May 6th, 12:00 AM

Syncope or Seizure?

Joseph Heron
Rowan University

Kevin Dwyer
Virtua Health - Voorhees

Syncope or Seizure?

Joseph Heron DO, Kevin Dwyer DO
Emergency Medicine Residency and Department of Emergency Medicine, Rowan University SOM

ABSTRACT

Syncope is a common complaint in the emergency departments accounting for 1-2% of visits and can approach admission rates of a staggering 85% (1). The causes and conditions can be numerous, ranging from benign to life threatening. A good background history can go a long way in determining the etiology of the patient's syncope. We describe a case of an elderly male who presented with a reported chief complaint of seizures, another syncope-mimic seen in the emergency department. He had a history of CAD, HTN, AAA, and osteoarthritis but no prior history of cardiac arrhythmia, MI, or structural heart disease. The wife noticed collapse followed by seizure-like activity after opening the refrigerator. The initial EKG was non-specific. During his course in the ED there was a witnessed syncopal episode with upper extremity shaking and return to mental baseline within a minute of the event. A repeat EKG showed complete heart block. Seizures are a common presentation and chief complaint in the emergency department. It is easy to get tunnel vision in the clinical pathway and treatment plan. Other less common presentations of loss of consciousness with rhythmic shaking include convulsive syncope and should be considered. It is important to cast a wide net in the initial workup and always consider convulsive syncope, especially in an elderly patient with risk factors.

INTRODUCTION

Syncope is defined as a transient loss of consciousness and postural tone caused by a period of inadequate cerebral perfusion. This is most often caused by an abrupt drop of systemic blood pressure so it is not surprising that one of the most common clinically apparent causes of syncope is cardiac in nature. Neurally mediated syncope is the most common type and has a benign course, whereas cardiac syncope is associated with increased morbidity and mortality(9). Insufficient cerebral perfusion is of a relatively brief duration and is transient and self limited. Further examining the definition of syncope leads us to realize that loss of postural tone is inevitable with loss of consciousness(LOC), and as a result syncope is usually associated with collapse. Recovery, however, is typically rapid and rarely lasts longer than one to two minutes. If symptoms reside longer then other causes should be explored. Syncope can be caused by a variety of etiologies including orthostatic hypotension, arrhythmias, epileptic seizures, psychogenic non-epileptic seizures, subclavian steal syndrome, subarachnoid hemorrhage, intoxications, metabolic disturbances or even a breath holding spell among many more (3).

Case Report

A 75 year old man presented to the emergency department after walking to the kitchen and falling in front of his fridge with subsequent upper extremity shaking. The episode of seizure-like activity lasted for thirty seconds and resolved on its own. He did bump his head slightly after collapsing to the ground. Shortly after, the patient returned to his baseline mental status and was able get himself up and return to a seated position on his couch. While sitting on the couch, the patient slumped over and had another shaking episode. It was at this point that the wife called 911 and brought the patient to the ED.

The patient did not receive any medications en route.

Vital signs on arrival were blood pressure of 122/70, heart rate of 72 beats per minute, respiratory rate of 18 breaths per minute and temperature of 98.7 F orally with a pulse oximetry of 98% on room air.

The patient's other past medical history was significant for abdominal aortic aneurysm, hypertension, and osteoarthritis. He had his wisdom teeth removed many years ago but no other surgeries. He had a remote history of smoking but quit over forty years ago. He drank alcohol socially but never exceeded one or two beers in one sitting. There was no history of prior cardiac events. The patient was allergic to peanut butter and bee stings. He did not take any over the counter medication.

The patient was lucid on arrival but a poor historian and unable to provide a detailed history of the events. Due to visitor restrictions, his wife did not accompany him. Shortly after the patient arrived the wife was contacted via phone. She reported the patient had a similar episode a couple years ago and was admitted to the hospital and worked up for seizure. Upon discharge, she was under the impression he was diagnosed with a seizure disorder. This information was relayed to EMS and reported as a past medical problem. He was not currently taking any anti-epileptics.

