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A) COMMENTS ON PUBLISHED ARTICLES

Antidiabetics in chronic kidney disease: new questions to new and traditional drugs

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To the Editor:

After reading the editorial by Martinez-Castelao A et al.,[1] I must congratulate the authors for the clarity and pragmatism of their article.

The topic of chronic kidney disease (CKD), diabetes mellitus, and hypoglycaemic drugs continues to be a source of controversy among nephrologists and doctors from all areas who come into contact with and must make decisions regarding patients with renal failure who require these drugs.

Although the aforementioned editorial removes many of the doubts that may arise for primary care physicians who read the original article,[2] it still leaves certain aspects somewhat unclear that we wish to highlight.

Why do classification systems for CKD continue to be in use when evaluating drugs that are not those proposed by the KDIGO several years ago?

The editorial includes the indications for new and traditional anti-diabetic drugs (Tables 2 and 3) based on the level of altered renal function in the patient, with glomerular filtration rates (GFR) >50ml/min, 30-50ml/min, or <30ml/min, referred to as mild, moderate, or severe, respectively. This classification system that differs from the more commonly used 5 stages established by the KDIGO (soon to undergo review) appear not only in this article, but also in the technical data sheets of several drugs, thus hindering the comparison between studies or protocols in the management of these drugs.

Due to what pathophysiological mechanisms, which are not sufficiently explained, is acarbose contraindicated in stage 4 and 5 patients, given that its mechanism of elimination is <2% renal?

A relatively large body of literature is available regarding the use of metformin, but very little is known regarding the adverse effects of acarbose.

New dipeptidyl peptidase-4 inhibitors and glucagon-like peptide-1 receptor agonists are good resources to use when other drugs are contraindicated, but at times doubts can arise when treating patients with CKD, especially considering the fact that reduced doses for use in patients with CKD are often not available in Spain.

Has a pharmaco-toxic mechanism been isolated based upon which doses should be adjusted in the event of deteriorated capacity for renal elimination?

Should we guide ourselves based on dosage or administration interval based on which drug is being administered?

Although we are approaching a more updated and realistic version of the modern CKD patient with the imminent release of the S.E.N.-semFYC consensus document (and the debate continues whether a decrease in GFR<60 constitutes CKD, as held by the "huge" equation authors[3]), and while the precision of estimates of GFR with cystatin-C alone or combined with serum creatinine is increasing,[4] there are still doubts surrounding aspects of the treatment of this disease that have been around for some time.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

1. Martínez-Castelao A, Górriz JL, Sola E, Morillas C, Jover A, Coronel F, et al. A propósito de las discrepancias entre documentos de consenso, guías de práctica clínica y normativa legal en el tratamiento de la diabetes tipo 2. Nefrologia 2012;32(4):419-26.
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Authors reply: About the discrepancies between consensus documents, clinical practice guidelines, and legal regulations in the treatment of type 2 diabetes

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To the Editor:

In his letter to *Nefrología* published in this issue,[1] J. Serra Tarragon makes a