

9. Kröger E, Rohmann H, Osten B, Falkenhagen U. *Serratia marcescens* infections in chronic hemodialysis patients. *Z Urol Nephrol.* 1975;68:657-63.

Silvia González Sanchidrián*, Jesús Pedro Marín Álvarez, Javier Deira Lorenzo, Pedro Jesús Labrador Gómez, Juan Ramón Gómez-Martino Arroyo

Servicio de Nefrología, Complejo Hospitalario Universitario de Cáceres, Hospital San Pedro de Alcántara, Cáceres, Spain

* Corresponding author.

E-mail address: silvia_goz@hotmail.com (S. González Sanchidrián).

2013-2514/© 2017 Sociedad Española de Nefrología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>). <https://doi.org/10.1016/j.nefro.2017.10.001>

Hyperkalemia of non-renal origin in a haemodialysis patient[☆]

Hiperpotasemia de causa no renal en paciente en hemodiálisis

Dear Editor,

Potassium, the primary intracellular cation, is essential in physiological processes such as cell membrane excitability, as well as solute and ion transport. Maintenance of potassium homeostasis is an essential physiological function. Under normal conditions, 90% of dietary potassium intake is absorbed in the small intestine, and an amount equivalent to that absorbed is excreted in the distal tubules of the kidney. The colon's contribution to potassium absorption and excretion is insignificant. The amount of potassium that is excreted in the faeces of a healthy person is close to 10 mmol per day.¹ Multiple studies, the oldest of which were published in the 1960s, demonstrate the importance of maintaining potassium homeostasis through the gastrointestinal tract in patients with end stage renal disease.²

We present the case of a 74-year-old patient with end stage renal disease failure since 1999 likely secondary to a glomerular disease which was not biopsied with a decrease in kidney mass (right nephrectomy due to type I clear-cell papillary renal carcinoma in 2011), who started haemodialysis in November 2011. In 2014, the patient was diagnosed with attenuated familial adenomatous polyposis and underwent endoscopic resection of several polyps. Later on, in September 2015, a subtotal colectomy was performed, due to his high risk of developing colorectal cancer. A resection of the ileum up to the sigmoid colon-rectum and a termino-terminal anastomosis were performed without complications. The patient was discharged a week after surgery. Two weeks after discharge, the patient showed a gradual increase in plasma potassium, with a peak level of 7.1 mEq/l in February 2016 (Fig. 1). A review of the patient's prior venous blood gases revealed that

he had never had metabolic acidosis, that could potentially explain his hyperkalaemia. Moreover, his dialysis suitability parameters were correct and had not changed in the last 6 months (online haemodiafiltration, with convective volumes of 28-30 l/session, Kt/V of 1.6-1.8 and Kts of 56-61).

An interview of the patient about his diet confirmed that he followed a suitable low-potassium diet with an estimated potassium intake of around 30-40 mEq/day. Particular emphasis was placed on an ongoing basis on the importance of maintaining a low-potassium diet. Even so, it was necessary to start treatment with calcium polystyrene sulphonate at a dose of one sachet (15 g) per day, initially on alternating days, then every 24 h. Despite these measures, the patient's potassium

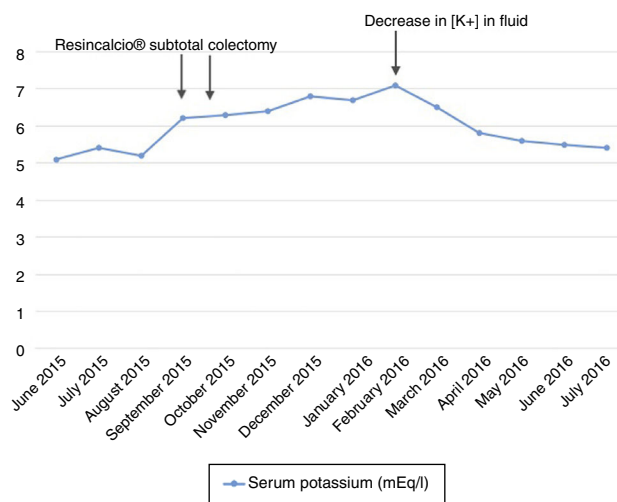


Fig. 1 - Changes over time in potassium laboratory values.

DOI of original article:

<http://dx.doi.org/10.1016/j.nefro.2017.01.005>.

