from the time at which we performed the renal biopsy and a favourable response to steroids resulted in the rapid recovery of renal function (Cr 1.1 mg/dL) with disappearance of the proteinuria (260 mg/day) in only 2 months.

Following the result of the renal biopsy, the haematology department was consulted. Having ruled out an associated chronic lymphoproliferative process, and given the absence of symptoms, we decided to adopt a watch and wait approach with close follow up of the patient's clinical progress. After 9 months, the patient remained asymptomatic, with normal renal function and no proteinuria, with persistence of the monoclonal IgM-kappa component. Although the initial diagnosis suggested a poor short-term prognosis, the absence of systemic involvement seems to have contributed to a favourable outcome.

REFERENCES

1. Cohen LJ, Reenke HG, Laubach JP, Humphreys BD. The spectrum of kidney involvement in lymphoma: a case report and review of the literature. Am J Kidney Dis. 2010;56:1191–620.
2. Pelstring RJ, Essell JH, Kurtin PJ, Cohen AR, Banks PM. Diversity of organ site involvement among malignant lymphomas of mucosa-associated tissues. Am J Clin Pathol. 1991;96:738–45.
3. Isaacson PG, Wright DH. Malignant lymphoma of mucosa-associated lymphoid tissue: a distinctive type of B-cell lymphoma. Cancer. 1983;52:1410–6.
4. Kowalewska J, Nicosia RF, Smith KD, Kats A, Alpers CE. Patterns of glomerular injury in kidney infiltrated by lymphoplasmacytic neoplasms. Hum Pathol. 2011;42:896–903.
5. Stokes MB, Wood B, Alpers CE. Membranoproliferative glomerulonephritis associated with low-grade B cell lymphoma presenting in the kidney. Clin Nephrol. 2002;57:303–9.

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Serratia marcescens bacteraemia outbreak in haemodialysis. Comment on "Serratia marcescens bacteraemia outbreak in haemodialysis patients with tunnelled catheters due to colonisation of antiseptic solution. Experience in 4 hospitals"☆

Brote de bacteriemia por Serratia marcescens en hemodiálisis. Comentario a «Brote de bacteriemia por Serratia marcescens en pacientes portadores de catéteres tunelizados en hemodiálisis secundario a colonización de la solución antiséptica. Experiencia en 4 centros»

Dear Editor,

We read with particular interest the article published recently by Merino et al., entitled "*Serratia marcescens* bacteraemia outbreak in haemodialysis patients with tunnelled catheters due to colonisation of antiseptic solution. Experience in 4 hospitals",[1] to which we would like to contribute our experience.

As reported in the aforementioned article,[1] between December 2014 and January 2015, we recorded several cases of catheter-related bacteraemia (CRB) due to *Serratia marcescens*

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(*S. marcescens*) in our haemodialysis (HD) unit. On 19 December 2014, the Spanish Agency of Medicines and Medical Devices (AEMPS) ordered the withdrawal from the market of BohmClorh® chlorhexidine 2% aqueous antiseptic solution for healthy skin as a result of the contamination of several lots with *S. marcescens*.[2] Several subsequent warnings were issued extending the restriction of its use to the remaining lots and presentations and, finally, on 9 January 2015, the master formulae developed by the company were withdrawn.[3]

Between 1 December 2014 and 16 January 2015, we recorded 14 cases of blood culture-confirmed CRB due to *S. marcescens*, 26.9% of the patients in our unit; all had been or were catheter users.

Suspicion of an epidemic outbreak arose after the appearance in the same week of 4 cases of bacteraemia caused by a microorganism that was unusual in the HD unit, in patients on different shifts, with differing dialysis stations and healthcare staff.

The Medical Prevention Service was informed and the corresponding protocol was carried out, reviewing the asepsis measures for catheter handling, and investigating the unit consumables and non-consumables and healthcare staff as possible foci and vectors of transmission.

A total of 9 men and 5 women with mean age 64 ± 20 years were affected. Twelve patients (85.7%) were catheter users: 11 had a tunnelled catheter while only one had a temporary catheter; the remaining two recently had temporary catheters. A total of 26 episodes were recorded (mean of 1.85 episodes per patient), considering these as the onset of general malaise, hypotension and fever during the dialysis session, with blood cultures positive for *S. marcescens*. Two patients had up to 4 episodes of bacteraemia, one patient had 3 episodes, 4 patients had 2 episodes, and 7 patients had a single episode before the causal agent was identified. The catheter had to be removed in 5 patients, all of who were found to have infection with the same epidemic agent. Half the patients required hospitalisation for sepsis, with an average stay of 3.3 days. There were no deaths. One patient died due to CRB associated with a different microorganism.

In the early cases, patients were empirically administered intravenous vancomycin (1 g post-dialysis) and gentamicin (at a dose of 1 mg/kg/weight post-dialysis) until the blood culture results became available. Ciprofloxacin treatment was then instigated in all patients according to antibiotic susceptibility testing (AST), and the catheter was locked with the same antibiotic solution; one case required carbapenems due to poor clinical progress, and no antibiotic resistance was noted in the AST.

