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Review

Focal EEG abnormalities and focal ictal semiology in generalized epilepsy

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ABSTRACT

In clinical practice, the diagnosis of focal vs generalized epilepsy dictates the management of the patient. The distinction between generalized and focal epilepsy is at times imperfect and some epilepsies have features that fall in between these two extremes. An example is the occurrence of focal interictal and focal ictal abnormalities in generalized epilepsies. As a part of the special issue on "The epileptogenic zone in pediatric epilepsy surgery", this focused narrative review will discuss different focal abnormalities seen in generalized epilepsy. An overlap of focal and generalized epileptiform abnormalities may support a continuum between focal and generalized epilepsy. When evaluating patients in the "gray zone", other factors such as ictal semiology, neuroimaging, genetic testing and functional deficits may need to be considered to reach an accurate diagnosis. Being aware of possible occurrence of focal clinical and EEG features in generalized epilepsy will help clinicians select more preferred AED (s), avoiding potential iatrogenic side effects and inappropriate consideration for epilepsy surgery.

1. Introduction

In clinical practice, the dichotomy of focal vs generalized epilepsy dictates the choice of antiepileptic drugs (AEDs) for treatment and subsequent management of epileptic patients. In general, patients with focal epilepsy are good candidates for excisional epilepsy surgery, whereas those with generalized epilepsy are not. In most cases, the diagnosis of focal vs generalized epilepsy is clear after a careful consideration of the seizure semiology and results from diagnostic testing, namely video electroencephalography (VEEG), neuroimaging, and genetic testing.

While there are some variations by different authors to date, several proposed theories attempt to explain the pathophysiology of generalized epilepsy. A brief introduction of the three theories outlined below provide a theoretical underpinning to further discussion of generalized epilepsy. For detailed discussions of the proposals, the readers are encouraged to review the corresponding references.

Penfield and Jasper [1] described the centrencephalic theory based on a generator seated in the midline subcortical gray matter, the thalamus. Later on, Gloor [2] added a concept of a diffuse cortical hyperexcitability as an essential factor in generalized epilepsies which would respond to volleys from the thalamus (cortical-reticulo theory). Lastly, the cortical theory stated that only diffuse, hyperexcitable cortex is solely responsible for the epilepsy, disregarding a deep midline generator [3].

The purpose of this review is to discuss a selected body of literature published mostly in the early 1990's and 2000's on the topic of focal abnormalities in generalized epilepsy, with a focus on idiopathic generalized epilepsy (IGE), specifically juvenile myoclonic epilepsy (JME). Although JME may have a genetic basis given that about one third of the patients have family history of epilepsy [4], the few reported genes at the present do not sufficiently account for the phenotype of JME.The diagnostic criteria of JME is well established owing to the criteria outlined by Janz [4] and Wolf [5]. The referenced studies in this review have been selected for rigorous inclusion criteria, including interictal and ictal VEEG recording. The term "generalized epilepsy" refers to all types of generalized epilepsies, while "IGE or GGE" refers to specific types of generalized epilepsy. In addition, semiological seizure classification was used to highlight localizing and lateralizing signs.

In this review, we hope to underscore the fact that focal clinical and EEG features in generalized epilepsy may mimic focal epilepsy. Being aware of possible occurrence of these features in generalized epilepsy

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will aid clinicians select more preferred AED (s), avoiding potential iatrogenic side effects and inappropriate consideration for epilepsy surgery. In this review, we have used the "generalized epilepsy" as a more inclusive term unless specified otherwise.

2. Anamnesis

During anamnesis it is crucial to separate the description of a seizure given by the patient from that given by the witness. In addition, the patient should be reminded not to describe what he/she was told, but only what he/she remembers. Following these simple rules will often help discern between epileptic vs non-epileptic paroxysmal event and generalized vs focal epilepsy. However, focal signs especially during bilateral tonic clonic seizures are more difficult to reliably elicit during an interview. This difficulty is most likely related to the "untrained eye" of a distressed witness, who is obviously more focused on the safety of the patient rather than on the observation of the ictal symptoms. Further, an observer may only report a focal feature during a generalized tonic-clonic (GTC) or myoclonic seizure (in a patient with generalized epilepsy) that may lead to an incorrect preliminary diagnosis of focal epilepsy with incorrect lateralization. Therefore, the most accurate way to analyze ictal semiology and lateralizing signs is with a VEEG.

