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Case Report

Management of hypokalemic periodic paralysis in pregnancy: Anaesthetic considerations for a 34-week gestation patient

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Abstract

Hypokalemic periodic paralysis (HypoPP) is a rare autosomal disorder characterized by episodic hypokalemia leading to acute muscle weakness, precipitated by various factors like physical exertion, infection, pregnancy, carbohydrate overload and thyroid abnormalities. We present a successful anaesthetic management of a parturient who underwent emergency cesarean section with a diagnosis of HypoPP complicated with premature rupture of membranes. Ensuring the health and safety of both the mother and fetus requires meticulous anaesthetic planning during delivery to optimize outcome as it is complicated due to the condition's impact on the motor system, cardiac function and potential respiratory involvement. Emergency cesarean section was planned as the patient was not progressing, remote risk of infection with increase in time, and for pain relief also as stress can surge catecholamine release worsening hypokalemia. Regional anaesthesia was preferred being a cesarean section eliminating the need for neuromuscular blockade which may further worsen her weakness.

Keywords: Anaesthetics, Cesarean section, Hypokalemic periodic paralysis, Muscle weakness, Sodium channels.

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1. Introduction

Hypokalemic periodic paralysis is a rare entity but poses a serious concern during the perioperative period due to the possibilities of developing respiratory failure,[1] cardiac arrhythmia,[2] residual neurological deficits,[3] etc. Even though the incidence of patients who develop HypoPP during pregnancy are not much reported, few available literatures show they may cause serious morbidity in maternal and fetal outcome. Early and optimal intervention of hypokalemia is mandatory for preventing any worsening of symptoms and for early recovery. Here we present a 24-year-old G2P1 patient presented with premature rupture of membrane (PROM) necessitating an emergency caesarean section owing to nonprogress of labour probably because of the muscular weakness. She was successfully optimized in the available time left before taking for cesarean section and spinal anaesthesia was instituted to deliver the baby timely without any maternal and fetal complications. Peri operatively the mother's hypokalemia was corrected with the potassium infusion based upon the requirement leading to complete recovery of motor power without any residual weakness.

2. Case Report

A 24-year-old G2P1 woman at 34 weeks of gestation was admitted with a three-day history of progressive lower limb pain that advanced to quadriparesis. On neurological examination, motor power was graded 1/5 in both upper and lower limbs (Medical Research Council scale). Laboratory investigations revealed severe hypokalemia, with a serum potassium (K^+) level of 1.7 mEq/L, and acute tubular acidosis. Arterial blood gas analysis showed pH 7.3, PCO_2 21.3 mmHg, and bicarbonate (HCO_3^-) 12.5 mmol/L.

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A multidisciplinary team involving nephrology, neurology, obstetrics, and anaesthesiology was engaged to manage the patient's complex condition. Intravenous correction of metabolic acidosis was initiated with 50 mEq of sodium bicarbonate diluted in 500 mL of Ringer's lactate every 8 hours. Potassium repletion commenced with 40 mEq of K^+ in 500 mL of 0.9% normal saline, administered at 20 mEq/hour. Subsequent doses were reduced to 10 mEq/hour. A total of 100 mEq of potassium was administered over 8 hours, with serum K^+ levels monitored every two hours to guide further management.

The following morning, the patient experienced premature rupture of membranes. In light of non-progressing labor, an emergency cesarean section was indicated. Preoperative reassessment showed partial neurological improvement, with upper limb motor power at 3/5 and lower limbs at 2/5. Flaccid paralysis persisted, impairing voluntary muscle activity, including abdominal contractions. Airway examination was unremarkable, and systemic findings were within normal limits.

Electrocardiogram (ECG) demonstrated sinus rhythm with flattened T waves (**Figure 1**). Echocardiography was normal. Thyroid function testing was conducted to evaluate possible thyrotoxic periodic paralysis, but results confirmed a euthyroid state. Preoperatively, serum potassium had improved to 2.8 mEq/L.

After thorough counseling regarding anesthetic options, the patient opted for spinal anaesthesia. Fetal well-being was continuously monitored via non-stress test and ultrasonography until transfer to the operating room. Due to the patient's flaccid paralysis, identification of the loss of resistance was challenging; however, spinal anaesthesia was successfully administered by a senior anaesthesiologist in the left lateral decubitus position. A dose of 2.4 mL of 0.5% hyperbaric bupivacaine was delivered intrathecally at the L3–L4 interspace using a 26G Quincke needle. A T6 sensory level was confirmed via loss of cold sensation.

