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Case Report

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Secondary Hyperkalemic Paralysis: a Case Report and an Update Review of 128 Cases

Túlio Marcos Coimbra[1*], Sara Terrim[1], Guilherme Diogo Silva[1].

[1] Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, Universidade de São Paulo, São Paulo, Brazil
* Corresponding author. E-mail address: *tulio.coimbra@hc.fm.usp.br*

ABSTRACT

Introduction: Acute flaccid paralysis (AFP) is characterized by progressive weakness with signs of impairment of the lower motoneuron. Secondary hyperkalemic paralysis is a cause of AFP that must be quickly recognized to prevent cardiac and neurological deterioration. **Methods:** We present a case of secondary hyperkalemic paralysis admitted to the emergency department and then we updated a systematic review of published cases of secondary hyperkalemic paralysis published in 2015, including case reports published from 2014 to 2021. **Results:** We present a 69-years-old man admitted to the emergency department due to acute ascending generalized weakness. The neurological examination showed symmetrical tetraparesis (strength 2/5 in lower limbs and 4/5 in the upper limbs). Sensory and cranial nerves testing were unremarkable. He also had intense fasciculations in the cervical region and in the proximal muscles of the upper limbs. Initial laboratory showed potassium of 9.3 mEq/dL. The correction of hyperkalemia led to a complete reversal of weakness and fasciculations. Our patient was diagnosed with renal impairment secondary to a systemic lupus erythematosus was the cause of the secondary hyperkalemic paralysis. **Discussion:** We found 128 cases of secondary hyperkalemic paralysis in Literature. The most common symptom was flaccid tetraparesis. Sensory and cranial nerves testing are usually unremarkable. Electrocardiogram is abnormal in almost all patients, particularly with a tall peaked T wave or wide QRS complex. Renal failure is the most common cause of secondary hyperkalemic paralysis. Correction of hyperkalemia is associated with excellent motor outcomes.

Keywords: Paralysis, Case report, Secondary hyperkalemic paralysis, Fasciculation

Introduction

Acute flaccid paralysis (AFP) is a neurological emergency characterized by progressive weakness with signs of impairment of the lower motoneuron. Common causes of AFP include Guillain Barre syndrome and myasthenia gravis.

Hyperkalemic paralysis is an uncommon cause of AFP. Primary hyperkale mic paralysis occurs in genetic chanalopathies. Secondary hyperkalemic paralysis (SHP) is associated with acquired causes.

A systematic review of hyperkalemic paralysis published in 2015 demonstrated that the most common causes of secondary hyperkalemic paralysis were kidney injury, drugs and adrenal insufficiency. We believe more case reports have been published from 2015 to 2021 and an update systematic review can improve our understanding of the clinical characteristics and the causes of secondary hyperkalemic paralysis.

Methods

We present a case report to illustrate a case of secondary hyperkalemic paralysis as a cause of AFP. We describe patient demographics, clinical presentation, laboratorial testing and follow up.

A systematic review of hyperkalemic paralysis published in 2015 was the starting point for our update of new case reports published up to July 2021. Then,

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to identify more cases of SHP, we performed a systematic Literature review using the terms “hyperkalemic” AND “paralysis” in Pubmed, Embase and Scopus databases in July 2021. As this previous systematic review was published in 2015[1], we limited date of publication from 2015 to 2021.

To be included, studies had to present clinical features of secondary hyperkalemic paralysis, described in complete case reports. We excluded: case reports about hyperkalemic paralysis primary or genetic, case reports about hyperkalemia, case reports of hyperkalemia in the context of other unmanifested conditions with secondary hyperkalemic periodic paralysis, general review about secondary paralysis without a case report, studies developing animals or describing the pathophysiology of hyperkalemic periodic paralysis, case reports involving muscle weakness but in the context of hypokalemia, studies with drugs in primary hyperkalemic periodic paralysis (like clinical trials), book chapter citation, incomplete texts and/or abstracts only. Two different reviewers independently screening manuscripts for eligibility. We did not perform any specific bias evaluation as we considered only low-quality evidence (case reports) were included.

We collected the following data from published case reports: (1) age, (2) sex, (3) neurological examination, (4) electrocardiogram characteristics and (5) the cause of the secondary hyperkalemic paralysis. We described summary data in narrative and tabular manner.

Results

Case Report

A 69-year-old white male presented a 2-hour history of lower limb weakness. He denied sensory or bladder symptoms. He had primary arterial hypertension and was under investigation of an autoimmune thrombocytopenia.

