drug-induced destructive thyroiditis. AIT can be difficult to treat because of the long half-life of amiodarone. We describe a patient who developed AIT 15 months following cessation of amiodarone.

CASE

A 58-year-old male presented with sudden onset palpitations and hand tremors. He had history of ventricular tachycardia, for which he had undergone Implantable Cardiac Device (ICD) insertion. He had been on amiodarone for two years, from September 2021 to February 2023, following which the medication was discontinued. At the time of admission, amiodarone had been stopped for 15 months. On examination, he was tachycardic with heart rate of 150/min. The electrocardiogram (ECG) showed sinus tachycardia with spontaneous ICD shock. Thyroid function test indicated TSH <0.08 m IU/L (0.35-4.95), FT3 11.8 pmol/L (2.9-4.9), and FT4 >64 m IU/L (9.01-19.05). T4/T3 ratio was >4. Considering the patient's history of amiodarone use, a diagnosis of AIT was established. The patient was prescribed carbimazole 30 mg daily and fT4 remained >64 m IU/L after 1 week. Prednisolone 40 mg daily was added to treat mixed AIT 1 and 2. Thyroid ultrasonography revealed heterogeneous echogenicity in both thyroid lobes, with no focal lesion. Doppler study demonstrated a normal pattern of vascularity. The thyroid peroxidase antibody was 185 IU/L (<35) and the patient is currently awaiting thyroid scintigraphy.

CONCLUSION

The differentiation between type 1 and type 2 AIT can be challenging and indistinct, and some patients exhibit mixed forms of AIT. In such cases, combination therapies are often employed. It is critical to emphasize the importance of maintaining a high index of suspicion for AIT, irrespective of treatment duration or the time elapsed after discontinuation of amiodarone, due to the prolonged half-life of the drug.

EP_A156

METASTATIC MEDULLARY THYROID CARCINOMA DESPITE PROPHYLACTIC TOTAL THYROIDECTOMY IN MULTIPLE ENDOCRINE NEOPLASIA (MEN) 2A

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INTRODUCTION/BACKGROUND

Medullary thyroid carcinoma (MTC) is a rare neuro-endocrine tumour originating from parafollicular cells. Seventy five percent of MTC occur in a sporadic form while 25% are hereditary and associated with Multiple Endocrine Neoplasia (MEN) type 2. As the age of onset of MTC and penetrance of MTC in MEN2 varies by subtype, prophylactic thyroidectomy is recommended for patients with the highest risk of pathogenic variants. We describe a patient with MEN2a with metastatic MTC 10 years following prophylactic thyroidectomy.

CASE

A 29-year-old female was diagnosed with MEN2a at the age of 15 years. The diagnosis was made through genetic screening after her mother was diagnosed with the same condition with RET proto-oncogene mutation. She underwent prophylactic thyroidectomy at 16 years old. Her tumour markers, calcitonin and carcinoembryonic antigen (CEA) were within the normal ranges. She had no loco-regional recurrence during serial follow-up. However, her tumour markers were noted to increase 10 years after surgery. CEA doubling time was 19 months indicating progressive disease. Markedly elevated serum calcitonin at 783 pg/ml (<7.6) indicated high tumour burden and likely metastases. Neck ultrasonography did not reveal any disease recurrence. Serial CT scan of the abdomen demonstrated multiple liver lesions suspicious of metastases. Ga-68 DOTATATE and F-18 FDG PET-CT scan showed multiple non-somatostatins nor FDG-avid liver lesions. There were FDG-avid cervical lymph nodes suspicious of nodal metastases. Histopathologic analysis of the liver biopsy specimen confirmed metastatic MTC. She is planning for a multi-targeted tyrosine kinase inhibitor as systemic therapy for metastatic disease.

CONCLUSION

Management of MTC in the context of MEN2a is challenging and advances in molecular diagnosis and risk stratification systems have led to better individualized treatment and follow-up strategies. Prophylactic thyroidectomy as early

as 6 months for the highest risk and before 5 years old for other high-risk codon mutations is crucial in reducing the risk of micro-metastases.

