Since 1943, when bone metabolic disorders associated with chronic kidney disease (CKD) were described as ‘Renal Osteodystrophy’ [1], the knowledge and scope of this disorder have experienced relevant changes and progress. The growth and the almost worldwide spread of renal replacement therapies (RRT) have largely contributed to the expansion and current daily use of this term among the medical community. As a result of changes in strategy, new active treatments and longer survival in RRT, the term ‘Renal Osteodystrophy’ began progressively gathering several bone-associated mineral disorders, clearly explained in the K/DIGO paper in this issue of NDT [2], expanding its meaning.

The first and classical definition of Renal Osteodystrophy included only a combination of bone histological lesions, such as secondary hyperparathyroidism, osteomalacia, osteosclerosis and osteoporosis [1]. By then, no specific biochemical parameters were available, thus the possibilities to translate this term in clinical practice was difficult. Later, the better knowledge of this disorder, the possibility of more precise measurements of bone metabolic parameters, mainly the availability of parathormone (PTH) assays, and the introduction of undecalcified bone biopsies, were crucial in the understanding and dissemination of the term ‘Renal Osteodystrophy’. The combination of a few biochemical parameters such as serum calcium, phosphate, alkaline phosphatase and PTH, together with the X-ray of specific areas using appropriate techniques and the undecalcified bone biopsy, converted the term ‘Renal Osteodystrophy’ in a meaningful and useful clinical term. In fact, the histological classification of Renal Osteodystrophy in High and Low Bone Turnover forms and its reasonable correlation with PTH has brought the term extensively into use in clinical practice and it has been the main base for the therapeutic management of bone and metabolic disorders associated with CKD during the last two decades.

The first description of ‘Renal Osteodystrophy’ did not include two important associated conditions: vascular calcifications and bone fractures [3]. However, already, these two frequent findings were associated with bone and mineral disorders related to CKD. In fact, since the beginning of the expansion of dialysis as a current RRT, poor control of calcium and phosphate and extra osseous calcifications were described as associated clinical findings. As an example, in 1967, it was mentioned that ‘dialysis patients achieved a better control in several biochemical parameters but they were turning to stone’ [4].

The new definition, evaluation and classification proposed by the K/DIGO initiative is far more complete and covers in one concept all the mineral and bone disorders associated with CKD, such as laboratory and bone abnormalities, vascular calcifications and their hard outcomes; cardiovascular disease, fractures and mortality. That is the main strength of the concept and its great value for the future. However, there might be some practical limitations to spread its use.

First the well and widely used term ‘Renal Osteodystrophy’ is simpler than the new proposed term: ‘Chronic Kidney Disease-Mineral and Bone Disorder’ of the K/DIGO initiative is far more complete and covers in one concept all the mineral and bone disorders associated with CKD, such as laboratory and bone abnormalities, vascular calcifications and their hard outcomes; cardiovascular disease, fractures and mortality. That is the main strength of the concept and its great value for the future. However, there might be some practical limitations to spread its use.

First the well and widely used term ‘Renal Osteodystrophy’ is simpler than the new proposed term: ‘Chronic Kidney Disease-Mineral and Bone Disorder’ and its abbreviation, CKD-MBD. And secondly, there is a possible language barrier in the new terminology. Renal Osteodystrophy is a term that in almost all languages can be expressed using two simple words. On the contrary, the new proposed abbreviation, CKD-MBD, is less meaningful by itself than ‘Renal Osteodystrophy’ and it is difficult to ensure that it makes similar sense in different languages. The concept ‘Chronic Kidney Disease-Mineral Bone Disorder’ includes six words, its translation in several languages will imply the use of different words starting with others letters, thus the English abbreviation ‘CKD-MBD’ may lose part of its meaning and strength. For example, in Spanish the abbreviation of the same words will be ‘ERC-AMO’, far away from CKD-MBD. This simple but important fact can hinder the implementation of the terminology in ‘non English-speaking countries’.

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The K/DIGO, and specifically the “Bone working group”, has made great efforts to evaluate, redefine, classify and update the concept of ‘Renal Osteodystrophy’, a subject highly needed. The position statement from K/DIGO represents an important step forward, to better describe a broader clinical syndrome and a systemic disorder of mineral and bone metabolism secondary to CKD, which implies not only abnormalities in mineral and bone metabolism but also extra-skeletal manifestations. The worldwide adoption of the new recommendations will be useful in clarifying and enhancing international scientific and academic communication in English. However, as mentioned earlier, special efforts must be made in each language to render this new concept in a few and meaningful words, to convert this new terminology into a handy and useful term to replace ‘Renal Osteodystrophy’.

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Contrast media nephropathy—how to diagnose and how to prevent?

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Introduction

An increasing number of individuals are being exposed to iodinated contrast media (CM). This derives from both the technical advances that have enhanced the role of imaging in the diagnostic and therapeutic arena and the changing demographics of the population. There are more elderly individuals with a burden of chronic diseases including hypertension, diabetes, kidney disease and heart disease, for which the tools of the radiologist and interventionalist are particularly appropriate. It is therefore not surprising that increasing attention in the medical literature is being given to the renal adverse effect of CM, contrast-induced nephropathy (CIN) and strategies to minimize its incidence.

Before attempting to synthesize from this literature a reasoned approach to the prevention of CIN, I would like to emphasize the pitfalls of this body of work. To begin with, a uniform definition of CIN does not exist. This is no small issue, as the incidence of CIN can vary 2-fold in the same population depending upon whether one uses an absolute increase in serum creatinine (≥0.5 mg/dl increase) or a relative increase in serum creatinine (≥25% increase) or a combination of the two measured 48–72 h post-CM exposure. When a common definition of CIN is not used, comparisons between clinical trials are very difficult. Second, clinical trials in this area tend to be small and single centre (less than a few hundred patients). They often lump together patients with diverse risk factors, routes of CM administration, different types of CM and different reasons for imaging. Particularly in small trials, attempts to adjust for these confounders often lack sufficient power.

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