

EP_A034**EUGLYCEMIC DIABETIC KETOACIDOSIS PRECIPITATED BY HYPERTRIGLYCERIDEMIA-INDUCED PANCREATITIS, LIVER ABSCESS AND SODIUM-GLUCOSE COTRANSPORTER-2 INHIBITOR USE IN A PATIENT WITH FAMILIAL HYPERTRIGLYCERIDEMIA**

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INTRODUCTION/BACKGROUND

Euglycemic diabetic ketoacidosis (EDKA) has a worse outcome than typical DKA as it is relatively rare and remains a diagnostic challenge. Conditions such as sepsis, pancreatitis, use of sodium-glucose cotransporter-2 inhibitors (SGLT-2i), pregnancy and starvation are known to be associated with EDKA. We report a case of a patient with Type 2 Diabetes Mellitus (T2DM) and familial hypertriglyceridemia on SGLT-2i who presented with hypertriglyceridemia-induced pancreatitis (HTGP) concurrently with EDKA.

CASE

A 31-year-old female presented with epigastric pain, vomiting and lethargy. The clinical exam revealed tender epigastrium with no guarding and negative Murphy's. Serum amylase was 242 U/L (Imrie score 2, BISAP 1) and C-reactive protein (446 mg/L). The ultrasound of the abdomen revealed an ill-defined collection (2.3 x 3.2 cm) at segment V of the liver with findings suggestive of chronic pancreatitis. She had three prior admissions due to acute pancreatitis and once complicated by an infected pancreatic pseudocyst. She was diagnosed with T2DM and familial hypertriglyceridemia five years ago, with poorly controlled glucose and lipid profile (HbA1c 8.4%, triglycerides 33.4 mmol/L). She is on an SGLT2 inhibitor, amongst other medications, which she continued taking despite her illness. She developed EDKA in the ward (pH 7.43, PCO₂ 20, HCO₃ 14, serum ketone 3.6, lactate 0.8). She was started on DKA treatment, then continued with variable rate insulin sliding scale, fasting, statin, fibrates and intravenous antibiotics. Dietary and lifestyle advice were reinforced. She was discharged well after two weeks (triglyceride 4.2 mmol/L, C-reactive protein 2 mg/L) with resolved symptoms and liver lesions.

CONCLUSION

EDKA should be a well-recognised diagnosis in an era where there is growing use of SGLT2i, especially in patients with multiple precipitating factors. Physicians must have a high clinical suspicion in patients who are on SGLT-2i in acute illness. In addition, we need to consider that EDKA can precipitate HTGP and vice versa. In both conditions, early initiation of continuous intravenous insulin infusion can improve outcomes.

EP_A035**A REVIEW OF CLINICAL PROFILE AND GLYCEMIC CONTROL OF PATIENTS WITH YOUNG-ONSET TYPE 2 DIABETES MELLITUS ON INTENSIVE INSULIN THERAPY**

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INTRODUCTION/BACKGROUND

Young-onset type 2 diabetes mellitus (T2D) is a more aggressive subgroup of T2DM with rapid disease progression and rate of complications. Many patients progress to intensive insulin therapy early in the disease process due to decompensation and poor glycaemic control.

METHODOLOGY

We aimed to review the demographic profile, glycaemic control, and prevalence of complications in patients with young T2DM on intensive insulin therapy at the Endocrine Institute of Hospital Putrajaya. A retrospective audit was conducted using electronic medical records. Patients with T2DM between the age of 18-40 years on basal-bolus insulin therapy attending the outpatient diabetes clinic between January 2022 – March 2024 were included. Data about the demographic profile, insulin therapy, glycaemic control and complications were collected. A descriptive analysis using SPSS version 25.0 was performed.

RESULTS

The analysis involved a total of 72 cases, with a mean age of 33.7 years. Females comprised two-thirds (68.1%), with Malays being the majority (81.9%). The mean weight was 85.3 kg and the mean BMI was 32.1 kg/m². The mean duration of diabetes was 10.1 years. Among them, 62.5% have comorbidities such as hypertension and dyslipidaemia, and 48.6% are obese. The average duration of insulin therapy was 5.9 years. The mean HbA1c was 10.3% before insulin therapy and 9.5% on current intensive insulin therapy. Microvascular complications were prevalent (73.6%), with

nephropathy being the most common (59.6%), followed by retinopathy and neuropathy. Approximately 5% of patients had macrovascular disease. More than two-thirds (70.8%) were on statin and half (56.9%) were on anti-proteinuria therapy.

