heart rate along with his temperature normalized. He was eventually weaned off inotropic support. Repeat TFTs after 6 days showed T4 of 11.1 pmol/L and TSH of 9 m IU/L.

CONCLUSION

In this case, the presence of TB meningoencephalitis obscured the diagnosis of severe hypothyroidism, resulting in treatment delay. In cases with high clinical suspicion of myxoedema coma, stress doses of hydrocortisone and thyroxine replacement are vital even prior to laboratory confirmation to enhance survival.

EP_A163

CLINICAL AUDIT ON REFLEX-FREE T3 TESTING AT HOSPITAL PUTRAJAYA, MALAYSIA

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INTRODUCTION

Reflex-free T3 (fT3) testing has long been used to optimize the use of laboratory tests in Hospital Putrajaya. It involves the automatic addition of fT3 reflexively when the TSH level is below the normal range and free T4 (fT4) is within normal limits. Excessive reflex testing can lead to an added economic burden. The objective of this audit was to determine the usefulness of reflex fT3 testing using different TSH cut-offs.

METHODOLOGY

Previously, fT3 was performed automatically when TSH was below the normal limits (<0.38 m IU/L) with normal fT4 (7.9 to 14.4 pmol/L). A new workflow was implemented in March where reflex fT3 was only done when TSH is <0.1 m IU/L with normal fT4. This reflex testing is only applied to adults above 18 years old. Patients who underwent reflex fT3 testing three weeks before (Group 1) and after (Group 2) implementation of the new workflow were identified. Patients who would have had reflex fT3 testing with the old workflow but not in the new workflow (TSH 0.1 - 0.37 m IU/L with normal fT4) were also identified (Group 3). Data on patient characteristics were retrospectively collected and analysed.

RESULT

There were 105 patients in Group 1, 66 in Group 2 and 41 in Group 3. The new TSH cut-off of <0.1 resulted in a 38% reduction in reflex fT3 testing. Only 9 (4.25%) out of the 212 patients in the 3 groups had clinical necessity for fT3 testing. The fT3 result changed management in only 6 cases. The other 3 cases were planned for follow-up with repeat tests as clinically euthyroid.

CONCLUSION

Reflex fT3 testing was unnecessary in a large number of cases. The usefulness of reflex fT3 testing in this cohort was very low. Hence, reflex fT3 testing is being discontinued at our centre. Further evaluation is needed to determine strategies that can optimise the ordering of fT3 tests.

EP_A164

WHEN THIONAMIDES ARE CONTRAINDICATED: OUTCOME OF CHOLESTYRAMINE THERAPY IN HYPERTHYROID PATIENTS: A SINGLE TERTIARY CENTRE EXPERIENCE

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INTRODUCTION

Cholestyramine, a bile acid sequestrant, binds to thyroid hormones in the intestine and enhances their clearance. Thionamides are the mainstay in the treatment of hyperthyroidism, however, this may not be an option in the presence of profound hepatitis and agranulocytosis. Here, we aim to assess the efficacy and tolerability of cholestyramine therapy in patients with hyperthyroidism where thionamides are contraindicated.

METHODOLOGY

A one-year retrospective review of patients with hyperthyroidism who were treated with cholestyramine was performed from April 2023 to April 2024.

RESULT

A total of 10 patient medical records (8 females and 2 males) were reviewed. The mean age was 51.7 years old and the median duration of hyperthyroidism was 7.5 years. Graves' disease was the underlying aetiology in 7 cases, and the rest was a toxic multinodular goitre. Six of our patients already had atrial fibrillation, with four of them

having cardiomyopathy. The reason for conversion from carbimazole to cholestyramine was transaminitis for one patient, and the remaining was due to neutropenia and thrombocytopenia. Seven patients (70%) received Lugol's iodine for not more than 10 days, relying on its Wolff-Chaikoff effect. One patient received prednisolone as an adjunct therapy for hyperthyroidism. The total daily dose of cholestyramine commenced was 12 g given in TDS dosing for a median duration of 1.4 months. Median FT4 level pre- and post-cholestyramine therapy were 50.2 pmol/L and 25.5 pmol/L respectively (NR 7.86-14.41), $p = 0.028$. The median TSH level was <0.005 m IU/L. We were able to rechallenge six patients (60%) with carbimazole as they showed an improvement in their laboratory parameters. Only two patients underwent subsequent definitive therapy with RAI and thyroidectomy. None of our patients developed any adverse side effects from cholestyramine.