3. Focal interictal EEG abnormalities in generalized epilepsy

Numerous studies have demonstrated *focal interictal* abnormalities in patients with generalized epilepsy, mostly JME (Fig. 1, Table 1). Focal interictal abnormalities found in these studies were intermittent focal slowing, asymmetric generalized epileptiform discharges, and independent focal epileptiform discharges – all mimicking the diagnosis of focal epilepsy. These abnormalities usually, but not always, shifted in locations. The selected studies reported the occurrence of *focal interictal* abnormalities in 14 %–56 % of the patients. Most of the studies reported the frequency of *independent focal epileptiform* activity (mimicking focal epilepsy) occurring in 7–35 % of the patients. The occurrence of these abnormalities was variable among different studies. This variability is probably related to differences in the length of EEG recordings (the longer the recording, the more likely to capture a particular abnormality) and the threshold set by each reader who classifies a waveform as epileptiform.

4. Focal ictal EEG abnormalities in generalized epilepsy

A limited number of studies exist in the area of focal *ictal* EEG abnormalities in generalized epilepsy. Three selected studies utilizing VEEG monitoring illustrate these occurrences. Usui et al. [13] studied 26 patients with JME in the epilepsy monitoring unit (EMU). The diagnosis of JME was based on the results of ictal and interictal VEEG recording. Three patients (11 %) exhibited lateralized ictal activity during generalized tonic clonic seizures (GTCs). The initial EEG seizure patterns were generalized, but after a few seconds paroxysmal alpha waves lateralized to one hemisphere. If a focal symptom like a head version occurred, this symptom was contralateral to the lateralized paroxysmal activity. In the second study, Oguni et al. [15] analyzed 302 myoclonic seizures in five patients with JME using VEEG. He and associates used the criteria of JME by Wolf, 1992. Asymmetry of the myoclonic jerks was noted in 4/5 patients. Predominance of one side or the other was variable. Furthermore, the authors also described patients who reported unilateral myoclonic jerks, but indeed these jerks had bilateral myoclonic jerks when analyzed on VEEG. The diagnosis of JME in the above two studies is likely reliable given the established diagnostic criteria of JME by Janz [4] and Wolf [5]. A third retrospective study by Linane et al. [16] reported six patients with idiopathic generalized epilepsy (IGE) with similar lateralized ictal EEG findings correlating with contralateral focal symptoms, like the head version or fencing posturing. The diagnosis of IGE was reached based on epilepsy characteristics and risk factors, brain MRI results, neurologic examinations, ictal and interictal EEG tracings, as well as video-recorded semiology. Because the authors analyzed multiple clinical, neuroimaging, and neurophysiological data the validity of the diagnosis is probably high. The results of these studies at the least should alert clinicians that focal semiological features during a GTC seizure need to be cautiously interpreted.

5. Focal ictal semiological findings in generalized epilepsy

Generalized interictal epileptiform discharges support the diagnosis of generalized epilepsy, while there may be some exceptions. There are characteristics interictal and ictal EEG patterns that correlate with and produce specific ictal symptomatology. For example, bursts of 3 Hz spike and wave complexes correlate with and may produce absence seizures, while bursts of slow spike and wave complexes correlate with and may produce atypical absence seizures. An interictal EEG pattern of

