Thyrotoxic hypokalaemic periodic paralysis

Introduction

Thyrotoxic hypokalaemic periodic paralysis is an endocrine emergency, and an infrequent neuromuscular complication of hyperthyroidism. It is not usually described as a complication in the western literature, and it is therefore likely to be missed when it presents, especially in migrants.

With increasing immigration, thyrotoxic hypokalaemic periodic paralysis is likely to occur more frequently. Ethnic minorities form 6.5% of the population in the UK, and it is forecast that by 2025 the UK population will increase by 10% to 64 million, with immigration accounting for approximately two-thirds of the increase.

This article reports two cases of acute onset of weakness resulting from thyrotoxic hypokalaemic periodic paralysis.

Case Report 1

A 24-year-old male Chinese student from southern China presented with sudden onset of generalized weakness. The evening before admission he had played football for 3 hours, and eaten a Chinese takeaway plus two hamburgers. He awoke in the early hours of the following morning unable to move his arms or legs. There was no history of any recurring illness and in particular there were no symptoms suggestive of hyperthyroidism.

On examination the only abnormality was a profound symmetrical muscle weakness more marked proximally than distally (Medical Research Council (MRC) classification grade 1–2). Sensation to light touch and pinprick was normal. There were no signs of thyrotoxicosis.

Laboratory investigations revealed the following (normal values in parentheses): serum potassium 1.2 mmol/litre (3.5–5.0); urinary potassium 14 mmol/litre (20–60); phosphate 0.18 mmol/litre (0.8–1.45); sodium, urea, and creatinine levels were normal. Electrocardiogram showed first degree atrioventricular block, prolonged QT interval and right axis deviation.

He was given 120 mmol of intravenous potassium supplement over 12 hours. Over the next 24 hours his serum potassium returned to 4 mmol/litre, with complete resolution of the weakness.

Subsequent investigations showed the following thyroid hormone measurements: thyroid-stimulating hormone (TSH) <0.08 mU/litre (0.5–6.0), triiodothyronine (T_3) 3.7 nmol/litre (1.0–2.5), free thyroxine (T_4) 53.7 pmol/litre (9.0–25.0), and thyroid peroxidase antibodies 124 IU/ml (0–75). He was HLA B46 and D9 positive. Ultrasonography of the thyroid gland revealed a mildly enlarged, heterogeneous thyroid with no discrete nodules and increased vascularity throughout.

He was started on treatment with propranolol and carbimazole and returned home to China to seek further medical treatment.

Case Report 2

A 33-year-old Afro-Caribbean male had awakened from sleep with generalized weakness, and collapsed when attempting to get out of bed. He reported a similar previous episode which had resolved spontaneously. There were no precipitating factors with meals or exercise.

On physical examination, he had a palpable smooth goitre, but no clinical features of hyperthyroidism. He had proximal muscle weakness in all extremities but more pronounced in the legs. He had normal tone, sensation and reflexes.

The results of laboratory investigations were as follows: serum potassium 1.9 mmol/litre, magnesium 0.57 mmol/litre. Electrocardiogram revealed sinus tachycardia with diffuse ST/T wave changes. After treatment with 80 mmol/litre of intravenous potassium supplement, his serum potassium returned to 3.7 mmol/litre over the next 24 hours, with complete resolution of the weakness.

Thyroid hormone measurements showed thyroid-stimulating hormone (TSH) <0.01 mU/litre, free thyroxine (T_4) 68.2 pmol/litre, free triiodothyronine (T3) 27.1 pmol/litre, and thyroid peroxidase antibodies 605 IU/ml. He was HLA A23 positive. The patient was commenced on propranolol and carbimazole.

Discussion

Thyrotoxic hypokalaemic periodic paralysis was first described in the German literature in 1920 by Rosenfeld. The first case in the English language was reported in 1931 by Dunlap and Kepler from the Mayo clinic.

Ninety per cent of all cases reported in the literature are Orientals, with an incidence of 13–24% mainly among Japanese and Chinese (McFadzean and Yeung, 1967). The exact incidence is not known among other ethnic groups, but it occurs less frequently (Kilpatrick et al, 1994; Dixon and Jones, 2002) and is unusual in Europeans. The onset is usually between the ages of 20 and 40 years, with a male:female ratio of 20:1 (McFadzean and Yeung, 1967).

While a family history is rare, thyrotoxic hypokalaemic periodic paralysis has been associated with some human leukocyte antigens such as HLA-DRw8, A2, Bw22, Cw3, B5, DRw9, Bw46 and A23. The disorder is characterized by transient recurrent episodes of muscular weakness or paralysis; proximal muscles are affected more severely than distal muscles. Sensation and mental functions are unaffected. The onset is sudden, and paralysis typically resolves within 3–36 hours. Hypokalaemia is the biochemical abnormality during an attack. The mechanism of hypokalaemia is thought to be an influx of potassium into muscle by increased activity of sodium–potassium pumps.

Elevated thyroid hormone levels upregulate the activity of muscle, kidney, liver, and platelet sodium–potassium adenosine triphosphate (ATP)-ase. Sodium and calcium channels located on the sarcoplasmic reticulum may play a role in thyrotoxic hypokalaemic periodic paralysis sus-

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ceptibility, but the mechanism remains to be elucidated (Ko et al, 1996). Possible precipitating factors for occurrence of paralysis include ingestion of a high carbohydrate load and strenuous physical activity followed by a period of rest (McFadzean and Yeung, 1967), as in case 1. Other precipitating factors include alcohol ingestion, trauma, cold exposure, emotional stresses, and medications such as diuretics, adrenalin, insulin, and acetazolamide.

Treatment of thyrotoxic hypokalaemic periodic paralysis includes the administration of supplemental potassium to hasten recovery of muscle function and to avoid cardiac complications. The definitive treatment is the management of the hyperthyroid state. While awaiting normalization of thyroid status, avoidance of precipitating factors is recommended. BJHM

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A case of emphysematous pancreatitis

A 58-year-old man with a history of diabetes mellitus, hypertension and ischaemic heart disease attended the accident and emergency department with a 24-hour history of generalized abdominal pain. Clinical examination revealed features of septic shock. Biochemical abnormalities included an elevated serum amylase (2047 μmol/litre). Computed tomography (CT) of the abdomen was performed with intravenous and oral contrast medium (*Figure 1*). This demonstrated evidence of extensive air within the pancreas in keeping with emphysematous pancreatitis. Despite immediate resuscitation, antibiotic therapy and pancreatic necrosectomy, the patient died.

Discussion

Pancreatitis is most commonly caused by alcohol or gallstones and has a mortality of up to 50%. Abscess formation in the necrotic pancreatic tissue occurs in 20% of cases and carries a poor prognosis (Bazan and Kim, 2003). Fermenting bacteria generate carbon dioxide and nitrogen, resulting in an emphysematous pancreas. Diabetic patients are especially vulnerable to developing gas as a consequence of impaired mechanisms of glycolysis (Grayson et al, 2002).

Percutaneous intervention with CT guidance may be indicated if pseudocysts have formed. Evidence of necrotic pancreatic tissue on CT indicates an increased risk of failure to respond to conservative management. Surgical resection of necrotic tissue is often required as was the case for this patient (Bazan and Kim, 2003). This case confirms that CT is the modality of choice to evaluate the severity of acute pancreatitis and its complications (Daly et al, 1995). BJHM

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Figure 1. Computed tomography scan of the abdomen with oral and intravenous contrast shows complete replacement of the pancreas by gas as a result of severe necrotizing emphysematous pancreatitis, with no normally enhancing pancreatic tissue and stranding of the mesenteric fat.