Inappropriate Secretion of ADH Caused by Obstruction of Ventriculoatrial Shunts*

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"Salt-wasting" is a condition of hypotonicity of body fluids and continued renal excretion of excessive amounts of sodium chloride which is not caused by adrenal or pituitary disease. It has been encountered in patients with tuberculosis meningitis, pulmonary tuberculosis, and carcinoma of the lungs. In this report we are concerned with cerebral salt-wasting reported by Peters and associates. The mechanism of the salt-wasting syndrome was elucidated by Schwartz, et al., who suggested that it was due to inappropriate secretion of the antidiuretic hormone (ADH). Under normal conditions, as body fluid tonicity decreases, secretion of ADH is stopped and water diuresis ensues, resulting in an increase of body fluid tonicity toward normal. In patients with salt-wasting, however, secretion of ADH continues in spite of lowered body fluid osmolality, and if intake of fluid is not restricted, progressive hypotonicity of body fluids and expansion of extracellular fluid occur with increased renal excretion of sodium. The mechanism of excessive sodium excretion may be related to inhibition of aldosterone secretion, caused by the expanded extracellular fluid volume, increased glomerular filtration rate, or suppression of proximal tubular reabsorption of sodium.

From a number of reports, it appears that inappropriate secretion of ADH is also the basic mechanism involved in cases of cerebral salt-wasting. The two patients reported here developed inappropriate secretion of ADH and symptomatic hypotonicity during a period of obstruction of previously placed ventriculoatrial shunts. When a functioning shunt had been re-established, with presumed relief of hydrocephalus, the inappropriate secretion of ADH was also relieved.

Case Reports

Case 1. This 24-year-old man was first found to have hydrocephalus at the age of 7 years. He had had several operative procedures for the relief of this condition before a ventriculoatrial shunt (Pudenz-Heyer valve) was inserted in 1964. This shunt had required revision on several occasions. In July, 1965, he was admitted to the University of California Medical Center at 5 p.m. in an unresponsive state. His family stated that during the prior 3 days he had developed increasing drowsiness, nausea, and vomiting. On the morning of admission he became unresponsive and his blood pressure was 90/60.

Examination. At the time of admission serum sodium concentration was 122 mEq/l (Fig. 1), and the patient was given 500 cc of 3% saline intravenously. On the following morning serum sodium concentration was 137 mEq/l, and the patient was oriented and more alert; he resumed oral intake a few days later. His loss of 2.7 kg of body weight during his first 4 days in the hospital suggested that he had had water retention in the period immediately preceding admission.

Twelve days after admission, RISA was injected into a lateral cerebral ventricle and a scan suggested that the shunt was blocked. Over the next 3 days the patient became progressively more lethargic and ataxic; on the 15th day after admission, serum sodium concentration was again 122 mEq/l (Fig. 1) and urine osmolality was 828 mOsm/kg. He was again given 500 cc of 3% sodium chloride intravenously. The next morning his serum sodium concentration was 144 mEq/l, and he was more alert. The obstructed shunt was revised on the 29th hospital day and he remained alert afterwards.

Second examination. The patient was readmitted to the General Clinical Research Center for follow-up studies in October, 1965.

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He was alert and his shunt seemed to be functioning properly. Determinations of blood volume, response to water loading, and aldosterone secretion were within normal limits. Water balance could be maintained on a daily fluid intake of 1200 cc. When fluid intake was raised to 3000 cc/day, urine volume promptly increased and serum electrolytes and osmolality remained at normal levels. Serum urea nitrogen was 19 mg% and endogenous creatinine clearance on two successive days was 66 cc/min and 81 cc/min.

It appears that when this patient’s shunt became blocked he developed inappropriate secretion of ADH and water intoxication (hypotonicity). Since urine osmolality was only 195 mOsm/kg, on admission, ADH secretion presumably had begun to decline. Serum sodium concentration rose promptly after intravenous intake of hypertonic saline followed by the excretion of some of the retained water. His shunt, however, was not working well and he again developed inappropriate ADH secretion and water intoxication. Unfortunately, body weights were not determined at this time, but when his serum sodium concentration had dropped to 122 mEq/l, his urine osmolality was 828 mOsm/kg, which would indicate ADH secretion in the presence of a normal glomerular filtration rate. He again improved with 3% sodium chloride solution given intravenously and the shunt was subsequently revised. When studied while the shunt was working normally, there was no evidence of inappropriate ADH secretion.

Case 2. This 41-year-old woman had had a ventriculocisternostomy in October, 1965, for aqueductal stenosis and spontaneous rhinorrhea. Subsequently the shunt was revised on several occasions and a right frontal craniotomy was done for repair of rhinorrhea. A ventriculostial shunt was inserted in February, 1966, and she remained well until March, 1967, when she began to note intermittent fever.

Examination. On April 15, 1967, the patient was admitted to the University of California Medical Center in a stuporous condition with nuchal rigidity, elevated body temperature, and moderate edema of the face and eyelids. Her pupils were small and unresponsive to light and her eyes conjugately fixed down and to the right. Spinal puncture revealed CSF pressure of 170 mm H2O, and 890 white cells in the spinal fluid, of which 790 were polymorphonuclear leukocytes. Antibiotic therapy was started with penicillin, Chloramphenicol, and Gantrisin. On the fol-