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.

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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