Initial and Postoperative Hyponatremia Associated with Pituitary Adenoma: A Case Report

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Summary

This 67 year-old man experienced 3 episodes of symptomatic hyponatraemia. Radiological examination revealed a sellar lesion and the tumour was removed via the transsphenoidal route. Thereafter, he simultaneously developed intractable diabetes insipidus and serious hyponatraemia with persistent natriuresis. His level of atrial natriuretic peptide was not significantly elevated, however, his plasma aldosterone concentration was low. The oral administration of salt gradually improved his hyponatraemia as well as the coincident symptoms. By the administration of a mineralocorticoid, fludrocortisone acetate, we succeeded in maintaining his serum sodium level without salt replacement. We discuss the mechanism(s) and treatment of hyponatraemia associated with pituitary tumour.

Keywords: Hyponatraemia; syndrome of inappropriate secretion of antidiuretic hormone (SIADH); cerebral salt wasting syndrome (CSW); fludrocortisone acetate.

Introduction

Hyponatremia occasionally occurs in patients with central nervous system disorders, especially subarachnoid haemorrhage and severe head injury [7, 10, 12, 14, 17]. In patients with pituitary adenomas, hyponatremia as an initial symptom and as a postoperative complication after transsphenoidal surgery has been reported, respectively. Only rarely does hyponatraemia precede other common symptoms of pituitary tumour; studies on postoperative hyponatremia in these patients have been published. The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and the cerebral salt-wasting syndrome (CSW) have been considered as underlying mechanism(s) [7, 12, 14, 18].

We treated a patient with a pituitary adenoma who presented with hyponatraemia as the initial symptom and experienced severe hyponatraemia with diabetes insipidus after transsphenoidal surgery. We discuss the mechanisms of initial and postoperative hyponatremia, and the treatment for this phenomenon.

Case Report

This 67-year-old man developed headache, nausea, vomiting, and confusion on November 1, 1994. He had been treated medically for hyperthyroidism when he was 20 years old. At the time of this admission, his thyroid functions were normal. No adrenal dysfunction was disclosed by examination of his ACTH-, cortisol-, and aldosterone levels. The serum sodium concentration was 112 mmol/L. Upon intravenous administration of hypertonic saline his clinical symptoms improved. Magnetic resonance imaging (MRI) showed an iso-intense mass in the pituitary gland, the stalk was deviated (Fig. 1, left). Although a pituitary tumour was suspected, he refused further treatment. The second episode of hyponatremia occurred two months after the first, on January 8, 1995. At that time, his serum sodium level was 111 mmol/L, the urinary sodium concentration was 20 mmol/L, and the daily renal excretion of sodium totaled 22 mmol. Water-loading tests revealed disturbance of water diuresis (Fig. 2). Restricting his water intake resulted in normalization of his serum sodium level. He returned in June, 1995 when he experienced a third episode of hyponatremia. MRI showed growth of the previously suspected sellar tumour with mild suprasellar extension and evidence of intratumoural haemorrhage (Fig. 1, right). He exhibited neither visual disturbance nor other clinical symptoms of hypopituitarism. He agreed to undergo surgery and the tumour was removed via the transsphenoidal route. The tumour was grayish-yellow and soft, easy to remove by the suction and so was totally removed. The finding of intratumoural haemorrhage was unclear. Histopathological examination returned the finding of pituitary adenoma of sinusoidal type (Fig. 3). Immunohistochemical staining showed no tumour cells positive for antibodies against any pituitary hormones. Final diagnosis of the tumour was non-functioning pituitary adenoma.

Diabetes insipidus appeared on the second day after the operation. His daily urine volume was 3000 to 5000 ml (Fig. 4); the urinary specific gravity was less than 1.007. The intravenous admi-
Fig. 1. Coronal T1-weighted MR image. The image on the initial study shows an iso-intense mass lesion in the sella turcica with stalk deviation (left). The second MRI study demonstrates increased volume of the mass lesion with intratumoural high-intensity lesions indicating intratumoural haemorrhage (right).

Fig. 2. Result of the water-loading test. After 1000 ml water intake, the urine osmolarity remains higher than the serum osmolarity throughout the examination period. The urine volume, measured at 30-min intervals, never exceeds 40 ml.

Fig. 3. Photomicrograph of the pituitary tumour removed by transsphenoidal surgery. The tumour is composed of cells which have oval abundant cytoplasms and round nuclei, and which are arranged in cords or clusters with some bleeding. Mitotic figures are absent. Haematoxylin and cosin staining, original magnification \( \times 200 \)

Administration of aqueous pitressin failed to control the urine volume. Sixteen days after the operation, he complained of headache and nausea; his serum sodium at that time was 114 mmol/L. The urinary excretion of sodium was 780 mmol on the previous day. Despite the intravenous and oral administration of sodium chloride, moderate hyponatraemia persisted for 15 weeks (Fig. 5). His plasma concentration of antidiuretic hormone (ADH; normal < 3.1 pg/ml) was always less than 0.1 pg/ml even while he was receiving pitressin. Plasma level of atrial natriuretic peptide (ANP; normal < 40 mg/ml) peaked at 65 pg/ml; baseline level of plasma aldosterone (normal 2–12 pg/ml) was 25.9 pg/ml, while his levels of plasma ACTH and cortisol were within the normal range. Fifteen weeks after surgery, oral salt supplementation (15 g/day) stabilized his serum sodium level. His polyuria gradually improved upon the intranasal instillation of 1-deamino-8-arginine-vasopressin. The administration of a mineralocorticoid, 0.4 mg/day of fludrocortisone acetate, was begun 33 weeks after the operation. His urinary excretion of sodium was markedly decreased and his serum sodium level was normalized. His supplemental salt intake was then stopped (Fig. 6). His course was satisfactory and one year after the operation he was normonatraemic.