The cesarean section proceeded uneventfully, resulting in the delivery of a healthy male neonate weighing 2.6 kg, with APGAR scores of 7 and 9 at one and five minutes, respectively. Intraoperative hemodynamics remained stable without vasopressor requirement.

Postoperatively, potassium supplementation continued with 40 mEq/day in 500 mL of 0.9% saline, guided by serial electrolyte monitoring (**Figure 2**). The patient's motor function showed progressive improvement: on postoperative day one, upper limbs were graded 4/5 and lower limbs 2/5. By the second postoperative day, motor strength had returned to 5/5 in the upper limbs and 4/5 in the lower limbs. She resumed independent mobility and was capable of performing routine activities, including newborn care, by day three.

The neonate, though premature, required only routine observation and was transferred to the mother's side after three days, following stabilization.

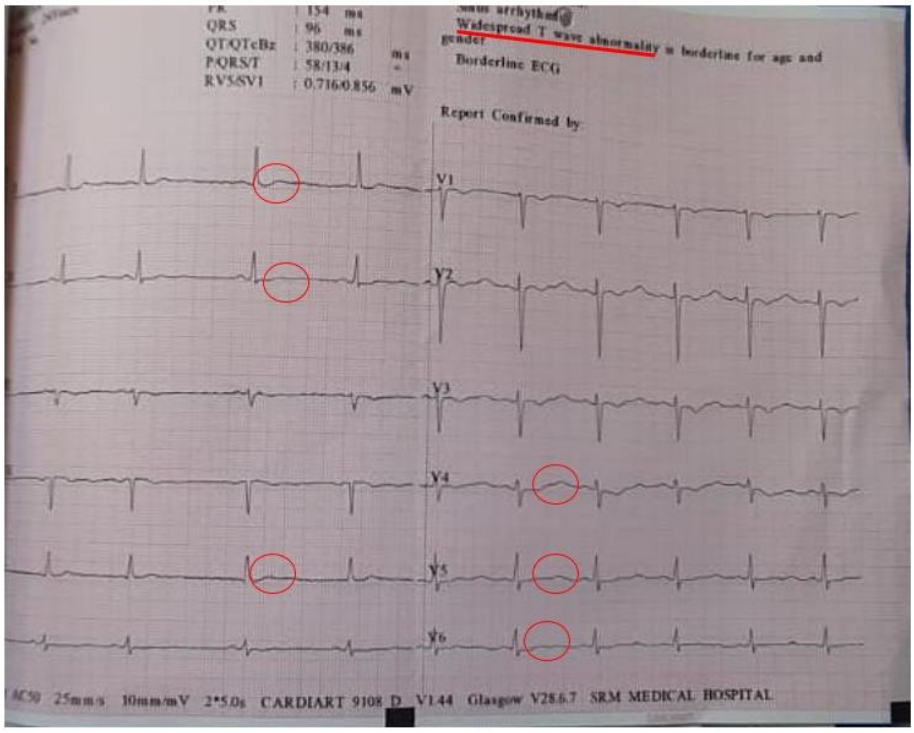

Figure 1: ECG showing sinus rhythm with flattened 'T' waves

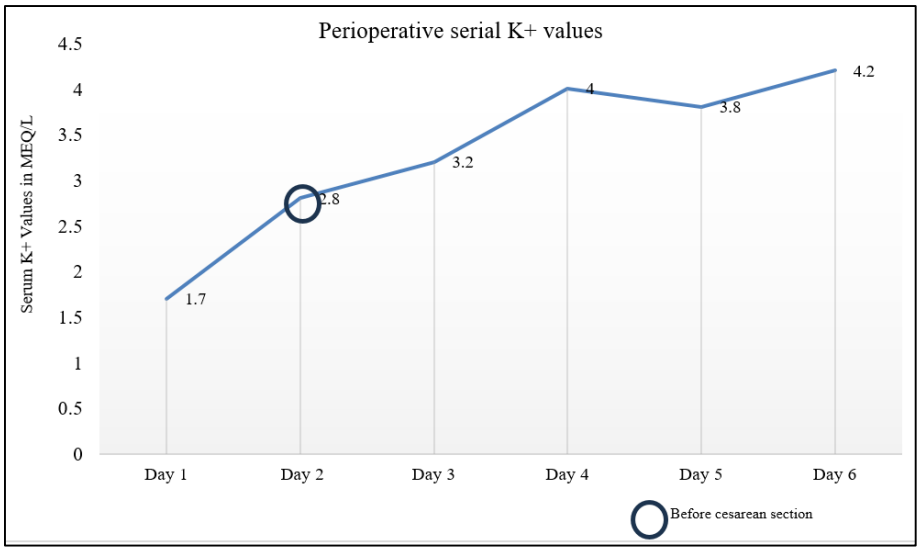

Figure 2: Perioperative serial K+ values

3. Discussion

In our patient the preoperative bicarbonate correction and K^+ supplements were continued until the morning of surgery. Additional measures included withholding glucose infusions and beta-adrenergic agonists, moderate levels of sedation and convection warming to prevent shivering. On the day of surgery patient had quadriparesis with lower limb power of 3/5 and preserved sensory function, so regional anaesthesia was preferred to avoid polypharmacy and complications associated with general anaesthesia as cited above.

The role of neuromuscular blocking agents in HypoPP are controversial. Postoperative muscle weakness has been described following the use of depolarizing muscle relaxants whereas the non-depolarizing muscle relaxant use is usually considered safe. Therefore, avoidance of neuromuscular blocking agents in HypoPP have been recommended.[4] Regional anaesthesia offers maternal awareness of the child birth and prevent fetal exposure to the anaesthetic and opioid drugs. The potential complications associated with general anaesthesia were mitigated by utilizing regional anaesthesia which offered a safer and more controlled approach.[5]

Perioperative alleviation of anxiety with good premedication, avoidance of stress and postoperative adequate analgesia is vital in preventing attacks.

