Case Report

Thyrotoxicosis: an unusual cause of syncope

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A B S T R A C T

Syncope is a common emergency department (ED) chief complaint, with many known but also unknown causes. Here we present a novel ED presentation of a young woman with new-onset hyperthyroidism that masqueraded as a syncopal event with head trauma. A 21-year-old woman arrived in the ED with head trauma as the result of seemingly unprovoked syncope, due to her history as well as the nature of her trauma. Persistent tachycardia during her ED course after an unremarkable full trauma evaluation prompted ordering of additional lab testing, which revealed evidence of thyrotoxicosis. Here we consider the possibility of thyroid dysfunction resulting in syncope.

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1. Introduction

Accounting for nearly one in fifty ED visits, syncope is a quite common presenting chief complaint [1]. The differential is extensive, ranging from benign causes including vasovagal and orthostatic causes to life-threatening ones such as subarachnoid hemorrhage (SAH) or pulmonary embolism (PE). In about one third of patients who present with a chief complaint of syncope, no definitive diagnosis is made at either ED discharge or during hospital admission [2]. Here we identify a curious case of new-onset thyrotoxicosis, masquerading as a syncopal event in a young patient.

2. Case report

The patient was a 21-year-old otherwise healthy woman who presented to the ED with “syncope, found down outside a store”. Her vital signs were: BP 145/87, Pulse 114, Temperature 37.3 °C, Respirations 20, SpO2 99%.

She was fully oriented and did not recall any prodrome of headache, lightheadedness, palpitations, chest pain, or shortness of breath. She complained of left-sided headache, felt weak and nauseated. A full review of systems was negative with no recent illnesses, neck pain or vision changes, fevers, chills, gastrointestinal or genitourinary disturbances. She denied drug or excessive caffeine use.

Her physical exam was significant for a large area of ecchymosis over the left temporal region extending periorbital, with normal visual acuity and no other focal findings. Point-of-care echocardiography1 was negative for any remarkable pathology. Emergent CT head and CT of the cervical spine did not demonstrate any acute findings. An EKG revealed sinus tachycardia at 119 bpm without any ST changes, nor signs of arrhythmia or cardiac ischemia (Fig. 1). Pertinent initial labs including a CBC and chemistry panel were within a normal range.

The initial ED treatment included IV fluids, acetaminophen, and ondansetron, which improved her headache and nausea. Despite this, during her period of monitored ED observation, her heart rate increased without any other symptoms or suggestion of additional pathology; it ranged from 120 to 150 bpm, sinus on a repeat EKG. We then decided to consult the Internal Medicine service for admission, continued monitoring and work-up.

Surprisingly, while removing our patients’ hard cervical collar after clearance of the C-spine by imaging, she had uniform thyromegaly without any palpable masses. Given these findings, thyroid function tests (TFT) were ordered and she was given IV propranolol prior to admission, with good response. Other than tachycardia, she remained stable throughout her ED course, and her minor wounds were treated.

A chart review revealed: TSH: <0.01mcIU/ml, Free T4: 4.7 nl/dl, and T3: >800 ng/dl. Her hospital course was uncomplicated, and she was discharged on oral propranolol, started on oral methimazole, and follow-up was scheduled for further testing.

3. Discussion

In summary this was a 21-year-old healthy woman who presented as unprovoked syncope causing head trauma. Her lack of a pattern of injury on the hands or arms, suggestive of protective instincts, was thus

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1 It is understood that point-of-care ultrasound (POCUS) in the emergency department is a helpful tool, but there are guidelines and policies to its use clinically.
suspicious. It was the persistent, worsening tachycardia combined with a detailed neck examination that prompted the ordering of TFTs. Given the initial workup, it was less likely neurologic or neurogenic in nature; SAH, PE, heart disease, and ectopic pregnancy were not supported as well. In her inpatient stay, our patient was found to have evidence of severe hyperthyroidism. This was a noteworthy discovery that led to a brief review on thyroid disease.

Thyrotoxicosis is a state in which a patient is symptomatic from excess thyroid hormone, with well-known symptoms including heat intolerance, palpitations, diarrhea, resting tremor. Thyrotoxic crisis, or thyroid storm, represents a clinical diagnosis with severe effects on the body’s hemodynamics and metabolic states, and multiple precipitating factors [3]. Interestingly, our patient did not exhibit most other classic signs of hyperthyroidism.

In recent emergency medicine literature and guidelines, the disposition of patients with syncope has evolved to a risk stratification approach [4,5]. Many patients are discharged even without an established diagnosis—reasonable in low risk populations. Ultimately, our decision to admit was due to unexplained prolonged tachycardia, though given the physical exam we had suspicion for undiagnosed thyroid disease.

Independent of tachycardia, many studies have reflected the effects of thyrotoxicosis on the heart including heart failure and AV block, both of which have been documented causes [6–8]. One such case out of a cardiology journal in Turkey describes a syncopal event that was possibly caused by thyrotoxicosis [9], which we also found in our case. A large review by Shen et al.[7], who examined the cardiac effects of thyroid disease, does not mention syncope. We wonder whether hyperthyroidism secondarily caused an arrhythmia or other high-output heart failure state, which resulted in the transient loss of consciousness.

4. Conclusion

While the direct mechanism in our hyperthyroid patient presenting with syncope is unclear, it is none-the-less a notable finding that warrants further research. Regardless, our ultimate hope is that clinician readers will be reminded to use their best clinical judgment along with a careful history and physical to safely guide a patient’s disposition, and consider hyperthyroidism in unexplained persistent tachycardia.

References