

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.