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A Super-Aged Case with Marked Hyponatremia Reaching 98 mEq/L; Clinical Implications of Low Serum Sodium

Takami Nakao[1], Yasuhide Kitazawa[1], Yasuro Obana[1], Naoki Hashimoto[2]

[1]Kindai University Nara Hospital, Ikoma City, Japan
[2]Emergency Center, Sanda City Hospital, Sanda City, Japan
Email: kitaz@med.kindai.ac.jp

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Abstract

A 79-year-old man, an in-patient at a long-term hospital suffers from Alzheimer dementia and thyroid hypofunction. Recently, he had also suffered from pneumonia followed by impaired consciousness and referred to our hospital for further examination. At the time of admission, the level of consciousness was JCS III-200 and other vital signs were normal. He had no edema on his limbs but had dry skin. According to chest CT, we detected pneumonia. Based on the laboratory data on admission, we detected low osmolality of 206 mOsm/kg of water and serum Na 98 mEq/L, beside urinary Na 54.1 mEq/L. We confirmed severe hyponatremia of hypersecretion type. Fluid replacement therapy was started mainly with Ringer's solution which is similar to extracellular fluid. We diagnosed it as SIADH induced by adrenal crisis based on the significant lower serum Na value and low blood sugar. Consequently, the patient was administered Hydrocortisone and Fludrocortisone acetate. On the 14th day, serum Na level improved from 98 mEq/L to 140 mEq/L. After daily monitoring of serum Na and urinary Na, administration of fludrocortisone acetate was terminated. The patient was discharged on 25th day, since serum Na was stabilized with oral administration of hydrocortisone and oral salt supplement. This case report shows that adrenal crisis can be triggered by pneumonia. In cases of impaired consciousness, if hyponatremia and hypoglycemia are observed, we may have to suspect the possibility of adrenal crisis. Therefore, urinary biochemical examination is an important part of the laboratory tests.

Keywords

Adrenal Crisis, SIADH, MRHE

1. Introduction

Super elderly people are often transported to an Emergency Department in a hospital with symptoms such as loss of appetite, nausea, vomiting and weakness, associated with impaired consciousness. Many of these patients show a tendency towards hyponatremia or hypoglycemia. We report a case of secondary adrenal insufficiency that presented with marked hyponatremia triggered by pneumonia.

2. Case Presentation

Patient: 79 years old, male.

Chief complaint: pneumonia with disturbed consciousness.

Past history: The patient suffers from Alzheimer's disease and hypothyroidism.

Present illness: He suffered from aspiration pneumonia at the geriatric family. He was referred to our hospital because he had impaired consciousness during the treatment. Body weight 52 kg, height 160 cm, BMI 20.3, conscious: Japan coma scale III-200; Glasgow Coma scale 5 (E-1, V-1, M-3). Vital signs: BP 104/58 mmHg, pulse rate 60/min regular, body temperature 35.9˚C, respiratory rate 15/min, pupil diameter bds 3 mm with prompt and complete light reflex. No edema in the extremities. Head CT showed no intracranial lesions that may have caused disturbance of consciousness. However, Chest CT showed pneumonia in both lungs.

Blood test data on admission are presented in Table 1, and the endocrinological data are presented in Table 2. The medications taken by the patient while in the hospital and the patient's progress after hospitalization are presented in Figure 1. Laboratory blood test findings showed significant hyponatremia, serum Na 98 mEq/L, while urinary biochemistry showed concentrated urine; urinary Na 54.1 mEq/L and urinary osmolarity 536 mOsm/kg of water. We diagnosed it as hypotonic dehydration with salt-wasting in urine.

We immediately started infusion therapy using Ringer's solution which is similar to extracellular fluid. Tazobactam piperacillin was administrated to treat pneumonia. On the 3rd day, glucose of 50% concentration was intravenously injected to treat mild hypoglycemia. However, the effect of glucose injection did not improve the hypoglycemia condition and blood glucose remained around 60 - 80 mg/dl. Judging by hyponatremia with hypoglycemia under euthyroid condition, we suspected adrenal dysfunction. Hence, after intravenous injection of Methylprednisorone 125 mg, blood glucose gradually returned to normal level, and the urinary Na concentration decreased on the next day.

