Thyrotoxicosis Presenting as Hypokalemic Periodic Paralysis

Dilshad Siddiqui, MBBS, MRCP(UK)*

A case of hypokalemic periodic paralysis secondary to thyrotoxicosis is reported. The patient presented with intermittent weakness of limbs and history of weight loss. Serum potassium was 1.8 mmol/L on the day of admission. Although rare, thyrotoxicosis should be excluded as a cause of hypokalemic periodic paralysis.

Bahrain Med Bull 1998;20(2):58 - 60.

Although complaints of intermittent weakness are frequently encountered in clinical practice, the disorders that cause periodic paralysis are very rare and evaluation of such symptoms is challenging because examination is often normal between attacks. Diagnosis is based on patient's history, examination and evaluation of serum electrolytes during the attack[1].

Attacks of hypokalemic periodic paralysis can occur in patients with thyrotoxicosis, especially in young Latin Americans or Asian men where up to 10% of thyrotoxic patients may have periodic paralysis[2].

Acute attacks respond to potassium administration. Treatment of underlying thyrotoxicosis abolishes attacks. Beta-adrenergic blocking agents reduce the frequency and severity of attacks while measures to control thyrotoxicosis are being instituted.

THE CASE

A 33 year old Bahraini male was admitted through BDF Hospital Accident and Emergency Department in June 1996 with the complaint of intermittent weakness of both lower limbs since morning. The first episode of weakness occurred early in the morning, after getting up from sleep, when he felt his legs weak and heavy. Initially, inspite of weakness, he was able to perform the usual routine work like washing and praying but later on the weakness in the legs increased and he was unable even to stand. The weakness was not associated with fever, parasthesia or numbness and lasted for about one hour then resolved spontaneously.

The second episode of weakness in limbs occurred about 2 hours later, while he was driving on his way to work. This time weakness was associated with nausea and sweating. He was unable to drive and was brought by a friend to A/E Department where he vomited food particles once only.

The patient gave a history of similar weakness in both lower limbs about a year ago which lasted for about two hours. That time, he had attended A/E Department of another hospital and was told that his serum potassium was low. The patient was not known to have any systemic disease and was not on any medications. Review of systems reveled history of weight loss since one year but he did not know the exact weight loss, and he felt that for the past 2-3 months, his weight was steady around 56.5 kgs. The patient has 3 children. He used to smoke around one packet of cigarette per day and there was no history of alcohol intake. There was no history of similar problem in the family. His mother had died due to heart attack.

On examination, he looked sick and was unable to move out of the bed. He was not pale, jaundiced or cyanosed, and was afebrile. Pulse: 100/min, BP: 130/70 mmHg, respiratory rate: 20/min, weight was 56.4 kg.

Thyroid was diffusely enlarged but there were no signs of thyrotoxicosis. Cardiovascular system, respiratory and abdominal examination was unremarkable. Neurologically, patient was alert and oriented. Cranial nerve examination was normal. Tone was decreased in both upper and lower limbs. Power in the upper limbs was 4/5 and lower limbs 3/5. Deep tendon reflexes were normal. Plantars were flexors and sensory examination was normal.

Routine laboratory tests showed the following results: FBC: WBC 3.2 x 10^9/L, Hb: 13.4 g/L, Platelets: 220 x 10^9/L; ESR: 2 mm in 1 hour. U & E, Creatinine: Sodium: 141 mmol/L, Potassium: 1.8 mmol/L, TCO_2: 24.8 mmol/L, Urea: 6.0 mmol/L, creatinine: 69 umol/L, random blood sugar: 9.3 mmol/L, chest x-ray and MSU were normal. ECG: sinus rhythm rate 100/min. U waves present in most of the leads (Fig 1). Further investigations were requested ie, serum cortisol, urine 24 hours for electrolytes and thyroid function test. Serum cortisol was normal, 571 nmol/L in the morning sample, and urinary electrolytes were normal as well.

