Acute Hypokalaemic Paralysis Complicating Pregnancy: A First Experience in a Tertiary Institution in Southwest Nigeria

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Abstract

Acute hypokalaemic paralysis complicating pregnancy presents a unique challenge and an uncommon phenomenon, and when it occurs, presenting symptoms can range from mild to life-threatening ones. This condition occurs in the presence of severely low levels of potassium and precipitating factors can be congenital or acquired. An important acquired factor related to pregnancy is hyperemesis gravidarum. This case report centers on a 31-years old primigravida at about 22 weeks gestation, who presented with progressive bilateral lower limb weakness over a period of 4 days, with a background history of previous management for hyperemesis gravidarum in our facility at 11 weeks and several episodes of vomiting in index pregnancy managed at private facility. Potassium level at presentation was 1.25mmol/l. Following a diagnosis of acute hypokalaemic paralysis, she had potassium replacement and the presenting symptom improved with correction of the potassium. Her pregnancy progressed to term and she was delivered of her baby with no adverse events recorded. Early diagnosis and prompt institution of care are important for a good outcome when faced with acute Hypokalaemic paralysis complicating pregnancy.

Keywords: Acute hypokalaemic paralysis, High-risk,pregnancy,Hyperemesis gravidarum, Hypokalaemia, Myopathy in pregnancy,Pica, potassium supplementation.

Introduction

Women in the reproductive age group can have varying degrees of fatigue and muscular weakness, more so, when they are pregnant and these fatigue and weaknesses can be related to hyper- or hypokalaemia(1). Hypokalaemic paralysis as an entity is rare and could be life-threatening especially when not suspected and diagnosed on time. However, if the diagnosis is timely and prompt treatment instituted, patients affected recover fully and without any clinical sequelae(2). Hypokalaemic periodic paralysis occur with a prevalence of about 1 in 100,000 and the causes of myopathy are idiopathic in majority of cases and when there is a known cause, it could be as a result of metabolic, endocrine, genetic or inflammatory conditions(3). We present the first case of hypokalaemic-induced paralysis in our centre.

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Case Presentation

The patient was Mrs. A.B, a 31-years old booked primigravida at estimated gestational age of 21 weeks + 6 days who presented with weakness of both the lower limbs of 4 days duration. The weakness was said to be mild initially, not ascending, associated difficulty with walking and worsened over a period of 4 days following which she could no longer stand on her feet. There was associated swelling of both legs. No history trauma to the legs and both upper limbs were normal. There was no history of similar symptoms in the past. There was no difficulty with breathing, cough, orthopnoea or paroxysmal nocturnal dyspnoea. She was not a known hypertensive or diabetic. The index pregnancy was booked early at estimated gestational age of 6 weeks and all the booking parameters were normal. She had one dose each of intramuscular tetanus toxoid and intermittent preventive therapy for malaria. There was history of hospitalization in our facility at estimated gestational age of 11 weeks + 4 days on account of hyperemesis gravidarum. She also said she had been in and out of a private facility where she was admitted and managed for vomiting in index pregnancy.

On examination, she was fully conscious with a Glasgow coma score of 15. She was not pale, not febrile, not dehydrated, but she had bilateral pitting pedal edema up to below her knee. Her vital signs were all normal; blood pressure was 110/70mmHg. There were no muscle wasting in her lower and upper limbs. Power in both upper limbs were 5, while that in both her lower limbs were 2. The symphysio-fundal height was 26cm and the fetal heart sound was heard and regular.

A provisional diagnosis of myopathy in pregnancy ? cause to rule out Guillain-Barre syndrome (GBS) was made. With further review the possibility of the diagnosis being GBS was discarded. Obstetrics ultrasound scan showed a huge fibroid in the lower utero-cervical region measuring 10.1 x 11.0cm. The full blood count was normal as well as retroviral screening and hepatitis B surface antigen. The electrolyte, urea and creatinine result showed normal findings except for the potassium, which was severely low with a value of 1.25mmol/l. A diagnosis of acute hypokalaemic paralysis complicating pregnancy was made and with a potassium deficit of 62.1mmol, patient was commenced on parenteral potassium supplementation by adding 20mmol of potassium chloride into alternate pints of intravenous fluid. The potassium level gradually increased, culminating in improvement in presenting symptom with total resolution following normalization of potassium level. Correction was achieved over a period of about 4 days and the patient was placed on oral potassium supplements. She was discharged home after a nine-day hospital stay.

