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

to initial treatment with diuretics and required multiple abdominal paracentesis. Echocardiogram showed presence of loculated pericardial effusion at posterior wall measuring 1.15 - 1.44 cm. Thyroid function test (TFT) was then done, showed severe hypothyroidism (Free T4 <5.41 pmol/L and TSH 89.71 mIU/L) secondary to Hashimoto's thyroiditis (anti-TPO 205 IU/mL). Patient was started on L-Thyroxine 150 mcg OD. There was significant resolution of ascites with normalisation of TFT.

It was postulated that low level of T3, increases level of Hyaluronic acid (HA), HA then induces capillary leak which results in fluid accumulation. Literature suggests that in patients with ascites who have SAAG less than 1.1 g/dL, high protein level (>2.5 g/dL), and predominant cell count of lymphocytes, hypothyroidism should be suspected. Early suspicion of hypothyroidism prevents patients from undergoing unnecessary procedures while its treatment provides resolution of ascites.

CONCLUSION

Hypothyroidism should be considered in patients with unexplained cause of ascites.

EP_A077

A RARE PRESENTATION OF SYMPTOMATIC COMPLETE HEART BLOCK IN A MILDLY HYPERTHYROID PATIENT

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INTRODUCTION/BACKGROUND

Hyperthyroidism mainly causes sinus tachycardia and atrial fibrillation. Complete heart block is an extremely rare complication of hyperthyroidism with very few cases reported, mainly in association with acute inflammatory disease, hypercalcemia, administration of drugs, or structural heart disease.

CASE

Here, we report a case of a 62-year-old male with underlying DM, hypertension and cerebrovascular accident. He was brought in for syncopal attack with lethargy, profuse sweating, and dizziness. He denied any history of fever or anginal chest pain. Upon arrival BP was 151/49 mmHg, with heart rate of 27-34 bpm. Systemic examination was unremarkable; there was no goitre or thyroid eye sign present. Serial ECG revealed complete heart block and he was initially managed with intra- venous infusion (IVI) of adrenaline and dopamine, IV atropine boluses and followed by transvenous pacemaker (TPM) insertion. Laboratory investigation including FBC, RP, electrolytes, liver enzymes, and cardiac enzymes were all within normal range.

However, thyroid function test showed mild hyperthyroidism with free T4 of 16.4 pmol/l (7.86-14.41), and TSH 0.115 mIU/L (0.38-5.33). TSH receptor antibody was negative. Echocardiography demonstrated good left ventricular systolic function with ejection fraction of 55%, and no regional wall motion abnormalities which made an ischemic aetiology as unlikely. We commenced carbimazole at a dose of 10 mg daily. Subsequently permanent pacemaker was inserted due to dependency on TPM. He had an uneventful recovery and was discharged well.

CONCLUSION

The exact mechanism of complete atrioventricular (AV) block remains unclear. Few reports postulated that interstitial inflammation of the AV node and His-bundle or focal myocarditis around the AV node could have led to cumulative damage to the cardiac conduction system. This case reiterates the importance of recognizing the association between complete AV block and hyperthyroidism due to the rarity of this condition. There is still insufficient information regarding the optimal management of this condition.

EP_A078

THE INNOCENT CARBIMAZOLE

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INTRODUCTION/BACKGROUND

Agranulocytosis is a life-threatening condition with mortality rate of 21.5% seen in 0.30.6% of patients taking thionamides. However, thionamides may not be the only culprit and other aetiologies should be considered.

CASE

We describe here a 29-year-old male with Grave's disease diagnosed since 2017 who was recently restarted on high dose carbimazole after a period of non-adherence leading to raised Free T4 53.7 pmol/L [13.1 - 21.3] with