This case report highlights the multidisciplinary approach to managing a pregnant patient with quadriparesis and hypokalemia in the perioperative period. Hypokalemia can rarely occur in pregnancy as a result of hemodilution.[6] The majority of HypoPP cases are hereditary or familial. The familial form is a rare channelopathy resulting from mutations in either the calcium or sodium ion channels, predominantly affecting skeletal muscle cells. Acquired cases can occur in hyperthyroidism. The disease-causing mutation in HypoPP, specifically in the CACNA1S gene, was identified by Jurkat-Rott et al in 1994.[7] In the absence of an identified genetic mutation in approximately 30% of patients, periodic paralysis subtypes can be distinguished on the basis of clinical presentation, serum potassium levels during attacks, and pattern of abnormalities on long exercise testing.

If primary periodic paralysis is suspected but cannot be confirmed by genetic testing, further examination should be undertaken to confirm that the symptoms are not secondary to other conditions such as thyrotoxicosis or secondary causes by gastric K^+ loss. Several symptoms or signs, or test results, can suggest an alternative diagnosis. The first attack most commonly occurs in the first two decades of life, and rarely after 30 years of age. Prominent sensory symptoms or pain or autonomic symptoms during the attacks may indicate Guillain-Barré syndrome or spinal cord injury. The absence of history of trauma as well as lack of symptoms such as fever, vomiting and diarrhea ruled out the possibility of the above-mentioned diagnoses.[8] The patient was euthyroid which excluded the possibility of thyrotoxicosis. HypoPP may present as either paralytic form which presents as acute onset limb paralysis or may progress as a myopathic form which leads to fixed weakness of limbs especially in lower limbs.

HypoPP was a diagnosis of exclusion in this patient as the she did not consent for further genetic and diagnostic evaluation. She demonstrated many of the clinical features of HypoPP, including proximal muscle weakness sparing the respiratory, facial, and pharyngeal musculature with severe hypokalemia responding to treatment.

4. Conclusion

This case highlights the importance of individualizing the anaesthetic approach in high-risk obstetric patients, particularly those with neuromuscular conditions such as hypokalemic periodic paralysis. Early and coordinated multidisciplinary involvement was essential in optimizing both maternal and fetal outcomes. The choice of anaesthetic

technique should be guided by the patient's neurological status, surgical requirements, and the anticipated need for postoperative care. Additionally, regular antenatal follow-up and the avoidance of known precipitating factors are critical in maintaining electrolyte balance and preventing acute decompensation in susceptible individuals.

5. Declaration of Patient Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images

6. Source of Funding

None.

7. Conflict of Interest

None.

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