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Seizure or syncope: lessons over time

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Abstract

A 25-year-old woman with recurrent syncopal episodes presented with a first time generalized tonic clonic (GTC) seizure. She had experienced two prior fainting spells lasting seconds and associated with diet pills and dehydration. She had another similar spell prior to falling, sustaining a laceration to the right posterior occiput, and having a witnessed GTC seizure. Her scalp electroencephalography (EEG) showed left temporal slowing with sharp features. T1-weighted and T2-weighted MRI revealed two moderately enhancing focal lesions within the left frontal and temporal regions. These findings raised the possibility of an underlying seizure focus. Repeat imaging studies of this patient 1 month later, however, demonstrated resolution of these findings and an area of encephalomalacia, consistent with a traumatic coup contrecoup injury. A repeat EEG was normal. Therefore, the cause of the loss of consciousness was due to syncope with the consequent head injury giving rise to an isolated seizure. Understanding the underlying cause of a seizure is important in dictating treatment. In this setting the patient was not initiated on seizure medication and has done well.

Keywords

Coup contrecoup; Seizure; Syncope

1. Introduction

Transient loss of consciousness (TLOC) is a spontaneous loss of consciousness with complete recovery. It is estimated to effect one-quarter to one-half of the population at some point during a lifetime. TLOC can result from a seizure disorder, cardiogenic causes (usually vasovagal disorders but also arrhythmias) and psychogenic attacks [1] [2]. Guidelines from the National Institute for Health and Clinical Excellence (NICE) first suggest determining whether the patient has had an episode of TLOC, followed by EKG evaluation. Referral for urgent cardiology evaluation is suggested in the setting of syncope with exertion, dyspnea on exertion, heart murmur/failure, family history of sudden death, or abnormal EKG. A neurogenic cause to TLOC is suspected if the presentation involves one or more of the following: a bitten tongue, head turning to one side, memory loss, unusual posturing, prolonged jerking of limbs, post event confusion, or prodromal déjà vu, or jamais vu [3]. The history usually guides treatment but at times it can be ambiguous and evaluations for both cardiac and neurogenic causes are often simultaneously pursued.

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2. Case report

A 25-year-old female with a prior history of syncope (lightheadedness, followed by LOC for a few seconds, and no ictal or post ictal phenomena) presented with a witnessed generalized tonic clonic (GTC) seizure. She had recently returned from Switzerland and was otherwise well. She had eaten. She was walking, followed by running on a treadmill, felt a little faint for a second or so and then lost consciousness. She was unable to brace herself, fell backwards and sustained a head laceration on the posterior, right back of her head. The event was witnessed by a physician, and she was noted to have rhythmic shaking for 30 to 45 s. She bit her tongue, had post-ictal confusion, but no bowel or bladder incontinence.

The head and cervical spine CT scans were unremarkable. Her brain MRI with contrast was notable for 2 enhancing lesions in the left frontal and temporal lobes (Fig. 1A, B). It was unclear whether these lesions were intra-axial or extra-axial given their small size and peripheral location. Differential of prior infection/granulomatous disease including tuberculosis and sarcoid, small meningiomas, and neoplastic seeding was raised. Her electroencephalogram (EEG) was notable for left temporal slowing with some sharp features (Fig. 1C). Lumbar puncture showed zero red blood cells and white blood cells. Tests for cerebrospinal fluid protein, glucose, gram stain and culture, acid-fast bacilli, cytologic examination, angiotensin-converting enzyme (ACE), and cysticercosis antibody were all negative. Blood erythrocyte sedimentation rate, C-reactive protein, ACE, anti-nuclear antibody (ANA) and antineutrophil cytoplasmic **antibodies** (ANCA) were only notable for positive ANA of 1:320. EKG showed no PR shortening, ST wave abnormalities or QTC prolongation.

The patient underwent repeat imaging and testing 1 month post event. Her head CT was notable for a small region of encephalomalacia in the left frontal region (Fig. 2A). No enhancement was seen on axial T1-weighted MRI following contrast administration (Fig. 2B, C). Her sleep-deprived EEG was normal. The patient was not initiated on anti-convulsant medication and has done well since.

