CASE REPORTS

HYPOKALEMIC PERIODIC PARALYSIS IN A HYPERTHYROID BLACK WOMAN

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In this case of periodic paralysis and thyrotoxicosis, investigation of the patient's family revealed other members similarly affected. To the best of the authors' knowledge, it represents the first reported instance of this familial association in the case of a black woman.

The familial periodic paralyses are a group of disorders characterized by attacks of flaccid paralysis. Three varieties of the disorder are generally recognized; one with reduced plasma concentration of potassium during attacks, another with elevated potassium, and a third with normal potassium. Periodic paralysis also occurs in association with metabolic disorders, but this form is not known to be familial. The following is a case of hypokalemic periodic paralysis in a black woman with thyrotoxicosis.

CASE REPORT

In June 1985, a 41-year-old black woman with a history of hyperthyroidism (diagnosed in 1975) and hypertension was brought to the emergency room complaining of inability to walk. The patient had been previously admitted twice to the hospital for similar symptoms, mainly, marked weakness of proximal musculature with inability to walk.

Two years prior to her current admission, the patient had been maintained on diuretics (hydrochlorothiazide) for hypertension, and had subsequently become weak to the point where she could no longer walk. At admission to the hospital at that time, she was told her potassium was very low. She was given oral potassium supplements and was discharged with control of hypertension achieved without diuretics. She did well at home, continuing without diuretics. One year later, she again developed sudden inability to move arms and legs. Again she was admitted to the hospital, treated with intravenous and oral potassium supplements, and discharged in three days upon return of her strength. At home, she was satisfactorily maintained on propylthiouracil, 50 mg daily, clonidine, 0.3 mg every 8 hours, and propanolol, 40 mg by mouth twice a day.

Four days before admission in June 1985, the patient noted muscle cramping, polyuria, and increased irritability. On the day she was brought to the emergency room, she had fallen asleep in a chair and found upon awakening that she could not arise from the chair.

On physical examination, she was found to be a well-developed 41-year-old woman with a blood pressure reading of 140/100 mmHg, pulse 76 beats per minute, respirations 15/min, and temperature 98.8 °F (37.1 °C). Her skin was warm and dry. Exophthalmos was obvious, and she had a pronounced stare. Her thyroid gland was diffusely enlarged to three times normal. Her muscle strength was determined to be one fifth in the upper and lower extremities, with most pronounced weakness of the proximal musculature. There was generalized hyporeactivity of the tendon reflexes.

The serum potassium was 2.1 mmol/L, sodium, 147 mmol/L, chloride, 105 mmol/L, and carbon dioxide combining power 32 mEq/L. Her chemistry profile was normal with the exception of a serum lac-

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tate dehydrogenase level of 456 IU/L. Serum total thyroxine (T_4) was 13.9 μg/dL (normal 4.0 to 14.0) and T_3 resin uptake, 31.9 percent (normal 24 to 36 percent). Thyroid stimulating hormone level was 2.7 μU/mL (normal 1.0 to 10.0). The complete blood cell count revealed a white blood cell count of 8,900 with 45 percent neutrophils, 54 percent lymphocytes and 1 percent eosinophils, hematocrit 37.9 percent and hemoglobin 12.6 percent with a platelet count of 380,000 mm^3. At admission, evaluation of urine electrolytes revealed a urine sodium and potassium of 101 mEq/L and 10 mEq/L, respectively. Arterial blood gas revealed a pH of 7.47, pO_2 84, pCO_2 36 and HCO_3 27. An electrocardiogram showed a QU interval of 0.60 and a sinus arrhythmia.

A total of 340 mEq of potassium was given orally and intravenously in the first 24 hours after admission. This dosage was tapered in the next two days. Within 24 hours, the patient was able to resist gravity with all muscle groups, and by 48 hours the patient felt "almost back to baseline." Oral and intravenous supplementation was discontinued at 48 hours. Serum potassium was measured at 5.0 mmol/L at 72 hours after admission. The patient was placed on acetazolamide, 250 mg by mouth daily.

Subsequently the patient has had one episode of weakness requiring hospitalization, at which time her serum potassium was 2.5 mmol/L. She again responded to intravenous and oral supplementation and was discharged. During that hospitalization, the patient admitted noncompliance with acetazolamide. She has been asymptomatic since that time.

DISCUSSION

Hypokalemic periodic paralysis is part of a group of disorders characterized by attacks of flaccid paralysis. Several varieties of the disorder are recognized; one with reduced plasma concentration of potassium during attacks, another with increased potassium, and a third with normal potassium. All are inherited as autosomal dominant. Periodic paralysis may also occur secondary to thyrotoxicosis and other metabolic disorders. This hyperthyroidism-associated form is most frequently seen in Oriental patients; there is a reported incidence of 1.9 percent in Japanese patients with hyperthyroidism.[1] To the authors' knowledge, there are two previously reported cases of this form in black male patients[2] and no cases reported in black women.

The different varieties of periodic paralysis resemble one another in clinical presentation. Attacks occur

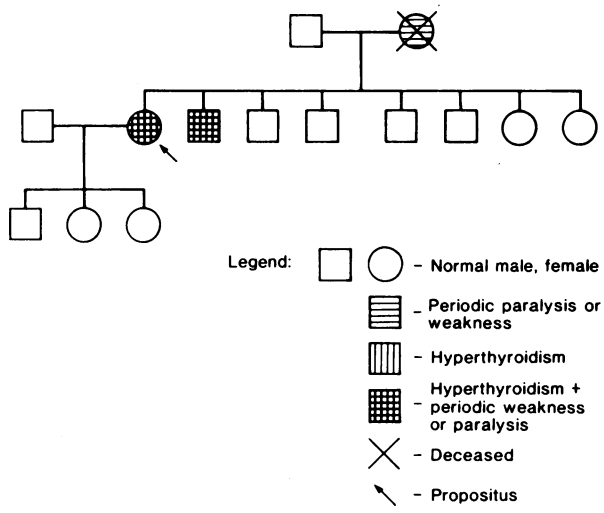

Figure 1. Family genogram of 41-year-old black woman with hyperthyroidism and periodic paralysis

at rest, after exertion, after a high carbohydrate diet, or upon exposure to cold or other stress. They may be induced by insulin with glucose, adrenal cortical steroids and epinephrine, or ACTH.[3] With regard to the specific entity of hypokalemic paralysis, the plasma potassium is reduced while the net total body potassium is normal. The pathogenesis remains unclear.

The pedigree of this patient's family also is of interest (Figure 1). The limited information available reveals at least two individuals with hyperthyroidism and symptoms of episodic weakness or periodic paralysis and one individual with episodic weakness. The data reported here are suggestive of an autosomal dominant inheritance as has been previously described.[4] Investigation of this family continues.

Acknowledgment

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