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).