Case Report

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Dropped Head Syndrome Associated with Hypothyroidism and Hypokalemia

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Abstract

Dropped head syndrome (DHS) is known entity and has multiple causes. Dropped head syndrome has been reported both in hypokalemia secondary to several causes and in hypothyroidism as such. But its association with hypothyroidism and hypokalemia has not been reported so far. We present a case of DHS associated with hypothyroidism and hypokalemia. A 35-years-woman presented with dropped head syndrome. Biochemical evaluation revealed hypokalemia and hypothyroidism. Other causes of hypokalemia have been ruled out. The patient responded well to intravenous potassium initially but after institution of thyroxine potassium again started falling down. Therefore oral potassium chloride was continued in small doses for next four weeks. At end of six weeks, her TSH normalized and potassium was maintained in normal range without replacement.

Keywords: Dropped Head Syndrome; Hypothyroidism; Hypokalemia.

Introduction

Dropped head syndrome (DHS) is characterized by severe neck extensor weakness, resulting in chin-on-chest deformity in the standing or sitting position, which is correctable by passive neck extension. The syndrome has a number of causes. It may be a presentation of a variety of neuromuscular disorders or it may occur in isolation (1-3). Hypothyroidism is often accompanied by myopathy with proximal distribution. Hypothyroidism has been described as one of the cause of DHS (4). DHS has also been described as lone manifestation of hypokalemia (5-6). DHS with hypothyroidism and hypokalemia to our best knowledge has not been reported in literature so for. We illustrate a case of DHS associated with hypothyroidism and hypokalemia.

Case Report

35 year old women presented with weakness of extensor muscles of neck to our emergency. There was no history suggestive of altered sensorium, convulsion, visual, respiratory, bulbar weakness or weakness in any other part of body or. Initially we thought it could be part of conversion disorder, but subsequent to laboratory reports we were clear in our mind that it was most likely DHS due to hypokalemia. She had no symptom suggestive of hypothyroidism but had history of menorrhagia.

She was having thin built, had no pallor or facial puffiness. Her blood pressure was normal. She had normal higher mental functions. Her cranial nerves, sensory and sphincter functions were intact. The patient had weakness of extensor neck muscles, no other muscle weakness; deep tendon reflexes were normal and she had flexor plantar response. She had no thyromegaly. Rest of the systemic examination was normal.

Laboratory investigations revealed normal hemoglobin, normal ESR, low potassium (initial potassium 1.7meq/L) and normal sodium. Serum creatine phosphokinase (CPK) level was high (800 IU/L). Her ECG showed ST segment depression and U wave pattern in V2-V3. Electromyography and nerve conduction studies were normal.

She was put on potassium supplementation and

her condition improved in next twenty-four hours. Her thyroid stimulating hormone was high (98.45μIU/ml) and triiodothyronine (T3) was normal, but thyroxine (T4) was low (1.0 μg/ml). She had positive serum anti-TPO antibody titre (300IU/ml). DHS associated with hypothyroidism and Hypokalemic was diagnosed based on clinical and biochemical parameters. No other cause of hypokalemia could be detected. Her 24-hour urinary potassium excretion was 15.10 meq/L (normal range 25-120 meq/L). She had normal serum magnesium and urinary calcium excretion which ruled out the likelihood of Gitelman's syndrome. Her Urine pH was within normal limit, plasma rennin activity (PRA) was measured normal (2.7 ng/ml/h) and computed tomography (CT) of abdomen showed no abnormality in both adrenal glands.

During hospitalization, the patient was treated with intravenous potassium (IV potassium chloride in normal saline, via peripheral vein at a rate of 20 meq/h) and simultaneously started on oral potassium chloride (KCL) 20-30 meq QID. Patient showed improvement in her weakness with in twenty four hours with near normalisation of potassium. Potassium supplementation was stopped as potassium completely normalised on next day. After starting oral levothyroxine replacement (levothyroxine 50μg/day), potassium levels again started falling down. Therefore patient was put all over again on oral supplementation of potassium chloride solution (20meq thrice daily) for another 4 weeks after which KCL supplementation was stopped to see whether she could maintain potassium levels without any replacement. Serum TSH normalised in six weeks with normal serum potassium levels. With adequate control of hypothyroidism, the patient did not have the need to take potassium supplementation and had no further episode of DHS or any occurrence of hypokalemic periodic paralysis during eighteen months follow up.

Discussion

In a patient presenting with DHS, associated conditions must first be considered [1]. Hypokalemic paralysis may be caused by a temporary shift of potassium into cells, or a generously proportioned deficit of potassium as a consequence of severe renal or gastrointestinal potassium loss. Hypokalemic periodic paralysis is the most common periodic paralysis, but a infrequent channelopathy manifested by episodic flaccid weakness secondary to abnormal sarcolemal excitability. Thyrotoxicosis is the most frequent cause of secondary hypokalemic periodic paralysis [7,8]. Hypokalemic paralysis is an unusual presentation of hypothyroidism [9-11]. Disturbances of potassium equilibrium can produce a wide-ranging disorders including myopathy, marked muscle wasting, diminution of muscle tone, power, and reflexes and dropped head syndrome [12].

In our patient, there was no history of diarrhoea, vomiting, or diuretic abuse. There was no suggestion of Barter's syndrome on clinical or biochemical justification. Normal serum magnesium and urinary calcium excretion ruled out the possibility of Gitelman's syndrome. Similarly, clinical features of renal tubular acidosis were not seen in this patient [13]. Laboratory finding such as normal urinary pH and lack of hyperchloremia during episode of DHS also excluded the possibilities of renal tubular acidosis.

The persistent hypokalemia during early periods of thyroxine replacement can be due to the fact that thyroxine in pharmacological doses can cause increased potassium excretion and water diuresis in patients with myxedema during initial part of therapy [14]. This may result in hypokalemia, particularly in a patient with malnutrition and low stores of total body potassium.

Conclusion

The association of periodic hypokalemic paralysis with hypothyroidism has been described in only few case reports, but association of dropped head syndrome with hypothyroidism and hypokalemia has not been reported so far.

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