Case Report

RFP Gastroenterology International
Volume 8 Number 1, January - June 2023

Loss of Conciouness is not always Neurological Aetiology, Must not Forget Cardiogenic Event: A Single Case Study

Mayank Chugh[1], Satender Tanwar[2]

How to cite this article:
Mayank Chugh, Satender Tanwar/Loss of Conciouness is not always Neurological Aetiology, Must not Forget Cardiogenic Event: A Single Case Study/RFP Gastroenterology International. 2023;8(1):23-25.

Abstract

Medical education relies upon the through clinical examination and history taking in exact and proper diagnosis of the patient.[1] The patient who has the symptoms which has the presentation of the multiple system sharing the same presentation need[2] judicious examination and through evaluation. Similarly the article case study selected here is the syncope[2] vs Siezure where both have the common feature such as the LOC– Loss of consciousness need the through evaluation for the same.[3]

Differentiating between syncope and seizures, a relatively easy task, is not quite so simple in the ED.[4] Transient loss of consciousness can occur from seizure or syncope, and the emergency clinician must distinguish between the two general[5] conditions, especially if it's the patient's first episode, and direct the appropriate initial evaluation and follow-up.[6]

Ten percent of patients diagnosed as having a seizure do not have a seizure disorder but rather a cardiovascular event that caused transient loss of consciousness.[7] Basic ED labs and an ECG, even an out patient EEG, are not always sensitive enough to differentiate seizures[8] from syncope. Long-term ECG monitoring, as well as tilt table testing, are some tools that can further reveal the origin of the transient loss of consciousness.[9]

Keywords: Cardiogenic; ECG; EEG; Holter; LOC; Syncope; Siezures.

INTRODUCTION

There was no problem calling this patient's event a seizure, and there was no confusing it with syncope.[10] It was observed by physicians, there was a tonic phase of muscular activity, clonic movements lasted 60 seconds,[11] the patient remembered nothing of the event, and he was disoriented for 15 minutes.[12] Differentiating a seizure from syncope in an event that occurred outside the hospital that was witnessed only by non medical personnel makes the task more formidable.[13]

Differentiating between syncope and seizures, a relatively easy task, is not quite so simple in the ED.[14] Transient loss of consciousness can occur from seizure or syncope, and the emergency clinician must distinguish between the two general conditions, especially if it's the patient's first episode, and direct the appropriate initial evaluation and follow-up.[15] If one concludes that the event was

Author Affiliation: [1,2]Consulting Gastroenterologist & Hepatologist, Department of General Medicine and Gastroenterology, Chugh Hospital, Bhiwani 127021 Haryana, India.

Corresponding Author: Satender Tanwar, Consulting Gastroenterologist & Hepatologist, Department of General Medicine and Gastroenterology, Chugh Hospital, Bhiwani 127021 Haryana, India.

E-mail: drsatendertanwar@gmail.com

Received on: 20.09.2023

Accepted on: 03.10.2023

syncope, it's usually from a cardiovascular event, and some can be serious or even fatal.[16]

If one believes that the episode was caused by a seizure, neurological testing and consultation is required. That would seem to straight forward, but 10-20 percent of patients diagnosed as having a seizure do not have a seizure disorder but rather a cardiovascular event that caused transient loss of consciousness.[17] A seizure resulting from syncope is termed convulsive syncope, and seizure activity occurs in up to 20 percent of episodes of syncope. Seizures can result from an occult cardiac etiology, and some causes, such as an episodic arrhythmia, can escape elucidation in the ED. Basic ED labs and an ECG, even an outpatient EEG, are not always sensitive enough to differentiate seizures from syncope. Long-term ECG monitoring, as well as tilt table testing,[18] are some tools that can further reveal the origin of the transient loss of consciousness. The commonly used short-term Holter cardiac monitor is a popular intervention but of minimal actual value.[19]

Most clinicians would simply believe the diagnosis if a patient comes to the ED with loss of consciousness and gives a history of prior seizures. He would then check the anticonvulsant level, adjust any necessary derangements in the drug levels, and send the patient on his way.[20] We usually add another drug or attribute seizures to noncompliance. As it turns out, a significant number of patients who are told they have a seizure disorder actually do not. This may be one reason we see patients on anticonvulsants who continue to seize. It's important to take a good history to distinguish seizure from syncope with the hope of getting the patient directed to the right consultant.[21]

CASE STUDY

- A 56 years old male presented with repeated episodes of LOC.
- He has been evaluated by neurosurgeon and investigated same.
- After investigation started on the AED – Anti -epileptic Drugs.
- Even after the AED he was continues to have LOC.
- No ECG and Cardiac evaluation done at primary level.

Thereby one day when he had the LOC he was taken to the ED at Physician available nearby where he had been evaluated and found to have ECG Suggestive of CHB.

- ECG repeat which was suggestive of same CHB.

