cases of peritonitis in dialysis patients, since it can grow in glucose-rich media (gardener's peritonitis?)[3–5] and in carriers of invasive devices, in the paediatric population it can cause sepsis[6] and it has also been cultivated in bile samples from patients with cholangitis and choledocholithiasis.[7]

The patient was kept on tobramycin treatment for 14 days, showing an excellent sensitivity to cephalosporins, aminoglycosides and ciprofloxacin.

Following this new peritonitis episode, after identifying errors in performing the exchange during retraining sessions and due to his advanced age, we decided to transfer the patient to carer-assisted automated peritoneal dialysis.

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Calcium mass balance with citrate dialysate is lower than with acetate☆

El balance de calcio es menor con un líquido de diálisis con citrato que con acetato

Dear Editor,

The topic of what is the ideal calcium concentration in dialysis fluid (DF) is currently on the agenda of most scientific dialysis meetings. With a calcium concentrations of 1.25 mmol/L, haemodialysis tolerance worsens, and with calcium levels of 1.5 mmol/L, positive calcium balance together with the development of alkalemia at the end of the hemodialysis session may favour vascular calcification. Dealing with this issue is not easy because the concept of "balance" (concentration measured before and after the haemodialysis session) is interpreted as the theoretical "gradient" that is not measured nor calculated. The reason for this letter is to try to clarify these concepts and to provide some data.

Calcemia usually increases significantly during the haemodialysis session using acetate DF with a calcium concentration of 1.5 mmol/L, this is more evident if the initial serum calcium concentration is low.[1,2] If the acetate of the DF is replaced by citrate, the increase in the concentration of both total and ionic serum calcium after hemodialysis or online haemodiafiltration (OL-HDF) is less or or even null.[3–5] It could be thought that by using a DF with 1.5 mmol/L of calcium, the smaller increase in calcaemia at the end of the citrate session generate a positive calcium gradient from the DF to the blood, which would induce a positive balance for the patient, which would cause the patient's calcification; or, it could be interpreted that the smaller increase in calcemia observed with citrate is the result of a lower, or even negative, calcium balance.

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Table 1 – Total calcium and ionic calcium concentrations measured in the dialysis fluid at the dialyser entry point.

	No.°	Average Ca++ (mmol/L) (DE)	Average total Ca (mg/dL) (DE)	Theoretical Ca in concentrate (mg/dL)	Total Ca – theoretical Ca (mg/dL)
Citrate DF	7	1.05 (0.156)	6.34 (0.097)	6.5	−0.16
Acetate DF	13	1.22 (0.102)	5.84 (0.18)	6	−0.16
p		0.04			NS

On 7 occasions, the DF contained 1 mmol/L of citrate and 3.25 mEq/L of calcium, and on 13, 3 mmol/L of acetate and 3 mEq/L of calcium. The same dialysis monitors (AK200 Us (Baxter®)) were used.

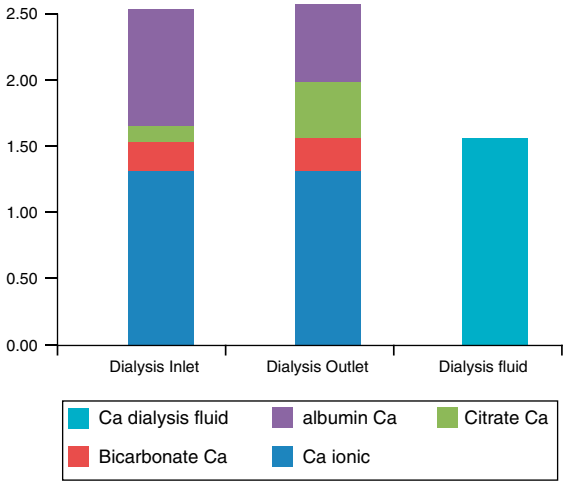

Fig. 1 – Concentrations of calcium in the blood (mmol/L) entering and leaving the dialyser, with the different components. Ionized calcium plus that bound to citrate and bicarbonate form the diffusible calcium in blood; all DF calcium is diffusible. Note that the concentration of blood-diffusible calcium is greater than that of the DF leaving the dialyser.

The work of Steckiph[6] compares the calcium balance in OL-HDF using a DF with citrate vs acetate, both with the same calcium concentration. It was a cross-sectional, randomized study in 18 patients; calcium and citrate balance were measured in the entire collection of DF. The calcium concentration in DF during the 2 periods was the same: 1.5 mmol/L. The result was that the mean calcium balance at the end of OL-HDF was −274 (260) mg with DF containing citrate and +125 (174) mg with acetate DF.

