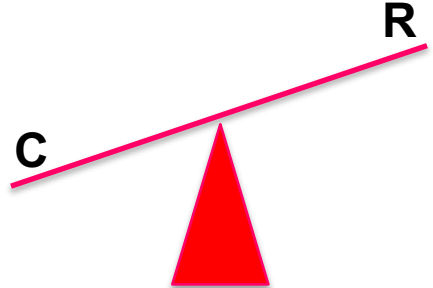
Q4. Would your management be different if the patient had biventricular failure secondary to triple vessel coronary artery disease?

Case 2

Cardiorenal syndrome



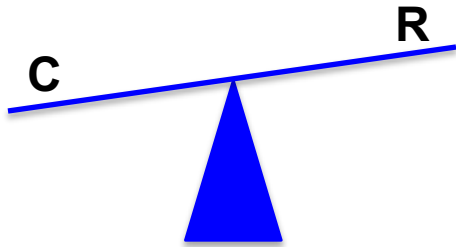
morbidity



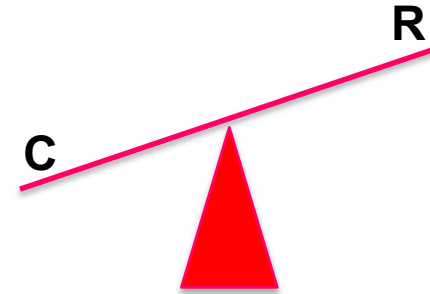
mortality

Case 2

Cardiorenal syndrome



morbidity



mortality

(Renal) Management

- Medical – not just pharmacological
- ?? Ultrafiltration/Renal Replacement Therapy
- Palliative

NICE CG 187 Acute Heart Failure (Oct 2014)
(not AKI or CKD)

Do not routinely offer ultrafiltration to people with acute heart failure.

Consider ultrafiltration for people with confirmed diuretic resistance.

Diuretic resistance is defined as dose escalation beyond a person's previously recognised dose ceiling or a dose approaching the maximum recommended daily dose without incremental improvement in diuresis.

From 'diuretics and ultrafiltration in acute decompensated heart failure' (Felker and Mentz 2012).

Q5

What factors would you take into account when considering UF/RRT?

What modalities are available?

What are the potential disadvantages?

Case 2 learning points:

Case 2 learning points:

- Treat the patient rather than the numbers (...usually)

Case 2 learning points:

- Treat the patient rather than the numbers (...usually)
- You might have to accept a deterioration in biochemistry

Case 2 learning points:

- Treat the patient rather than the numbers (...usually)
- You might have to accept a deterioration in biochemistry
- You are likely to have to accept a compromise between fluid status and degree of 'uraemia'

Case 2 learning points:

- Treat the patient rather than the numbers (...usually)
- You might have to accept a deterioration in biochemistry
- You are likely to have to accept a compromise between fluid status and degree of 'uraemia'
- Prognosis is likely to be determined by cardiac status and potential for improvement.

Case 2 learning points:

- Treat the patient rather than the numbers (...usually)
- You might have to accept a deterioration in biochemistry
- You are likely to have to accept a compromise between fluid status and degree of 'uraemia'
- Prognosis is likely to be determined by cardiac status and potential for improvement.
- UF/RRT is probably only appropriate if there is a realistic chance of improvement in cardiac status

Case 2 learning points:

- Treat the patient rather than the numbers (...usually)
- You might have to accept a deterioration in biochemistry
- You are likely to have to accept a compromise between fluid status and degree of 'uraemia'
- Prognosis is likely to be determined by cardiac status and potential for improvement.
- UF/RRT is probably only appropriate if there is a realistic chance of improvement in cardiac status
- Palliative care is often forgotten or delayed

Case 3

A 76 year old man with an 8 year history of prostate cancer, treated medically, is referred to EAU by his GP after presenting with malaise, nausea, anorexia and mild weight loss. GP had checked some blood tests: SCr 452 $\mu\text{mol/L}$, K 5.4 mmol/L , Hb 98 g/L .

Baseline SCr 4 months previously was 142 $\mu\text{mol/L}$.

PMH included hypertension treated with amlodipine but nothing else of note.

Q1 What else would you like to know from the history?

Case 3

A 76 year old man with an 8 year history of prostate cancer, treated medically, is referred to EAU by his GP after presenting with malaise, nausea, anorexia and mild weight loss. GP had checked some blood tests: SCr 452 $\mu\text{mol/L}$, K 5.4 mmol/L , Hb 98 g/L .

Baseline SCr 4 months previously was 142 $\mu\text{mol/L}$.

PMH included hypertension treated with amlodipine but nothing else of note.

Q1 What else would you like to know from the history?

Longstanding nocturia x2-3; no obvious reduction in U/O
No new medicines

O/E

Thin, BP 164/78, slightly dry. Bladder not palpable.

Urinalysis: trace blood, protein +

Q2 What initial investigations would you arrange?

Case 3

U/S KUB

Right kidney 9.5 cm, slight thinning of cortex

Left kidney 10.5 cm, no obvious abnormality

No hydronephrosis

Q3 What is your differential diagnosis?

Q4 What further investigations and management would you arrange?

Case 3

Differential diagnosis?

Case 3

Differential diagnosis?

1. Obstruction

Case 3

Differential diagnosis?

1. Obstruction

2. Obstruction

Case 3

Differential diagnosis?

- 1. Obstruction**
- 2. Obstruction**
- 3. Something else**

Case 3

Non dilated obstructive uropathy

- ? 5-10% of obstructive uropathy
- Hypovolaemia
- Early obstruction
- Reduced compliance of perirenal tissue
 - retroperitoneal fibrosis
 - cancer: prostate, bladder, colon, cervical
 - lymphoma

Case 3

Learning points

Case 3

Learning points

- High index of suspicion

Case 3

Learning points

- High index of suspicion
- No hydronephrosis does not mean no obstruction

Case 3

Learning points

- High index of suspicion
- No hydronephrosis does not mean no obstruction
- Consider CT, urology ref (retrograde studies)

Case 3

Learning points

- High index of suspicion
- No hydronephrosis does not mean no obstruction
- Consider CT, urology ref (retrograde studies)
- Early decompression where possible