Case Report

Hypokalemic Periodic Paralysis in Pregnancy: A Case Report

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Abstract

Background: Myopathy is a disorder of skeletal muscles and has a rare occurrence in pregnancy. It may present with numbness/weakness. The occurrence of isolated weakness involving all the limbs is alarming to the patient and the diagnosis can be challenging to the Obstetrician. We present a case of hypokalaemic paralysis in pregnancy.

Case report: A 32-year-old grand multipara presented at 31 weeks gestation with numbness in all limbs for nine days and one-day history of weakness in all limbs. She had a similar episode in her last pregnancy with complete resolution by the end of puerperium. On examination, she was conscious with a Glasgow Coma Scale score was 15/15, had no signs of meningeal irritation, and no cranial nerve palsy. She had normal muscle bulk; the power of 4/5 in both upper limbs and 3/5 in both lower limbs. There was no clearly defined sensory level. Planter reflex was flexor symmetrically. A review of other systems was unremarkable. Her PCV was 35% and random blood glucose was 4.2mmol/l. Serum biochemistry showed severe hypokalaemia of 1.8mmol/l with normal levels of sodium and chloride. Urinary potassium level was normal. She had parenteral correction of potassium with complete resolution of weakness and she was maintained on oral potassium supplements. She had an uneventful delivery at 37 weeks gestation.

Conclusion: Measuring the serum level of potassium should be part of the initial workup when evaluating pregnant women presenting with muscle weakness. Multidisciplinary management leads to early diagnosis, prompt management, and a good prognosis.

Keywords: hypokalemia, muscle weakness, pregnancy.

Introduction

The main causes of myopathy could be genetic, inflammatory, endocrine, metabolic, and idiopathic in many cases. The heterogeneity of the causes makes it more difficult to diagnose. Hypokalemic myopathy is characterized by acute muscular weakness with low levels of potassium (<3.5mmol/l)[1]. Hypokalemic periodic paralysis (HPP) is the most common of the periodic paralyses but is still quite rare with an estimated prevalence of 1 in 100,000[2].

The weakness may be trivial or sometimes life-threatening[3]. The occurrence of weakness in

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Access this article online

Quick Response Code:

Website: www.nigerianmedjournal.org

How to cite this article: Aliyu RM, Bawa US, Abubakar SA, Abdullahi AS, Suleiman HM, Sarkin-Pawa Z, Yusuf RY, Abdul MA. Hypokalemic periodic paralysis in pregnancy: a case report. Niger Med J 2021; 62; (2):92-95

pregnancy is alarming to the patient and the diagnosis can be challenging to the Obstetrician. The challenge lies in prompt recognition of the cause and prompt treatment. A multidisciplinary approach is vital in the management of such patients. We present a case of HPP in pregnancy.

Case report

Mrs. I.M was a 32-year-old gravida 6, para 5 whose last childbirth was five years ago. She presented at 31 weeks gestation with numbness in all limbs of nine days and one-day history of weakness in all limbs. The numbness started from the lower limbs and then involved the upper limbs. She was still able to walk unsupported till a day before the presentation when she began to have profound weakness starting in the same sequence as the numbness. She was unable to stand unsupported or walk and she could not lift her hand to the level of her head necessitating her presentation. However, she still had normal neck control and optimal sphincteric function.

She had a two-day history of catarrhal symptoms. There was no associated loss of consciousness, body swelling, or fever. She had no difficulty with breathing or swallowing. There was no preceding history of diarrhoea or vomiting.

This was the second episode of such weakness in the patient's life. The first episode occurred during her last pregnancy at term. She presented to a General Hospital and had an uneventful induction of labour. The weakness slowly began to resolve and she was able to walk with support by the fourth day postpartum, and had complete resolution of weakness at the sixth week postpartum.

She had never experienced such weakness following intake of a large meal, upon waking up from sleep, or following rest after exertion. She was not a known diabetic or hypertensive. There was no history of drug ingestion except for the prescribed haematinics. She had no family history of similar symptoms.

Index pregnancy was booked at 20 weeks gestation in our hospital. Infection screens for HIV, Syphilis, and Hepatitis B and C were negative. The pregnancy had been uneventful till the onset of these symptoms.

