THE CASE

A 22-year-old woman presented to the emergency department (ED) with a 24-hour history of nausea, vomiting, diarrhea, generalized abdominal pain, and mild headache. She denied shortness of breath, chest pain, or anxiety, and didn’t have a history of cardiac problems. The physical examination revealed tachycardia (heart rate, 135 beats/min) and a respiratory rate of 24 breaths per minute. The patient was diagnosed with dehydration and was given 3 liters of intravenous (IV) fluids. After fluid administration, her heart rate decreased to 94 beats/min and she was discharged home.

The patient returned to the ED later that same day with recurrent nausea, vomiting, and a mild fever. This time she reported a several week history of palpitations, heat intolerance, agitation, mild cognitive impairment, and difficulty sleeping. Her mother accompanied her to this visit and added that the patient had unintentionally lost 13 pounds over the past 2 weeks. The patient denied pain or enlargement in her neck, obstructive symptoms, hives, pruritus, or changes in vision. Reexamination revealed tachycardia (132 beats/min) with no murmurs, rubs, or gallops; increased respiratory rate (26 breaths/min); and diffuse thyromegaly without distinct nodules. The thyroid was nontender to palpation. The patient was also found to have a fine resting tremor, hyperactive deep tendon reflexes, and clonus in her lower extremities. Bibasilar crackles were noted on lung exam.

THE DIAGNOSIS

An electrocardiogram (EKG) revealed sinus tachycardia with some sinus arrhythmia. A chest radiograph revealed prominent pulmonary vasculature and the presence of Kerley B lines consistent with marked pulmonary edema. Laboratory testing revealed an elevated N-terminal pro b-type natriuretic peptide level of 2420 pg/mL (normal range: <100 pg/mL). Evaluation of thyroid function revealed overt hyperthyroidism with an elevated free thyroxine of 4.6 ng/dL (normal range: 0.8-1.8 ng/dL), a total triiodothyronine of 199 ng/dL (normal range: 60-181 ng/dL), and a suppressed thyroid-stimulating hormone level of <0.02 mcU/mL (normal range: 0.35-5 mcU/mL). A subsequent thyroid ultrasound showed a diffusely enlarged thyroid gland with a thickened isthmus, but no nodules.

The patient’s results were discussed with the on-call endocrinology provider at the time of her revisit to the ED. The patient was started on antithyroid medications (methimazole 20 mg/d) and a beta-blocker (atenolol 25 mg/d). Arrangements were made for an outpatient endocrine consultation within 3 days of her visit to the ED.

Upon evaluation in the outpatient endocrinology clinic, a thyrotropin receptor antibody test was positive, confirming Graves’ disease. The patient was given a diagnosis of thyrotoxicosis secondary to hyperthyroidism due to Graves’ disease. Her marked pulmonary edema was secondary to thyrotoxicosis and aggressive hydration with IV fluids.

CONTINUED
DISCUSSION
Hyperthyroidism is a common metabolic disorder with prominent cardiovascular manifestations.1 Classically, patients with hyperthyroidism develop irritability, heat intolerance, emotional lability, muscle weakness, menstrual abnormalities, and weight loss (despite an increased appetite). Cardiovascular manifestations include palpitations in up to 85% of patients, and dyspnea on exertion and fatigue in approximately 50% of patients.2 Hyperthyroidism has also been shown to produce changes in cardiac contractility, myocardial oxygen consumption, cardiac output, blood pressure, and systemic vascular resistance.3,4 Hyperthyroidism may complicate preexisting cardiac disease or may cause cardiac complications in individuals without structural abnormalities. (Our patient had no known structural abnormalities.)

In a small subset of patients with severe hyperthyroidism and exaggerated sinus tachycardia or atrial fibrillation, rate-related left ventricular dysfunction may cause heart failure.5 The assessment of thyrotoxic manifestations, especially potential cardiovascular complications, is essential to formulating an appropriate treatment plan.6 Cardiac evaluation may require an echocardiogram, EKG, Holter monitor, or myocardial perfusion studies.

Beta-blockers, diuretics among treatment options
Treatment with beta-blockers to reduce heart rate should be first-line therapy.7 In patients with overt heart failure involving pulmonary congestion, the use of diuretics may be appropriate.8

Our patient continued to take the medications prescribed during her ED visit: methimazole 20 mg/d and atenolol 25 mg/d for her Graves’ disease. A chest radiograph one month later revealed resolution of her pulmonary edema.

THE TAKEAWAY
The cardiovascular manifestations of hyperthyroidism remain some of the most common signs and symptoms of thyroid disease. Pulmonary edema and congestive heart failure, however, are uncommon. Physicians need to be aware of this rare—but important—clinical presentation of a common condition. JFP

References