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.

EP_A067

SUBARACHNOID HEMORRHAGE: A RARE BUT DEVASTATING COMPLICATION OF DIABETIC KETOACIDOSIS

Hui Min Tan[1], Mohd Irfan bin Moktar[1], Hwee Ching Tee[2]

[1]Department of Medicine, Hospital Beaufort

[2]Department of Medicine, Hospital Queen Elizabeth II

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

Khoirun Mukhsinin Putra[1], Yulianto Kusnadi[1], Ratna Maila Dewi[1], Alwi Shahab[1]

[1]Division of Endocrinology, Metabolism, and Diabetes, Faculty of Medicine, Sriwijaya University/Dr. Mohammad Hoesin General Hospital, Indonesia

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.