

Sporadic hypothyroidism-related hypokalemic paralysis: Diagnosis in a resource-poor setting

Nadasha Kadeeja¹, Nivetha Senthilnathan¹, Stalin Viswanathan¹,
Rajeswari Aghoram²

¹Department of General Medicine, Indira Gandhi Medical College and Research Institute, ²Department of Neurology, JIPMER, Puducherry, India

ABSTRACT

Hypothyroidism and distal renal tubular acidosis causing hypokalemic paralysis (HP) have been described only in four female patients. HP as the initial manifestation of uncomplicated diabetes has been reported only in three young males. We report two middle-aged patients presenting with gradual-onset areflexic quadriparesis and neck flop, associated with urinary potassium losses, and recovering over 3 days. The male patient with alcohol abuse had urine pH >5.5 and hyperchloremic metabolic acidosis due to renal tubular acidosis and hypothyroidism. The second, a hypertensive female, had metabolic alkalosis, hypomagnesemia, and diabetes mellitus diagnosed at admission. Both these patients improved with intravenous and oral potassium supplementation.

Keywords: Diabetes mellitus, hypokalemic paralysis, hypothyroidism, renal tubular acidosis

Introduction

Neuromuscular manifestations of hypokalemia include fatigue, lethargy, skeletal muscle paralysis, ileus, urinary retention, and rhabdomyolysis.^[1] Thyroid myopathy presents as stiffness, cramps, weakness, and rhabdomyolysis.^[2] Hypokalemic paralysis (HP) with thyroid disease is seen in younger individuals with thyrotoxic periodic paralysis and more common in Asians.^[3] HP as the initial presentation of hypothyroidism is unusual. We describe two middle-aged individuals presenting with HP and found to have hypothyroidism with distal renal tubular acidosis (DRTA) (Case 1) and diabetes (Case 2), respectively.

Case Reports

Case 1

A 56-year-old alcohol consumer presented with weakness of

all four limbs. Two-day ago, he had difficulty in walking; within 24 h, he developed quadriparesis. There was no bowel/bladder, oculopharyngeal, or respiratory weakness. His sensorium remained normal. He had had speech and hearing impairment since childhood (11 years). There was no preceding diarrhea, vomiting, fever, or drug intake. He had binged alcohol on the morning of the weakness. His past and family histories were noncontributory.

On examination, he was thinly built, with pulse 78/min, blood pressure 130/90 mmHg, and respiration 20 breaths/min. He had hypotonic flaccid quadriparesis (1/5 MRC lower limbs; 3/5 upper limbs), hyporeflexia (upper limbs), areflexia (lower limbs), mute plantars, neck flop, and a single-breath-count of 5. The thyroid gland was not palpable. Investigations are in Table 1. In view of normal anion-gap hyperchloremic metabolic acidosis and urine pH >5.5, a diagnosis of DRTA was made. Other urinary electrolytes (Ca, Cl, and HCO₃), urine osmolality and autoimmune workup (for hypothyroidism, systemic lupus, and Sjogren's) could not be performed.

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Kadeeja N, Senthilnathan N, Viswanathan S, Aghoram R. Sporadic hypothyroidism-related hypokalemic paralysis: Diagnosis in a resource-poor setting. *J Family Med Prim Care* 2017;6:862-4.

Address for correspondence: Dr. Stalin Viswanathan,
Department of General Medicine, Indira Gandhi Medical College
and Research Institute, Kathirkamam, Puducherry - 605 009, India.
E-mail: stalinviswanathan@gmail.com

Access this article online

Quick Response Code:



Website:
www.jfmpc.com

DOI:
10.4103/jfmpc.jfmpc_215_17

He was treated with oral and intravenous K⁺ 480 mmol (first 24 h) and shifted to Intensive Care Unit (ICU). He received 240 mmol/day for the next 48 h. Electrocardiography and muscle power were monitored continuously. His K⁺ improved to 3.8 on day 3 and he was shifted out of ICU. He was unwilling for further therapy in hospital and was discharged at request after initiation of sodium bicarbonate 500 mg BID and Levothyroxine 100 µg OD. He did not return for follow-up.

Case 2

A 55-year-old hypertensive female presented with weakness of all four limbs. Ten days ago, she had developed multiple episodes of vomiting and fever, followed by bilateral thigh pain of 2 days' duration. On the day before the presentation, she developed inability to walk, requiring assistance to the toilet. There was no diurnal variation in her weakness, oculopharyngeal, or respiratory weakness. She was constipated and had dysuria with frequency. The past history was significant only for hypertension (amlodipine 5 mgOD). On examination, she appeared dull looking, with dry and coarse skin, bradycardia (60 beats/min), hypertension (140/100 mmHg), and tachypnea (28 breaths/min). She had flaccid quadriparesis (3/5 power MRC), hyporeflexia, floppy neck, and flexor plantar response. Mentation was intact without cranial neuropathy, sensory signs, or sphincter involvement. Investigations are in Table 1.

