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Case Report

Secondary hypokalemic non-periodic paralysis in a case of intractable hyperemesis gravidarum – A maternal near miss

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ABSTRACT

Background: Hyperemesis Gravidarum is a debilitating early pregnancy complication causing Hypokalemia and associated morbidity if timely recognition and intervention is not initiated. A rare sequelae of intractable hyperemesis is Non periodic Hypokalemic paralysis.
Case: A 26 years old primigravida at 13 weeks 6 days of gestation was admitted with hyperemesis gravidarum preceded by nausea and vomiting of 10 days duration. Her sonography showed a single live fetus of corresponding maturity. She was treated with intravenous fluid therapy and parenteral antiemetics. As her hyperemesis continued despite all medications, she was started on steroid therapy, she however developed hypokalaemic paralysis with serum potassium of < 2.3mmEq/L and ECG changes of prolonged QT interval with u waves. Timely detection and potassium supplementation prevented adverse maternal outcome and patient recovered totally within 48 hrs of potassium infusion initiation.
Conclusion: Hypokalemia remains a morbid sequelae of hyperemesis gravidarum which can be missed in the early stages as patient may remain asymptomatic. It thus becomes essential to serially monitor such patients with daily serum electrolytes especially when on corticosteroid therapy for intractable Hyperemesis to prevent any maternal near miss condition.

1. Introduction

Hyperemesis Gravidarum (HG) was first identified as "pernicious vomiting of pregnancy" by Antoine Dubois, who was a consultant and chief obstetrician to second wife of Napoleon Bonaparte, Empress Marie Louise, in 1852. It was C.S. Bacon in 1897 who described in detail this multifactorial disease and attempted to separate this medical condition from hysteria.[1]

HG is defined as severe, protracted nausea and vomiting associated with more than 5% of pre pregnancy weight loss, dehydration, electrolyte imbalance and ketonuria or ketonemia. The incidence varies from 0.3-3.6% in India and approximately 2% of all pregnancies are affected.[2] The chief causes of mortality are hypokalemia, thyrotoxicosis, Wernicke's encephalopathy, and severe dehydration.

Hypokalaemia remains the most life-threatening complication of hyperemesis gravidarum. It can cause sudden cardiac arrest subsequent to ventricular fibrillation. There can be precipitous hypokalemic paralysis which if recognized timely, can prevent maternal mortality.

2. Case

26 years old primigravida reported with complaints of unwarranted nausea, vomiting and cough of 10 days duration not responding to oral antiemetics at 13 weeks 6 days of gestation. Her admission pulse and BP were

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100/min and 110/60 mm Hg, with signs of mild dehydration. Clinically her uterus was just palpable (14wks size). Sonography showed a singleton live fetus consistent with period of amenorrhoea. Her spot urine ketones tested Positive (4 +). She was given intravenous fluids of 3L per day (normal saline and ringer's lactate) and antiemetics like Inj Ondansetron 4mg iv 8hrly and Inj Ranitidine 50mg iv 12hrly. She was already on oral doxylamine which was continued. Her admission blood workup showed normal electrolytes (Table 1).

