

EP_A065

A UNIQUE FAMILIAL CLUSTER OF TYPE 1 DIABETES: CLINICAL COURSE AND TREATMENT CONSIDERATION

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INTRODUCTION

Familial type 1 diabetes occurs up to 10% in large databases. This case series illustrated a family with three new onset type 1 diabetes diagnosed due to elevated blood sugar and positive diabetes autoantibody.

CASE

The index case was a 15-year-old male who presented with progressive osmotic symptoms over 1 month in 2024. He was admitted for severe diabetic ketoacidosis (DKA). Initial hemoglobin A1c (HbA1c) was 10.7%, and anti-GAD was >2,000 IU/mL. He was treated with basal bolus insulin.

The youngest brother was a 12-year-old male, presented with osmotic symptoms for 1 week in 2025. Random blood glucose elevated to 36 mmol/L. HbA1c was 8.9%. Anti-GAD was >2,000 IU/mL. He was admitted with impending DKA and discharged with basal bolus insulin.

The second brother was a 15-year-old male currently asymptomatic. Random blood glucose was normal. Anti-GAD was elevated at 1,307 IU/mL. Discussion regarding teplizumab to prevent further progression to type 1 diabetes was halted due to high cost and limited availability of the drug.

CONCLUSION

Active screening for siblings with diabetes autoantibodies among siblings with type 1 diabetes should be considered. Treatment with teplizumab for asymptomatic persons should be considered.

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INDIVIDUALIZING THERAPY WITH REPAGLINIDE: A SINGLE-CENTRE EXPERIENCE

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INTRODUCTION

Repaglinide, a meglitinide analogue, has a rapid onset and short duration of action. It offers prandial glucose control while reducing hypoglycemia risk compared to sulfonylureas. We describe its use in our special cohorts of diabetic patients where repaglinide was prescribed to reduce hypoglycemia in vulnerable patients or to optimize glycemic control through pharmacotherapy intensification or deintensification.

CASES

Cohort A comprised 12 elderly patients with multiple comorbidities (mean age: 70.3 years; mean DM duration: 18.3 years) characterized by high glucose variability and problematic hypoglycemia. In seven patients, low-dose gliclazide was replaced with repaglinide with reduction in hypoglycemic episodes. One patient on low-dose pre-mixed insulin was successfully switched to preprandial repaglinide. Two patients on basal insulin were able to reduce their insulin requirements and stabilize their glucose levels. A younger patient with autonomic dysfunction had hypoglycemia while on insulin glulisine, while its omission led to severe hyperglycemia. Substituting glulisine with repaglinide resolved the glucose variability. Repaglinide effectively reduced hypoglycemic episodes while maintaining glycemic stability, with a mean hemoglobin A1c (HbA1c) reduction of 0.36% over 3–6 months.

Cohort B consisted of six younger patients (mean age: 35.3 years) with focus on optimizing HbA1c and improving treatment adherence. Two patients on basal-bolus regimes successfully transitioned off bolus insulin to repaglinide, significantly improving compliance. One patient on basal insulin was successfully transitioned to an all-oral regimen. Three patients on existing oral hypoglycemic agents were started on repaglinide to close the glycemic gap. Cohort B achieved a robust mean HbA1c reduction of 2.1% within 3–6 months.

CONCLUSION

Repaglinide remains an important armamentarium in personalized diabetes management. Its pharmacokinetics and flexible dosing allow intensification of glycemic control while minimizing the risk of hypoglycemia. It also offers a viable strategy for selected patients struggling with complex insulin regime.

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SUBARACHNOID HEMORRHAGE: A RARE BUT DEVASTATING COMPLICATION OF DIABETIC KETOACIDOSIS

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INTRODUCTION

Diabetic ketoacidosis (DKA) is a common complication of type 1 diabetes mellitus (T1DM). Neurological complications of DKA are rare in adults. We report a case of young lady with T1DM who presented with severe DKA and complicated with subarachnoid hemorrhage (SAH).

CASE

A 19-year-old female was diagnosed with T1DM following an admission for DKA 2 months earlier. Her hemoglobin A1c was 17.2%, and autoimmune markers showed positive anti-glutamic acid decarboxylase (anti-GAD) antibodies (71 IU/mL) with negative anti-insulinoma-associated antigen 2 (anti-IA2) and islet cell antibodies. Her C-peptide level was low at 69 pmol/L. She was discharged with basal bolus insulin analogues, but she defaulted on her medications. She presented to the hospital 2 months later with a history of epigastric pain, vomiting, and breathlessness for 2 days, and was then found unconscious at home. There was no history of trauma. She was intubated for a poor Glasgow Coma Scale (E1V1M1). Blood results showed severe DKA with glucose 25 mmol/L, blood gas pH 6.572, bicarbonate 2.6 mmol/L, and ketone more than 8.8 mmol/L. She was started on insulin infusion and fluid resuscitation. DKA resolved after 24 hours. Computed tomography (CT) of the brain showed SAH in the right frontal and left frontotemporal regions. CT angiography of the brain reported no evidence of an intracranial aneurysm. She was extubated on day 4 of admission. Subsequent cerebral digital subtraction angiography was unremarkable. The patient achieved complete neurological recovery on follow-up.

CONCLUSION

This case highlights that severe DKA may rarely be associated with spontaneous SAH, which increases morbidity and mortality. Proposed mechanisms include vascular injury, oxidative stress from severe ketoacidosis, and endothelial dysfunction related to inflammatory mediators. Clinicians should have a high suspicion and consider neuroimaging in DKA patients with unexplained reduced consciousness or atypical neurological findings.

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ASSOCIATION OF TESTOSTERONE WITH INSULIN RESISTANCE AND BETA-CELL FUNCTION IN TYPE 2 DIABETES MELLITUS

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INTRODUCTION

Insulin resistance and progressive beta-cell dysfunction are key components of the pathophysiology of type 2 diabetes mellitus (T2DM). The triglyceride-to-high-density lipoprotein cholesterol (TG/HDL) ratio has been widely used as a marker of insulin resistance. In addition, testosterone deficiency has been associated with adverse metabolic profiles and T2DM. However, the relationship between testosterone levels, insulin resistance, and beta-cell function in patients with T2DM remains incompletely understood.

METHODOLOGY

A cross-sectional study was conducted involving 25 male patients with T2DM attending the diabetic clinic at Dr. Mohammad Hoesin General Hospital. Serum testosterone, C-peptide, TG, and HDL levels were obtained from routine clinical data. The TG/HDL ratio was calculated as an index of insulin resistance. Normality was assessed using the Shapiro-Wilk test, and due to the non-normal distribution of key variables, Spearman correlation analysis was applied.

RESULTS

A strong inverse correlation was observed between testosterone levels and TG/HDL ratio ($r = -0.860$, $p < 0.001$), indicating that higher insulin resistance was associated with lower testosterone levels. In addition, TG/HDL ratio showed a strong negative correlation with C-peptide levels ($r = -0.741$, $p < 0.001$), suggesting reduced beta-cell function in the presence of increased insulin resistance.