as well as material support from the manufacturer of the BTM, Fresenius Medical Care, Germany.

Conflict of interest statement. None declared.

Institute of Physiology Daniel Schnedtiz Medical University of Graz

E-mail: daniel.schnedtiz@meduni-graz.at


doi: 10.1093/ndt/gfm741

Advance Access publication 5 November 2007

Reply

Sir,

We thank Dr Schnedtiz for his interest in our study. In his comments, the hypothesis that low blood glucose levels without symptoms could not be real, but attributable to recirculation of venous line blood in the vascular access when using glucose-free dialysate, is interesting and apposite.

Hypoglycaemia without symptoms has been previously described in diabetic patients, in circumstances other than dialysis [1] and during regular haemodialysis in diabetics and non-diabetics [2–4]; some mechanisms have been implicated in the explanation for the absence of symptoms [5–7]. This was, in fact, the motivation and the first purpose of our study: to evaluate the frequency of this phenomenon, and suggest a way to prevent it.

In our study, the possibility of recirculation in the vascular access was not directly verified, but there is some evidence against its presence: the URR of all patients enrolled in the study was regularly under 0.30 in the previous months and in the study (an indication of adequate dialysis dose, not achievable with recirculation in the vascular access); hypoglycaemia was repeatedly not observed in all blood samples of the same patient, as would be expected in the presence of significant recirculation (in this case, certainly present in the whole session of the dialysis).

Furthermore, other studies have demonstrated significant reduction of glucose levels in the blood running out of the dialyzer when using glucose-free dialysate [8], and we and others [2,3,9] found a significant loss of glucose in the dialysate leaving the dialyzer, all pointing to the possibility of a ‘real’ occurrence of systemic hypoglycaemia.

Finally, we agree that, in such a case, blood samples obtained from the peripheral circulation must be preferred, to avoid this possible kind of bias.

Conflict of interest statement. None declared.

Unidade de Medicina Renal Jayme Eduardo Curso de Medicina Burmeister Universidade Luterana do Aline Scapini Brasil Diego da Rosa Miltersteiner Marcelo Generali da Costa Bruno Machado Campos

E-mail: burmeister@via-rs.net


doi: 10.1093/ndt/gfm742

Advance Access publication 28 November 2007

Is PTH a risk factor for cardiovascular calcifications in haemodialysis?

Sir,

We read with interest the article in NDT by Coen et al. [1], on the association between serum intact parathyroid
hormone (iPTH) levels higher than 600 pg/ml and the severity of coronary calcifications in haemodialysis (HD) patients. In their manuscript, the authors divided 197 HD patients into four groups of serum iPTH levels: (A) 0–150 pg/ml; (B) 150–300 pg/ml; (C) 300–600 pg/ml; (D) >600 pg/ml. Surprisingly, a higher coronary calcification score was not associated with lower serum iPTH levels, but only HD patients with evidence of secondary hyperparathyroidism (SHPT) show significant increases in arterial calcium deposition (group D). To our knowledge, this is the first observation in which the association between severe SHPT and cardio-vascular calcification is clearly documented. Nonetheless, we would like to raise a point of discussion, because other studies have shown different results.

In normal renal function postmenopausal women, atherosclerosis and osteoporosis are major causes of morbidity and mortality, and a graded inverse cross-sectional association between the extent of aortic calcification and metacarpal bone mass and density was found [2]. Furthermore, London et al. [3] studied the bone histomorphometry and arterial calcification (AC) score in 58 HD patients. In contrast to the results of this letter, patients with higher AC scores had lower serum iPTH levels and bone histomorphometry, suggestive of low bone activity and adynamic bone disease. Conversely, HD patients either without AC or with lower AC scores had similar serum iPTH levels and bone histomorphometry. In addition, during the recent ERA-EDTA Meeting in Barcelona, Asci et al. [4] showed that coronary artery calcification is associated with low bone activation frequency in 224 prevalent HD patients who underwent bone biopsy.

Interestingly, the pathogenesis of vascular calcification (VC) in chronic kidney disease has been extensively investigated [5]. Nowadays we know that VC is an active process, in which different bone regulatory proteins play a crucial role, because of their ability to induce or inhibit mineral deposition in the vessels. Considering the complexity of the VC process, it is no surprise that the article by Coen et al. [1] showed opposite results, compared to the previous studies.

In conclusion, we fully agree with the authors’ conclusions about the ‘special attention’ to be paid to HD patients with severe SHPT, using correct bath calcium concentrations, reducing oral calcium intake and prescribing vitamin D receptor activators with minimal effects on serum calcium and phosphate levels.

Conflict of interest statement. None declared.

Department of Nephrology Renal Division, S. Paolo Hospital University of Milan Italy E-mail: mariocozzolino@hotmail.com


do: 10.1093/ndt/gfm687

Advance Access publication 8 December 2007

Reply

Sir,

We thank Drs Cozzolino and Brancaccio for their interest in our publication on PTH as a risk factor of coronary calcifications in uraemic HD patients. Some explanations for the apparent inconsistencies of our results with those published in the international literature have already been given in the article. For example, we expressed some reservations on London et al.’s much quoted publication [1] concerning the population cohort they studied. As discussed in our article, quite a number of patients in the London et al. paper had been previously parathyroidectomized; by definition, therefore, they had a past history of secondary hyperparathyroidism. As for the article by Hak et al. [2], I do not feel that their results are at odds with our results. Patients with increased postmenopausal osteoporosis are probably in a relatively increased bone resorption state compared to bone formation, a condition which, similarly to our cases with secondary hyperparathyroidism, may favour calcium phosphate deposition in extraskeletal tissues. Some of the experimental results of Price et al. [3,4] may favour increased bone resorption as a cause of the extraskeletal calcification process. The latter, according to these authors’ experimental results, can be suppressed or prevented by administration of bisphosphonates. As for the abstract presented at the ERA-EDTA 2007 Congress, by Asci et al. [5], I must conclude that the levels of PTH serum levels explored in their study were much lower than in our publication, therefore excluding the cases of severe secondary hyperparathyroidism. Our group with PTH levels >600 pg/ml and significantly higher calcification scores had an average value of intact PTH of 1186 ± 511 pg/ml, while in the Asci et al. study the high turnover group had a value of PTH 343 ± 341 pg/ml. Asci et al. did not explore really severe secondary hyperparathyroidism and for that reason we felt that our data cannot be compared to their study. In addition, in the abstract there is no information about vitamin D and calcium administration in the low turnover patients.

I could add, as stated in our article, that our results are more in line with epidemiological observations [6], suggesting that mortality is increased in patients with elevated PTH values, and considering that cardiovascular mortality