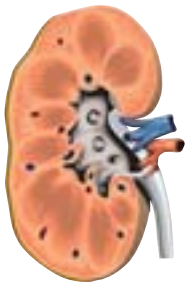

Atherosclerotic renal artery stenosis (ARAS) & chronic renal failure

Use ACE inhibitors first, not stents

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Although excellent technical results can be achieved by percutaneous techniques, controlled studies show that these procedures should be reserved for specific acute indications, where kidney damage is still reversible. ACE inhibitors or angiotensin receptor blockers (ACEIs/ARBs) should be used in all cases for renoprotection, provided there is no more than the expected 30% rise in serum creatinine.

ARAS is common, being found in up to 40% of patients with clinical peripheral vascular disease, typically elderly males with orificial or proximal renal arterial involvement. More extensive vascular disease correlates with increasing risk of both ARAS and cardiovascular death from any cause. In contrast, fibromuscular dysplasia typically affects younger females, involving the more distal renal artery, with milder degrees of usually non-progressive stenosis.

ARAS should be suspected in the patient with vascular disease in three scenarios associated with activation of the renin-angiotensin axis from renal ischaemia:

- Refractory hypertension (especially with relative hypokalaemia),
- "Flash" pulmonary oedema, and
- Rapidly deteriorating renal function (especially within a month of commencement of an ACE inhibitor or Angiotensin receptor blocker in a patient with a solitary functioning kidney).

Renal ultrasound with Doppler examination may provide the diagnosis, and give critical information on kidney size. The technique is slow however, with best results from an experienced operator and a slim patient.

ARAS is usually static, with recent prospective Doppler studies showing 60-80% of those with > 60% stenosis to have stable renal function and renal size. Patients with unilateral complete occlusion or bilateral stenoses were at higher risk of progression, presumably having more advanced or aggressive disease.

ARAS, patent or stenosed, is usually associated with renal parenchymal damage. Whilst the artery is patent, the kidney is exposed to the same atherosclerotic insults as its artery, producing renal lesions such as hypertensive nephrosclerosis, focal glomerular sclerosis, atheroemboli and/or diabetic nephropathy. When arterial stenosis worsens, the kidney is protected from the hypertensive damage, but then suffers ischaemic nephropathy, with cortical atrophy and glomerular tuft collapse.

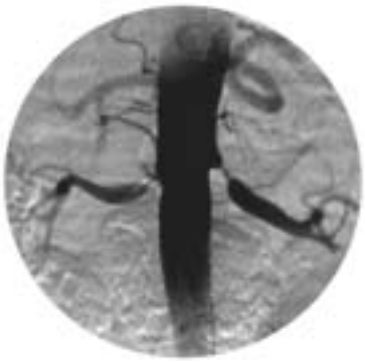
Renal parenchymal damage correlates with proteinuria and atrophy, not arterial patency. Proteinuria does not always imply irreversible renal damage however, and can reflect the degree of intraglomerular hypertension, rather than the extent of glomerular damage.

Aggressive medical management is mandatory to correct all vascular risk factors and optimise renoprotection. Even in the absence of ARAS, an average of three antihypertensive agents is required to achieve the target BP of < 130/80 for patients with renal impairment. Tight control of dyslipidaemia and diabetes, cessation of smoking, and use of antiplatelet agents are all necessary, as atherosclerosis is a systemic condition.

ACEIs or ARBs alone (or even better, in combination) provide better renoprotection than other antihypertensives, especially if proteinuria is present. A rise in serum creatinine of up to 30% occurs in most patients with renal impairment after commencing these agents, due to correction of intraglomerular hyperfiltration. This creatinine rise on ACEIs/ARBs does not imply the presence of renal artery stenosis, provided a subsequent serum creatinine has stabilised. Often, all that is required is an ACEI/ARB dose reduction, temporary omission or cessation of any diuretic, and then increased calcium channel blockade.

Theoretically, monitoring total renal function by serum creatinine alone may mask the effect of change in unilateral renal lesions. Use of ACEI/ARBs in the presence of a unilateral ARAS may cause deterioration in the function of the affected kidney, but little change in the serum creatinine if the function of the contralateral kidney improves simultaneously. Most clinicians are currently accepting this as a calculated risk, provided the serum creatinine stabilises; serial isotopic measurement of split renal function is expensive and loses accuracy in the presence of poor renal function.

Intervention with percutaneous renal angioplasty (PTRA), stenting or surgery is usually technically successful in >90% of cases. Functional success is much less certain however, with less than a third of patients achieving improved blood pressure control or preservation of renal function. Five randomised controlled trials have compared medical therapy, PTRA, stenting or surgery in varying combinations, with no treatment showing superior efficacy to another. Morbidity and mortality was least with medical therapy, and highest with surgery (as expected in the presence of diffuse vascular disease).



ASTRAL Trial. The Angioplasty and STent for Renal Artery Lesions trial is currently recruiting 1000 patients worldwide over the next four years in a randomised study to compare efficacy of optimal treatments. See www.astral.bham.ac.uk, or speak with local investigator Dr Ashley Irish at Royal Perth Hospital (9224 2244, ashley.irish@health.wa.gov.au).

SUSPECT:

Vascular disease or risk factors plus:

- Refractory hypertension (especially with relative hypokalaemia),
- "Flash" pulmonary oedema, and
- Rapidly deteriorating renal function (especially with ACEIs/ARBs)

INVESTIGATE:

- S. creatinine and calculated GFR
- Spot urine protein:creatinine ratio
- Renal Ultrasound (for renal length and atrophy)
- Renal arterial Doppler

TREAT:

- BP, cholesterol, diabetes, smoking aggressively
- Use ACEIs/ARBs (or both) where CRF or proteinuria are present
- Expect up to 30% increase in serum creatinine

DISCUSS:

- Risk:benefit of intervention (PTRA, stent, surgery)
- Possible ASTRAL Trial eligibility

Footnote. Kidney Health Australia (formerly the Australian Kidney Foundation) has initiated a nation-wide education project, Kidney Check Australia Taskforce (KCAT), to reduce endstage renal disease through earlier detection and management of mild chronic renal failure. The key elements are investigation of high risk groups, initiation of appropriate therapy and timely referral. The opinions expressed in the above article are those of the author and not necessarily endorsed by KCAT.

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