

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.

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OVERT HYPOTHYROIDISM COMPLICATED BY STROKE IN A YOUNG PREGNANT WOMAN

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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.

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ACUTE MYOCARDIAL INFARCTION MASKING THYROTOXICOSIS IN PREGNANCY

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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.