



## Nephrology and clinical chemistry: friends for life!

Renal diseases are frequently *pauci*- or asymptomatic. It is not rare that patients with end-stage chronic kidney disease (CKD) or severe acute kidney injury (AKI) are admitted to the emergencies with only recent symptoms, even if, in case of CKD, the pathophysiological process is ongoing for months or years. In this context, one can only underline the fundamental importance of the clinical laboratory to diagnose CKD or AKI. Both blood and urine analyses are of paramount importance to detect, as early as possible, kidney injuries with the objective of preventing and treating kidney diseases. Both guidelines on AKI and CKD management propose definitions for AKI and CKD (1,2). In these situations, serum creatinine remains the mostly used biomarker to estimate renal function. However, serum creatinine is far from perfect both from a clinical and laboratory points of view (3,4). Bargnoux and colleagues extensively reviewed all limitations of serum creatinine. Among these limitations, numerous analytical interferences have been described (5). Also, in the current era of equations to estimate glomerular filtration (GFR), the standardization of serum creatinine takes a long time but is mandatory (6). For both aspects (standardization and interferences), the added value of enzymatic assays needs to be emphasized (6,7). For AKI diagnosis, Makris summarized the apparently simple recommendations, but then, reviewed into details all the difficulties in applying the AKI criteria, especially serum creatinine changes and urine output (2). Ebert and Schaeffner summarized the current creatinine-based equations used to GFR rate. Then, they discuss in their review the potential role for other renal biomarkers. Cystatin C is certainly the most studied until now (8-10), but other avenues of research exist, such as beta-trace protein (11,12) or panel of biomarkers proposed with a metabolomic approach (13). Classical creatinine- and new biomarkers-based equations still remain estimation of GFR, and they all share, more or less, the same degree of imprecision (4,14). In specific situations and/or in specific subjects, a highly precise method is required to measure (not estimate) GFR (15,16). Once again, the clinical laboratory plays a key role in the development of reference methods to measure GFR. Indeed, today, the most frequently used method is the plasma clearance of iohexol. Carrara and Gaspari extensively reviewed this method, insisting both on analytic strengths and safety. Measuring GFR, as a reference method, is probably underused in Nephrology whereas it is an easy and relatively cheap method (16,17). CKD is associated with non-renal complications such as anemia, acidosis and/or hyperkalemia (18). Among these complications, bone disorders are very frequent and represent a particularly complex disease (19,20). The so-called renal osteodystrophy is mainly (but not only) associated with abnormalities in bone turnover (20). Bone biopsy is the gold standard, for example, to make the distinction between low-bone turnover (or adynamic bone disease) and high-bone turnover (or osteitis fibrosa), but this technique is cumbersome, costly and painful. Here again, the role of the laboratory is fundamental with the measurement of parathyroid hormone (PTH) and bone biomarkers (21). Cavalier reviewed which biomarkers are the most useful in the context of CKD and, still more, how they must be interpreted.

The present special issue of *Journal of Laboratory and Precision Medicine (JLPM)* underlines, if needed, the essential links between specialists in clinical chemistry and nephrologists.

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