

A Case of Recently Diagnosed Uncontrolled Hyperthyroidism Associated with Bilateral Pulmonary Embolism

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A Case of Recently Diagnosed Uncontrolled Hyperthyroidism Associated with Bilateral Pulmonary Embolism

Gomez Casanovas J, Bartl M, Pedraza L, Fleires A, Suarez A

Background

Pulmonary embolism (PE) is a relatively common acute cardiovascular disorder with considerable mortality, despite advances in diagnosis and treatment. In 25 to 50% of first-time cases, no readily identifiable risk factor can be found. Several studies have suggested hyperthyroidism to be a potential hypercoagulable and hypofibrinolytic state. In this case, we present a patient with uncontrolled hyperthyroidism with incidental bilateral PE.

Case Presentation

A 47-year-old Hispanic lady with past medical history of recently diagnosed hyperthyroidism who was not compliant with medical therapy, presented to the emergency department with 4-hour history of chest pain. She described it as sudden onset, pressure-like pain that occurred during exertion and radiated to the back. She had associated palpitations, diarrhea, arthralgias and dyspnea that did not improve with rest. She also states having poor appetite and some weight loss for at least 1 year. The patient does mention having been diagnosed with hyperthyroidism one month ago by an endocrinologist in Mexico, who prescribed her propranolol and methimazole, which she was not taking as prescribed. Her vital signs were temperature of 98.1, heart rate of 124, respiratory rate of 18 and blood pressure of 112/84 mm Hg with a SpO₂ of 99% on room air. Upon physical examination, she was alert, anxious and in mild distress. She was tachycardic, with no murmur or gallop and lungs were clear to auscultation. She did not have any skin lesions. Laboratory findings were remarkable for elevated D-dimer of 643, alkaline phosphatase of 228 with liver function tests within normal range and troponin I of 0.27. Thyroid function test revealed TSH of 0 uIU/mL, total T₃ 493 ng/dl, free T₃ 22.5 pg/ml, T₃ uptake of 62.5 %, total T₄ 24.9 ug/dl and free T₄ of 5.06 ng/dl. CT of the chest with contrast revealed subsegmental bilateral lower lobe pulmonary emboli. It also revealed a soft tissue prominence within the anterior mediastinum. Thyroid US revealed an enlarged thyroid gland with heterogeneous echotexture and hyperemic Doppler flow, compatible with active thyroiditis. Burch-Wartofsky score was 35 points. Patient was admitted for uncontrolled hyperthyroidism with impending thyroid storm as well as bilateral PE with possible right heart strain. She was started on Propranolol, Methimazole, potassium iodide and heparin drip. Patient status overall improved and Echocardiogram revealed EF 60-65% without signs of right heart strain. Thyroid workup then revealed TSI of 297, positive ANA with nuclear pattern and TPO of 151 IU/mL. She was later discharged with Eliquis, methimazole and propranolol for close follow up.

Conclusions

Hyperthyroidism has well known effects on the cardiovascular system, however, further data suggests that it modifies physiologic processes of hemostasis, leading to bleeding or thrombosis.

This is due by upregulating adhesion molecules and endothelial marker proteins. Most studies have shown that hyperthyroidism is related to venous thromboembolism risk, however just a few have focused on specifically its association with PE. There are currently no recommendations in regard to prophylactic anticoagulation in hyperthyroid state, however physicians should be alert for possible thrombotic events with these patients.