CASE REPORT

Thyrotoxic periodic paralysis in a white woman

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A 24 year old white woman presented with sudden onset of flaccid quadriparesis and hypokalaemia. She was later found to be thyrotoxic. Paralysis resolved with potassium supplements, and after initiation of antithyroid medication she had no further episodes of hypokalaemic paralysis. To the best of the authors' knowledge, and after a Medline search, thyrotoxic periodic paralysis has not been described previously in a white woman.

A 24 year old white woman of English ancestry presented with a sudden onset of flaccid quadriparesis. There was no history of recent strenuous physical exercise. She denied any symptoms of hyperthyroidism: there was no weight loss, palpitations, heat intolerance, irritability, diarrhoea, or menstrual disturbance. She had no significant past medical history and no family history of thyroid disease or muscle disease.

On examination, pulse rate was 100 beats/min, blood pressure 120/70 mm Hg, and there was reduced tone and absent reflexes in all four limbs. She had a mild tremor but no lid lag and no signs of thyroid eye disease. She had a moderate size goitre with a soft bruit.

Investigations showed a serum potassium of 2.1 mmol/l (3.7–5.0) with normal sodium, urea, and creatinine. Electrocardiography showed a sinus tachycardia. She was given intravenous and oral potassium supplements (60 mmol over 12 hours) and the paralysis resolved rapidly with normalisation of serum potassium to 4.8 mmol/l. Subsequent investigations showed the following thyroid function tests: free thyroxine >77 pmol/l (10–25), thyroid stimulating hormone <0.01 mmol/l (0.4–4.0), and antithyroid peroxidase antibodies were strongly positive at >1000 IU/l (0–35).

The patient was started on carbimazole 40 mg daily and was advised to avoid strenuous exercise and carbohydrate-rich meals. Over the next few weeks her thyroid function tests normalised and she had no further episodes of hypokalaemic paralysis. She did not require β-blockers or any further potassium supplements after the presenting episode.

DISCUSSION

Thyrotoxic periodic paralysis with hypokalaemia occurs in 13%–24% of Orientals with thyrotoxicosis.[1] In this race it occurs 70 times more frequently in males than females[1] and usually occurs between the ages of 20 and 40 years reflecting the age of onset of thyrotoxic Graves' disease. It has also been previously reported in white people, native American Indians, black people, and Aborigines.[2] However, previous case reports involving white people have all described male patients.[3 4] To our knowledge, and after a Medline search using the key words hypokalaemia, thyrotoxicosis, and thyrotoxic periodic paralysis, this is the first described case of hypokalaemic thyrotoxic periodic paralysis in a white woman.

Our case is consistent with previously described cases of hypokalaemic thyrotoxic periodic paralysis in that the symptoms and signs of thyrotoxicosis are subtle and may be missed if they are not specifically looked for. Since the signs are subtle it is important to check thyroid status in all patients with hypokalaemic paralysis.

The mechanism of hypokalaemia is of a sudden influx of potassium into muscle by increased activity of sodium-potassium pumps. There is an increase in sodium-potassium ATPase activity in all patients who become thyrotoxic but in those individuals who are susceptible to periodic paralysis the increase in sodium-potassium ATPase activity is much higher despite similar degrees of hyperthyroidism.[1] In all patients with hyperthyroidism, with or without periodic paralysis, sodium-potassium ATPase activity returns to normal with establishment of the euthyroid state.[1] Increase in sodium-potassium ATPase activity is mediated by the influence of β-adrenergic stimulation and thyroid hormone. Exercise causes release of adrenaline and a carbohydrate load in a thyrotoxic state can cause a brisk insulin response, both of which promote potassium influx into cells. However, the mechanism seems to be different from that observed in familial hypokalaemic periodic paralysis since administering thyroxine in these patients does not precipitate episodes of paralysis[5] and once a patient with thyrotoxic periodic paralysis has become euthyroid episodes of hypokalaemic paralysis cannot be induced by strenuous exercise or carbohydrate-rich meals.[6]

One puzzling aspect of hypokalaemic thyrotoxic periodic paralysis is why it should affect males so much more frequently than females when Graves' disease effects females much more commonly than males. A study in Japanese men showed a 2.5-fold increase in frequency of HLA-DRw8 in those with thyrotoxic periodic paralysis compared with those with thyrotoxicosis without periodic paralysis.[7] This would suggest one HLA type to be involved in susceptibility of periodic paralysis in the thyrotoxic state and another for the susceptibility for Graves' disease. Indeed, thyrotoxic periodic paralysis is associated with other causes of hyperthyroidism other than Graves' disease such as a thyrotropin-secreting pituitary adenoma.[8] However, as to why males with a certain HLA type are more susceptible to periodic paralysis than females remains unanswered.

Management of the patient during an episode of hypokalaemic paralysis requires cardiac and electrolyte monitoring because of the risk of cardiac arrythmia, although cardiac arrest is uncommon.[9] Treatment of an episode is usually with potassium supplements, but recovery time does not correlate with the amount of potassium replaced and often there is a rebound hyperkalaemia. Because of this, others have recommended the use of non-selective β-blockers as first line treatment of paralysis since this will antagonise the hyperadrenergic state that has been implicated in the pathogenesis of thyrotoxic periodic paralysis and there has been no reported cases of rebound hyperkalaemia.[10] The risk of recurrent episodes of hypokalaemic paralysis may be reduced by β-blockade and avoidance of strenuous exercise and carbohydrate-rich meals and treatment with antithyroid

Summary points

- In patients with hypokalaemic periodic paralysis thyroid function tests must be requested since signs and symptoms of hyperthyroidism are often mild.
- If patients are treated with potassium supplements during an acute episode serum electrolytes must be closely monitored because of the risk of rebound hyperkalaemia.
- Alternatively, non-selective β-blockers may be used during an acute episode.
- Paralytic attacks only occur when patients are hyperthyroid.
- Risk of recurrent episodes of hypokalaemic paralysis may be reduced by β-blockade and avoidance of strenuous exercise and carbohydrate-rich meals while antithyroid drugs take effect.

drugs. Once patients are euthyroid they are not at risk of hypokalaemic paralysis. There is no evidence that potassium supplements reduce the frequency of hypokalaemic episodes and they are not usually necessary, as was the case with our patient.

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