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P246 -Setting targets for the management of mineral bone disorders in haemodialysis –do they help us?

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Introduction. Renal Association guidelines recommend two audit measures in the assessment of mineral bone disorders (MBD) in ESKD – the proportion of patients with serum phosphate <1.7mmol/L and with all three bone parameters in range (calcium 2.2-2.5mmol/L, phosphate 1.1-1.7mmol/L and PTH 16-72pmol/L). Guidelines are based on associations between biochemistry and mortality of unproven causality. Implicit assumptions include that low serum phosphate is related to malnutrition and low/normal PTH is associated with hypercalcaemia/adynamic bone disease.
The UK Renal Registry publishes units' MBD outcomes. We explore why a specific unit's performance was identified as a statistical outlier for poor performance. For this unit, calcium and phosphate (<1.7) outcomes were achieved in 87.9% and 76.8% of patients respectively. Only 43.4% were in range for PTH (13.9% >72; 42.8% <16). Low PTH (42.8%) and low phosphate (27.1%) were the commonest reasons for non-compliance with the stated target in the 2017 Report.

Method. To explore the relevance of these findings, we investigated the current haemodialysis cohort (n=200). Medical and medication histories were reviewed in the 100 subjects with PTH <16. Adherence to unit protocol was assessed. The protocol recommends that serum vitamin D is measured at first dialysis and deficiency/insufficiency corrected with oral colecalciferol; calcium is maintained in the range 2.2-2.5, through manipulation of the dialysate [calcium] plus oral calcium supplements in patients with hypocalcaemia despite high dialysate calcium; phosphate is maintained <1.7 through dietary advice and non-calcium based binders, except in the presence of hypocalcaemia (where calcium-based binders are substituted); low dose alfacalcidol (1-alfa) is prescribed for hypocalcaemia if there is evidence of a proximal myopathy or secondary hyperparathyroidism and higher dose 'pulsed' 1-alfa is used to manage tertiary hyperparathyroidism. Calcimimetics and surgical parathyroidectomy are considered only for uncontrolled tertiary hyperparathyroidism.

Results. Excluding 15 patients who had a surgical parathyroidectomy, 24 were currently on 1-alfa, all of whom had previously elevated PTH levels. 33 had never received 1-alfa. The remainder had received 1-alfa in the past when PTH was elevated. Dosing appeared appropriate to the PTH level at initiation of prescription (median PTH in untreated patients = 18.4; median PTH in patients receiving low dose 1-alfa = 31.4; median PTH in those receiving pulse 1-alfa = 65.5). None were taking calcimimetics. A low phosphate (<1.1 mmol/L) was not associated with evidence of malnutrition, dietary restrictions, nor prescription of phosphate binders. There was no relationship between PTH and serum [calcium] or [phosphate].

Discussion. Defining unit performance on biochemical outcomes may create false impressions. The assumption that PTH <16 or phosphate <1.1 is associated with worse outcomes does not take account of individual patients' trajectories nor the reasons underlying the biochemical results. We don't know whether preventing secondary hyperparathyroidism in the first place or returning PTH to near normal values through correction of vitamin D deficiency and good control of calcium and phosphate is harmful. Until that is established, should we strive towards preventing secondary hyperparathyroidism, rather than being discouraged from doing so?