Reply

We thank Dr Grzegorzewka for sharing her results on serum OPG and RANKL levels in dialysis population. With respect to her comments regarding our study, our objective was not to focus on markers, but to use them as a tool for interpreting phosphate elimination. However, we found (results not shown in the manuscript) that OPG serum levels were lower in patients with adynamic disease than in those with hyperparathyroidism \(7.8(2.3) \text{ vs } 18.2(11.3) \text{ pmol/l}, P < 0.004\). There were no statistically significant differences in serum RANKL. Therefore, we agree with Dr Grzegorzewka that OPG is higher in patients with hyperparathyroidism. The problem is that the biochemical diagnosis of bone disease is quite difficult, and it is possible that if groups are separated only by using iPTH concentration below or over 100 pg/ml, no differences are found. In fact, our biochemical measurements tried to identify real differences between individuals using extreme values of iPTH in combination with bone alkaline phosphatase \([1]\).

At the time of writing our manuscript, no study measuring RANKL levels in the haemodialysis population could be retrieved in a Medline search. We found that RANKL values were in the normal range of reference from healthy volunteers or transplant patients. Also, no correlations were found between RANKL and bone activity markers. More studies are needed to explain the differences between Dr Grzegorzewka’s study and our studies. Finally, the positive relationship between OPG and age found in normal individuals is maintained in end-stage renal disease according to our own and many results from others. At present, the mechanisms that regulate OPG secretion are largely unknown and we only can speculate about this mechanism.

Conflict of interest statement. None declared.

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Dermal and extra-dermal manifestations of gadolinium-triggered nephrogenic fibrosing dermopathy

Sir,

The article [1] about gadolinium (Gd)-containing contrast agent as a potential trigger for nephrogenic fibrosing dermopathy (NFD) in renal failure patients raises the following questions:

What was the indication for magnetic resonance imaging?

Did the amount of Gd differ between the patients who did and did not develop NFD? Did the patients with and without NFD differ regarding history of skin problems, allergy, previous administration of Gd, renal function parameters and erythropoetin dosage? Was development of NFD also observed in patients who had not received Gd?

Did the five patients with NFD derive from the same location, and could there be another environmental toxic influence?

NFD has been reported to be associated with muscle abnormalities \([2]\). How many of the patients had muscle involvement? Was anyone seen by a neurologist? Did any of the patients undergo muscle biopsy?

How were new cases observed? Was NFD diagnosed in not Gd-exposed cases? As long as these open questions remain unanswered, Gd as a trigger of NFD remains speculative.

Conflict of interest statement. None declared.

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