Introduction
Epilepsy is a common neurological disorder prevalent among 7 out of 1,000 people in general population [1]. Before determining whether paroxysmal events represent an epileptic disorder, one must consider 2 alternatives: (a) non epileptic events mimicking epileptic seizures and (b) true epileptic seizures caused by a non-neurological condition. We report a patient with complete Atrioventricular (AV) block presenting with misleading symptoms suggesting seizure.

Case Presentation:
A 76-year-old, male, received at home, with a history of collapse and multiple episodes of ongoing unusual jerky movements of the body and amnesia to current events, no post ictal confusion. The vital signs measured at home were: blood glucose: 279 mg/dL, Heart Rate (HR)-36/min, respiratory rate: 10/min, blood pressure-80/60 mm hg.

Management and Outcome:
In view of recurrent seizures and unstable bradycardia patient was managed with injection atropine 0.6 mg IV bolus, injection Midazolam 2 mg IV bolus, injection Phenytin 1 gm IV loading dose and external cardiac pacing on demand mode (30/70). Patient continued seizing, with decreased level of consciousness. In view of low Glasgow Coma Scale (GCS) and risk of aspiration, patient was intubated and mechanically ventilated, shifted to cath lab for temporary pacemaker implantation and was planned for permanent pacemaker and coronary angiography, transferred to the Cardiac Care Unit for further monitoring and stabilization. Coronary angiography reported Triple Vessel Disease (TVD). 2nd day of hospitalization, patient was extubated, maintained all vital signs and was neurologically intact.

Discussion:
This study points to the difficulty one faces in confusing seizure with syncope and vice versa during diagnosis. Hence, reinforcing the importance of differentiating one from the other, recognizing repeated, convulsive syncope’s without provocation with a normal sinus rhythm or any other rhythm disturbance, are suggestive of cardiac syncope. Such patients should have a cardiac evaluation before discharge and stress should be laid on noninvasive cardiovascular evaluation such as Holter ECG monitoring, especially in patients with infrequent attacks and less dramatic rhythms.

Abstract

Introduction: Epilepsy is a common neurological disorder prevalent among 7 out of 1,000 people in general population. (1) Before determining whether paroxysmal events represent an epileptic disorder, one must consider 2 alternatives: (a) non epileptic events mimicking epileptic seizures and (b) true epileptic seizures caused by a non-neurological condition. We report a patient with complete Atrioventricular (AV) block presenting with misleading symptoms suggesting seizure.

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Epilepsy is a common neurological disorder prevalent among 7 out of 1,000 people in general population [1]. An epileptic seizure occurs due abnormally excessive or synchronous neuronal activity in the brain manifesting in a transient occurrence of signs and/or symptoms. Fundamentally, it is a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures [2], which can vary from brief and nearly undetectable to long periods of vigorous shaking [3]. In epilepsy, seizures tend to recur, and have no immediate underlying cause [4]. However, seizures that occur due to a specific cause are not deemed to represent epilepsy [2]. Before determining whether paroxysmal events represent an epileptic disorder, one must consider 2 alternatives: (a) non-epileptic events mimicking epileptic seizures and (b) true epileptic seizures caused by a non-neurological condition. However, many cardiovascular disorders may cause blackouts, complicated by abnormal movements due to generalized cerebral hypoxia, which may appear similar to epileptic seizures, leading to an incorrect diagnosis of epilepsy [5].

We report a patient with a complete Atrioventricular (AV) block presenting with misleading symptoms suggesting seizure.
Case Presentation

A 76-year-old, male patient was received at home, where he was found lying on the floor, conscious and talking. The patient last remembered working on his computer when he suddenly felt dizzy and didn’t remember anything thereafter. The vital signs measured at home were: blood pressure: 110/70 mm Hg, blood sugar via finger stick test: 279 mg/dL. While talking to the patient, he again had an episode of generalized tonic-clonic seizure lasting a couple of seconds, following which patient regained consciousness and had no post-ictal confusion. Patient was immediately put on a Cardiac Monitor and 1 large I/V bore cannula placed in the left antecubital vein. Repeat vital signs were: Heart Rate (HR)-36/min, respiratory rate: 10/min, blood pressure-80/60 mm Hg. Thereafter, Patient again had an episode of generalized seizure lasting a couple of seconds. Subsequently he regained consciousness. In view of unstable bradycardia, Injection Atropine 0.6 mg IV bolus pushed.

