

CASE REPORT

A CASE OF THYROTOXIC HYPOKALEMIC PARALYSIS

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An Indian male patient with acute quadriplegia with hypokalemia as a rare initial presentation of thyrotoxicosis is presented in this case report.

Key words : thyrotoxicosis, quadriplegia, hypokalemia

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Introduction

Thyrotoxic hypokalemic paralysis (THP) is an unusual complication of a fairly common disease, hyperthyroidism – it is most commonly a complication of Graves' disease but can occur with any etiology of thyrotoxicosis (1).

THP is characterized by transient, recurrent episodes of flaccid paralysis affecting proximal muscles more severely than the distal muscles. During the episodes of weakness, hypokalemia is invariably present and the severity of weakness correlates with the degree of hypokalemia. It is most commonly seen in patients between the third and fifth decade and is more frequently observed in males than in females (2). Manifestations of thyrotoxicosis vary from subtle to quite obvious and usually precede or coincide with onset of the paralytic attack.

We describe a case of a male who presented with acute onset quadriplegia with hypokalemia as the initial presenting feature of thyrotoxicosis.

Case report

A 40 year old male patient presented with complaints of sudden onset weakness in all four limbs, for 3 days duration. The weakness occurred simultaneously in all limbs and reached a peak 4 hours prior to admission. There was no proximal\distal predilection, sensory loss, bladder or bowel involvement. There was no past history or family history of similar episodes. The patient also had complaints of palpitations and diarrhoea-off and

on for 1 month prior to the onset of weakness. There was no history of trauma, chest pain, dyspnoea, swelling over body, perception of missed beats, weight loss or change of appetite. On examination the patient was afebrile, well built, oriented in time, place and person with a pulse of 150/min, regular, bounding and BP 180/90mmHg. On neurological examination higher mental functions and cranial nerves were normal. The muscle bulk and tone was normal in all four limbs. The power was 0/5 in all four limbs. The deep tendon reflex (DTR) in upper limb and lower limb were exaggerated. There was no clonus. Sensory and posterior column examination was normal. Bilateral plantar response was flexor. Rest of the general physical examination and systemic examination including spine was normal. There were no eye or skin signs suggestive of thyrotoxicosis.

All the routine biochemical investigations including liver and renal functions were normal. Serum potassium was 1.9meq/L and creatine phosphokinase was 88U/L. The ECG revealed sinus tachycardia. In view of hypokalemia associated with quadriplegia and brisk DTR with a past history of palpitations and diarrhea a possibility of THP was thought and thyroid function was assessed. The thyroid function revealed T3 = 250 ng/dl, T4 20mg/dl, serum TSH .01 /mU/L, confirming the diagnosis of thyrotoxicosis and associated THP. The patient was given treatment for hypokalemia and neomerzazole 10mg tid along with propranolol 20 bid. was administered. There was a dramatic clinical improvement in patient's motor power in the ensuing

24 hours and complete neurological recovery was achieved in the next 2 days. Further investigations into the cause of thyrotoxicosis revealed the presence of a multinodular goiter on ultrasound of neck. On follow up after 3 months, the repeat T3 180mg/dl, T4 12mg/dl and TSH 0.1 mU/L were obtained.

Discussion

Periodic paralysis is a condition characterized by episodic weakness of the muscles and may be hypo, normo or hyperkalemic type. The commonest of these is hypokalemic periodic paralysis. The mechanisms of periodic paralysis is believed to be an inability of the muscle membrane to propagate an action potential (3). Periodic paralysis could be primary or secondary when associated with thyroid, renal or gastrointestinal disease leading to potassium loss or retention.

In the presence of hyperthyroidism, periodic paralysis is invariably of the hypokalemic variety (4). The incidence of periodic paralysis with thyrotoxicosis is very low in the Western population but is particularly high in oriental races where it occurs in 8-16% of thyrotoxic males and 0.2-0.45% of females (4). From India, reports of THP are scanty (5). The precipitating factors include high carbohydrate diet, stress and episodes of heavy physical activity. Attacks occur usually at rest or at night and are seen more frequently during the warmer months (6).

The clinical features of thyrotoxic periodic paralysis are similar to that of primary hypokalemic periodic paralysis with most patients presenting with acute flaccid paralysis with depressed/absent DTRs. In a minority of patients, the DTRs may be brisk and may provide the clue to the underlying thyrotoxicosis. Thyroid hormones increase the activity of sodium pump resulting in increased intracellular potassium. This may be a reason why periodic paralysis seen with thyrotoxicosis is typically hypokalemic (7).

The treatment of THP has two components, Immediate correction of hypokalemia is required to improve muscle weakness. Close attention should be given with parenteral potassium replacement since the hypokalemia is due to an intracellular shift and hyperkalemia can result quickly with restoration of normal sympathies. The other component is effective treatment of thyrotoxicosis because paralytic episodes cease after the euthyroid state is achieved. Initial management with beta blockers and antithyroid medication work well, but recurrence of

hyperthyroidism and paralytic episodes can occur after discontinuing antithyroid medications. Propranolol has been used for prophylaxis and reduces the incidence of spontaneous episodes of THP (3, 8). Acetazolamide which is effective in the prophylactic treatment for familial periodic paralysis, however provides no protection for THP and may worsen an attack (3, 8).

To summarise, the possibility of THP should always be kept in any patient who presents with hypokalemic paralysis especially with brisk/exaggerated tendon reflexes. The clinical signs of thyrotoxicosis may vary from subtle to quite obvious and a high index of suspicion is required to establish the diagnosis.

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