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CASE REPORT

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A rare case of thyrotoxic periodic paralysis revealing Graves' disease in a young Malian

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Abstract

Sporadic thyrotoxic periodic paralysis (TPP) is a rare muscle disorder that manifests with abrupt muscle weakness and hypokalemia associated with hyperthyroidism. It is mostly reported in the Asian population, and rare in Caucasians. Only few cases have been reported in people with black ancestry. Here, we report a rare case of thyrotoxic periodic paralysis revealing Graves' disease in a young Malian. A 17-year-old man was admitted in the Neurology clinic with rapid proximal tetraplegia that started after strenuous physical activities at the school. Clinical examination confirmed the proximal weakness. In addition, he had bilateral ptosis, exophthalmia, and horizontal ophthalmoplegia. Laboratory testing showed normal serum potassium and creatinine, low calcium and TSH levels. However, CK, FT4, thyroid stimulating hormone antibody, and acetylcholine receptor antibody levels were high. In addition, electrocardiogram was normal while thyroid Doppler-ultrasound showed heterogeneous, hypoechogenic, hypertrophic, and hyper vascularized gland. Patient had completely recovered his limb weakness within the following hours with symptomatic treatment. The clinical findings were consistent with Graves' disease, and he was put on Neomercazole. He did not present another episode of paralysis after 4-years of follow up. This is a first case of thyrotoxic periodic paralysis reported in Mali and one of the rare cases in sub-Saharan Africa. Despite its scarcity, all patients with acute weakness consecutive to effort, whether recurring or not, should be screened for TPP.

KEYWORDS

Africa, Graves' disease, Mali, Normokalemia, thyrotoxic periodic paralysis

1 | INTRODUCTION

Sporadic thyrotoxic periodic paralysis (TPP) is a rare muscle disorder that is mostly reported in the Asian population with a prevalence of 2%.[1] It is a serious neurological complication of hyperthyroidism that threaten the life of patients without efficient care. Its diagnosis is established in presence of acute muscle weakness, hypokalemia, and hyperthyroidism. The onset is usually between the second and the fourth decades. Its clinical manifestation is

typically a painless abrupt muscle weakness with no sensory and no cognitive impairments.[2] Potassium shifting into the cells due to the Na-K ATPase pomp that increases active transport of potassium (K+) ions into the intracellular compartment causes hypokalemia without total body potassium deficit secondary to the hyperthyroidism.[3,4] Although hypokalemia is typically observed, rare cases of TPP with normokalemia or hyperkalemia have been reported.[5,6] TPP is a clinical entity that has common similar symptoms with the familial periodic paralysis (FPP) which is a dominant condition and is not linked to a hyperthyroidism. It is extremely rare in populations with black ancestry with only few cases reported in Africa and in Africans from the diaspora.[4,7,8] Here we report a case of a Malian adolescent who was admitted in the Department of Neurology of the Teaching Hospital of Point G for acute tetraparesis after effort.

