A 73-year-old man is transported to the emergency department (ED) by ambulance for nausea, vomiting, diarrhea, and weakness of three days’ duration. Earlier today, he presented to his primary care provider with these symptoms and was found to be hypotensive; he was advised to go to the ED but instead went home against medical advice.

The patient’s medical history is significant for type 2 diabetes, stage 3b chronic kidney disease, dyslipidemia, hypertension, coronary artery disease, and benign prostatic hyperplasia. He has undergone stent placement and triple coronary artery bypass graft surgery. His medication list includes insulin glargine, glimepiride, liraglutide, atorvastatin, benazepril, carvedilol, amlodipine, clopidogrel, and tamsulosin.

Upon admission, the patient has a pulse of 98 beats/min; temperature, 98.2°F; respiratory rate, 18 breaths/min⁻¹; and PO₂, 98 mm Hg. An ECG, chest radiograph, and CT (without contrast) of the head, chest, and abdomen are all within normal limits. Lab evaluation is significant for severe thyrotoxicosis (see Table 1).

Endocrinology consult is requested. Further testing yields the following findings:

- **Thyroid-stimulating immunoglobulin:** 309% (reference range, < 30%)
- **Nuclear medicine thyroid scan with uptake:** 6-hour uptake of 70.3% (10%-25%) and 24-hour uptake, 81.8% (15%-35%)
- **Homogeneous radiotracer uptake within the thyroid gland:** no evidence of hot or cold nodules
- **Thyroid ultrasound:** bilateral enlarged heterogeneous gland and multiple sub-centimeter nodules (largest measuring 6 × 7 mm)

These results confirm a diagnosis of Graves’ disease. Treatment options, including antithyroid medications, radioactive iodine ablation (RAI), and surgery, are discussed. The patient is treated with RAI therapy (10 mCi) and discharged from the hospital.

Six days later, however, he returns to the ED with severe intermittent dizziness and lightheadedness of two hours’ duration, new-onset atrial fibrillation (A-fib), and mild shortness of breath. His vital signs include a pulse of 116 beats/min; temperature, 98.1°F; respiratory rate, 18 breaths/min⁻¹, blood pressure, 154/88 mm Hg; and PO₂, 100 mm Hg.

His lab values include:
- TSH < 0.005 uIU/mL
- Free T4, 8.01 ng/dL
- Free T3, 3.701 pg/dL
- eGFR, 60 mL/min/1.73 m²

Cardiology consult is requested. A pacemaker is placed for bradycardia-tachycardia syndrome, and the patient is put on rivaroxaban for stroke prevention.

The endocrinologist suspects post-RAI thyroiditis or ineffective RAI treatment. The patient is started on methimazole (10 mg bid), and his carvedilol is replaced with metoprolol (50 mg bid).
Two weeks postdischarge, the patient returns to the office. Although he says he’s doing better, he seems uneasy and agitated and has a pulse of 120 beats/min. His methimazole and metoprolol are increased (to 10 mg tid and 50 mg tid, respectively).

Another two weeks later, lab results still show elevated thyroid levels—now with increased enzyme levels on liver function testing. The patient reports worsening dizziness and shortness of breath. He is sent back to hospital and admitted for inpatient management, with urgent surgical consult for thyroidectomy. Total thyroidectomy is successfully performed, and the final pathology report shows a benign goiter.

**DISCUSSION**

Thyroid storm is an extreme form of thyrotoxicosis with an associated mortality rate of 8% to 25%.1 When thyroid hormone levels are elevated, adrenaline receptors are upregulated—but, while it is possible for persistent thyrotoxicosis to progress to thyroid storm on its own, a surge of adrenaline is usually needed. Most cases are triggered by acute stressors (ie, myocardial infarction, surgery, anesthesia, labor and delivery) in the context of underlying thyrotoxicosis.1

Diagnosis of thyroid storm is made clinically in patients who are thyrotoxic and present with systemic decompensation (ie, altered mental status, cardiovascular dysfunction, hyperpyrexia). Although no universally accepted criteria currently exist, the Burch-Wartofsky Point Scale (BWPS; see Table 2, page 24) can be used to assess disease severity and guide the extent of treatment and monitoring.2 However, this measure should not replace clinical judgment—the distinction between compensated thyrotoxicosis and decompensating thyrotoxicosis (thyroid storm) should be made by sound but prompt clinical assessment.

