A higher rate of early discontinuation of psychotropic medications was considered as a major problem in a clinical practice. Patients who had such beliefs easily discontinued their therapeutic sessions. In other research, fear of dependency and addiction are samples of false beliefs which influenced treatment.[2,3] Other false beliefs which existed in our culture may not be seen in other countries, although these false beliefs may be seen in another forms in other developing countries. The origin of these false beliefs is not well understood. They may originate from superstitions and lack of psycho education in the general community. It was suggested that patient education does have a positive effect on patient's compliance to medication.[4] To improve patient's compliance; many methods were used. In a recent article, research that examined such methods of interventions was reviewed. Forty-nine percent of the interventions were associated with a statistically significant increase in medication adherence and only 17 reported statistically significant improvements in treatment outcomes. Almost all the interventions that were effective for long-term care were complex, including combinations of more convenient care, information, counseling, reminders, self-monitoring, reinforcement, family therapy, and other forms of additional supervision or attention.[5]

In conclusion, psychiatrists, especially those who work in developing countries, are advised to carefully evaluate patients' compliance and their beliefs in psychiatric medications and management. This preliminary assessment could facilitate further management.

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Hypokalemic periodic paralysis as a presenting manifestation of thyrotoxicosis

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Hypokalemic periodic paralysis is an uncommon complication of thyrotoxicosis that occurs exclusively in male Orientals. A few case reports have emphasized its relative rarity among non-oriental populations, particularly among Arabs.[1-5] We report 5 patients who presented with hypokalemic paralysis and were treated in the King Fahad Central Hospital, Gizan, Kingdom of Saudi Arabia.

Patient 1 was a 25-year-old Saudi soldier from Faifa who presented with a 10-day history of recurrent and mostly nocturnal attacks of weakness of all limbs. The attack was aborted only when he was treated with intravenous infusion and potassium in the local hospital, on each occasion. The review of systems was normal. He denied dyspnea, weight loss, fever, heat intolerance, diarrhea, or vomiting. There was no facial weakness. He reported a few similar attacks approximately 4 months earlier. He was taking no drugs. Physical examination showed no jaundice, pallor, peripheral adenopathy, leg edema or exophthalmos. The pulse was 80/min with a blood pressure of 120/80 mm Hg, and the cardiorespiratory systems and abdomen were normal. He was conscious and well oriented with normal cranial nerves and intact sensory system. The muscle power was reduced to grade II in all groups of muscles in the upper and lower limbs. The reflexes were diminished and the plantar reflexes down going. The laboratory investigations are summarized in **Table 1**. Complete blood count (CBC), arterial blood gases (ABG) and chest x-rays were normal. Creatinine phosphokinase (CPK) was 82 U/L, urine pH was 6, sodium was 21 mmol/L and potassium was 9 mmol/L. He was treated with intravenous potassium chloride (KCl) infusion. He regained full muscle power within 6 hours and was discharged in a stable condition on carbimazole therapy.

Patient 2 was a 26-year-old Filipino electrician who had been working in Saudi Arabia for 4 years. He presented with a 4-hour history of difficulty in getting up from his bed and walking. He had several, similar and intermittent attacks during a period of 4 months prior to his presentation to the hospital. The review of systems was negative, and the family history was non-contributory. On

Table 1 - Laboratory data in patients with hypokalemic paralysis and thyrotoxicosis.

Test (normal range)	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
K (3.5-5.3 mmol/L)	1.5	2.3	1.9	2.6	2.5
Na (135-153 mmol/L)	146	143	143	138	141
Creatinine (50-100 umol/L)	82	41	72	88	74
BUN (2.5-6.4 mmol/L)	4	3.6	4.2	3.9	N
Ca (2.20-2.62 mmol/L)	2.37	2.1	2.6	na	na
T_3 (0.8-2.7 nmol/L)	4.1	6.3	12	8.5	4.3
T_4 (62-165 nmol/L)	170	195	260	>260	>260
TSH (mU/L)	<0.1	<0.1	<0.1	<0.1	<0.1
Thyroid scan	+		+	+	

