VIEWPOINT

EDUCATIONAL CORNER

An Elderly Woman With Recurrent Transient Loss of Consciousness Preceded by Hallucinatory Attacks



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ABSTRACT

We report a patient with transient loss of consciousness preceded by hallucinations caused by cardiac syncope. The diagnosis was confirmed by simultaneous video-electrocardiogram-electroencephalogram recording of an event. The attacks no longer occurred after pacemaker implantation. Clinical features helpful in guiding decision making in transient loss of consciousness are discussed. (Level of Difficulty: Beginner.) (J Am Coll Cardiol Case Rep 2020;2:1824-7) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

n 89-year-old woman presented at the emergency department with the following paroxysmal events that started 6 days earlier and occurred approximately 3 times a day. First, she experienced visual and auditory complex hallucinations. Subsequently, she would scream or yell out a short phrase and shake her arms for several seconds,

LEARNING OBJECTIVES

- To make a differential diagnosis in cases of transient loss of consciousness based on history-taking and interictal examinations.
- To recognize atypical cases of transient loss of consciousness and be aware of the need to register an event to establish a definitive diagnosis in such cases.
- To be aware that hallucinations can occur in syncope, either before loss of consciousness or when regaining consciousness.

followed by loss of consciousness for 5 to 10 s. When unconscious, her eyes were open and her gaze was directed upward. Afterward, she would immediately be oriented and could provide details of the experienced hallucinations. The events occurred in both upright and supine position. She did not bite her tongue nor was she incontinent during these events. A video recording of an event is provided in the online supplement (Video 1).

PAST MEDICAL HISTORY

Her medical history consisted of transient ischemic attacks due to right-sided carotid artery stenosis (treated conservatively at the patient's request), deep vein thrombosis, paroxysmal atrial fibrillation for which she used warfarin and digoxin, chronic obstructive pulmonary disease, moderate left ventricular hypertrophy with a left ventricular ejection fraction of 50%, moderate aortic valve stenosis (mean

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pressure gradient 28 mm Hg), and moderate to severe mitral and tricuspid valve insufficiency. She had no prior psychiatric disorders.

DIFFERENTIAL DIAGNOSIS

Epileptic seizure, cardiac syncope, reflex syncope, delirium.

INVESTIGATIONS

On admission, her blood pressure was 150/104 mm Hg and she had a regular pulse of 58 beats/min. Upon cardiac auscultation, she had a mild crescendodecrescendo systolic murmur at the right upper sternal border and moderate holosystolic murmur at the apex. Basic neurological examination was unremarkable. Laboratory examinations and chest X-ray were normal. The electrocardiogram (ECG) showed normal sinus rhythm, normal conduction, and signs of left ventricular hypertrophy with strain. Cranial computed tomography showed an old silent brain infarct in the left basal ganglia. During simultaneous video, ECG, and electroencephalogram (EEG) recording of an event, the ECG recording showed an atrioventricular block followed by an asystole. After several seconds of asystole, symptoms of hallucinations, screaming, and

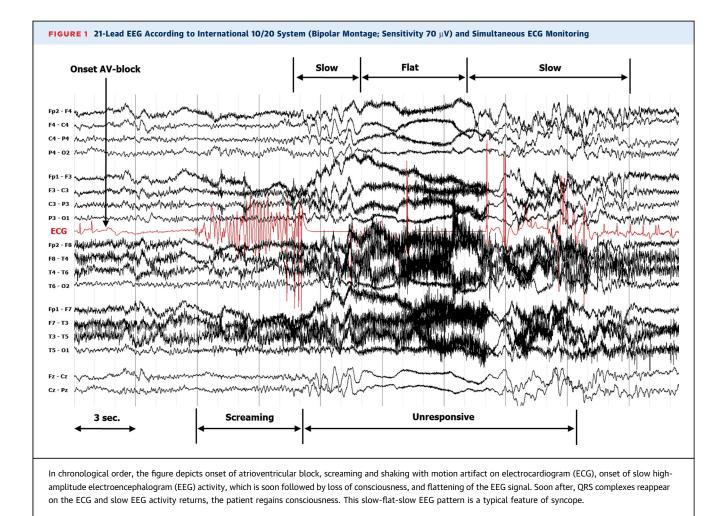
shaking began, followed by loss of consciousness (**Figure 1**, Video 2). The EEG showed diffuse highamplitude slow activity after which the EEG became isoelectric, a typical EEG pattern for syncope (1).

