



## HYPOKALEMIC PARALYSIS DUE TO HYPEREMESIS GRAVIDARUM: A MATERNAL NEAR MISS

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**ABSTRACT** **Background:** Hypokalemia is one of the complications of hyperemesis gravidarum which can lead to severe morbidity and mortality if unrecognized. Hypokalemic paralysis is rare and this case is reported as it occurred in a woman with prolonged duration of vomiting with less frequency.

**Case:** A 28 years old primigravida at 15 weeks of gestation was admitted with hyperemesis gravidarum of 3 months duration through OPD. On admission her pulse rate was 150 bpm and BP was 140/80 mm Hg and her urine ketone were positive. Ultrasound revealed single fetus corresponding to the period of gestation. She was managed with intravenous fluids and injectable antiemetics and her haematological and biochemical profile was done on emergency basis. Her serum potassium was <2.5 mEq/L and the reports were not accessed immediately and acted up on. After 12 hours of admission she suddenly developed neck rigidity and weakness of lower limbs which further deteriorated and she was unable to stand up and walk. She was shifted to ICU and repeat serum potassium was 2.3 mEq/L and ECG showed T wave inversion. She was diagnosed as hypokalemic paralysis. Hypokalemia correction was done by 20 mEq of potassium in 500 ml normal saline slowly over 4 hours and her potassium was normalized to 3.7 mEq/L in 24 hours time. She recovered completely after 24 hours and was able to walk.

**Conclusion:** Hypokalemia results in severe morbidity and women are asymptomatic until the levels are dangerously low. It can even result in maternal mortality (as reported in literature) if unrecognized and not corrected timely and hence it is a maternal near miss.

**KEYWORDS :** Hyperemesis gravidarum, Hypokalaemic paralysis , maternal near miss

### INTRODUCTION

Hyperemesis gravidarum (HG) refers to intractable vomiting during pregnancy that leads to weight loss and volume depletion, resulting ketonuria and or ketonemia<sup>1</sup>. As per Cochrane database 2015, it occurs in approximately 2% of all pregnancies<sup>2</sup>. Incidence of hyperemesis gravidarum in India is 0.3-3%<sup>3</sup>. Hyperemesis gravidarum can lead to morbid conditions like liver dysfunction, renal failure, Boerhaave syndrome, hypokalemic paralysis, Wernicke 's encephalopathy<sup>4</sup>. Although maternal death is rare, there are still reported cases of deaths from complications of hyperemesis gravidarum. The causes of mortality due to hyperemesis gravidarum recently reported in literature are hypokalaemia , Wernickes encephalopathy, thyrotoxicosis and severe dehydration<sup>5,6</sup>.

Hypokalemia is a life threatening complication of hyperemesis gravidarum. Hypokalemia occurs in most of the patients of HG.<sup>7</sup> Hypokalaemia leads to Ventricular fibrillation and causes sudden cardiac arrest It can be a maternal near miss if not recognized and treated on time. It can lead to sudden onset paralysis called hypokalemic paralysis and timely recognition and prompt management can save the life of the pregnant woman<sup>8</sup>. The present case is reported to highlight that prolonged vomiting though less severe can result in hypokalemia and paralysis and it can be a maternal nearmiss if the reports are not checked timely and acted up on.

### CASE

A 28 years old primigravida presented at 15 weeks 3 days period of gestation to out-patient department with complaint of excessive vomiting for a period of 3 months duration . On admission her pulse was 150/min and BP was 140/80 mm HG and there was no dehydration. Abdominal examination revealed uterus of 14 weeks size. Ultrasound showed singleton fetus corresponding to the period of amenorrhoea. She was admitted as she had 4 episodes of vomiting during the waiting period in OPD. Urine ketones were Positive. She was given intravenous fluids and supportive treatment. Her haematological and biochemical profile sent at admission was shown in Table 1. All were normal except Potassium which was < 2.5 meq/L and the reports were not accessed immediately and acted up on.

The next day (after 12 hours of admission) she suddenly developed weakness of lower limbs and neck rigidity which deteriorated further and she was not able to walk after 12 hours. There was no history of trauma, headache, neck pain, family history or previous history of similar such episodes. Power in upper limbs was normal, whereas in lower limbs it was grade 3 (medical research council grading). Deep tendon reflexes were intact and cranial nerve function was normal. Repeat Biochemical investigations showed a potassium of 2.3 mEq/L,

while other investigations were unremarkable. Hypokalaemic Paralysis was suspected and she was shifted to Obstetric ICU and monitored by connecting to multi- parameter monitor and ECG was done which showed T wave inversion. She was started on IV potassium correction 20 mEq in 500 ml Normal saline slowly over 4 hours three times a day along with other supportive measures for hyperemesis.(Table 2) Electrolytes monitoring was done 4<sup>th</sup> hourly.

