


Hyperemesis Gravidarum-induced Encephalopathy, Kidney Injury, and Fetal Death – A Case Report

Siti Zulaikha Risqiyani[1,2], Fikhy Rizky Hapsari[1,3]
[1]Faculty of Medicine, Jember University, Jember, East Java, Indonesia,
[2]RSUD Moh Saleh Probolinggo, East Java, Indonesia,
[3]Obstetric and Gynecology Department, RSUD dr. Soebandi Jember, East Java, Indonesia

ABSTRACT

Introduction: Hyperemesis gravidarum (HG) is a severe form of nausea and vomiting during pregnancy that can become life-threatening for the mother and fetus if not treated properly. Although most cases resolve with conservative management, rare instances progress to severe multisystem complications, including encephalopathy, acute kidney injury, and fetal demise. **Case:** A 22-year-old G1P0A0 woman at 13 weeks of gestation presented with decreased consciousness following four weeks of intractable vomiting since 8 weeks of pregnancy. The patient has been vomiting more than five times a day, and consciousness began to decrease over the last two days, with complaints of blurry vision and tremors. Physical examination revealed hypotension (blood pressure 80/45mmHg), tachycardia, and cold extremities. Laboratory findings demonstrated acute kidney injury (serum creatinine 8.1 mg/dL), severe electrolyte disturbances (sodium 115.7 mmol/L, potassium 2.07 mmol/L, chloride 77.8 mmol/L), and hepatocellular injury. Transabdominal ultrasound confirmed fetal demise at 13 weeks of gestation. **Discussion:** HG can precipitate life-threatening complications, including Wernicke's encephalopathy, acute kidney injury, and fetal loss through mechanisms of severe dehydration, malnutrition, and electrolyte imbalance. Early recognition with aggressive fluid resuscitation, electrolyte correction, and nutritional support is essential to prevent irreversible multisystem deterioration and improve maternal-fetal outcomes. **Conclusion:** Multisystem complications in HG are extremely dangerous for both mother and fetus. Appropriate preventive measures and early intervention must be taken to prevent worsening outcomes and reduce the risk of maternal and fetal mortality.

Keywords: Case report, encephalopathy, fetal demise, hyperemesis, kidney injury.

ABSTRAK

Pendahuluan: Hiperemesis gravidarum (HG) adalah bentuk mual dan muntah yang parah selama kehamilan yang dapat mengancam jiwa ibu dan janin jika tidak diobati dengan benar. Meskipun sebagian besar kasus sembuh dengan penanganan konservatif, beberapa kasus jarang berkembang menjadi komplikasi multisistem yang parah termasuk ensefalopati, cedera ginjal akut, dan kematian janin. **Kasus:** Seorang wanita berusia 22 tahun, G1P0A0, pada usia kehamilan 13 minggu, datang dengan penurunan kesadaran setelah empat minggu muntah yang tak kunjung berhenti sejak usia kehamilan 8 minggu. Pasien muntah lebih dari lima kali sehari, kesadarannya mulai menurun dalam dua hari terakhir, mengeluhkan penglihatan kabur dan tremor. Pemeriksaan fisik menunjukkan hipotensi (tekanan darah 80/45 mmHg), takikardia, dan ekstremitas dingin. Temuan laboratorium menunjukkan cedera ginjal akut (kreatinin serum 8,1 mg/dL), gangguan elektrolit berat (natrium 115,7 mmol/L, kalium 2,07 mmol/L, klorida 77,8 mmol/L), dan cedera hepatoseluler. Ultrasonografi transabdominal mengonfirmasi kematian janin pada usia kehamilan 13 minggu. **Pembahasan:** HG dapat memicu komplikasi yang mengancam jiwa termasuk ensefalopati Wernicke, cedera ginjal akut, dan kehilangan janin melalui mekanisme dehidrasi berat, malnutrisi, dan ketidakseimbangan elektrolit. Pengenalan dini dengan resusitasi cairan agresif, koreksi elektrolit, dan dukungan nutrisi sangat penting untuk mencegah kerusakan multisistem yang ireversibel dan meningkatkan hasil ibu dan janin. **Simpulan:** Komplikasi multisistem pada HG sangat berbahaya bagi ibu dan janin. Tindakan pencegahan yang tepat dan intervensi dini harus dilakukan untuk mencegah memburuknya hasil dan mengurangi risiko kematian ibu dan janin. **Siti Zulaikha Risqiyani, Fikhy Rizky Hapsari. *Hyperemesis Gravidarum-induced Encephalopathy*, Gagal Ginjal, dan Kematian Janin – Laporan Kasus.**