MANAGEMENT

Our patient was diagnosed with syncope due to intrinsic paroxysmal atrioventricular block. After pacemaker implantation (dual chamber pacemaker), the events no longer occurred.

DISCUSSION

Transient loss of consciousness is a commonly encountered symptom and may pose a clinical dilemma, as the underlying condition can be difficult to establish. The major causes are epileptic seizure,



ABBREVIATIONS AND ACRONYMS

ECG = electrocardiography

EEG = electroencephalography

cardiac syncope, reflex syncope, and syncope due to orthostatic hypotension (1). An important first step is to attempt to distinguish epilepsy from syncope. Signs suggestive of epilepsy are a lateral tongue bite, postictal confusion, lateral head deviation, unusual posturing, preceding aura, or focal neurological manifestations (e.g., aphasia, unilateral limb shaking) (2,3). However, discriminating epilepsy from syncope based on symptoms alone can be difficult, as there is substantial overlap in symptomatology. During any type of syncope, individuals can exhibit either loss of muscle tone, myoclonic jerks, or stiffening of arms and legs (4,5). A video analysis of 42 cases of induced syncope showed that myoclonus occurred in 90% of cases (although myoclonus never preceded the loss of consciousness), 79% had other motor activity (e.g., lateral head deviation, automatisms, head raising, or sitting up), 40% vocalized (moaning sounds), and visual and auditory hallucinations occurred in 60% of individuals (6). Importantly, patients assigned these hallucinations to the period of unconsciousness or during the period afterward, but did not experience them before losing consciousness (6,7). Prodromal hallucinations would normally be suggestive for epilepsy (8,9). Therefore, a diagnosis of epilepsy was initially suspected in our patient, which was the reason for performing the ECG-EEG recording that established the diagnosis of cardiac syncope instead. Thus, the limb shaking, vocalizations, and hallucinations in our patient were caused by cerebral hypoperfusion. Of note, cardiac arrhythmia can occur secondary to an epileptic seizure (10), but this was ruled out with the EEG registration and further substantiated by the fact that no more events occurred after pacemaker implantation.

In accordance with established guidelines, the initial diagnostic workup of a patient suspected of syncope should include a careful history taking, including the situation in which syncope occurs, the nature and duration of prodromal symptoms, bystander observations, post-event symptoms, prior medical history and medication use, and family history with emphasis on syncope and sudden unexplained death (10). In the current case, high-risk features suggesting cardiac syncope were the presence of structural heart disease, a short history of syncope, age >60 years, very short prodromes, and syncope occurring in supine position (10). A diagnosis of reflex syncope should be considered in case of more prolonged prodromal signs, absence of a prior history of heart disease, younger age, specific triggers (e.g., dehydration, pain, stress, micturition) and a long history of syncope (10). Syncope occurring in the setting of positional change (supine to sitting, sitting to standing) is suggestive of orthostatic hypotension. Recommended physical examination in case of syncope includes blood pressure and heart rate and rhythm, testing for orthostatic hypotension if the patient's history is compatible with this condition, cardiac auscultation, and a basic neurological examination. Performing an ECG in syncope is recommended and performing targeted blood tests (e.g., to exclude anemia) is reasonable, whereas routine and comprehensive laboratory testing is not considered useful (10). After pacemaker implantation, the events no longer occurred.

CONCLUSIONS

Patient-reported information combined with interictal examinations are paramount to diagnosing the cause of transient loss of consciousness, but are not always sufficient. Our case demonstrates that, although prodromal hallucinations are usually indicative of epilepsy, this phenomenon also can occur in cardiac syncope. In atypical and recurrent cases, registration of an event is essential and should be pursued.

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TAPPENDIX For supplemental videos, please see the online version of this paper.