EP_A157

SIGHT-THREATENING ACTIVE GRAVES' OPHTHALMOPATHY WITH NEWLY DIAGNOSED HEPATITIS B

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INTRODUCTION/BACKGROUND

Graves' Ophthalmopathy (GO) is an orbital autoimmune disease that is the most frequent extrathyroidal expression of Graves' disease. Full-blown disease is associated with disfiguring features (exophthalmos, stare), inflammatory signs and symptoms, ocular dysfunction (diplopia), and rarely, visual loss due to compressive Dysthyroid Optic Neuropathy (DON). The prevalence of GO in Malaysia was 34.7%.

CASE

A 66-year-old Orang Asli female, active smoker with underlying type 2 diabetes, hypertension and dyslipidaemia presented to us in March 2024 with complaints of eyesore and redness for 3 months. In addition, she had photophobia, insomnia, and intermittent headache for 1 month. She had exophthalmos and DON with a clinical activity score (CAS) of 6. Her thyroid stimulating hormone (TSH) level was 40 IU/L. She was started on oral carbimazole 30 mg once a day. In view of sight-threatening DON, she was commenced on high-dose intravenous methylprednisolone 1 gm daily for 3 days, subsequent tapering dose of 500 mg weekly for 6 weeks, and then 250 mg weekly for another 6 weeks. However, her Hepatitis B surface antigen (HBsAg) was reactive and she had mild transaminitis which were relative contraindications for corticosteroid pulse therapy. She was co-managed with the Gastroenterology team and started on oral tenofovir 300 mg once daily. Post IV methylprednisolone, her eye signs and vision had clinical improvement. Her liver function tests remained stable.

CONCLUSION

This case highlights the challenges in managing severe sight-threatening active Graves' ophthalmopathy in a patient with newly diagnosed hepatitis B. Prompt treatment is crucial to prevent further deterioration of her eye condition.

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A LETHAL CASE OF SEVERE CARBIMAZOLE-INDUCED AGRANULOCYTOSIS

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INTRODUCTION/BACKGROUND

Agranulocytosis is a severe complication of carbimazole, the primary drug for treating hyperthyroidism. It is rare with an incidence rate of 0.3–0.6% and mortality rate of 21.5%. Onset may develop within 7 days of initiation of anti-thyroid drug therapy. This case report highlights the deleterious effect of carbimazole-induced agranulocytosis in an elderly female.

CASE

A 70-year-old female with newly diagnosed hyperthyroidism (baseline TSH: 0.002 m IU/L, free T4: 58 pmol/L) was initiated on carbimazole 30 mg once daily at a health clinic. After approximately one month on carbimazole, she developed fever, sore throat, and multiple oral ulcers. On examination, she exhibited a spiking temperature of 39.4°C, injected throat, multiple oral ulcers over the hard palate, tongue, and lower lip, and a diffuse goitre. She had leucopoenia with total white blood cell count of 1.0, with immeasurable absolute neutrophil count (ANC) and no blast cells. Repeat TSH was 0.003 m IU/L and fT4 was 39.55 pmol/L. Chest radiograph showed consolidation over the right lower lung zone. Initial treatment included intravenous piperacillin-tazobactam, subcutaneous granulocyte colony-stimulating factor (G-CSF) 300 mcg daily, cholestyramine, Lugol's iodine, and propranolol. Due to the deterioration in her clinical condition, we promptly escalated her antimicrobials to meropenem, micafungin and increased her G-CSF dosing to 300 mcg two times a day. Her ANC remained at 0.01-0.02 (10^9/L). Despite treatment escalation, she succumbed to severe sepsis after 8 days of admission.

CONCLUSION

The primary treatment for carbimazole-induced agranulocytosis involves discontinuing the offending drug and administering intravenous broad-spectrum antibiotics. G-CSF may be used to expedite haematological recovery. Clinical vigilance is crucial when initiating carbimazole, especially in high-risk patients such as the elderly and those receiving high doses initially, by conducting early repeat blood investigations. This approach enables early intervention to mitigate adverse outcomes and ensure a favourable prognosis.