goitre and fine tremors. Ultrasonography of the neck was done with the impression of subacute thyroiditis. These findings fulfilled the Japanese Thyroid Association (JTA) criteria for subacute thyroiditis. A Tc-99m Pertechnetate also confirmed the findings of thyroiditis. The patient was commenced on oral prednisolone 25 mg daily (0.5 kg/BW/day) with a tapering regimen over 2 weeks. He also received oral celecoxib 200 mg daily for 5 days and oral propranolol 20 mg daily. He gradually improved upon the 2-week clinic review and all medications were discontinued. Upon re-evaluation at 3 months, thyroid function tests normalized, and the thyroid ultrasound displayed the resolution of thyroiditis characteristics, accompanied by amelioration of all symptoms.

CONCLUSION

Subacute thyroiditis is an inflammatory thyroid condition characterized distinctly by painful enlargement of the thyroid. Transient hyperthyroidism is a hallmark of subacute thyroiditis where the inflamed thyroid gland releases unregulated excessive thyroid hormone into the bloodstream, leading to thyrotoxicosis symptoms. In this case, we treated our patient with a short-term 2-week combination of steroids + NSAIDs which showed non-inferior efficacy to the traditional long-term steroids (4-8 weeks).

EP_A150

HUMAN CHORIONIC GONADOTROPHIN (HCG) AND HYPERTHYROIDISM: RARE BUT PARALLEL CAUSE

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INTRODUCTION/BACKGROUND

Choriocarcinoma is a hCG-producing malignancy, with the beta subunit being structurally similar to TSH, allowing it to bind to TSH receptors on thyroid follicular cells and at high levels to elicit biochemical hyperthyroidism.

CASE

We report a case of choriocarcinoma-induced hyperthyroidism in a 29-year-old female. She was initially admitted for breathing difficulties and was found to have a posterior mediastinal mass, which was later histologically confirmed to be choriocarcinoma with B-HCG levels of 466,511 that peaked to 825,316. Thyroid functions measured shows TSH of 32.07, T4 of 0.009 with negative thyroid antibodies (Anti Thyroid Peroxidase <9, Anti Thyroglobulin 11.9). Ultrasonography showed bilateral thyroid nodules, ACTR TR3 (1.5 cm), however, staging CT did not pick up an overt goitre. Patient was started on carbimazole and planned for chemotherapy with thyroid function monitoring.

CONCLUSION

TSH and Beta-HCG are highly homologous and can cross-link to produce elevated thyroid hormone levels. The prevalence of hyperthyroidism in choriocarcinoma is not known; however, prolonged exposure to high HCG levels is required for it to occur. It is generally accepted that 25,000 IU/L of HCG is equivalent to 1 mU/L of TSH activity, with suggestions to measure thyroid function in patients with HCG >50,000 IU/L. Patients with symptomatic hyperthyroidism are treated with antithyroid drugs and the primary choriocarcinoma is treated with chemotherapy. Reduction or normalisation of the beta hCG levels quickly induces euthyroidism. Beta-HCG-induced hyperthyroidism is rare; however, with high levels found in choriocarcinoma, suspicion of concurrent hyperthyroidism should be raised. Patients with HCG-secreting tumours should be evaluated for hyperthyroidism and may benefit from treatment until the underlying cause is treated.

EP_A151

DOUBLE WHAMMY: CIRCULATORY COLLAPSE AND LIVER DYSFUNCTION IN THYROID STORM

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INTRODUCTION/BACKGROUND

Liver dysfunction is not an uncommon association in patients presenting with thyroid storm and could limit the treatment armamentarium. Circulatory collapse precipitated by the use of long-acting non-cardioselective beta-blockers in certain groups of patients can complicate the course of the disease.

