

## Hyperemesis Induced Acute Quadriparesis in Pregnancy with Missed Miscarriage: A Case Report

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### Abstract

Reports on quadriparesis in pregnancy secondary to gastrointestinal losses are rare. We describe a previously healthy 22-year-old primigravida at approximately 20 weeks gestation who presented to us with weakness in all the four limbs for 3 days and vomiting off and on in the last 6 weeks gestation. This report is unusual but important as it is a rapidly treatable or reversible paresis. Herein this report.

**Keywords:** Hypokalemic; Hyperemesis Gravidarum; Pregnancy; Quadriparesis; Reversible

### 1. Introduction

Hyperemesis gravidarum (HG) is persistent and excessive vomiting starting before the end of the 22nd week of gestation, leading to carbohydrate depletion, dehydration and electrolyte imbalance as defined by the International Statistical Classification of Disease and Related Health Problems, 10th Revision.<sup>1</sup> It occurs in 0.3-10.8% of pregnancies.<sup>2</sup> Until the 1950's, maternal deaths were commonly caused by hyperemesis gravidarum.<sup>3</sup> Morbidity and mortality associated with unrecognized disease include respiratory failure, cardiac arrest, Wernicke's Encephalopathy and Korsakoff's Psychosis, pulmonary embolism and death<sup>4-9</sup> from malnutrition, dehydration and electrolyte.

Hypokalaemia from hyperemesis gravidarum can lead to acute hypokalaemic paralysis which is an uncommon cause of acute weakness. Hence, it is imperative for physicians to be knowledgeable about the causes of hypokalemic paralysis, and consider them diagnostically. The hypokalaemic paralyzes represent a heterogeneous group of disorders with a final common pathway presenting as acute weakness and hypokalaemia. Most cases are due to familial hypokalaemic paralysis,<sup>10</sup> however, sporadic cases are associated with diverse underlying etiologies including gastrointestinal potassium losses as is the case in our patient, thyrotoxic periodic paralysis, barium poisoning, renal tubular acidosis, primary hyperaldosteronism and licorice ingestion. The approach to the patient with hypokalemic paralysis includes a vigorous search for the underlying etiology and potassium replacement therapy. Further therapy depends on the

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etiology of the hypokalemia. Disposition depends on severity of symptoms, degree of hypokalemia, and chronicity of disease.<sup>10</sup>

## 2. Case Report

We describe a previously healthy 22-year-old primigravida whose first day of her Last Menstrual Period (LMP) was 27 July 2023 and presented at 20 weeks + 1 day gestation with weakness in all the four limbs 3 days prior to her presentation and vomiting which was off and on for 6 weeks gestation. She had no difficulty in respiration or swallowing or weakness of face and neck muscles. There was no history of trauma, fever, headache, recent diarrhea, neck-pain, lower urinary tract symptoms and previous such episodes prior to onset of weakness. Her parents and siblings had no history of similar episode or any other significant illness. There was no history of the use of corticosteroid prior to presentation. On admission she was afebrile with a temperature of 36.50C, dehydrated with a pulse rate of 100 beats/minute and a blood pressure of 100/70 mmHg. She was conscious and oriented to time, place and person however power in all the four limbs were grade 2 by MRC (Medical Research Council) scale with diminished deep tendon reflexes in both upper and lower limbs and normal plantar reflexes. Cranial nerve functions were intact and so was the sensory function. Guillain-Barre syndrome was ruled out. Examination of respiratory system and cardiovascular system was unremarkable.

Abdominal examination revealed uterus of 18–20-week size. The abdominopelvic Ultrasonography performed showed missed miscarriage and she had medical evacuation without complications. Computed tomography (CT) scan of the brain as well as Magnetic Resonance imaging (MRI) of the spine were all essentially normal. Her total white blood cell count was normal as well as the platelets.

She was administered intravenous fluids and given supportive management keeping in view vomiting and dehydration. The biochemical profile report at the time of admission revealed a very low serum potassium levels of 1.0 mmol/L (as shown in Figure 1). Thyroid profile was normal. Intravenous potassium was replaced which led to remission of symptoms. Her power improved and she was discharged after 3 weeks of hospitalization with potassium supplements and physiotherapy and was advised frequent follow ups for maternal wellbeing and preconception care. The serum potassium level at discharge was 3.9mmol/L (Figure 2).

