Medicina Universitaria. 2015;17(67):122–125

MEDICINA UNIVERSITARIA

www.elsevier.es/rmuanl

EXPERT'S CORNER: A PERSONAL APPROACH

Is it an epileptic seizure?

C.E. Muñiz-Landeros*

Neurology Service of the ''Dr. José Eleuterio González'' University Hospital at the UANL, Monterrey, N.L., Mexico

Received 20 January 2015; accepted 21 January 2015
Available online 7 May 2015

One of the most frequent situations which occur in medical practice is the moment of categorically defining if the symptomatology to which a patient or another reliable source refers corresponds to an epileptic seizure or any of the clinical events presented as differential diagnoses, ranging from lypotimia, a syncope, hyperventilation syndrome, anxiety, somatization, conversion or pseudo seizure.[1,2]

The main element physicians have is the information provided by the patient or the person accompanying the patient. It depends on the way the doctor obtains precise data for the symptomatology study in question, that is, the semiology. Thus, it is essential to keep in mind that clinical training is substantial to collect only useful, complete information and discard data which can cause distraction or confusion. Therefore, the semiology study must include very specific characteristics like: type of symptom, form of onset, mode of installation, duration, intensity and quality of the symptoms, as well as temporal evolution, frequency of presentation, precipitating factors, or symptoms accompanying or following initial onset, in addition to the symptomatology which exacerbates or diminishes the symptom. This information will be best evaluated if obtained from the patient or a trustworthy source who had witnessed the event. This will make it possible to obtain the highest degree of data, which will be useful for the study of the symptom.

In medical literature a series of useful questions for the study of these types of clinical phenomena has been reported:

- Is there a symptom premonitory to the seizure?
- What is that symptom?
- Which is the pattern of presentation of the seizure?
- How long does the seizure last?
- Does the patient keep his state of consciousness during the seizure?
- What symptoms are there after the seizure?
- How long does the patient take to recover?
- How frequent are the seizures?
- Are there any changes in the patient with treatment?

These 10 questions allow us to cover the minimum required information to begin the study of a patient's symptomatology.

A well-structured clinical history is key for diagnosis, because there are several medical conditions which can mimic convulsive and non-convulsive epileptic seizures, either partial or generalized. Differential diagnosis will vary according to the patient's age, medical co-morbidity and characteristics of presentation. For example, generalized tonic–clonic seizures can be confused with a ''convulsive syncope'' or non-epileptic psychogenic events; moreover, temporal lobe epilepsy can be confused with panic attacks or a cognitive disorder. Patients must be evaluated based on their personal pathological background and establish the possibility of presenting potential risk factors for developing epileptic seizures, like: a head injury, encephalitis, meningitis, febrile seizures, or a family history of epilepsy.

The presence of past medical history of one or more of these risk factors may help strengthen a clinical diagnosis of epilepsy when a patient presents recurrent seizures. At the same time, it can also be useful in locating the onset of the seizures. For example, a history of complicated febrile seizures is observed more commonly in patients with mesial temporal lobe epileptic seizures. Simple febrile epileptic seizures are really common and are not considered a risk

* Corresponding author at: Ave. Francisco I. Madero y Gonzalitos Col. Mitras Centro, Monterrey, N.L. C.P. 64460, Mexico.
E-mail address: drmuniz_landeros@yahoo.com.mx

http://dx.doi.org/10.1016/j.rmu.2015.03.002

factor in the development of epilepsy.[3] Reports show that nearly 10% of the population may experience some epileptic seizure throughout their lives.[4]

Seizure classification is more than a simple academic exercise, because it can determine subsequent decisions in its evaluation and treatment. Epileptic seizures can be divided into **provoked** and **non-provoked seizures**. Provoked seizures are denominated, acute and symptomatic and are attributed to an acute central nervous system injury (CNS), or a metabolic alteration like hypoglycemia. By definition, provoked seizures occur close in time to the triggering event. These seizures represent about 40% of all seizures (excluding pediatric febrile seizures) and have an incidence of 29 to 39/100,000 habitants.[5,6]

They can occur at any other age, but are more frequent during childhood and adulthood. In adults the most frequent etiologies are cerebrovascular disease, drug or alcohol suppression, brain injury and cerebral neoplasias.[4] These are usually isolated, non-recurrent seizures, thus the treatment is aimed at the primary etiology and antiepileptic medications are not required regularly.

Epilepsy is a condition with a tendency of non-provoked recurrent seizures and is generally diagnosed by having presented two or more episodes. Non-provoked seizure incidence in the general population is 57 to 63/100,000 habitants and for epilepsy is 46 to 48/100,000 habitants.[7–9] Epilepsy is not considered a single disease, but a group of alterations which have the presence of seizures (convulsive and non-convulsive epileptic) in common.