* Senior Resident
Department of Internal Medicine
Bahrain Defence Force Hospital
State of Bahrain

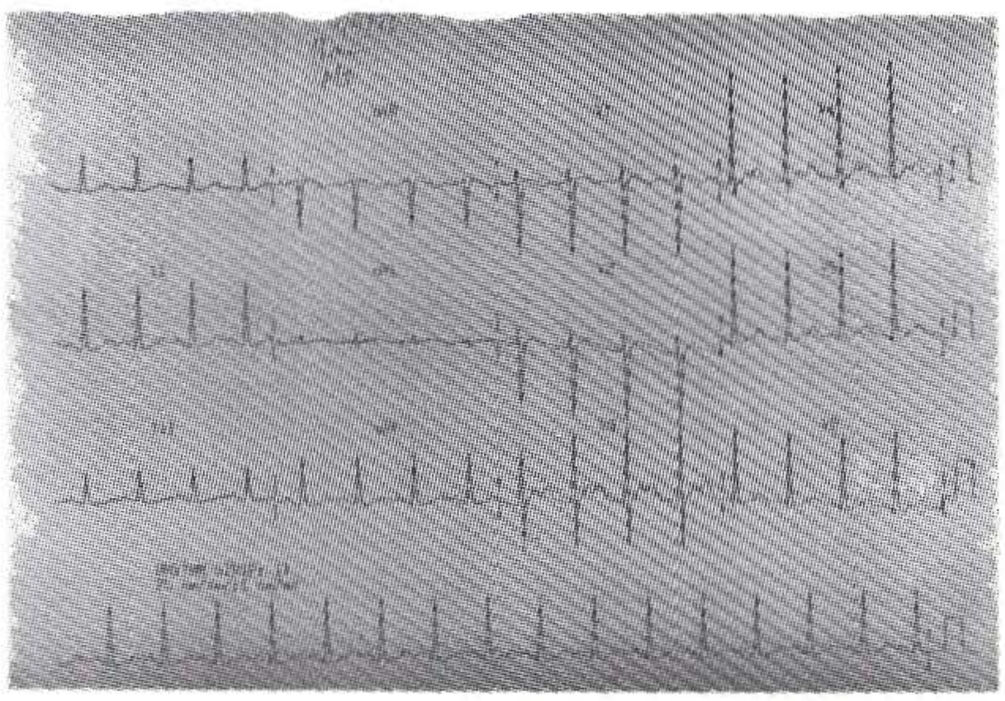

Fig 1. ECG showing changes in hypokalaemia

Hospital course and management: After admission, patient was given 60 mmol of KCL in 1 litre 5% Dextrose water over 8 hours. He felt better and at the end of the infusion, his serum potassium was 4.7 mmol/L and repeat ECG showed disappearance of "U" waves (Fig 2). Next day, the patient had no weakness of limbs and neurological examination was normal. His serum potassium was 5.4 mmol/L and serum sodium was 142 mmol/L. He was discharged in a satisfactory condition.

