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HYPOKALAEMIC PERIODIC PARALYSIS IN PREGNANCY- A RARE CASE

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ABSTRACT Hypokalemic Periodic Paralysis (HPP) is a rare skeletal muscle disorder characterized by intermittent episodes of focal or generalized weakness of skeletal muscles which can last for hours to even days, with concomitant hypokalemia[2]. Pregnancy has been previously reported to exacerbate the symptoms. We present a case of a 35-year-old G3P2L2 at 31 weeks pregnancy with previous 2 LSCS, who had an acute attack of paraplegia and was diagnosed as HPP with serum potassium levels of 1.8 mmol/L on admission secondary to Type 1 Renal Tubular Acidosis (RTA). She reported improvement in her symptoms after parenteral potassium chloride(KCL) correction and requiring minimal oral potassium supplementation throughout rest of her pregnancy. Diligent care was taken to avoid known triggers, with emphasis on dietary modification and potassium supplementation. The patient progressed till 37 weeks and underwent Elective Caesarean Section under spinal anaesthesia without complications for obstetric indication. Postpartum period was uneventful and patient was discharged after 48 hours from hospital with normal potassium levels. This case highlights the importance of an individualized and multi-disciplinary approach when managing patients with rare disorders such as Hypokalemic Periodic Paralysis in an obstetric scenario.

KEYWORDS : Hypokalemic Periodic Paralysis, renal tubular acidosis, paraplegia, pregnancy.

CASE REPORT

Mrs. H, 35 year old female with married life of 12 years (consanguineous) with G3P2L2 with previous 2 LSCS, 1st childbirth 11 years back and 2nd childbirth 8 years before the present pregnancy. She was a known case of RTA Type 1 (on oral potassium supplements) and Hereditary Spherocytosis diagnosed 1st time prior to her 1st pregnancy, on treatment for both the clinical conditions. Her family history revealed RTA and Hereditary Spherocytosis in her younger sister. She had history of GDM in both of her previous pregnancies. Her 1st visit to the hospital was at 6 weeks 5 days. She was diagnosed with Overt Diabetes at 10 weeks in the current pregnancy and was advised for Insulin administration by the Endocrinologist. Dating scan showed a thin sub-chorionic hemorrhage which settled over the subsequent follow-up scans. NT scan was normal. First trimester quadruple test suggested low risk for pre-eclampsia. She received Iron and Calcium supplements in her 2nd trimester and was started on prophylactic Aspirin as she had a high risk pregnancy. She was on regular consultation with Hematologist and Endocrinologist for Hereditary Spherocytosis, RTA and DM. She was detected with moderate anemia at 15 weeks of gestation and was managed with oral Iron supplements as per Hematologist's advice. Anomaly scan detected no anomalies. At 31 weeks of gestation she was admitted to hospital with complaints of sudden difficulty in moving her upper and lower limbs since 1 day, with vomiting. Her history suggested non-compliance with potassium supplements despite advise after diagnosed with RTA. She was diagnosed with HPP with serum K+ of 1.8mmol/L on admission. She was aggressively managed in ICU for 1 day with Intravenous potassium (KCl) infusion along with supportive care. Bicarbonate (9.7mmol/L) correction was done with oral sodium bicarbonate supplements. Multidisciplinary approach was provided in her management through a team comprising of Obstetrics, Endocrinology and Intensive care specialists. She was symptomatically better after potassium correction and hence was shifted to ward for observation and discharged in a stable condition with advice of K+ supplements (KCl syrup- POTKLOR®). Serum K+ at discharge was 3.89 mmol/L. She was diagnosed with fetal growth restriction (FGR) at 31 weeks during her ICU admission with normal Doppler parameters. She was followed up with serial growth scans at two-weekly intervals which suggested fetal weight along the lower centile, however with normal Doppler studies. Serum K+ levels remained within normal limits for the rest of the antenatal period. She underwent Elective Caesarean Section with Tubectomy at 37 weeks and delivered a Female baby of 2.27 kg. Post-operatively, she received adequate IV fluids, analgesics, antibiotics, thrombo-prophylaxis and potassium supplements. Her potassium levels were monitored during her postoperative stay and were maintained within normal limits. Postoperative recovery was satisfactory. The patient was reviewed in the outpatient department at 1 week and 6 weeks postpartum, the potassium levels were normal and the postoperative recovery was satisfactory.