Non-provoked seizures are a result of chronic or functional structural alterations, which affect the neuronal cortical function and are denominated symptomatic. In adults, the most common causes are brain injury, cerebrovascular disease and cerebral neoplasias. [7–9] Among the elderly, cerebrovascular disease is responsible for two-thirds of symptomatic seizures, followed by neurodegenerative alteration.[7–9] In most cases the etiology can be unknown. When a structural injury or functional alteration is suspected but not proven, the epilepsy is classified as cryptogenic.

Idiopathic epilepsy is considered genetically mediated. In some papers described in medical literature the reported incidence ranges from 33 to 42% symptomatic, from 21 to 53% cryptogenic and 14 to 37% idiopathic. [9,10]. Non-provoked seizures can occur at any age. However, its highest incidence is in children and the elderly. Non-provoked seizures are subdivided by their anatomic origin and by the specific epileptic syndrome. The types of generalized primary seizures include: generalized tonic–clonic, myoclonic, atonic, tonic, clonic and absences.[7–9] Generalized seizures are more frequent during childhood and adolescence. [9]. Partial seizures start in a region of the brain and may be subdivided into those that remain aware (simple partial) or those where awareness is affected (complex partial). Some may progress into generalized and are called secondarily generalized. First-time epilepsy in adults and elderlies is usually partial.[9] Partial seizures start more frequently in the frontal and temporal lobes.[11]

These types of seizures have distinctive characteristics and can be identified by their clinical description. The seizures originating at the parietal and occipital lobes are characterized by sensitivity and visual phenomena, respectively. For the syndromic classification of epilepsy the following information is needed: type of seizure, associated neurological symptoms, age of onset, electroencephalographies and family or genetic history. This will allow a proper prognosis and treatment.[12]

The physician must keep in mind that a convulsive or non-convulsive epileptic seizure is subdivided into three stages: **pre-ictal, ictal and post-ictal**:

Pre-ictal period – characteristic precipitant factors are rarely reported. These occur frequently during the sleeping period (frontal lobe seizures). Some patients report non-specific neurological lateralization premonitory symptoms, like: dizziness, a strange feeling, focal paresthesias, and complex abdominal, olfactory, hearing and visual sensations. Fainting, acral paresthesias and palpitations can indicate hyperventilation. It is important to mention that any real epileptic seizure cannot be ''induced''.[12]

Ictal period – During this stage the mental state is altered, with poor or no response. The stereotyped manifestation is considered characteristic in epileptic seizures, thus considering certain characteristics to be ''typical'' in some epileptic seizures, for example, in Jacksonian seizures, or frontal lobe epilepsy. In these ones, they present a prone position, atonic posture with abduction of upper extremities, a short duration, and occurrence during sleep. In all seizure types, there is usually an abrupt onset (sudden) between 1 and 2 min long. The patient displays a poor response to verbal stimuli. This may sometimes occur during complex partial seizures (CPS). In the motor phase of the generalized clonic–tonic convulsive seizures (GCTCS) there is a tonic posture or clonic response of the upper and lower extremities, varying according to the onset and whether it is partial motor with generalization or generalization from onset. The opisthotonic posture ''*arc de cercle*'' is never adopted; there may be intense body movements during frontal lobe epilepsy, as well as lateral head movements, but the version of lateral rotation of the head is more frequent in the GCTCS. In case of pelvic movement, it will be toward the posterior region, and never toward the front. In this tonic phase of the GCTCS the rigid posture is characteristic, there is rarely a fluctuating pattern or motor response pauses and it is usually continuous. There is no motor response to any external stimuli. There is vocalization or ''*epileptic scream*'', characterized by a stertortous sound of moderate intensity. Palpebral closure is extremely rare, if it occurs there is no resistance to manual opening, it generally tends to occur during ocular opening with an occasional deviated look. There are no visual fixations to head rotation maneuvers (Henry and Woodruff sign), which consist of: ''the eyes open and the head rotates to the sides, then the gaze diverts to the ground, in the contralateral rotation the eyes deviate again to the ground''.

Extensor plantar response occurs (Babinski sign) bilaterally, usually in the 5 min subsequent to the seizure. The presence of urinary incontinence is frequent in GCTCS. Occasionally, there is a tongue injury, more often than not in the lateral regions. The respiratory pattern is usually regular, interrupted by the tonic phase, ''noisy'', intense and prolonged. Corneal reflex response is altered.

Post-ictal phase-In this stage the degree of recovery is generally insidious and sometimes prolonged. There is rarely a full sudden recovery. The patient presents a state of confusion, headache, lethargy, or he may refer to having fatigue or

diffuse myalgias. As a general rule, he/she does not remember the ictal event. It is important that a physician does not assume that the ''first seizure'' is in fact the ''first seizure''.