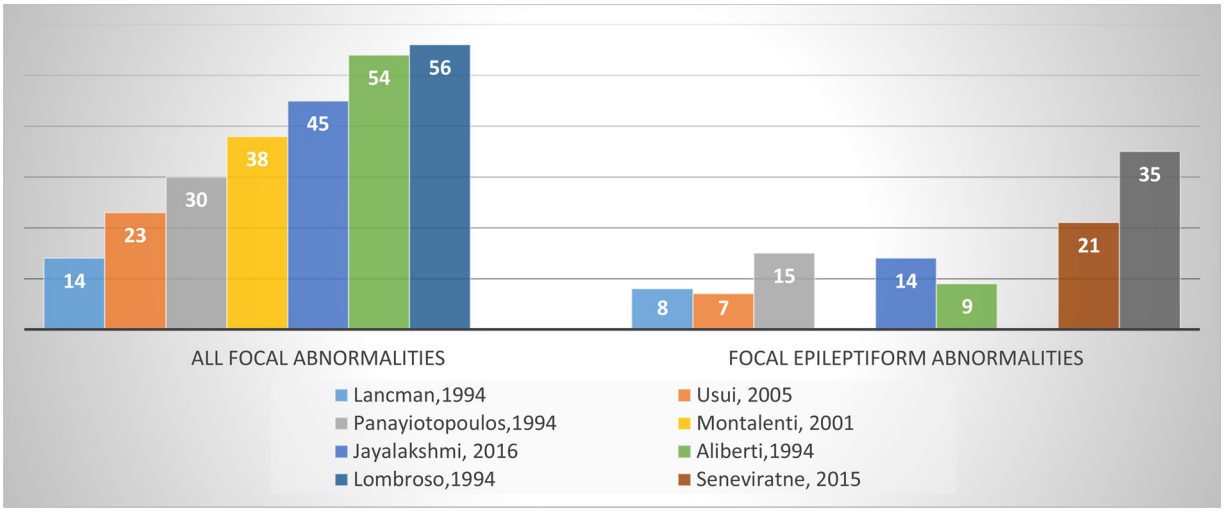

Fig. 1. Focal interictal EEG features in generalized epilepsy: A summary of selected studies illustrates the percentage of *focal interictal* (epileptiform and non-epileptiform) abnormalities in patients with generalized epilepsy. See corresponding details in Table 1.
All focal abnormalities (left half of the figure): *All* focal abnormalities as reported in each study.
Focal epileptiform abnormalities (right half of the figure): Only the focal *epileptiform* abnormalities extracted from *all* focal abnormalities.

Table 1
A summary of selected studies illustrating the percentage of *focal interictal*, non-epileptiform and epileptiform, EEG abnormalities in patients with generalized epilepsy.

Study	Epilepsy	# of patients	Focal abnormalities[a] (% of total # of patients)	IEA (% of total # of patients)
Seneviratne et al. [6]	GGE	120	89 %	66.4 %
Panayiotopoulos et al. [7]	JME	66	30%	15 %
Aliberti et al. [8]	JME	22	54%	9 %
Lancman et al. [9]	JME	85	14 %	7 %
Lombroso [10]	IGE	58	56%	–
Montalenti et al. [11]	JME	63	38 %	–
Leutmezer et al. [12] (VEEG)	IGE	20	–	35 %
Usui et al. [13] (VEEG)	JME	26	23%	15 %
Jayalakshmi et al. [14].	JME	202	45 %	14 %

Legend: IEA: interictal epileptiform abnormalities.
JME: juvenile myoclonic epilepsy.
GGE: genetic generalized epilepsy (childhood absence epilepsy, genetic generalized epilepsy unspecified, juvenile absence epilepsy, juvenile myoclonic epilepsy, generalized epilepsy with tonic-clonic seizures only).
IGE: idiopathic generalized epilepsy.
Dash: indicates unavailable datum.
[a] See corresponding graphs in Fig. 1.

