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HYPEREMESIS GRAVIDARUM WITH MULTI-ORGAN DYSFUNCTION

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ABSTRACT Severe hyperemesis gravidarum, though rare, poses potentially fatal risks in pregnancy due to complications such as dehydration, electrolyte imbalances, malnutrition, Wernicke's encephalopathy, and renal dysfunction. A primigravida who had multiple admissions for hyperemesis gravidarum in early pregnancy, presented to the emergency department at 13 weeks with complaints of excessive fatigue and lethargy of one-week duration. Evaluation of the patient resulted in a diagnosis of hyperemesis gravidarum with multisystem involvement. She responded to supportive therapy of intravenous fluids and multiple doses of thiamine. This case report describes the typical clinical presentation of hyperemesis gravidarum with multiorgan dysfunction.

KEYWORDS : Hyperemesis Gravidarum, complications, dehydration, electrolyte imbalance, Wernicke's encephalopathy, multisystem involvement, thiamine

INTRODUCTION

Nausea and vomiting are common in early pregnancy, affecting 70–85% of women. Hyperemesis gravidarum, characterized by severe nausea, vomiting leading to ≥5% weight loss, is less frequent (0.5–1%) and can lead to complications like acute renal failure, liver dysfunction, and neurological issues such as Wernicke's encephalopathy.

Renal complications: Acute kidney injury or acute renal failure is characterized by a sudden decline in kidney function occurring within seven days of injury onset. In pregnant women, it can arise due to severe hyperemesis, presenting with oliguria and azotemia despite no prior history of kidney disease.

Liver dysfunction: Women experiencing hyperemesis gravidarum may develop liver dysfunction typically within 1-3 weeks of severe vomiting onset. This condition is marked by moderate increases in aminotransferases and occasional hyperbilirubinemia, which rarely exceeds 3-4 mg/dl. Severe and prolonged vomiting correlates with higher bilirubin levels.

Neurological Complications: Central and extra pontine myelinolysis (Osmotic Demyelination Syndrome, ODS) manifests with classic signs such as confusion, loss of consciousness, horizontal gaze paralysis, and spastic quadriplegia. Delirium is also common. ODS results from the destruction of the myelin sheath covering nerve cells in the brainstem (pons), often triggered by electrolyte imbalances, particularly rapid shifts in sodium or potassium levels in women with hyperemesis gravidarum (HG). It frequently co-occurs with Wernicke's Encephalopathy (WE) and other complications linked to malnutrition and electrolyte disturbances. Close monitoring, nutritional support, and systematic electrolyte replacement are crucial to mitigate maternal and fetal morbidity and mortality in HG cases. Deficiencies in vitamins, particularly thiamine (B1), play a significant role, exacerbated when glucose-containing fluids are administered before correcting these deficiencies. Lab results for hormones, electrolytes, and nutrients can vary, necessitating a comprehensive clinical assessment rather than relying solely on laboratory findings. Standard multivitamin infusions typically contain 6 mg of thiamine, insufficient for HG management; therefore, HG patients often require a minimum of 100 mg of thiamine daily in their intravenous fluids.

CASE REPORT

A primigravida at 13 weeks of gestation who had multiple admissions for hyperemesis gravidarum in early pregnancy presented in emergency with complaints of excessive fatigue and lethargy for 1 week, vomiting - multiple episodes till 2 days back and decreased intake of food and water since past 1 week. During this period, the patient was unable to tolerate solids or liquids. She denied any fever, chills, rash or sore throat. Her medical history was unremarkable, and there was no history of drug abuse, nonsteroidal anti-inflammatory drug use, recent antibiotic use, psychiatric illness, or eating disorder. There was no history of hypertension or kidney disease. Physical examination revealed a cachectic, lethargic, sick-looking, dehydrated woman weighing 37 kg with multiple oral ulcers. She had mild pallor and icterus, with a pulse rate of 110 beats per minute, blood pressure of 90/60 mm Hg, and temperature of 37.4-degree C. Her initial blood reports showed deranged RFT, LFT, and electrolytes. peripheral blood smear showed mild normocytic anemia and relative neutrophilia.

The patient was admitted to the ICU with Riles tube aspiration fluids and I/O charting and maintained good urine output. The electrolyte imbalance was corrected. Tropical infections like dengue, leptospirosis, and scrub typhus were evaluated as the cause of jaundice and were tested negative. Viral serology was also tested negative. Blood cultures were negative initially, but the third sample showed staphylococcus aureus. Procalcitonin was 3.77 and was initiated on antibiotics according to specific sensitivity. She was given thiamine injections of 100 mg daily and symptoms improved with supportive care and management.

