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Hypokalaemia and the thyroid—is there a link?

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Case

A previously healthy 40-year-old male was admitted to investigate the second episode of an acute flaccid paralysis that occurred within 3 months. Both began suddenly in the early morning as generalized weakness evolving to paralysis within 2 h, and sparing respiratory and facial muscles. The paralysis was painless, and there was no disturbance of consciousness. Vital signs, including heart rate, were within normal limits. Physical examination revealed flaccidity in both legs, with reduced deep tendon reflexes. Laboratory test results at admission are shown in Table 1. He was started on intravenous potassium, with complete recovery of muscle strength in few hours.

Question

What is your diagnosis?

Answer

Thyrotoxic hypokalaemic periodic paralysis (THPP)

Discussion

The incidence of THPP is highest among Asian persons (approximately 2%) [1]. In North America, the incidence rate was reported to be around 0.1–0.2% [2]. Patients are usually young adult males and Graves' disease is the most common cause of hyperthyroidism associated with THPP [3], as was the case of the patient described here. TSH-receptor antibody (TRAb) was positive and an ultrasound revealed a diffuse enlargement of the thyroid gland (total volume 16.3 cm^3).

THPP is characterized by muscle weakness that occurs due to a massive intracellular shift of potassium without total body K+ deficit. Respiratory muscles are rarely involved [4]. Spontaneous regression ensues in 30 min to 6 h, and complete recovery is the rule [2]. As in our case, most patients do not report thyrotoxic symptoms. Despite the fact that serum thyroid stimulating hormone (TSH) was suppressed and free T4 was elevated (Table 1), the hyperthyroidism was clinically silent [1].

The specific mechanism for THPP is not entirely clear. It appears that patients with THPP have an underlying predisposition for activation of Na/K-ATPase activity, either directly by thyroid hormone or indirectly via adrenergic stimulation [1]. Despite similarities between THPP and familial hypokalaemic periodic paralysis (FHPP), an autosomal dominant neurological channelopathy [5], mutations in voltage-gated cationic channel genes are rarely described [3].

Potassium measurement helps differentiate THPP from other causes of acute muscle weakness such as stroke, Guillain–Barré syndrome, botulism, alcoholic neuropathy and hysteria [1]. THPP is not associated with urinary potassium loss, and the transtubular K concentration gradient (TTKG) is low (<2). To determine the aetiology of the patient's hypokalaemia, we measured serum aldosterone and renin levels and they were found to be normal.

Table 1. Biochemistry

		Follow-up		
Laboratory tests	Range	At admission	48 h	72 h
Serum				
Sodium (mEq/l)	135–145	140	134	142
Potassium (mEq/l)	3.5–5.0	1.6	5.6	3.6
Creatinine (mg/dl)	0.6–1.2	0.87		0.7
TSH (nUI/ml)	0.4–4.0			0.001
Free T4 (ng/l)	0.8–1.9			4.37
Urine				
Sodium (mEq/24 h)	40–220			264
Potassium (mEq/day)	25–125			60
Creatinine (mg/24 h)	800–2000			2816

During the crisis, administration of potassium is important to avoid arrhythmias and accelerate muscle recovery. However, the dose of KCL should be as small as possible to prevent rebounding hyperkalaemia, as happened in this patient (Table 1). While the patient waits for specific treatment, crisis can be avoided by the use of beta-blockers. Our patient received definitive treatment with radioiodine therapy.

Conflict of interest statement. None declared.

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