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Sudden Paralysis in an American Football Player! Monster Football?

Chmiel, Corinne ; Giambarba, Christian ; Trachsler, Johannes

Abstract: This case of ascending paralysis, following an episode of diarrhea, was initially misinterpreted as Guillain-Barré syndrome. The prominent hypokalemia led to the search for other differential diagnoses, initially interpreted as a rare case of periodic hypokalemic paralysis, which usually occurs in Asians after intake of large amounts of starch foods, such as Spaghetti, or rest after heavy exercise. In this case, the reason for the hypokalemia with associated paralysis was caused by a hyperhyreosis factitia through denied intake of T3. **Keywords:** Guillain-Barré syndrome; Guillain-Barré-Syndrom; Hyperhyreose factitia; Hypokalemia; Hypokaliämie; Notfallmedizin; emergency medicine; hyperhyreosis factitial; hypokalemic ascending paralysis; hypokaliämische aufsteigende Lähmung.

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Case Report: Monster Football

PD Dr. med. Corinne Chmiel^{1, 2, , *3}, Dr. med. Christian Giambarba³, Dr. med. Johannes Trachsler³

¹ University Hospital of Zurich, Institute of primary care, Switzerland

² Private Practice Zurich, Switzerland

³ City hospital Waid, Zurich, Switzerland

*Corresponding author

Institute of primary care

University and University Hospital of Zurich

Pestalozzistrasse 24

8091 Zürich, Switzerland

corinne.chmiel@usz.ch

www.hausarztmedizin.uzh.ch

Tel: +41 (0)44 255 98 55

Fax: +41 (0)44 25 5 90 97

Abbreviations: General practitioner (GP).

Case Report

A 28-year-old Caucasian muscular man was brought to the emergency department by ambulance due to ascending paralysis of the legs without loss of sensibility. By the time of admission, he was hardly able to move his arms. He also reported of non-hemorrhagic diarrhea since three weeks with slight abdominal pain. The medical history included appendectomy three years earlier, intake of **Bisoprolol 5mg daily since years** due to arterial hypertension, and a seldom intake of Bromazepamum in case of nervousness. He was taking oral Testosterone until four months ago for muscular improvement. He had been working as a professional football player in the USA until three years ago; currently he was working in an office in Switzerland.

Temperature was 36.8°C (98.2°F), heart rate regular 92 beats/min, blood pressure 150/60 mmHg, and respiratory rate 20 /min. The skin was hyperemic and sweaty. The palpation of the heart showed a palpable thrill. Abdomen sounds were practically lacking in all four quadrants with diffuse tenderness in the lower left quadrant without peritonistic signs. Sensation of touch was preserved but slightly diminished at the feet. Distal and proximal muscles of the legs were paralytic (without rigidity), although the toes could be slightly moved. Strength of the proximal and distal muscles of the arms was reduced (M3). Reflexes were not electable symmetrically. Babinski sign was negative. The patient was unable to walk.

The electrocardiogram revealed a broadened QRS-complex, diffuse ST-depressions and an AV-Block I° (Figure 1). Chest x-ray was normal. Initial laboratory reports were normal besides ALAT/GPT 120U/l (<50U/l), creatinin-kinase 373 U/L (<308U/l). Potassium massively reduced with 1.3 mmol/l (>3.4mmol/l) and phosphate 0.3mmol/l (>0.87mmol/l). Urinary potassium concentration measured shortly after beginning intravenous potassium substitution was 21 mmol/L, potassium/creatinine ratio 20.3 mmol/g. Acid base status was normal. Therefore, the differential diagnosis of periodic hypokalemic paralysis was considered. The surprising results of Thyroid hormones, which arrived with delay, were: TSH < 0.01 uU/l (0.3-4.2), fT3 31.3 pmol/l (3.1-6.8), fT4 1.2 pmol/l (12.0-22.0).

Following resolution of hypokalemia by means of intravenous substitution in the intensive care unit, the weakness and ECG abnormalities rapidly resolved. Further diagnostic testing revealed no new insights: negative HIV serology, normal levels of serum cortisol and parathyroid hormone, negative serum and urinary toxicologic tests, normal levels of vitamin E, B1, B2, selenium, normal cranial MRI, normal Electromyography and nerve conduction studies, except for a slightly accelerated nerve conduction velocity, compatible with hyperthyreosis. Thyreoperoxidase and thyreoglobulin antibodies were negative, TRAk was slightly elevated 1.9 IU/l (<1.75).

