□ SHORT COMMUNICATION □

Change of the Image of the Posterior Pituitary in a Patient with Mineralocorticoid-responsive Hyponatremia of the Elderly: Comparison of Findings before and after Treatment

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Key words: hyponatremia, SIADH, mineralocorticoid, MRI, posterior pituitary

(DOI: 10.2169/internalmedicine.46.6152)

Text

On October 5, 2005, an 80-year-old woman with no contributory past history consulted our hospital with a complaint of headache. Laboratory studies showed serum sodium of 119 mEq/l and potassium of 4.6 mEq/l. Urinary excretion of sodium was 61 mEq/l. Blood urea nitrogen was 11 mg/dl; serum creatinine, 0.5 mg/dl; serum uric acid, 2.0 mg/dl; and hematocrit, 38.0%. The plasma osmolality was low at 247 mOsm/kgH₂O, while urinary osmolality was

somewhat elevated at a concentration of 333 mOsm/kgH₂O. The thyroid stimulating hormone (TSH) was 1.87 μ IU/ml and free thyroxine, 1.3 ng/dl. Adrenocorticotropin (ACTH) was 55.0 pg/ml, cortisol, 22.7 μ g/dl and arginine vasopressin (AVP) was relatively high at 0.4 pg/ml compared to the low plasma osmolality. The patient was diagnosed as having a syndrome of inappropriate secretion of antidiuretic hormone (SIADH). She had not taken any medication that would induce SIADH. Computed tomographic (CT) scan of the whole body did not demonstrate pleural effusion, ascites or findings of malignancy. On cranial magnetic resonance imaging (MRI), there were no abnormal findings except for the absence of high intensity of the posterior pituitary on T1-weighted images (Fig. 1A). Despite restriction of water intake, the serum sodium level decreased to 116 mEq/l, and amnesia appeared. Therefore, administration of fludrocortisone acetate 0.05 mg was initiated on suspicion of mineralocorticoid-responsive hyponatremia of the elderly (MRHE). The serum sodium level improved to 122 mEq/l on October 18 and 134 mEq/l on October 26. With improvement of the serum sodium level, her headache disappeared and amnesia improved. Serum sodium was 139 mEq/l; serum uric acid, 4.6 mg/dl; and hematocrit, 38.9% on December 14, then administration of fludrocortisone acetate was discontinued. Laboratory data showed a serum sodium level of 135 mEq/l on December 21; plasma osmo-

Figure 1. Cranial magnetic resonance imaging (MRI) demonstrated an absence of high intensity of the posterior pituitary on T1-weighted images (Fig 1A). After normalization of the serum sodium level and plasma osmolality, high intensity of the posterior pituitary on T1-weighted images was improved (Fig 1B).

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Received for publication July 28, 2006; Accepted for publication October 13, 2006

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lality, 279 mOsm/kgH₂O; plasma renin activity, 0.6 ng/ml/h; and plasma aldosterone concentration, 34 pg/ml. Both the serum sodium level and the plasma osmolality were maintained within normal ranges without further administration of fludrocortisone acetate, and cranial MRI on March 16, 2006 demonstrated improved high intensity of the posterior pituitary on T1-weighted images (Fig. 1B).

Cranial MRI shows the absence of a high intensity of the posterior pituitary on T1-weighted images in patients with SIADH as well as central diabetes insipidus (1). The following two views have been proposed to explain the reason. 1) In patients with SIADH dependent on ectopic production of AVP, negative feedback suppresses the formation of normal AVP by the neurohypophysis. 2) In patients with SIADH derived from the neurohypophysis, depletion of its stores in the posterior pituitary due to persistent release may result in the absence of a high intensity of the posterior pituitary on T1-weighted images. It is postulated that increased AVP synthesis and secretion are related to a reduction in extracellular fluid volume and changes in the sodium level. In an animal experiment that suppressed AVP secretion by salt-loading for one week, it was reported that after one week of rehydration, AVP immunoreactivity in the supraoptic nucleus had recovered to almost the same level as that in control rats (2), but the recovery period of AVP secretion in humans is not clear, and there are no reports describing the interval

until amelioration of high signal of the posterior pituitary on MRI.

Based on the clinical course and changes on imaging of the posterior pituitary, it is obvious that the secretion of AVP from the neurohypophysis played a role in the pathogenesis of hyponatremia in this case. Most cases of SIADH are caused by a relative increase in AVP compared with plasma osmolality, and water restriction or sodium load improves the condition. However, Ishikawa et al proposed the disorder concept of MRHE, which resembles SIADH in clinical findings but the pathogenic mechanisms and treatments differ (3). MRHE is thought to be caused by degradation of the response to the renin-aldosterone system in addition to increasing weakness of the renal capacity for sodium retention due to aging. In this case, the plasma aldosterone concentration level was low after treatment, suggesting that a low level of aldosterone secretion was involved in hyponatremia.

We encountered a case of MRHE that improved dramatically following transient administration of fludrocortisone acetate to correct the serum sodium level. We consider that in a case of SIADH that does not improve with water restriction and shows an absence of high intensity of the posterior pituitary on T1-weighted images on cranial MRI, physicians should consider prescribing fludrocortisone acetate.

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