Antiemetics, pantoprazole, intravenous K⁺ at 20 mmol/h, and ceftriaxone 2 g OD for probable urinary infection were given. She denied the history of alcohol use/abuse. She tolerated oral feeds the next day, and oral K⁺ 240 mmol/d, fruit juices, and tender coconut water were administered. Her K⁺ 24 h after admission had fallen further to 2 mmol/L, without new gastrointestinal or renal losses. Intravenous magnesium 1 g was also given. Her K⁺ improved to 3.2 mmol/L on day 3. She began ambulating on her own and L-thyroxine 75 µg was initiated, with K⁺ supplementations being continued. She was discharged on day 7 with K⁺ remaining consistently above 3.5 mmol/L.

Discussion

Hypokalemia (reduced K⁺) may be either due to K⁺ deficiency (negative K⁺ balance) or due to intracellular shifts of K⁺.^[1] Potassium deficiency may not always manifest as hypokalemia, as in diabetic ketoacidosis (DKA).^[1] Hypokalemia may occur over hours (intracellular shift and thyrotoxicosis) or gradually (increased excretion and poor intake).^[3]

HP with hypothyroidism has been reported on 15 occasions previously (16 adults, 2 children).^[4-12] Eleven (11/18) patients with HP had hypokalemic periodic paralysis (HPP). One patient had resistance to thyroid hormone and was already on L-thyroxine without improvement in his episodic paralysis.^[9] Both our patients came with their first episodes of weakness. Five reports had females with both DRTA and hypothyroidism.^[5,10] Three (3/5) were already hypothyroid and had HPP. The remaining two patients (2/5) with sporadic HP at presentation were female

Table 1: Laboratory investigations of the two patients

Lab value (Normal ranges)	Case1	Case2
Serum Potassium (3.5-5mmol/L)	1.7	2.4
Serum sodium (130-145mmol/L)	139	123
Serum Chloride (102-108mmol/L)	110	94
Serum creatinine (44-105µmol/L)	103	100
Serum calcium (2.2-2.6 mmol/L)	1.92	2.52
Serum magnesium (0.62-0.95 mmol/L)	0.78	0.49
Asparate transaminase (0.20-0.65 µkat/L)	0.32	4.2
Alanine transaminase (0.12-0.70 µkat/L)	0.21	1.35
Creatine kinase (0.66-4.0 µkat/L)	2.33	23.43
Urine pH	6.8	6.5
Urine potassium spot	16	16
pO ₂ (9.6-13.8kPa)	12.26	7.9
pH (7.35-7.45)	7.39	7.69
HCO ₃ (22-30 mmol/L)	18.5	38.5
pCO ₂ (4.3-6.0 kPa)	1.47	3.11
Serum osmolality (275-295 mOsmol/kg)	292	250
Anion gap (8-12)	11.1	9.5
TSH (0.5-5.5mIU/L)	12.06	36.08
Hemoglobin (120-160g/L)	111	94
Total leukocyte counts (4-10.5×10 ⁹ /L)	10.3	13.8
Neutrophilia %	83	90
Platelets (150-450×10 ⁹ /L)	121	417
Electrocardiogram	Flat T waves, ST depression, LAE, LVH and U waves	Bradycardia, prolonged PR and flattened T waves
Chest radiography	Normal	Cardiomegaly
Echocardiography	Normal	Minimal pericardial effusion

and had both Hashimoto's thyroiditis and Sjogren's; the second patient had systemic lupus erythematosus (SLE), additionally.^[10] Ours was a male patient with DRTA and hypothyroidism, both diagnosed at presentation. DRTA has been described with hyperthyroidism, autoimmune/nonautoimmune hypothyroidism and diseases such as SLE, chronic active hepatitis, Sjogren's, and myeloma.^[10] Due to unavailable laboratory facilities, we could not confirm what predisposed him to hypothyroidism and DRTA. Metabolic acidosis leads to extracellular K⁺ shift, and serum levels are generally maintained. Potassium losses in urine (diabetes, toluene) and stools (diarrhea), insulin lack (DKA) and defects in renal acidification (DRTA) lead to hypokalemia and its manifestations.^[3] Only the last cause was relevant in Case 1.