K - serum potassium, Na - serum sodium, BUN - blood, urea, nitrogen, Ca - calcium, T_3 - triiodothyronine, T_4 - thyroxine, TSH - thyroid stimulating hormone, na - not available, N - normal complete blood count

examination the patient was well nourished with no jaundice, pallor, pyrexia, adenopathy, or neck goiter. There was no exophthalmos or lid lag. Examinations of the eyes, abdomen and the cardiorespiratory systems were normal. The blood pressure (BP) was 130/70 mm Hg with a pulse of 90/min. Central nervous system (CNS) examination showed normal cerebral functions, cranial nerves and sensory system. The muscle power was grade III in the proximal muscles of the pelvic and shoulder girdles, and was grade IV distally. Deep tendon reflexes were diminished in all limbs and plantar reflexes were down going bilaterally. The relevant laboratory results are shown in **Table 1**. Complete blood count and ABG measurements were normal. The patient recovered completely within 4 hours of treatment with KCl infusion and was later treated with an anti-thyroid drug.

Patient 3 was a 26-year-old Bangladeshi expatriate worker who presented with a 2-day history of recurrent inability to walk or elevate his shoulders or arms. The review of systems provided a history of palpitations, heat intolerance, and weight loss; but no fever, vomiting or diarrhea. On examination the patient had a BP of 150/80 mm Hg, a pulse rate of 140/min, tremors of outstretched hands, proptotic eyes, lid retraction and lid lag. The neck showed a diffuse moderate thyromegaly with a bruit over it. The cardiorespiratory systems and abdomen were normal. Higher cerebral functions and the cranial nerves were normal, but the reflexes were diminished globally with down going plantar reflexes. In the upper and lower limbs, the muscle power was grade III proximally and was normal (grade V) distally. A thyroid scan showed a diffuse thyromegaly with raised uptake of isotope. Electrocardiography showed some changes consistent with hypokalemia. He was treated with intravenous fluids and KCl. His muscle power improved to normal within 2-3 hours. He was started on Carbimazole 60 mg/day, Propranolol 40 mg three times a day. The serum potassium improved from 1.9 to 4.3 mmol/L and he was fully ambulant at the time of discharge.

Patient 4 was a Sudanese prisoner (30-years-old) who was admitted with a 2-month history of recurrent attacks of weakness of all limbs that were worse in the lower more than the upper limbs. The presenting episode started during the previous night. Before then there had been 3 similar attacks which occurred usually after dinner. Review of systems confirmed tremulousness, palpitations, heat intolerance and weight loss, despite a good appetite but he had no diarrhea or vomiting. Examination revealed bilateral exophthalmos BP of 160/75 mm Hg, regular pulse of 120/min, diffuse, thyromegaly (soft texture, grade 2) and tremors. The cardiorespiratory systems and abdomen were normal; CNS examination was normal except for absent reflexes in the limbs. The muscle power was diminished to grade III in the upper limbs and to grade II in the lower limbs. The muscle power improved markedly after intravenous infusion of potassium. He received 12 mci of radioactive iodine for the hyperthyroidism and was discharged on propranolol. He was readmitted with another episode of weakness a week later. Serum potassium was 1.9 mmol/L with an arterial pH of 7.45 and, abnormal ECG changes (T wave inversion). He responded to the treatment with intravenous KCl

infusion, and he was started on carbimazole and propranolol on discharge. He became euthyroid 4 months after radioactive iodine was used for thyroid ablation. There was no recurrence of paralysis during the follow-up period of one year.

Patient 5 was a 26-year-old Saudi male who was diagnosed to have thyrotoxicosis 2 years earlier. He was taking a tapering dose of neomercazole until 3 months prior to presentation when he discontinued the drug. He presented with a 12-hour history of progressive weakness that started in the lower limbs and later in the upper limbs. The attack followed a heavy breakfast. Past medical history indicated that he had a similar episode 2 years before the diagnosis of thyrotoxicosis and its treatment. Physical findings included proptosis, mild lid lag and tremors of both the hands; regular pulse 100/min, BP of 130/80 mm Hg, normal examination of the chest, abdomen, cardiovascular and CNS. The motor power was reduced to grade II in all the muscles of the upper and lower limbs. Arterial blood gases were normal and the ECG showed depressed T-waves in all leads. Diffuse thyromegaly and an increased radioisotope uptake were documented by thyroid scan. The motor deficit disappeared completely within 8 hours after infusion of potassium; and the serum potassium rose to 4.3 mmol/L. He was discharged on neomercazole and remained well during the follow-up period.