On the 4th day, his consciousness recovered to the prehospital state with JCS I-1, GCS 14 (E-4, V-4, M-6). At that time, the serum Na was still low at around 110 mEq/L. As a result, oral administration of Hydrocortisone was started at 10 mg/day, followed by gradual dose increase to 40 mg/day. Nevertheless, the serum Na level remained at about 130 mEq/L but still below normal range. Based on this result, we changed our treatment protocol by adding oral administration

of Fludrocortisone acetate 0.1 mg/day from the 9th day. Fludrocortisone is a mineral corticoid drug.

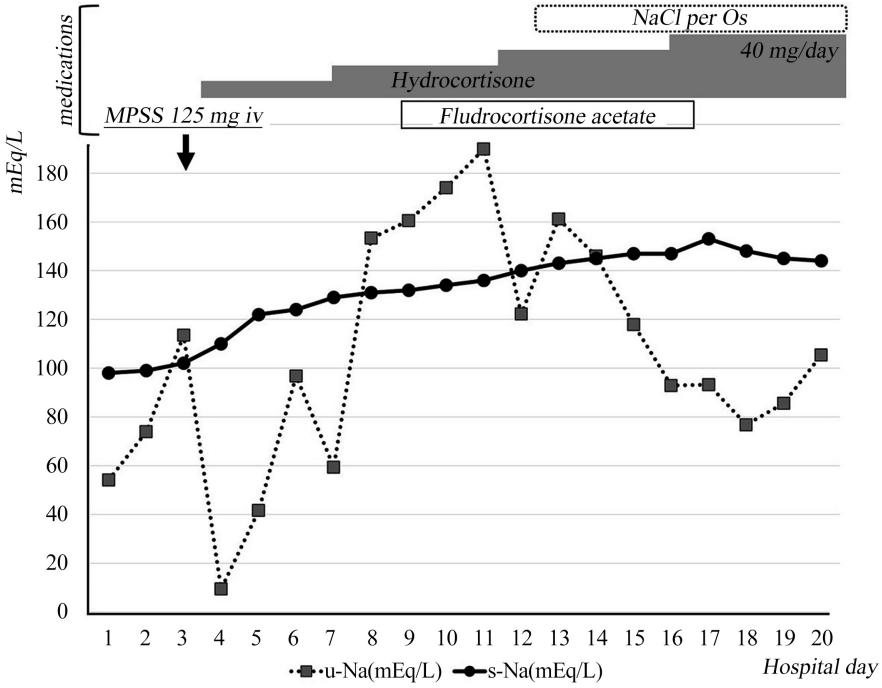

Figure 1. Medications and changes in serum/urinary Na levels, after admission. MPSS: Methylprednisolone Sodium Succinate, intravenous injection.

Table 1. Laboratory data on admission.

Blood Cell Counts			*Biochemical test*		
WBC	13.2	$\times 10^3/\mu L$	Na	98	mEq/L
RBC	3.96	$\times 10^6/\mu L$	K	4.9	mEq/L
Hb	10.5	g/dL	Cl	69	mEq/L
Ht	30.8	%	Ca	7.8	mg/dL
Plt	19.6	$\times 10^4$/mL	CRP	5.33	mg/dL
			TP	6.6	g/dL
			Alb	2.9	g/dL
Urine electrolyte concentrations			BUN	17.1	mg/dL
u-Na	54.1	mEq/L	Creatinine	0.2	mg/dL
u-Cl	67	mEq/L	UA	1.3	mg/dL
u-Osm	536	mOsm/kg of water	AST	215	IU/L
			ALT	214	IU/L
			γ-GTP	97	IU/L
			t-Chol	111	mg/dL
			TG	39	mg/dL
			s-Osm	206	mOsm/kg of water

Table 2. Hormonal concentrations.

	Results		Reference range	
Sampled on the 2nd hospital day				
ADH (AVP)	1.6	pg/mL	0.3 - 3.5	pg/mL
TSH	2.28	μIU/mL	0.45 - 4.50	μIU/mL
FT_3	1.6	pg/mL	1.7 - 4.0	pg/mL
FT_4	0.99	ng/dL	0.7 - 1.5	ng/dL
Cortisol	15.2	μg/dL	5.0 - 17.9	μg/dL
Aldosteron	106.2	pg/mL	36 - 240	pg/mL
BNP	44.9	pg/mL	<18.4	pg/mL
Sampled on the 4th hospital day				
ACTH	<1.5	pg/mL	7 - 56	pg/mL

With the start of enteral nutrition, the amount of infusion was reduced, and administration of sodium chloride was also started. During this period, serum Na level and urinary Na level were monitored. On the 17th day, blood test results showed serum Na 153 mEq/L giving rise to hypernatremia. Hence, Fludrocortisone acetate was discontinued. Eventually, administration of Hydrocortisone 20 mg/day and salt 1.5 g/day resulted in maintaining serum Na at about 140 mEq/L, and the patient was discharged from hospital on the 25th day.

