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HYPONATREMIA SECONDARY TO A SODIUM DEFICIT IN PATIENTS ON CONTINUOUS OUT-PATIENT PERITONEAL DIALYSIS

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INTRODUCTION

Hypotonic hyponatremia is an electrolytic disorder which can be caused by different mechanisms such as: an excess of body water and/or a deficit of sodium and/or potassium[1-2].

In patients on continuous out-patient peritoneal dialysis, hyponatremia can also be caused by some of these mechanisms and that, secondary to an excess of body water is the most frequent due to a failure in ultrafiltration[3].

In this report, we describe a clinical case of a patient who developed hyponatremia, while under continuous ambulatoy peritoneal dialysis, due to a deficit of sodium in the body secondary to a negative balance of sodium: body deficit of sodium.

CASE REPORT

58 year old patient with a history of:

- Arterial hypertension on a hyposodic diet (4 gr/day). Since the beginning of the dialysis treatment no antihypertensive drugs were required.
- Chronic kidney failure on peritoneal dialysis for one year with residual diuresis of 100 cc/day and a dialytic scheme of 4 volumen exchanges: 1700 cc, concentration: 1.5 % (x4) y 2.3% (x1), time of permanence: 6 hours, obtaining an ultrafiltration rate of 1200 cc/day. This was enough for an adequate doses of dialysis: weekly Kt/V: 2.00

In the routine monthly tests it was possible to observe asymptomatic hyponatremia (128 mmol/l), with normal glucemia, haemogram, proteinogram and lipidogram. At the time of the physical exam she had normal blood pressure, without pulmonary edemas or crackles. She was indicated hydric restriction of 800 cc/day and was instructed to return for a visual check-up in 48 hs. Nevertheless, in that check-up she was found to be thinner, more hypotense and not having recovered from her hyponatremia. She was not on any medication which could potentially induce hyponatremia (psychiatric, opioids and antiepileptic drugs).

Since the performed studies: encephalon, thorax and abdominal CT scans, and hormonal dosages (cortisol, thyroid hormone) showed normal values, then we decided to ask her to return to her normal hydration intake level (similar to the sum of urinary volume and peritoneal ultrafiltration), but also to start ingesting 6 gr/day of sodium. When after 48 hours she was re-assessed, the patient was already normotense and her natremia had increased to 138 mmol/l.

DISCUSSION

As it is clearly expressed by the Edelman ecuation:

$$\text{Natremia (mmol/l)} = \frac{\text{total body sodium + total body potassium}}{\text{Total body water}}$$

Hyponatremia can be induced by an increase in body water, as well as, a decrease in its content of sodium and/or potassium. It is already known that hyponatremia secondary to a disbalance of water/sodium, is caused by the alteration of the relationship of sodium/water which natremia represents.

In the case of hyponatremia secondary to a decrease in body potassium, it could be brought about as a consequence of adding sodium (cation) to the intra-cellular compartment, in compensation for the missing potassium (cation), to maintain electroneutrality of the intracellular medium. As a consequence of this, the concentration of sodium in the intravascular compartment would decrease and so would natremia [1-2]. Something similar would happen in states of malnutrition, where even in the case of normal kalemia, the intracellular deficit of potassium would induce hiponatremia due to this mechanism[3].

Hyponatremia in the patient on peritoneal dialytic treatment can be caused as a consequence of a disbalance in the sodium-water relationship in favor of the latter in relation with the quantity of water and salt ingested by the patient, as well as that excreted through urinary and peritoneal secretion (if it is preserved). It must be taken into account that during peritoneal dialysis sodium goes through the plasma to the peritoneal cavity due to the forces of diffusion and convection. However, as sodium is relatively sieved by the peritoneal membrane, the peritoneal fluid is usually hypotonic, which means it is richer in water than in salt, which in theory fosters the appearance of hypernatremia, especially in cases of short term permanence, as it happens in the case of the automatized case, if it weren`t for the release of vasopressin which stimulates thirst and the intake of fluids, thus normalizing the patient´s natremia.

Hyponatremia has been described in patients on peritoneal dialysis in the context of malnutrition, children fed with hyposodic formulas, hyperglucemia (dilucional hyponatremia) and using icodextrin. In the latter, osmotic retention of active metabollic derivatives in the intravascular would induce the passage of water from the intracellular and thus hyponatremia[4-7].

Hyponatremia is usually caused by an excess of body water; nevertheless when hyponatremia is secondary to a deficit of body sodium it has been described in very particular clinical situations such as: salt-losing interstitial nephritis, cerebral salt wasting syndrome and senile salt wasting syndrome[8-10]. In this report we describe a clinical case where the presence of hyponatremia is explained by a negative balance of sodium due to a low diet intake of this cation (hyposodic diet) and a relatively high and sustained egress of such (dialysed sodium), in the context of a hydric balance (ingested water – water excreted in urine and dialysis) neutral. It is for this reason, we observed that this hyponatremia did not improve when restricting the hydric intake, but increased sodium one.

CONCLUSION

In this report we have documented a case of hyponatremia secondary to a sodium deficit in a patient on continuous out-patient peritoneal dialysis.

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