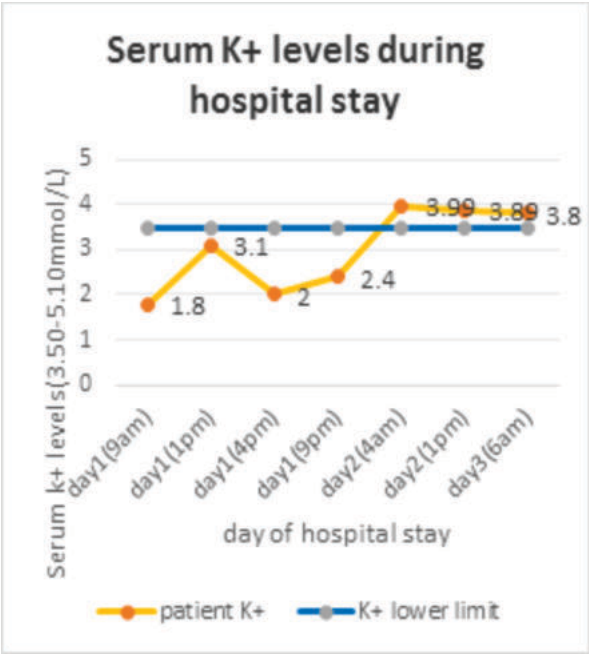

Chart 1- Serum K+ Levels During Hospital Stay

DISCUSSION

Hypokalemic Periodic Paralysis (HPP) is a rare neuromuscular disorder characterized by skeletal muscle ion channel mutations (calcium or sodium channels). It is characterized by episodic severe muscle weakness (generalized or focal flaccid paralysis) which is sudden in onset, associated with low blood serum potassium levels (K+ <3.5mmol/L), which can last for several hours before resolving spontaneously[3]. Our patient Mrs. H had serum K+ value of 1.8 mmol/L at the time of presentation with paraplagia. Generally, the flaccidity is more marked proximally than distally, with normal-to-decreased deep tendon reflexes. Our patient has complete flaccidity of all her four limbs with decreased deep tendon reflexes.

Etiology of HPP can be either Hereditary (familial) or Acquired. Periodic muscle weakness can also result from hypokalemia due to potassium loss secondary to renal and gastrointestinal issues such as RTA, gastroenteritis, or endocrine-related causes. The major trigger in our case was familial renal pathology, i.e. RTA. Endocrine factors such as thyrotoxicosis and Insulin-dependent diabetes can also trigger hypokalemia. Our patient, though diagnosed to have overt DM and was advised for Insulin administration, was highly non-compliant with her medications. Thyrotoxic periodic paralysis and Anderson-Tawill syndrome (Long QT syndrome type 7) were ruled out, by virtue of normal thyroid function tests and ECG findings respectively in our patient [1]. Additional triggers can include strenuous exercise, carbohydrate-rich meals, cold, stress, excitement, fear, salt intake, prolonged immobility, use of glucocorticoids, alcohol, and anaesthetic procedures[4]. A myopathy may occur independent of paralytic symptoms and may be the sole manifestation of HPP.

Treatment for hypokalemic periodic paralysis can vary depending on the intensity and duration of the paralytic attacks. Minor attacks may resolve spontaneously. Moderate attacks may be self-treated in a non-medical setting by oral potassium supplements. Severe attacks similar to our patient's will typically require more intensive medical management with intravenous potassium infusion with serial measurement of serum potassium levels(CHART 1). Possible respiratory involvement, and cardiac involvement (QT interval prolongation, U wave, mild ST depression) should be continuously monitored. There were no such vital organ involvements in our patient due to prompt and quick correction of hypokalemia. Cardiac involvement due to hypokalemia can result in arrhythmias such as torsades-de-pointes and ventricular tachycardia. HPP has no known curative treatment till present day; only prompt potassium correction may help to maintain muscle strength. Prevention of HPP can be achieved by avoidance of triggering factors, with diet rich in potassium, with oral potassium supplements.

CONCLUSION

Hypokalemic Periodic Paralysis in pregnancy is often apprehensive for the pregnant women and challenging for the treating clinicians. The heterogeneity of the causes makes it more difficult to diagnose and manage the underlying condition. The holistic treatment goals aim to identify and mitigate triggering factors, manage acute manifestations, prevent complications, and reduce the frequency of muscle weakness attacks with an aim of preventing complications in both mother and fetus. Trigger identification and dietary modification may be helpful along with medications to maintain the potassium levels within normal limits. This seemingly alarming disorder may be amenable to simple correction of electrolytes. If left undetected, it may lead to dangerous respiratory paralysis with patient ending up on a ventilator and fetal complications. With timely detection and prompt management, there is a scope for delivery without any maternal and fetal complications.

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