Kata Kunci: Laporan kasus, ensefalopati, kematian janin, hiperemesis, cedera ginjal.

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Alamat Korespondensi *siti.zulaikhar99@gmail.com*


INTRODUCTION

Nausea and vomiting often occur during pregnancy and affect about 80% of pregnancies, being considered early signs of pregnancy.[1,2] Although 80% of pregnant women experience nausea or vomiting during pregnancy, only 0.3%–2% of pregnant women are diagnosed with hyperemesis gravidarum (HG).[1] Uncontrollable vomiting during pregnancy is a condition of pregnancy known as hyperemesis gravidarum (HG).[3–5] Weight loss (> 5% of pre-pregnancy weight), ketonuria, dehydration, electrolyte imbalance, acid-base imbalance, and arrhythmia are the most frequent causes of hospitalization among women with HG. With potentially harmful effects on the mother and the fetus, HG can manifest in different degrees of severity.[6,7] Sometimes, HG is linked to potentially fatal consequences in other illnesses. Although severe HG is uncommon, if treatment is not received, it can be fatal.[6]

Complications that can occur include Wernicke's encephalopathy due to thiamine deficiency, esophageal rupture (Mallory Weiss syndrome), splenic avulsion, pneumothorax, transient gestational thyrotoxicosis, and acute kidney injury (AKI).[8–10] We encountered a rather rare case in our area, a 22-year-old woman who came to the emergency unit with severe hyperemesis gravidarum presented decreased consciousness, acute kidney injury, and fetal demise at 13 weeks of gestation.

CASE

A 22-year-old G1P0A0 female at 13 weeks of gestation came to the emergency room with decreased consciousness, preceded by nausea and vomiting more than five times a day for 4 weeks since her 8th week of pregnancy, and worsened over the past 2 days; accompanied by blirred vision and tremors. She lost her appetite over the previous month and did not eat over the past two days. The patient lost 5 kg during the last month. Urine production has drastically decreased to nearly zero in the past 24 hours. History of kidney disease, diabetes, or high blood pressure were unknown. The patient only had one antenatal care (ANC) visit at 5 weeks of pregnancy.

At physical examination, the patient weighed 62 kg, was 158 cm tall, and was disoriented and agitated. All extremities are cold. Blood pressure 80/45 mmHg, persistent tachycardia up to 120 beats per minute, pulse rate of 117 beats per minute, respiratory rate 22 breaths per minute, and a body temperature of 36.9 °C. No fluxus nor blood from genitalia.

The laboratory values show serum urea 70 mg/dL (normal reference 6 to 20 mg/dL) and serum creatinine 8.1 mg/dL (0.5 to 1.1 mg/dL), indicating acute kidney injury. Electrolyte imbalance also detected with sodium 115.7 mmol/L (135 to 155 mmol/L), potassium 2.07 mmol/L (3.5 to 5 mmol/L), and chloride 77.8 mmol/L (90 to 110 mmol/L). Liver function was impaired, marked by SGOT 134 U/L (10 to 31 U/L), SGPT 496 U/L (9 to 36 U/L), albumin 2.9 g/dL (3.4 to 4.8 g/dL), total bilirubin 1.56 mg/dL < 1.2 mg/dL), direct bilirubin 1.01 mg/dL (0.2 to 0.4 mg/dL). Tests for hepatitis B, syphilis, and human immunodeficiency virus were negative. Urine analysis showed 25 to 50 red blood cells, 10 to 25 white blood cells, proteinuria +1, ketones +2, positive bacteria, and positive yeast. Blood gas analysis result: pH 7.56 (7.35 to 7.45), pCO2 26.4 mmHg (35 to 45 mmHg), HCO3 24 mmol/L (22 to 26 mmol/L).

