

Comment on “Eliminating chronic kidney disease... as a diagnosis”

To the Editor I have recently read an article by Prasad and Cifu,¹ in which they presented the concept of eliminating the name “chronic kidney disease” (CKD) as a diagnosis.¹ The authors do not seem to be fully aware how much clinical and epidemiological studies have benefited from the introduction of a separate disease entity of “CKD” and its individual stages. Despite what the authors stated at the beginning of their article, the entity of CKD is as old as the disease itself, and it has always had a broader meaning than just “higher creatinine levels”. Furthermore, the authors state that the diagnosis of CKD itself does not really mean much for the patient because it does not affect the treatment administered by the general practitioner, and moreover, that such diagnosis necessitates an array of complex laboratory and imaging tests as well as consultations that do not clearly benefit the patient.

In my opinion, the authors are not fully aware of the fact that the diagnosis of CKD may lead to profound changes in the patient’s lifestyle, diet, and physical activity, thus slowing down the progression of renal disease. It may also affect pharmacological treatment, especially in the case of comorbidities. Moreover, the diagnosis of CKD has serious implications for diagnostic procedures and treatment if the patient is consulted by physicians who are not nephrologists.

According to the authors,¹ a referral to a nephrologist is not necessary because it does not affect the treatment outcome in any way. I was also surprised by the statement that mortality is not associated with the use of erythropoietin and that nephrologists do not know target phosphate levels in patients with CKD. Moreover, the authors¹ suggested that the use of “CKD” is “disease mongering”, that is, “broadening of diagnosis categories to increase the utilization of medical resources with no evidence of corresponding health benefit”. I think that the authors did not have any contact with CKD patients in the times when erythropoietin was not available or when little was known on calcium–phosphate imbalance. For some reasons, the authors chose not to discuss the beneficial effects of erythropoietin and calcium–phosphate balance on patients’ lives.

In my opinion, the introduction of “CKD” as a disease entity has marked a significant advancement in nephrology (in terms of epidemiological studies, assessment of nephroprotective effects of drugs, and pathophysiological study of CKD of different etiology).

I believe that patients should know that they have been affected by chronic renal disease and they should tell their doctors, whatever the specialty, about the disease. Despite what the authors say, CKD patients should be referred to nephrologists because they have the best experience in the treatment of the disease according to current medical knowledge. The authors seem to be unaware how important it is to introduce an individualized treatment of CKD depending on numerous confounders that have not been included in numerous clinical trials. After all, the results of the trials: Evaluation of Cinacalcet HCl Therapy to Lower Cardiovascular Events (EVOLVE)² and Paricalcitol Capsules Benefits Renal Failure Induced Cardiac Morbidity in Subjects With Chronic Kidney Disease Stage 3/4 (PRIMO)³ have shown that lack of hard evidence does not always downgrade the value of drugs because it may be caused by limitations in study design. The introduction of “CKD” minimizes the risk of unreliable results, especially in cases where they do not correspond to the clinical status of the patient.

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Authors' reply In our original article, we argued that the diagnosis of chronic kidney disease (CKD) should be eliminated.¹ In order for a diagnosis to be valuable, it must lead to changes in treatment that improve patient outcomes beyond what would occur without the diagnosis. We provided an argument detailing why CKD is unlikely to meet this standard. Since drafting our article, other groups have reached similar conclusions based on similar reasoning.²

Dr. Kokot disagrees with our position. He argues that CKD is a useful diagnosis and cites 2 trials to support this claim. First is the EVOLVE study, which found that cinacalcet did not reduce the risk of death or major cardiovascular events among patients with secondary parathyroidism who were on dialysis.³ It is odd that Dr. Kokot cites this trial, as it only included patients who were on dialysis, which was not the subject of our original discussion. Additionally, the EVOLVE trial was a negative study.

Next is the PRIMO study, which included patients with CKD and mild-to-moderate left ventricular hypertrophy. The trial found that treatment with paricalcitol did not improve the primary endpoint of left ventricular mass.⁴ Dr. Kokot hints that this trial may have been positive had it been conducted differently. Such speculation, however, does not change the fact that the trial was negative, nor does it validate CKD as a diagnosis, instead it provides further support for our claim.

Dr. Kokot argues that we are not aware of the benefits of the diagnosis of CKD or prompt referral to a nephrologist. We are not aware of these benefits because they do not exist. Dr. Kokot provides no evidence for said benefits.

Finally, at times, Dr. Kokot mischaracterizes our position. He states we claimed, "nephrologists do not know target phosphate levels." We said no such thing. Instead, we stated that the calcium phosphate product at which point therapy is recommended has been a moving target over the last 2 decades.

In short, Dr. Kokot disagrees with our argument, but fails to offer evidence to rebut any of our claims.

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