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Sporadic hypokalemic periodic paralysis: A case report and literature review

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ABSTRACT

The hypokalemic periodic paralysis is a rare (1:100000) inherited channelopathy of skeletal muscles characterized by muscle weakness coinciding with low potassium levels, many classifications exist. However sporadic, familial, and thyrotoxic is the commonest, we presented a thirty-eight-year-old Filipino Female with sporadic periodic paralysis with quadriparesis. The literature of the published case reports, the genetic background, investigation, and treatment was reviewed.

1. INTRODUCTION

The hypokalemic periodic paralysis is a rare (1:100000) inherited channelopathy of skeletal muscles characterized by muscle weakness coinciding with low potassium levels, they are seldom fatal, however, persistent weakness may develop with major disability [1]. The culprit genes encoding calcium and potassium cause the same periodic paralyzes of the same type (hypokalaemic periodic paralysis or Andersen-Tawil Syndrome characterized by facial dysmorphism, periodic paralysis, and cardiac arrhythmia), while gene mutations in sodium channels cause hypokalemic, normokalemic, or hyperkalemic periodic paralysis [2]. Many classifications exist, one is familial and non-familial (including thyrotoxic, and sporadic paralysis) and secondary causes [3]. We reported a case of sporadic hypokalemic periodic paralysis presenting with proximal muscle weakness and rhabdomyolysis.

2. Case report

A thirty-eight-year-old Filipino Female with no significant past medical history presented to the emergency room with acute quadriparesis.

The weakness was bilateral and involved proximal muscles of both upper and lower limbs. She had no respiratory or swallowing difficulty and was able to move her neck and facial muscles. She denied any sensory symptoms, as well as any recent diarrhea, chest pain, shortness of breath, or weight change.

She reported that her symptoms came on after heavy exercise related to her nature of work (she worked as a servant), she denied use of alcohol or drugs, she had similar episode 2 years ago but didn't receive any

diagnosis. She had a history of cervical spondylosis. To her knowledge, she has no history of thyroid disorders and she denied any family history of similar condition.

On physical exam, the patient's heart rate was 90 and blood pressure was 110/70, respiratory rate 20, and oxygen saturation 98%. She was alert (Glasgow Coma Scale(GCS) 15/15, she has no goiter or enlarged lymph node. The cardiac exam revealed normal S1 and S2 and no murmurs. Examination of the lungs and abdomen were unremarkable and has no skeletal or skin abnormalities.

Neurologic exam revealed proximal symmetrical flaccid paralysis involving her extremities with intact sensation with hyporeflexia and down going planters. All cranial nerves were intact, and she has no cerebellar signs.

Urinalysis, routine chemistry, liver enzymes, and complete blood count were normal except for a potassium level of 1.8 (3.5–5 mmol/L) and calcium which was initially normal but on next day of admission, she developed symptomatic hypocalcemia (calcium 1.8). Electrocardiogram revealed evidence of hypokalemia.

Thyroid function test was normal (TSH 2 3.85µIU/mL, free T3 3.81pmol/L, free T4 14.22 pmol/L)

Vitamin D was 33ng/mL and parathyroid hormone (PTH) was 23.80pg/mL both were low, phosphate was 1.2 mmol/L, and Creatine kinase was 1300 (38-176 N/L in females) which may be moderately elevated in hypokalemic paralysis.

EMG, Muscle biopsy, and genetic studies for channelopathies were not done.

The patient was admitted to high dependency unit with cardiac monitor, a central line was inserted and KCL 20 mmol /hour along with oral potassium 600mg 8/hourly and regular checkup of serum potassium every 4 hours, the patient showed a dramatic improvement of power from 1/5 to 4/5 MRC after 8 hours of treatment, On next day, the patient developed carpopedal spasm, but no chovestick or trousseaus' signs. Her calcium was 1.8 mmol; therefore, the patient received calcium gluconate 10% 10 ml over 20 minutes followed with an infusion of 40 ml over 24h. She also received Vitamin D 10000 IU every other day. Thereafter symptoms resolved, and serum calcium backed to normal.

The patient was discharged after attaining her usual power on acetazolamide 250 mg BID for one month, spironolactone 25mg OD, and potassium 600mg OD for two weeks and given OPD after one month with advice to avoid eating much carbohydrate as well as avoiding extraneous exercise. And seek medical advice if she develops similar episodes. The absence of family history, the recurrent attacks, the absence of glucose and protein in her urine analysis, the normal acid-base, and the normal urinary potassium excluded familial and renal tubular damage (the absence of diarrhea and vomiting, dieresis, drug intake, the normal acid-base and urinary potassium, the absence of a high blood pressure makes diuretic and laxative abuse, surreptitious vomiting, Bartter syndrome, Gitelman syndrome, and Primary aldosteronism unlikely. Thyrotoxic periodic paralysis was excluded by the normal T4, T3, and TSH levels and Gitelman syndrome is unlikely due to the normal calcium/creatinine ratio in her urine.

