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The role of FGF-23 in CKD patients still needs to be clarified

Sir,
I read with interest the article in the Journal by Zoccali [1] on the role of FGF-23 in dialysis patients. In his Editorial Comment to the recent article by Gutierrez et al. [2], the author underlines how we do not have enough proofs to suggest FGF-23 measurement in the routinely practice. I totally agree with this opinion. At the same time, I would like to raise a point of discussion on the potential role of this phosphatonin in CKD.

In fact, advanced CKD patients develop hyperphosphataemia due to impaired renal phosphate (P) excretion. Different hormonal mechanisms have been proposed to understand the P homeostasis regulation. PTH effectively reduces renal P reabsorption and increases renal calcitriol synthesis. The recent discovery of FGF-23 represents a major pathogenetic milestone, even if the role of FGFs 1 to 22 still remains unknown.

Ben-Dov et al. [3] described a new axis for the physiological interactions between parathyroid, kidney, and bone in P homeostasis. In normal parathyroid tissue FGF-23 decreases calcitriol, serum PTH andPTHmRNA levels (the ‘bone–parathyroid axis’). But, does FGF-23 play a different role in physiology and in renal disease? In normal conditions, high FGF-23 causes hypophosphataemia, reduces calcitriol, enhances PTH and causes rickets/osteomalacia. Because FGF-23 is produced by the osteocytes and mediates fundamental actions in the kidney, the new endocrine system was coined: ‘bone–kidney axis’. Probably, the situation is more complicated and the ‘bone–kidney–intestine–parathyroid axis’ works with different pathways under physiological conditions and in CKD. Serum FGF-23 levels are increased and FGF-23 synthesis may be induced by P retention in CKD. Unfortunately, the stimuli driving the elevation of serum FGF-23 in CKD remain unclear.

In conclusion, not only I deeply agree with the author’s conclusions that by now ‘the clinical nephrologist does not need to measure FGF-23 levels in her/his patients’, but I also believe that additional data are necessary to better understand its role in CKD.

Conflict of interest statement. None declared.

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Reply

Sir,
I thank Dr Cozzolino for his letter. The pathway to discovery is an endless enterprise. Almost one century after Tiegerstedt’s discovery of renin we are still amazed by the new knowledge that is almost daily published on this enzyme. FGF23 is a fresh territory and, understandably, here the list of open questions goes far beyond hyperparathyroidism. We will certainly see this growth factor growing in the years to come and look forward to see contributions by Mario Cozzolino and other investigators in this fascinating area.

Conflict of interest statement. None declared.

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Diagnosis of pulmonary tuberculosis among dialysis patients by enzyme-linked immunospot assay for interferon-γ

Sir,
We read with interest the analysis of the usefulness of a whole-blood interferon-γ (INF-γ) release assay-QuantiFERON (QFT) test in the diagnosis of active tuberculosis in dialysis patients [1]. The authors suggested that the QFT test may be a useful supplementary tool for the detection of active tuberculosis among dialysis patients. An enzyme-linked immunospot (ELISPOT, Oxford Immunotec Ltd, Oxford, UK) assay, like the QFT test, was developed to detect IFN-γ produced by activated T cells after exposure to two specific antigens of Mycobacterium tuberculosis, i.e. early secretory antigenic target 6, and culture filtrate protein 10 [2]. Several studies have demonstrated that ELISPOT assays are useful for diagnosis.