Persistent and symptomatic post-transplant hyperparathyroidism: a dramatic response to cinacalcet

Sir,

We read with interest the articles by Kruse et al. [1] and Serra et al. [2], which demonstrated the beneficial effect of cinacalcet HCl in the treatment of persistent hyperparathyroidism following renal transplantation. We would like to report our own experience of one selected case of post-transplant hyperparathyroid bone disease, which highlights not only normalization of laboratory-measured parameters but also a dramatic response in symptomatology.

A 79-year-old lady with stable graft function (serum creatinine 96 µmol/l) 13 years post-transplant on cyclosporin monotherapy developed widespread bone pain and myalgia requiring opiate analgesia and eventual hospitalization. Investigations revealed the following: plasma intact parathyroid hormone (PTH) 2690 pg/ml, alkaline phosphatase (ALP) 689 U/l, calcium 2.07 mmol/l and phosphate 0.98 mmol/l. X-ray of her hands showed evidence of early hyperparathyroid bone disease.

Her hyperparathyroidism was refractory to vitamin D therapy and, due to multiple comorbidities, she was deemed unfit for parathyroidectomy. She was, therefore, treated with cinacalcet at a starting dose of 30 mg daily, reducing to alternate day dosing following the development of hypercalcemia. PTH fell to 470 pg/ml after 2 weeks of therapy, this response being sustained over almost 4 months (327 pg/ml at 15 weeks). ALP fell to 342 U/l and calcium and phosphate levels remain within normal limits at 15 week follow-up. Most importantly, her severe bone pain and myalgia have improved dramatically allowing withdrawal of opiate analgesia, significantly improving her quality of life. There has been no change in graft function, blood pressure control or other medication during this time and she has suffered no side effect attributable to cinacalcet.

This patient demonstrates a dramatic and sustained response to cinacalcet, characterized by a 91% fall in PTH (Serra et al. [2] observed an 18% reduction) and resolution of symptomatic hyperparathyroidism, previously effected only in renal transplant patients by parathyroidectomy. We would, therefore, advocate a role for cinacalcet in transplant recipients with symptomatic persistent hyperparathyroidism who are not suitable for parathyroidectomy.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfk018

Estimation of glomerular filtration rate in patients with normal serum creatinine undergoing primary PCI: is it really normal?

Sir,

Kidney disease and cardiovascular disease seem to be lethally synergistic and both approach a level of epidemy, particularly in the elderly. The 2002 DOQI (Dialysis Outcomes Quality Initiative) states that individuals with a reduced glomerular filtration rate (GFR) are at greater risk for cardiovascular disease (CVD) and cardiac deaths [1]. The ability to identify chronic renal insufficiency may allow early implementation of treatments that could arrest or delay the progression of renal damage, enable effective treatment of its complications and reduce the risk of drug-induced nephrotoxicity. Contrast nephropathy is a potentially serious complication of diagnostic angiography and percutaneous coronary intervention (PCI) [2]. Unfortunately, the cardiovascular risk increases in a concentration range of serum creatinine where this parameter is very insensitive to changes in GFR. The current Kidney Disease Outcomes Quality Initiative (K/DOQI) guidelines advocate creatinine-based equations for estimating the GFR to identify patients with potential kidney disease and to classify them into different stages on the basis of these values [1].

The aim of our study was to establish the prevalence of kidney dysfunction in patients with normal serum creatinine

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfk003