Peritoneal dialysis as a perpetuating cause of hyponatremia☆

La diálisis peritoneal como causa perpetuadora de hiponatremia

Dear Editor,

Hyponatraemia is one complication of peritoneal dialysis[1–4] which has been referenced as associated with the retention of osmolytes (icodextrin), hyperglycaemia, catabolic states, intracellular potassium or phosphorus loss[5] and ultrafiltration failure.[6] A positive correlation has been demonstrated between the loss of sodium and the ultrafiltrate volume.; thus adjusting salt intake to said volume is therefore recommended.[3] This aspect of peritoneal dialysis can complicate clinical recovery from hyponatraemia.

We present the case of a 28-year-old woman with ESRD on peritoneal dialysis, (CAPD [continuous ambulatory peritoneal dialysis]) and rheumatoid arthritis, receiving treatment with prednisone. She arrived for consultation due to vomiting and diarrhoea that had persisted for a week. Her regular peritoneal dialysis regimen included two 1.36% glucose exchanges (4-h dwell time), one exchange with 2.27% glucose (6-h dwell time) and a fourth exchange with icodextrin (7.5%). With a CAPD she achieved a daily negative balance of around 1900 ml. Residual diuresis was counted at around 200 ml per day. The analytical control demonstrated evidence for hyponatraemia (Na: 119 mEq/l), hypochloraemia (Cl: 78 mEq/l) and hypokalaemia (K: 3.2 mEq/l). Hydroelectrolytic reposition was begun with physiological saline supplemented with potassium chloride and the CAPD regimen was modified, eliminating the exchange with icodextrin and substituting it with a 1.36% glucose exchange. The three exchanges at 1.36% were maintained with a dwell time of 5 hours. Dwell time for the 2.27% glucose exchange was 9 h. Microbiological studies were negative. Symptomatology was self-limited to the few hours after admission, during which volume reposition was completed and potassium levels were normalised, although there was no evidence that the hyponatraemia was resolved. The sodium sieving level was 15%. During the study, it was observed that, at the end of the dwell time for a 1.36% glucose exchange, the ratio between the sodium concentration of the peritoneal dialysate effluent and that of the plasma was 100%, which meant that, based on the plasma sodium concentration at 122 mEq/l, the final sodium concentration in the dialysate effluent was 122 mEq/l. The final exchange volume was 2300 ml. The initial sodium concentration from the exchange was 134 mEq/l, which would have corresponded to a theoretical final concentration of 116.5 mEq/l. This discrepancy between the real value (122 mEq/l) and the theoretical value (116.5 mEq/l) pointed to a negative sodium balance and the exchange dwell time as the perpetuating causes of hyponatraemia. The next day, starting with a plasma sodium concentration of 120 mEq/l, the exchange dwell time was limited to 3 h and the plasma sodium increased to 124 mEq/l.

This case emphasises the importance of the influence of the peritoneum in sodium homeostasis in patients on peritoneal dialysis, going beyond the concept of sodium sieving in order to evaluate ultrafiltration failure. Free water transport through aquaporins (AQP1) induced by the osmolality of the peritoneal fluid is assessed by sodium sieving, which is considered to be reduced if a decrease of less than 5 mEq/l of sodium in the peritoneal fluid occurs (glucose concentration at 3.86%) after 60 min of infusion.[7] In our patient, who maintained normal sodium sieving, the comparison between the theoretical sodium concentration in the dialysate effluent and the actual measurement at the end of the dwell time pointed towards the aetiology of persistent hyponatraemia. Although some concepts have still not been elucidated with respect to peritoneum physiology, adjusting peritoneal dialysis regimens to the serum sodium concentration may prevent the perpetuation of hydroelectrolytic imbalance. The theoretical/real sodium ratio with respect to dialysate volume upon completion of the exchange dwell time could be used to quickly evaluate the tendency of the sodium balance in peritoneal dialysis.

REFERENCES

1. Bargman J. Non-infectious complications of peritoneal dialysis. In: Khanna R, Krediet R, editors. Nolph and Gokal's textbook of peritoneal dialysis. New York: Springer; 2009. p. 571–609.

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2. Kang SH, Cho KH, Park JW, Yoon KW, Do JY. Characteristics and clinical outcomes of hyponatraemia in peritoneal dialysis patients. Nephrology. 2013;18:132–7.
3. Uribarri J, Prabhakar S, Kahn T. Hyponatremia in peritoneal dialysis patients. Clin Nephrol. 2004;61:54–8.
4. Zanger R. Hyponatremia and hypokalemia in patients on peritoneal dialysis. Semin Dial. 2010;23:575–80.
5. Cherney DZ, Zevallos G, Oreopoulos D, Halperin ML. A physiological analysis of hyponatremia: implications for patients on peritoneal dialysis. Perit Dial Int. 2001;21:7–13.
6. Port F, Young E. Fluid, electrolyte disorders in dialysis. In: Kokko J, Tannen R, editors. Fluids and electrolytes. Philadelphia: W.B. Saunders; 1996. p. 533–60.
7. Gómez AM, Pérez M, Rodríguez A, Sastre A, Díaz E, López A, et al. Categorization of sodium sieving by 2.27% and 3.86% peritoneal equilibration test. A comparative analysis in the clinical setting. Nephrol Dial Transpl. 2009;24:3513–20.

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Kidney failure caused by tubular intersticial nephropathy with Fanconi syndrome after treatment with zoledronic acid☆

Insuficiencia renal por nefropatía tubulointersticial con tubulopatía proximal tipo Fanconi tras tratamiento con ácido zoledrónico

Dear Editor,

The relationship between kidney disease and bisphosphonates is illustrated in the medical literature[1]: the vast majority of cases involve glomerulonephritis and acute tubular necrosis. We discuss a case of tubulopathy related to the administration of zoledronic acid. This association has been described, although infrequently.

80-Year-old man with COPD and dyslipidaemia treated with inhaled indacaterol and aclidinium bromide, oral atorvastatin and omeprazole, who presented with acute kidney failure in October 2013 due to obstructive uropathy. He was diagnosed with Gleason score 10 prostate cancer and, after placement of urinary catheter, he recovered his baseline glomerular filtration rate (Cr: 1.1 mg/dl). An extension study showed osteoblastic metastasis in the iliac wing. He was treated by a transurethral resection of the prostate and hormonal therapy (bicalutamide and leuprolide), to which intravenous zoledronic acid was added at a dosage of 4 mg per month from November 2013. His glomerular filtration rate progressively deteriorated and he developed hypophosphataemia, hyperphosphaturia, hypouricaemia, hyperuricosuria, non-nephrotic range mixed proteinuria, metabolic acidosis with normal anion gap, and glycosuria with aminoaciduria, which was the reason for consultation. His accumulated dose of zoledronic acid was 116 mg over 29 months. With these findings, he was diagnosed with kidney failure due to interstitial nephritis with Fanconi syndrome. The zoledronic acid was discontinued and substituted by denosumab. After 6 months of follow-up, the aminoaciduria and glycosuria disappeared, the uricosuria was reduced and the phosphataemia was normalised. Proteinuria continued to decrease, however, he only partially recovered glomerular filtration rate (Fig. 1).

Bisphosphonates are bene anti-reabsorbing agents used to treat corticosteroid-induced post-menopausal osteoporosis and to prevent pathological fractures in Paget's disease, prostate or lung cancer and multiple myeloma. Strong biphosphonates have a nitrogenous chain in their structure. They may be administered orally or intravenously. In the former, they do not tend to induce nephrotoxicity. Intravenous administration involves circumventing the hepatic metabolism, interaction with the P450 enzymatic system and its unaltered excretion in the kidney, through glomerular filtration

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