MRI BRAIN showed no neuroparenchyma abnormality. Complement C3, and C4 were normal and ANA was negative

Renal ultrasound revealed normal-sized kidneys with parenchymal echogenicity and no evidence of hydronephrosis, obstruction, or calculi. An NT scan was taken 16 weeks prior to her discharge and revealed a viable 16-week gestation with normal NT and nasal bone was visualized.

She was discharged on day 14. She came for regular antenatal checkups. Her blood investigations were within normal limits. She was counseled regarding appropriate diet and calorie intake. Iron and calcium supplements were given. Weight gain was adequate. USG at 36 weeks showed a SLIUG, cephalic~34w+2 days, AFI-11cm, EFW-2.24kg, less than 10th centile, placenta -fund posterior, along left lateral, upper segreant, grade 2, doppler -normal.

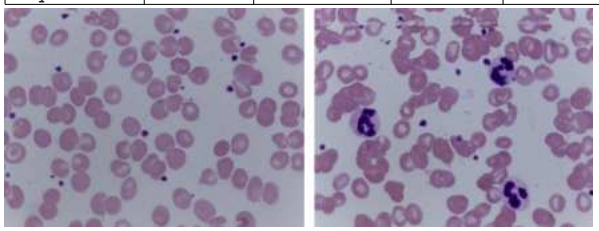
Her third-trimester investigations were within normal limits. At

38 weeks of gestation, she went into normal labor but underwent Emergency LSCS (Indication:CPD) and delivered a term female baby of weight 2.580 kg. There were no intrapartum or postpartum complications.

At 8 months of age, the baby has achieved regular milestones.

Table 1: Blood Investigation

Day	S. Urea (mg/dl)	S. creatinine (mg/dl)	S sodium	S potassium (mmol/l)	S calcium (mg/dl)	S Mg (mg/dl)	T. Bilirubin (mg/dl)	SGOT (IU/L)	SGPT (IU/L)
Day1	278	4.5	22	2.0	6.0	2.3	6.7	24	110
Day 3	169	2.8			7.8				
Day 5	79	1.8	23	3.1		2.1	3.0	43	75
Day 7	50								
Day10	13	1.3			8.2			32	75
Day12	8	1.2	24	3.8	8.7				
Day13						1.1	1.5	25	68



DISCUSSION

Hyperemesis gravidarum represents a serious complication during pregnancy that poses potential risks to the mother's life. Persistent vomiting can lead to dehydration and subsequent renal failure. Our patient, without a history of renal disease and with normal-sized kidneys, suggests her renal failure was due to an acute process. It's critical to differentiate between the three types of acute renal failure—prerenal, intrinsic, and postrenal—as intrinsic causes carry higher morbidity and mortality rates. Prerenal failure, resulting from mild to moderate renal hypoperfusion, is the most common form, progressing to ischemic injury and intrinsic renal failure with severe hypoperfusion. The patient's sodium level of 115 mmol/L and potassium level of 2.0 mmol/L indicate an intrinsic renal cause, supported by renal scan findings.

Several hypotheses exist regarding the etiology of hyperemesis gravidarum, including psychosocial factors such as adjustment difficulties and sexual function disturbances. Human Chorionic Gonadotropin (high) is a leading cause, acting on the TSH receptor to elevate free T4 and suppress TSH levels.

Hyperemesis gravidarum not only threatens maternal life but also raises concerns about adverse pregnancy outcomes, including low birth weight, preterm delivery, and fetal malformations. Studies have highlighted increased risks of central nervous system and skeletal malformations in the offspring of affected women, particularly those with significant weight loss. Early malnutrition has profound effects on brain development, as demonstrated by research showing reduced brain cell numbers and sizes in malnourished pregnant women.

Though rarely fatal, hyperemesis gravidarum can lead to significant maternal morbidity due to nutritional deficiencies, electrolyte imbalances, Wernicke's encephalopathy, and renal failure. In this case, acute renal failure was likely due to severe intravascular volume depletion from hyperemesis gravidarum. Following eight days of adequate fluid resuscitation and electrolyte correction, renal and liver functions normalized, resulting in favorable maternal and perinatal outcomes.

CONCLUSION

Hyperemesis Gravidarum is a reversible condition and can prevent its complications if diagnosed and treated early. She should be closely monitored and electrolyte imbalances should be corrected at the earliest. We would like to emphasize the importance of thiamine supplementation in these women with prolonged vomiting even before starting parenteral nutrition

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