So what is the true calcium gradient in the dialyser? The gradient is not the same across all capillaries of the dialyser and changes during the dialysis session. Further, the gradient is established as a function of diffusible calcium, which includes the ionized calcium and the bound calcium, which is essentially bound to bicarbonate and citrate present in both DF and blood.

With DF containing citrate, the blood entering a dialyser capillar at the beginning of the session has a low concentration of both citrate and calcium-citrate complexes (Fig. 1). At the end of the capillar, the citrate concentration increased from 0.1 to 1 mmol/L and the calcium -citrate complexes are up to 0.3–0.4 mmol/L[7] since citrate is being transferred from the DF. This calcium that is now bound to the citrate comes mainly from that bound to albumin, but also from free ionic calcium. At the end of the dialyser, the total blood-diffusible calcium has increased. This is not the case with the DF with acetate, which has a much lower affinity for calcium than citrate.

The concentration of calcium in the DF is different from the theoretical concentration. Table 1 shows the actual measurements of calcium in the DF with both citrate and acetate; the total calcium concentration is 0.16 mg/dL (0.04 mmol/L) less than the theoretical concentration. This is explained by the fact that, in the hydraulic circuit of haemodialysis machines, there is always a certain degree of microprecipitation, which is more pronounced if both bicarbonate and calcium levels are increased. The DF with citrate has a significantly lower ionic calcium concentration, due to the calcium-citrate complexes; therefore, the greater concentration of diffusible calcium in the blood and the lower concentration in the DF explain a lower Calcium balance with citrate than with acetate, as demonstrated by those who have measured it.[6]

It is difficult to know the "gradients" of calcium during haemodialysis. Changes in iPTH variations during the dialysis session is an indirect manner to assess changes in calcaemia. In publications on DF with citrate,[3–5] even with calcium concentrations of 1.65 mmol/L, the iPTH increases or remains unchanged, but such changes in concentration may not be identical to the balance.

Thus, we believe calcium balances should be calculated using the entire DF, including the ultrafiltration. Baxter™ recommends increasing the calcium concentration in the DF with citrate by 0.15 mmol/L compared to the DF with acetate in order to maintain the same balance.[7] It could be that the DF with citrate with 1.5 mmol/L is an intermediate solution between the 1.25 and 1.5 mmol/L with acetate.

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Sudden death in patients with advanced chronic renal disease☆

Muerte súbita en pacientes con enfermedad renal crónica avanzada

Dear Editor,

We have read the article by Caravaca et al. entitled "Sudden death in patients with advanced chronic kidney disease"[1] and we would first like to congratulate the authors.

It is a subject of great interest among both kidney failure patients and the general population, which is a cause for social concern and requires a complex approach.

We believe its complexity is based on several factors. The first is the definition of sudden death (SD) itself. The one used by the authors is the most widely accepted and includes natural death occurring within an hour of the onset of symptoms, although they also include unexplained death during sleep. The latter definition can be assimilated to those that propose other time intervals (2, 6 and 24 h) between the onset of symptoms and death for specific circumstances such as unwitnessed death, which reduce the probability of heart-related death the longer the time that elapses between death and the onset of symptoms.[2] This variability in the time period in question is one of the factors that contribute to the differences found in the prevalence of SD in different studies. Caravaca et al. found an annual incidence of 1.8%, not including that of death during sleep. In a study conducted at our hospital on a dialysis population, the annual incidence was 1.7% when we considered an interval of one hour, and 2.9% when the interval was extended to 24 h.[3] The lack of autopsies in the Caravaca et al. study, as in ours (something that was particularly inexplicable for the reviewers of the American journal in which it was published), poses many obstacles when it comes to establishing the cause of death. In a Japanese study in which autopsy was performed on 81.4% of patients who had SD, strokes and aortic dissection accounted for more than 45% of the causes of death.[4]

Another aspect to be taken into account when analysing SD is the need to search for those factors that identify the population at greatest risk in order to come up with targeted and profitable prevention strategies. Caravaca et al. found through Cox regression analysis that greater age and greater comorbidity were associated with a higher probability of SD, and the use of antiplatelet therapy had a protective effect. In our study,[3] with the same analysis, we identified a combined variable that included a documented history of coronary disease and electrocardiographic abnormalities (abnormal Q waves, subendocardial lesion, negative T-wave and QRS >120 ms) which clearly identified two risk groups. Patients who had none of the aforementioned findings had almost the same probability of SD as the general population; however, those who had one of them had a probability of SD similar to that shown by patients in the general population with left ventricular dysfunction and who have been included in studies demonstrating the efficacy of the automatic implantable defibrillator.[5,6] The lack of availability of electrocardiography records in the Caravaca et al. study, which the authors point out as a limitation, makes it impossible

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