CONCLUSION
Despite the absence of a significant statistical difference, more patients presented with more severe DKA with longer recovery during the COVID-19 pandemic. A larger multi-centre study is needed to evaluate the magnitude of the impact of COVID-19.

EP_B005

DOES THE INCIDENCE OF DIABETIC KETOACIDOSIS IN PATIENTS WITH TYPE 1 DIABETES MELLITUS DIFFER DURING COVID-19 PANDEMIC?

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INTRODUCTION
Recent studies have observed increased numbers of newly diagnosed type 1 diabetes patients and higher incidence of diabetic ketoacidosis in T1DM patients during the COVID-19 pandemic. The care of non-COVID-19 patients was compromised due to the tremendous burden of managing critical cases of COVID-19 patients. However, this finding is not consistent. This study compares the incidence of DKA and its severity during the pandemic with the similar timing prior to it.

METHODOLOGY
This is a retrospective cross-sectional study on all of patients who were either newly diagnosed or pre-existing patients with T1DM aged one to 18 years old treated at University Malaya Medical Centre for DKA. from September 2017 until August 2022. Data on demographics, first DKA presentation, recurrence, severity, pediatric ICU admission, duration of recovery, COVID-19 status, duration from symptoms to presentation, and biochemical values were obtained.

RESULT
A total of 96 DKA cases were reported from September 2017 until August 2022. An equal number of DKA cases (48) was seen in each period. Fifty patients (69%) were newly diagnosed. Recurrent DKA occurred more during the COVID-19 period (27 versus 21). More patients presented with severe DKA during the pandemic (24 versus 19). Although not statistically significant, newly diagnosed T1DM presented with more severe DKA with lower pH (7.05 versus 7.12, p=0.417) and took longer to recover compared to the pre-pandemic period (48 versus 36 hours, p=0.150). Three newly diagnosed patients were COVID-19 positive with COVID-19 Category 2b and presented in moderate to severe DKA.

CONCLUSION
There was no difference in the number and severity of DKA cases during the pre-pandemic and COVID-19 pandemic period. A larger-scale study is needed to analyse the impact of COVID-19 on the incidence and severity of DKA.

EP_B006

INSULIN ANTIBODY MEASUREMENTS: SHEDDING LIGHT ON HIRATA'S DISEASE

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INTRODUCTION
The Endocrine Society Clinical Practice Guideline recommends measurement of insulin autoantibodies (IAA) upon confirmation of endogenous hyperinsulinism. The differential diagnosis of endogenous hyperinsulinism include insulinoma, post-bariatric hypoglycaemia, nesidioblastosis and insulin autoimmune syndrome (IAS). IAS, also known as Hirata's disease, is a rare immune-mediated disorder characterised by hyperinsulinaemic hypoglycaemic episodes. It is increasingly being recognized in Malaysia because of accessibility to IAA testing.

CASE
We describe two cases of newly diagnosed IAS with varied clinical presentations and treatment approaches. The first patient is a 57-year-old male with Graves' disease who experienced severe and recurrent hypoglycaemia during fasting and postprandial states. The second patient is a 56-year-old female with hypertension and bronchial asthma who developed recurrent hypoglycaemia despite cessation of insulin therapy following the treatment for severe refractory diabetic ketoacidosis.

Laboratory findings for both patients showed elevated serum insulin and C-peptide during the hypoglycaemic event, with insulin/C-peptide ratio >1. Pancreatic antibodies were negative. Serum insulin autoantibodies measured on

chemiluminescent immunoassay revealed remarkably high titres in both cases. Triggering factors were identifiable in both cases: in the first, exposure to carbimazole; and in the second patient: exposure to pantoprazole, amlodipine, metoprolol, perindopril and amoxicillin clavulanate. The first patient improved with dietary modification and alpha-glucosidase inhibitor. The second patient was treated with steroids.

CONCLUSION
Our case series highlighted the importance of measuring insulin antibody titer after confirming endogenous hyperinsulinism. High insulin concentration along with insulin/C-peptide molar ratio >1 should raise the clinical suspicion of IAS. Early recognition of this syndrome can avoid the need for laborious and costly investigation of presumed insulinoma with appropriate therapeutic approach.

KEYWORDS
endogenous hyperinsulinism, hypoglycaemia, insulin autoantibodies

EP_B007

HYPERGLYCAEMIA-INDUCED MOVEMENT DISORDER

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INTRODUCTION/BACKGROUND
Dyskinetic syndromes are one of the rarer initial presentations of undiagnosed diabetes. We report a case of hyperglycaemia-induced movement disorder in a newly diagnosed diabetes mellitus patient.

CASE
A 27-year-old male with no known medical illness presented to our centre in September 2019 with involuntary movement of left facial and left upper limb for five days, along with lethargy and persistent vomiting. Initial tests showed serum glucose 17.7 mmol/L, serum ketone 2.2 mmol/L and metabolic acidosis on blood gas, indicating non-ketotic hyperglycaemia. Serum osmolarity was 292 with deranged renal profile. His HbA1c level was 16.8%. He had a strong family history of DM. In the ward, he was treated with insulin infusion and adequate hydration. Risperidone tablet was started for new-onset chorea. Cranial CT during this initial admission showed hyperdense areas at both basal ganglia and thalamus, suggesting changes that correlated with his hyperglycaemic state. He presented again in November 2019 for lower gastrointestinal bleed secondary to rectal ulcer. During this admission, his involuntary movement progressed into hemidystonia of the left facial and left upper limb. He was then started on Baclofen tablet. Blood glucose was controlled during this admission. Brain MRI showed hyperintensity T1-weighted sequence, heterogenous hypointensity on T2-weighted and FLAIR sequences at bilateral caudate nuclei, bilateral lentiform nuclei and posterior thalamus. There were no other focal lesions to suggest other causes of the movement disorder.

Metabolic derangement in the absence of focal vascular lesions at the basal ganglia area are the common cause of hemiballism and hemichorea as was observed in our patient.

CONCLUSION
The case illustrates that abnormal movement may persist despite adequate glycaemic control. Appropriate medical therapy should be initiated to control the complication.