3. Discussion

In the above case, there was no history of oral antihypertensive drugs or diuretics being administrated that could induce hyponatremia, moreover, sodium chloride had been supplemented in the tube feeding at the long-term care insurance facility (LTCF). Hence, the hyponatremia condition was considered to be an acute onset, because the serum Na was within normal range a week before this event. This led us to diagnose this case as SIADH due to adrenal crisis triggered by pneumonia. Although the precise diagnosis was not determined, in order to be safe, we started steroid replacement therapy.

Daily monitoring of serum and urinary Na contributed to a good clinical outcome and well-being of the patient. Super-elderly people often visit Emergency Department or require ambulance transportation to medical centers with complaints of loss of appetite, nausea, vomiting, feeling lethargic and altered level of consciousness. In some patients without abnormal findings on CT or MRI of the brain, general blood test data may indicate hyponatremia sometimes. In addition, there are some cases where hyponatremia occurs even in elderly people who have been hospitalized for a long period and are on long-term enteral nutritional management protocol with sodium chloride supplement, while undergoing regular blood tests.

There are various causes of hyponatremia, including diuretic medication, diarrhea, heart failure, liver disease, renal disease, and SIADH [1]. The syndrome

of inappropriate secretion of antidiuretic hormone (SIADH) is most frequently the cause of hyponatremia, although hyponatremia associated with volume depletion of the extracellular fluid also occurs commonly [2]. In these cases, the level of secretion of antidiuretic hormone was deemed "inappropriate". With extensive research done, this antidiuretic hormone in human was found to be arginine vasopressin. There have been proposed various diagnostic algorithms and criteria for SIADH [3] [4] [5].

One of the diagnostic criteria for SIADH is shown in **Table 3** [5]. Based on these criteria, our patient showed 1) reduced serum osmolarity, 2) relatively high urinary osmolarity and sodium, 3) normal thyroid function, and 4) elevated plasma ADH levels despite the presence of hypotonicity.

In recent years, the pathological condition called MRHE (Mineral corticoid Responsive Hyponatremia of the Elderly) has been attracting attention as a cause of hyponatremia in the elderly. This disease is characterized by hyponatremia, low serum osmolarity, hypertonic urine, low serum renin levels, high serum aldosterone levels, and relatively high levels of ADH. Reabsorption of Na and water in the proximal and distal convoluted tubule of the kidney decreases with

Table 3. Diagnostic criteria for SIADH.

Essential features
Decreased effective osmolality (<275 mOsm/kg of water)
Urinary osmolality > 100 mOsm/kg of water during hypotonicity
Clinical euvolemia
No clinical signs of volume depletion of extracellular fluid
No orthostasis, tachycardia, decreased skin turgor, or dry mucous membranes
No clinical signs of excessive volume of extracellular fluid
No edema or ascites
Urinary sodium > 40 mmol/liter with normal dietary salt intake
Normal thyroid and adrenal function
No recent use of diuretic agents
Supplemental features
Plasma uric acid < 4 mg/dl
Blood urea nitrogen < 10 mg/dl
Fractional sodium excretion > 1%; fractional urea excretion > 55%
Failure to correct hyponatremia after 0.9% saline infusion
Correction of hyponatremia through fluid restriction
Abnormal result on test of water load (<80% excretion of 20 ml of water per kilogram of body weight over a period of 4 hours), or inadequate urinary dilution (<100 mOsm/kg of water)
Elevated plasma AVP levels, despite the presence of hypotonicity and clinical euvolemia

Referred from Ellison DH, et al.[5] *with minor modifications.*

age, combined with decreased responsiveness to aldosterone subsequently ending in increased Na excretion leading to hyponatremia. The distinction between SIADH and MRHE is important. However, in actual clinical setting, since it takes longer time to get the test results from laboratory, it becomes difficult to diagnose dehydration condition in a patient on admission, therefore, delaying the diagnosis to differentiate between SIADH and MRHE. MRHE is said to have a lower blood cortisol level and a higher ACTH level compared to SIADH [6]. In our case, endocrine test data on admission showed that cortisol was within normal range and ACTH was lower compared to normal range which was reported at a later date (**Table 2**). Based on these data, SIADH diagnosis is a reasonable conclusion rather than MRHE. MRHE and SIADH require strict discrimination. In cases similar to our case where there is a strong concern about the complication of adrenal crisis, corticoid replacement therapy must be prioritized against the fatal risk of glucocorticoid deficiency. However, the usefulness of mineralocorticoid replacement therapy for salt-wasting hyponatremia of any type may be considered universal.