Examination Reqd.		Nationality		Religion		Doctor's signature	
Age	22y	Sex	F	Tribe	1960	Lab. Ref. No	
TEST & UNITS		CON. LIMITS	RESULT	TEST & UNITS		CON. LIMITS	RESULT
<input type="checkbox"/> GLUCOSE (F)	mmol/L	3.6-5.6	4.5	<input type="checkbox"/> TOTAL PROTEIN	g/L	62-80	
<input type="checkbox"/> GLUCOSE *	mmol/L	3.6-6.7		<input type="checkbox"/> ALBUMIN	g/L	30-50	
<input type="checkbox"/> GLUCOSE (PP)	mmol/L			<input type="checkbox"/> GLOBULIN	g/L	18-30	
<input type="checkbox"/> BILIRUBIN TOTAL	umol/L	8.0-17.0		<input type="checkbox"/> CSF PROTEIN	g/L	0.15-0.4	
<input type="checkbox"/> BILIRUBIN DIRECT	umol/L	Less than 8		<input type="checkbox"/> CSF SUGAR	mmol/L	2.5-5.0	
<input type="checkbox"/> ALKALINE PHOSPHATASE	Iu/L	25-92		<input type="checkbox"/> CAERULOPLASMIN	mg/L	300-600	
<input type="checkbox"/> ALANINE TRANSAMINASES	Iu/L	3-40		<input type="checkbox"/> IRON	mmol/L	13-32	
<input type="checkbox"/> ASPARTATE TRANSAMINASES	Iu/L	5-40		<input type="checkbox"/> TIBC	mmol/L	45-70	
<input type="checkbox"/> SODIUM	mmol/L	135-145	129	<input type="checkbox"/> MAGNESIUM	mmol/L	0.72-1.0	
<input type="checkbox"/> POTASSIUM	mmol/L	3.5-5.0	1.0	<input type="checkbox"/> UPASE	Iu/L		
<input type="checkbox"/> BICARBONATE	mmol/L	21-31	17.0	<input type="checkbox"/> ALDOLASE	Iu/L		
<input type="checkbox"/> CHLORIDE	mmol/L	97-108		<input type="checkbox"/> ALPHA FETOPROTEIN			
<input type="checkbox"/> URIC ACID	mmol/L	2.5-8.5	18	<input type="checkbox"/> G6PD			
<input type="checkbox"/> CREATININE	umol/L	44.2-198.5	246.4	<input type="checkbox"/> PREGNANCY TEST			
<input type="checkbox"/> CPK (MALE)	Iu/L	5-55		<input type="checkbox"/> PSA	ng/ml	<4.0	
<input type="checkbox"/> CPK (FEMALE)	Iu/L	5-35		<input type="checkbox"/> HbA1C	%	<7.0	
<input type="checkbox"/> CPK (MB)	Iu/L	Less than 8		<input type="checkbox"/> FSH	mIU/mL		
<input type="checkbox"/> TOTAL CHOLESTEROL	mmol/L	3.6-6.2		<input type="checkbox"/> LH	mIU/mL		
<input type="checkbox"/> HDL	mmol/L	0.8-1.7		<input type="checkbox"/> PROGESTERONE	ng/ml		
<input type="checkbox"/> LDL	mmol/L	1.9-3.6		<input type="checkbox"/> PROLACTIN	ng/ml		
<input type="checkbox"/> VLDL	mmol/L	0.4-1.3		<input type="checkbox"/> TSH		0.4-4.2	
<input type="checkbox"/> TRIGLYCERIDE	mmol/L	0.51-2.2		<input type="checkbox"/> T3	ng/ml	0.8-2.0	
<input type="checkbox"/> ACID PHOSPHATASE (V)	Iu/L	0.9-9.1		<input type="checkbox"/> T4	ng/dl	4.5-11.6	
<input type="checkbox"/> ACID PHC. PROSTATIC	Iu/L	Less than 10		<input type="checkbox"/> OTHERS			
<input type="checkbox"/> AMYLASE	Iu/L	130-370					
<input type="checkbox"/> CALCIUM	mmol/L	2.2-2.8					
<input type="checkbox"/> INORGANIC PHOSPHORUS	mmol/L	1.3-1.6					
<input type="checkbox"/> URIC ACID	mmol/L	0.08-0.4					

