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SODIUM LOSS INDUCED HYPONATREMIA IN THE ELDERLY ON A LOW SODIUM DIET

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RESUMEN
La hiponatremia es un desorden electrolítico que consiste en un aumento relativo a nivel corporal del contenido acuoso respecto del salino.

La retención de agua libre o la pérdida de sodio son sus mecanismos causales principales, siendo el primero de ellos el más frecuente. Sin embargo, dado que los ancianos normales poseen un aumento en la excreción urinaria de sodio respecto de los jóvenes, pueden desarrollar hiponatremia secundaria a deplección salina cuando han estado en forma prolongada bajo una dieta hiposódica.

En este reporte presentamos un caso de hiponatremia que fue resuelto mediante la restitución de sodio y no mediante la restricción hídrica. Analizamos también la baja confiabilidad de la excreción fraccional de urea en la interpretación de la etiología de la hiponatremia en los ancianos.

Palabras clave: hiponatremia, pérdida urinaria de sodio, anciano

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ABSTRACT
Hyponatremia is an electrolyte disorder which consist of a relative increase in body free water

content respect to sodium one. Free water retention or sodium loss are its possible mechanisms, being the former more frequent than the latter. However, since urinary sodium loss is increased in healthy elderly, sodium depletion can be a frequent etiology of hyponatremia in this people when they have been on a low sodium diet for a long period.

In this report a case of hyponatremia that solved with sodium restitution instead of water restriction is presented, and the name of senile sodium leakage hyponatremia is proposed for this entity. It is also analyzed the low reliability that fractional excretion of urea seems to have in the evaluation of this syndrome.

Keywords: hyponatremia, urinary sodium loss, elderly

INTRODUCTION

Hyponatremia is an electrolyte disorder which consist of a relative increase in body free water content respect to sodium one. Either free water retention or sodium loss are possible mechanisms of hyponatremia, being the former more frequent than the latter[1].

Since hyponatremia is one of the most frequent electrolyte disorders in the elderly[2], and urinary sodium loss is usually increased in this population [3], sodium depletion can be a frequent mechanism of hyponatremia in the aged people on low sodium diet for a long period. In the present report we present a clinical case which is a good example of a senile sodium loss induced hyponatremia since it solved with sodium restitution instead of water restriction.

CASE REPORT

An Eighty years-old, nursing-home patient who had the following antecedents:

- **Immobility syndrome secondary to severe Alzheimer dementia and parkinsonism.**
- **Gastrostomy-feeding due to altered deglutition mechanism.**

He was admitted in our hospital presenting an acute febrile syndrome. Blood and urinary samples for laboratories and cultures were taken.

Hypotonic hyponatremia (124 mmol/l) and a positive urine culture to *streptoccocus faecalis* were the main laboratory findings. Urinary infection was treated with intravenous ampiciline.

He was taking no hyponatremia inducing drugs, and he had a high fractional excretion of urea (FEU: 83%), with normal extracellular fluid on examination (neither hypotension nor edema), plasma potassium (3.7 mmol/l) and renal function markers (plasma creatinine: 0.8 mg/dl and plasma urea: 30 mg/dl). Then, hyponatremia was interpreted as induced by a syndrome of inappropiate antidiuretic hormone secretion (SIADH), and free water restriction was started. This therapy practically did not modify his plasma sodium level (125 mmol/l) while FEU became lower (FEU: 22%), meaning this fact that despite water restriction had not been effective to solve the hyponatremic state, it had induced renal urea reabsorbtion, proving that water restriction had been reached (Table 1)

TABLE 1: Main laboratory data

	PNa	FEU	FENa
Day 1	124	83	3.3
Day 2	125	59	3.3
Day 4	125	22	2.3
Day 7	127	49	3.4
Day 15	136	64	6.5

PNa: plasma sodium (mmol/l), FEU: fractional excretion of urea (%), FENa: fractional excretion of sodium (%)

Since there was not improvement in the hyponatremia after four days of water restriction, a change in the therapeutical strategy was done: fluid restriction was stopped and gastrostomy feeding was reiniciated adding sodium supplements (4 g/day).

After that plasma sodium normalized while fractional excretion of urea and sodium increased, reflecting an augment in salt and water apport. No clinical signs of water and salt overload were detected (Table1).

