Hypercalcaemia

In chronic renal failure of long standing, hypercalcaemia is usually caused by the use of vitamin D derivatives and phosphate binders, and sometimes by tertiary hyperparathyroidism. [See section on Osteodystrophy]. Usually stopping vitamin D and calcium containing phosphate binders is enough, but be cautious if patient is on a significant dose of vitamin D or has had a parathyroidectomy – calcium may plummet.

In acute renal failure calcium is usually low-normal. A high-normal or high calcium should lead to suspicion that the renal problem is caused by hypercalcaemia itself, or by the same disease as is responsible for hypercalcaemia — e.g., myeloma.

Treatment of Hypercalcaemia

Fluid repletion – with saline improves renal impairment caused by hypercalcaemia and has a small effect on calcium level.
Diuretics – should be avoided unless adequate volume expansion with normal saline has been achieved. Thereafter loop diuretics can be used with caution, but thiazides tend to increase serum calcium.
Corticosteroids – are effective in sarcoidosis, some haematological malignancies and allegedly also in vitamin D poisoning.
Bisphosphonates – are effective in all circumstances. Disodium pamidronate can be given as a single dose of 30-90mg over 2-4 hours (maximum infusion rate 20mg/h in renal impairment; maximum concentration 60mg in 250ml 0.9% NaCl). Calcium falls over days, reaching a nadir at 3-5 days, and usually remaining suppressed for several weeks, when the infusion can be repeated. Note that high doses of pamidronate may be nephrotoxic – associated with proteinuria and FSGS.

For dialysis patients, adjusting calcium content of dialysate may be helpful.

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