Adult E-Poster

October 2024. During this hospitalization, C-peptide levels increased to 287 pmol/L. Given his ongoing difficulties with insulin compliance, a new trial with basal insulin and a DPP-IV inhibitor was initiated.

CONCLUSION

The initial diagnosis of Type 1B diabetes was suggested by low C peptide and negative autoantibodies. Persistent hypoglycaemia despite low insulin doses and the challenge of non-compliance led to consideration of MODY variants, prompting trials with oral glucose-lowering drugs. Further genetic studies are needed for a definitive diagnosis.

EP_A041

FASTING, FEASTING, AND FALLING GLUCOSE: A CASE OF NON-INSULINOMA PANCREATOGENOUS HYPOGLYCEMIA SYNDROME FOLLOWING WEIGHT LOSS AND KETOGENIC DIET DISCONTINUATION

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INTRODUCTION/BACKGROUND

Non-insulinoma pancreatogenous hypoglycemia syndrome (NIPHS) is a rare cause of endogenous hyperinsulinemic hypoglycemia, distinct from insulinoma, often linked to β-cell dysfunction after bariatric surgery in adults.

CASE

This case report describes a previously well 31-year-old male with recurrent hypoglycemia symptoms following intentional weight loss of 44 kg (120 to 76 kg) from practising ketogenic diet for one year. The patient experienced recurrent episodes of giddiness, palpitations and syncope, with lowest capillary blood glucose levels documented as 1.8 mmol/L. These symptoms emerged after resuming a regular carbohydrate diet, after he developed severe constipation with ketogenic diet. The episodes were erratic but reported to be more common after prolonged fasting.
Initial evaluation revealed a normal HbA1c (4.7%) with normal hemoglobin (16.5 g/dL), normal morning cortisol and renal and hepatic functions. A supervised 72-hour fast demonstrated symptomatic venous hypoglycemia (2.7 mmol/L) at the 36th hour with inappropriately elevated insulin (5.67 μIU/mL) and C-peptide (496 pmol/L) levels, confirming endogenous hyperinsulinemia. Prolonged oral glucose tolerance testing (OGTT) revealed exaggerated insulin secretion (peak insulin: 43.92 mIU/L at 1 hour; glucose: 8.1 mmol/L), followed by non-suppression of insulin (5.65 mIU/L) and C-peptide (1244 pmol/L) at 4 hours despite low blood glucose of 3.5 mmol/L. Pancreatic CT was normal, supporting a diagnosis of NIPHS. Patient's symptoms improved following small frequent meals and avoidance of large amounts of simple carbohydrates.

CONCLUSION

This case highlights NIPHS as a consequence of altered β-cell function following prolonged ketogenic diet, likely exacerbated by rapid dietary carbohydrate reintroduction.

EP_A042

BILATERAL OSTEOPOROTIC FEMUR FRACTURES IN A YOUNG WOMAN: AN AFTERMATH OF EMPTY SELLAR SYNDROME

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INTRODUCTION/BACKGROUND

Empty sella syndrome (ESS) is characterized by the radiological finding of a flattened pituitary gland within the empty sella turcica due to subarachnoid space expansion, commonly associated with hormonal deficiencies. We report a rare case of panhypopituitarism due to primary ESS in a young female who presented with bilateral osteoporotic femur fractures.

CASE

A 38-year-old aboriginal female was first brought to the orthopedic team for persistent right hip pain and a limping gait for several years. There was no prior history of trauma, surgery or irradiation. CT of bilateral hip joints showed generalized osteopenia with non-union bilateral femur fracture. Bone mineral density revealed osteoporosis of the lumbar spine and left radius with Z-score of -4.3 and -6.6, respectively. Further evaluation for secondary osteoporosis revealed short stature with a low BMI of 17 kg/m2. Notably, she has primary amenorrhea and delayed puberty, with Tanner stage 1 breast and pubic hair development. Family history was unremarkable except for one younger sister with short stature. Anterior pituitary hormone profile revealed central hypothyroidism (TSH: 2.05 mIU/L, fT4: 6.7 pmol/L), hypogonadotrophic hypogonadism (serum estradiol <55.1 pmol/L, FSH: 0.3 IU/L, LH <0.2 IU/L), low