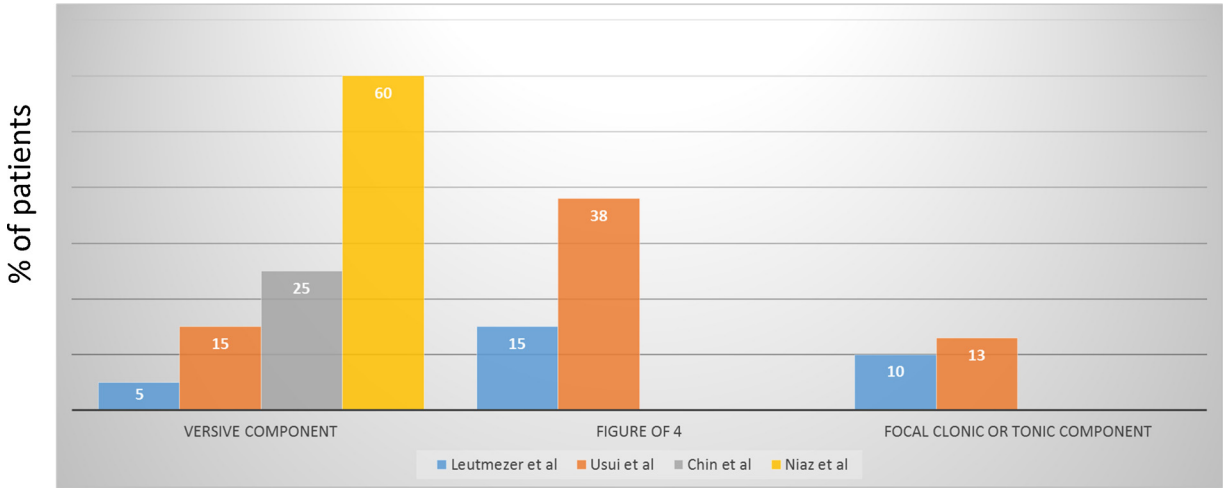

Fig. 2. Focal ictal semiology in generalized epilepsy: A summary of selected studies shows the percentage of patients with three different *focal ictal* symptoms, mimicking lateralizing signs during generalized tonic-clonic or generalized clonic seizures. See corresponding details in Table 2.

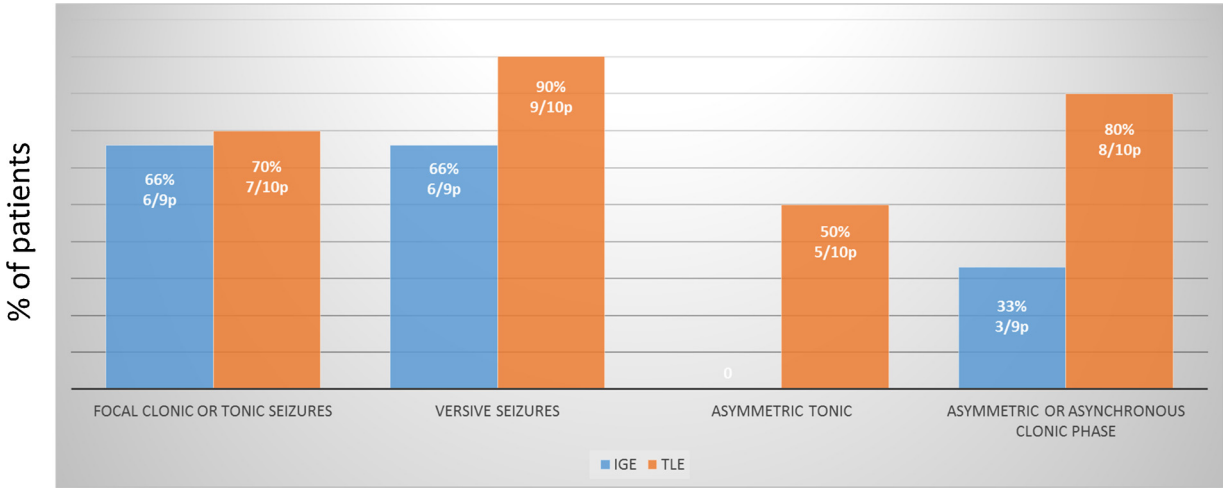

Fig. 3. Result of study by Niaz et al. [20] illustrating focal symptomatology in bilateral tonic-clonic seizures in generalized vs focal epilepsy. See corresponding details in Table 2.

Table 2
Focal ictal semiology in generalized epilepsy: A summary of selected studies shows the percentage of patients with different *focal ictal* symptoms, mimicking lateralizing signs during generalized tonic clonic or generalized clonic seizures. See accompanying supplemental information summarizing each study.

VEEG study	# of Patients	Type of epilepsy	Figure-of-4 sign[b]	Focal tonic[c]	Postictal nose wiping	Focal myoclonic	Focal clonic[c]	Versive[a]	Postictal hemi-paresis	Asymmetric seizure termination after GTC	Lateralizing symptoms	Focal ictal symptoms
Leutmezer et al. [12]	20	IGE	4 (15 %)	2(10 %)	2 (10 %)	–	–	1 (5 %)	1 (5 %)	–	7 (35 %)	–
Usui et al. [13]	26	JME	5 (38 %)	–	–	11 (42 %)	2 (15 %)	2 (15 %)	–	–	12 (46 %)	always on same side
Chin and Miller [21]	20	IGE	–	–	–	–	–	5 (25 %)	–	–	–	variable direction
Walser et al. [22]	19	IGE	5 (26 %)	–	–	–	–	7 (37 %)	–	8 (42 %)	–	–
Niaz et al. [20]	9	IGE	–	–	–	–	–	6 (66 %)	–	–	–	–