Accumulated incidence of the presence of epileptic seizures for age 74 in most reported studies in the literature is close to 4–5%.[13,14] The risk of relapse after an isolated non-provoked seizure is close to 30%.[15] A history of seizures can modify the risk; therefore, proper interrogation must be done. It is important to interrogate about the ''spaced out'' episodes, independently from their length. This will rule out the possibility of absence seizures or complex partial seizures. It is important to obtain information about whether or not the patient woke up with oral injuries, like tongue bites, or urinary incontinence. This will help detect the possibility of epileptic seizures during the sleep phase. Likewise, the presence of involuntary movements like muscular twitches right after waking up, which would rule out the possibility of myoclonus or myoclonic seizures. Some patients may report myoclonic movements of the physiological type at the beginning of the sleep phase or in a state of somnolence, these are not considered pathological.

The presence of abnormal movements of the convulsive type is not always an indicator that we are dealing with epilepsy, we must rule out the possibility of convulsive syncope. Therefore, we will approach the patient if there is a trigger factor (trauma, excessive heat, the sight of blood), which could trigger a vasovagal syncope.[16]

Patients with convulsive syncope usually report premonitory symptoms, which could be explained by a cerebral hypoperfusion, such as: loss of equilibrium, dizziness, flaccidness, reduced vision (darkened or blurry), tunnel vision, palpitations, diffuse cold and hot sensations and muscle tone loss. Diaphoresis and paleness are usually present. The patient presents a sudden, complete recovery, and in this phase myoclonic, short and generally transitory abnormal movements are reported.[17–19]

The presence of stereotyped anxiety symptoms may be confused with either a panic attack, or frontal lobe epilepsy. In the latter, the anxiety symptoms are followed by confusion, alteration in response or loss of consciousness, in addition to being associated with automatisms (repetitive movements of the mouth or extremities). However, there are reports of ictal events of fear and panic in frontal or parietal lobe epileptic seizures.[20,21]

The presence of nonspecific or ''strange'' attacks is not always psychogenic, as in frontal lobe seizures, since there are reports of sudden ''pedaling-type'' stereotyped movements or ''bed shakes'' with a short duration. In these cases, history and the use of simple or videoed electroencephalograms during sleep phases will help diagnosis.[7,8] The presence of forced or palpebral closure with resistance suggests a mainly psychogenic event.[22,23] This is associated with intense shaking movements, anterior pelvic movements, swinging head movements, emotional or behavioral changes, ictal ''stuttering'' or the presence of ''multiple seizures'' at the moment of clinical consultation.[23,24]

The electroencephalogram (EEG) is a tool used to confirm an epileptic seizure diagnosis, as well as to classify the type of epilepsy and epileptic syndrome. We must keep in mind that obtaining an EEG with ictal activity in a standard ambulatory study in adult patients is not common; nevertheless, interictal epileptiform discharges (IEDs) are highly linked to a tendency to clinical seizures. A normal EEG does not rule out a clinical diagnosis of epilepsy. Epileptiform discharges are present in the initial EEG only in 25–50% of patients with epilepsy, since the sensitivity of EEGs may vary considerably depending on the frequency of the seizures, epileptic syndrome and site location of the seizure. EEG's diagnostic performance is incremented with low stimulation studies like: sleep deprivation, hyperventilation and photostimulation, in addition to using the prolonged video-electroencephalogram technique. It is important to keep in mind that there are abnormal variants of non-epileptic cerebral graphoelements, since this could lead to an incorrect diagnosis, hence the diagnosis interpretation must be done within the context of clinical history.[25–28]

Conclusion

Once again, like in most situations in medical attention, performing a complete clinical history with all its semiologic elements of study is the diagnostic cornerstone in ''seizure'' cases. A proper integration of the characteristics of presentation will allow us to make a probable diagnosis for epileptic events (convulsive or non-convulsive) more precisely. Thus, being able to make an accurate differential diagnosis, among the main medical causes, in addition to being able to create a proper diagnostic approach with laboratory studies and paraclinical procedures like electroencephalogram, or neuroimaging if necessary, and being able to begin an efficient medical treatment, avoiding the possibility of sub-diagnostics, over-diagnostics or iatrogeny.

Conflict of interest

The authors have no conflicts of interest to declare.

Funding

No financial support was provided.