3. Review of case reports

Among the 16 cases identified, 50% were from Asia, and 50% were from Europe, nearly two-thirds were sporadic, 31.3% were thyrotoxic, while 1(6.3%) was familial.

[4] reported a case of fatal thyrotoxic periodic paralysis, a study published in Spain [4] reported a familial case with epilepsy and subclinical hypothyroidism on replacement therapy, a plausible explanation is that the patient may be thyrotoxic due to over-replacement. [6] reported a rare case of sporadic hypokalemic paralysis with differential recurrent right brachial weakness and cognitive dysfunction, [7] from Taiwan reported a similar case of recurrent periodic paralysis. [8] showed no mutations in calcium, sodium or potassium channels in a case with thyrotoxic paralysis, similarly, [9], [10] reported cases with Grave's and Basedow diseases respectively. A sporadic (secondary) with gastroenteritis and quadriparesis was reported by [11], [12] documented the usefulness of muscle fiber conduction. Rare cases of bilateral facial palsy and cognitive function, rhabdomyolysis in a sporadic case due to distal renal tubular acidosis, and ACE dysfunction in sporadic cases were reported by [13- 15] respectively. In the present review, [16] reported a rare case of T3 toxicosis in Japan. Sporadic cases of hypokalemic paralysis were published in Serbia [17] and Italy [18], [19].

3.1 Gene mutations

There were 14 studies (35.7% case-control), more than a half were from Asia, while 28.6% were from South America and Europe (14.3% each).

3.2 Thyrotoxic hypokalemic periodic paralysis genes mutations

[20] studied15 patients with the thyrotoxic form and found a potassium channel gene mutations, and [21] found no mutations in calcium genes.

3.3 Familial hypokalemic periodic paralysis genes mutations

Regarding the familial type of hypokalemic periodic paralysis, [22] identified a sodium channel gene in skeletal muscle, while [23] conducted a case-control of 60 patients and their families and 50 controls and concluded that: Only two families reported calcium channel gene mutations which are shared in one family with sporadic, no thyrotoxic cases showed the abnormality. [25], [24] mapped the genes of patients with familial periodic paralysis and found a calcium channels deficiency.

3.4 Shared mutations between familial, sporadic, and thyrotoxic

[26] in Taiwan showed that CTD-2378E21.1 is shared between thyrotoxic and sporadic that may negatively regulate potassium channels in muscles, while [27] in Germany in their case-control study showed that potassium channels alterations are seldom pathogenic. The latter study included a larger sample which included Asians and Non-Asians patients. A study conducted in Taiwan [27] support the observation of shared mutations between sporadic and thyrotoxic periodic paralysis. [28], [29] in their study in Taiwan showed that only a minority of sporadic cases shared the genes with familial types (maybe a variant). Furthermore, those without the mutations showed a late age of onset, fewer attacks, and no precipitating causes, similar findings were concluded by [30], [31] conducted a study and concluded similar findings. [32] studied thyrotoxic and sporadic periodic paralysis and found thatKir2.6 are associated with sporadic as well as thyrotoxic suggesting a decreased outflux of potassium and paradoxical depolarization during attacks, leads to sodium channel inactivation, but the study included only four cases.

3.5 Gitelman mutations

[33] studied two cases and identified additional mutations of thiazide-sensitive Na-Cl cotransporter (TSC) gene in the form of deletion, insertion or missing mutations in exons 5, 6, and 21.

Table 1. Case-reports

Author	Year	Country	Result

[4]	2018	India	A fatal case of thyrotoxic hypokalemic periodic paralysis
[5]	2014	Spain	Familial hypokalemic periodic in a female with epilepsy and subclinical hypothyroidism on replacement therapy
[6]	2009	India	Sporadic with recurrent differential right brachial weakness and cognitive dysfunction
[7]	2009	Taiwan	Recurrent sporadic paralysis
[8]	2008	Italy	A 37 Italian male with thyrotoxic paralysis and no mutations in the candidate exons of calcium (CACN1AS), potassium (KCNE3) and sodium (SCN4A) channel genes.
[9]	2004	Germany	A Chinese man with thyrotoxic Grave's disease
[10]	2003	France	A 37-year-old Caucasian man with Basedow's disease (hyperthyroidism).
[11]	1998	Turkey	A sporadic (secondary) with gastroenteritis and quadriparesis
[12]	1992	Netherlands	Reported a case of sporadic paralysis and concluded that muscle fiber conduction is helpful.
[13]	1990	China	Bilateral facial palsy and speech disturbances in sporadic
[14]	1990	Malaysia	Rhabdomyolysis in a sporadic case due to distal renal tubular acidosis
[15]	1986	Japan	ACE dysfunction syndrome of sporadic type
[16]	1984	Japan	T3 toxicosis
[17]	1981	Serbia	Sporadic
[18]	1969	Italy	sporadic
[19]	1968	Italy	Sporadic

Table 2. Type and country of origin of the studied case reports

Character	No%
Country	
Asia	8 (50%)
Europe	8 (50%)
Classification	
Sporadic	10 (62.3%)
Thyrotoxic	5 (31.3%)
Familial	1 (6.3%)