The patient was put into the ambulance for shifting him to the hospital, while pacing pads were placed over the chest and injection Midazolam 2 mg IV bolus and injection Phenytoin 1 gm IV loading dose, was administered. Patient continued seizing, with decreased level of consciousness without regaining consciousness this time. External cardiac pacing on demand mode (30/70) was started.

Patient had no history of trauma, headache, fever, chills, diplopia, dysarthria, dyspnea, chest pain or tightness, tinnitus, or hearing impairment. He had a history of diabetes mellitus type 2, not taking any regular medication; prior to this episode he was active, doing all routine activities.

Patient was shifted to the Emergency Room for further evaluation.

Physical examination

On being shifted to the hospital, a physical examination of the patient was carried out. The report is as under: Patient had poor general condition.

Primary survey: Airway: Compromised; Breathing: Labored; Circulation: Peripheral; Pulses: Weak.

Systemic examination: Heent: No significant abnormality detected, pupils were equal and reactive to light.

Chest: Lungs were clear, bilateral air entry was equal with vesicular breath sounds.

CVS: Auscultation of the heart revealed a normal S1 and S2 without murmur or gallop.

Abdomen: Abdominal examination was unremarkable.

CNS: Altered sensorium, agitated, GCS- E1V2M3- 6/15.

Investigations

Laboratory findings were as follows: Complete Blood Count (CBC): within normal limits. Renal Profile: sodium- 133 mEq/L (normal, 136 mEq/L-145 mEq/L); potassium- 4.7 mEq/L (normal, 3.5 mEq/L-5.1 mEq/L); bicarbonate- 16.8 mEq/L (normal, 23 mEq/L-29 mEq/L); glucose- 229 mg/dL (normal, 74 mg/dL-106 mg/dL); blood urea- 29.8 mg/dL (normal, 10 mg/dL-26 mg/dL).

Cardiac biomarkers (CPK, CPK-MB, TROPONIN I) were within normal limits.

There were no remarkable findings on chest radiography.

12 lead ECG showed ventricle paced rhythm with right bundle branch block, Heart Rate (HR): 86/min, pacing spikes seen (Figure 1).

Management

Patient was continued on transcutaneous pacing.

Post external pacing, vital signs were: Heart Rate (HR): 74/min; blood pressure: 124/60 mm Hg; respiratory rate: 20/min; oxygen saturation on ventilator: 100%; temperature: 98.3°F.

In view of low Glasgow Coma Scale (GCS) and risk of aspiration patient was intubated and mechanically ventilated.

Cardiology and neurology consultation were taken.

Patient was then shifted to Cath Lab, for temporary pacemaker implantation and was planned for permanent pacemaker and coronary angiography, and was transferred to the Cardiac Care Unit for further monitoring and stabilization.

Trans-thoracic echo Doppler reported an akinetic posterior wall, distal IVS, distal lateral wall, distal anterior wall and mid basal
inferior wall. Left Ventricular Ejection Fraction (LVEF)-40%. Dilated LV (6.5 cm/5.4 cm) & LA (4.9 cm), tethering of PML, moderate to severe Mitral Regurgitation.

Coronary angiography reported Triple Vessel Disease (TVD). Carotid Doppler USG reported bilateral lateral CCA showing diffuse irregular plaques not causing any significant narrowing/ hemodynamic abnormality.

2nd day of hospitalization, patient was extubated, maintained all vital signs and was neurologically intact (Figure 2).

Patients was advised for an early Coronary Artery By-pass Grafting (CABG) with mitral valve replacement and further follow up in the Cardiology outpatient department. Unfortunately, patient as per his preference wanted to be shifted to another center. Patient was discharged on day 5 from our hospital with temporary pacemaker in situ (Figure 3).

Discussion

In this case, the tonic-clonic activity was confusing, as the symptoms could be a “seizure” or “syncope”. Syncope is a symptom, defined as a transient, self-limited loss of consciousness with a relatively rapid onset and usually leading to fall; the subsequent recovery is spontaneous, complete, and usually prompt. The underlying mechanism is a transient global cerebral hypo perfusion. Seizure is synonymous with an epileptic fit, which is the manifestation of a paroxysmal discharge of abnormal rhythms in some part of the brain [6].