Legend: Superscripts ([a,b,c]): See accompanying bar graphs in Fig. 2.
JME: juvenile myoclonic epilepsy.
IGE: idiopathic generalized epilepsy.
Dash: indicates unavailable datum.

hypsarrhythmia is often seen in infants with epileptic spasms, while bursts of polyspike and waves correlate with and may produce myoclonic seizures.

Generalized epilepsy may produce dialeptic seizures (defined by altered mentation during seizure and inability to recollect seizure [17–19], myoclonic seizures, and relatively symmetric bilateral tonic-clonic or clonic seizures. However, prior studies have also noted focal ictal semiology in patients with generalized epilepsy. Many of these studies are based on chart review of clinical history, which may not be reliable. Other studies have analyzed ictal symptomatology in patients with IGE, utilizing a more reliable method of VEEGs. In general, focal ictal findings on VEEG in patients with generalized epilepsy are focal myoclonus, version, figure-of-4 sign, focal clonus, and asymmetric motor symptoms at the end of seizures [12,13,20–22]. These focal ictal symptoms mimic lateralizing signs typically seen in focal epilepsies (Figs. 2 and 3, Table 2).

The frequency of "lateralizing signs" in patients with IGE varies among different studies. This variability is most likely related to differences in the number of seizures captured for each patient (the more seizures captured, the more likely to find a focal symptom in a particular patient). The difference in interpretations of seizure semiology by different investigators may also play a role. For example, interpretation of head turns as lateralizing signs may vary among different reviewers. Versive seizures are a valuable lateralizing sign in focal epilepsy when the version is forced, sustained, and unnatural. When these criteria are not met, the number of "versions" that are classified as versive seizures may increase as many patients may turn their heads to one side or the other during epileptic seizures.

The distinction between generalized and focal epilepsy, sometimes, is imperfect and some epilepsies have features that fall in between these two extremes. For example, a patient diagnosed with IGE may have focal ictal symptomatology and/ or focal EEG findings. In such case, a coexistence of generalized and focal epilepsy may need to be considered [23]. In other words, there may be a continuum between focal and generalized epilepsies as proposed by Lüders et al. [3]. This continuum is defined by *the presence of multiple epileptic foci and the tendency towards secondary generalization.* Capturing multiple seizures and seeing "lateralizing signs" that alternate sides will probably be helpful to disregard one sole epileptic focus. Therefore, one could discuss about mostly-focal epilepsies (resection of the epileptogenic focus can be considered) and mostly-generalized epilepsies (as having multiple epileptogenic focus making excisional surgery impossible).

The presence of ictal motor signs in focal epilepsy is important for lateralizing the epileptogenic zone [24]. However, relying solely on a report of an ictal focal symptom may lead to an incorrect diagnosis of focal epilepsy in a patient with generalized epilepsy. In focal epilepsy, an ictal motor sign occurs when a seizure discharge (if strong enough) invades an eloquent cortex. For example, a patient with right temporal lobe epilepsy may have left head version associated with a seizure discharge spreading to the contralateral frontal eye field [25]. A patient with JME can also have head version during a generalized convulsive seizure. The difference between these two scenarios is the following: In the case of temporal lobe epilepsy, an isolated seizure discharge from the right temporal lobe induces the left head version with involvement of the frontal eye field. In the patient with JME, the head version occurs in the context of a generalized seizure discharge (Fig. 6B). It is possible that as the seizure discharge spreads diffusely in the latter case, the head version occurs when an eloquent cortex with a lower threshold for ictal symptom (in this case, the frontal eye field) is stimulated.

