CONCLUSION

Maternal gestational hyperthyroidism causes a hyperthyroid fetal environment due to increased thyroxine transfer which leads to suppression of the fetal hypothalamic-pituitary-thyroid axis and central hypothyroidism of the newborn. Primary hypothyroidism could be a result of transplacental passage of antithyroid drugs(ATD) during pregnancy or transplacental passage of maternal blocking antibodies. Early diagnosis and adequate treatment of mothers with GD is imperative to prevent the deleterious consequences of thyroid impairment during the neonatal period. Infants of maternal GD should be monitored for both hyperthyroidism and hypothyroidism.

PE-08

CASE SERIES OF NEONATAL DIABETES WITH KCNJ11 MUTATION_ TRANSFER FROM INSULIN TO SULPHONYLUREA

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INTRODUCTION

Permanent neonatal diabetes, presenting before 6 months old, signifies a monogenic cause. Mostly, it involves mutation of KCNJ11 gene that encodes the Kir6.2 subunit of the ATP-sensitive potassium channel (KATP). The landmark findings by Gloyn et al (NEJM 2004) on oral sulphonylurea (SU) binding to KATP and closing it by a non-ATP dependent route has markedly changed the landscape of management.

METHODOLOGY

Cross-sectional review of medical records.

RESULTS

3 patients (A, B, C) included. (A) presented at day 14, while (B&C) both at 2-month-old. (A) had hyperglycemia without ketosis while (B&C) had severe DKA. (B) also had seizures with delayed motor development (possibly intermediate DEND). All were initiated with subcutaneous insulin at diagnosis. Genetic tests were performed at 8-year-old, 1-year-old, and 5-month-old, respectively. Both (B&C) were similarly heterozygous for a pathogenic KCNJ11 missense variant with p.(Arg201Cys). Transfer to SU was performed based on the published protocol by Prof Andrew Hattersley from the University of Exeter. Time to SU varied with the slowest transfer at 8-year-9-month-old and quickest at 1-year-7-month-old. All transfers were successful with insulin weaned off. Noticeable improvement of HbA1c and C-peptide were demonstrated after 12 weeks. HbA1c decreased from 8% to 5.7%, 8.9% to 6.2%, and 9.6% to 5.8%; C-peptide improved from undetected (<33 pmol/L) to 185 pmol/L, 861 pmol/L, 73 pmol/L, respectively. (B) showed minimal response initially to gliclazide but an excellent response to glibenclamide. Initial glibenclamide dose varied from 0.8 mg/kg/day to 1.6 mg/kg/day. No hypoglycemia or GI complications. (A) needed to restart insulin at 13-year-8-month-old, 5 years after the transfer. (A) was the last to transfer to SU and required a higher initial dose.

CONCLUSION

Neonatal diabetes warrants rapid and focused genetic analysis to identify the genotypes with modifiable outcomes. Early genetic confirmation facilitates the transfer to oral SU for better glycaemic and neurodevelopmental outcomes and potentially improves the durability of the treatment.

PE-09

NEWLY DIAGNOSED T1DM PATIENTS - A DESCRIPTIVE STUDY IN A CHILDREN'S HOSPITAL

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INTRODUCTION

Type-1-diabetes mellitus (T1DM) is the most commonly diagnosed type of DM in children and adolescents. Typically, the presentation of T1DM is either as classic new onset DM, silent DM, or diabetic ketoacidosis (DKA). We aim to describe the epidemiological profile, clinical presentation, and factors related to delayed diagnosis in our patients.

METHODOLOGY

We retrospectively evaluated all newly diagnosed T1DM patients that presented to our centre from January 2015 till May 2021. Diagnosis of T1DM is based on clinical phenotype, with or without antibody confirmation.