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■ Case Report

Hypokalaemia-Induced Paralysis in Pregnancy

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ABSTRACT

Hypokalaemia-induced paralysis is characterized by acute muscular weakness with low levels of potassium (<3.5 mmol/l). Clinical features may range from numbness/weakness to complete paralysis. We present a case of a 26-year-old G1P0 at GA of 32 weeks with preeclampsia, who presented with acute onset of weakness of both upper and lower limbs. She was diagnosed to have Hypokalaemic paralysis with potassium levels of 1.3 mmol/L. The medical condition improved promptly on intravenous potassium replacement. Pregnancy was continued till 34 weeks with close feto-maternal surveillance, use of antihypertensive and regular monitoring of serum potassium levels. Postpartum period was uneventful, patient regained full control of her upper and lower limbs and she was discharged after two weeks when potassium levels and BP returned to normal.

Keywords: case report, hypokalaemia, paralysis, pregnancy

INTRODUCTION

Hypokalaemia in pregnancy is a rare occurrence[1,2] and It occurs in around 1% of pregnancies.[3] Common causes of hypokalaemia during pregnancy are: dilution effect; diarrhoea; hyperemesis gravidarum;[4] use of diuretics;[1,5] and use of antibiotics like gentamicin and carbenicillin.[4] Other causes are Gitelman syndrome, Bartter syndrome etc. Hypokalaemia, depending on its severity may present with weakness, muscle cramps, muscular paralysis, confusion, constipation, an abnormal heart rhythm or an irregular heartbeat, tingling or numbness and increased urination.[2] The treatment is to replenish serum potassium with a potassium supplement and correct the underlying condition.

CASE HISTORY

R.A. was a 26-year-old G1P0 at a gestational age (GA) of 32 weeks, who presented to the emergency room with complaint of sudden onset of weakness of the upper and lower limbs of 5-days duration. She noticed weakness of all her extremities and she experienced difficulty in walking and carrying objects with weakness progressively worsening with time. She had no difficulty with breathing or swallowing and She was able to move her neck and facial muscles. There was no history of pain or paraesthesia. Pregnancy was spontaneously conceived and had been uneventful until GA of 20 weeks when she was found to have elevated blood pressure and She was commenced on anti-hypertensive.

She presented at the source of referral, a primary level of care, where she was evaluated and noted to be have elevated blood pressure (BP 170/110mmHg) and then, was referred to our facility for expert care. She was placed on tablet (Tab) labetalol, Tab alpha methyl-dopa and Tab nifedipine. There was no history of alcohol intake or significant changes in her diet. No history of hypothyroidism or similar episodes in her family. She had not been previously diagnosed with any medical illness. There was no history suggestive of trauma to the back, faecal or urinary incontinence.

Clinical Findings

She was afebrile, not pale, not dehydrated and She had no pedal oedema. Her vital signs were normal except for the elevated blood pressure (140/90 mmHg). She had a Glasgow coma scale score of 15/15. The muscle bulk was preserved across all limbs. The tone and reflexes were normal across all joints. She had a power grading of 2 in both upper limbs, and lower limbs. Light sensation was intact across the dermatomes. The joint position sense was preserved. On abdominal examination, the symphysio-fundal height was 30cm; a singleton foetus in longitudinal lie and cephalic presentation was palpated. Foetal Heart Rate was 148 per minute, and regular. She had a Packed cell volume of 39%, random blood glucose was 98mg/dl, urinalysis was significant for haematuria (+++) and proteinuria (+). The serum electrolyte showed hypokalaemia of 1.3mmol/L; while the serum urea and creatinine were within normal limits.

A diagnosis of pre-eclampsia with hypokalaemia? cause was made. She was co-managed with the Neurologist who made an assessment of quadriparesis? Cause, with possibility of Guillain-Barre syndrome (GBS). However, the presentation and the findings on examination were not in keeping with the diagnosis of GBS. No other cause was found for the quadriparesis asides the hypokalaemia. A magnetic resonance imaging (MRI) of the brain was requested for but this could not be done due to shortage of finances. She was placed on IV Magnesium sulphate for seizure prophylaxis and Intramuscular Dexamethasone for foetal lung maturity. Potassium correction was commenced with Potassium chloride (KCl) 20mmols into alternate pint of fluid after calculating potassium deficit to be 131.6mmol/L and this was corrected over 4 days. Her blood pressure control was optimal while she was on admission. She had an urgent caesarean section done 6 days into admission and was delivered of a live female neonate, birth weight-2.0kg, Apgar score-9 and 10 at the first and fifth minute. She started physiotherapy on the 2nd day post-delivery. Her clinical state improved upon potassium correction with serum potassium value of 3.58mmol/l and she was able to walk and carry out her usual activities by the 12th day of admission. She was discharged home on oral potassium chloride, and she was seen in the outpatient department two weeks after. She had no complaints; her potassium level was normal, and the infant was well.