2 | CASE PRESENTATION

A 17-year-old adolescent male of Moorish ethnicity was admitted in the Neurology Department for acute onset muscle weakness. He had no familial history of muscle weakness. Symptoms began with tingling in the legs and muscle pain that occurred after an intense physical exercise at school. Few hours later, he presented with muscle weakness in lower limbs, and he fell down when he was hurrying to cross the road due to painful cramps. The following day he could not get out of his bed due to continued pain and weakness in all four limbs. At the admission he was in good shape in general with normal blood pressure (130/60 mmHg), normal temperature (36.3°C), normal pulse (88 beats/minute) and normal respiratory rate (17 cycles/minute). Neurological examination found a painful proximal and symmetrical muscle weakness in both upper and lower limbs, diffuse absent tendon and idiomuscular reflexes, decreased muscle tone and bilateral palpebral paresis (Figure 1A). He also presented with bilateral exophthalmia, and ophthalmoplegia particularly in upper, inner, and lateral gazes for the right eye and in the inner gaze for left eye. He did not have sensory or cognitive impairment. The thyroid gland was found to be swollen on physical examination which was confirmed by ultrasound that showed diffuse hypoechogenic, slightly heterogeneous, hypertrophic, and hyper vascularized gland. Serum electrolytes found normal potassium, sodium and magnesium, and low calcium. The TSH was low, and TSH receptor antibody and free FT4 were high, consistent with a hyperthyroidism. The CK levels were high (427 UI/L) but myoglobinemia was normal. Myoglobinuria testing was not available but we noticed darkened urines. The total blood cell counts, sedimentation rate, serum creatinine, C reactive protein were normal. He had normal hemoglobin electrophoresis profile (AA). The electrocardiogram was normal particularly no signs of hypokalemia or hyperkalemia. The clinical and laboratory findings are summarized in Table 1. Initially, he was put on bed and benefitted of a rehydration on the basis of 1 liter of lactate solution, 1 g of Paracetamol and 100 mg of Ketoprofen intravenously, leading to almost a total motor recovery within 13 h. The clinical presentation and the progression of the symptoms were consistent with sporadic TPP. The morphology and function of the thyroid in addition to exophthalmia are in favor of Graves' disease. The patient was referred to the Department of Internal Medicine where he was put on Neomercazole 40 mg per day and calcium supplement. He hadn't had any other episode of paralysis after 4 years and the other symptoms decreased gradually (shown in Figure 1). However, he was not compliant with the treatment, and had relapses of the ptosis.

3 | DISCUSSION

TPP is a sporadic form of periodic paralysis (PP) that may occur in patients with hyperthyroidism. It is characterized by an abrupt onset of muscle weakness, hypokalemia, and hyperthyroidism.[1] The mechanism by which hyperthyroidism produces hypokalemic PP is still not well understood. But it is known that thyroid hormones increase tissue responsiveness to beta-adrenergic stimulation, which, along with thyroid hormone, increases sodium-potassium ATPase activity on the skeletal muscle membrane and explains hypokalaemia.[3] Cases with normokaliemia or hyperkaliemia were also reported.

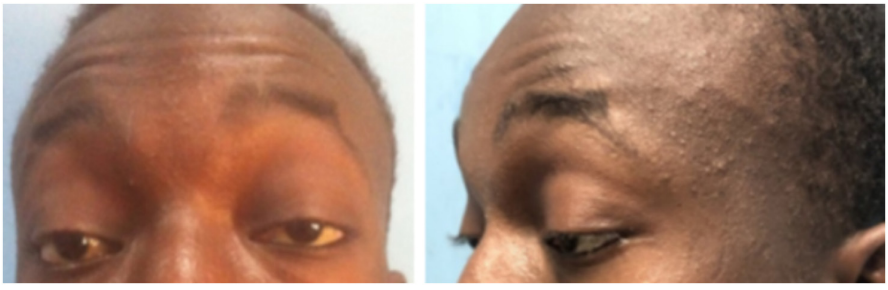

FIGURE 1 Clinical description of the patient showing a bilateral palpebral paresis and exophthalmia.

TABLE 1 Sociodemographic, clinical and laboratory findings.

Sociodemographic and clinical aspects	
Age (year)	17
Sex	M
Ethnic	Moorish
Neurological signs and symptoms	Acute onset of painfull and proximal muscle weakness after strenuous activities, abolished tendon and idiomuscular reflexes, low muscle tone
Laboratory analysis	
K+ (mmol/L)	3.90 (N = 3.50–5.50)
Na + (mmol/L)	147 (N = 135–155)
Ca2+ (mmol/L)	1.18 (N = 2.15–2.55)
Mg2+ (mmol/L)	0.70 (N = 0.66–1.07)
CK (UI/L)	427 (N = 26–192)
TSH µUI/mL	0,005 (N = 0,27-4,7)
FT4 (pmol/mL)	101 (N = 9–20)
TSHr antibody (UI/L)	>40 (N < 1.75)
AChol-r antibody (nmol/L)	1.11 (N < 0.25)
Creatinine (µmol/L)	65 (N = 45–84)
CRP	Negative
VS 1H/2H	Normal
Blood cell count	Normal
ECG	Normal

Abbreviations: AChol-r, acetylcholine receptor; CK, creatine phosphokinase; CRP, C reactive protein; ECG, electrocardiogram; H, hour; L, litter; mmol, millimole; M, male; N, normal; TSH, thyroid stimulating hormone, TSHr, thyroid stimulating receptor; VS, sedimentation rate; UI, international unity.

