

An Unexpected Case of Postnatal Diabetic Ketoacidosis: First Presentation of Autoimmune Pancreatitis

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INTRODUCTION

Gestational diabetes mellitus (GDM) is defined as the first onset of glucose intolerance in pregnancy without prior known diabetes. While it is commonly associated with metabolic risk factors like obesity and hypertension, a small percentage of women with GDM have underlying autoimmune causes, with presence of pancreatic autoantibodies resulting in autoimmune-mediated destruction of the pancreas. **We present a case of idiopathic postpartum pancreatitis precipitating fulminant diabetic ketoacidosis (DKA) in a patient with otherwise well-controlled GDM during pregnancy**, and subsequent findings of pancreatic antibody (anti-GAD). This is the first presentation of autoimmune diabetes diagnosed postnatally in a woman who has no previous significant past medical or family history.

CASE REPORT

A 33-year-old healthy primigravida booked early with a singleton pregnancy. Body mass index (BMI) was 19.2kg/m². GDM was diagnosed at 25 weeks using the IADPSG criteria with a three-point oral glucose tolerance test (OGTT). She maintained normal hypocount readings on diet control. Oral Metformin was started at 36 weeks for suboptimal control but she was non-compliant.

A healthy baby boy weighing 2935g was delivered at 39 weeks via an emergency caesarean section. She developed acute onset abdominal pain on POD 3 associated with fever and tachycardia. She was distressed despite absence of an acute abdomen. Stat capillary blood glucose (CBG) was unrecordable. There was raised total white cell count of 37 x 10⁹/L, C-reactive protein 86mg/L, serum lactate of 2.5mmol/L and procalcitonin of 2.62 µg/L. Liver function test, amylase and lipase levels were normal. CT abdomen and pelvis showed normal gallbladder, biliary tree and pancreas. She became **acutely delirious** requiring an urgent transfer to the Obstetric Intensive Care Unit (ICU). Serum glucose returned at 33mmol/L and serum ketones was raised at 2.4mmol/L, suggesting **diabetic ketoacidosis**. Insulin infusion and treatment for metabolic acidosis commenced, which remained refractory. Urgent transfer was arranged to a tertiary Medical ICU for continuous renal replacement therapy (CRRT). Amylase and Lipase were elevated at 1187U/L and >600U/L respectively 15 hours after the onset of her symptoms. A clinical diagnosis of **Glasgow 3 pancreatitis** was made. She was later found positive for **anti-glutamic acid decarboxylase (GAD) autoantibody**, which suggested a new manifestation of `insulin-dependent diabetes mellitus.

DISCUSSION

GDM is one of the most common metabolic disorders in pregnancy, characterized by glucose intolerance in pregnancy in prior healthy individuals. This hyperglycaemic state is expected to improve postnatally. **Up to 10% of women with GDM have antibodies to pancreatic islet or to β cell antigens such as anti-GAD antibodies**(1), suggesting autoimmune-mediated destruction of pancreatic β cell, behaving like patients with Type 1 DM. The presence of autoimmunity against β-cells in women with GDM have a higher risk of progression Latent Autoimmune Diabetes of Adulthood (LADA) after pregnancy (2-3). **It is also found that women with autoimmune GDM have similar clinical characteristics as patients with T1 DM, such as younger age, low BMI, presence of ketones or other autoimmune conditions and persistence of impaired glucose regulation postnatally** (4). Therefore, while it is not routine to screen for pancreatic autoimmune antibodies in all women with GDM, it remains a consideration to test women with the above clinical features, especially if they require insulin early in pregnancy or develop ketoacidosis in the course of pregnancy or early postpartum. The presence of these antibodies do not affect the maternal or fetal outcomes during pregnancy but may provide clinicians a low index of suspicion for lethal complications including DKA and pancreatitis.

CONCLUSION

This is an unexpected presentation of postpartum acute fulminant diabetic ketoacidosis in a patient with GDM in pregnancy. We note that:

- **Presence of anti-GAD autoantibody** suggested an autoimmune-mediated destruction of pancreatic β cells.
- Awareness should be raised for pregnant women with the following clinical features: **Younger age, low BMI, presence of ketones or other autoimmune conditions**.
- Postnatal DKA in a low-risk woman with GDM may catch the clinician off guard. There should be **a low index of suspicion amongst clinicians for this potentially lethal condition in women with GDM** when they become acutely unwell.

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