She continued to remain symptomatic and refused oral feeds. Her intravenous fluids were increased to 4L per day and dose of antiemetics increased in a step ladder pattern. Her biochemical profile was repeated every day. However, she continued to have vomiting and ketonuria even on day 3 of admission. She also had a significant weight loss of 4.9% of pre pregnancy weight. In view of the above she was started on intravenous hydrocortisone 100mg twice daily which was continued for 5 days. Serum electrolytes were assessed before starting steroid and every day. By Day 3 of steroids, her vomiting had reduced to 2-3 episodes per day and oral intake improved minimally. However, ketonuria persisted for which high carbohydrate diet along with intravenous vitamin B supplementation was started. Intravenous steroids were stopped after 5 days (Day 8 of admission) and tapering dose with T. Prednisolone 10mg started. On day 9 there was no ketonuria and vomiting. But patient complained of generalized weakness and muscle pain. On day 10 patient developed weakness of lower limb and inability to get up from bed in the morning. She gave no history of headache, any trauma, neck pain, any previous similar episodes nor any family history of same. As per medical research council grading system, her upper limbs power was 4/5, lower limbs 3/5 and 0/5 at hips. Cranial nerve function along with bowel and bladder functions remained unaffected. There were absent deep tendon reflexes in lower limbs. Her potassium was 2.3 mEq/L and ECG showed QT prolongation and 'U waves. (Figure 1). With the suspicion of Hypokalaemic Paralysis she was transferred to ICU from ward HDU and put on continuous cardiac monitoring. Intravenous potassium correction therapy was started with 40 mEq of potassium chloride in 500 ml normal saline given slowly (over 4 hours) thrice a day along with adjuvant therapy for hyperemesis. Electrolyte monitoring was done 6 hourly. (Table 2) Her abdominopelvic ultrasonography showed gall bladder sludge but no suprarenal or any abdominal mass lesion. Foetal ultrasound showed a single live fetus with corresponding biometry at 15w3d. Her arterial blood gas analysis in ICU showed metabolic alkalosis with pH 7.6, bicarbonate (HCO_3^-) of 30mmol/L and P_aCO_2 of 48 mmHg.Detailed neurological examination was done to rule out other causes of paralysis with areflexia. Her thyroid function tests (S. TSH= 2.3 mIU/ml, fT4 = 1.2 ng/dl) were normal. Potassium correction was continued for 48 hrs. Patient was walking with support on day 12 and without support by day 13. She was discharged with dietary advice, oral doxylamine succinate and was scheduled to review with her anomaly scan at 18-20 wks in view of turbulent early second trimester events.

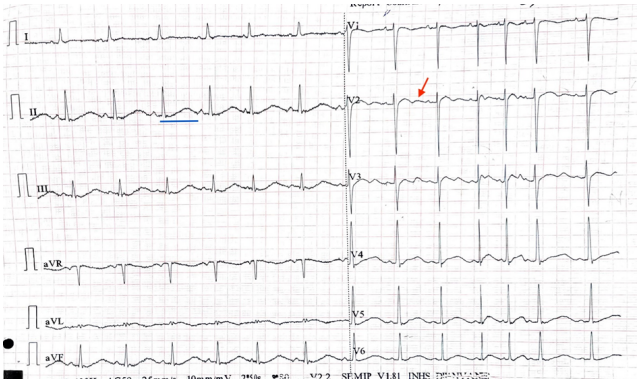

Fig. 1: ECG showing 'u' wave (red arrow) and QT prolongation (corrected QT 480 ms – blue line)

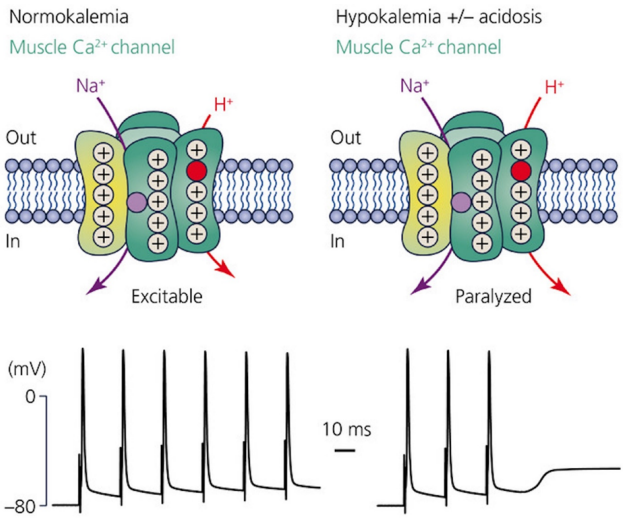

Fig. 2: Hypokalaemic non periodic paralysis showing slow activation rate of Ltype Calcium channels and reduced ATP dependent K channel current and abnormal depolarization causing paralysis

3. Discussion

Hypokalaemia is a known life-threatening complication of hyperemesis gravidarum; incorrect diagnosis and improper management can lead to maternal cardiac arrest. Our patient presented with complaints of protracted nausea and vomiting, and persistent ketonuria, which remained nonresponsive to standard step ladder pattern of antiemetic therapy. The patient lost almost 5% of her pre-pregnancy

