

EP_A053**BALANCING HORMONAL CHAOS: A CASE REPORT ON TYPE 1 DIABETES MELLITUS AND LACTATION-INDUCED HYPOGLYCEMIA**

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INTRODUCTION/BACKGROUND

Type 1 Diabetes Mellitus (T1DM) accounts for 0.6% of diagnosed diabetes cases in Malaysia, presenting unique challenges for young adults of childbearing age. Women with T1DM encounter numerous hurdles not only before and during pregnancy but also in the postpartum period. Here, we present a case study of a young patient with T1DM who experienced recurrent hypoglycaemia during lactation.

CASE

A 31-year-old mother of two was diagnosed with T1DM at the age of 15 and was managed with multiple daily insulin (MDI) injections. Prior to her second pregnancy, her HbA1c was 6.1%. During her second pregnancy, she was on prandial insulin aspart six units and glargine 18 units. The patient's insulin requirement was further reduced immediately postpartum. Despite these adjustments, she encountered frequent hypoglycaemic episodes, particularly during breastfeeding. During her clinic visits, she was advised to take small snacks before nursing and to use a continuous glucose monitor (CGM). However, at four months postpartum, she presented with facial nerve palsy and was admitted for transient ischemic attack (TIA) due to severe hypoglycaemia. She initiated a sensor-augmented insulin pump trial at 16 months postpartum, which resulted in the cessation of hypoglycaemic episodes and subsequently, better glycaemic control.

CONCLUSION

Several factors contribute to lactation-induced hypoglycaemia in women with T1DM, including hormonal and physiological changes. Increased energy demand for milk production, elevated oxytocin and prolactin levels that enhance insulin sensitivity, and unpredictable timing and duration of breastfeeding sessions can all exacerbate hypoglycaemia. This case highlights the challenges of managing T1DM during lactation, specifically the increased risk of hypoglycaemia due to the energy demands of breastfeeding. Personalized treatment plans, in collaboration with endocrinologists, and the expanded utilization of CGM and insulin pumps can significantly

enhance glycaemic control and minimise the risk of hypoglycaemia in breastfeeding mothers with T1DM.

EP_A054**FAMILIAL HYPERTRIGLYCERIDEMIA MANIFESTING RECURRENT PANCREATITIS, ERUPTIVE XANTHOMAS, LIPEMIA RETINALIS IN A YOUNG FEMALE WITH TYPE 1 DIABETES MELLITUS AND PRIMARY OVARIAN FAILURE**

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INTRODUCTION/BACKGROUND

Severe hypertriglyceridemia is one of the etiologies of pancreatitis and is associated with diabetes mellitus.

CASE

We present a case of a 22-year-old female with DM diagnosed at 17 years old with presentation of osmotic symptoms. On examination, she was normotensive with a BMI of 23 kg/m², no features of Cushing syndrome but with sexual immaturity (Tanner stage 1). Baseline laboratory showed HBA1C of 10%, elevated AST 132 U/L, ALT 138 U/L, triglyceride (TG) 16.62 mmol/L, LDL 0.3 mmol/L, HDL 0.8 mmol/L, macroalbuminuria, normal renal profile, thyroid function test and cortisol level. Serum FSH of 24.2 IU/L, LH of 8.02 IU/L and estrogen levels of 71.1 pmol/L confirmed primary ovarian failure and karyotyping excluded Turner's syndrome. Ultrasound of the abdomen showed a fatty liver, a small uterus and ovaries. Initial treatment included an oral hyperglycaemic agent, basal insulin, fenofibrate and statin.

On subsequent follow-up, diabetes control remains poor, with HbA1c persistently above 10%, requiring intensification with basal-bolus insulin. Fundoscopy showed bilateral lipemia retinalis but no retinopathy. She developed acute pancreatitis two years after diagnosis of DM, and imaging confirmed pancreatitis without calculi. Lipid levels were not available due to lipemic samples. She later noted xanthomas over her extremities and presented again with severe pancreatitis and uncontrolled diabetes. Markedly elevated triglyceride level at 59.73 mmol/L reduced to 5.49 mmol/L with continuous insulin infusion. Omega-3 oil tablets were added, and an outpatient review showed improved HbA1c levels from 12.3% to 10.4% and triglyceride of 5.36 mmol/L. DM autoantibodies confirmed autoimmune diabetes.