All the patients reported here had recurrent acute limb weakness, low serum potassium ranging from 1.5-2.6 mmol/l and thyrotoxicosis that was indicated by elevated serum T3 and T4 and low thyrotropin (TSH). The duration of the episodes lasted from 2-6 hours. All the patients responded to potassium infusion. Two of the cases had Grave's ophthalmopathy. The thyrotoxicosis was treated with anti-thyroid drugs in 3 patients and radioactive iodine in one patient. One of the patients who received radioactive iodine therapy had a recurrence of flaccid paralysis a week after treatment, but further attacks were prevented by achieving an euthyroid status with the addition of anti-thyroid drugs. The findings in all of the patients reported are consistent with hypokalemic periodic paralysis in patients with thyrotoxicosis.[1-5] The hypokalemic paralysis associated with thyrotoxicosis shares similar characteristics with the familial form, which is inherited as autosomal dominant trait in two-thirds and occurs sporadically in the other third.[5] None of our patients had a family history of periodic paralysis.

Characteristically, thyrotoxic hypokalemic period paralysis (THPP) occurs most commonly in young men as was found in our patients. In a study of 45 Chinese adults with this disease, all but one, were male.[1] Four of the 5 cases reported here demonstrated the relatively uncommon occurrence of hypokalemic periodic paralysis (HPP) as the initial presentation of thyrotoxicosis.[1] This is in agreement with the observations in earlier reports.[2] For example, only 29.5% of 45 patients had a known history of thyrotoxicosis before the presentation with HPP.[1] In another report, THPP was the presenting symptoms in 6 of 8 patients.[4] Clinical manifestations of thyrotoxicosis were present in 3 of 5 cases in this report and in 60% (27 of 45) among Chinese patients presenting with paralysis.[1] Episodes of acute limb weakness occur most frequently during the night and are often precipitated by meals that are rich in carbohydrates.[1,4] Delay in the diagnosis of thyrotoxicosis in patients with HPP was highlighted in a report of 8 patients[4] and is exemplified by 2 of our patients who had a few episodic attacks that responded each time to KCl infusion in the referring medical centers without the recognition or diagnosis of thyrotoxicosis.

The pathogenesis of HPP in thyrotoxicosis is unknown. It has been hypothesized that it may involve the hyperactivity of the sodium:potassium pump of the cell membrane, the activity of which is stimulated by thyroid hormones.[1] Correction of hypokalemia, the mainstay of initial therapy of this disorder, usually leads to a dramatic improvement.[1,2,4] However, a rebound hyperkalemia that may complicate therapy must be avoided.[1] None of the patients reported here had a serum potassium level of >5mMol/L during treatment or at the time of discharge. It is to be emphasized that HPP may be recurrent unless the underlying thyrotoxicosis is recognized and treated adequately.[2,3] This is exemplified by patient 5 who had the reappearance of the syndrome after discontinuing the anti-thyroid drugs; and by patient 4 who had an attack during the interval between radioactive iodine treatment and the attainment of an euthyroid status.

Several reports stated that THPP occurred exclusively among young Asian or Oriental men and was extremely rare in Caucasians, African-American, Africans, Hispanics and Arabs.[1-4] The cases reported here including 2 Saudi Arabs, confirm the occurrence of this syndrome among persons of various nationalities. Non-availability of biochemical tests for thyrotoxicosis may be a major cause of under-diagnosis and therefore, under-reporting in many primary and secondary health care centers in the developing world. Furthermore, a low index of suspicion of this disorder is probably reinforced by previous reports of its rarity among non-Oriental populations. The diagnosis of THPP should always be considered in all patients with acute muscle weakness and prompt diagnosis, and treatment of thyrotoxicosis in such patients may prevent recurrence of this syndrome.

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