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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10.1136/bmjpo-2024-ASPED.54

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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10.1136/bmjpo-2024-ASPED.55

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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10.1136/bmjpo-2024-ASPED.56

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

titer was significantly elevated at 1156 IU/mL (normal range: <5 IU/mL). Anti- GAD antibodies are associated with autoimmune destruction of beta cells in T1D. The persistence of positive anti-GAD antibodies in this case was intriguing, as it is often considered a marker of ongoing autoimmunity.

The patient's clinical course was closely monitored, and he continued to maintain stable blood glucose levels without insulin therapy. Regular follow-up assessments, including HbA1c, C-peptide, and anti- GAD antibody measurements, were performed to track his progress.

Conclusion(s) This case highlights a rare and fascinating scenario in pediatric T1D. The 7-year-old patient exhibited an extended honeymoon period, during which he displayed an exceptional sensitivity to insulin therapy, allowing for the discontinuation of insulin without compromising glycemic control. The presence of persistently elevated anti-GAD antibodies raises intriguing questions about the underlying immunological processes at play. Further research and long-term follow-up of this patient are warranted to better understand the mechanisms responsible for this unique presentation of T1D. This case underscores the importance of personalized management in T1D and the need for ongoing research into the immunological aspects of the disease to develop novel therapeutic approaches.

57 PERITONEAL DIALYSIS FOR TREATING REFRACTORY ACIDOSIS IN A CHILD WITH DIABETIC KETOACIDOSIS A CASE REPORT

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10.1136/bmjpo-2024-ASPED.57

Background Severe Diabetic Ketoacidosis (DKA) has been reported frequently in the literature, however, a DKA case requiring peritoneal dialysis exclusively for acidosis treatment has not been reported. Here, we reported the first child in the literature who suffered severe DKA as the first presentation, and treated using peritoneal dialysis for his ketoacidosis.

Case Report(s) A two-year old Filipino male child presented to the emergency room (ER)of a tertiary care center (Royal Hospital) with history of polyuria, polydepsia and weight loss of one week. He was seen in a private clinic two days before presentation to our ER with history of cough and difficulty in breathing, managed as viral infection with symptomatic treatment and discharged home. Child's condition did not improve, so he presented next day to the same private clinic. He was found to be tachyponic, recessing and desaturating. A diagnosis of acute exacerbation of bronchial asthma was entertained. Hence the child received hydrocortisone intravenously, salbutamol nebulization and aminophylline intravenously. General condition deteriorates as he developed shallow breathing and started to have drowsiness. At this stage, the patient was referred to our ER. The child was admitted urgently to the Pediatric Intensive Care Unit (PICU) with severe metabolic acidosis (PH: 6.8, bicarbonate: 4.9 Base Excess: -22). Hence, he was intubated and ventilated.

The child remained critically ill with severe acidosis. He received several doses of sodium bicarbonate, however, the PH continued to be in range of 6.9–7.1 (normal 7.35–7.45). In addition, his blood pressure continued to drop. He received several boluses of normal saline (10–20 ml/kg), and adrenalin infusion was also added.

Brain MRI was done on the 2nd day of admission, and showed moderate cerebral edema (CE). He was put on brain protection regimen. On the same day, he developed left lower limb swelling, and had femoral line inserted at the left femoral vein. Clinically the limb was worm with femoral and pedal pulses felt, and there were no signs of infection. Ultrasound with venous Doppler of the left limb showed evidence of deep vein thrombosis (DVT) in left common femoral vein. Hence the IV heparin infusion started with close monitoring of the coagulation profile.

Although his serum sodium remained in range of 136–140 mmo/l, he developed hyperchloremia which reached 123 mmol/l (normal 98–107 mmol/l).

In day 3 post admission, the general child's condition did not improve. He continued to be acidotic (PH: 6.9-7.1, bicarbonate below 10, base excess >-20). In addition, he had a drop in blood pressure secondary to the severe persistent acidosis which affected the cardiac function. He continued requiring two the inotropes (dopamine and adrenalin) to maintain his blood pressure.

The peritoneal dialysis (PD) was started on day 3 after admission. The dialyzer was constituting of 1.36% dextrose with lactate base solution and heparin 500 iu/l (FRAZENIUS-2013). The blood gas improved and normalized within 24 hours of staring PD (PH 7.35, bicarbonate 14.4 then 18 and base excess -13.5 then -8). The PD continued for 48 hours, and the general condition of the child improved. The blood pressure was maintained, hence the inotropes tapered then stopped. In addition, the ventilator support tapered to minimum. Subsequently, with further improvement he was extubated on day 7 post admission. Then, subcutaneous insulin injection was initiated with insulin Regular ® for one day, then both intermediate (NPH) and insulin ® were given. The dose was adjusted based on the blood glucose and child's oral feed and activity.

Conclusions This is first case reported with DKA among children managed by PD worldwide. Managing children with diabetic ketoacidosis in well-established centers with all facilities and experienced endocrinologist and multidisciplinary team is crucial to improve the patient outcome. Using peritoneal dialysis for treating acidosis is an invasive procedure and has its own risk, however, it can be inevitable treatment for resisting acidosis. However further studies are needed to confirm its effectiveness and weighing its risk.

58 A CASE REPORT 'YOUTH ONSET T2DM'

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10.1136/bmjpo-2024-ASPED.58

Background Diabetes is one of the most common chronic diseases with day by day increasing incidence(1). Globally,one in eleven adults have diabetes mellitus (90% have type 2 diabetes mellitus), and South East Asia is the epicentre of this global T2DM epidemic. Factors responsible for T2DM can be irreversible such as, genetic, age ethnicity race, and or revisable such as physical activity diet, and smoking.(2,3) Dietary choices and physical inactivity are the major reasons for the rapidly rising incidence of DM (3). The prevalence of type 2 diabetes in youth is increasing and pathophysiology clearly differs from type 1 diabetes: insulin resistance and nonautoimmune β-cell failure are underlying factors for T2DMbut little