Since no reason for the thyreotoxicosis with associated hypokalemic paralysis was found we consulted the patient's general practitioner (GP). Three years earlier the patient had consulted the GP because of an appendectomy after returning to Switzerland. The GP, which knew the patient since childhood, remembered being amazed by the bodily transformation the patient has undergone since he lived in the USA to become a professional football player. The patient then also mentioned, that in the USA subclinical hypothyroidisms was diagnosed, which is why he had received hormone replacement therapy (Novothyral (T3 and T4)). However, the patient never followed the request of the GP to present the laboratory values from the USA, it is therefore unclear whether a subclinical hypothyroidisms ever really existed. The hormone levels recorded by the GP were FT3 3.9 uU/l (3.1-6.8), FT4 13.6 uU/l (12.0-22.0), TSH <0.02 uU/l (0.3-4.2). Due to the hyperthyreotic constellation, the GP wanted to stop replacement therapy, but the patient insisted in continuing its intake. 15 months later the patient again consulted the GP due to daily recurring episodes of nausea and vomiting, diarrhea 7-8 times daily, thirst with a drinking volume of 8 to 10 liters daily, reddish coloring of the skin, muscular weakness and intermittent paralysis. The patient reported that he had received Thybon Avensis (T3) in a Pharmacy in Germany without prescription. The TSH was repeatedly suppressed with normal T4 and slightly elevated T3 levels and elevated serum Creatininkinase of 2000 E/L. Testing for pheochromozytoma due to hypertension and rash as well as diabetes mellitus and toxicological screening was negative. He increased dosage of bisoprolol and in the same week forwarded the patient to the endocrinology department at the University Hospital of Zurich. There the patient reported that the GP had diagnosed the patient with a hypothyroidism, which is why he was prescribed Novothyral (T3 and T4). The endocrinologist did not believe these statements when seeing the laboratory results of the GP, which were unchanged at the endocrinologist. The search for additional differential diagnoses by the endocrinologist was negative. Testosterone was elevated probably due to intake of anabolic substances with suppression von LH und FSH, Prolactin slightly elevated, Ferritin elevated, Albuminuria probably due to hyperthyreotic state. The endocrinologist assessed the situation as Hyperthyreosis factitia due to negated hormone intake (Novothyral T3 and T4).

When confronting the patient with the information received by the GP and endocrinologist, he admitted the following: since he moved from Switzerland to the US to become a professional football player, he had been shifting his diet from initially food supplements for muscular improvement, to the intake of Tribulus Terrestris, a phytotherapeutic of which players thought that it increases endogenous testosterone production, to finally mixes of different juices which circulated among team players mainly purchased over internet: Monster-Pump=Cyto-Sport, BSN=Syntha6, JackT3 oder Jack 3d, Monster 100, Mega Tribu X, Tyrosine. Among other ingredients, they contain T3 and ephedrine as well as amphetamines. Subsequently he

was diagnosed with “subclinical hypothyroidisms” without prescribed therapeutic consequences. From then on, he started to take Iodine tablets (150ug Taxofit) as suggested to him by trainers and team members. Later he continued taking Novothyral (T3 and T4) with Thybon (T3), as well as oral testosterone. Since he felt recurring episodes of paralysis, he took a few Jack 3d Shakes, and when feeling better, started with his training, which he could pursue without any problems. In the evening then symptoms began with progressive ascending paralysis of the limbs, which is why he called the ambulance bringing the patient to us. Throughout the hospitalization (of 6 Weeks?), all symptoms resolved eventually with accompanied slow normalization in T3 levels (last measured fT3 1.8 pmol/l (3.1-6.8), fT4 4.5 pmol/l (12.0-22.0). TSH rose steadily until 5.37 uU/l (0.3-4.2) at the time of discharge. **without any hormone substitution?** Before discharge from hospital, the patient was offered psychological guidance to tackle his dependency issues, which was denied. The patient was unfortunately not available for follow up, since he moved away from Zurich and changed his phone number.

