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

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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

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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.

Electrocardiography done showed ST elevation at inferior lead with lateral involvement. CKMB done was 724 u/L (< 24). Thrombolysis achieved no resolution of ST elevation post thrombolysis. Echocardiography shows basal posterior dyskinesia. Coronary angiography revealed normal coronary arteries. Thyroid function test (TFT) reveals TSH <0.005 mIU/L (0.4 – 4.0), free T4 (fT4) 58.31 pmol/L (7.8 – 14.4) and T3 16.73 pmol/L. TSH-receptor antibody <0.80 iu/L (<1.75). The liver enzymes were deranged with AST 833 u/L (<75) and ALT 185 u/L (<45). She was diagnosed as Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) with thyroxicosis. She was started on Lugol's solution in the ward. Prior to discharge, her repeat TSH was <0.005 mIU/L and fT4 14 pmol/L after 5 days of Lugol's. She was discharged with Carbimazole 15 mg daily which was stopped at 19 weeks as TFTs normalized.

CONCLUSION

Thyrotoxicosis can have a variety of presentations and AMI is one manifestation. It should be considered in a patient presenting with acute MI who do not fit the usual demography and has no obvious risk factors for coronary artery disease.

EP_A075

SEEING BLUES ALL AROUND: A CASE OF PROPYLTHIOURACIL-INDUCED CYANOPSIA

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INTRODUCTION/BACKGROUND

Cyanopsia is a subjective symptom characterized by a bluish appearance of the overall visual field and has been reported among patients taking phosphodiesterase-5 inhibitors. Propylthiouracil (PTU) is a member of the thiouracil group and widely used for the treatment of thyrotoxicosis. Despite being associated with various side effects, such as hepatitis, agranulocytosis, body rash, and antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis, PTU-related cyanopsia has not been reported.

CASE

In this report, we describe a case of PTU-induced cyanopsia, including the results of biochemical laboratory tests. We also discuss treatment strategies and include literature review.

A 33-year-old female presented with palpitations and tremors. Clinically, she appeared anxious with hand tremor and diffuse goiter. Her pulse was regular, and other systemic examinations were unremarkable. Thyroid function tests (TFTs) showed overt hyperthyroidism with suppressed thyroid-stimulating hormone (TSH) levels (<0.008 miu/L) and high free T4 levels (55.9 pmol/L). She was treated for Grave's disease with carbimazole but developed significant urticaria. PTU was then introduced, but she developed a bluish appearance of her surrounding vision right after the second dose. The symptoms subsided after one day of discontinuing PTU. Re-challenging with PTU at a lower dose also resulted in a similar effect. Eye assessment by ophthalmology was normal. She was then given propranolol and cholestyramine to control her thyroid status. Definitive treatment with radioactive iodine will be administered once her thyroid function improves.

PTU is one of the mainstay oral medications for hyperthyroidism and is generally well-tolerated. However, PTU-induced cyanopsia may limit oral treatment options, although the phenomenon appears reversible after stopping medication, regardless of the dose. It may cause significant distress and lead to discontinuation of treatment.

CONCLUSION

We report here, the first known case of PTU-induced cyanopsia.

EP_A076

MYXEDEMA ASCITES: A RARE INITIAL PRESENTATION OF HASHIMOTO THYROIDITIS

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INTRODUCTION/BACKGROUND

Ascites due to hypothyroidism is rare and only occurs in less than 4% of cases. Here, we present a case of severe hypothyroidism due to Hashimoto's thyroiditis, where the patient's initial presentation was gross ascites.

CASE

A 45-year- old male who has hypertension, presented with worsening abdominal distension for 1 month. Examination showed gross ascites and bilateral lower limb oedema with no other stigmata of chronic liver disease. Peritoneal fluid Serum-Ascites Albumin Gradient (SAAG) was 0.5 g/dL, suggesting a non-portal hypertension cause of ascites with high protein level of 2.9 g/dL and presence of lymphocytes count of 30 cell/mm^3. Peritoneal fluid examination, imaging and endoscopy findings excluded the usual causes of ascites. Patient showed no response