RCPE: Renal Medicine

Thursday 22 April 2021

Antineutrophil cytoplasmic antibody (ANCA) associated vasculitis in 2021 - Renal: Dr Kate Stevens & Extrarenal: Dr Lucy McGeoch,

Q. In patients with renal involvement, does treatment improve renal function*?*

A. Broadly speaking it depends - We give treatment with the aim of preventing further damage. If there is a lot of acute inflammation within the kidney, there is a reasonable chance of improvement in function with immunosuppression. If there is a lot of sclerosis / chronic damage then there is less chance of seeing any improvement. The kidney biopsy (as per slides in talk) can help predict chance of developing ESKD.

Still immunosuppress if dialysis dependent at presentation and from there decision to continue is often individualised but broadly if no extra renal involvement and no evidence renal recovery or ongoing renal involvement then probably only 3-6 months of treatment. If extra-renal manifestations then treat as would otherwise. If ongoing renal involvement ie non visible haematuria but dialysis dependent then likely to continue as if not on dialysis but this will depend upon patient / circumstances.

Sodium-glucose cotransporter-2 (SGLT2) inhibitors - treatment for diabetic and non-diabetic kidney disease - Associate Professor Will Herrington

Q. ***Is there any benefit / merit in their use with T1DM / nephropathy****?*

A. Use in T1DM should be by diabetologists under close supervision only, with only dapagliflozin licenced for use in T1DM at a lower dose than normal. There have not been trials of sufficient size or length to confirm the likely renal and cardiovascular benefits, but the short trials with glycaemic outcomes (E.g. the EASE trials and inTandem3) show the absolute risk of ketoacidosis is much higher in T1DM than in T2DM, and ketone meters and careful patient selection would be advisable.

Q. ***Cardiologists very keen to start in cold and Hf common issue, is this off licence?***

A. Not sure what cold means – sorry. Dapagliflozin has a licence for use in stable reduced ejection fraction heart failure following the DAPA-HF trial (down to an eGFR of 30), and data from EMPEROR-REDUCED are consistent with DAPA-HF. Data in HFpEF from EMPEROR-PRESERVED are due later this year.

Q. ***I feel the risks are stressed more than the potential benefits at times - might this be putting patients off accepting them?***

A. Yes, in the studied populations in the large trials in T2DM, CKD and HFrEF, there is clear evidence of net benefit on hard clinical outcomes and putting this in a quantitative way to patients is a challenge. Simple participant information leaflets are needed (we are preparing some for patient with CKD as part of UK Kidney Association guidelines, and I believe diabetologists from ABCD have already produced such templates).

Q. ***What are the challenges with the non-use of these drugs in patients with eGFR < 25 but not dialysis-dependent? Lack of evidence or increased adverse events?***

A. These agents don't have an important antiglycaemic effect at low eGFR, and so safety data in such participants is sparse due to their exclusion from the safety trials in T2DM. Data is emerging from the CKD trials (and a small amount of data at low eGFR from the HFrEF trials), but there are still very few years of participant follow-up in those with an eGFR<25. EMPA-KIDNEY will hopefully report next year and provide important data in those at low eGFR.

How I manage renal stone disease - Dr Graham Stewart

Q. ***Which ones do you really not want to miss? Are they usually obvious?***

A. Cystinuria and primary hyperoxaluria should be identified and treated as early as possible, as they have the potential to cause progressive renal damage without intervention.

Cystinuria is easily identified by testing for the presence of amino acids in a spot urine sample. Genetics for primary hyperoxaluria are diagnostic and should be sent on any patient with a 24hour urine oxlalate>1mmol/day.

I had assumed that these 2 groups of patients would be more obvious in their presentation with histories starting in childhood and frequent interventions, however, the few that I have picked up have presented in adult life and did not have particularly frequent symptoms.

The other group who benefits from early detection are calcium phosphate (brushite) stone formers. This is because they will recur frequently, require more intensive intervention to impact on frequency of recurrence and there is the potential to make the situation worse with use of potassium citrate leading to urine alkalinisation. The only way to identify these patients is with stone analysis.

This is the book I could not remember the name of when asked for a good, pragmatic guide to investigation and management. I would recommend it highly. £35 on amazon.

How to approach acid-based disorders - Dr John Neary

Q. ***Interested in the occasional finding of high lactate in some cases of toxic alcohol ingestion?***

A. My original short answer to this was that, yes, we do see Lactic Acidosis in the setting of Toxic alcohols, but I'm not sure how much of the problem is the toxic alcohol or the fact that the patient is otherwise unwell with Vomiting and hypotension – ie low BP more common cause of Lactic acidosis. I should also add that in that setting, I would treat other causes such as hypotension as a priority and not just assume that the raised lactate is due to toxic alcohol. However, it is listed in all the tables of causes of Lactic acidosis and according to the excellent review article in NEJM*, is due to "interference with oxidative phosphorylation" (Kraut and Madias, NEJM 2014; 371; 2309-19).