Kidney news
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INTERSTITIAL NEPHRITIS (IN)

The case

One of your regular patients, who usually keeps very well, presents with two weeks of persistent nausea. There are no other GI symptoms. She has been taking St John's Wort and MenoLife® for post-menopausal symptoms. Further systems interrogation is non-contributory. Examination reveals a 78kg woman, BP 174/88, JVP + 2cm, with mild leg oedema. No ascites. No other evidence of fluid overload. No rashes or joint swelling. No other findings of significance. She denies any other medications. She has been off her food a little, but no weight change has occurred.

Routine tests revealed normal blood count; liver tests all normal, Hb 132g/l; electrolytes normal. Plasma creatinine is 0.7mM, and urea 37mM. MSU has no blood and no protein and 44WBCs per high power field. Calculated GFR is 0.13ml/sec/1.73m^2 (8ml/min/1.73m^2).

WHAT NOW?

The most important aspect is to exclude acute renal failure, with a check of the records for an old plasma creatinine.

Result: 0.07mM, 12 months prior. No other values.

THE PROBLEM IS SEVERE, PRESUMED ACUTE, RENAL FAILURE

The presumption is, without recent past creatinine measurements, the creatinine rise is related to her symptoms, and that severe renal failure has been present for days to several weeks (duration of symptoms) and urgent management is required. If the old creatinine was normal last week, that would confirm ARF. In the absence of such a recent result, the issue must be managed as acute. The problem may turn out to be chronic; but an unnecessary "acute renal failure referral" for what turns out to be CRF is better than missing an ARF – that is potentially reversible.

Management requires urgent renal referral. Not a letter; a telephone call with possible admission.

She was admitted. Repeat blood tests confirmed the severe renal failure. Ultrasound scan confirmed two kidneys at the upper limit of normal size (11.1cm and 10.9cm in length).

Normal renal length on USS is between 9 and 11cm. Small adults will have smaller kidneys, and vice versa.

Renal biopsy revealed all the glomeruli were normal, and there was severe interstitial nephritis.

Interstitial nephritis is often found on presentation to have no abnormality in the urine – which may delay the diagnosis. An elevated creatinine with history of drug exposure will help, however. Sterile pyuria (usually < 100WBCs per high power field), eosinophiluria, and proteinuria (without haematuria) are the typical urinary findings.

Drugs of all modalities – OTC, prescription, illicit and herbal may invoke interstitial nephritis.

Removal of the offending drug is the initial management. If the creatinine is mildly elevated (the GFR is > 60ml/min/1.73m^2) then stopping the offending drug(s) may be all the therapy required. **Alternate day monitoring of the plasma creatinine should be performed. Usually within one week, the creatinine will fall**.

If the creatinine continues to rise, or does not settle within one week of drug removal, referral is indicated. Further management including renal biopsy (to confirm diagnosis, and exclude other causes of the renal failure), and initiation of prednisone therapy is likely.

Prednisone (1mg/kg) is the mainstay therapy. A course of several weeks may be required before the creatinine declines back to baseline.

Often the creatinine does not return to baseline, and there is permanent persistent mild CRF. This is often a result of the delayed diagnosis from late presentation. Renal failure symptoms are not present until severe renal failure is present. The patient does not present, and therefore the damage may have been progressing for some weeks to months before presentation. The prolonged damage results in interstitial fibrosis, and permanent kidney damage. A high degree of suspicion of IN by the GP is required for early detection of this reversible condition.

Lifetime monitoring of renal function (3 monthly to annually) and BP and avoidance of future nephrotoxins are all long-term management actions.

**Kidney news is produced in the interest of education of all medical practitioners in the management of kidney disease or general conditions that may affect the kidneys.
Previous issues of kidney news are available at www.kidney.net.nz/newsletters.htm.**

Common drugs associated with IN

Antibiotics
- Ciprofloxacin
- Cephalosporins
- Penicillins
- Rifampicin

Omeprazole
Cimetidine
Sulphonamides containing agents
- Trimethoprim
- Frusemide
- Bumetanide

NSAIDs
Sulphasalazine and aminosalicylates

Common nephrotoxins "herbal" compounds (usually causing IN or fibrosis)

Aristolochic acid
St John's wort
Juniper (Juniperis communis)
Squill (Uriginea maritima)

Black cohosh (Cimicifuga racemosa) – not proven

Nephrotoxins "herbal" compounds
(associated with hypertension)

Broom
Blue cohosh
Capsicum
Ginger
Ginseng
Liquorice

A comprehensive history of all medications and herbal therapies is essential in detecting renal damage from ingested "medications".

Most illicit drugs (designer, street, recreational drugs, and similar terminology) have central nervous system or hepatic toxic effects. Only occasionally have they renal toxicity – and may produce interstitial nephritis.

Cessation of the suspicious or causative agent usually is adequate to allow renal recovery.

Routine follow-up long term of nephrotoxicity from agents causing interstitial nephritis is MSU (for WBCs and proteinuria); plasma creatinine (to assess deterioration in GFR); and regular BP checks. **Prolonged interstitial nephritis will develop into interstitial fibrosis – an adverse prognostic factor in renal disease**.

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Management of urinary tract infections
Investigation of urinary calculi
Investigation of proteinuria and haematuria
Early detection, investigation and management of impaired renal function.
Renal nutrition.

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