DISCUSSION

Hypokalaemia-induced paralysis during pregnancy is a rare occurrence. It manifests as acute muscular weakness associated with low potassium levels.[1] In normal pregnancy, there are alterations in fluid, electrolytes and various gestational hormones and there are several physiologic changes, like hyperemesis gravidarum, that can lead to hypokalaemia specific to pregnancy.[5] The aetiology of hypokalaemia may be varied, ranging from congenital to acquired causes.[2] A careful insight into the past history regarding the time of onset, episodes of weakness; the precipitating factors like: exercise, carbohydrate load, increased salt intake etc, may be helpful to make a diagnosis of congenital abnormalities.[2] R.A. did not give such history in the past, nor were there any other members in the family who suffered from a similar condition. Also, history of hyperemesis gravidarum was not elicited. A report of hypokalaemia-induced paralysis following glucose screen has been reported in literature,[1] but no such precipitating factors exist in R.A. The electrolyte profile was normal in this patient except for hypokalaemia. Hypokalaemia is divided into three categories: mild (K^+:3.0-3.5mmol/L), moderate (K^+:2.5-2.9mmol/L), and severe (K^+<2.5mmol/L).

Most patients become symptomatic when the serum potassium falls to <2.5mmol/L. R.A. had a serum potassium of 1.3mmol/L, thus, she had severe hypokalaemia.

Some syndromes associated with hypokalaemia in pregnancy are Bartter, Gitelman and Geller syndrome.[4,6,7] These are inherited hypokalaemia salt-losing tubulopathies. Bartter and Gitelman syndromes are inherited as autosomal recessive traits,[6] while Geller syndrome is an autosomal dominant disease, which presents with hypokalaemia and hypertension during pregnancy, and usually resolves post-partum.[7] Patients who are diagnosed with Bartter or Gitelman syndromes have normal or low blood pressure with elevated renin and aldosterone levels.[6] R.A. had elevated blood pressure in index pregnancy, thus ruling out the possibility of having these two syndromes. However, in R.A.'s case, Geller syndrome may be a differential as it usually presents with hypokalaemia and hypertension during pregnancy.

The occurrence of paresis in a pregnant woman may sometimes be severe enough to warrant mechanical ventilation if the respiratory system is compromised.[2] The challenge lies in timely recognition of the cause and prompt correction. The acute episode may be managed with intravenous potassium replacement and maintenance with oral potassium supplements. In R.A., routine investigations done led to the early recognition of this condition, hence preventing more serious complications like respiratory paralysis or arrhythmias.

The proper management of a pregnant woman with hypokalaemia requires emergency stabilization for patients with moderate to severe hypokalaemia, identification of the underlying cause and titration of K^+ based on the cause and magnitude of the deficit.[3] Hypokalaemic paralysis is promptly reversible with potassium administration and vaginal delivery is usually possible with careful monitoring. The goal is to normalize the serum potassium level by administering potassium chloride (KCl) in oral or parenteral forms to alleviate the symptoms of muscle weakness. R.A. had an initial correction with parenteral KCl before changing to oral formulation. She had a caesarean section as she was diagnosed with preeclampsia with an unfavourable cervix. Also, the neonate may have episodes of flaccid paralysis and or respiratory distress at birth, therefore, feeding and respiration may be a problem; thus, the neonate has to be closely monitored under expert supervision.[2] At birth, R.A.'s baby was very healthy, with no feeding or respiratory problems.

CONCLUSION

Hypokalaemic paralyses represent a heterogeneous group of disorders with a final common pathway presenting as acute weakness and hypokalaemia. Though the aetiology of the hypokalaemia was uncertain in the case presented, it improved with potassium correction over time. With timely intervention and prompt management, adverse outcomes may be averted.

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REFERENCES

1. Kushna DK, Drake JG, Block WA. Hypokalemic periodic paralysis in pregnancy after 1-hour glucose screen. Obs Gynecol. 2000;95(6):1037.
2. Kulkarni M, Srividya T, Gopal N. Hypokalemic Paraplegia in Pregnancy. JCDR. 2014;8(6):03–4.
3. Mbongozi XB, Businge CB, Mdaka ML, Wandabwa JN. The practice of geophagia and the predisposing factors to hypokalaemia among pregnant women in rural eastern cape province, south africa. S Afr J Obstet Gynaecol. 2019;25(3):84–8.
4. Shinar S, Gal-oz A, Weinstein T, Levin I, Maslovitz S. Gitelman syndrome during pregnancy – from diagnosis to treatment: a case series and review of the literature. CRPM. 2014;3(1):39–43.
5. Yang CW, Li S, Dong Y. The Prevalence and Risk Factors of Hypokalemia in Pregnancy-Related Hospitalizations: A Nationwide Population Study. Int J Nephrol. 2021;2021.
6. Shibli A Al, Narchi H. Bartter and Gitelman syndromes: Spectrum of clinical manifestations caused by different mutations. World J Methodol. 2015;5(2):55.
7. Hindosh N, Hindosh R, Dada B, Bal S. Geller Syndrome: A Rare Cause of Persistent Hypokalemia During Pregnancy. Cureus. 2022;14(6):6–9.