CONCLUSION

The above said patient presented with the complaints of repeated episode of syncope and LOC which either have cardiogenic as well as neurological cause which needs the judicious evaluation and thorough examination.[22]

The case initially treated by the neurosurgeon, started with the antiepileptic's but patient continues to have the loss of consciousness and similar complaints even after starting the AEDs, that's is suggestive of that pathology is not of neurological originated. The case later evaluated by physician and found out to CHB which life is threatening if not treated at timely manner.[23]

REFERENCES

1. Seltzer S., McCabe B. F. Perilymph fistula: the Iowa experience. *Laryngoscope*. 1986;96:37–49.
2. Minor L. B., Solomon D., Zinreich J. S., *et al.* Sound- and/or pressure-induced vertigo due to bone dehiscence of the superior semicircular canal. *Arch Otolaryngol Head Neck Surg*. 1998;124:249–258.
3. Carey J. P., Minor L. B., Nager G. T. Dehiscence or thinning of bone overlying the superior semicircular canal in a temporal bone survey. *Arch Otolaryngol Head Neck Surg*. 2000;126:137–147.
4. Headache Classification Subcommittee of the International Headache Society. *The International Classification of Headache Disorders. 2nd ed*. Oxford, England: Blackwell Publishing; 2003. pp. 24–36.
5. Neuhauser H. K., Leopold M., von Brevern M., *et al.* The interrelations of migraine, vertigo, and migrainous vertigo. *Neurology*. 2001;56:436–441.
6. Neuhauser H. K., Lempert T. Diagnostic criteria for migrainous vertigo. *Acta Otolaryngol*. 2005;125:276–279.
7. Kim H. Y., Chung C. S., Moon S. Y., *et al.* Complete nonvisualization of basilar artery on MR angiography in patients with vertebrobasilar ischemic stroke: favorable outcome factors. *Cerebrovasc Dis*. 2004;18:269–276.
8. Grad A., Baloh R. W. Vertigo of vascular origin. Clinical and electronystagmographic features in 84 cases. *Arch Neurol*. 1989;46:281–284.
9. Colman N., Nali K., Ganzeboom K. S., Shen W. K., Reitsma J., Linzer M., *et al.*. (2004). Epidemiology of reflex syncope. *Clin. Auton*. Res. 14 Suppl. 1, i9–i17. 10.1007/s10286-004-1003-3.
10. Goldstein D. S., Sharabi Y. (2009). Neurogenic

orthostatic hypotension: a pathophysiological approach. *Circulation* 119, 139–146. 10.1161/Circulation AHA.108.805887.

11. Healey J., Connolly S. J., Morillo C. A. (2004). The management of patients with carotid sinus syndrome: is pacing the answer? *Clin. Auton.* Res. 14Suppl. 1, 80–86. 10.1007/s10286-004-1012-2.
12. Kapoor W. N. (2002). Current evaluation and management of syncope. *Circulation* 106, 1606–1609. 1161/01.CIR.0000031168.96232.BA.
13. Lewis D. A., Dhala A. (1999). Syncope in pediatric patient. *Pediatr. Clin. North Am.* 46, 205–219. 10.1016/S0031-3955(05)70113-9.
14. Lewis T. (1932). A lecture on vasovagal syncope and the carotid sinus mechanism. *Br. Med. J.* 873–876. 10.1136/bmj.1.3723.873.
15. Lipsitz L. A., Wei J. Y., Rowe J. W. (1985). Syncope in an elderly institutionalized population: incidence, prevalence and associated risk. *Q. J. Med*. 55, 45–54.
16. Luciano G. L., Brennan M. J., Rothbberg M. B. (2010). Postprandial hypotension. *Am. J. Med*. 123, e1–e6. 10.1016/j.amjmed.2009.06.026.
17. Medow M. S., Stewart J. M., Sanyal S., Mumtaz A., Sica D., Frishman W. H. (2008). Pathophysiology, diagnosis and treatment of orthostatic hypotension and vasovagal syncope. *Cardiol. Rev.* 16, 4–20. 10.1097/CRD.0b013e31815c8032.
18. Moya A., Sutton R., Ammirati F., Blanc J. J., Brignole M., Dahm J. B., *et al*.. (2009). Guidelines for the diagnosis and management of syncope. *Eur. Heart J.* 30, 2631–2671. 10.1093/eurheartj/ehp298.
19. Numerosos F., Mossini G., Lippi G., Cervellin G. (2013). Evaluation of the current progno-stic role of cardiogenic syncope. *Intern. Emerg. Med*. 8, 69–73. 10.1007/s11739-012-0889-3.
20. Osiro S., Zurada A., Gielecki J., Shoja M. M., Tubbs R. S., Loukas M. (2012). A review of subclavian steal syndrome with clinical correlation. *Med. Sci. Monit.* 18, RA57–RA63. 10.12659/MSM.882721.
21. Potter B. J., Pinto D. S. (2014). Subclavian steal syndrome. *Circulation* 129, 2320–2323. 10.1161/Circulation AHA.113.006653
22. Raj S. R., Coffin S. T. (2013). Medical therapy and physical maneuvers in the treatment of the vasovagal syncope and orthostatic hypotension. *Prog. Cardiovasc. Dis.* 55, 425–433. 10.1016/j.pcad.2012.11.004.
23. Rosanio S., Schwarz E. R., Ware D. L., Vitarelli A. (2013). Syncope in adults: systematic review and proposal of a diagnostic and therapeutic algorithm. *Int. J. Cardiol.* 162, 149–157. 10.1016/j.ijcard.2011.11.021.