There is an increasing recognition of the problem of misdiagnosis of epilepsy. The extent of the problem remains unclear, but it is estimated that approximately 20% of the patients do not have epilepsy [7].

Stokes-Adams syndrome, named after two Irish physicians—Robert Adams (1791-1875) and William Stokes (1804-1877), is defined as a sudden, transient episode of syncope, occasionally featuring seizures especially when cerebral perfusion is diminished for a prolonged period of time because of arrhythmias like complete heart block, ventricular fibrillation and pulse less ventricular tachycardia [8-10].

Syncope, complicated by involuntary movements, such as myoclonus or myoclonic jerks, is not an uncommon event, and has been reported in 12% of blood donors [11], and in 38 (90%) of 42 syncopal episodes among 56 patients were provoked by a combination of hyperventilation, orthostatic and a valsalva maneuver tilt induced vasovagal syncope [12], which is associated with periods of asystole [13,14], resulted in abnormal movements in 64% of tilt-positive patients in this study. In a study by, Grubb et al. [15] on 15 patients with recurrent, unexplained seizure-like episodes, who were unresponsive to anticonvulsant medication syncope with tonic-clonic seizure like activity was seen in 10 patients (67%). After cardiac drug or device therapy, all 10 patients were tilt-negative and free from seizure-like episodes.

Thus, it is very important for emergency physicians to understand the difference between a seizure and a syncope and be on the watch for cardiovascular syncope, as misdiagnosing these seizure-like symptoms can be life threatening. An episode of convulsive seizure lasting for a few seconds with spontaneous recovery (regaining consciousness with no post ictal confusion phase), most-likely implies syncope and less-likely seizure [5]. All patients should have subsequent 12 lead ECG [16] and continuous cardiac monitoring or holter monitoring, which should commence from the emergency department itself. Missing a cardiac arrhythmia could be fatal and in addition, patients may be inappropriately treated with potentially harmful anticonvulsant drugs [5].

Various rhythms have been reported in the course of Stoke Adams syndrome like complete Atroventricular (AV) block, ventricular tachycardia, ventricular fibrillations and one of the rarest is standstill of the ventricle as an episode during normal rhythm [17]. As an Emergency Physician, one should be aware of uncommon manifestations.

There are specific and sensitive test for the diagnosis of epilepsy—namely, video telemetry monitoring with electroencephalography—but this may not be widely available or practical in patients with infrequent attacks [7].

Emergency department clinicians should be familiar with any automatic gain in their ECG machine as tachycardia interpreted as narrow complex tachycardia can actually be atrial activity that had been amplified by the automatic gain to look like small amplitude QRS complexes [5].

In this case, the uncontrolled diabetes mellitus, coronary angiography report suggesting a Triple Vessel Disease (TVD) with moderate to severe mitral regurgitation could be a cause of the patient leading to a complete heart block and thus presenting as syncope.

As per telephonic conversation with the patient, he had a permanent pacemaker implantation, was again advised coronary artery bypass graft with mitral valve replacement. Patient refused for any intervention. At present is doing well and has not had such episodes again.

Conclusion

This case report points to the difficulty one faces in confusing seizure with syncope and vice versa during diagnosis. However, a good and accurate clinical history is most important in reaching a correct conclusion. Simple noninvasive cardiovascular evaluation can help diagnosing cardiovascular syncope. Hence, reinforcing the importance of differentiating one from the other, recognizing repeated, convulsive syncope’s, without provocation, are suggestive of cardiac syncope. Patients can present with Stokes Adams attack with a normal sinus rhythm or any other rhythm disturbance as well. Such patients should have a cardiac evaluation before discharge and stress should be laid on noninvasive cardiovascular evaluation such as Holter ECG monitoring. [Type a quote from the document or the summary of an interesting point. You can position the text box anywhere in the document. Use the drawing tools tab to change the formatting of the pull quote text box].

This case report emphasizes the importance of recognition of cardiac syncope and differentiating the above from a seizure in a busy ED; especially for the young aspiring EM physicians and trainees and have a broad approach to common presentations in the ED which can sometimes be misleading.

References