Table 1. Patient's electrolyte profile

	Potassium (3-5) mmol/l	Sodium (135-145) mmol/l	Chloride (90-110) mmol/l	Urea (2.5-5.8) mmol/l	Creatinine (60-129) mmol/l
Admission	**1.25**	**135.8**	**90.5**	**2.4**	**62.1**
Day 3	**2.13**	-	-	-	-
Day 4	**2.93**	-	-	-	-
Day 5	**3.53**	-	-	-	-
Day 6	**4.02**	-	-	-	-
Day 9	**3.98**	-	-	-	-

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Her potassium level remained normal and the rest of her pregnancy was uneventful. She had an elective Caesarean section at 38 weeks gestation on account of huge low-lying uterine fibroid and was delivered of a live female neonate with APGAR score of 7 and 9 at the first and fifth minutes respectively and a birth weight of 3.05kg. The delivery and puerperium were not complicated.

Discussion

Hypokalaemia-inducedparalysis
complicating pregnancy makes it a high-risk pregnancy and it should be managed as such, with close monitoring and institution of a multidisciplinary approach in the management(4). Hypokalaemia is divided into three categories, which are mild, with potassium level of 3 – 3.5mmol/l, moderate with a level of 2.5 – 2.9mmol/l and severe when potassium level is <2.5mmol/l. Paralysis induced by hypokalaemia is a rare event, usually occurring at severely low levels of potassium(5). The case of Mrs. A.B. presented here was therefore paralysis induced by severe hypokalaemia since her serum potassium at presentation was 1.25mmol/l. The aetiology of hypokalaemia ranges from congenital to acquired causes. In ruling out the congenital causes, it is important to make enquiries into episodes of weakness following exposure to risk factors such as exercise, heavy carbohydrate and increased salt intake(4,5). None of such precipitating factors were found in Mrs. A.B. It is important also to rule out other congenital conditions such as thyrotoxic periodic paralysis, Anderson Tawill syndrome and familial hypokalaemic periodic paralysis (FHPP)(4). An important acquired cause of hypokalaemia specific to pregnancy is hyperemesis gravidarum which result in loss of potassium from the gastrointestinal system(6). The index patient was admitted at a gestational age of 11 weeks and 4 days, wherein she was managed as a case of hyperemesis gravidarum. She also gave an account of being managed in a private facility for several episodes of vomiting in pregnancy. Other acquired causes of hypokalaemia include poor intake, diarrhea and urinary loss. Pica in form of clay eating, known as geophagia has also been shown to cause hypokalaemia by binding to potassium in the gut, thereby preventing absorption(3) but this was not the case in the index patient.

Clinical presentation depends how low the level of potassium is(3). Most patients become symptomatic when the potassium level is <2.5mmol/l and paresis could be severe enough to warrant mechanical ventilation, if the respiratory system is compromised(5). If there is a rapid fall in the level of potassium, symptoms of hypokalaemia could manifest at higher levels of potassium(7). Aside muscle weakness, other symptoms of hypokalaemia include cramping, fatigue, constipation and palpitation.(8) Severely depleted levels of potassium leads to arrhythmias and paralysis and such levels herald changes in electrocardiogram (ECG) which include T-wave inversion, U-wave elevation and ST-segment depression(9). The index patient had a normal ECG report but presented with paralysis of her lower limbs.

The major factor in the management of hypokalaemia-induced paralysis is early recognition and prompt treatment. The patient had early diagnosis made from baseline investigations requested at presentation. Key in the treatment is avoidance of triggers and potassium supplementation to correct hypokalaemia(10). Mrs. A.B. had early treatment with parenteral potassium which was later converted to oral potassium supplementation. Following initiation of treatment, her potassium level gradually increased, and this was followed by

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continuous improvement in her symptom with eventual resolution after potassium level had been corrected. Her baby was closely monitored for episodes of flaccid paralysis after birth(4) but the baby remained healthy.

Her treatment was prompt before any other symptoms or complication such as arrhythmia or respiratory failure could occur.

Conclusion

Acute hypokalaemic paralysis complicating pregnancy is a rare but potentially life-threatening condition which requires a high index of suspicion, prompt diagnosis and timely correction of serum potassium to prevent adverse maternal and fetal outcomes. This case underscores the importance of routine electrolyte monitoring in pregnant women who present with acute muscle weakness. Early diagnosis and swift institution of appropriate treatment not only improves maternal outcome, but also safeguard fetal wellbeing.

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