Kidney news
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GLOMERULONEPHRITIS (gn)

Glomerulonephritis leads to reduced ability to filter the water soluble waste products; the appearance of lipids, protein and/or blood in the urine. Persistent proteinuria is renally toxic, and will result in progressive nephron, and therefore GFR, loss.
Interstitial nephritis was discussed in a previous newsletter.

Detection and Diagnosis of glomerulonephritis
The dipstick urine test, complemented with microscopy of urinary sediment, is essential in the detection of urine abnormalities. The urine dipstick tests for albumin and not protein; hence immunoglobulins (and light chains of myeloma) are not detected. The sulpho-salicylic acid (SSA) assay in the biochemistry laboratory will test for all urinary proteins (including myeloma).
Red corpuscular casts imply glomerulonephritis or vasculitis.
White cell casts imply pyelonephritis or interstitial nephritis.
Definitive diagnosis will always require a renal biopsy; with or without serological laboratory blood tests.
Therapy is aimed at good blood pressure control <130/80; and minimisation of proteinuria (<1G/24 hours); and some conditions require immunosuppressive therapy (eg. prednisone, azathioprine, cyclophosphamide) to dampen the inflammatory processes, and improve GFR and reduce proteinuria.

Nephrotic syndrome
Heavy proteinuria and lipiduria, and uncommonly haematuria. Often not associated with hypertension, unless there is an associated disease with which there may separately be hypertension (e.g. lupus or diabetes mellitus).
Common diagnoses in children are minimal change (MCN) and focal and segmental gn (FSGS); and in adults: MCN; membranous; FSGS; amyloid and diabetes mellitus.
Focal glomerulonephritis
Often insidious presentation, and mild proteinuria and haematuria. Minority of kidney tissue involved. Common diagnoses are: post-infectious gn (especially children); IgA nephropathy; HSP; thin-basement membrane disease.
Diffuse glomerulonephritis
Majority of renal tissue involved, with often hypertension, loss of GFR, and heavy proteinuria.
Common diagnoses are: post-infectious gn (children); IgA nephropathy; membranoproliferative gn; vasculitis; lupus.

IgA nephropathy (=IgA-positive focal gn)
This is the most common glomerulonephritis. IgA nephropathy is more common in the Asian and Caucasian ethnic groups. It is frequently first detected on routine MSU for insurance or immigration medicals. The other common presentation is with synpharyngitic (occurring at the same time as the sore throat) macroscopic haematuria. History is important here to distinguish IgA from the delayed haematuria of post-infectious gn.
Prognosis is defined mainly by the four features: hypertension; reduction of GFR, proteinuria and scarring on renal biopsy. Microscopic haematuria without proteinuria, reduced GFR or hypertension has a relatively benign prognosis; and renal biopsy may not be necessary at initial presentation. The severity of haematuria does not define prognosis.
Monitoring (once to twice per annum) for the development of proteinuria (MSU) and to ensure renal function remains stable is all that is necessary in such mild cases. These adverse signs usually develop before hypertension. IgA nephritis (nephropathy) progress is stable in most cases (80 - 90%); or a slow relentless progressive course of chronic renal failure (10 - 20%).
The rate of GFR loss is typically slow (1-3ml/min/year (cf. 0.8ml/min/year from aging itself)) hence potent immunosuppressive therapy is uncommonly required or indicated. As proteinuria is a particularly adverse prognostic feature, therapy is aimed at maximal tolerated dose of ACE inhibitor or ARB to minimise proteinuria. Even in the absence of hypertension there is an indication for the use of ACEI and/or ARB. Studies have shown ACEI or ARBs are better than other classes of anti-hypertensives at delaying progression of renal failure
One small trial has shown the combination of ACEI (enalapril) and ARB (losartan) together reduced the proteinuira than either agent alone. This is a similar finding in the management of type 1 diabetic nephropathy associated proteinuria.
Omega 3 or fish oil has been researched and the evidence is conflicting. The largest trial (106 patients) showed a benefit of 12G fish oil (which is a lot of capsules).
Therapy has to be for 2 to 4 years; and benefit may only be for those who have >3G of proteinuria. With only little possibility of benefit, and the fact that fish oil capsules are not subsidised; such therapy becomes an expensive 2 to 4 year venture for the patient.

Thin basement membrane (TBM) disease
TBM disease (=benign familial haematuria) presents with microscopic haematuria. On renal biopsy the basement membrane is approximately half the normal thickness. This is the only abnormality noted. It appears to be the manifestation of a heterozygous gene defect of the homozygous disease form that results in Alport syndrome

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(associated with deafness and progressive renal failure). There is a weak association with hypercalciuria and hyperuricaemia in TBM; increasing the risk of urinary tract stones.

TBM requires no treatment, has an excellent prognosis; and almost never progresses to ESRF. Future monitoring is not necessary; however one does need to exclude renal tract malignancy in such cases, especially in patients over the age of 45 years.

Minimal change nephropathy (MCN)

Minimal change disease is common in children; and frequently seen in young adults as the cause of nephrotic syndrome. It is uncommonly associated with Hodgkin's, NHL and leukaemia. Some food allergies, NSAIDs, quinolones, lithium and pamidronate therapy are also rare causes of MCN.

Corticosteroids are the main stay of therapy. Therapy is often prolonged (months), although intermittent because of remission and relapses. Relapses seem to be less frequent with slower dose reduction. Bone calcium sparing treatment (bisphosphonates are often adjunctive therapy in adults). Dose reduction can commence once proteinuria is normal (<300mg/day). GPs can often manage the prednisone dose reduction, with proteinuria monitoring.

Occasionally cyclophosphamide is required to obtain prolonged remission.

Membranous nephropathy (MN)

More common in adults. Uncommonly associated with solid tumours – especially lung and colon, Successful treatment of the cancer resolves the MN. Can be caused by penicillamine or gold; associated with hepatitis B and C. As therapy is immunosuppressive based, these causes need to be excluded before therapy. *Most commonly MN is idiopathic*.

More difficult to treat and treatment with prednisone may be prolonged in adults – 3 to 6 months at 1-1.5mg/kg per day.

Focal and Segmental Glomerulosclerosis (FSGS)

FSGS is a frequent cause of renal failure in New Zealand; presenting with reduced GFR, hypertension and proteinuria. Frequent relapsing or steroid resistant MCN in children often is FSGS; and renal failure will ensue. FSGS is usually primary. Secondary cases often develop (and therefore have a less favourable prognosis) from nephron loss – common examples would include other glomerulonephritides; or reflux nephropathy. Obesity is a cause of FSGS.

The rate of progressive loss of GFR is variable, may be aggressive and rapid, or may take many years.

Immunosuppressive therapy may be indicated. FSGS is more resistant to immunosuppressive therapy; and often will progress along a relentless course to end-stage renal failure.

Good blood pressure control (<130/80mmHg) with ACEIs or ARBs (with their additional proteinuria-reducing effect) is the basis of therapy.

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Special Interests

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

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