Once thyroid storm is suspected, aggressive treatment should be implemented to improve the systemic thyrotoxic state. Propylthiouracil (PTU) is preferred over methimazole, as it blocks T4 to T3 conversion in addition to blocking new hormone synthesis. Propranolol is the best choice of β-blocker because it also blocks T4 to T3 conversion and controls cardiac rhythm.

Iodine can rapidly block new hormone synthesis and release; it is often used to reduce thyroid hormone levels prior to emergency thyroid surgery. However, it should be given at least one hour after a dose of PTU. Hydrocortisone is given prophylactically for relative adrenal insufficiency (due to rapid cortisol clearance during thyrotoxic state); it may block T4 to T3 conversion as well. Volume resuscitation, respiratory care, temperature control (eg, antipyretics, cooling blankets), and nutritional support should also be incorporated, ideally in the intensive care unit (ICU). During or after thyroid storm management, treatment of the precipitating event/illness and of hyperthyroidism should be initiated to prevent recurrence.1

The patient’s initial BWPS was 30 (gastrointestinal [GI] score 10 + central ner-

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**TABLE 1**

<table>
<thead>
<tr>
<th>Lab test</th>
<th>Patient values (Reference range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TSH</td>
<td>&lt; 0.005 uIU/mL (0.358-3.740)</td>
</tr>
<tr>
<td>Free T4</td>
<td>6.48 ng/dL (0.76-1.46)</td>
</tr>
<tr>
<td>Free T3</td>
<td>1,497 pg/dL (218-398)</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>11.2 g/dL (14.0-18.0)</td>
</tr>
<tr>
<td>Hematocrit*</td>
<td>33% (40-51)</td>
</tr>
<tr>
<td>eGFR</td>
<td>39 (&gt; 60)</td>
</tr>
<tr>
<td>Glucose</td>
<td>180 mg/dL (82-115)</td>
</tr>
<tr>
<td>AST</td>
<td>94 U/L (15-37)</td>
</tr>
<tr>
<td>ALT</td>
<td>94 IU/L (16-61)</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>78 U/L (45-117)</td>
</tr>
<tr>
<td>Troponin I</td>
<td>0.10 ng/mL (0.00-0.50)</td>
</tr>
<tr>
<td>Urinalysis</td>
<td>WNL</td>
</tr>
</tbody>
</table>

* Complete blood count otherwise WNL.

Abbreviations: AST, aspartate aminotransferase; ALT, alanine aminotransferase; eGFR, estimated glomerular filtration rate; WNL, within normal limits.
vous system [CNS] score 10 + without precipitating factor 10), which put him in the “impending storm” category. At his second ED visit, his BWPS was 40 (cardiovascular score 10 + A-fib 10 + GI score 10 + CNS score 10 + precipitating factor [RAI ablation] score 0)—still in the “impending storm” category but certainly indicating a worsened state.

RAI for hyperthyroidism can transiently increase thyroid hormone levels due to inflammation of the gland. To prevent exacerbation of the thyrotoxic state, pretreatment with methimazole should be considered in patients with risk factors (eg, older age, cardiovascular complications, cerebrovascular disease, pulmonary disease, renal failure, infection, trauma, and poorly controlled diabetes). Patients should also be placed on β-blockers prior to treatment, in anticipation of a transient rise in thyroid hormone levels.

Due to this patient’s age, severity of thyrotoxicosis, and multiple risk factors, strong consideration should have been given to pretreating him with antithyroid medication and a β-blocker before definitive treatment was given. This would have potentially averted his subsequent hospital visits and urgent need for thyroidectomy.

**CONCLUSION**

Thyroid storm is an uncommon but serious medical condition with a high mortality rate. Prompt recognition and an aggressive multimodal treatment approach, ideally in the ICU, are paramount to stabilize patients and seek definitive treatment.

**REFERENCES**