CONCLUSION

Most patients with young-onset T2DM have poor glycaemic control despite being on intensive insulin therapy. Most patients fit the phenotype of obesity with metabolic syndrome suggesting possible insulin resistance, as opposed to depletion, as the key factor driving disease progression. Treatment strategies employed should focus on intensive lifestyle intervention and pharmacotherapy targeting weight reduction and insulin resistance as opposed to excessive insulin in this subgroup.

EP_A036

SCREENING AND TREATMENT OF DIABETIC KIDNEY DISEASE IN TYPE 2 DIABETES MELLITUS (T2DM) PATIENTS: A CLINICAL AUDIT AT HOSPITAL SULTAN HAJI AHMAD SHAH TEMERLOH, MALAYSIA

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INTRODUCTION/BACKGROUND

Diabetic kidney disease (DKD) is a global health challenge that has garnered increasing attention due to its significant impact on individuals and healthcare systems worldwide. In Malaysia, DKD accounted for the majority of new dialysis patients, increasing cardiovascular risk and hence, escalating healthcare expenses.

METHODOLOGY

This clinical audit aims to assess the screening and treatment of DKD among T2DM patients in Hospital Sultan Haji Ahmad Shah (HOSHAS), Temerloh, Pahang. All T2DM patients attending the diabetes clinic in HOSHAS from June to July 2023 were included in this clinical audit. Electronic medical records were assessed for demographic data, blood pressure and glycaemic targets, screening and treatment of macro- or microalbuminuria.

RESULTS

We included 141 patients in this audit. Of those, 63.8% were females, with a mean age of 52.8 ± 15.0 years and an average duration of diabetes of 13.0 ± 8.4 years. The screening rate for albuminuria was high (93.6%) but only 25.5% of the

patients had further quantification of albuminuria. Overall, 31.9% achieved a blood pressure target of below 140/80 mmHg but only 19.0% with albuminuria achieved a BP target of below 130/80 mmHg. A total of 19.1% of patients achieved HbA1c of less than 7%. Among the patients with albuminuria, 71.2% were on ACE-i/ARB and 39% were prescribed SGLT2 inhibitors.

CONCLUSION

This audit highlights the importance of early detection and appropriate management of DKD in T2DM patients. Microalbuminuria assessment, optimal blood pressure and renal-modulation therapy are essential in preventing the progression of albuminuria and reducing the risk of ESKD in patients with diabetes.

EP_A037

GLUTAMIC ACID DECARBOXYLASE (GAD) ANTIBODIES-ASSOCIATED LIMBIC ENCEPHALITIS AND DIABETES: A CASE REPORT

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INTRODUCTION/BACKGROUND

Glutamic acid decarboxylase (GAD) is an enzyme involved in producing the major inhibitory neurotransmitter Gamma-Aminobutyric Acid (GABA). GAD antibodies have been implicated in the pathogenesis of insulin-dependent diabetes mellitus (IDDM) and a few neurological diseases such as the case below.

CASE

A 24-year-old male presented with a one-week history of fever, gradual memory impairment, behavioural changes and seizure. On arrival, he was confused and disoriented. His blood glucose was 18 mmol/L, HbA1c of 12.3% with acidosis at pH 7.30, bicarbonate of 19.7, serum osmolarity of 282 mmol/L and urine FEME showed ketone 2+, glucose 3+.

The lumbar puncture CSF sample was acellular with normal cerebrospinal fluid protein. Serum autoimmune and paraneoplastic panels were negative. EEG showed seizure activity at the right frontotemporal region with clinical evidence of piloerection. His brain MRI was abnormal with hyperintensity and swelling of the right medial temporal lobe. Correlating the history, EEG and radiological changes, his diagnosis is supportive of limbic encephalitis with newly diagnosed diabetes mellitus. Intravenous