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Case Report

Vasovagal Syncope Treated as Epilepsy for 16 Years

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Abstract

The differentiation of vasovagal syncope and epileptic seizure is sometimes problematic, since vasovagal syncope may mimic epileptic seizures in many ways. The present report describes a patient who had been diagnosed and treated as having epilepsy with medically-refractory seizures for 16 years. Often, unlike epileptic seizures, tonic-clonic convulsions and postictal confusion are uncommon features of vasovagal syncope, but these may occur. Our patient was subjected to subcutaneous injection of one ml normal saline, which caused asystole leading to hypoxia and consequently a typical tonic-clonic convulsion. This patient was proved to have vasovagal syncope. The findings in the present case suggest that the possibility of vasovagal syncope should always be taken into consideration when evaluating patients with medically-refractory or unusual pattern of seizures. In such a circumstance, simultaneous video-electroencephalogram/electocardiogram monitoring may help achieve the correct diagnosis.

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Keywords ● Epilepsy ● syncope ● EEG

Introduction

Vasovagal syncope may mimic epileptic seizures in many ways. This makes the differentiation of the two events sometimes problematic.[1] Syncope is the impairment of cardiovascular autonomic control, which results in gradual failure of cerebral perfusion. Vasovagal syncope is the most common type of neurally mediated syncopes.[2] Patients with vasovagal syncope may occasionally have clonic movements, tonic posture and even myoclonus during an episode. These movements may be confusing to a physician. The present case report describes a patient who had been treated as having epilepsy with medically-refractory seizures for 16 years. The patient, however, was proved to have vasovagal syncope.

Case Description

A 22-year-old, right-handed woman presented for the evaluation and treatment of uncontrolled seizures. Her seizures began when she was six years old. Her seizures were described as seeing flashing lights for about five seconds, then having loss of consciousness with rhythmic limb movements and jaw locking for 1-2 minutes, and urinary incontinence, followed by post-ictal fatigue and confusion for about 30 minutes. She used to have one or two attacks per month. These episodes never happened during sleep. The episodes were often triggered by pain induced by stimulations such as dental work or needle insertion for blood

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sampling. However, sometimes there was no identifiable triggering factor. She had been admitted to hospital repeatedly for her seizures.

She had no history of tobacco, alcohol or drug abuse. Her mother did not have any pregnancy complications. The patient had normal development as well as normal school and university performances. In review of her systems, she complained of having a migraine-type headache and daytime hypersomnolence. She did not have any past history of major febrile illnesses, central nervous system infections, febrile convulsions or significant head traumas. Her medical and neurological examinations, routine blood tests including hematology, blood chemistry, and liver function tests, brain MRI and electrocardiogram (ECG) were normal. Repeated interictal electroencephalograms (EEGs) were normal as well.

The patient's condition had been diagnosed as epileptic seizures, and she had been treated with various anti-epileptic drugs (AED) for the preceding 16 years. She did not have reasonable drug compliance despite her repeated seizures, because she believed that drugs were not effective for her illness. She was referred to us by her dentist, because she had experienced a seizure during dental work after injecting an anesthetic agent. At the time of admission, she was taking lamotrigine 50 mg daily.

She was admitted to epilepsy care unit, Nemazee Hospital, Shiraz University of Medical Sciences, Shiraz, Iran for a video-EEG/ECG monitoring. She had no seizure attack during the study, and the recorded EEG and ECG were entirely normal. After obtaining her consent, it was decided to provoke her seizure using the pain of subcutaneous injection of one ml normal saline as a triggering factor. A few seconds later, her heart rate dropped, and she developed asystole, which lasted for about 100 seconds. During asystole, she developed a clonic-tonic-clonic seizure for one minute, and post-ictal confusion for about 15 minutes. The attack terminated spontaneously without any intervention. The diagnosis of vasovagal syncope was confirmed, and she was referred to a cardiologist after being instructed to discontinue taking lamotrigine. According to her recent office visit, she had been seizure free for eight months since she was referred to the cardiologist. In the meantime, she was taking propranolol 20 mg twice per day, and was exercising appropriate instructions and considering the related precautions to prevent any further attacks.

Discussion

One important step in the management of a patient with a history of seizure(s) is correct diagnosis of the illness. An important and sometimes problematic issue in the management of seizure is the differentiation of epileptic seizures from non-epileptic ones.[1] Basically, epilepsy is a clinical diagnosis. Unless one happens to observe a seizure while recording the EEG, which is a rare event in general clinical practice, the diagnosis relies on the judgment of a physician or other health care providers. This judgment ultimately rests on the history provided by the patient or others, which may be misleading and results in maltreatment, similar to the scenario, which happened in the case of the present patient. The history of seeing flashing light was interpreted as epileptic aura. The triggering factors such as pain and inspecting blood were overlooked, and motor phenomena, loss of consciousness and post-ictal confusion led to the misdiagnosis of the illness. She had been diagnosed as having epilepsy for 16 years, however, normal EEGs and AED-unresponsiveness had been ignored. As others have mentioned in similar case reports, no one, even physicians who witness the events, is immune to making a false clinical diagnosis of epilepsy.[3]

Syncope is a condition that is most commonly misdiagnosed as epilepsy.[4] The precise mechanism of vasovagal syncope is not fully understood. It is proposed that the failure of sympathetic efferent vasoconstrictor traffic occurs episodically and in response to a triggering agent such as fear, anger or pain.[5] There is evidence for the involvement of both neural and chemical pathways.[6] Presyncopal symptoms include lightheadedness, generalized muscle weakness, tinnitus and visual blurring, but up to a third of patients will have little or no prodrome. Often, unlike epileptic seizures, tonic-clonic convulsion, other motor phenomena, and post-ictal confusion are uncommon features, but may occur.[7] In the present case, injection (psychogenically)-induced asystole led to hypoxia, which in turn caused a typical tonic-clonic convulsion.

Conclusion

The possibility of vasovagal syncope should always be taken into consideration when evaluating patients with medically-refractory or unusual pattern of seizures. In such circumstances, simultaneous video-EEG/ECG monitoring may help achieve the correct diagnosis, particularly if the physician applies a triggering agent(s) after obtaining the patient's consent.

Conflict of Interest: None declared

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