On examination, she was a young woman who was anxious, afebrile, and was not pale. Her pulse rate was 86 beats per minute, regular, and of normal volume. Her blood pressure was 120/80 mmHg and the heart sounds were normal. Her Glasgow Coma Scale score was 15/15; had no signs of meningeal irritation and no cranial nerve palsy. She had normal muscle bulk and sensation was preserved in all limbs. The power in both upper limbs and both lower limbs was 4/5 and 3/5 respectively; deep tendon reflexes were reduced and plantar reflex was flexor bilaterally. There was no clearly defined sensory level. An initial diagnosis of myopathy in pregnancy was made. The neurological review was sought for and diagnosis of periodic paralysis in pregnancy was suspected to rule out acute inflammatory demyelinating polyradiculoneuropathy (AIDP).

Her Packed Cell Volume was 35% and her blood glucose was 4.2mmol/l. Serum biochemistry showed severe hypokalaemia of 1.8mmol/l and normal levels of sodium, bicarbonate, chloride, urea, and creatinine levels. Urinary electrolytes were within normal limits. A diagnosis of HPP in pregnancy was finally established.

She was commenced on parenteral potassium supplementation with gradual correction of hypokalaemia. There was a gradual improvement of the weakness which completely resolved with normalisation of serum potassium level by the seventh day of admission (see Table 1). She was commenced on an oral potassium supplement. Serum potassium levels remained within normal limits for the rest of the antenatal period. She had an uneventful spontaneous vaginal delivery at 37 weeks gestation to a live infant that weighed 3.25kg with an Apgar score of 9 and 10 in the first and fifth minute respectively. The puerperium was uneventful.

Table 1: Trend of patient's electrolyte profile

Timing	Potassium (3.6mmol/l-5.2mmol/l)	Sodium (136mmol/l-145mmol/l)	Chloride (94mmol/l-108mmol/l)	Bicarbonate (24mmol/l-32mmol/l)	Urea (2.5mmol/l-6.5mmol/l)
Admission	1.8	136	88	24	2.5
Day 2	2.2	136	87	24	2.4
Day 4	3.2	139	89	26	2.5
Day 7	4.2	140	90	24	2.3
37 weeks gestation	4.1	142	106	24	2.8
2 weeks postpartum	4.3	142	102	26	3.0

Discussion

HPP in pregnancy is a rarity. It usually manifests with varying degrees of weakness associated with a low level of serum potassium. This patient presented with

initial numbness which progressed to profound weakness and led to an inability to walk. Her serum potassium level was 1.8mmol/l which represents severe hypokalaemia thus an emergency. Irrespective of the cause of hypokalaemia, this acute episode should be managed with potassium replacement. This patient initially had parenteral correction of potassium with complete resolution of symptoms. This degree of hypokalemia is consistent with other reports in the literature[1,4].

Hypokalemia could result from poor intake, urinary loss, or redistribution into the cells. The aetiology of hypokalemia in this patient is likely due to redistribution into cells because she had no history of external loss of potassium from diarrhoea, vomiting, or urinary loss. Other reported causes of hypokalemic myopathy in pregnancy include hypokalemic periodic paralysis, pica (especially clay eating), and thyrotoxic periodic paralysis[1,2,4-6].

HPP is an autosomal dominant channelopathy that classically has an onset during adolescence and presents with weakness which is usually precipitated by some triggers like intake of a very large carbohydrate meal or rest after exertion. Sporadic occurrence though less common has also been reported[7]. Though this patient had the first episode of weakness in adulthood and had no history of experiencing weakness following intake of a large meal, upon waking up from sleep, following rest after exertion, or family history of similar illness, it may suggest the existence of an atypical variant of HPP. Pregnancy and delivery have been reported to exacerbate familial HPP[8].

Reports of clay eating causing severe hypokalemia and muscle weakness exist in literature[4,10]. Clay has been found to bind potassium in the intestines but this patient had no history of clay eating. Thyrotoxic hypokalaemic paralysis could also present with episodes of weakness and even quadriplegia but this was unlikely in this patient based on the absence of thyrotoxic symptoms like weight loss, tachycardia, goitre, tremor, and ophthalmopathy.

Hypokalemic paralysis in pregnancy has been reported to occur following glucose screen test, anaesthesia, and betamethasone injection but these events were absent in this patient[1,11].

A pregnancy complicated by HPP is a high-risk pregnancy because cardiac and respiratory failure associated with hypokalemia can easily lead to maternal death[12]. Fortunately, this alarming presentation is readily reversed with the restoration of potassium to normal levels and other supportive therapy. Measuring serum levels of potassium should be part of the initial workup when evaluating pregnant women presenting with muscle weakness. Multidisciplinary management leads to early diagnosis, prompt management, and good prognosis[11].

Source of support: None

Acknowledgment: None

Conflict of interest: None to be declared

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