Case Report

Hyperemesis Gravidarum as a Cause of Pregnancy-related Acute Kidney Injury: A Case Report from Ile-Ife, South West Nigeria

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Abstract

Hyperemesis gravidarum (HG) complicated by acute kidney injury (AKI) is not commonly encountered in practice. Despite the extreme rarity of HG as a cause of AKI, health-care providers should be aware of the likelihood of such a complication. The aim of the study was to report a case of HG complicated by AKI. A 35-year-old G2P1^{+0} (one alive) female presented with progressively worsening nausea and vomiting with associated weight loss and reduced urinary output at the gestational age of 17 weeks. Serum creatinine and urea values at presentation were 883 μmol/L and 56 mmol/L, respectively. She, however, responded to intravenous fluid therapy/replacement with subsequent normalization of serum urea and creatinine values and restoration of renal function. AKI can be a complication of HG. Individualized care of affected women will improve the prognosis.

Keywords: Acute kidney injury, hyperemesis gravidarum, fluid therapy

Introduction

Nausea and vomiting occur commonly in pregnancy affecting about 80% of pregnancies and are considered presumptuous signs of pregnancy.[1] Various theories have been propounded to explain the link between vomiting and pregnancy. These theories include hormonal, immunological, infectious, and psychosocial.[1,2]

In some cases, however, vomiting in pregnancy can be severe and may occur in association with dehydration, electrolyte/metabolic derangement and weight loss. When this occurs, the clinical entity is termed hyperemesis gravidarum (HG).[3] HG is said to affect approximately 0.3%–3% of pregnancies and occurs more in the first trimester, though there have been cases of the condition persisting till term.[3,4] It is a condition that occurs with varying degrees of severity, with possible deleterious implications to the mother and fetus.

HG may sometimes be associated with life-threatening complications. Complications that could result from HG include Wernicke's encephalopathy from thiamine deficiency, esophageal tears (Mallory–Weiss syndrome) due to retching, splenic avulsion, pneumothorax, gestational transient thyrotoxicosis, and acute kidney injury (AKI).[5]

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AKI is said to occur when there is a sudden loss of kidney function with associated elevated levels of serum creatinine; when it occurs due to complications arising from pregnancy, it is called pregnancy-related acute kidney injury (PRAKI).[6]

PRAKI is a rare complication of HG with very few cases reported in literature. We present a case of PRAKI caused by HG, which, to the best of our knowledge and from a detailed literature search, is the first reported case in Nigeria.

Case Report

A 35-year-old unbooked gravida 2 para 1^{+0} (one alive) female presented at the accident and emergency department at the estimated gestational age of 17 weeks with a history

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of persistent vomiting. Vomiting started at about 8 weeks of pregnancy. However, about 4 weeks prior to presentation, it had progressively worsened with associated weight loss. There was an associated decrease in urinary output over the past week, and she was also noticed to be talking irrationally about 5 days prior to presentation.

She was not a known hypertensive, diabetic, or chronic kidney disease patient; she was not a known psychiatric patient. Her previous pregnancy was uneventful with no similar illness.

General physical examination revealed a woman who was conscious but restless, pale, anicteric, dehydrated, afebrile, and without pedal edema.

She weighed 75 kg. Her pulse rate was 114 beats per minute, blood pressure was 90/60 mmHg, respiratory rate was 24 cycles per minute, and her temperature was 37.2° C.

Her serum electrolytes at presentation were deranged with sodium - 129 mmol/L (normal range, 135–150 mmol/L), potassium - 2.7 mmol/L (normal range, 3–5 mmol/L), creatinine - 883 µmol/l (normal range, 50–132 µmol/L), urea - 56 mmol/L (normal range, 2.5–5.8 mmol/L), and bicarbonate - 28 mmol/L (normal range, 20–30 mmol/L). Her complete blood count revealed a packed cell volume of 30%, white blood cell count of 12,400 cells/mm^3, and platelets of 216,000 cells/mm^3; clotting profile was normal. She was also negative for HIV I and II and hepatitis B surface antigen. Urinalysis was positive for ketones, but negative for protein and leukocyte esterase. An abdominopelvic ultrasound scan revealed a nonviable fetus at 17 weeks of gestation with no obvious fetal anomaly; the kidneys, liver, and spleen had a normal sonographic appearance. A diagnosis of HG complicated by AKI with fetal demise was made.

Nephrologists were invited to co-manage the patient. A urethral catheter was inserted to monitor the urine output, and she was commenced on intravenous fluid therapy with antiemetics (intravenous metoclopramide 10 mg 12 h for 72 h). She was placed on 1 L of 0.9% normal saline over 30 min initially and then 1 L of normal saline to alternate with 1 L Ringer's lactate 6 h. She initially had oliguria (producing only 70 ml of urine over the first 6 h); however, with the continuation of intravenous fluid, there was an improvement in the urine output. The missed abortion was managed with prostaglandin 5 days into admission due to a delay in the patient giving her consent and she subsequently expelled an abortus that weighed 350 gm. Serial measurements of serum electrolytes on the 3rd, 5th, 8th, 14th and 18th day into admission respectively revealed a progressive reduction in urea and creatinine levels as depicted in Table 1. Her clinical condition improved remarkably and she was subsequently discharged in stable condition.

Table 1: Serial measurements of urea and creatinine

Day	Creatinine (µmol/L*)	Urea (mmol/L**)
1	883	56.0
3	453	65.5
5	185	43.2
8	123	9.0
14	88	3.2
18	56	2.0

*µmol/L: Micromoles per liter, **mmol/L: Millimoles per liter

Discussion

PRAKI is a rare complication of HG and may occur due to hypovolemia from excessive vomiting and associated poor intake with resultant hypoperfusion of the kidneys (also known as prerenal AKI).

The management of AKI from hypovolemia could either be conservative with the replacement of fluid, which would lead to the restoration of intravascular volume with adequate renal perfusion or hemodialysis.[6] The patient presented, responded well to appropriate fluid therapy with electrolyte supplementation and did not need hemodialysis. This is similar to a case of HG with AKI reported by Chatwani and Schwartz,[7] who also responded to fluid therapy. However, two other cases reported by Hill *et al*.[8] and Shim *et al*.[9] needed hemodialysis for the restoration of renal function. This was due to the failure of fluid therapy.

Pregnancies complicated by HG have also been shown to be associated with certain pregnancy outcomes such as low birth weight, preterm delivery, and, in some rare cases, termination of pregnancy if all therapies fail.[3,7] There has, however, been no association between HG and congenital anomalies.[10] A plausible explanation for the adverse fetal outcome in the case presented may be as a result of fetal exposure to uremic toxins.

Although AKI is a rare complication of HG, caregivers should preempt it and take necessary measures to prevent its occurrence. When it does occur, management should be tailored to the patient's individual needs.

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Conflicts of interest

There are no conflicts of interest.

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