References

1. Hopp J. Neurologic pearls. Semin Neurol. 2010;30:82–5.
2. Noe KH. Seizures: diagnosis and management in the outpatient setting. Semin Neurol. 2011;31:54–64.
3. Nelson KB, Ellenberg JH. Predictors of epilepsy in children who have experienced febrile seizures. N Engl J Med. 1976;295:1029–33.
4. Berg AT, Shinnar S. The risk of seizure recurrence following a first unprovoked seizure: a quantitative review. Neurology. 1991;41:965–72.
5. Annegers JF, Hauser WA, Lee JR, Rocca WA. Incidence of acute symptomatic seizures in Rochester, Minnesota, 1935–1984. Epilepsia. 1995;36:327–33.
6. Loiseau J, Loiseau P, Guyot M, et al. Survey of seizure disorders in the French southwest. I. Incidence of epileptic syndromes. Epilepsia. 1990;31:391–6.
7. Hauser WA, Annegers JF, Kurland LT. Incidence of epilepsy and unprovoked seizures in Rochester, Minnesota: 1935–1984. Epilepsia. 1993;34:453–68.

8. MacDonald BK, Cockerell OC, Sander JW, Shorvon SD. The incidence and lifetime prevalence of neurological disorders in a prospective community-based study in the UK. Brain. 2000;123:665–76.
9. Olafsson E, Ludvigsson P, Gudmundsson G, et al. Incidence of unprovoked seizures and epilepsy in Iceland and assessment of the epilepsy syndrome classification: a prospective study. Lancet Neurol. 2005;4:627–34.
10. Loiseau P, Loiseau J, Picot MC. One-year mortality in Bordeaux cohort: the value of syndrome classification. Epilepsia. 2005;46 Suppl. 11:11–4.
11. Manford M, Hart YM, Sander JW, Shorvon SD. National General Practice Study of Epilepsy (NGPSE): partial seizure patterns in a general population. Neurology. 1992;42:1911–7.
12. Krumholz A, Wiebe S, Gronseth G, et al., Quality Standards Subcommittee of the American Academy of Neurology; American Epilepsy Society. Practice parameter: evaluating an apparent unprovoked first seizure in adults (an evidencebased review): report of the Quality Standards Subcommittee of the American Academy of Neurology and the American Epilepsy Society. Neurology. 2007;69:1996–2007.
13. Forsgren L, Bucht G, Eriksson S, et al. Incidence and clinical characterization of unprovoked seizures in adults: a prospective population-based study. Epilepsia. 1996;37:224–9.
14. Hauser WA, Annegers JF, Rocca WA. Descriptive epidemiology of epilepsy: contributions of population-based studies from Rochester, Minnesota. Mayo Clin Proc. 1996;71:576–86.
15. Annegers JF, Shirts SB, Hauser WA, Kurland LT. Risk of recurrence after an initial unprovoked seizure. Epilepsia. 1986;27:43–50.
16. Morrell MJ. Differential diagnosis of seizures. Neurol Clin. 1993;11:737–54.
17. Abboud FM. Neurocardiogenic syncope. N Engl J Med. 1993;328:1117–20.
18. Benbadis SR, Wolgamuth BR, Goren H, et al. Value of tongue biting in the diagnosis of seizures. Arch Intern Med. 1995;155:2346–9.
19. Benbadis SR. Tongue biting as a lateralizing sign in partial epilepsy. Seizure. 1996;5:175–6.
20. Sazgar M, Carlen PL, Wennberg R. Panic attack semiology in right temporal lobe epilepsy. Epileptic Disord. 2003;5: 93–100.
21. Alemayehu S, Bergey GK, Barry E, et al. Panic attacks as ictal manifestations of parietal lobe seizures. Epilepsia. 1995;36:824–30.
22. Syed TU, Arozullah AM, Suciu GP, et al. Do observer and self-reports of ictal eye closure predict psychogenic nonepileptic seizures? Epilepsia. 2008;49:898–904.
23. Vossler DG, Haltiner AM, Schepp SK, et al. Ictal stuttering: a sign suggestive of psychogenic nonepileptic seizures. Neurology. 2004;63:516–9.
24. Burneo JG, Martin R, Powell T, et al. Teddy bears: an observational finding in patients with non-epileptic events. Neurology. 2003;61:714–5.
25. Worrell GA, Lagerlund TD, Buchhalter JR. Role and limitations of routine and ambulatory scalp electroencephalography in diagnosing and managing seizures. Mayo Clin Proc. 2002;77: 991–8.
26. Benbadis SR. Errors in EEGs and the misdiagnosis of epilepsy: importance, causes, consequences, and proposed remedies. Epilepsy Behav. 2007;11:257–62.
27. Benbadis SR, Tatum WO. Overinterpretation of EEGs and misdiagnosis of epilepsy. J Clin Neurophysiol. 2003;20: 42–4.
28. Krauss GL, Abdallah A, Lesser R, et al. Clinical and EEG features of patients with EEG wicket rhythms misdiagnosed with epilepsy. Neurology. 2005;64:1879–83.