CASE

A 15-year-old male with underlying pineal gland germinoma previously treated with surgery and cranial irradiation presented with a COVID-19 infection. He was treated for panhypopituitarism with thyroxine, hydrocortisone and DDAVP. Low dose growth hormone (GH) (0.016 mg/kg/day) was started after 5-years clinical remission. Pre-GH, his BMI was 24.2 kg/m^2 and the HbA1c was 4.9%. He complained of fever, respiratory distress, lethargy and reduced oral intake. At presentation, the plasma glucose was 52.2 mmol/L with high serum ketones of 7.6 mmol/L. Blood gas was acidotic (pH 7.25, bicarbonate 14.2 mmol/L). The HbA1c was 12.5% and the C-peptide was low. His COVID-19 PCR was positive. Fluid bolus was delivered, and he was managed as per DKA protocol. Stress dose hydrocortisone was given. After 12 hours he was transitioned to basal bolus subcutaneous insulin. After 1 month, he had recurrent hypoglycaemia prompting a reduction in the insulin doses and discontinuation after 2 months. The HbA1c and C-peptide level without insulin were 6.2% and 2.9 mg/ml respectively. His diabetes autoantibodies were negative.

CONCLUSION

COVID-19 infection is a potential trigger for development of new onset diabetes mellitus due to glucose dysregulation or autoantibody development. In our case, antibodies were negative and insulin dependency was temporary despite classically presenting with DKA. Long term follow up is required to monitor his glycaemic status.

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CENTRAL CONGENITAL HYPOTHYROIDISM IN AN INFANT OF A MOTHER WITH GRAVES' DISEASE

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INTRODUCTION

We report a case of central congenital hypothyroidism in a preterm baby born to a mother with hyperthyroidism.

CASE

The mother has been on treatment for hyperthyroidism since 2018. She developed thyrotoxicosis at 30 weeks pregnancy requiring Lugol's iodine, carbimazole and propranolol. She subsequently developed pre-eclampsia and went into labour. Her Thyroid Stimulating Hormone (TSH) receptor antibody levels were high, while anti-peroxidase and anti-thyroglobulin antibodies were negative. The child was born at 31 weeks gestation.

Initially the cord TSH level was 0.021 mIU/L. Subsequently, regular thyroid function tests continued to show very low TSH with normal T4. At one month of life, the T4 levels were low with persistent very low TSH and therefore the child was started on L-thyroxine. TSH receptor antibodies were positive. He was noted to have constipation and an umbilical hernia during this review. The diagnosis of central congenital hypothyroidism (CCH) was made, and the child was started on L-thyroxine. After initiation of therapy, T4 levels have normalised.

CCH is a rare condition with prevalence of 1 in 180 000 children. The risk is significantly increased in infants born to mothers with Graves' disease. In Graves' disease, patients may have TSH-blocking antibodies that bind to TSH receptors but do not initiate intracellular signaling, resulting in hypothyroidism. These antibodies can freely cross the placental, especially during the second and third trimester. Fetal thyroid matures functionally at around 25 weeks of gestation and because of this the hypothalamic-pituitary-thyroid axis can be affected in utero or postnatally.

CONCLUSION

This case highlights the importance of monitoring T4 and TSH levels in infants born to women with Graves' disease.