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.

CASE
Our patient was a 73-year-old male diagnosed with recurrent transglottic squamous cell carcinoma (T2N0M0) and had total laryngectomy, total thyroidectomy and bilateral neck dissection done. Oral thyroxine replacement 100 mcg (1.3 mcg/kg/day) daily was started 3 days post operatively. Patient's post operative recovery was complicated with neck wound breakdown with suspicion of pharyngo-cutaneous fistula and was subsequently started on total parental nutrition by day 12 post operation. Patient was referred to endocrine team due to worsening hypothyroidism FT4 7.68 pmol/L (12.0-22.0) TSH 12.8 mIU/L (0.27-4.20). On examination, patient was conscious, alert, GCS full, BP 127/75 mmHg, pulse rate 86 beats per minute, on vacuum dressing over neck wound, reflexes normal, clinically asymptomatic of hypothyroidism. Patient was converted to per rectal administration of levothyroxine. Levothyroxine tablet was crushed and mix with 10 mls of water and 20 mls of lignocaine gel, pushed into rectum with nasogastric tube.

Patient was initially started on 2.6 mcg/kg/day per rectal thyroxine and subsequently increased to 4 mcg/kg/day 5 days later due to the lack of adequate biochemical response. Thyroid function normalized after 3 weeks of therapy, with FT4 18.4 pmol/L (12.0-22.0) TSH 2.80 mIU/L (0.27-4.20).

CONCLUSION
In conclusion, per rectal administration of levothyroxine can be a useful, safe, and effective alternative to oral levothyroxine in conditions precluding oral administration. We should advocate for increased availability of rectal levothyroxine preparations worldwide.

EP_A081

A CASE OF TRIPTORELIN-INDUCED THYROIDITIS

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INTRODUCTION/BACKGROUND
Gonadotrophin-releasing hormone agonists (GnRHa) therapy has been associated with thyroid dysfunction, including thyroiditis. Triptorelin, a GnRHa, is used as androgen deprivation therapy (ADT) in men with prostate cancer. We present a case of triptorelin-induced thyroiditis in a patient with locally advanced prostate cancer.

CASE
An 83-year-old male with underlying stable prostate cancer presented with an acute transient episode of abnormal behaviour. After completed radiotherapy, he was on 3-monthly SC leuprorelin before recently changing to 3-monthly SC triptorelin. He had received the second dose of SC triptorelin 4 weeks prior to presentation. A plain brain CT ruled out a space-occupying lesion. His renal profile, serum calcium, glucose and dementia workup were normal, except for a deranged TFT [suppressed TSH (0.01 mIU/L, N:0.55-4.78), high fT4 (55.1 pmolL, N:11.5-22.7), high fT3 (13.0 pmol/L, N:3.5-6.5)]. Baseline TFT taken two years ago was normal. He had no fever, neck pain, dysphagia, respiratory or thyrotoxicosis symptoms. He denied any family history of thyroid disease, recent vaccination, or supplement use, including biotin. He was clinically euthyroid, and there was no evidence of Graves' ophthalmopathy, tremor, atrial fibrillation, or a goiter. Serum anti-TPO antibody was raised (67.2 iu/mL, N<35.0). Lumbar puncture findings were normal and the cerebrospinal fluid anti-TPO antibody was not detected. Technetium-99m uptake scan reported reduced uptake in both thyroid lobes, suggestive of thyroiditis. Hence, a diagnosis of triptorelin-induced thyroiditis was made. Upon discharge, his behaviour normalized and his TFT improved (TSH 0.02 mIU/L, fT4 32.3 pmol/L, fT3 6.7 pmol/L) without antithyroid drugs or glucocorticoids. During clinic review two months after he completed ADT, he was clinically euthyroid and his TFT had normalized (TSH 1.96m IU/L, fT4 12.0 pmol/L).

CONCLUSION
Patients with thyroid autoimmunity are more susceptible to thyroid dysfunction after GnRHa administration, probably due to GnRHa immunostimulatory actions, emphasizing need for TFT monitoring during GnRHa treatment. Those with persistent thyroid dysfunction after discontinuation of GnRHa therapy may require treatment.

EP_A082

ONE-YEAR AUDIT OF PATIENTS ADMITTED WITH THYROID DISORDERS TO THE MEDICAL DEPARTMENT, SIBU HOSPITAL

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INTRODUCTION
The prevalence of thyroid disorders in Malaysia is around 3.4%. It is the second most common endocrine disorder after diabetes. Organ dysfunction related to thyroid disorders such as hyperthyroidism and hypothyroidism may result in significant morbidity and mortality.