CR-D-45

SEVERE INSULIN RESISTANCE REQUIRING HIGH DOSE INSULIN IMPROVED WITH ADD-ON EMPAGLIFOZIN THERAPY: A CASE REPORT

https://doi.org/10.15605/jafes.034.02.S89

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INTRODUCTION

Empagliflozin, an SGLT-2 transporter inhibitor is known to improve glucose control with weight loss. Its role in reducing insulin resistance is under-recognised.

CASE

A young patient with BMI of 47.4 kg/m² and diabetes has been treated with high dose multiple daily insulin injection totalling 600 iu/day (4.2 iu/kg/day). Despite compliance, lifestyle intervention and add-on therapy of metformin and vildagliptin, insulin requirement only reduced to 275 iu/day(2 iu/kg/day) and remained static. We subsequently started him on 25 mg daily of empaglifozin.

CONCLUSION

In an obese patient with insulin resistance, empaglifozin improved insulin sensitivity leading to significant reduction in insulin requirement

KEY WORDS

insulin resistance, obesity, empaglifozin

CR-D-46

A CASE OF HYPOGLYCAEMIA IN A NON-DIABETIC ADULT

https://doi.org/10.15605/jafes.034.02.S90

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INTRODUCTION

Hypoglycaemia is diagnosed based on the Whipple's Triad. Nondiabetic adult hypoglycaemia may be related to critical illnesses, drugs, cortisol or glucagon insufficiency, insulinoma or it can be factitious. Post prandial hypoglycaemia may be an early sign of prediabetes.

CASE

We report a 26-year-old female with recurrent symptomatic hypoglycaemia with symptoms of sweating, giddiness and lethargy for 6 months. She has no known medical illnesses but has a strong family history of type 2 diabetes mellitus. She denies taking traditional medications. The hypoglycaemic symptoms occur after 3-4 hours of her main meals. She had documented glucose level of 1.8-2.8 mmol/L by finger prick testing. The symptoms resolve after consuming sugary drinks. Body mass index was 20 kg/m², HbA1c was 4.5%, eGFR 60 ml/min. A prolonged oral glucose tolerance test was performed over 5 hours. At 0 hour her glucose was 4.9 mmol/l, 60 min 10.3 mmol/L, 120 min 8.9 mmol/L and at 5 hours it was 2.8 mmol/L. Her cortisol was 579 nmol/l, insulin level 7.24 uIu/mL (NV 2.6-24.9) and C-peptide 509.3 pmol /L (NV 370-1470). The occurrence of biochemical hypoglycaemia at 5 hours post OGTT is likely suggestive of inappropriate hyperinsulinemic state of an individual with prediabetes.

CONCLUSION

Hyperinsulinemic hypoglycaemia is biochemically due to unregulated secretion of insulin from the pancreatic beta cells during low blood glucose levels. This mechanism occurs in prediabetic patients as a counterregulatory effect towards insulin resistance leading to prolonged insulin secretion. Further research is needed to better understand the underlying mechanism.

KEY WORDS

hypoglycemia, hyperinsullinemic, prediabetic