**Table 1: Baseline investigations**

Parameter	Value
Haemoglobin	11.9 g/dl
TLC	9,790 mm3
Platelet count	2.43 L
Urea/Creatinine	9/0.30
Na/K/Cl	137/<2.5/103meq/L
AST/ALT	85/53
ALP	54
Bilirubin(T/D)	1.19/0.39

**Table 2: Serum potassium levels**

Time	K+ (mEq/L)
Admission	<2.5
Next day (0 h) Paralysis	2.3
4h	2.04
8h	2.5
12h	2.7
16h	3.0
20h	3.5
24h	3.7

Neurologist opinion was sought for further evaluation to rule out other causes of paralysis.. She was advised to continue potassium correction and nerve conduction studies were done to rule out underlying neuropathy, which were found to be normal. Patient significantly improved next day and recovered completely in two days and was able to walk. She was discharged on doxylamine , nutritional advice and was scheduled regular follow up in the antenatal clinic.

### DISCUSSION

Hypokalemia is one of the complications of hyperemesis gravidarum and failure to diagnose and correct it timely can even lead to fatal consequence and hence it is a maternal near miss. In this case the patient was a primigravida at 15 weeks of period of gestation who presented with complaint of vomiting of less frequency but for a prolonged time of 3 months duration and developed hypokalemic paralysis.

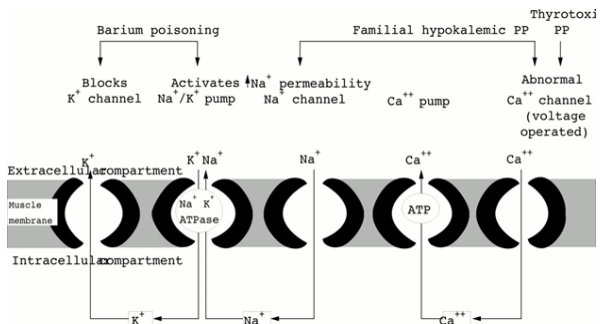
The etiology of hypokalemic paralysis varies and most common cause is hereditary and other causes are hyper aldosteronism, distal renal tubular acidosis, Bartter or Gitelman syndrome, excessive vomiting, drugs like diuretics, Amphotericin B<sup>9</sup>. So the approach to any patient with hypokalemic paralysis should be to search for the cause of hypokalemia and timely replacement of potassium.

There are two cases reported where hypokalemic paraplegia occurred secondary to betamethasone injections in pregnancy and these were not due to hyperemesis<sup>10,11</sup>. Hernandez Pacheco JA reported two cases where patients presented with hypokalemic paralysis in pregnancy and cause for hypokalemia was found to be distal tubular acidosis and Bartter's syndrome respectively<sup>12</sup>. In our patient, urinary calcium levels were normal, so these conditions were ruled out. There is another case report where the hypokalemia was caffeine induced causing paralysis in a pregnant woman<sup>13</sup>. Thyroid function test should be done in all patients with hypokalemic periodic paralysis to differentiate it from thyrotoxic periodic paralysis.

Fejzo and colleagues reviewed the literature and found that five out of 6 maternal deaths presented with hypokalemia were secondary to hyperemesis gravidarum. Two of them did not receive prompt hypokalemia treatment and were discharged who later came with cardio-respiratory arrest. Hypokalemia was completely missed in another woman, while the fourth patient developed central pontine myelinolysis and died of multiorgan failure. The sixth woman presented with hypokalemia and subsequently developed seizures leading to death<sup>14</sup>.

There are two other reported cases where hyperemesis induced hypokalemia that led to cardiac arrest and death<sup>14,15</sup>. Hypokalemia can cause ventricular fibrillation and sudden cardiac death<sup>16</sup>. In hypokalemic paralysis, there is slow activation rate of L type Calcium channels and reduced ATP dependent K channel current and abnormal depolarization.

This figure explains the slow activation of calcium channels in hypokalemic paralysis as compared to thyrotoxic paralysis in which there is abnormal opening of calcium channels.



Hyperemesis poses increased risk of prematurity, low birth weight and SGA babies<sup>7</sup> and hence subsequent antenatal care should be like that of high risk pregnancy.

## CONCLUSION

Hypokalemia results in severe morbidity during pregnancy and women are asymptomatic until the levels are dangerously low. Hence it is mandatory to screen all women with nausea and vomiting of pregnancy and HG for electrolyte imbalance and give timely correction.

## RECOMMENDATION

Those women who are at risk of developing hyperemesis include women of teenage, low body mass index, multiple pregnancy, molar pregnancy and a prior history of HG should be identified during the first antenatal visit and nutritional advice should be given to prevent hyperemesis. Screening can be undertaken by objective method using PUQE (Pregnancy Unique Quantification of Emesis) score<sup>17</sup>

Adequate intake of potassium for pregnant women is 2900 mg/day. So women with nausea and vomiting in pregnancy should be counseled to have more of potassium rich foods like soybeans, potatoes, milk, yoghurt, fish, meat and nuts. Those who are on thiazide diuretics and laxatives should be emphasized more about the need to take potassium rich foods<sup>18</sup>.

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