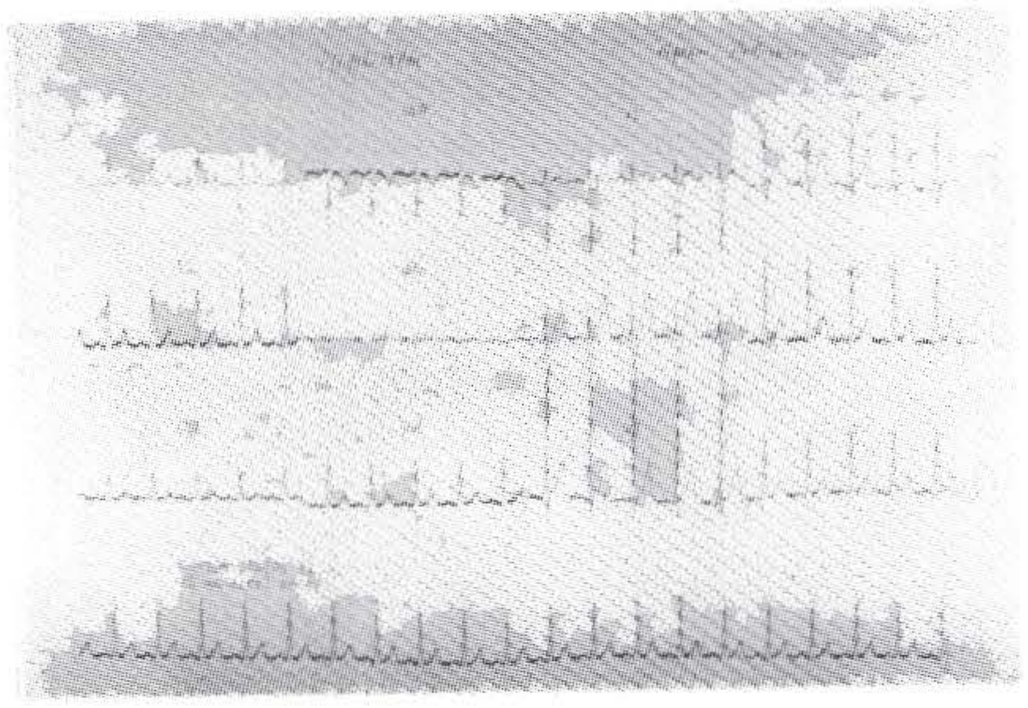

Figure 2. ECG after potassium administration. The 'U' waves have disappeared

In the first outpatient follow-up visit, the results of the TFT's were available which showed evidence of hyperthyroidism. TSH: <0.05 uu/ml (N=0.23-4.0), Free T_3: 32.7 Pmol/L (N=5.4-9.3), Free T_4: 63.5 Pmol/L (N=11.8-24.6).

Thyroid ultrasound was reported as diffuse enlargement with multiple tiny hypoechoic areas suggestive of colloid, feature favouring hyperactive thyroid gland. He was started on Carbimazole 15 mg TDS and Tab Propranolol 40 mg TDS, and the dose was titrated according to the TFT's result. He started gaining weight but his thyrotoxicosis was difficult to control with antithyroid drugs.

In May 1997, he was treated with radioactive iodine and later the doses of Propranolol and Carbimazole were gradually reduced and finally stopped in July 97, when his TFT's results were as follows:

TSH <0.01 uu/ml, Free T_3 4.7 Pmol/L, Free T_4 4.9 Pmol/L. His last clinic visit was in August 1997 when he was clinically and biochemically euthyroid. His weight was 72 Kg (gained 15.6 kg since day of admission). TFT's were TSH 4.0 uu/ml, Free T_3 6.9 Pmol/L, Free T_4 10.6 Pmol/L. Serum potassium remained in the normal range throughout follow-up ie, between 3.8 to 4.4 mmol/L.

DISCUSSION

There are several causes of sudden onset of severe weakness and/or paralysis. Periodic paralysis could be primary or secondary to electrolyte disturbances, neuromuscular junction disorders eg. myasthenia gravis, Lambert Eaton syndrome, central nervous system causes eg. cataplexy and sleep paralysis associated with narcolepsy and multiple sclerosis and transient ischaemic attacks. Electrolyte disturbances include hypokalemia, hyperkalemia, hypocalcemia, hypercalcemia, hyponatremia, hypophosphatemia, and hypermagnesemia.

Hypokalemic periodic paralysis may be primary and familial or it may be secondary to renal gastro-intestinal potassium loss. Major causes of secondary hypokalemia include renal tubular acidosis, diuretic or laxative abuse, primary aldosteronism, Bartter's syndrome and villous adenoma of the colon.

One of the underlying disorders is hypokalemic thyrotoxic periodic paralysis (HTPP) which is an uncommon disorder. HTPP is characterised by periodic occurrences of muscle weakness during attacks of hyperthyroidism[3]. Thyrotoxic periodic paralysis is a reversible cause of paralysis[4]. This disorder affects predominantly Asian males, where up to 10% of the thyrotoxic males may present with hypokalemic periodic paralysis[5]. It is very rare among Caucasians[6]. The typical signs and symptoms of thyrotoxicosis may be absent and thyrotoxicosis may remain undiagnosed for years. Here the case illustrates the HTPP presentation with only weight loss and periodic paralysis. In its clinical presentation, thyrotoxic periodic paralysis (HTPP) is identical to primary familial hypokalemic periodic paralysis (FPP). FPP is autosomal dominant in two-thirds of cases and sporadic in one third of cases. HTPP affects predominantly males, but is rarely associated with a positive family history, and has a later onset of presentation than FPP. Graves diseases is the most common cause of hyperthyroidism, but any cause of thyrotoxicosis can trigger attacks of HTPP in susceptible subjects[7].