Figure 1 Results of serum electrolyte and creatinine at admission

Examination Reqd. <i>Seher</i>			Consultant <i>D. Gupta</i>		
Age <i>34</i>	Sex <i>F</i>	Tribe <i>Kho</i>	Nationality	Religion	Doctor's signature <i>[Signature]</i>
LABORATORY REPORT (For Lab Use Only)			Lab. Ref. No.		
TEST & UNITS	CON. LIMITS	RESULT	TEST & UNITS	CON. LIMITS	RESULT
<input type="checkbox"/> GLUCOSE (F)	mmol/L 3.6 - 5.6		<input type="checkbox"/> TOTAL PROTEIN	g/L 62-80	
<input type="checkbox"/> GLUCOSE @	mmol/L 3.6 - 6.7		<input type="checkbox"/> ALBUMIN	g/L 30-50	
<input type="checkbox"/> GLUCOSE (PP)	mmol/L		<input type="checkbox"/> GLOBULIN	g/L 18-30	
<input type="checkbox"/> BILIRUBIN TOTAL	umol/L 8.0 - 17.0		<input type="checkbox"/> CSF PROTEIN	g/L 0.15-0.4	
<input type="checkbox"/> BILIRUBIN DIRECT	umol/L Less than 8		<input type="checkbox"/> CSF SUGAR	mmol/L 2.5-5.0	
<input type="checkbox"/> ALKALINE PHOSPHATASE	Iu/L 25-92		<input type="checkbox"/> CAERULOPLASMIN	mg/L 300-600	
<input type="checkbox"/> ALKALINE TRANSAMINASES	Iu/L 3-40		<input type="checkbox"/> IRON	mmol/L 13-32	
<input type="checkbox"/> ASPARATE TRANSAMINASES	Iu/L 5-40		<input type="checkbox"/> TIBC	mmol/L 45-70	
<input checked="" type="checkbox"/> SODIUM	mmol/L 135-145	<i>136</i>	<input type="checkbox"/> MAGNESIUM	mmol/L 0.72-1.0	
<input type="checkbox"/> POTASSIUM	mmol/L 3.5-5.0	<i>3.9</i>	<input type="checkbox"/> UPAASE	Iu/L	
<input type="checkbox"/> BICARBONATE	mmol/L 21-31	<i>21.4</i>	<input type="checkbox"/> ALDOLASE	Iu/L	
<input type="checkbox"/> CHLORIDE	mmol/L 97-108	<i>99</i>	<input type="checkbox"/> ALPHA FETOPROTEIN		
<input checked="" type="checkbox"/> UREA	mmol/L 2.5-8.5	<i>3.6</i>	<input type="checkbox"/> G6PD		
<input checked="" type="checkbox"/> CREATININE	umol/L 44.2-194.5	<i>1.88</i>	<input type="checkbox"/> PREGNANCY TEST		
<input type="checkbox"/> CPK (MALE)	Iu/L 5-55		<input type="checkbox"/> PSA	ng/ml <4.0	
<input type="checkbox"/> CPK (FEMALE)	Iu/L 5-35		<input type="checkbox"/> HbA1c	% <7.0	
<input type="checkbox"/> CPK (MB)	Iu/L Less than 8		<input type="checkbox"/> FSH	mIU/mL	
<input type="checkbox"/> TOTAL CHOLESTEROL	mmol/L 3.6-6.2		<input type="checkbox"/> LH	mIU/mL	
<input type="checkbox"/> HDL	mmol/L 0.8-1.7		<input type="checkbox"/> PROGESTERONE	ng/ml	
<input type="checkbox"/> LDL	mmol/L 1.9-3.6		<input type="checkbox"/> PROLACTIN	ng/ml	
<input type="checkbox"/> VLDL	mmol/L 0.4-1.3		<input type="checkbox"/> TSH	0.4-4.2	
<input type="checkbox"/> TRIGLYCERIDE	mmol/L 0.51-2.2		<input type="checkbox"/> T <sub>1</sub>	ng/ml 0.8-2.0	
<input type="checkbox"/> ACID PHOSPHATASE (T)	Iu/L 0.9-91		<input type="checkbox"/> T <sub>2</sub>	ng/mL 4.5-11.6	
<input type="checkbox"/> ACID PHOS. PROSTATIC	Iu/L Less than 10		<input type="checkbox"/> OTHERS		
<input type="checkbox"/> AMYLASE	Iu/L 130-320				
<input type="checkbox"/> CALCIUM	mmol/L 2.2-2.8				
<input type="checkbox"/> INORGANIC PHOSPHORUS	mmol/L 1.1-1.6				
<input type="checkbox"/> URIC ACID	mmol/L 0.08-0.4				