Table 1: Investigations on admission D1

Hematological Parameters	**Hb g/dl**	**TLC /mm^3**	**DLC**	**PCV %**	**Plt /mm^3**	**TSH mIU/ml**
Value	12.6	16,600	$N_{87}L_6E_4M_3$	38.6	333000	2.3
Biochemical Parameters	**LFT**	**RFT**	**S. Na^+ mEq/L**	**S. K^+ mEq/L**	**RBS mg/dl**	**Urine Ketones**
Value	S.Bil 1.6 mg/dl SGOT 39 IU/ml SGPT 46 IU/ml	S.Urea 24mg/dl S.Creat 0.8mg/dl	142	3.8	110	4+

Table 2: Electrolytes during hypokalemic paralysis

Electrolytes mEq/L	**D 10**	**D11**	**D12**	**D13**	**D14**
K^+	2.3	2.4	2.5	4.8	4.7
Na^+	129	132	134	136	138

weight and was hence started on intravenous steroid therapy. Despite a tight watch on her electrolytes patient progressed to hypokalaemic paralysis which could have been precipitated by the steroid therapy.

In Hypokalemic periodic paralysis (hypoPP), the affected individuals may experience paralytic episodes subsequent to hypokalemia (serum potassium <3.5 mmol/L). These paralytic attacks are characterized by muscle flaccidity of proximal muscles more than the distal group with decreased to absent deep tendon reflexes. Though they develop acutely over minutes to hours they often show spontaneous recovery.[2] Hereditary or familial and acquired etiology of hypokalemic periodic paralysis have been acknowledged. Familial hypokalaemia periodic paralysis is caused by either calcium or sodium ion channel gene mutation. Secondary hypokalemia leading to periodic muscle weakness can also result from renal and gastrointestinal potassium losses as in hyperemesis gravidarum, renal tubular acidosis, gastroenteritis, Barttter or Gitelman syndrome, drugs like Amphotericin B or secondary to endocrine causes.

In hypokalemic paralysis, there is abnormal depolarization due to slow activation rate of L type Calcium channels and reduced ATP dependent K channel current. (Figure 2)

A systematic approach to diagnosis and treatment of any patient with hypokalemic paralysis should aim at identifying the etiology of hypokalemia and timely replacement of potassium. HypoPP, has been shown to be precipitated by betamethasone steroid injections during pregnancy.[3] This occurs due to glucocorticoids' influence on sodium-potassium adenosinetriphosphatase (Na^+-K^+ ATPase), resulting in a altered distribution of potassium across the membrane of skeletal muscle cells and producing an extracellular to intracellular shift. Glucocorticoids cause insulin resistance, causing hyperinsulinemia and hyperglycemia, leading to a larger shift of potassium into the intracellular space. Our case report associates the paralysis to hydrocortisone used for intractable HG.

In two cases reported by Hernandez Pacheco JA, hypokalemic paralysis in pregnancy was associated with distal tubular acidosis and Bartter's syndrome.[4] These conditions were ruled out in our patient as her urinary calcium levels were normal. In a systematic review of literature by Fezjo et al. it was found that hypokalaemia causing five out of six maternal deaths were secondary to HG. Consequently, patients with the triad of hypokalaemia, HG and steroid therapy epitomize a perilous subgroup that necessities vigilant monitoring and treatment until complete resolution of potassium and other electrolyte imbalances is achieved.

4. Conclusion

Hypokalaemia leads to severe maternal morbidity and women continue to be asymptomatic even with dangerously low potassium levels. This case highlights the importance of strict electrolyte monitoring in patients with HG and on steroid therapy. Our patient was showing signs of progressive hypokalaemia despite repeated normal lab results, till she developed paralysis and metabolic alkalosis. Timely intervention by potassium supplementation corrected the hypokalaemia and the patient recovered from this near miss event. Cautious use of steroid therapy in intractable hyperemesis should be done weighing in the risks associated with precipitating hypokalaemia.

5. Source of Funding

None.

6. Conflict of Interest

None.

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