normal FT4, elevated FT3, and suppressed TSH. Thyroid ultrasound and contrast computed tomography (CT) scan unveiled a large left hemithyroid mass with retrosternal extension and contralateral tracheal displacement. Metastatic lesions were observed in the lungs, pleura, left scapula, cervical, and mediastinal lymph nodes. Needle aspiration of the thyroid mass showed a follicular nodule, while biopsy of the left scapula confirmed metastatic follicular thyroid carcinoma. The patient underwent total thyroidectomy with left modified radical neck dissection. Histopathologic examination revealed widely invasive follicular thyroid carcinoma, with areas of transformation to anaplastic thyroid carcinoma.

CONCLUSION
The coexistence of T3 thyrotoxicosis and thyroid cancer, particularly the follicular subtype, is uncommon and warrants careful consideration in clinical practice.

EP_A167

PROPRANOLOL-INDUCED CARDIAC DECOMPENSATION IN THYROID STORM

https://doi.org/10.15605/jafes.039.S1.178

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INTRODUCTION/BACKGROUND
Propranolol is one of the preferred beta-blocking agents used in thyroid storm. It is highly lipid-soluble and effective in reducing T3 concentration up to 30% if given in high doses. However, only a few cases reported on the side effects of this drug, especially life- threatening complications in thyroid storm.

We reported 4 cases of propranolol-induced circulatory collapse in patients with thyro-cardiac disease who presented with thyroid storm between 2022- 2024.

CASE 1
A 28-year-old male diagnosed with Graves' disease developed thyroid storm with cardiac decompensation post-wound debridement. He received carbimazole 30 mg and propranolol 40 mg prior to surgery. The propranolol was withheld following the unfortunate event and he recovered after 3 days.

CASE 2
A 32-year-old female with Graves' disease presented with acute heart failure and tachyarrhythmia. She was initially normotensive on arrival; however, she developed circulatory collapse after receiving propranolol 40 mg. She was managed in the ICU before succumbing to her death due to severe cardiac decompensation.

CASE 3
A pregnant female at 34 weeks AOG presented with an impending thyroid storm and premature uterine contraction. She was normotensive and tachycardic on presentation. The condition was complicated by cardiogenic shock and acute heart failure right after propranolol 40 mg administration. She was placed on mechanical ventilation but had an intrauterine foetal loss.

CASE 4
A 43-year-old female presented with thyroid storm and unstable atrial fibrillation. She was intubated and received synchronized cardioversion at 150 J together with antithyroid and glucocorticoid drugs. Her condition worsened after she was given oral propranolol 20 mg and she eventually succumbed due to cardiac decompensation.

CONCLUSION
Long-acting beta-blockers should be used with caution in thyroid storms with pre-existing thyro-cardiac disease as they can potentially impede the compensatory mechanism and consequently cause hemodynamic instability.

EP_A168

A CHALLENGING CASE OF GRAVES' DISEASE WITH MYELODYSPLASTIC SYNDROME

https://doi.org/10.15605/jafes.039.S1.179

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INTRODUCTION/BACKGROUND
Graves' disease is an autoimmune condition where antibodies are produced against the thyrotropin (TSH) receptors on the thyroid gland. The condition can be associated with haematologic manifestations.

CASE
A 44-year-old male with underlying Graves' disease, Schizophrenia, Chronic Hepatitis B and Myelodysplastic Syndrome presented with a week's history of loose stools and vomiting. On examination, blood pressure was 115/78 mmHg and heart rate was 97 bpm. He had pallor, tremors, sweaty palms, and a small goitre. Thyroid function tests were: TSH <0.001 m IU/L (0.27-4.2), T4 21.9 pmol/L (12.0-22.0), T3 2.65 pmol/L (3.1-6.8). His complete blood count was: Hb 11.7 g/dl (13-17), WBC 3.52 x 10^9/L (4-10), ANC 1.68 x 10^9/L (2.0-7.0), Platelets 69 x 10^9/L (150-410).

He was started on a thionamide with close monitoring of blood counts. However, the thionamide was withheld in view of his reducing absolute neutrophil count. He was then treated with steroids, lithium and cholestyramine with no improvement in his thyroid function tests.