*[Signature]*  
MEDICAL LABORATORY SCIENTIST

*30/06/23*  
DATE

Figure 2 serum electrolyte with normal potassium level at discharge

### 3. Discussion

Hyperemesis gravidarum is a severe clinical disease in pregnancy where patients have intractable nausea and vomiting with increased morbidity and even mortality.<sup>10</sup> Incorrect diagnosis and improper management can lead to maternal cardiac arrest and maternal death.<sup>6,9</sup> In hyperemesis gravidarum, rarely, patients present with severe vitamin deficiency, causing a neurological emergency called Wernicke's encephalopathy.<sup>7</sup> Though this was not seen in our patient. Our patient presented with complaints of protracted nausea and vomiting, dehydration, electrolyte abnormalities, and a weight loss of over 5% of her pre-pregnancy weight with weakness involving the four limbs and missed miscarriage. The occurrence of electrolyte abnormalities, hypokalaemia in particular in this condition is the cause of flaccid paralysis. A similar hypokalaemia with missed miscarriage was reported by Suwanwongse and his colleague in Lincoln Medical Center, New York City, USA,<sup>11</sup> however, the presenting potassium level was 2.8 mmol/L which was higher than the value of 1.0 mmol/L seen in our patient. The intractable vomiting was for a duration of 2 days<sup>11</sup> which was shorter compared to a duration of over 6 weeks experienced in our patient. A case of hypokalaemic flaccid paralysis with type 2 respiratory failure due to hyperemesis gravidarum with a potassium level of 1.8 mmol/L was reported by Naik and his study group in India.<sup>5</sup> The thyroid profile tests showed hyperthyroidism and would have accounted for the manifestation of type II respiratory failure which was absent in our patient who had normal thyroid function profile. Another case of secondary hypokalaemic non-periodic paralysis following the use of corticosteroid due to intractable nausea and vomiting of pregnancy was reported by Khan and his colleagues.<sup>12</sup> This was not the case in our patient as she did not use corticosteroid before she developed quadriparesis. Gupta and his colleague equally reported hyperemesis induced acute quadriparesis in a 22-year-old primigravida at approximately 20 weeks gestation who presented with weakness in all the four limbs of 3 days duration and vomiting off and on for 6 weeks. The biochemical profile at the time of admission revealed a very low potassium levels of 1.42 mEq/L. Thyroid profile was normal. She had intravenous potassium replacement which led to remission of symptoms and pregnancy continued uneventfully. Her power improved and she was discharged with potassium supplements.<sup>13</sup> This case scenario was almost similar to what obtained in our patient who was also a 22 year old primigravida, developed quadriparesis at about same gestational age of 20 weeks + 1 day and with similar duration and pattern of nausea and vomiting however there was no miscarriage probably due to a bit higher level of serum potassium compared to a very low level of serum potassium obtained in our patient.

#### 4. Conclusion

Hypokalemia in Hyperemesis gravidarum is a life-threatening condition and delay in the diagnosis may lead to flaccid paralysis. Strict and regular electrolyte monitoring and early correction of potassium will avert the associated mortality. Timely intervention by potassium supplementation corrected the hypokalaemia and the patient recovered from this near miss event.

#### Compliance with ethical standards

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##### *Statement of Ethical approval*

This case report is exempt from ethical approval in our institute

##### *Disclosure of conflict of interest*

There was no conflict of interest to declare.

##### *Statement of informed consent*

Informed consent was obtained from the patient whose case was reported.

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