Letter regarding the article “The impact of hypomagnesemia on erectile dysfunction in elderly, non-diabetic, stage 3 and 4 chronic kidney disease patients: a prospective cross-sectional study”

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Dear editor

In an article published in a recent issue of *Clinical Interventions in Aging*, Toprak et al¹ found that, among patients with stage 3–4 chronic kidney disease (CKD), the prevalence of erectile dysfunction was higher in patients with hypomagnesemia. This finding is clinically relevant because it supports the hypothesis that hypomagnesemia may lead to inflammation² and endothelial dysfunction, two causes of erectile dysfunction. Toprak et al¹ concluded that the detection of the serum magnesium level in non-diabetic elderly men with CKD could be useful to assess the risk of erectile dysfunction. Consequently, it is important to assess the causes of hypomagnesemia in patients with CKD, in particular the causes that are potentially reversible. With this in mind, hypomagnesemia could be caused by the long-term use of proton pump inhibitors,^{2,3} widely used in patients with CKD but not reported in the study of Toprak et al,¹ alone or in combination with diuretics² or cyclosporine.⁴ Hypomagnesemia may also be associated with low levels of parathyroid hormone (slightly reduced in the cohort of patients with hypomagnesemia enrolled by Toprak et al¹), calcemia (not reduced in the cohort of patients with hypomagnesemia enrolled by Toprak et al¹) and kalemia (not evaluated in the study of Toprak et al¹). Moreover, an association between short-term use of proton pump inhibitors and erectile dysfunction has been previously reported.^{5,6} In conclusion, it could be useful to investigate and report, if available, the pharmacological history and serum and urinary cation levels in patients with CKD and hypomagnesemia in order to evaluate whether a proton pump inhibitor or cyclosporine are used and if hypokalemia is present.

Disclosure

The authors report no conflicts of interest in this communication.

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Authors' reply

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Dear editor

We are in full agreement with Fatuzzo et al that it is important to assess the causes of hypomagnesemia in patients with chronic kidney disease.^{1,2} Hypomagnesemia may be caused by malabsorption, inflammatory bowel disease or gastrointestinal disorders, diarrheal diseases, alcohol abuse, use of laxatives, diuretics, corticosteroids, oral contraceptives, bile acid sequestrants, proton pump inhibitors, amphotericin B, aminoglycosides, tetracycline, chemotherapy drugs (cisplatin, amsacrine), immunosuppressants (cyclosporine, sirolimus), bisphosphonates, beta adrenergic agonists, foscarnet, pentamidine, hypokalemia, hypocalcemia, inherited renal tubular defects, and kidney transplantation.²

Proton pump inhibitors are widely used and are one of the causes of hypomagnesemia. In our study, patients' medical histories were reviewed, and comorbidities, risk factors, and medications with the potential to affect erectile dysfunction

and magnesium levels were recorded.³ We recorded all pharmacological history of the patients involved in our study. We checked the use of proton pump inhibitor usage in all patients and a non-significant result was found between the study groups. Fourteen hypomagnesemia patients (7.7%), and 17 normomagnesemia patients (8.8%) used proton pump inhibitors ($P=0.732$). None of our patients used cyclosporine. We also recorded the potassium levels of the patients in our study and found that there was no statistically significant difference between the study groups. The level of potassium was 3.92 ± 0.71 mEq/L in the hypomagnesemia group and 4.11 ± 0.72 mEq/L in the normomagnesemia group ($P=0.123$).

In conclusion, we agree with Fatuzzo et al that the pharmacological history and electrolyte levels are important to evaluate hypomagnesemia in patients with chronic kidney disease.

Disclosure

The authors report no conflicts of interest in this communication.

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