Gout and hyperuricaemia

Uric acid levels are raised in chronic renal failure, and in view of that, gout sometimes seems less common than expected.

- Asymptomatic hyperuricaemia should usually not be treated.
- Patients with very early onset gout should be screened for predisposing genetic abnormalities.
- Lead poisoning should be remembered as a cause, but is rarely the explanation for gout with renal impairment and hypertension.
- Tophaceous gout may be associated with interstitial granulomatous nephritis with crystal formation – but rarely except in inherited severe gout.

Allopurinol

Is more likely to cause toxicity in renal impairment and initial dose should be 100mg. It may rarely cause interstitial nephritis. Its introduction may precipitate acute gout and this needs to be protected against with nonsteroidals or colchicine.

Allopurinol inhibits metabolism of azathioprine. Although some advice suggests reducing azathioprine dose by two thirds, consideration should be given to alternatives, or cessation of azathioprine, if allopurinol is clinically necessary.

Nonsteroidals

Are not necessarily completely contraindicated in moderate renal failure, if used with monitoring and for a limited period.

Colchicine

Is the first choice for treatment of acute gout in patients with significant renal impairment. It has a narrow therapeutic ratio, toxicity manifesting as diarrhoea, nausea and vomiting. Reduced dosing is recommended at GFR 10-50, and extreme caution if GFR is less than 10. The BNF recommendations (2mg then 500mcg every 2-3h) will often cause severe side-efffects; safer to start with 500mcg tds and increase as necessary (reduce if necessary). Start with less in very small people or with severe renal impairment.

Lower doses (e.g. 500mcg 1-3 times daily) are useful prophylactically.

Ref: Morris et al 2003. Colchicine in acute gout. Br Med J 327:1275-6

Uricosurics

Are of little value in the presence of significant renal impairment. In addition to the conventional uricosurics probenecid and benzbromarone, losartan and fenofibrate have some uricosuric activity.

Acute hyperuricaemia

Occurs particularly in tumour lysis syndromes (though prophylactic allopurinol usually prevents it), or in high turnover haematological malignancies such as AML. Causes acute renal failure through crystalluria. Treatment has three elements:

- Remove urate by high intensity haemodialysis – will require long and frequently repeated treatments.
- Prevent uric acid formation with allopurinol – high doses indicated.
- Recombinant uricase (urate oxidase) appears effective, beginning to lower uric acid levels in hours when administered prophylactically.

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