Table 3. Genetic mutations

Author	**Year**	**Country**	**Methods**	**Conclusion**
[26]	2016	Taiwan	Case-control of 77 thyrotoxic, 33 sporadic and 1730 controls not having the known genes associated with the disease.	shared genetic predisposition betweenthyrotoxic and sporadic forms

				(CTD-2378E21.1 and may negatively regulate KCNJ2 expression.
[27]	2016	Germany	A case-control of 474 of controls (400 Caucasians, 74 male Asians) and 263 unrelated patients with periodic paralysis	KCNJ18 alterations are seldom pathogenic
[28]	2012	Taiwan	Case-control of 151 (90 thyrotoxic and 61 sporadic) and 100 age and sex-matched controls.	The same susceptible gene variant rs623011 was found in both.
[29]	2012	Taiwan	60 patients assumed to have sporadic by an exclusion of other etiologies and 8 familial case testes for the associated genes	The majority of sporadic had no genes similarity to familial, while a minority shared the familial genes (maybe a variant). Those without the mutations showed a late age of onset, fewer attacks, and no precipitating causes.
[32]	2011	USA	Two thyrotoxic and two sporadic investigated	Kir2.6 are associated with sporadic as well as thyrotoxic suggesting a decreased outflux of potassium and paradoxical depolarization during attacks, leads to sodium channel inactivation.
[22]	2006	China	The mutations of 23 Chinese patients with different forms of hypokalemic periodic paralysis were analyzed.	Mutations at codon 672 in the skeletal muscle sodium channel gene(SCN4A) gene was identified to segregate with the disease in the familial form and not in others
[23]	2005	Taiwan	Case-control of 60 patients and their families and 50 controls	Only two families reported CACNA1S gene mutations which are shared in one family with sporadic, no thyrotoxic cases showed the abnormality.
[21]	2003	Switzerland	Studied 5 Chinese patients with thyrotoxic periodic paralysis	No mutation was found on the whole CACNA1S gene
[20]	2002	Brazil	15 patients with the thyrotoxic form	Found a mutation in the potassium KCNE3 gene
[33]	2002	Japan	Two Japanese with Gitelman	Identified additional mutations of thiazide-sensitive Na-Cl cotransporter (TSC) gene in the form of deletion, insertion or missing mutations in exons 5, 6, and 21.
[31]	2002	Brazil	A case-control study 14 patients with THPP, 13 sporadic cases and one with a family history. An FHPP family was selected as a positive control.	Mutations linked to familial hypokalemic periodic paralysis in the calcium channel alpha1 subunit gene (Cav1.1) are not associated with thyrotoxichypokalaemic periodic paralysis. However, polymorphisms are present.
[24]	2001	Japan	Gene analysis of patients with hypokalemic periodic paralysis	Functional deficiency of the skeletal muscle ion-channels identified in familial forms.
[25]	1997	Japan	Mapped the autosomal dominant hypokalemic periodic paralysis (HypoPP)	Locus to chromosome 1q31-32, where the dihydropyridine-sensitive calcium channel alpha 1 subunit (CACNL1A3) is located.

[30]	1997	USA	Studied the mutations of the dihydropyridine-sensitive calcium channel alpha 1 subunit	Found that mutations are associated with earlier onset and severe disease

Table 4. Type and country of origin of the studied case reports

Character	No%
Country	
Asia	8 (57.1%)
Europe	2(14.3%)
USA	2(14.3%)
South America	2(14.3%)
Type of studies	
Case-control	5 (35.7%)
Other experimental studies	9 (64.3%)

3.6 Investigations

- Arterial blood gases and urine potassium sugar, and proteins for renal tubular acidosis
- Free T4, T3, and TSH
- Urinary calcium /creatinine ratio to detect hypercalcuria in Gitelman syndrome
- Gene analysis
- EKG. and EMG (exercise) are helpful [34], [35]

3.7 Treatment

Oral aqueous potassium is the mainstay for hypokalemia (avoid slow release preparation), typically 40-60mEq/day for adults that raise serum K by 1-1.5mEq, if no response after half an hour 0.3mEq/KG is given up-to 100mEq. Typically not more than 200mEq, if intravenous potassium is needed (cardiac arrhythmia or airway compromise due to ictal dysphagia or accessory respiratory muscle paralysis), 5% mannitol is preferred because both dextrose and saline may exacerbate the weakness. IV potassium chloride 0.05-0.1 mEq/kg a bolus. Only 10 mEq at a time should be infused with intervals of 20-60 minutes, unless in situations of cardiac arrhythmia or respiratory compromise. Sequential potassium checks are mandatory to avoid hyperkalemia Carbonic anhydrase inhibitors have been shown to reduce the frequency of attacks and can be used for prophylaxis, Dichlorophenamide 50-100mg q./12 hours or Acetazolamide 250-500mg/8hourly. Potassium sparing diuretics, eplerenone, triamterene, and spironolactone may show benefit (no need for potassium supplimentation) [36- 38]. Treatment of the underlying cause when applicable.

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