

kidney news

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Introduction

2000 Christmas Edition.



MERRY CHRISTMAS

I am delighted to take this opportunity to wish you, your family and members of your practice, a Merry Christmas, and wonderful New Year!

Thank you all for your referrals. I hope you and your patients have found the information beneficial. My practice this year has been busier than any previous year, even allowing for my absences. I have enjoyed the wide variation of referrals.

Some of you prefer me to E-mail these newsletters. No problem just let me know. You should receive the E-mail copy about the same time, or a few days before the hard copy. If you haven't received an E-mail copy, as you read this, E-mail me, and I will return same straight away. E-mail is also the easiest way for me to forward back copies. You will probably still receive a hard copy in the mail-out from either Eastcare or Takanini Care.

Previous topics covered include nephrotic syndrome, urinary infections, uncontrolled hypertension/renal artery stenosis, haematuria.

This year, I started a service in South Auckland, based at Takanini Care – every Thursday night from 17:30. Hopefully that will help provide a closer-to-home service.

Very little is new in renal medicine this year. The same problems of stones, hypertension, impaired renal function, and proteinuria have predominated. This newsletter topic is aimed at what to do when you find the creatinine is elevated. I have also tried to address the choice of radiological investigation too.

Case Study: Stella, a 34 year old mother of 3 (youngest is 5 years) presents with symptoms of a headache that is probably one of her usual migraines – but more serious with a prolonged duration (two days) this time. BP is atypically elevated at 158/90. Examination is normal. As part of the routine assessment, you check the renal function. Electrolytes normal, urea 8.2mmol/l, and creatinine 0.15mmol/l. FBC, glucose, liver tests were normal. No other test was done. **What now?**

Case discussion: The most important question is: **Is this high creatinine new or old?** Are there any old creatinines – other GPs, hospital, or in the lab records? Or does Stella know of previous results elsewhere (e.g. Insurance medical examinations). A helpful part of the history in women is: were there any obstetric complications, especially hypertension, oedema or UTIs in pregnancy? For a normal pregnancy the mother needs good kidneys, as the GFR increases around 40% of baseline, most of which occurs by the end of the first trimester. **Next paramount question is medications, including nutritional/health supplements.** Many medications are nephrotoxic, and problems slowly creep up on the patient, especially when they are OTCs. NSAIDs are a problem here. St John's Wort is another example, as are some herbal extracts. Some Chinese herbal products are toxic, but it is often difficult to ascertain whether the toxicity is due to the whole agent, or one particular ingredient. Generally I find a short time off the suspected agent tells all. This works if the offending agent hasn't been taken for many months, otherwise permanent scarring may have occurred – and renal damage (and serum creatinine) is irreversible.

WHAT'S IN HERE THIS TIME?

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The GFR has already declined approximately 50% by the time the serum creatinine rises above the upper limit of normal (about 0.10mmol/l for a mid-thirties woman). **What should be done now?** Repeat the history, especially for medications (prescribed and non-prescribed), diet history (especially for multi-vitamin (e.g. some supplements have high vitamin D and calcium content, that may cause hypercalcaemia) and for herbal agents). Family history renal disease, and hypertension, and check BP, hydration status (JVP should be visible above the clavicle), look closely for rashes (e.g. connective tissue diseases, lupus) and dipstick the urine. The **urine dipstick** is an excellent screen. If blood is present hypertensive damage, glomerulonephritis or stones are some of the more common possibilities. Proteinuria implies renal damage – either glomerular or interstitial, and requires many further tests, and almost invariably a **renal biopsy**. Further investigations are also needed now. The minimum laboratory tests: MSU (for blood protein, and microscopy for casts), repeat creatinine (is it getting worse by the day), serum urea (helps with assessment of hydration status) and electrolytes. Serum calcium (high from thiazide diuretics, hyperparathyroidism, or excessive intake of calcium or vitamin D, or myeloma) and phosphate. Serum albumin (nephrotic), full blood count (if not previously done), 24 hour urinary protein if more than 1+ protein on dipstick/MSU result. There may be other serological tests, but I like to be selective about these – often after the renal biopsy, if needed.

Now is the time for the radiology. The **ultrasound scan is the first best radiological test** in almost all cases. The length of the kidneys must be measured, recorded and reported by the radiographer/ radiologist. Very obese patients are the only real exception to good views being obtained. Normal sized kidneys (9-11cm) or mildly enlarged (10-13cm) generally suggests acute renal deterioration and more prompt referral (within a week or two). If the creatinine significantly (more than 10%) rises in the few days from the first routine check to the follow-up repeat – then referral/ telephone discussion that day is advised. Large kidneys (>13cm) suggest diabetes mellitus or infiltrative diseases e.g. myeloma or lymphoma, or sarcoidosis. Cysts are common over the age of 50 years. Several cysts in each enlarged kidney after the age of 30 years is highly suggestive of polycystic kidney disease. It is unwise to reassure someone doesn't have PCKD until the absence of cysts on an ultrasound scan performed over the age of 30 – false sense of security may be set. **One kidney** (congenital in 1:500 births) should be large (compensatory hypertrophy) – at about 13-15cm in size. Less than this is abnormal. A solitary kidney of 12cm length is abnormal. One (or two) small kidneys suggest chronicity. The catch here is one kidney may have been small for years (and asymptomatic), and now a disease affecting the one remaining kidney results in the presentation/symptoms or the abnormal finding. A good example of this is **renal artery stenosis**. Increased echogenicity of the renal parenchyma is not disease specific. It merely reflects renal damage, often chronic or interstitial inflammation. This is the **time for referral**. These tests do not need to be done before referral, but I will do them first, if not already done.

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Interests

Investigation of renovascular disease and hypertension

Management of urinary tract infections

Investigation of urinary calculi

Investigation of proteinuria and haematuria

Investigation and management of impaired renal function.

Renal nutrition.

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