Thyrotoxic Periodic Paralysis

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SUMMARY

A case of thyrotoxic periodic paralysis in a young Chinese male is reported. Attacks of paralysis ceased once the patient was rendered euthyroid on appropriate treatment. The features of thyrotoxic periodic paralysis are briefly outlined.

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Periodic paralysis complicating hyperthyroidism occurs not uncommonly in Oriental patients, but only rarely in White patients. Engel[1], in a review of the literature, found that 90% of the patients on record were Japanese, in whom the incidence of periodic paralysis varied from 2% to 8% of all thyrotoxics.[2-4] In reports from Hong Kong[5] and Singapore[6] thyrotoxic periodic paralysis occurred in about 2% to 5% of Chinese patients, whereas in Taiwan[7] this complication is surprisingly rare. This report describes a case of thyrotoxic periodic paralysis in a South African male of Chinese origin.

CASE REPORT

A 21-year-old Chinese male with an attack of paralysis was seen at his home on 12 February 1973. He first experienced attacks of severe weakness of his limbs in October 1971, as a student in Cape Town, with further attacks in February and July 1972, and again one week prior to the present episode. All the attacks were usually preceded by a strenuous game of tennis, followed by a heavy meal in the evening. The onset was heralded by stiffness of the leg muscles about 3 hours after supper, which progressed to symmetrical weakness of both upper and lower limbs to the extent that he could not move his limbs at all. Spontaneous recovery ensued after 24 - 28 hours. Since February 1972 he had lost 30 kg in weight, and he had a poor appetite during the 3 - 4 months prior to the first consultation. For 6 weeks he had experienced palpitations, dyspnoea and tiredness on exertion. He sweated profusely and preferred cold weather. He also tended to lose his temper very easily, but denied any increased prominence of his eyes or any change in bowel habits. There was no family history of thyroid or paralytic diseases.

Examination showed an alert, moderately anxious patient with a pulse rate of 140/min, and a blood pressure of 130/60 mmHg. There was marked lid lag, with normal ocular movements and no exophthalmos. Moderately diffuse enlargement of the thyroid gland with a continuous bruit was found. Skin and hair were normal, but the palms were warm and sweaty. Neurological examination during the attack revealed marked weakness of the upper and lower limbs, involving mainly the proximal muscles. There was no muscle wasting. The tendon reflexes were diminished, and there was slight weakness of the trunk muscles. The cranial nerves were intact. There was no sensory abnormality. On recovery, his muscle power and reflexes returned to normal, but a fine finger tremor persisted. Serum potassium estimations done during and after the attack are shown in Table I.

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TABLE I. SERUM POTASSIUM LEVELS DURING AND AFTER ATTACKS OF PARALYSIS

Date	Serum K^+ (mEq/litre) During attack	After attack	Duration of attack
12 February 1973	**2,6**	**4,5**	**28 hours**
25 February 1973	**3,3**	**4,2**	**19 hours**

The following laboratory results were normal: haemoglobin, haematocrit, total and differential white cell count, serum sodium, chloride and urea. Blood sugar was 105 mg/100 ml, serum calcium 10,0 mg/100 ml and phosphorus 3,5 mg/100 ml.

Thyroid function studies: The Trilute ^{125}I column test (T_3) was 87% (normal for males 43 - 60%). Serum thyroxine iodine (T_4) by column was 11,7 μg/dl (normal 3,0 - 7,0). The free thyroxine index (T_7) was 20,0 (normal for males 2,9 - 9,2). Radio-iodine uptake by the thyroid was 79% in 6 hours (normal 15 - 45%) and 45% in 24 hours (normal 20 - 50%).

On 16 February 1973 the patient was started on 40 mg propranolol and 10 mg carbimazole every 6 hours. He was also instructed to take 2 Slow-K tablets every 6 hours at the onset of muscle stiffness. On 25 February 1973 after a heavy supper he experienced stiffness of his leg muscles, and took the potassium tablets as instructed. This did not prevent the onset of paralysis, although the duration of the attack was shortened (Table I). During the attack he had a pulse rate of 96/min and a blood pressure of 130/60 mmHg.

After 6 months of antithyroid treatment, the patient was clinically euthyroid, on a maintenance dose of 5 mg carbimazole twice a day. Results of repeat thyroid function tests were: T_3—62%; T_4—3,7 μg/dl; and T_7—4,5. To date, the patient has not had any further attacks of paralysis, in spite of having returned to his previous habit of a vigorous game of tennis, followed by a heavy evening meal.

DISCUSSION

Thyrotic periodic paralysis (TPP) and familial periodic paralysis (FPP) are similar in many respects. Both forms occur predominantly in males, although TPP tends to occur at a later age. During attacks, flaccid areflexic paralysis of skeletal muscle, which on electromyography demonstrates electrical inexcitability, occurs. The attacks are precipitated by excessive carbohydrate loading or by rest after exercise. The serum potassium is lowered, often to subnormal levels, with improvement of muscle weakness on treatment with potassium. Histologically, vacuolisation of muscle and dilatation of sarcoplasmic reticulum or T-system have been shown to occur in both forms.

There are, however, a few important differences between TPP and FPP. A family history is absent in TPP in contrast with FPP, although sporadic cases of FPP do occur. In TPP, spontaneous or induced attacks occur only during the hyperthyroid state, and cease once the patient is rendered euthyroid by appropriate therapy. With relapse to the hyperthyroid state, recurrence of paralytic attacks occurs. It appears that these patients have a latent metabolic defect which only becomes manifest in the presence of excessive thyroid hormone. In contrast, patients with FPP do not suffer more frequent attacks when rendered thyrotoxic by the administration of tri-iodothyronine or thyroid-stimulating hormone,[1] and they may, in fact, show improvement.[8]

Adrenergic blockade by means of reserpine has no effect on paralytic attacks in TPP,[9] which was confirmed by our patient who suffered a spontaneous attack while on adequate propranolol dosage. It is almost certain that there is an influx of extracellular potassium into muscle cells during attacks of paralysis, although the reason for this is not clear at present. Hyperaldosteronism does not play a role.[10] The glucose-insulin test is used to provoke an attack in susceptible patients because it initiates or accentuates this tendency for potassium to shift into muscle cells. The study of serum potassium levels, although valuable in diagnosis, does not correlate with the degree of muscle weakness during attacks,[9] intracellular potassium probably being more relevant.

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