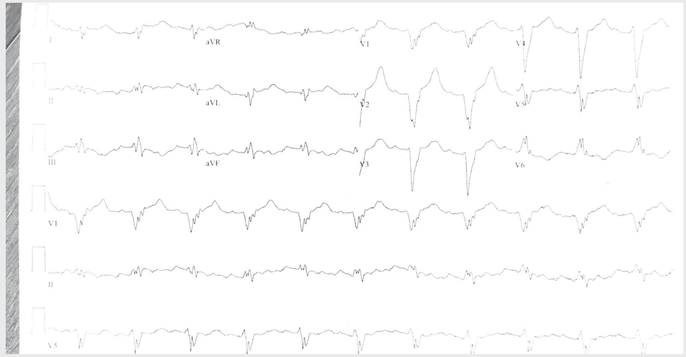
The patient was awake, alert and oriented on arrival and in no acute distress. He transferred himself from the EMS stretcher to the hospital bed. During the preceding three days he reported generalized weakness and lightheadedness. On the afternoon of his arrival, the patient admitted feeling generally weak, which he attributed to being hungry. During the time leading up to his ED visit, he had not experienced chest pain, palpitations, diaphoresis, pallor, lightheadedness, or shortness of breath. He denied fevers, syncope, abdominal pain, nausea, or vomiting.

On initial exam his head was normocephalic and atraumatic. Neck was supple without lymphadenopathy, tracheal deviation, or jugular venous distention. The heart exam revealed no murmurs, rubs or gallops, a normal rate and rhythm, and no displacement of point of maximal intensity. The lungs were clear to auscultation bilaterally. The abdomen was soft and non-distended. There was no peripheral edema. His skin was warm and dry with capillary refill of 2-3 seconds. The neurological exam was unremarkable.

Diagnostic studies showed glucose of 130. Initial rhythm on the monitor was sinus rhythm at 66 beats per minute. Initial EKG showed sinus rhythm with first degree AV block, normal axis, QRS interval of 90ms, QTc interval of 450ms, and non specific T wave changes in the V4-V6. Initial laboratory data was unremarkable. The chest x ray revealed no evidence of cardiomegaly, effusions, pneumothorax, or infiltrates. Initial head CT was negative for acute intracranial pathology.

Shortly after lab work resulted, the patient had an episode of bradycardia on the cardiac monitor as low as 40 beats per minute. He experienced transient loss of consciousness twice during monitoring, which responded to vigorous stimulation. Re-evaluation of the patient yielded no complaints. Blood pressure was 100/60. Transcutaneous pacer pads were placed. Repeat EKG showed 2nd degree AV block with left bundle branch block. EKG was reviewed with cardiology, who recommended temporary pacing and transfer for permanent pacemaker placement.

Shortly thereafter, the patient became further bradycardic into the mid 30s. He proceeded to have a brief period of loss consciousness with witnessed shaking of his upper extremities. After thirty seconds, he returned to his previous mental status. He stated that he felt "good." The patient experienced two more of these episodes in the period of fifteen minutes, all with brief episodes of extremity shaking, stiff truncal posture, and brisk return of baseline mental status. The decision was made to temporarily transvenously pace the patient for stabilization, which was successful.



Discussion

Discussion: It is often difficult to develop appropriate management plans for patients experiencing paroxysmal loss of consciousness. Many of these presentations are lumped together and gravitate toward a more global diagnosis of syncope. Frequently, the etiology is never found in the ED and left to further workup and investigation in the hospital.
Syncope is a common yet vague complaint of patients presenting to the ED. Approximately 1% to 2% of all ED visits occur due to a chief complaint of syncope, which amounts to around 1 million patient visits per year. (3) Lifetime prevalence rates range from 10.5 to 19 percent. (6)

Patients often remain undiagnosed despite exhaustive diagnostic testing. Patients with an identified cardiac cause for syncope have twice the risk of death compared to general population, and patients with a neurologic cause have a 50 percent increased risk. Those with an unknown cause of syncope have a 30 percent increased risk of death, whereas patients with vasovagal syncope are at no increased risk. (6)

Syncope is a common chief complaint in the emergency department. It accounts for an estimated annual average of 600 per 100,000 visits per year and a very high percentage of these patients are admitted for further evaluation. A conservative estimate of the total annual costs for syncope-related hospitalizations was $2.4 billion, with a mean cost of $5,400 per hospitalization(10).
The etiologies of syncope can be numerous, and it is imperative to rule out life threatening etiologies. Furthermore, it is important to cast a wide net in initial evaluation and pay close attention to history if available. Underlying etiologies include neuro cardiogenic, orthostatic, neurologic, cardiac, and pulmonary causes. Cardiac arrhythmias have been identified as the most common source(3).