The condition that results from the absolute and relative deficiency of glucocorticoids due to various causes is called the adrenal crisis [7]. **Table 4** shows a list of features that would suggest the diagnosis of secondary adrenal crisis. The combination of hyponatremia, hypoglycemia, and impaired consciousness in this patient strongly indicated adrenal crisis. However, the cortisol level on the day after admission was within normal range, and an absolute deficiency of glucocorticoid was ruled out. The ACTH value was difficult to evaluate because it is based on a day later after rapid administration of Methylprednisolone. It can be inferred that there was a relative deficiency of glucocorticoid under acute pneumonia. Factors that may induce adrenal crisis are suggested to be 63% for infectious diseases, 6% for surgery, and 6% for trauma [8]. It has also been reported

Table 4. Clinical features of adrenal crisis.

Symptoms
Weight loss
Fatigue
Myalgia
Gastrointestinal symptoms
Vertigo
Conscious disturbance
Biochmistry
Hyponatremia
Normokalemia
Hypoglycemia
Norma thyroid function
Norma acid/base balance

that 28% of patients with pneumonia (bacterial, viral and fungal) develop hyponatremia [9]. Even enteral nutritional patients who receive salt by tube are at risk of developing hyponatremia due to infectious diseases such as erroneous swallow pneumonia. Therefore, periodic checking on serum and urinary Na levels is important for super-elderly patients.

4. Conclusion

We reported a super-aged patient with marked hyponatremia. The patient was diagnosed as SIADH due to adrenal crisis. Appropriate hormone replacement therapy recovered the patient. Urinary Na should be measured at first in order to diagnose a patient with abnormal serum Na level.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Dineen, R., Thompson, C.J. and Sherlock, M. (2017) Hyponatraemia-Presentations and Management. *Clinical Medicine Journal*, **17**, 263-269. https://doi.org/10.7861/clinmedicine.17-3-263

[2] Berghmans, T., Paesmans, M. and Body, J.J. (2000) A Prospective study on Hyponatremia in Medical Cancer Patients: Epidemiology, Aetiology and Differential Diagnosis. *Supportive Care in Cancer*, **8**, 192-197. https://doi.org/10.1007/s005200050284

[3] Thomas, C.P. (2021) Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH), Differential Diagnoses, Medscape. https://emedicine.medscape.com/article/246650

[4] Olsson, K., Enhörning, S., Öhlin, B. and Melander, O. (2016) Hyponatremia in the Emergency Department: Could Biomarkers Help in Diagnosis and Treatment? *Open Journal of Emergency Medicine*, **4**, 11-22. https://doi.org/10.4236/ojem.2016.41003

[5] Ellison, D.H. and Berl, T. (2007) The Syndrome of Inappropriate Antidiuresis. *The New England Journal of Medicine*, **356**, 2064-2072. https://doi.org/10.1056/NEJMcp066837

[6] Ishikawa, S., Saito, T., Futagawa, A., Higashiyama, M., Nakamura, T., Kusaka, I., *et al.* (2001) Close Association of Urinary Excretion of Aquaporin-2 with Appropriate and Inappropriate Arginine Vasopression-Dependent Antidiuresis in Hyponatremia in Elderly Subject. *The Journal of Clinical Endocrinology & Metabolism*, **86**, 1665-1671. https://doi.org/10.1210/jcem.86.4.7426

[7] Oelkers, W. (1996) Adrenal Insufficiency. *The New England Journal of Medicine*, **335**, 1206-1212. https://doi.org/10.1056/NEJM199610173351607

[8] Jung, C. and Inder, W.J. (2008) Management of Adrenal Insufficiency during the Stress of Medical Illness and Surgery. *Medical Journal of Australia*, **188**, 409-413. https://doi.org/10.5694/j.1326-5377.2008.tb01686.x

[9] Nair, V., Niederman, M.S., Masani, N. and Fishbane, S. (2007) Hyponatremia in Community Acquired Pneumonia. *American Journal of Nephrology*, **27**, 184-190. https://doi.org/10.1159/000100866