Hypokalemia with metabolic alkalosis is seen in excessive vomiting, diuretics, mineralocorticoid excess, Gitelman, Bartter's, and Liddle's syndromes. Due to hypertension, mineralocorticoid excess/Liddle's was a possibility, but she had hyponatremia and hypertension was mild and easily controlled. There was no evidence of hypertension-related target organ damage. Hypomagnesemia-hypokalemia-metabolic alkalosis also led to consideration of Gitelman, but she had had hypertension for

3 years. We did not have the facility for analyzing urinary calcium and magnesium, and hormones – plasma renin, cortisol, and aldosterone. Case 2 probably had urinary infection-related vomiting, poor intake, and polyuria-related K⁺ loss leading to HP and was found to have diabetes and hypothyroidism at admission. There are three cases of HP and diabetes reported in young males.^[13,14] Four cases with HP in diabetic emergencies have also been reported.^[3,15] Six (6/7) cases of HP in diabetes are from India. These men did not have hypothyroidism, and HP was the presenting symptom of diabetes alone. Case 2 also had elevated transaminases and creatine kinase, attributable to both hypokalemia and hypothyroidism-related myopathy. Urine did not show evidence of rhabdomyolysis.

Conclusion

HP with hypothyroidism is being described mostly among Asian-Indians. HP leading to a diagnosis of diabetes is described in the same cohort. We report the first male with HP having both hypothyroidism and DRTA, and also the first woman to have both diabetes and hypothyroidism presenting as HP. Investigating HP in the primary and secondary care of developing countries such as ours is fraught with disappointment in the inability to reach a conclusive etiological diagnosis. Hence, many patients are often treated for symptoms alone, leading to poor reportage. Apart from blood sugars and thyroid profile, hormonal analysis and urinary electrolytes will go a long way to diagnose individuals with hypokalemic weakness.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

- Allon M. Disorders of potassium metabolism. In: Gilbert S,

Weiner DE, editors. National Kidney Foundation Primer on Kidney Diseases. 6th ed. New York: Elsevier Saunders; 2014. p. 90-9.

- Khadilkar S. Neurological manifestations of thyroid and parathyroid disorders. In: Gupta SB, editor. Medicine Update. Mumbai: Association of Physicians in India; 2005. p. 609-12.
- Lin SH, Davids MR, Halperin ML. Hypokalaemia and paralysis. QJM 2003;96:161-9.
- Sinha U, Sengupta N, Sinharay K, Sahana PK. Recurrent hypokalemic paralysis: An atypical presentation of hypothyroidism. Indian J Endocrinol Metab 2013;17:174-6.
- Malkarnekar S, Naveen L. Adult-onset distal renal tubular acidosis with hypokalemic quadriparesis in a patient with autoimmune hypothyroidism. J Integr Nephrol Androl 2014;1:82.
- Singh TK, Sangme N, Singh LN, Singh AJ. Hypokalaemic periodic paralysis associated with hypothyroidism. Indian J Phys Med Rehabil 2012;23:79-81.
- Chaudhary SP, Gupta K, Guta RK, Tahilaramani H. Hypokalemic paralysis during management of hypothyroidism. Indian Pediatr 2000;37:1261-3.
- Mohapatra BN, Lenka SK, Acharya M, Majhi C, Oram G, Tudu KM, *et al.* Clinical and aetiological spectrum of hypokalemic flaccid paralysis in western odisha. J Assoc Physicians India 2016;64:52-8.
- Ma S, Hu M, Yang H, Lian X, Jiang Y. Periodic paralysis as a new phenotype of resistance to thyroid hormone syndrome in a chinese male adult. J Clin Endocrinol Metab 2016;101:349-52.
- Velarde-Mejía Y, Gamboa-Cárdenas R, Ugarte-Gil M, Asurza CP. Hypokalemic paralysis: A hidden card of several autoimmune diseases. Clin Med Insights Arthritis Musculoskelet Disord 2017;10:1179544117722763.
- Sharma CM, Kumawat BL, Garg AK, Aggarwal P. Etiological and biochemical profile of hypokalemic paralysis in North-West India. Int J Sci Res 2017;6:203-6.
- Kayal AK, Goswami M, Das M, Jain R. Clinical and biochemical spectrum of hypokalemic paralysis in north: East India. Ann Indian Acad Neurol 2013;16:211-7.
- Amalnath DS, Dutta TK. Hypokalemic paralysis as the presenting manifestation of diabetes in two patients. Indian J Endocrinol Metab 2013;17:1127-8.
- Vishnu VY, Kattadimma A, Rao SA, Kadhiraivan T. Sporadic hypokalemic paralysis caused by osmotic diuresis in diabetes mellitus. J Clin Neurosci 2014;21:1267-8.
- Aggarwal HK, Jain D, Kaverappa V, Yadav S, Jain P. Quadriparesis as a presenting manifestation of diabetic ketoacidosis: A rare case report. Diabetol Croat 2013;42:60-4.