1.2 Neuropsychiatric Manifestations of Hypokalemia

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Abstract

Potassium deficiency can cause an acute organic brain syndrome. Older patients are more sensitive to hypokalemia because of decreased total exchangeable potassium associated with the aging process. Two patients who developed hypokalemia secondary to prolonged treatment with diuretics are described. The neuropsychiatric manifestations of hypokalemia may take a pseudoneurotic form and masquerade as depressive conversion and anxiety reactions.

Hypokalemia influences muscle and nerve by alternating the resting potential and by changing intra-cellular enzyme activity, therefore disturbing the cellular function (1). Acute organic brain syndrome has been described in hypokalemia but the extent of brain participation in the general body deficiency of potassium is unknown (2, 3).

Two patients with acute organic brain syndrome, secondary to hypokalemia caused by prolonged treatment with Chlorthalidone (Hygroton) are described. The subject is presented as case report because in trauma, comatose or critically ill patients a basic situation of potassium deficiency may lie subjacent and be overlooked as the sole cause of unconsciousness, unless prompt corrections of hypokalemia are automatically made in the intensive care unit.

Case Report

1. A 68 years old women suffering from diabetes mellitus, atherosclerotic heart disease and hypertension treated with Methyldopa. A month before her hospitalization treatment with 100 mg daily of Chlortalidone was started. Three days before her hospitalization a change in her behavior was noted by her family. Approximately from the same time sudden constipation appeared which did not respond to enemata. During the examination the patient spoke to herself. She did not recognize her surroundings, did not know her name nor her birth date. Her abdomen was swollen with faint peristalsis. Her blood pressure was 200/100 mm mercury.

Laboratory examinations: BSR 5/18 mm (Westergreen) HB 13.4 g\% Hematocrit 38\%, Wbc 8200/cmm with a normal differential count. The urinary sediment was normal, Urea 28 mg\%, Glucose 160 mg\%, Potassium in repeated examination 2.9-2.8 mEq/l, Sodium 136 mEq/l Chloride 98 mEq/l. The liver function tests were normal. The E.C.G. revealed nonspecific ischemic changes. On plain x-ray of the abdomen fluid levels were not seen. The patient was treated with potassium chloride intravenously and per os. After two days of treatment the blood level of potassium slowly raised to 3.4-4.9 mEq/l. The patient stopped to speak to herself, was aware of her hospitalization and could tell her name, adress and date of birth. Bowel movements normalized and her abdomen was no more swollen. She was discharged with Methyldopa Trichlormetiazide (Nydor) and “slow” potassium therapy.
2. A 65 years old women known to have elevated blood pressure was treated with Methyldopa and 100 mg Chlortalidone every two days. She was hospitalized because of progressive weakness which started six weeks prior to her hospitalization. According to her family the patient was lately dropping things from her hands and because of weakness in her legs was most of the time in bed.

In the examination the patient did not recognize her surroundings, and didn’t know her age and birth. She did not know the name of the president of the State. The patient did not succeed to grip forcefully the hand of the examiner. Her blood pressure was 160/110 mm mercury. Laboratory examinations: BSR 30/50 mm (Westergreen) HB 13.6 g% Hematocrit 39.5%. The urinary sediment was normal, blood urea 30 mg%, glucose 118 mg%, potassium in repeated examination 2.6-2.7 mEq/l, sodium 140 mEq/l, Chloride 100 mEq/l. The liver function tests were normal. The E.C.G. revealed non-specific ischemic changes.

The patient was treated with potassium chloride intravenously and per os and the potassium in the serum raised gradually to 3-4.4 mEq/l.

On the fourth day of treatment the patient was able to grip forcefully the hand of her examiner, knew her age, date of birth and the name of the State’s president. She admitted that she did not take Methyldopa, but instead took 200 mg Chlorthalidone every day. She was discharged with Methyldopa, Trichlormethiazide (Nydor) and “slow” potassium therapy.

Discussion

Organic brain syndrome is a neuropsychiatric disturbance caused by or linked with brain malfunction. The latter is secondary to irreversible loss of brain cells.

Acute organic brain syndrome can appear suddenly with fluctuating disturbances of consciousness, from mild disorientation to stupor or coma, impairment of intellectual function and judgment or emotional instability. Defects in cognitive function may be complicated by confabulation. Acute brain syndrome may be superimposed upon, may coexist with or may complicate a preexisting chronic brain syndrome or functional disorders. One of the causes that can induce it, is hypokalemia (1, 4).

The two patients developed acute brain syndrome secondary to hypokalemia. The hypokalemia developed after using a diuretic agent without potassium salt for prolonged period. On the top of the brain syndrome each patient presented with another hypokalemia effect. The first suffered from constipation and the second from myasthenia (1).

All manifestations disappeared when the serum potassium level returned to normal. Neuromuscular manifestations secondary to hypokalemia appear only after losing one third to one half of the total body potassium (5). Old patients over 60 years are specially sensitive to hypokalemia (6, 7). With increasing age the acellular components of the body (collagen, skeleton) which are potassium poor increase progressively. The fat-free body mass which is potassium rich progressively decreases with age. The decrease in exchangeable potassium is connected with the ageing process.

The neuropsychiatric manifestations of hypokalemia can present also as pseudoneuroses. They can resemble depression (anorexia, constipation, asthenia, and depressive mood), a convulsive reaction (myasthenia or paralysis) since there is no disturbance in the sensorium and intellectual function and the deep reflexes remain normal until very low serum potassium levels. Hypokalemia can manifest also as anxiety (headache irritability, nervousness, paresthesiae and visual disturbances) (8).