She was treated with L-thyroxine 100 mcg daily. GnRH Agonist (Leuprorelin) was initiated for a total of 18 months to halt premature puberty and to achieve age-appropriate target height.

CONCLUSION

Sexual precocity in a short, obese child with delayed bone age is a harbinger of VWGS. High TSH levels act through FSH receptors inducing an FSH like effect causing the prepubertal response seen in VWGS. Early puberty accelerates growth and promotes bone maturation, leading to early fusions that cause a decrease in final adult height (FAH). In our case, Leuprorelin was used to suppress the secretion of sex hormones, inhibit rapid bone maturation, and prolong the growth period, which improved FAH. This case highlights the importance of recognizing VWGS, so that thyroxine treatment can be initiated.

EP_A071

SYNDROME OF RESISTANCE TO THYROID HORMONE

https://doi.org/10.15605/jafes.038.S2.89

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INTRODUCTION/BACKGROUND

Resistance to thyroid hormone (RTH) is a rare genetic disorder characterized by clinically mild hyperthyroidism and biochemically elevated circulating free thyroid hormone levels with unsuppressed serum thyroid stimulating hormone. Here we reported the case of a 18-year-old male who was previously under paediatric follow-up for hyperthyroidism but with non-suppressed thyroid stimulating hormone (TSH). When treated with anti-thyroid drug, his thyroid hormone levels normalized but TSH increased, suggesting thyroid resistance.

CASE

We present a case of an 18-year-old male who was under paediatric follow-up since infancy. His mother was diagnosed with hyperthyroidism during her third pregnancy, and she underwent radioactive ablation after delivery. His initial cord T4 was 124 nmo/L (124-244 nmol/L), and subsequent serial thyroid function tests revealed persistently high free T4 (FT4), so he was started on propylthiouracil at the age of 1 year and 8 months. After starting an antithyroid medication, his TSH became elevated while his FT4 returned to normal. His TSH returned to normal and FT4 increased after discontinuing the anti-thyroid medication. Even with elevated FT4 and non-suppressed TSH, he remained euthyroid. Clinically, there was no goitre. All systemic examinations, including his mental development and learning, were normal. His thyroglobulin antibody and thyroid microsomal antibody were positive. Neck ultrasound and TSH receptor antibody levels were both normal. Magnetic resonance imaging of the pituitary revealed no evidence of pituitary adenoma. Because the patient was asymptomatic, the decision was made to discontinue carbimazole. He remained asymptomatic despite having an FT4 in the upper range. Thyroid hormone resistance syndrome was eventually diagnosed. We had our limitations to further workup because the genetic test was not available in our country. He has not needed any antithyroid medication since then.

CONCLUSION

This case demonstrates not all hyperthyroidism must be treated with antithyroid medications. Early recognition could avoid unnecessary treatment.

EP_A072

THE SILENT ATTACK: PANCYTOPENIA AS AN ATYPICAL PRESENTATION OF HASHITOXICOSIS PHASE OF HASHIMOTO'S DISEASE

https://doi.org/10.15605/jafes.038.S2.90

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INTRODUCTION/BACKGROUND

Autoimmune thyroid disease (AITD) has been linked to cytopenia with hyperthyroidism causing pancytopenia, while hypothyroidism is linked with anaemia. However, pancytopenia rarely occurs in hypothyroidism. We present a case of alternating hyperthyroidism and hypothyroidism presenting initially with pancytopenia.

CASE

A 70-year-old female presented with prolonged fever for three weeks without any other symptoms. Physical examination was normal, but initial blood tests showed cytopenia of all cell lineage with no identified cause. Screening tests for malnutrition, infection, tuberculosis, and connective tissue disease were normal.

CECT Thorax Abdomen and Pelvis showed only multiple thyroid nodules. Thyroid function showed hyperthyroidism with TSH levels <0.01 ml/UL, fT4 28pmol/L, elevated anti-TG, and anti-TPO, while TSI was normal. Carbimazole 5 mg daily was initiated for hyperthyroidism. Two months later, the patient showed symptoms and signs of

hypothyroidism, TSH 57.10 ml/UL and fT4 2.72 pmol/L, consistent with diagnosis of Hashimoto's disease. Carbimazole was discontinued, L-thyroxine replacement initiated. Follow up period noted normalization of thyroid function, but poor recovery of pancytopenia under further evaluation and management by haematologist.

