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

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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.