3. Discussion

In this patient, the initial diagnostic testing was misleading. Suggestion was raised of a structural lesion due to an infectious, inflammatory, or malignant process and the EEG provided a correlative focus of potential epileptogenicity. Under these criteria, longer term management with an anticonvulsant might be considered a mainstay of treatment despite the first time presentation of a seizure. Repeat imaging studies in this patient, however, demonstrated a traumatic head injury, likely a coup contrecoup insult [4], that resulted from the impact on the right posterior occiput and caused the left frontal and temporal abnormalities. Repeat EEG also was normal. Resultant seizures have been reported with this type of insult [5]. Given the patient's history, syncope rather than underlying seizure disorder was the likely cause of the episodes of TLOC.

Seizures that occur singly or in a cluster can be triggered by an acute episode such as brain injury. They appear to reflect an acute reaction of the brain to cortical damage with little independent impact on the management of head injury and potential for development of a chronic seizure disorder [6]. Moreover, seizures within the first 2 weeks of a brain injury do not necessarily mean that a chronic seizure disorder will develop, although prophylactic anticonvulsants are effective in reducing seizures in the first week post-injury in adults. Anticonvulsants do not reduce the likelihood of developing after the first week [7]. EEG and MRI findings may help determine whether the condition is likely to become chronic.

Although there continues to be some disagreement surrounding treatment of first time unprovoked GTC seizures [8] [9], our patient's seizure likely was caused by the traumatic head injury and no treatment with anticonvulsant medication was initiated. The patient has since done well without any further episodes. The unusual findings in this very common presentation reinforce the need to understand underlying cause for abnormal tests before making a diagnosis and pursuing a treatment plan.

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References

1. Martikainen KK, Seppa K, Viita PM, et al. Outcome and consequences according to the type of transient loss of consciousness: 1-year follow-up study among primary health care patients. *J Neurol.* 2007; 258:132–6. [PubMed: 20689962]
2. Simon RP. Syncope and transient loss of consciousness. Differential diagnosis and treatment. *West J Med.* 1975; 123:164–70. [PubMed: 1179732]
3. Westby M, Bullock I, Cooper PN, et al. Transient loss of consciousness--initial assessment, diagnosis, and specialist referral: summary of NICE guidance. *BMJ.* 341:c4457.
4. Morrison AL, King TM, Korell MA, et al. Acceleration-deceleration injuries to the brain in blunt force trauma. *Am J Forensic Med Pathol.* 1998; 19:109–12. [PubMed: 9662103]
5. Kross R, Ohler K, Barolin GS. Effect of heading in soccer on the head--a quantifying EEG study of soccer players. *EEG EMG Z Elektroenzephalogr Elektromyogr Verwandte Geb.* 1983; 14:209–12. [PubMed: 6418516]
6. Wiedemayer H, Triesch K, Schafer H, et al. Early seizures following non-penetrating traumatic brain injury in adults: risk factors and clinical significance. *Brain Inj.* 2002; 16:323–30. [PubMed: 11953003]
7. Teasell R, Bayona N, Lippert C, et al. Post-traumatic seizure disorder following acquired brain injury. *Brain Inj.* 2007; 21:201–14. [PubMed: 17364531]
8. Kilpatrick C. An unprovoked tonic-clonic seizure should generally be treated. *J Clin Neurosci.* 2001; 8:187–8. discussion 189. [PubMed: 11484668]
9. Mackenzie R. An unprovoked tonic-clonic seizure should generally not be treated. *J Clin Neurosci.* 2001; 8:188. discussion 189. [PubMed: 11486749]

Figure 1.

Initial MRI and electroencephalogram (EEG) findings: (A) axial fluid-attenuated inversion recovery MRI of the brain showing: (A) a 7 mm × 5 mm lesion in the left frontal region (black arrowhead) and a 9 mm × 6 mm lesion in the anterior left temporal region (black arrowhead); (B) post-contrast axial T1-weighted MRI showing lesion enhancement of both lesions; and (C) the EEG was notable for left temporal slowing with some sharp features (outlined area).

Figure 2.

Repeat imaging 1 month post-event showing: (A) head CT scan with a small region of encephalomalacia (white arrowheads) in the left frontal lobe; (B) axial fluid-attenuated inversion recovery brain MRI of the same lesions in the left frontal and temporal regions; and (C) the same lesions unenhanced on post-contrast axial T1-weighted MRI.