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Sudden weakness in a young Chinese man

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A 31-year-old man from mainland China presented with sudden onset weakness of limbs of eight hours duration. He had woken up from bed with weakness. He was not on any medication; both parents and brother are healthy. On examination he had motor weakness with diminished deep jerks. Non-tender hepatomegaly was present.

Investigations revealed normal blood counts, urea, creatinine, glucose and electrolytes were normal, creatine kinase 1105 U/l (reference range: 10–195), alanine transaminase 68 U/l (5–40), aspartate transaminase 49 U/l (6–37), alkaline phosphatase 180 U/l (98–279), glutamyl transferase 128 U/l (10–50), plasma viscosity 1.57 mPas at 25°C (1.50–1.72). Electrocardiograms were normal and chest X-ray was clear. Peak flow was 560 l/min.

He recovered naturally and, while awaiting other results, was discharged. He was re-admitted within a week with hypokalemia of 1.8 mmol/l (3.5–5.5). The outstanding investigations were by then available. Free thyroxine 65.6 pmol/l (9–24), thyroid-stimulating hormone (TSH) <0.05 mU/l (0.49–4.67), free tri-iodothyronine 24 pmol/l (4–8.3). Antiparietal cell antibodies were positive, thyroid autoantibodies were not detected, ds-DNA, antinuclear antibodies, antismooth muscle, mitochondrial and extractable nuclear antigen, and Jo1 were negative. Muscle biopsy did not show significant abnormalities. Electromyography and nerve conduction studies were normal. By this time creatine kinase levels had normalised. Hepatitis B surface antigen and anti-Hb E were positive and Hb E antigen was negative.

Questions

- 1 What is your diagnosis?
- 2 How would you treat the weakness and what complications may arise from treatment?
- 3 Will the patient be prone to further paroxysmal weakness?
- 4 What other history would you consider important in this type of hepatitis and what will you inform the patient?

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Answers

QUESTION 1

Thyrotoxic periodic paralysis together with hepatitis B at low infective risk.

QUESTION 2

The patient was managed with parenteral potassium supplements with bedside monitoring. He was started on carbimazole and developed a rash which is a common adverse effect. The medication was changed to propylthiouracil which he tolerated well. Thyroid function tests after three weeks were: total free thyroxine 22 pmol/l and TSH <0.05 mU/l. The other complications of carbimazole include sore throat, mouth ulcers due to bone marrow suppression, and arthralgias. Patients should be informed of these side-effects; blood counts must be done and carbimazole stopped if agranulocytosis is noticed. These antithyroid drugs act by inhibiting oxidation and creating an intrathyroidal iodine depletion. Our patient is awaiting radioiodine therapy.

QUESTION 3

The episodes of weakness will cease once an euthyroid state is established. Meanwhile, short-term use of beta-blockers are effective. Avoiding strenuous exercise and avoiding high carbohydrate diets will help.

QUESTION 4

The condition suggests a possible history of parenteral drug use, tattoos and/or sexual activity. Our patient had not received any blood or blood products. The abnormal liver function tests are due to hepatitis. He should be informed that his blood and secretions are infectious to others and of the risks of chronic liver failure and hepatocellular carcinoma.

Discussion

Thyrotoxic periodic paralysis is a common cause of weakness in young people of Chinese ethnic origin and it rarely occurs in Caucasians. Interestingly, the subjects more often do not manifest the classical picture of thyrotoxicosis, which can be misleading. Thyrotoxic periodic paralysis classically starts in the early

Thyrotoxic periodic paralysis

- sudden onset weakness
- common in young Chinese men
- apparent lack of clinical signs of thyrotoxicosis
- treatment: correction of hypokalaemia, antithyroid drugs, beta-blockers, avoiding high carbohydrate diets and strenuous activity
- attacks cease with euthyroid status

Box 1

morning hours, usually following a day of strenuous activity. Speech is usually spared and weakness involves limbs. Almost all cases occur sporadically (95%) and in a series reported from Hong Kong, the subjects did not have a familial history of periodic paralysis.¹

Male preponderance is a recognised characteristic.² Thyrotoxic periodic paralysis is indistinguishable from idiopathic familial periodic paralysis but has a higher propensity for arrhythmia, cardiac arrest and respiratory failure. Hyperthyroidism *per se* can elevate liver enzymes.³ Hepatitis B may flare-up with thyrotoxicosis and achieving the euthyroid status may help remission.⁴

In thyrotoxic periodic paralysis, hypokalaemia develops due to an intracellular shift of potassium and not its depletion. A recent study identified increased Na⁺, K⁺-ATPase activity in platelets in thyrotoxic patients with or without periodic paralysis.⁵ *In vivo* sodium pump activity in muscle, kidney and liver is also increased in thyrotoxicosis and this activity is restored with treatment.⁶ A recent case report of a patient with Graves' disease and hepatitis,⁴ and our subject with hepatitis B, parietal cell antibodies and thyrotoxicosis, indicate the need for further studies to look for any immunological links in hepatitis B.

Final diagnosis

Thyrotoxic periodic paralysis.

Keywords: thyrotoxic periodic paralysis, hepatitis B, Chinese

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