Hashimoto's disease can present with alternating hyperthyroidism known as hashitoxicosis phase, followed by hypothyroid state. Hyperthyroidism-induced pancytopenia is caused by decreased production of haemopoietic cells or increased destruction by immunological mechanisms or hypersplenism. However, the exact mechanism of hypothyroidism-induced pancytopenia is not well understood, but it is thought to be related to the immune system's dysregulation leading to a decrease in the lifespan of certain blood cells.

CONCLUSION

Hashimoto's disease needs to be considered as a differential diagnosis for pancytopenia. Resolution of pancytopenia is usually achieved after reaching a euthyroid state, but further haematological evaluation may be needed if the condition persists.

EP_A073

OVERT HYPOTHYROIDISM COMPLICATED BY STROKE IN A YOUNG PREGNANT WOMAN

https://doi.org/10.15605/jafes.038.S2.91

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INTRODUCTION/BACKGROUND

Overt hypothyroidism in pregnancy presenting with stroke is infrequent. Physiological changes during pregnancy have the potential of masking hypothyroidism, making its diagnosis challenging. Hypothyroidism in pregnancy augments the hypercoagulable state leading to stroke.

CASE

We are reporting a previously well 25-year-old female, who was gravida 2 para 1 at 9 weeks of gestation who presented with sudden onset of right sided body weakness, slurring of speech and headache. She was found to have overt hypothyroidism. She had weight gain of 8 kg for 2 months associated with constipation. At presentation, she had a brief loss of consciousness followed by slurred speech and numbness over the right limb. Her GCS was E4V5M6 with blood pressure of 144/96 mmHg, regular pulse rate of 80 beats per minute and normal temperature. She has no goitre, coarse hair, and pitting oedema. National Institutes of Health (NIH) stroke scale was 9/42. MRI revealed left basal ganglia infarct with large vessel occlusion at M1, while the cerebral angiogram shown left M1 occlusion. As thrombolysis was contraindicated because of pregnancy, thrombectomy was attempted. Left M1 occlusion was recanalized but residual clot persisted at superior branch of left M1. Autoimmune work-up was negative. Additional laboratory work-up for young stroke, revealed significantly abnormal thyroid function tests with T4: 7 pmol/L (9-19 pmol/L) and TSH of 14.87 mIU/L (normal value: 0.35-4.94 mIU/L). With positive antithyroglobulin (TG) antibodies of 1189.4 IU/mL (normal value: <4.11 IU/ml) and anti- thyroperoxidase (TPO) antibodies of 1158.4 IU/ml (normal value: <5.61 IU/ml), a diagnosis of hypothyroidism secondary to Hashimoto's disease was made. She was given thyroxine replacement.

CONCLUSION

In conclusion, overt hypothyroidism in pregnancy is associated with an increased risk of stroke due to changes in lipid metabolism, inflammation, and blood coagulation. Pregnant women with hypothyroidism should receive appropriate management to reduce their risk of stroke.

EP_A074

ACUTE MYOCARDIAL INFARCTION MASKING THYROTOXICOSIS IN PREGNANCY

https://doi.org/10.15605/jafes.038.S2.92

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INTRODUCTION/BACKGROUND

Myocardial infarction in pregnancy is a rare incidence with cases ranging from 1 to 10 per 100 000 deliveries and only 2% are caused by vasospasm. A direct explanation or mechanism as to how hyperthyroidism can cause acute myocardial infarction is not fully understood, but a few mechanisms has been proposed and one of it is related to coronary artery vasospasm.

CASE

We report a case of a 28-year-old female with twin pregnancy at 11 weeks of gestation, presenting with severe left sided chest pain radiating to left upper limb. She had no previous thyroid disease and was clinically euthyroid. Blood pressure was 119/76 with pulse rate 100 beats per minute at presentation.