However, it is still not clear how these different potassium levels lead or are associated to the same symptoms. TPP is commonly reported in the Asian population, rare in Caucasians and exceptional in people with black ancestry.[7,8] Large cohort studies are lacking to explain these differences. While thyrotoxicosis is more frequent in women, the TPP are predominant in men,[1] which may suggest that the disease is not solely due to thyrotoxicosis but intercurrent factors may be involved. The age at diagnosis is usually between the second and the fourth decade of life,[9] and our patient was 17 when he presented symptoms. In fact, less cases with adolescent onset have been reported compared to older age groups worldwide.[4,9,10] The triad that characterizes patients with TPP is hypokalemia, acute onset muscle weakness and hyperthyroidism.[4,5,9] The abrupt muscle weakness presented by this patient is the commonly reported clinical presentation.[5,9,11] Intense physical activities may have probably provoked the paralysis in the patient reported here. Indeed, some factors such ingestion of carbohydrate, strenuous physical activities, trauma, infections, and genetic susceptibility are incriminated in inducing the PP.[12] Serum electrolytes can typically reveal hypokalemia associated or not to some degree of hypomagnesemia and hypophosphatemia. The patient presented here had normal serum potassium, and he did not have any cardioelectric sign of dyskalemia. In fact, supporting our findings, normokalemic TPP cases have been reported elsewhere.[5,6] Serum calcium was low in our patient which is not commonly seen in TPP, and may not be associated with the disease described herein. In addition, the patient had a confirmed rhabdomyolysis with high CK levels and darkened urines but normal serum creatinine, corroborating what is reported in the literature.[13] The endocrine and immune testing and thyroid ultrasound confirmed Graves' disease which is the predominant cause of TPP as reported in several others studies.[1,2,4] In addition, our patient had increased anti-AChR antibodies, which has been previously reported in patients with Graves' disease without a concomitant myasthenia gravis (MG).[14] However, an overlapping phenotype between TPP and MG is also possible. Sporadic TPP should be differentiated from FPP which is an autosomal dominant condition and does not associate hyperthyroidism.[3,4] TPP requires an urgent management which includes symptomatic treatment with careful correction of hypokalemia when it is the case, and associated with thyroid hormones to prevent the recurrence of attacks. Euthyroidism is the objective of the treatment and allows to prevent further periodic paralysis.[3] If well managed, patients generally recover within the next few hours like in the case we report here. The patient did not present another episode of paralysis after 4 years of follow up on Neomercazole despite the lack of compliance to treatment.

In conclusion, TPP is a rare muscle complication of hyperthyroidism. We report here a rare case of this disease in a young Black African. The disease can have a dramatic outcome when associated with hypokalemia and with a delayed diagnosis. Therefore, endocrine function of the thyroid should always be checked in cases of sporadic periodic paralysis even in the first episode of attack.

AUTHOR CONTRIBUTIONS

Mohamed Emile Dembélé: Formal analysis; investigation; writing – review and editing. **Abdoulaye Yalcouyé:** Validation; writing – original draft; writing – review and editing. **Mamadou Cissoko:** Investigation; validation. **Lassana Cissé:** Investigation; validation; visualization; writing – review and editing. **Cheick Oumar Guinto:** Conceptualization; methodology; supervision. **Guida Landouré:** Conceptualization; funding acquisition;

investigation; supervision; validation; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors do not have any conflict of interest to declare.

DATA AVAILABILITY STATEMENT

All data related to this case report are documented within this manuscript. Further enquiries can be directed to the corresponding author.

ETHICS STATEMENT

The study was approved by the Ethics Committee of the Faculty of Medicine, Pharmacy and Dentistry (FMPOS) of Bamako under number N° 2017/149/CE/FMPOS.

CONSENT

Written informed consent was obtained from the legal guardian of the patient to publish this report in accordance with the journal's patient consent policy.

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