A Grave Complication: Reactivated Thyrotoxicosis Manifesting as Diabetic Ketoacidosis

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OBJECTIVES

- Recognize risk factors for recurrence of Graves' thyrotoxicosis.
- Manage patients taking thionamide medication for suppression of Graves' Disease.

PATIENT BACKGROUND

- **ID:** 58 y/o M with Graves' Disease (on thyroid-sparing therapy) and Type 1 DM (on basal/bolus insulin)
- Year 2013:
  - Subclinical hyperthyroidism (TSH <0.02 w/o symptoms)
- Oct 2014:
  - Palpitations, anxiety, 25# weight loss in 1yr
  - Labile glycemic control
  - **Free T4: 2.1** ng/dL
  - Dx: clinical hyperthyroidism
  - **Started PO methimazole**
- May 2015:
  - No hyperthyroid symptoms
  - Incidentally found to have elevated thyroid-stimulating immunoglobulin (180% NL)
  - **Dx: Graves' Disease**
- Jul 2017:
  - Saw PCP for depressed mood & paresthesias.
  - **TSH 93.2** mIU/L
  - **Free T4: 0.3** ng/dL
  - Dx: clinical hypothyroidism
  - **Stopped methimazole**

PRESENTATION

- Aug 2017:
  - Came to the ED for 3 days of progressive nausea, emesis, anorexia, lethargy, and profuse sweating
  - No sick contacts or infectious symptoms
  - Reliably took usual doses of basal + correctional insulin even while feeling sick

INITIAL EVALUATION

Vitals: T 36.5C, **HR 117**, **BP 189/92**, RR 18

ECG: Sinus tachycardia

Exam: Alert, conversant, **anxious affect**.
Diaphoretic w/ mild tremor & endgaze nystagmus.
Heart, lungs, abdomen, & extremities WNL.

CBC: WBC 10.7 k/uL, Hgb 15.8 g/dL, Plt 137 k/uL

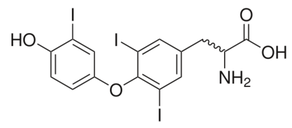
Chemistries:

CBG 437 mg/dL

Urine ketones 20 mg/dL
Anion gap 16 mg/dL
(baseline: 4-8 mg/dL)

Potassium 3.5 mg/dL
Bicarbonate 26 mg/dL

Endocrine labs:



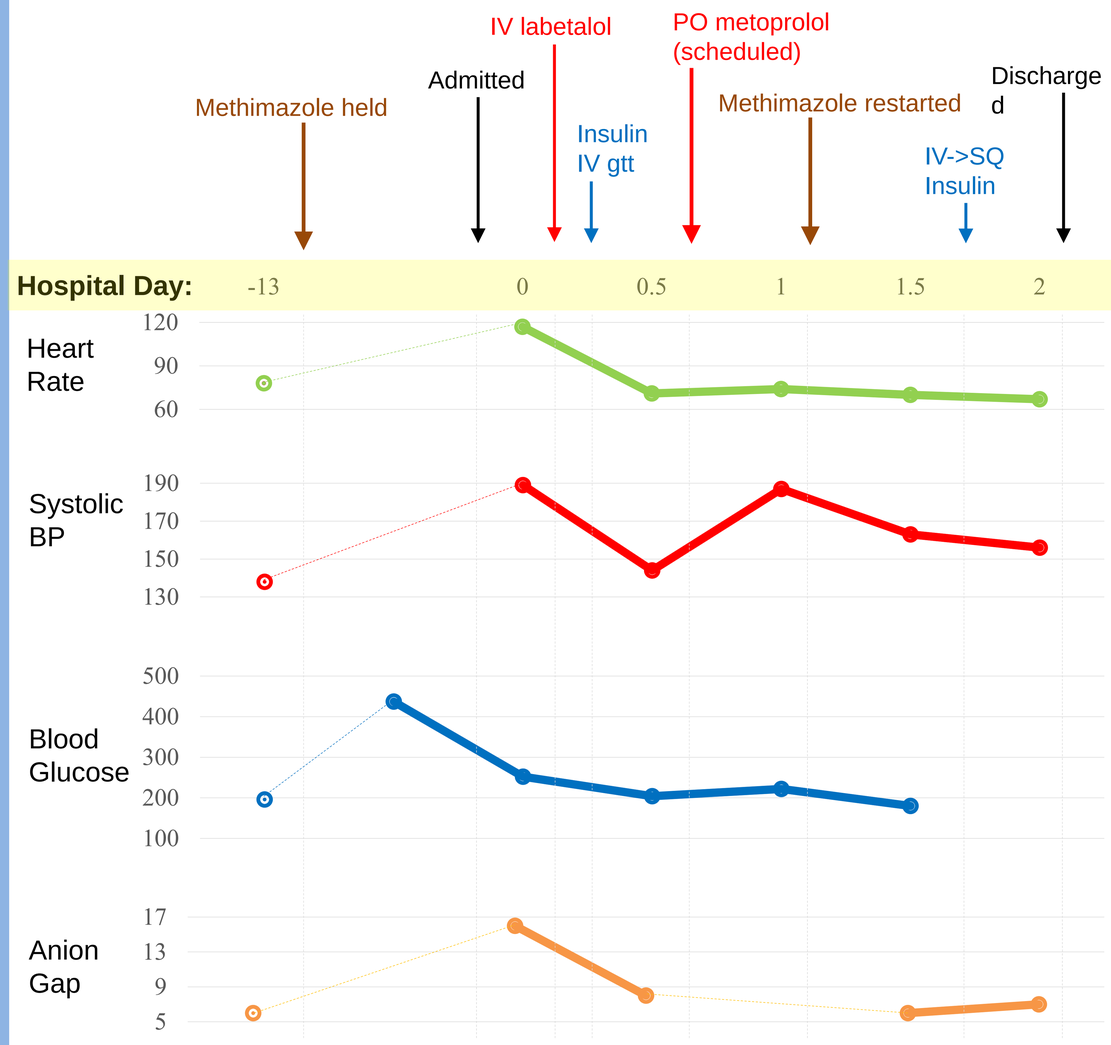
TSH 0.07 mIU/L

Free T4: 4.1 ng/dL

Total T3: 615 ng/dL
(ref: 87-196ng/dL)

HgbA1c 8.4%

CLINICAL COURSE



OVERVIEW OF GRAVES' DISEASE

- Autoimmune condition caused by antibodies against TSH receptor
- Diagnosis: clinical syndrome + elevated T3/T4 + elevated thyroid-stimulating immunoglobulin (TSI)
- Syndrome: thyrotoxicosis +/- ophthalmopathy, dermatopathy, comorbid autoimmunit(ies)
- Most often treated w/ radioactive iodine; however, patients can be managed using thionamide medication alone.
  - If thyroid tissue still present, can recur at unpredictable times
  - **Risk of recurrence** is positively associated w/ **circulating TSI titer**.
- Acute management of thyrotoxicosis should target multiple mechanisms:
  - Thyroid hormone-mediated sympathetic overdrive
  - Thyroid hormone synthesis, conversion, and release
- Diabetic patients w/ Graves' Disease can develop either **hyperglycemia** via adrenergic insulin resistance or **hypoglycemia** via autoimmune hyperinsulinism (Hirata's Disease).

LEARNING POINTS

- Clinical stability & hypothyroidism are poor markers of remission of Graves' Disease.
- In patients with medically-managed Graves' Disease, **check TSI levels** before discontinuing thyroid-suppressing medications.
- Thyrotoxic patients with Graves' Disease should receive both adrenergic blockade and thionamide medication (unless contraindicated).

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