Conclusion(s) Association of TS to autoimmunity is however widely known. Therefore, it is proposed that all patients with TS should be investigated for diabetes & the clinician must keep in mind the risks of metabolic complications when GH therapy applied in the presence of diabetes, but also aware that the treatment will improve their final height.

54 MODY-ABBC8 SUBTYPE; FROM DIAGNOSIS TO TRANSITION TO SULPHONUREA

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Background MODY (maturity onset diabetes of the young) are a group of inherited diabetes caused by genetic mutations. Mutations in ABCC8 affect the sulphonurea receptor 1 (SUR1) protein and can be associated with MODY, neonatal DM, and gestational diabetes.
Case Report(s) Eleven year old Raida Khalaf was admitted to our hospital in August, 2020 with hyperglycemia without Diabetic ketoacidosis. She has a strong family history of transient diabetes. Two of her brothers were diagnosed with diabetes from the age of 3 months which resolved at age of three years (now aged 14 and 17). Her sister had neonatal diabetes which resolved at the age of one year but returned at the age of 18 years. Raida was not diagnosed with neonatal or infantile diabetes. Her siblings were managed with insulin but were not investigated. Raida was started on mixtard insulin (30 R: 70 NPH) but had poor diabetes control and a high HBA1C. Due to her family history, she was sent to a tertiary center in Amman to investigate the possibility of MODY. A lab sample was sent to University of Exeter Medical University genomic lab and she was identified to be heterozygous for the Pathogenic ABCC8 missense mutation. Recommendations for the transition to sulphonurea and a guideline were provided by the lab. Raida was admitted to hospital and successfully transitioned to sulphonurea(glibenclamide) over the course of 10 days. Over the next few months, there was improvement in glycemic control and a significant drop in HBA1C.
Conclusion(s) ABCC8 mutation and other forms of inherited diabetes must be considered in a patient with strong family history of diabetes, for which genetic testing can be done and effective treatment can be started.

55 VKH WITH APECED IN A TWO-YEAR-OLD CHILD: A RARE CONCOMITANT DIAGNOSIS IN AN UNPRECEDENTED AGE

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Background Vogt Koyanagi Harada (VKH) is a syndrome most commonly presenting between the second and fifth decades of life.1,2 Its incidence in children is rare, with a much graver prognosis and worse visual outcome. Autoimmune polyendocrinopathy candidiasis ectodermal dystrophy syndrome (APECED) is a rare genetic (autosomal recessive) disorder. It was first reported in 1929 under the name of type 1 autoimmune polyglandular syndrome (APS1) or Whitaker's syndrome.3 It is characterized by the presence of two or more endocrine disorders with immunological pathogenesis, resulting in a hypofunctional state.

The clinical picture is variable, but chronic mucocutaneous candidiasis is typically the presenting symptom.4 Other autoimmune manifestations often ensue, most commonly Addison's disease, hypothyroidism, and gonadal failure.

Insulin-dependent diabetes, alopecia areata, vitiligo, ectodermal dystrophy, nail dysplasia, and ocular symptoms (keratoconjunctivitis, iridocyclitis, cataract, and optic atrophy) are less common manifestations.
Case Report(s) A two-year-old type 1 diabetic with hypothyroidism presented with impaired fixation. Ocular examination revealed right vitritis, choroiditis, a hyperemic disc, and an area of exudative detachment. At the same time, there was no fundus view in the left eye, and ultrasonographic assessment revealed vitritis and a thickened choroid. Patient developed sunset glow fundus with alopecia, poliosis and vitiligo and a diagnosis of complete VKH with APECED was made.
Conclusion(s) APECED is a rare endocrine disorder and has been reported to be associated with VKH twice. Likewise, VKH is commonly present in much older patients; this is the first time ever to be diagnosed in a two-year-old child.

56 PROLONGED HONEY MOON FOR 2 YEARS, VERY SENSITIVE TO INSULIN THERAPY

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Background Type 1 diabetes (T1D) is a chronic autoimmune disorder characterized by the destruction of pancreatic beta cells, resulting in a deficiency of insulin production. T1D typically presents in childhood and requires lifelong insulin therapy. However, in some cases, patients experience a transient 'honeymoon period' shortly after diagnosis, during which residual beta cell function may temporarily alleviate insulin requirements. This case report describes a 7-year-old boy diagnosed with T1D who displayed a prolonged honeymoon period and an intriguing sensitivity to insulin therapy, accompanied by positive anti-GAD antibodies in his laboratory results.
Case Report(s) A 7-year-old male child presented to our pediatric endocrinology clinic with a recent diagnosis of T1D. He had been experiencing polyuria, polydipsia, and unintentional weight loss over the preceding weeks. His initial blood glucose was markedly elevated at 400 mg/dL (normal range: 70-120 mg/dL), with a hemoglobin A1c (HbA1c) of 10% (normal range: 4-6%). Upon examination, the child was thin but appeared well. He had no family history of diabetes.

Upon admission, he was initiated on insulin therapy with basal and prandial insulin. Surprisingly, the patient exhibited remarkable sensitivity to insulin, with rapid normalization of blood glucose levels. After only a few days, his insulin requirements had dramatically decreased, and hypoglycemia became a recurrent concern. The clinical team decided to cautiously decrease and eventually halt his insulin therapy, given the potential for hypoglycemia. Despite discontinuing insulin, his blood glucose levels remained within the target range. This phenomenon was reminiscent of a prolonged honeymoon period, which is typically short-lived in T1D patients.

Laboratory results revealed an interesting finding. The patient's anti-glutamic acid decarboxylase (anti-GAD) antibody