CASE

We report three cases of thyroid storm with circulatory collapse and ischemic hepatitis complicating the use of beta-blockers and thionamides. All were females in their 40's. Two presented with rapid atrial fibrillation (ventricular rate 158- 196 per minute) and biventricular failure, and one with acute pulmonary oedema. All developed hypotension required inotropic support; two after beta-blocker and one after intubation. Their free T4 was 53.6 pmol/L to 74.3 pmol/L, Burch-Wartofsky scores were 60-95. All received ventilatory support and were treated for

sepsis. They received thionamides, glucocorticoid, Lugol's iodine, antiarrhythmic and one received cardioversion. All developed ischemic hepatitis with transaminases increased from the initial 2-7 x to 10-80 x upper limit of normal, and two had coagulopathy. Thionamide dose was reduced in two and withheld temporarily in one. Cholestyramine was added as an adjunct for all. All responded to therapy. Two were discharged with carbimazole and beta-blocker. Unfortunately, one succumbed despite initial improvement due to hospital-acquired infection.

CONCLUSION

Although beta blockers play an important role in the management of thyroid storm, caution should be exercised due to its potential life-threatening side effect especially in the presence of clinical or subclinical thyro-cardiac disease. Lugol's iodine and cholestyramine are useful adjuncts in the presence of severe liver dysfunction when choices of antithyroid drugs are limited.

EP_A152

A CHALLENGE IN MANAGING THYROID STORM WITH CONCURRENT PERFORATED GASTRIC ULCER PATIENT

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INTRODUCTION/BACKGROUND

Thyrotoxic crisis can be fatal if not treated promptly. Individuals with severe thyrotoxicosis who lack a functional gastrointestinal system present an uncommon but significant therapeutic challenge with a high fatality rate. We describe a case of Graves' disease in a thyroid storm with a concurrent perforated gastric ulcer.

CASE

A 36-year-old male presented to the emergency department with acute abdominal pain and vomiting. He also had heat intolerance and significant weight loss. He was borderline hypotensive, tachycardic with a regular pulse, and had generalized abdominal guarding. Chest radiograph showed air under the diaphragm. Thyroid function test (TFT) confirmed thyrotoxicosis with Thyroid Stimulating Hormone (TSH) <0.008 m IU/L and free T4 (FT4) 64.32 pmol/L. He was scheduled for an emergency laparotomy for a perforated gastric ulcer, thus contraindicated to taking anything by mouth. We gave him 200 mg of intravenous hydrocortisone. We did not administer an antithyroid drug because our centre did not have any intravenous or per-rectal antithyroid drug preparation. Post-operatively, his condition deteriorated, and he developed rapid atrial fibrillation which required inotropic support and synchronised cardioversion. After he was permitted to sip fluids for medication, he was started on oral propylthiouracil (PTU) and Lugol's iodine in addition to regular intravenous hydrocortisone. Following this, his general condition and thyrotoxic status improved. Upon discharge, he received oral carbimazole and propranolol.

In the 2-month follow-up, he was clinically euthyroid. His anti-thyroglobulin receptor antibody level was elevated, supporting the Graves' disease diagnosis. His TFT improved, with TSH <0.008 m IU/L and FT4 17.40 pmol/L. We further titrated down his oral carbimazole until the next appointment.

CONCLUSION

Managing a thyroid storm with concurrent perforated gastric ulcer is challenging due to limited antithyroid options other than oral medication and high mortality rates. The attending physician should collaborate with the surgical team to determine the optimal timing for oral antithyroid medication to manage the thyrotoxic crisis.

EP_A153

PITFALLS IN THE DIAGNOSIS OF AMIODARONE-INDUCED THYROTOXICOSIS: A CASE SERIES

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INTRODUCTION/BACKGROUND

One of the lesser known but unique mechanisms of action of amiodarone is its ability to inhibit the 5'deiodinase enzyme that converts T4 into the active T3. Inadvertently, this results in an elevated T4 and a lower T3 which suppresses pituitary TSH, giving the impression of a classic thyrotoxicosis. Only when this peripheral effect is overcome by the explosive release of T4 and T3 that they begin to manifest as amiodarone-induced thyrotoxicosis (AIT).