Although a cardiac origin is the most common underlying abnormality in transient LOC, a presentation of convulsive syncope can muddy the waters. Cardiac syncope with cerebral hypo perfusion can masquerade as seizure and easily lead the clinician down a different treatment pathway, which can have devastating effects on the patient. It may delay proper treatment and prophylaxis for syncope, and exacerbate and further deteriorate cardiac function. Misdiagnosis can lead to complications for driving and occupation. Furthermore, patients may be inappropriately treated with potentially deleterious anticonvulsant drugs, many of which have been reported to have cardio-toxicity.
Research suggests that between 20-40% of patients are misdiagnosed with epilepsy(2).

DISCUSSION

It is cardinal to differentiate between convulsive syncope and epileptic seizures, but difficulties in diagnosis can be complicated by overlapping features, a superficial history, and insubstantial time with the patient. Features of epileptic activity described by non-medically trained observers may be too readily attributed to primary brain dysfunction rather than secondary to effects of cerebral hypo perfusion. Inadequate cerebral perfusion leads to abnormal neuronal firing, which can lead to a seizure like episode. Important distinguishing factors in convulsive syncope include episodes characterized by a brief loss of consciousness and no post ictal state. Precipitation by pain, fear, or emotional distress suggest a cardiac cause. A prodrome of nausea, diaphoresis, and vomiting may reflect vaso vagal syncope. Brief myoclonic jerking of limbs or stiffening of posture starting after loss of consciousness may be seen in convulsive syncope. Facial pallor is often a tell-tale sign. Situations which increase intrathoracic pressure such as defecation or getting up in the middle of the night can decrease venous return and precipitate syncope 4).
In contrast, epileptic seizures are typically cause by pathologic patterns of brain cortex activity with excessive and synchronous discharge of neurons in the brain. Often times, an aura may be sensed prior. Tonic-clonic movements, as opposed to non-sustained myoclonic jerks, often are indicative of seizure. Lateral tongue biting may also be seen, or less commonly posterior shoulder dislocation. A prolonged post ictal state ensues, where the patient is often tired and sleeps(5).

Another hurdle in distinguishing cerebral from cardiac causes of syncope lies in the fact that epilepsy is often a clinical diagnosis. As with any disease process in question, the potential for misdiagnosis occurs thus leading to inappropriate treatment modalities. One of the best diagnostic modalities available for epilepsy is video telemetry monitoring with EEG, which is especially sensitive and specific. Unfortunately, it may not be widely available or even practical for those with infrequent attacks, thus making the diagnosis unclear and often based on clinical judgement. Frequently, the EEG is used by the neurologist to define the epilepsy syndrome of a patient and not to cement a diagnosis in patients with blackouts. Other modalities that may prove helpful in the evaluation include CT or MRI to rule out mass effect or intracranial processes causing seizure.

As many as 20% to 40% of epileptics have been misdiagnosed. Many of these patients have cardiovascular syncope with abnormal movements due to cerebral hypoxia, which may be difficult to differentiate from epilepsy on clinical grounds 2). Many screening tests may be implemented to pinpoint an accurate diagnosis when cardiovascular syncope is still in the differential. Perhaps a more thorough workup for cardiogenic source is warranted. These include but are not limited to noninvasive cardiac testing such as tilt test and carotid sinus massage during continuous ECG. Other modalities include holter monitor or loop recorder(9).

CONCLUSIONS

Syncope due to cardiogenic cause is much more prevalent than epilepsy in the general population. Similarities in presentation between convulsive syncope and epilepsy can lead to misdiagnosis of epilepsy with cardiac causes going unrecognized. It is also important to note that although rare, the phenomenon of epileptic seizure and non epileptic seizure occurring in the same attack has been reported with one likely provoking the other. The management of transient loss of consciousness with convulsion requires careful history, collaboration between multiple specialties, physical examination, and additional diagnostic tests.

REFERENCES

1. References available upon request