When Hyperemesis Gravida Becomes Life-Threatening: Severe Hyperemesis Gravidarum Leading to Diabetic Ketoacidosis. A High-Risk Obstetric Emergency with Significant Foetal Consequences.

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Background

While often perceived as an exaggerated form of morning sickness, hyperemesis gravidarum (HG) can have consequences extending far beyond maternal discomfort. Prolonged disease may result in the following:

Dehydration Electrolyte disturbances

Malnutrition Wernicke's Encephalopathy

and, rarely, life-threatening metabolic complications. In women with type 1 diabetes mellitus (T1DM), starvation secondary to severe HG may precipitate diabetic ketoacidosis (DKA), an obstetric emergency associated with significant maternal morbidity and foetal mortality. Along with this there can be complications for the foetus including intrauterine growth restriction, low birth weight and pre-term birth.

Aim

To highlight diabetic ketoacidosis as a rare but devastating complication of prolonged hyperemesis gravidarum and reinforce the importance of early recognition to optimise maternal and foetal outcomes.

Case: Presentation

A 28-year-old G4P1 woman at 29+2 weeks' gestation presented with severe nausea, vomiting and inability to tolerate oral intake for over 24 hours on a background of persistent HG requiring Hospital-in-the-Home support and prolonged corticosteroid therapy. Her pregnancy was complicated by pre-existing T1DM and previous pre-eclampsia. Her diabetes was treated with a hybrid closed loop insulin pump. Despite intravenous hydration, antiemetics and monitoring, her starvation ketosis progressed and she required admission for intravenous insulin infusion and electrolyte replacement. During admission, a MET call was triggered following seizure-like activity with headache, visual disturbance, hyperreflexia and clonus, prompting treatment for possible eclampsia with magnesium sulphate. Foetal assessment demonstrated minimal movements and a pathological CTG with foetal tachycardia and absent variability.

Initial workup Results

BSL: 15.6 Ketones: 2.9	Hb:120 Platelets: 227 WCC: 18.7	Na: 135, K:3.8, Mg: 0.56, pH: 1.02, eGFR: >90, Cr: 56 ECG – Sinus tachycardia 127
ALP: 145, GGT: 10, ALT: 24., AST: 24, Bilirubin: 18 Conjugated : 6	pH: 7.21, Bicarb: 12, BE: 014.7, Lactate: 7.4	CTG – FHR baseline 165-170, variability Bedside USS: initial minimal foetal movements

Case: Progress

Multidisciplinary management in intensive care improved maternal metabolic status. CTG abnormalities reflecting acute foetal compromise resolved following maternal resuscitation, with restoration of baseline variability and reactivity. Emergency delivery was avoided at that time. This patient had 3 admissions in total during pregnancy for a similar presentation and the baby was delivered by emergency caesarean section at 35 weeks gestation.

Discussion

This case highlights DKA as a rare but catastrophic complication of severe HG, associated with foetal loss rates. Starvation reduces circulating insulin and increases counter-regulatory hormones, promoting lipolysis and hepatic ketone production; in pregnancy, this accelerated ketosis can rapidly precipitate DKA, particularly in women with type 1 diabetes. Early recognition and coordinated multidisciplinary management are essential to optimise maternal and foetal outcomes.

DKA is associated with foetal loss rates reported up to 30–50%.