On transabdominal ultrasound, a 13-week intrauterine single fetus is visible with no movement, with the placenta was still present. Liver, spleen, and kidneys are normal. The conclusion was intrauterine fetal death. The diagnosis was G1P0A0 at 12 weeks of gestation with hyperemesis gravidarum accompanied by decreased consciousness suspected Wernicke's encephalopathy, acute kidney injury, and fetal death. According to the Caine criteria, Wernicke's encephalopathy (WE) should be suspected in any patient presenting with at least two of the following: nutritional deficiency, changes in mental status or confusion, oculomotor disturbances, or cerebellar dysfunction.[10–13]

The patient was admitted to the intensive care unit. The therapy included 3% NaCl 1000 mL/24 hours, 25 mEq KCl in 0.9% NaCl 1,500 mL/24 hours, meropenem 500 mg IV every 8 hours, Stronger Neo-Minophagen C containing monoammonium glycyrrhizinate, amino acid acetate, and L-cysteine HCl 20 mg IV every 8 hours for 3 days, ondansetron 8 mg IV every 8 hours, omeprazole 40 mg IV every 12 hours, and oral amino acids 40 mg and channa striata extract 500 mg each every 12 hours. Termination of non-viable fetus was performed on the second day of hospitalization using 600 mcg misoprostol per rectum, followed by digital exploration of the placenta and curettage. The patient was discharged on the 7th day, in good condition with clear consciousness.

DISCUSSION

Hyperemesis gravidarum (HG) is a severe form of nausea and vomiting during pregnancy, a condition that can lead to significant maternal malnutrition and weight loss. Nausea and vomiting affect up to 80% of all pregnancies,[2,6] and persistent vomiting is the most common indication for hospitalization during the first half of pregnancy. The PUQE-24 (The Pregnancy-Unique Quantification of Emesis and Nausea) score[14] is 15, means severe or hyperemesis gravidarum. HELP (HyperEmesis Level Prediction) score was 48, indicating severe clinical condition.[15] Symptoms worsen in up to 3% pregnancies resulting in weight loss, dehydration, and electrolyte imbalances.[10] HG can be associated with life-threatening complications, including Wernicke's encephalopathy (WE) and acute kidney injury (AKI).[11]

WE can be caused by nutrient deficiencies and hyperemesis gravidarum (HG).[12,13] WE can be diagnosed with a history, physical examination, and objective findings. A normal MRI or CT scan of the head does not rule out the condition.[13] It was not performed in this case due to limited facilities. WE is a clinical diagnosis, identification depends on clinical of the classic triad of ophthalmoplegia, confusion, and ataxia.[10,16–18] However, many patients only show one symptom of the triad.[19,20] HG is a significant risk factor for WE due to malnutrition caused by continuous vomiting,[21] which is associated with increased fetal mortality.[22] This case is suggestive of WE. Other differential diagnosis is metabolic disorders like hepatic encephalopathy due to liver dysfunction (SGOT 134 U/L, SGPT 496 U/L).

The neurological manifestations observed in this case, including decreased consciousness, disorientation, blurred vision, and tremors, were highly suggestive of Wernicke's


encephalopathy (WE).[12,13] Prolonged vomiting for approximately four weeks likely resulted in severe nutritional deficiency and depletion of thiamine stores. Thiamine is an essential cofactor in cerebral glucose metabolism, and its deficiency impairs neuronal energy production, leading to selective neuronal injury.[12] Although MRI and serum thiamine measurements were unavailable, the patient fulfilled the Caine criteria through the presence of nutritional deficiency and altered mental status.[14] Similar cases of HG-associated WE have been reported in the literature, particularly in patients with delayed presentation and prolonged vomiting.