DISCUSSION

Edelman equation describes the variables that determine plasma sodium level[4]:

$$\text{Plasma sodium} = \frac{\text{(body sodium + body potassium)}}{\text{body water}}$$

This formula shows that the physio-pathological inducing hyponatremia mechanisms can be:

A) High body water content: In this situation the body water/body sodium relation is modified in favour to the former, for instance water retention due to SIADH.
B) Low body sodium content: In this situation the body water/body sodium relation is modified against the latter, for instance sodium loss due to an interstitial nephritis.
C) Low body potassium content: This situation stinulates sodium - potassium interchange between the cells and the intravascular compartment: sodium enters into the cells and potassium shifts out of them, leading to hyponatremia.
D) Combined mechanisms.

One of the most significant renal physiology changes in healthy elderly is their trend to urinary sodium loss. This condition is generated by a reduced sodium reabsorbtion capability in the loop of Henle and collecting tubules.

A NaK2Cl cotransporter dysfunction in the loop of Henle and a sort of aldosterone resistance in the senile collecting tubules are proposed as their physio-pathological mechanisms[2,5].Then, this sodium loss state present in the healthy old people means a trend to develop hyponatremia in this population, specially when they are on low sodium diet for a long period.

In order to interpret and consequently to treat a hyponatremic state fractional excretion of urea (FEU) is usually used. Since urea is excreted by the kidney during body free water excess (eg: SIHAD) while it is saved during renal hypoperfusion states (eg: dehydration, cardiac insufficiency, etc), fractional excretion of urea is a useful marker in hyponatremic states. It helps to distinguish between hyponatremia with normal extracellular fluid (FEU higher than 65%) and hyponatremia with high or low extracellular fluid: renal hypoperfusion states (FEU lower than 35%)[6,7].

In our case the patient was initially interpreted as suffering from SIADH since he had high fractional excretion of urea (FEU: 98%) in a context of normal extracellular fluid. Because of that he was treated with free water restriction (water apport up to 800cc/day). But plasma sodium did not normalize and fractional excretion of urea became lower as it was expected under a free water restriction

state (FEU:22%).

Since water restriction did not normalized patient´s plasma sodium, and he had a sodium loss state: a high fractional excretion of sodium (FENa: 2.3 %) probably due to the senile reduced sodium reabsorbtion capability, and he also was on a chronic low sodium diet, then sodium supplement (4 g/day) was added to his gastrostomy feeding: 1500cc/day of liquid food.

After that, plasma sodium progresively bettered and normalized while fractional excretion of urea and sodium became higher (FEU: 64% and FENa: 6.5%), reflecting an augment in salt and water apport. No clinical signs of water and salt overload were detected (Table1).

We propose for this particular entity the name of senile sodium leakage hyponatremia. A name that reflects its physiopathologic mechanism and the age group where it takes place.

Another remarkable point in our case was the fact that patient´s high FEU made us to interpret this hyponatremia as a SIADH induced one. Since it has already been described that FEU is higher in the healthy elderly [8] a high fractional excretion of urea can be a confounding factor when it is used in order to find out the inducing hyponatremia mechanism in the elderly.

CONCLUSION:
Senile sodium leakage hyponatremia is an entity that should be taking into account in hyponatremic old patients and fractional excretion of urea seems to be a not realiable index in hyponatremia evaluation in this aged group..

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Comment of the reviewer Pedro Abáigar Luquín MD PhD. Nephrology Department. Hospital General Yagüe. Burgos. España
This is a report regarding a case of hyponatremia in an aged nursing home patient which is a frequent disorder in this population.

I would like to remark that conversely to healthy people where urinary volume is determined by the amount of their water intake, in patients suffering from the syndrome of inappropiate antidiuretic hormone secretion this volume is determined by the amount of solute that they have to excrete by urine. Besides, malnutrition could be another cause of hyponatremia in this group.

In this report, the whole solute apport: salt and urea, could have helped in normalizing the patient´s natremia.

It would have been better to document the patient´s plasma and urinary osmolality, as well as his weight before and after the water restriction and sodium apport maneuvers. These data would have been useful in order to interpret this case.

Comment of the reviewer Ramón Díaz-Alersi, MD. S. Medicina Intensiva. Hospital Puerto Real. Cádiz. España.
The old people, specially the frail and dependent ones, are prone to suffer water and electrolytes disorders, as is the case in the present report. In a hyponatremic patient, it should be firstly rouled out the presence of water excess and then the presence of a sodium lack. This was the way followed by the authors, but unfortunately their data was not enough to certificated the hyponatremia etiology of this case.

It would have been very interesting to have a description of the patient´s plasma and urinary osmolality (even calculated), blood preassure, and the appearance of symptoms related to his hyponatremia.

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