CONCLUSION

Our experience demonstrated that in selected cases, cholestyramine may be used as an effective and well-tolerated therapy when first-line options are contraindicated.

EP_A165

THYROTOXIC CARDIOMYOPATHY COMPLICATED BY FULMINANT HEPATIC FAILURE: A CASE REPORT

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INTRODUCTION/BACKGROUND

Thyrotoxic cardiomyopathy with cardiac failure can lead to liver congestion and ischaemic hepatitis. Fulminant hepatic failure secondary to thyrotoxic cardiomyopathy is rare.

CASE

We report a 45-year-old woman with strong family history of hyperthyroidism. She presented with palpitations and cardiac failure symptoms for a month. Electrocardiograph showed atrial fibrillation. Echocardiogram revealed a preserved ejection fraction (55%), mid-septal wall hypokinesia, severe mitral and tricuspid regurgitation, with pulmonary hypertension. She had an elevated free T4 (fT4) level of 16.4 pmol/L (7.86-14.41 pmol/L) and free T3 (fT3) level of 7.6 pmol/L (3.10-6.80 pmol/L). TSH receptor antibody was elevated 13.7 IU/L (<1.75 IU/L) consistent with Graves' Disease. She was treated for thyroid storm and initiated on an anticoagulant. She was discharged with carbimazole 30 mg OD and bisoprolol 2.5 mg OD.

After 10 days, she returned with worsening cardiac failure, high-grade fever and jaundice. Upon admission, the fT4 level was 12 pmol/L. Her liver transaminases were normal except for hyperbilirubinemia secondary to liver congestion. Subsequently, transaminases showed rapid progression of liver failure with peak aspartate aminotransferase (AST) of more than 10,000 U/L, total bilirubin of 481.3 umol/L (5.0-21.0 umol/L), and severe coagulopathy. She required mechanical ventilation due to hepatic encephalopathy. Ultrasonography of the hepatobiliary system showed cholelithiasis with acute cholecystitis. Budd-Chiari Syndrome was ruled out since the hepatic veins were patent. Viral hepatitis was likewise ruled out. She was managed with N-acetylcysteine, diuretics, and second-line anti-thyroid treatment (cholestyramine, hydrocortisone, and Lugol's solution). Her sepsis responded to intravenous meropenem. She was not suitable for liver transplantation due to multi-organ failure after consulting the hepatology team.

CONCLUSION

A comprehensive approach involving cardiac evaluation with echocardiogram, assessment of liver dysfunction, and consideration of autoimmune causes of liver failure is crucial in the management of patients with thyrotoxicosis and liver failure. Liver transplant is an option in the management of thyrotoxicosis with fulminant liver failure.

EP_A166

T3 THYROTOXICOSIS IN A PATIENT WITH METASTATIC FOLLICULAR THYROID CARCINOMA

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INTRODUCTION/BACKGROUND

Differentiated thyroid cancers are usually associated with normal thyroid function. Rarely, thyrotoxicosis can develop due to functioning metastatic thyroid carcinoma. We present a case of a male with metastatic follicular thyroid cancer associated with T3 thyrotoxicosis.

CASE

A 57-year-old male with underlying multinodular goitre presented with rapidly enlarging neck swelling, heat intolerance, loose stools, weight loss, and left shoulder pain over three months' duration. He exhibited a huge left goitre with right tracheal deviation. Laboratory tests revealed