On neurological exam, he had symmetric flaccid tetraparesis. Upper limb strength was grade 4. He had 2/5 proximal and 3/5 distal lower limb weakness. Deep tendon reflexes were absent in the lower limbs and diminished in the upper limbs. Sensory and cranial nerves testing were unremarkable. He also presented fasciculation in the cervical region (**Supplement file - Video 1**). One hour after admission, upper limb strength worsened to grade 2.

Laboratory testing revealed a hyperkalemia (9.3 mEq/L). Electrocardiogram showed tall peaked T wave, prolonged PR interval (200 ms) and wide QRS complex (**Figure 1a**). We prescribed insulin, dextrose, bicarbonate and calcium gluconate. Potassium levels decreased to 6.8 mEq/L. He presented a complete recovery of weakness. Fasciculations disappeared. Electrocardiogram also improved (**Figure 1b**).

The investigation of the cause of hyperkalemia revealed an acute kidney injury (Cr 2.3 mg/dL). Further laboratory testing revealed a positive antinuclear antibody (ANA) (1/640), positive anti-DNA double strand antibody and low C4 and C3 levels. Renal failure with thrombocytopenia and these positive immunological testing led to the diagnosis of systemic lupus erythematosus, according to the 2019 EULAR criteria. Unfortunately, patient developed a nosocomial infection and died during hospitalization.

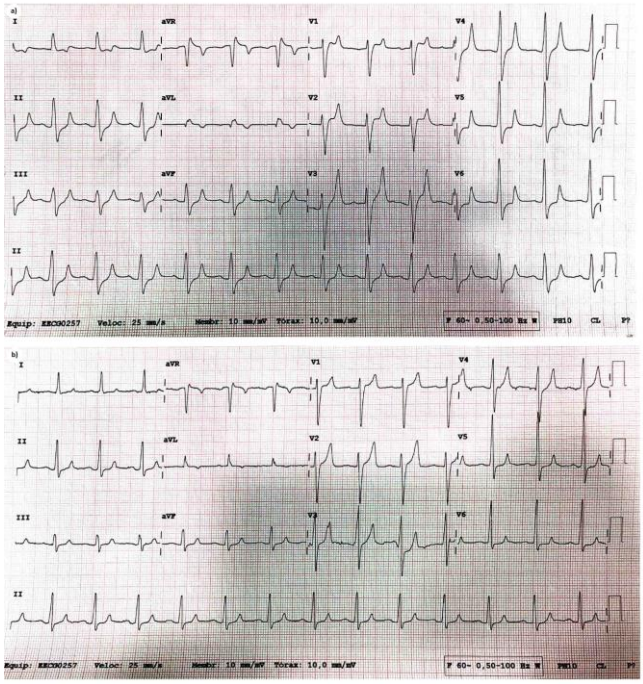

Figure 1. Electrocardiographic changes. In Figure 1a, electrocardiogram at admission (potassium levels of 9.3 mEq/L). In Figure 1b, Electrocardiogram after treatment (potassium levels of 6.8 mEq/L).

Systematic review

We found 226 records in databases searching and 3 additional records through other sources (references from review studies). After duplicates removal, we screened 78 records, from which we reviewed 8 full-text documents[2-9], all included in our qualitative and quantitative summary analysis. The major reason for excluding case reports was that the cause of the hyperkalemic paralysis was

primary/genetic or review articles. Other exclusion criteria are mentioned in the methodology and the frequency of each of them is available in **Supplementary Appendix**. The three included manuscripts from references from articles were not included in the previous systematic review of the topic[1], despite they have been published before 2015, because they were published after searching the previously published systematic review. **Figure 2** represents our flow diagram and in the **table 01** summarizes the individual characteristics of the new cases reported in the literature.

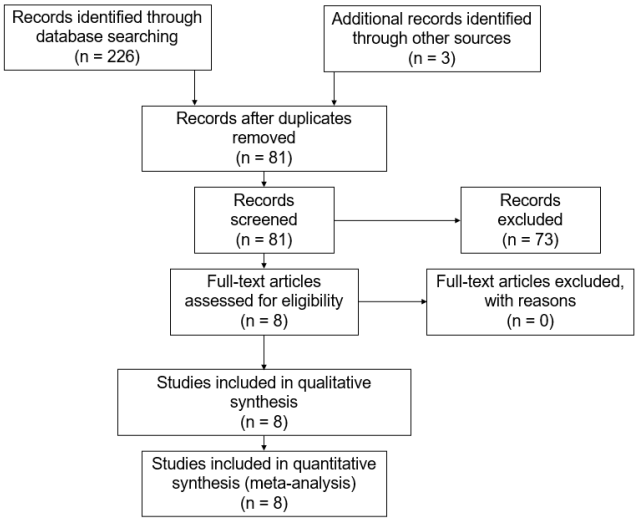

Figure 2. Flow diagram.

The systematic review published in 2015[1] includes a total of 118 patients (n = 118); 85 males; 33 females. Our review found 8 more case reports and, including our reported case, we added 9 new cases to the total number of patients analyzed, totaling 128 patients (n = 128). Subsequently, the tables present in the previously published systematic review were updated.

The mean age was 50.6 years old (range 15-86 years). Secondary hyperkalemic paralysis was more common in males, corresponding to 70% of patients (88 out of 128 patients).

As in our case, the most common presentation was flaccid tetraparesis (46.7%, 60 out of 128 patients). Sensory and cranial nerves abnormalities were rare. Respiratory symptoms occurred in almost one in five patients (18%, 22 out of 128 cases). **Table 02** summarizes clinical characteristics.

Acute fasciculations, as presented in our patient, may indicate a peripheral nerve hyperexcitability due to hyperkalemia. This finding occurs in primary hyperkalemic paralysis (sodium chanaolopathy) and was reported in one case of secondary hyperkalemic paralysis10 in our review. The presence of acute fasciculations may differentiate hyperkalemic paralysis from common causes of AFP as Guillain Barre or myasthenia gravis.

QRS Electrocardiogram was abnormal in almost all patients with secondary hyperkalemic paralysis. The most common finding was tall-peaked T wave (73.6%, 84 out of 114 patients), followed by wide QRS complex (59.6%, 68 out of 113 cases). Other electrocardiogram findings are described in **table 03**.

Presenting Clinical Features	**n (%)**
Main sign/symptoms	
Flaccid tetraparesis	60 (46.7)
Ascending flaccid paralysis	42 (32.8)
Muscular weakness	26 (20.3)
Associated sign/symptoms	
Paresthesia, dysesthesia	26 (20.3)
Difficulty breathing	22 (17.2)
Lethargy	12 (9.4)
Sensory loss	7 (5.5)
Dysphagia, difficult in mastication	6 (4.7)
Dysarthria	6 (4.7)
Others*	16 (12.5)

Table 02. Summary of Neurologic Clinical Findings of Patients with Hyperkalemic Paralysis (n = 128#). *Hyper-reflexia (n = 3), mental disorientation (n = 3), myalgia (n = 3); tremor (n = 2); fasciculation (n = 1), aphasia (n = 1), urinary retention (n = 1), bilateral facial palsy (n = 1), and hypertonia (n = 1). #Update of previously published data[1] (n = 118 + 9), based on our table 1

Causes of secondary hyperkalemic paralysis could be described as: a third of had chronic renal failure (33.8%, 42 out of 124 patients), a third had acute renal failure (31.4%, 39 out of 124 patients), and the last third had other causes. Other causes of secondary hyperkalemic paralysis included drug-induced hyperkalemia, adrenal insufficiency and hemolysis–cell lysis. **Table 04** summarizes the causes of secondary hyperkalemic paralysis.

Treatment strategies included insulin with dextrose, bicarbonate, salbutamol (inhalation), furosemide, sodium polystyrene sulfonate, and calcium chloride or gluconate. Correction of hyperkalemia was associated with excellent motor recovery.

Author	Age	Sex	Neurological examination	Electrocardiogram characteristics	Cause
Menegussi J et al.	54 years	Male	Flaccid tetraparesis	- Tall peaked T wave - Flattening of the P-wave - Prolonged PR interval - Wide QRS complex	- Drugs (Trimethoprim-sulfamethoxazole, Tacrolimus and Propranolol)
Hemachandra KHD et al	78 years	Male	Flaccid tetraparesis	- Tall peaked T wave - Sine waves	- Chronic renal failure - Addison's disease - Drugs (Losartan and Spironolactone) - Potassium supplements - Dietary excess (Coconut water)
Karmacharya P et al	68 years	Female	Flaccid tetraparesis	- Tall peaked T wave - Wide QRS complex	- Drugs (Spironolactone) - Potassium supplements
Kimmons LA et al	53 years	Female	Ascending flaccid paralysis	- Tall peaked T wave - Prolonged PR interval with first-degree AV block	- Acute renal failure - Drugs (Lisinopril and Spironolactone) - Potassium supplements
Narayanan S et al	66 years	Male	Ascending flaccid paralysis	- Tall peaked T wave - Flattening of the P-wave - Wide QRS complex	- Chronic renal failure - Drugs (Ramipril and Spironolactone)
Mishra A et al	60 years	Female	Flaccid tetraparesis	- Tall peaked T wave	- Addison's disease
Sasaki O et al	77 years	Female	Muscular weakness	- Tall peaked T wave - Flattening of the P-wave - Bradycardia	- Chronic renal failure - Drugs (Telmisartan, Spironolactone and Metoprolol Tartrate)
Garg SK et al	73 years	Female	Ascending flaccid paralysis	- Tall peaked T wave	- Drugs (Ramipril, Spironolactone and Atenolol)
Our case	69 years	Male	Ascending flaccid paralysis	- Tall peaked T wave - Prolonged PR interval - Wide QRS complex	- Acute renal failure secondary to systemic lupus erythematosus.

Table 01. Summarizes the individual characteristics of the new cases reported in the literature.

ECG Sign	n (%)
Tall peaked T wave	84 (73.6)
Wide QRS complex	68 (59.6)
Absent P wave	34 (29,8)
First-degree AV block	15 (13.1)
Sinus or nodal bradycardia	12 (10.5)
IV conduction abnormalities	9 (7.9)
Sine waves	7 (6.14)
Others*	12 (10.5)

Table 03. Summary of Electrocardiographic Pathologic Signs in Patients with Hyperkalemia and Abnormal Electrocardiogram (n = 114#). AV = atrioventricular; ECG = electrocardiogram; IV = intraventricular. * ST segment abnormalities (n = 3): prolonged QT duration (n = 2), loss of pacemaker capture (n = 2); low amplitude QRS (n = 1), and short QT duration (n = 1), flattening of the P-wave (n = 3). # Update of previously published data[1] (n = 105 + 9), based on our table 1.

Factor	n (%)
Chronic renal failure	42 (33.8)
Acute renal failure	39 (31.4)
Potassium intake (drug or feed)	28 (22.5)
Addison disease	17 (13.7)
Spironolactone	21 (16.9)
Dehydration	11 (8.8)
Hemolysis–cell lysis (cancer, rhabdomyolysis)	10 (8.6)
NSAIDs	7 (5.6)
ACE inhibitors	7 (5.6)
Hypoaldosteronism	4 (3.2)
Others*	23 (18.5)

Table 04. Summary of Factors Associated with Development of Hyperkalemia (n = 124#). ACE = angiotensin-converting enzyme; NSAIDs = nonsteroidal anti-inflammatory drugs. * Amiloride-hydrochlorothiazide (n = 3); co-trimoxazole (n = 3), diabetic ketoacidosis (n = 2), eclampsia (n = 2); arginine (n = 1), Cushing syndrome (n = 1), digoxin (n = 1), and thalidomide (n = 1); beta blocker (n = 3); tacrolimus (n = 1); coconut water (n = 1); angiotensin II receptor blockers (n = 2); # Update of previously published data[1] (n = 115 + 9), based on our table 1.

In **figure 3**, we propose an algorithm for approaching patients with acute flaccid paralysis, as well as highlighting the main measures to be taken in the emergency department when faced with a patient with hyperkalemia.

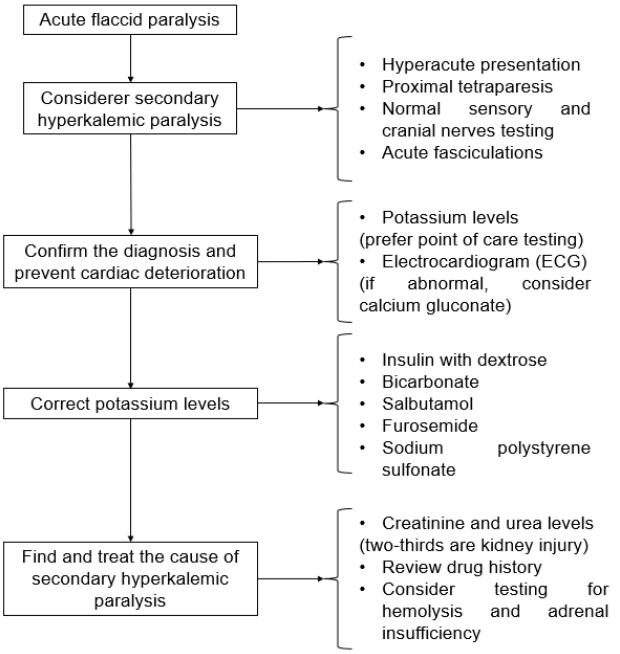

Figure 3. Algorithm for approaching patients with acute flaccid paralysis.

Conclusion

Secondary hyperkalemic paralysis is more common in middle-aged males. Symmetric acute tetraparesis without sensory or cranial nerves findings is the classical presentation. The most common causes are acute or chronic kidney injuries. Correction of potassium levels is associated with excellent recovery of the limb weakness.

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Informações sobre o Artigo

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