Discussion: Because patients with MODY 5 are at high risk for CKD3-4/ESRD, MODY 5 should be considered in young adults with diabetes who have negative islet autoantibodies and extra-pancreatic manifestations including elevated liver enzymes, renal cysts and nephrolithiasis; ADHD and learning difficulties; and pancreatic and hepatic morphological abnormalities. HNF1b-targeted genetic testing should be considered in patients with this clinical presentation.

Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

A Bizarre Case of Recurrent Hypoglycemia

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Introduction: Severe hypoglycemia is extremely rare in non-diabetic subjects and requires thorough evaluation due to its life threatening consequences. Any injuries resulting from medication use, including physical harm, mental harm, or loss of function is defined as adverse drug events (ADEs). Medication errors can occur due to the inappropriate use of medicine in any prescription stage. We hereby present an intriguing case of recurrent hypoglycemia in relation to it. Case presentation: A 65 year-old white male with surgically induced panhypopituitarism on replacement hydrocortisone, levothyroxine and testosterone was transferred to our hospital with recurrent serious hypoglycemic episodes. He was in his usual state of health until about 2 weeks prior to presentation when he became increasingly fatigued. He was found to have severe hypoglycemia with a blood sugar of 20 mg/dl. He was treated for hypoglycemia and was discharged home 2 days later with an increased dose of hydrocortisone and the provisional diagnosis of adrenal crisis. At home, his symptoms of hypoglycemia recurred. He was then referred to our facility where the Endocrinology service was consulted. At admission, his blood sugar was 69 mg/dl, but it rapidly dropped down to 39 mg/dl with confirmatory serum glucose level of 24 mg/dl. He was started on Dextrose 10%. On further inquiry his daughter told us that she had discovered two different medications in one prescription bottle which had been refilled about 2 weeks prior to this hospitalization, coinciding with the onset of symptoms. The bottle in question was supposed to be for hydrocortisone 10 mg tablets, but it contained 2 different tablets with inscription GXL10 and P10. An online search identified tablets as Glucotrol-XL 10 mg and hydrocortisone 10 mg. Urine drug screen (UDS) for sulfonylureas and glinide was positive for Glipizide (203 ng/ml, normal level 0). His blood sugar stabilized with Dextrose and he was safely discharged home. So it was concluded that recurrent hypoglycemia was secondary to inadvertent administration of hypoglycemic agents, Glucotrol-XL 10 mg, likely aggravated by missing doses of hydrocortisone. **Discussion:** The first report of medication errors was published in 1940. ADEs comprise the largest single category of adverse events experienced by hospitalized patients,accounting for about 19 percent of all injuries. ADEs are associated with increased morbidity and mortality, prolonged hospitalizations, and higher costs of care. Four medications or medication classes were implicated in 67% of hospitalizations: warfarin, insulins, oral antiplatelet agents, and oral hypoglycemic agents. ADEs in the outpatient setting are an important cause of emergency department visits and hospital admissions. This case emphasized the importance of taking a thorough history and focusing on patient safety and quality care in healthcare.

Diabetes Mellitus and Glucose Metabolism

DIABETES CASE REPORTS

A Case of Autoimmune Hypoglycemia (Insulin Autoimmune Syndrome)

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Introduction: Insulin Autoimmune Syndrome (IAS) is a condition caused by Insulin Autoantibody (IAA). IAS was initially reported in 1970 by Yukimasa Hirata from Japan. From 1970 to 2009, 380 cases were reported worldwide. We report a case of IAS in an African-American man who had recurrent episodes of syncope secondary to hypoglycemia. **Case Report:** A 50-year-old African American man with hypertension presented to emergency room after syncope. His initial blood glucose (BG) was 27 mg/dl. After Dextrose and glucagon injection, BG became 270 and he regained consciousness without any deficit. Vital signs and physical examination were unremarkable. He was not on any medication, and had no access to insulin or oral hypoglycemic agents. No family history of endocrine disorders, malignancy or autoimmune diseases were reported. He had been hospitalized two times for syncope secondary to hypoglycemia. During hospitalization, fasting insulin level was >1000 IU/ml with high C-peptide (3.5ng/ml). Hypoglycemic agents assay was negative. During outpatient follow-ups, fasting labs showed C-peptide 1.59 ng/ml, Insulin antibody 37.6 U/ml while venous BG was 65 mg/dl. Random BG at different occasions were 65, 68 and 55 mg/dl. HbA1C was normal. BMI was gradually increased from 32.4 to 34.8 in 6 months. During the third hospitalization, fasting venous BG were 45 and 57 on two different days, Insulin was 2111 IU/ml and C-Peptide was 1.78 ng/ml. Other labs including cortisol and TSH were normal. CT abdomen was unremarkable.
Discussion: In IAS, Insulin secreted after meal is bound by IAA. It causes hyperglycemia unchecked which triggers further insulin secretion. When the bound insulin dissociates from antibodies, it causes significant hyperinsulinemia and subsequent hypoglycemia. IAS can occur regardless of previous insulin exposure, as a solitary autoimmune manifestation or in association with other autoimmune disorders. It can be induced by drugs that contain sulfhydryl group. Etiology of IAS is still not fully understood but there are theories that genetic predisposition such as class II Human Leukocyte Antigen (HLA) and environmental triggers such as medications, viruses, hematological diseases etc. might play a role. In our patient, failure to test IAA in