Syndrome of Inappropriate Secretion of Antidiuretic Hormone Associated with Idiopathic Normal Pressure Hydrocephalus

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A 79-year-old woman suffering from urinary incontinence and unsteady gait was diagnosed as having idiopathic normal pressure hydrocephalus (NPH) with hyponatremia due to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). The concentration of antidiuretic hormone was high while the plasma osmolality was low in the presence of concentrated urine during the episodes of hyponatremia. Magnetic resonance imaging (MRI) of the head showed enlargement of the third and lateral ventricles. After ventriculoperitoneal shunt surgery, the symptoms of NPH and hyponatremia improved. It may be possibly explained that mechanical pressure on the hypothalamus from the third ventricle is responsible for hyponatremia.

Key words: hyponatremia

Introduction

The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) has been known since 1957 (1). It is characterized by low serum levels and an increased excretion of sodium with plasma hypoosmolality, urine hyperosmolality and low serum renin activity. Although many illnesses including disorders of the central nervous system (CNS) are reported to be associated with SIADH (2–6), idiopathic normal pressure hydrocephalus (NPH) has not been reported to be a causal disease of this syndrome. To the best of our knowledge, there is only a few case reports of SIADH in association with hydrocephalus (7). Here, we report a case of SIADH associated with idiopathic NPH.

Case Report

A 79-year-old woman with a past history of hypertension developed urinary incontinence and unsteady gait in April 1997. She had diarrhea and appetite loss associated with severe hyponatremia (117 mEq/l) in August 1997 and was admitted to the Tsuruga City Hospital. There was no history of head injury, meningitis or subarachnoid hemorrhage.

Physical examination showed no clinical dehydration. Her skin was warm and moist, and had normal turgor. No abnormal findings were observed in the lungs, heart and abdomen. Her blood pressure was 140/72 mmHg and heart rate was 92/min. She was alert, but disoriented and recent memory was unreliable. She was unable to memorize three object names and to subtract serial 7’s. The revised version of Hasegawa’s Dementia Scale (HDS-R) was 7 (full score 30) (8). She showed slightly wide based unsteady gait. There was no evidence of rigidity or limb ataxia. Her muscular power and sensory system was normal. Laboratory data showed relatively high urine osmolality (367 mOsm/kgH2O) despite a low plasma osmolality (256 mOsm/kgH2O). Blood urea nitrogen was 8.2 mg/dl, serum creatine 0.5 mg/dl and serum uric acid 2.3 mg/dl (Table 1). However, plasma antidiuretic hormone (ADH) level was high (14.0 pg/ml). The serum cortisol value was 27.8 µg/dl, the daily excretion of 17-ketosteroids (17-KS) and 17-hydroxycorticosteroids (17-OHCS) was 9.6 mg/day and 13.1 mg/day, respectively, which were all slightly high. Thyroid function test, plasma renin activity and the human atrial natriuretic peptide level were normal (Table 2). These laboratory results were consistent with SIADH (9). Lumbar puncture revealed cerebrospinal-fluid pressure of 80 mmH2O and the fluid was acellular. The protein was 31 mg/dl and the sugar 68 mg/dl. Radiographs of the chest were normal. Magnetic resonance imaging (MRI) of the head showed enlargement of the third and lateral ventricles. Periventricular high intensity was not observed on T2-weighted images (Fig. 1). Radioisotope cisternography disclosed ventricular reflux and slow clearance of the tracer from the ventricles. We diagnosed this patient as having idiopathic NPH (10), which might be responsible for the induction of.
SIADH.

A ventriculoperitoneal (VP) shunt was made on September 16, 1997 and the following day she showed a good response to verbal orders. Five days after surgery her walking was almost steady. Two weeks later, her urination was well controlled. HDS-R was 23. Five weeks later, the bicaudate cerebroventricular index, which indicates the degree of hydrocephalus (11) was changed from 38% to 36%. On October 3, 1997, serum sodium concentration recovered to the normal range (137 mEq/l). Also the hyponatremia disappeared when the third ventricle did shrink. He also commented that obstruction of the shunt, by inducing sudden dilation of the third ventricle and stretching its walls, seems to cause temporary impairment of function of the osmoreceptor and/or osmostat areas in the hypothalamus which influence ADH secretion. In the present case, VP shunt surgery improved the symptoms of both idiopathic NPH and hyponatremia. The fact suggests that idiopathic NPH may cause SIADH by the mechanism described above. With the recovery of the serum sodium concentration, the serum ADH level was normalized without water restriction or concentrated NaCl infusion. This case suggests that idiopathic NPH may be a causal factor of SIADH and close endocrinological examination should be performed in patients with idiopathic NPH.

Discussion

SIADH has been reported in various disorders, including malignant tumors, lung disease and disorders of CNS. How- ever, to the best of our knowledge, no case of SIADH associated with idiopathic NPH has been described in the literature. We report a patient who developed SIADH during the active phase of idiopathic NPH.

In the present case, enlargement of the third ventricle was detected by MRI. It has been shown that enlargement of the third ventricle is an important factor in the development of hyponatremia accompanied with the neurological disorders of SIADH. There were two cases of SIADH associated with Shy-Drager syndrome (12) or myotonic dystrophy (13), in which enlargement of the third ventricle and atrophy of the hypothalamus were detected. It may be possible that mechanical pressure on the hypothalamus from the third ventricle is responsible for hyponatremia. Wise (14) supported the idea from the observation that hyponatremia in patients with hydrocephalus persisted, when ventriculooatrial (VA) shunts became blocked and did not decrease the size of the third ventricle, whereas the hyponatremia disappeared when the third ventricle did shrink. He also commented that obstruction of the shunt, by inducing sudden dilation of the third ventricle and stretching its walls, seems to cause temporary impairment of function of the osmoreceptor and/or osmostat areas in the hypothalamus which influence ADH secretion. In the present case, VP shunt surgery improved the symptoms of both idiopathic NPH and hyponatremia. The fact suggests that idiopathic NPH may cause SIADH by the mechanism described above. With the recovery of the serum sodium concentration, the serum ADH level was normalized without water restriction or concentrated NaCl infusion. This case suggests that idiopathic NPH may be a causal factor of SIADH and close endocrinological examination should be performed in patients with idiopathic NPH.

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References

Figure 1. T1-weighted MR image (upper) and T2-weighted MR image (lower) of the head across the third ventricle (left) or lateral ventricles (right) showing enlargement of the third and lateral ventricles.