CASE REPORT

THYROTOXIC HYPOKALEMIC PERIODIC PARALYSIS

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Abstract:

Hypokalemic periodic paralysis is an uncommon complication of thyrotoxicosis. It usually affects men of Oriental origin and presents with acute and profound muscle weakness especially of the lower extremities. We report a 46 year old man from Kuwait with sudden paralysis of both legs. Physical examination showed signs of thyrotoxicosis and laboratory tests revealed severe hypokalemia, very low serum thyroid-stimulating hormone and very high free thyroxin. The motor deficit regressed within 8 hours of 80 mmol of KCl infusion. Awareness of such a problem is essential to avoid life threatening cardiac arrhythmia. Once recognized the treatment is simple and the prognosis is excellent.

Keywords: Hypokalemia-Periodic Paralysis; Thyrotoxicosis-Thyroxin

Introduction:

Hypokalemic periodic paralysis is a rare condition known to occur in two distinct forms: familial autosomal dominance with high penetrance in males or sporadic in association with hyperthyroidism. In both forms the classical presentation is recurrent weakness of the lower limbs in the morning after exercise or ingestion of a high carbohydrate meal. The typical patient would be a male aged between 20 and 40 years. In THPP the clinical features of hyperthyroidism such as hand tremors, exophthalmos or goiter may not be apparent. The diagnosis in such a case could be easily overlooked and considered to be hysterical or functional in origin. In fact paralysis might be the presenting symptom of hyperthyroidism. Serious complications like cardiac arrhythmias or respiratory failure may occur in neglected cases.

Case Report:

A 46-years old Kuwait man was brought to the emergency department of Al Amiri Hospital with severe weakness of the lower extremities. He woke up in the morning unable to get out of bed. He had a similar complaint one week prior to this but it was a mild episode that lasted for two hours and resolved spontaneously and completely. There was no history of palpitation, diarrhoea, diaphoresis or heat intolerance. He denied taking any medications or herbal products. There was no family history of thyroid disease or periodic paralysis.

Physical examination revealed a well-developed gentleman in no acute distress. Pulse was 65 beats/min, respiratory rate 16/min, blood pressure 140/80 mmHg and temperature 37°C. He was apprehensive with a fine hand tremor and diffuse thyroid swelling. Bruit was heard over the enlarged thyroid gland. Cranial nerves were intact. Power in the lower extremities was 1/5 with hyporeflexia. In the upper limbs the power was 2/5 with depressed biceps reflexes. There was no sensory deficit.

Electrolyte values on admission were sodium 133.8 mmol/L, potassium 1.76 mmol/L, magnesium 0.82 mmol/L, calcium 2.4 mmol/L and glucose 6.3 mmol/L.

The haemogram was normal and creatinine phosphokinase was not elevated (37 IU/L). The electrocardiogram (ECG) showed first degree heart block, right bundle branch block and the presence of U waves. There were no atrial or ventricular arrhythmias.

The patient received 80 mmol of intravenous potassium chloride over 8 hours during which there was a marked improvement of muscle power. The serum potassium was maintained at
Hypothyroid periodic paralysis has been associated with low serum potassium and biochemical thyrotoxicosis with or without clinical features of hyperthyroidism. The thyroxin excess need not be secondary to Grave’s disease as cases of THPP has been reported with toxic nodular goiter, solitary toxic nodule and with exogenous thyroxin intake used to reduce body weight⁴¹.

The hallmark of the condition is acute systemic weakness associated with low serum potassium and biochemical thyrotoxicosis with or without clinical features of hyperthyroidism. The thyroxin excess need not be secondary to Grave’s disease as cases of THPP has been reported with toxic nodular goiter, solitary toxic nodule and with exogenous thyroxin intake used to reduce body weight⁴¹.

The muscle weakness generally affects the lower extremities especially the proximal muscles. The upper limbs are involved to a lesser extent with deep tendon reflexes typically sluggish or absent all over⁶. The respiratory and cranial nerve musculature is rarely involved. The level of consciousness, cerebellar functions and sensation are always intact⁸⁰.

An ECG usually shows changes of hypokalemia: U waves, flat T waves and QT prolongation. Conduction defects and various forms of atrial and ventricular tacharrhythmias have been described. Our patient had first degree heart block and right bundle branch block in addition to the classical changes of hypokalemia. This form of ECG changes is similar to that described in the thyrotoxic Chinese patients of McFadzean and Yeung⁸. THPP has striking similarities to the familial form of periodic paralysis (FPP). Both present with a similar history, clinical findings and laboratory figures. In both conditions males are affected more than females. However the male/female ratio in THPP is 20:1 while in FPP the ratio is 3:1 to 12:1. Family history is common in FPP but it is rarely encountered in THPP⁶⁰. Strenuous exercise, heavy carbohydrate meal intake or alcohol ingestion predispose to the paralysis which typically occurs the morning following these activities²⁴.

The mechanism by which hyperthyroidism induces the paralysis is not well understood. However the emphasis has been directed to the Na⁺-K⁺-ATPase pump. The activity of this pump is increased by thyroxin resulting in increased intracellular trans-

Factors that augment the ATPase pump include heavy carbohydrate loads, insulin and androgens. Estrogen and progestrone inhibit the ATPase pump. These findings partially explain the predominance of males and Asians with THPP as rice and soft drinks are consumed in large amounts in Asia and in the Arabian Gulf region. Westernisation of the feeding habits to a greater protein intake has resulted in the decline of number of cases of THPP in Asian immigrants⁷. The occurrence of the paralysis in the morning might be explained by the diurnal variation in potassium movement with influx of potassium from the extracellular compartment into skeletal muscles at night and vice versa during the day⁸. In addition, thyroxin increases tissue responsiveness to beta adrenergic stimulation, also linked to increase in Na⁺–K⁺-ATPase activity. The use of a non-selective beta blocker such as propranolol helps to abort the acute attack and prevent further relapses⁹.

The cornerstone of treatment of the paralytic attack is potassium supplementation either by careful controlled parental infusion or by oral administration of potassium chloride. Careful monitoring to avoid hyperkalemia is essential as cases of rebound hyperkalemia have been described. This occurs because of the release of the potassium from the intracellular compartment as the paralysis improves and the muscles start to contract. Careful monitoring of cardiac arrhythmias induced by either hypoxia or hyperkalemia is advised⁹.

Propranolol has been shown to reduce the relapse rate and is useful in the treatment of the acute episode³⁵. Selective beta-1-blockers are not effective⁹. As the acute episode subsides attention should be directed toward the original problem which is thyrotoxicosis. This can be dealt with in the usual case i.e. radio-iodine therapy, surgery or with the use of anti-thyroid drugs.

Conclusion:

A young healthy-looking man with unexpected generalized paralysis and an intact sensorium might be considered hysterical by an unwary physician in a crowded emergency department. This would be especially true if the patient lacked any thyrotoxic symptoms or signs (which is not uncommon in THPP).

Serious complications could ensue if the patient was neglected or treated improperly. In fact, several patients described in the literature were “treated” initially with muscle ointments, analgesics and vitamins.

Prompt recognition safeguards against potentially serious outcomes. Potassium administration during the attack will improve the weakness dramatically and the use of propranolol and potassium supplements will prevent the attacks. The definite treatment is reversal of the thyrotoxic state.
References:


