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An unusual stroke mimic: A case report

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Abstract

Hypokalaemic paralysis is a rare disorder characterized by rapid onset of symmetrical flaccid skeletal muscle weakness in the presence of reduced serum potassium levels. It is categorized as primary or secondary depending on the aetiology. Asymmetric or unilateral muscle weakness in hypokalaemic patients is a rare presentation. In patients with comorbid cardiovascular risk factors, this atypical manifestation can mimic acute stroke. Only a few of such cases have been reported in the literature. This report discusses the case of a 46-year-old hypertensive Ghanaian woman who presented to a District Hospital with sudden-onset right-sided flaccid weakness and a high blood pressure. Acute stroke was ruled out with computed tomography scan of the brain. Further laboratory evaluation demonstrated reduced serum potassium level, which was corrected with subsequent dramatic resolution of the muscle weakness.

Keywords

Hypokalaemic paralysis, hypokalaemic periodic paralysis, unilateral paralysis, asymmetric paralysis, hypokalaemia, potassium chloride

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Introduction

Hypokalaemic paralysis refers to a disorder characterized by symmetrical flaccid skeletal muscle weakness occurring in the presence of low serum potassium levels. The muscle weakness often resolves completely with correction of hypokalaemia.[1]

Based on the aetiology, it is classified as primary or secondary.[1] Hypokalaemic periodic paralysis (the primary type) is further categorized into familial (hereditary) and acquired types. Familial hypokalaemic periodic paralysis is a channelopathy caused by mutations in either dihydropyridine-sensitive skeletal muscle calcium channel gene (*CACNA1S*) or voltage-sensitive sodium channel gene (*SCN4A*). Mutations in other genes such as *KCNJ2* and *KCNJ18* have also been identified.[2] It is an autosomal dominant disorder with reduced clinical expression in females as a result of lower penetrance in comparison to males, leading to fewer paralytic attacks in females.[3] About one-third of cases are sporadic and represent new mutations.[4] Acquired hypokalaemic periodic paralysis may be seen in patients with thyrotoxicosis.[5] The estimated prevalence of hypokalaemic periodic paralysis is 1 in 100,000.[6]

Hypokalaemia resulting from diarrhoea and vomiting, as well as renal potassium losses that occur in diuretic therapy, renal tubular acidosis and primary hyperaldosteronism, can also lead to muscle paralysis.[7] This is referred to as secondary hypokalaemic paralysis.

The final common pathophysiologic mechanism is hyperpolarization of muscle fibres, which renders them electrically inexcitable, resulting in failure of muscle action potential and ultimately leading to attacks of flaccid paralysis.[4] Here, we discuss the case of a middle-aged hypertensive Ghanaian woman who presented to our hospital with an attack of unilateral hypokalaemic paralysis masquerading as an acute stroke.

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Case presentation

A 46-year-old Ghanaian woman who had been hypertensive for 4 years but non-compliant with treatment presented to our hospital with right-sided weakness. She noticed the weakness upon waking up from sleep in the morning. She could not move her right upper and lower limbs making it difficult for her to get out of bed. She could, however, move her left extremities without any difficulty. There was no slurred speech, dysphagia, headache, seizures, loss of consciousness or loss of sensation. There was also no bladder or bowel dysfunction. Again, she denied diarrhoea, vomiting, heat intolerance or excessive sweating. There was no history of laxative or diuretic use prior to the onset of the right-sided weakness. She neither engaged in any strenuous physical activity nor took high carbohydrate diet before going to bed the previous night. She did not have a past or family history of stroke or any other form of muscle weakness. Her husband took her to a health centre where her blood pressure was found to be 172/130 mmHg. She was given nifedipine 30 mg stat by oral route and subsequently referred to our hospital (Methodist Hospital, Wenchi, Ghana) for further management.

On physical examination, she was afebrile with a temperature of 36.7°C, anicteric, not pale and not in respiratory distress. She had a regular pulse of 94 beats per minute and repeat blood pressure was 165/120 mmHg. She was alert, awake and oriented in time, place and person. She had supple neck and negative Kernig's sign.[8] On neurological assessment, muscle power was 3/5 and 2/5 in the right upper and lower limbs, respectively. Tone and deep tendon reflexes were reduced with flexor plantar response. She had intact sensation and normal cranial nerve examination findings.

Laboratory investigations showed normal full blood count, renal and liver biochemistries, lipid profile, fasting blood sugar (5.8 mmol/L) and negative HIV status. Based on the clinical findings, acute stroke was suspected. However, plain computed tomography (CT) scan of the brain was normal. Subsequent contrast-enhanced CT scan of the brain also did not reveal any space-occupying lesion. Magnetic resonance imaging of the brain was not done because she could not afford, and it is also not readily available in the part of our country where she was hospitalized. Further laboratory workup demonstrated reduced serum potassium level of 2.6 mmol/L. Other relevant electrolytes, thyroid stimulating hormone, plasma aldosterone concentration and plasma renin activity were all normal as shown in Table 1. Resting electrocardiogram (Figure 1) showed normal sinus rhythm with widespread ST segment and T wave changes with prolonged QT interval (corrected QT interval of 500 ms). Genetic testing for channelopathies was not done due to its unavailability in our country.

Following intravenous administration of 60 mEq of potassium chloride, the right-sided weakness completely resolved after 48 hours with serum potassium level rising to 3.5 mmol/L. Oral supplementation with potassium chloride 600 mg three times daily was given afterwards. Her blood pressure was controlled with nifedipine 30 mg daily and lisinopril 10 mg daily. Electrocardiographic changes returned to normal. Based on the clinical findings as well as the rapid and complete resolution of the right-sided weakness after correction of the hypokalaemia, a diagnosis of hypokalaemic paralysis was made. At follow-up 1 month and 4 months after discharge, her serum potassium was 4.3 mmol/L and 4.6 mmol/L, respectively, with no recurrence of muscle weakness.

Table 1. Results of other relevant laboratory tests.

Test	Result	Reference range
Serum potassium	2.6 mmol/L	3.5–5.5
Serum sodium	140 mmol/L	135–145
Serum magnesium	1.9 mg/dL	1.7–2.2
Serum chloride	105 mmol/L	98–107
Serum bicarbonate	26.27 mmol/L	22–29
Thyroid stimulating hormone	0.73 mU/L	0.51–4.30
Plasma aldosterone concentration	13 ng/dL	5–30
Plasma renin activity	1.9 ng/mL/hr	0.7–3.3
Aldosterone-to-renin ratio	6.8 ng/dL per ng/mL/hr	<20

Discussion

Prompt diagnosis of diseases mimicking acute stroke is necessary to avoid undue delays in the administration of appropriate therapy. Common stroke mimics include space-occupying lesions, subdural haematoma, migraine, hypoglycaemia and Todd's paralysis.

In hypokalaemic paralysis, the patients typically present with acute onset symmetrical flaccid muscle weakness predominantly involving proximal muscles and the lower limbs. Involvement of upper extremities is often not as severe as the lower limbs.[2] Additional typical features include preservation of consciousness level, absence of bladder and bowel dysfunction, intact sensation and sparing of bulbar, ocular and respiratory muscles,[9] although involvement of bulbar and respiratory muscles has been reported.[10] Classic neurological examination findings during a paralytic attack are reduced muscle power, hypotonia and hyporeflexia or areflexia.[4]

Hypokalaemic paralysis presenting with asymmetric or unilateral muscle weakness is a rare occurrence and only a few cases have been reported in the literature. A careful review of the reported cases shows varied clinical manifestations in these hypokalaemic patients. In three of the case reports, the patients had sudden-onset right-sided weakness.[11–13] Another patient also developed sudden onset of right-sided weakness which was associated with right facial

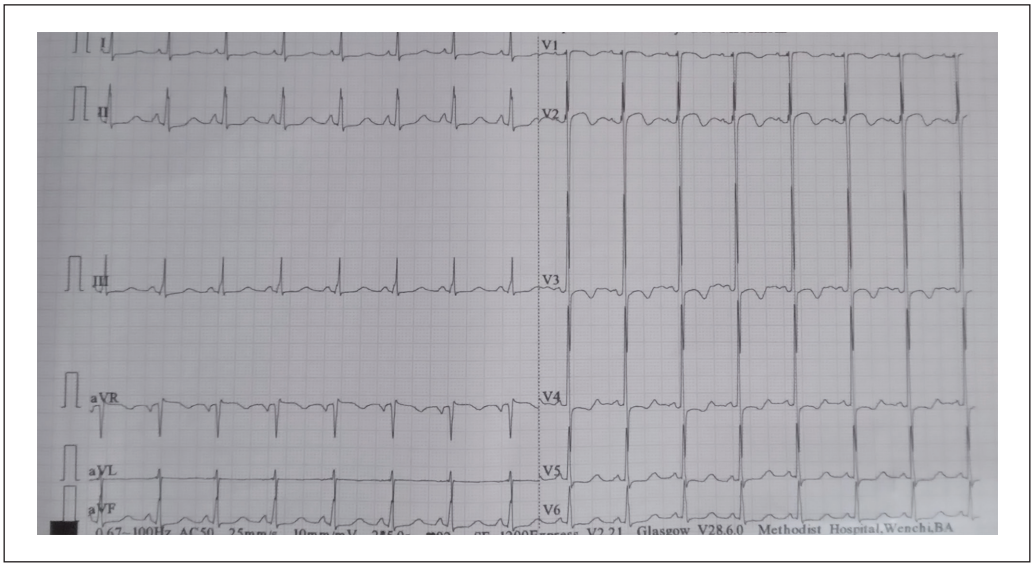

Figure 1. Standard 12-lead electrocardiogram showing normal sinus rhythm, widespread ST segment/T wave changes and QT prolongation.

droop and dysarthria.[14] This atypical manifestation of hypokalaemic paralysis can be misdiagnosed as acute stroke especially in individuals who have comorbid cardiovascular risk factors just as our patient did. Unlike in our case, the patients in some previous reports had monoparesis.[15,16] Other reported atypical patterns of muscle weakness associated with hypokalaemia include right hand weakness progressing to paraparesis,[17] right arm weakness with subsequent development of tetraparesis[18] and left thumb weakness.[19] Table 2 provides information on the clinical features, degree of hypokalaemia and how hypokalaemic paralysis was diagnosed in the aforementioned case reports.

Given how the patient presented, acute stroke was initially suspected. Another likely differential diagnosis was transient ischaemic attack. The diagnosis of stroke requires an objective evidence of central nervous system infarction or spontaneous intracerebral haemorrhage on neuroimaging. Transient ischaemic attack on the other hand is characterized by an episode of focal neurological dysfunction followed by complete resolution of symptoms without evidence of acute infarction. The symptoms typically last less than an hour. The diagnosis of transient ischaemic attack is particularly difficult due to the transitory nature of the symptoms and the lack of an established laboratory biomarker.[20,21] Our patient had a normal brain CT scan. Magnetic resonance imaging, however, could not be done. As shown in Table 2, one patient presenting with right-sided weakness had CT scan of the brain to exclude a stroke[12] just like in our case. One had both CT scan and CT angiography,[13] while another underwent both magnetic resonance imaging and CT scanning of the brain.[15]

The onset of hypokalaemic periodic paralysis usually occurs in the first or second decade of life. Although the patient's age (46 years) falls outside this typical age range, she met these consensus diagnostic criteria for hypokalaemic periodic paralysis: paralytic attack with documented serum potassium < 3.5 mmol/L, muscle weakness involving more than one limb lasting longer than 2 hours, improved symptoms with potassium replacement and exclusion of other causes of hypokalaemia (absence of gastrointestinal losses; no thyrotoxicosis; no hyperaldosteronism, renal tubular acidosis, use of diuretics or laxative abuse).[22] As observed in all the cases indicated in Table 2, complete recovery of muscle strength following the correction of hypokalaemia was an important consideration in making the diagnosis of hypokalaemic paralysis. Muscle weakness often resolves completely after correction of hypokalaemia with a mean recovery time of 38.6 ± 20.3 hours[23] as was observed in our patient. Even though testing for skeletal muscle channel gene mutations was not done due to its unavailability in our country, it is important to note that about 30% of patients with hypokalaemic periodic paralysis do not have any of the classic mutations.[22]

Conclusion

Asymmetric or unilateral muscle weakness is a rare occurrence in hypokalaemic paralysis. This atypical presentation can masquerade as acute stroke particularly in patients with coexisting cardiovascular risk factors. It is therefore important for clinicians to bear this in mind while evaluating patients with stroke-like symptoms to avoid delayed diagnosis and initiation of appropriate therapy.

Table 2. Clinical features, degree of hypokalaemia and how hypokalaemic paralysis was diagnosed in the reported cases.

Case report	Age (years)/ sex	Clinical manifestation	Serum potassium (mmol/L)	Underlying aetiology	How HP was diagnosed
Lu et al.[11]	52, male	Sudden onset of right-sided weakness	1.8	Unknown	Diagnosis was established on the basis of rapid reversal of muscle weakness 24 hours after correction of low serum potassium.
Chiang et al.[12]	24, female	Sudden onset of right-sided weakness	2.0	Distal renal tubular acidosis	Normal brain CT scan with complete resolution of weakness after correction of hypokalaemia.
Lajeunesse et al.[13]	48, male	Sudden onset of right-sided weakness	3.4	Grave's disease	Diagnosis was informed by previous episode of muscle weakness, normal CT scan/CT angiography of the brain and spontaneous resolution of muscle weakness with near normal serum potassium.
Al-Handola et al.[14]	61, female	Sudden onset of right-sided weakness with right facial droop and dysarthria	2.7	Grave's disease	Normal brain CT scan/CT angiography and MRI. Prompt potassium replacement with complete reversal of muscle weakness and dysarthria after 3 days of symptom onset.
Liu et al.[15]	80, female	Weakness of right lower limb	2.36	Gastroenteritis	Normal brain CT scan and MRI. Immediate potassium supplementation with complete recovery of muscle strength after 48 hours.
Katchanov et al.[16]	70, male	Progressive right leg weakness	1.5	Liquorice consumption	Potassium replacement with recovery of full muscle strength after 5 days of admission.
Ma et al.[17]	55, male	Right hand weakness progressing to paraparesis	2.83	Hypokalaemic periodic paralysis	Extensive diagnostic workup with normal findings but met some diagnostic criteria for HPP, including complete resolution of muscle weakness after potassium supplementation.
Sen et al.[18]	38, male	Right arm weakness progressing to tetraparesis	1.8	Crohn's disease	Correction of hypokalaemia with reversal of muscle weakness after 72 hours.
Chui et al.[19]	48, female	Recurrent left thumb weakness	1.9	Adrenal adenoma	Diagnosis of HP was based on the history of episodic focal muscle weakness and rapid resolution of weakness after replacement of serum potassium.

HP: Hypokalaemic paralysis; HPP: Hypokalaemic periodic paralysis; CT: Computed tomography; MRI: Magnetic resonance imaging.

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Author contributions

P.A. conceptualized the case report, collected the relevant data and wrote the original article. G.M.A. and M.A. reviewed the article and made significant input. All the authors have read and approved of the final article.

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Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

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