* Please cite this article as: Rodríguez P, Mérida E, Hernández E, Milla M, Praga M. Hiperpotasemia de causa no renal en paciente en hemodiálisis. *Nefrologia.* 2018;38:96-97.

levels remained high. Consequently, the level of potassium in his dialysis fluid was also lowered (from 2 to 1 mmol/l). After this, his potassium levels remained below 6 mEq/l.

A progressive decline in or maintenance of kidney function is associated with an increase in capacity for potassium secretion in the colon. The mechanism through which colon secretion of potassium is increased in these patients is complex and depends on several factors. On one hand, there is an increase in Na⁺/K⁺-ATPase pump activity in the basolateral membrane of the intestinal cell. This leads to an increase in intracellular potassium levels as well as an increase in potassium conductivity in apical cells. Colon secretion of potassium is also increased by aldosterone. Although its mechanism has not yet been fully reported, it appears to be related to an increase in expression of large-conductance potassium channels (BK channels) in the apical membrane of the intestinal cell.³

Konowa et al. published a case⁴ in 2013 on a patient with end stage renal disease undergoing haemodialysis who underwent surgical resection of the ileocaecal junction and a temporary ileostomy. After this, the patient developed hyperkalaemia, which resolved following reanastomosis of the colon. Various *in vitro* and *in vivo* studies have used immunohistochemistry techniques to show that there is greater BK channel expression in patients with end stage renal disease than in those with normal kidney function, in whom small- or intermediate-conductance potassium channel expression predominates.

In summary, potassium secretion in the gastrointestinal tract is a complex and essential mechanism for maintaining its homeostasis in patients with terminal end stage renal

disease. Our case demonstrated the importance of potassium secretion in the colon in patients with end stage renal disease.

REFERENCES

1. Mathialahan T, Maclennan KA, Sandle LN, Verbeke C, Sandle GI. Enhanced large intestinal potassium permeability in end-stage renal disease. *J Pathol.* 2005;206:46–51.
2. Hayes C, McLeod M, Robinson R. An extravenal mechanism for the maintenance of potassium balance in severe chronic renal failure. *Trans Assoc Am Phys.* 1967;80:207–16.
3. Sorensen M, Matos J, Sausbier M, Sausbier U, Ruth P, et al. Aldosterone increases KCa 1.1 (BK) channel-mediated colonic K⁺ secretion. *J Physiol.* 2008;586:4251–64.
4. Konowa N, Dickenman MJ, Jeong Kim M. Severe hyperkalemia following colon diversion surgery in a patient undergoing chronic hemodialysis: a case report. *J Med Case Rep.* 2013;7:1–4.

Paola Rodríguez*, Evangelina Mérida, Eduardo Hernández, Mónica Milla, Manuel Praga

Hospital Universitario 12 de Octubre, Madrid, Spain

*Corresponding author.

E-mail address: pao.rodriguezra@gmail.com (P. Rodríguez).

2013-2514/© 2017 Sociedad Española de Nefrología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).
<https://doi.org/10.1016/j.nefro.2017.01.028>

Denosumab and chronic kidney disease: Severe life-threatening hypocalcaemia[☆]

Denosumab y enfermedad renal crónica avanzada: hipocalcemia severa con riesgo vital

Dear Editor,

A few months ago, we published a case report of asymptomatic severe hypocalcaemia following a dose of denosumab in a patient with advanced chronic kidney disease.¹ We now report a new case, this time one that was life-threatening for the patient. We thought that it important to report this new case

due to its severity and, to show that it does no longer seems to be so rare.

We present the case of a 62-year-old woman whose medical history included slight intellectual disability, diabetes mellitus, long-standing dyslipidaemia and hypertension; and colon neoplasm 3 years earlier. She had been diagnosed of chronic kidney disease likely related to nephroangiosclerosis secondary to hypertension, currently in stage 4. Her regular treatment consisted of olmesartan, repaglinide, insulin glargine, atorvastatin, escitalopram, bromazepam, quetiapine and pantoprazole. Six months earlier, she suffered a hip fracture following an accidental fall and underwent implantation of a total hip prosthesis. The Traumatologists started

DOI of original article:

<http://dx.doi.org/10.1016/j.nefro.2017.01.004>.

[☆]Please cite this article as: Monge Rafael P, Martín de Francisco ÁL, Fernández-Fresnedo G. Denosumab y enfermedad renal crónica avanzada: hipocalcemia severa con riesgo vital. *Nefrología.* 2018;38:97–98.