Discussion

The occurrence of rapidly progressing ascending paralysis of the limbs without loss of sensibility after a history of three weeks diarrhea is highly suggestive of a Guillain-Barré syndrome, an acute immune-mediated polyneuropathy provoked by a preceding infection, in this case most likely gastroenteritis. Other differential diagnoses are peripheral neuropathy, neuroblastoma, marine animal stings or bites, Trichinosis, myopathies of systemic disease, Botulism, certain drugs, intoxications, tick bites and snake venoms. From the medical history although Beta blockers may be a possible cause for ascending paralysis, they seemed unlikely to have contributed to the current severe and rapid progression of symptoms, especially when considering that the medication has been taken for years. On the contrary, beta-blockers seemed to have masked the cardiac and vegetative symptoms of hyperthyroidism, and therefore the patient may not have become symptomatic earlier, resulting in delayed clinical evaluation. However, the beta-blocker did not block the metabolic consequences, such as provocation of hypokalemia, which was finally the reason for the patient presenting at the emergency department. Oral testosterone intake has been described to cause neuropsychiatric side effects but not peripheral neuropathy. The initial most striking finding was the profound hypokalemia (1.3mmol/L). As hypokalemia augments the transmembraneous potential (hyperpolarization), neuromuscular excitability is reduced, leading to fatigue, muscle weakness including rhabdomyolysis, constipation and cardiac arrhythmias. Therefore, the hypokalemia could very well explain the complaints of the patient as well as the findings of the ECG. Hypokalemia can be caused by transcellular shifts, reduced intake, augmented losses (renal

or extrarenal such as gastrointestinal) or a combination of these mechanisms. Since urinary potassium excretion was below 20 to 25 mEq (per day, liter, or g creatinine), renal potassium wasting is excluded. Therefore, extra renal (gastrointestinal) losses and transcellular shifts had to be considered. Potassium shift into cells may be caused through alkalosis, which was excluded, malnutrition, malabsorption, insulin or theophyllin application, adrenergic stimulation, hyperthyreosis, or history compatible with familiar hypokalemic periodic paralysis [1-3]. In this patient, no signs or symptoms of malnutrition or malabsorption were present. Insulin or adrenergic medication use was denied, and paralysis has never occurred before in the patient (other than during T3 or T4 intake) or his family members. Suppressed TSH and elevated fT3 indicate hyperthyroidism. Thyreotoxicosis may cause a sporadic, acquired form of periodic paralysis associated with hypokalemia caused by a sudden shift of potassium into cells. This genetic disorder is known as thyreotoxic periodic paralysis and occurs mainly in Asian men [4-6]. Sporadic cases are associated with numerous other conditions including barium poisoning, renal disorders, and gastrointestinal potassium losses. Although Graves' disease is the most common underlying thyroid pathology, other hyperthyroid disorders including exogenous thyreotoxicosis can be involved. Periodic hypokaliemic paralysis without hyperthyroidism is a different, hereditary entity with mostly autosomal dominant inheritance pattern that shares the same precipitating factors with thyreotoxic periodic paralysis, namely rest after heavy exercise or a high-carbohydrate meal. Treatment of periodic hypokalemic paralysis consists mainly of parenteral potassium supplementation which should occur with caution, since in many cases, this leads to rebound hyperkalemia, which was also seen in this case [7].

Summary

This case of ascending paralysis, following an episode of diarrhea, was initially misinterpreted as Guillain-Barré syndrome. The prominent hypokalemia led to the search for other differential diagnoses, initially interpreted as a rare case of periodic hypokalemic paralysis, which usually occurs in Asians after intake of large amounts of starch foods, such as Spaghetti, or rest after heavy exercise. In this case, the reason for the hypokalemia with associated paralysis was caused by a hyperthyreosis factitia through denied intake of T3.

Key words

Hypokalemia

Hypokalemic ascending paralysis

Hyperthyreosis factitia

Emergency medicine
Guillain-Barré syndrome

Conflicts of Interest

The authors declare no conflict of interest. No funding sponsors exist.

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