The patient presented with severe dehydration and electrolyte imbalances, likely resulting from hyperemesis gravidarum (HG) during early pregnancy. HG is a common cause of pregnancy-related acute kidney injury (PRAKI) during the first trimester. PRAKI is defined as acute kidney injury (AKI) resulting from complications of pregnancy and is a significant cause of maternal and fetal morbidity and mortality. A decrease or a complete lack of urine output is a key indicator of AKI, often combined with increased serum creatinine levels. The KDIGO (Kidney Disease: Improving Global Outcomes) clinical practice guideline for AKI defines AKI as an increase in serum creatinine by 0.3 mg/dL within 48 hours.[23] The patient had no prior history of kidney disease. In this case, the most likely cause of AKI was dehydration and fluid deprivation, indicated by serum creatinine of 8.1 mg/dL and oliguria (urine output < 400 mL/day). Dehydration leads to reduced renal perfusion, impairing the kidneys' ability to filter metabolic waste. AKI can be managed conservatively with fluid replacement and electrolyte correction or, if necessary, with hemodialysis (HD).

In this patient, persistent vomiting, poor oral intake, and a 5-kg weight loss resulted in severe intravascular volume depletion. The presence of hypotension (80/45 mmHg), oliguria, severe hyponatremia, and elevated creatinine strongly suggests a prerenal mechanism of AKI caused by renal hypoperfusion.[19] If untreated, prolonged hypoperfusion may progress to intrinsic renal injury. The improvement in renal function following fluid resuscitation without hemodialysis suggests that the kidney injury was predominantly reversible and prerenal.

Despite her significantly elevated creatinine levels, this patient responded well to intravenous rehydration and electrolyte supplementation, and hemodialysis was not performed. This clinical picture is consistent with a report by Taber and Shah,[24] that HG-induced AKI improved with fluid therapy alone. However, other cases reported by Shim, *et al*.,[8] and Fernanda, *et al*.,[25] required hemodialysis due to failed fluid therapy. Early and intensive hemodialysis, as shown in various studies, may improve both maternal and fetal outcomes in severe cases.[26–29]

Unfortunately, ultrasound at admission already revealed a non-viable fetus. The fetal demise was likely associated with prolonged maternal dehydration, as hydration is crucial for fetal oxygenation, nutrient transport, waste removal, and overall homeostasis during pregnancy.[30] Excessive maternal dehydration increases the risk of fetal hypoxia and acidosis, contributing to fetal death.[31] Fetuses in PRAKI cases have a 3.4 times higher risk of stillbirth and perinatal death compared to pregnancies without PRAKI.[32] Maternal hypotension, often accompanying severe dehydration, can worsen fetal outcomes by impairing placental perfusion and disrupting maternal-fetal gas exchange.[33,34] This condition has also been associated with apoptosis in fetal brain cells.

In addition to renal involvement, the patient showed neurological signs of decreased consciousness, disorientation, blurred vision, and tremors, consistent with Wernicke's Encephalopathy (WE) and peripheral neuropathy.[17] Before confirming diagnosis of WE, it is essential to rule out other causes of similar neuropsychiatric symptoms, such as hepatic encephalopathy.[35] WE is typically treated with high-dose thiamine injections, often in combination with other B vitamins.[36] However, due to limited therapeutic resources and facilities, this case was managed with supportive therapy, focusing on fluid and electrolyte balance and nutritional supplementation, including multivitamins. The patient's clinical condition improved following rehydration and electrolyte correction, which somewhat complicated the clinical confirmation of WE, as symptom improvement can occur from general supportive measures even without targeted thiamine therapy.

Although the patient's condition improved, the connection between thiamine deficiency and fetal death in this case remains unclear. It is possible that systemic complications like sepsis, potentially resulting from or worsened by thiamine deficiency, could have accelerated fetal demise.[37]

CONCLUSION

Hyperemesis gravidarum can cause multisystem problems severely affecting the health of both the mother and the fetus. Delays in the treatment of hyperemesis gravidarum can lead to encephalopathy, kidney injury, and even fetal demise.

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