In HTPP, hypokalemia is the most consistent laboratory abnormality, representing a trancellular shift rather than a total body deficit, the exact mechanism is unknown. The exercise test demonstrates distinct electromyographical abnormalities in those with periodic paralysis[8]. Though the mechanism of HTPP is unknown, there is a possibility that excess catecholaminergic activity might precipitate hypokalemia hence the benefit of intervening with IV propanolol in a reported case[9].

Potassium is frequently administered to hasten recovery and prevent cardiac arrythmias and respiratory arrest. Serum potassium must, therefore, be monitored carefully in these patients during treatment[10]. Beta- adrenergic blocking agents reduce the frequency and severity of attacks, while definitive treatment of thyrotoxicosis is being instituted. Correction of thyroid function is essential to treatment[11].

Occasionally, patients of HTPP who present with severe muscle weakness may not respond to potassium administration, intravenous propranolol may be tried in those patients.

CONCLUSION

Thyroid disorders are fairly common in Asian countries. HTPP is an unusual presentation of a fairly common disorder affecting young Asian males[12].

Therefore, when an Asian male presents with severe weakness and/or paralysis and hypokalemia, the physician should suspect hyperthyroidism. The clinical features of hyperthyroidism may be very subtle or absent in thyrotoxic hypokalemic periodic paralysis[13].

This is the first case of thyrotoxic periodic paralysis diagnosed at BDF Hospital, and additional cases may be found in Bahrain and other Gulf countries, if doctors exercise a high index of suspicion regarding the diagnosis and do thyroid function tests routinely in cases of unexplained muscle weakness.

REFERENCES

1. Weetman AP. Thyrotoxicosis. Journal of Medicine International 1997;25:24-8.
2. Robert C, Griggs. Periodic Paralysis. In: BraunwaldW, Petersdorf I,et al, eds. Harrison's Principles of Internal Medicine. 12 edn. International Edition: McGraw Hill Inc, 1991:2121-22.
3. Van Dam GM, Reisman Y, Van Wieringen K. Hypokalaemic thyrotoxic periodic paralysis: Case report and review of an oriental syndrome. Neth J Med 1996;49:90-7.
4. Akhter J, et al. Thyrotoxic periodic paralysis; a reversible cause of paralysis to remember. SDJ Med 1997;50:357-8.
5. Darrow M, Brammer WK, Rowley A. Thyrotoxic periodic paralysis: two case studies. Arch Phys Med Rehabil 1995;76:685-7.
6. Zabelle JE. Thyrotoxic hypokalemic periodic paraly sis. Harefuah 1989;117:368-9.
7. Ober KP. Thyrotoxic periodic paralysis in United States. Report of 7 cases and review of the literature. Medicine (Baltimore) 1992;71:109-20.
8. Neal JM. Thyrotoxic periodic paralysis in a caucasian man: recognition and diagnosis. Indiana Med 1991;84:696-9.
9. Shayne P, Hart A. Thyrotoxic periodic paralysis terminated with intravenous propranolol. Ann Emerg Med 1994;24:736-40.
10. Bergeron L, Sternback GL. Thyrotoxic periodic paralysis. Ann Emerg Med 1988;17:843-5.
11. Berwaerts J, Verhelst J, Vandenbrucke M, et al. Thyrotoxic periodic paralysis, an unusual cause of hypokalemic periodic paralysis. Acta Neurol Belg 1996;96:301-6.
12. Kilpatrick RE, Seiler-Smith S, Levine SN. Thyrotoxic hypokalemic periodic paralysis; report of four cases in black American males. Thyroid 1994;4:441-5.
13. Ben David D, Malnick SD, Evrow E, et al. Thyrotoxic hypokalemic periodic paralysis. Lancet 1997;22:349,883.