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

suppressed TSH. He presented with fever, sore throat, and malaise. Blood investigations revealed leukopenia with agranulocytosis and thrombocytopenia - total white cells of 1.81 x 10^3/ul, neutrophil count of 0.65 x 10^3/ul and platelet of 110 x 10^3/ul. Renal and liver profile were normal, but his lactate dehydrogenase was elevated at 716 U/L [135 - 225].

He was admitted with the initial suspicion of carbimazole-induced agranulocytosis. Hence, carbimazole was withheld and treatment with cholestyramine 16 g/day, broad spectrum antibiotics and subcutaneous Neupogen were commenced. Oral lithium was later added but he developed generalized maculopapular rash.

However, as physical examination revealed generalised lymphadenopathy, other differential diagnoses were also pursued. Finally histopathological examination of the excisional biopsy of the right inguinal lymph node showed necrotising histiocytic lymphadenitis consistent with Kikuchi-Fujimoto disease. Anti-nuclear antibody was negative and complements levels were normal. The rheumatology team initiated oral prednisolone and this was followed by prompt recovery of blood counts (total white cells 9.29 x 10^3/ul, neutrophil 3.39 x 10^3/ul and platelet 196 x 10^3/ul) a week later. He was restarted on carbimazole to render him euthyroid before definitive thyroidectomy.

CONCLUSION

This case describes a rare case of Kikuchi-Fujimoto disease and highlights the importance of considering and pursuing other aetiologies of agranulocytosis especially in a patient who has been on and off carbimazole for years.

EP_A079

SEVERE MARROW APLASIA SECONDARY TO CARBIMAZOLE

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INTRODUCTION/BACKGROUND

One of the adverse effects of thionamide therapy for Graves' disease is agranulocytosis. Generally, agranulocytosis recovers spontaneously after withdrawal of thionamide or with short course of granulocyte colony-stimulating factor (G-CSF).

CASE

We report a case of Graves' disease presenting with delayed recovery of severe agranulocytosis after treatment with Carbimazole.

A 26-year-old female diagnosed with Graves' disease with high antibody titre presented with fever and sore throat after one month treatment with carbimazole 30 mg daily. She was treated as neutropenic sepsis with severe agranulocytosis. The baseline absolute neutrophil count was 0.01 x 10^9/L (3.929-7.147). She was started on G-CSF and broad-spectrum antibiotics including Piperacillin/Tazobactam and subsequently escalated to Meropenem. Her thyrotoxicosis was treated with lithium, prednisolone, and cholestyramine. Haematology team was also consulted in view of delayed recovery of severe aplasia and she was prepared for possible bone marrow transplant. The patient's neutrophil counts recovered only after seven days of G-CSF treatment.

It was later observed that she was not responding to treatment after two months of optimized dose of lithium, prednisolone, and cholestyramine. Hence, the patient was planned for semi-urgent total thyroidectomy. During admission for surgery, her fT4 level was 58.9 pmol/l (7.88 - 14.41). She required 3 cycles of plasma exchange and Lugol's iodine prior to thyroidectomy as part of pre-operative optimization. She underwent total thyroidectomy with fT4 level of 33.5 pmol/l. The surgery was successful with transient hypocalcaemia postoperatively.

CONCLUSION

This case showed a rare incident of delayed recovery of severe marrow aplasia secondary to Carbimazole. In view of resistance to second line thyrotoxicosis treatment, the patient underwent semi-urgent total thyroidectomy with plasma exchange prior to surgery.

EP_A080

SUCCESSFUL TREATMENT OF HYPOTHYROIDISM WITH RECTAL LEVOTHYROXINE: A CASE REPORT

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INTRODUCTION/BACKGROUND

Appropriate hormone replacement therapy is the cornerstone of management and is typically in the form of oral levothyroxine. The aim of this case report is to describe an alternative route when the oral and parenteral routes are not available.