Hyponatremia is the most common electrolyte disorder. In most patients, the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is the common diagnosis for hyponatremia, but in about 30% of the patients, hyponatremia can be attributed to hyperglycemia, severe renal failure, or excessive administration of free water.

The early neurologic manifestations of hyponatremia are nonspecific and can easily be overlooked. Many patients may have headache, nausea, and vomiting, but more often patients with hyponatremia have symptoms of confusion, somnolence, or profound drowsiness. The most consistent clinical symptom in hyponatremia is progressive when serum sodium values reach 125 mEq/l. Patients with rapidly developing hyponatremia below 110 mEq/l become unresponsive, and may have generalized tonic-clonic seizures.

In 1957, Schwartz et al (1) described the findings of hyponatremia and urinary sodium wasting in association with impaired urinary dilution in two patients with bronchogenic carcinoma. They concluded that the syndrome was due to inappropriate secretion of antidiuretic hormone. It is now recognized that the SIADH occurs in a great variety of malignant and nonmalignant diseases, most commonly those involving the central nervous system and lung. The cardinal features of SIADH as defined by Bartter and Schwartz (2) are (a) hyponatremia with corresponding hypoosmolarity of the plasma, (b) osmolarity of the urine greater than that appropriate for concomitant plasma osmolarity, (c) excessive renal excretion of sodium, (d) absence of clinical evidence of edema-forming states or volume depletion, (e) normal renal and adrenal function.

SIADH is found in various disorders of the central and peripheral nervous system. SIADH is also a well-recognized complication of bronchogenic carcinoma, its combination with autonomic dysfunction seems much less common. In 1993, a patient with malignant thymoma, sensori-motor neuropathy, severe autonomic disturbances (orthostatic hypotension, neurogenic bladder and anhidrosis) and SIADH was reported (3). The authors suggested that afferent baroreceptor dysfunction may be associated with paraneoplastic neurological syndrome, since lesions in acute autonomic neuropathy are usually in the efferent fibers.

The Guillain-Barré syndrome (GBS) is a disease that affects primarily the peripheral nervous system. An association of SIADH with GBS has been documented in a number of previous reports (4, 5). The pathogenetic mechanism of SIADH in GBS is unknown. Few studies have included measurements of the plasma arginine vasopressin concentration. One of these studies has specifically assessed the plasma arginine vasopressin concentration as influenced by changes in plasma osmolarity (6). From this evidence, afferent neuropathy of cardiac volume and osmolarity receptors has been suggested (7). The relationship between diabetes mellitus and cardiac afferent nerve fiber damage is accepted (8). An association between SIADH and diabetes mellitus has been reported, and one of the five patients in the original series of SIADH in GBS also had diabetes mellitus (9). The cardiac afferent in preexistent diabetic neuropathy is conceivable as a predisposing factor. Exaggerated ADH release in response to postural changes has been discussed. However, SIADH continued when the patient with GBS was no longer able to leave the bed, arguing against a problem primarily of nonosmotic ADH release.

A wide variety of interesting autonomic nervous system disturbances occur in GBS, most without serious consequences. Sixty-five percent of patients with typical GBS had some dysautonomia (10). Dysautonomia is also more frequent in GBS patients with severe motor deficits and respiratory failure. However, dysautonomic dysfunction occurs infrequently in CIDP (11).

Most of the cases with SIADH have demonstrated autonomic dysfunction. Abnormalities of the peripheral autonomic afferent fibers arising from the vascular stretch receptors have been thought to reduce vagal inhibitory action on the release of antidiuretic hormone from the neurohypophysis. It is reported that plasma levels of vasopressin in a child with GBS and SIADH were increased (12). However, the relationship between GBS without dysauto-
nomia and SIADH is not clearly identified.

Chronic inflammatory demyelinating neuropathy (CIDP) is regarded as a different clinical entity from acute GBS, with diagnostic criteria proposed by Dyck and co-workers (13). CIDP is characterized by (a) a longer progression (2 or 3 months) before plateau, (b) a tendency for the illness to be less severe by than the typical acute disease, (c) fluctuation in the severity of symptoms over many years, (d) greater elevation in CSF protein concentration, (5) more profoundly slowed motor nerve conduction velocities including motor nerve conduction block, and temporal dispersion. Most patients with CIDP have elevated CSF protein concentrations, often greater than 150 mg/dl, and sometimes up to 1,000 mg/dl. Benign intracranial hypertension, though still rare, is more common than with acute GBS, and has been attributed to the greatly elevated CSF protein concentration typical of many cases of CIDP. Several CSF dynamic studies in GBS have demonstrated an increased absorptive resistance in CSF pathways (14), but others have failed to demonstrate an increase in resistance large enough to explain the elevation in CSF pressure (15). Thus, the mechanism of benign intracranial hypertension in GBS is still uncertain. Dysautonomia is uncommon in CIDP.

Respiratory failure, due mainly to diaphragmatic weakness, is the most common serious complication of acute GBS. It is severe enough to require mechanical ventilation in approximately one-third of patients. Most of these patients had more profound limb weakness, longer hospitalization, longer recovery, and more frequent serious residual deficits than patients who do not require ventilation. However, respiratory muscle weakness requiring ventilator assistance is also uncommon.

Recently, a case of CIDP with SIADH, dysautonomia, intracranial hypertension and use of mechanical ventilator was reported from Japan (16).

To our knowledge, this is the first report of SIADH as a complication of CIDP. This case had not only acute aggravation of CIDP with dysautonomia and requirement of ventilator assistance but also intracranial hyper tension. CIDP with these symptoms is quite uncommon.

References


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