Hence, he was eventually given radioactive iodine. Graves' disease with myelodysplastic syndrome proves to be challenging for endocrinologists to treat. The probable underlying pathophysiology is that high blood levels of thyroid hormones can be toxic to bone marrow cells leading to an increase in functional activity of reticuloendothelial cells, causing insufficient hematopoietic cells. In one study, free T3 and T4 were noted to be higher with lower TSH in patients with myelodysplastic syndrome. In view of the difficulty of treating hyperthyroidism with anti-thyroid drugs, our patient was treated with radioiodine ablation.

CONCLUSION

In conclusion, managing Graves' disease in individuals with myelodysplastic syndrome requires detailed evaluation and monitoring.

EP_A169

MANAGING THYROTOXIC ATRIAL FIBRILLATION IN A BIOCHEMICALLY EUTHYROID PATIENT

https://doi.org/10.15605/jafes.039.S1.180

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INTRODUCTION/BACKGROUND

Hyperthyroidism induces cardiovascular changes like increased heart rate and atrial automaticity, leading to conditions such as atrial fibrillation and heart failure, contributing to higher mortality rates. Despite achieving euthyroidism with treatment, cardiovascular manifestations may persist, necessitating further investigation into factors associated with persistent atrial fibrillation to guide appropriate anticoagulation therapy.

CASE 1

A 66-year-old Malay male with high blood pressure, dyslipidaemia, and thyrotoxic atrial fibrillation (TAF) due to Graves' disease of 5 years duration. He had two failed radioactive iodine treatments and thyroid surgery. He had periodic palpitations, dyspnoea, and left chest pain. His ECG revealed rapid atrial fibrillation. He has uncontrolled elevated blood pressure. The thyroid function tests were normal (T4 = 14.21, TSH = 4.78). He was eventually referred to the cardiology team who recommended cardiac ablation.

CASE 2

A 34-year-old female with Graves' disease and atrial fibrillation (AF) despite taking bisoprolol, went to the emergency department due to frequent palpitations and dizziness. She did not have chest pain. Her ECG showed atrial fibrillation. She had normal thyroid function tests (T4 = 15.21, TSH = 3.56) with elevated troponin levels. She was treated for symptomatic AF. She was subsequently referred to cardiology for cardiac ablation.

CONCLUSION

Thyroid hormones affect cardiovascular function, predisposing hyperthyroid individuals to atrial fibrillation even after achieving euthyroidism. The thromboembolic risk in TAF is reduced by oral anticoagulants. Treatment for TAF involves antithyroid medications to restore euthyroidism together with rate and rhythm regulation. Wong et al., found an unexpected relationship between decreased free thyroxine levels and chronic atrial fibrillation. TAF has a high thromboembolic risk even after euthyroidism, requiring anticoagulants and ongoing monitoring to prevent recurrence. Sometimes ablation is recommended, especially for persistent AF. In conclusion, hyperthyroidism-related AF therapy requires collaboration between endocrine and cardiovascular specialists. Prompt diagnosis and personalised treatment can improve the prognosis and reduce complications.

EP_A170

A RARE CASE OF FUNCTIONAL METASTATIC FOLLICULAR THYROID CARCINOMA WITH EGGSHELL CALCIFICATION

https://doi.org/10.15605/jafes.039.S1.181

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INTRODUCTION/BACKGROUND

Only a few cases of follicular thyroid carcinoma (FTC) with eggshell (or rim-like peripheral) calcification have been reported. Here, we report a rare case of functional metastatic FTC with eggshell calcification.

CASE

A 57-year-old female presented with progressive neck enlargement, dysphagia, and weight loss of 10 kg over 2 months. She also had a hoarse voice. On examination, she appeared thyrotoxic. She had a palpable 3 x 4 cm mass over the left neck, which was hard in consistency and immobile. Biochemically, she was hyperthyroid with suppressed TSH and high free T4 of 67.9 pmol/L (7.9-14.4). Her chest radiograph showed an eggshell calcification over the neck