Pharyngeal and perianal exudates were systematically collected from all patients in the unit. Only one of the patients was a pharyngeal carrier. In two of the controls, the pharyngeal exudate was positive for *S. marcescens*, but for a strain other than the original one in the outbreak.

There were cases in other hospital units and other Spanish hospitals.[4,5] Once the outbreak had been notified by AEMPS, the antiseptic product was cultured and confirmed as the reservoir of the epidemic. No further cases were recorded after the contaminated lots had been withdrawn.

Most CRB in HD patients is caused by Gram-positive bacteria.[6] *S. marcescens* is a gram-negative bacillus found predominantly in humid conditions; it is pathogenic for man and causes outbreaks of resistant infections, especially in immunosuppressed patients.[6]

The general rule in CRB is to remove the catheter, but conservative treatment is accepted in HD patients despite evidence of serious infection, as a measure to preserve the vascular access.[1,7]

In our experience, AST-directed treatment and catheter locking provided a good outcome, although the delay in identifying the source and probable formation of *biofilms* meant that a significant number of catheters had to be removed due to the high percentage of recurrences.

Although the finding of *S. marcescens* in HD units is rare, and identification of the reservoir in an antiseptic product is paradoxical, outbreaks have previously been described.[8,9] A methodological search for the causal agent and appropriate preventive strategies should therefore be instigated at an early stage when an epidemic outbreak is suspected. An initiative to record the experience in the HD units of all the affected hospitals would be interesting.

REFERENCES

1. Merino JL, Bouarich H, Pita MJ, Martínez P, Bueno B, Caldés S, et al. Brote de bacteriemia por *Serratia marcescens* en pacientes portadores de catéteres tunelizados en hemodiálisis secundario a colonización de la solución antiséptica. Experiencia en 4 centros. Nefrologia. 2016;36:667–73.
2. Agencia Española de Medicamentos y Productos Sanitarios. Nota informativa AEMPS: Retirada del mercado del antiséptico de piel sana BohmClorh solución acuosa 2% de clorhexidina, 250 ml [online]; 2014 [accessed Nov 2016]. Available from: http://cima.aemps.es/va/informa/notasInformativas/cosmeticosHigiene/seguridad/2014/COS_02-2014-Bohmclorh.htm
3. Agencia Española de Medicamentos y Productos Sanitarios. Nota informativa AEMPS: Retirada del mercado de las fórmulas magistrales a base de clorhexidina fabricadas por laboratorios Bohm, S.A. a partir del 8 de octubre de 2014 [online]; 2014 [accessed Nov 2016]. Available from: https://www.aemps.gob.es/informa/notasInformativas/cosmeticosHigiene/seguridad/2015/COS_01-2015-clorhexidina.htm
4. De Frutos M, López-Urrutia L, Domínguez-Gil M, Arias M, Muñoz-Bellido JL, Eiros JM, et al. Brote de *Serratia marcescens* producido por clorhexidina acuosa al 2% contaminada. Enfer Infecc Microbiol Clin. 2016, http://dx.doi.org/10.1016/j.eimc.2016.06.016.
5. Sistema Nacional de Vigilancia Epidemiológica. Brote por *Serratia marcescens* asociado a la utilización de un antiséptico de clorhexidina contaminado. Boletín Epidemiol Sem. 2016;24:85–101.
6. Aguinaga A, del Pozo JL. Infección asociada a catéter en hemodiálisis: diagnóstico, tratamiento y prevención. NefroPlus. 2011;4:1–10.
7. Krishnasami Z, Carlton D, Bimbo L, Taylor ME, Balkovetz DF, Barker J, et al. Management of hemodialysis catheter-related bacteremia with an adjunctive antibiotic lock solution. Kidney Int. 2002;61:1136–42.
8. Pereira BJ, Unnykrishnan P, Prasad KS, Macaden R. An epidemic outbreak of *Serratia marcescens* septicemia in a hemodialysis unit. Ren Fail. 1990;12:121–3.

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

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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

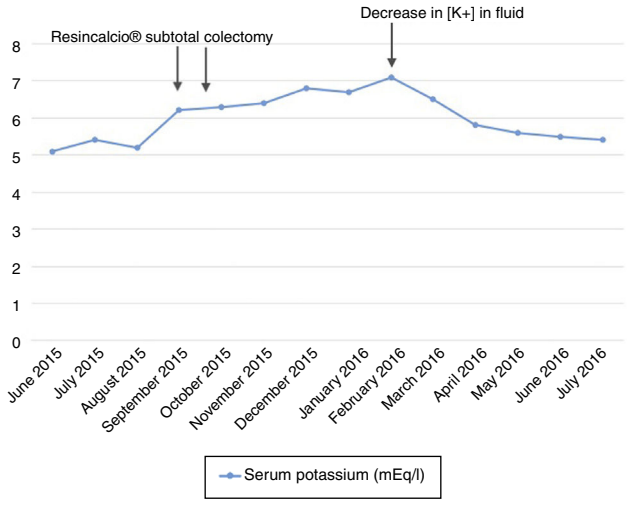

Fig. 1 – Changes over time in potassium laboratory values.

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