6. Cases

6.1. Case 1

An 11-year-old boy presented to the epilepsy clinic for an evaluation of paroxysmal episodes that began 2 months ago. The patient reported

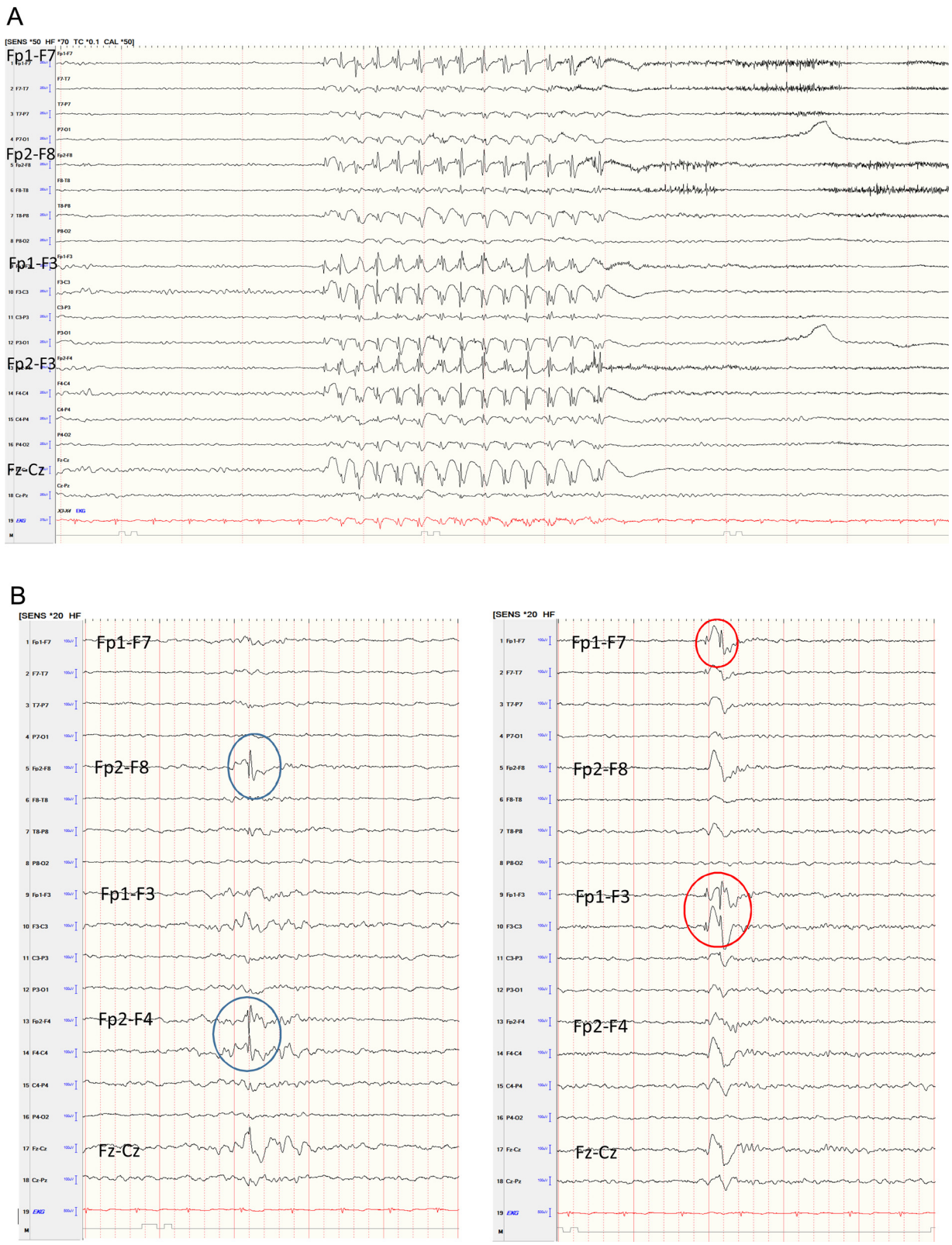

Fig. 4. Samples of interictal epileptiform activities seen in Case 1. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
(Bipolar montage with *five* chains).
(A) Shows 3 Hz generalized spike and wave discharges lasting for 5 s.
(Sensitivity 50 μV, HF 70 Hz, TC 0.1 s, Cal bar 50 μV).
(B) Shows independent right (blue circles) and prominent left frontal sharp waves (red circles).
(Sensitivity 20 μV, HF 70 Hz, TC 0.1 s, Cal bar 100 μV).

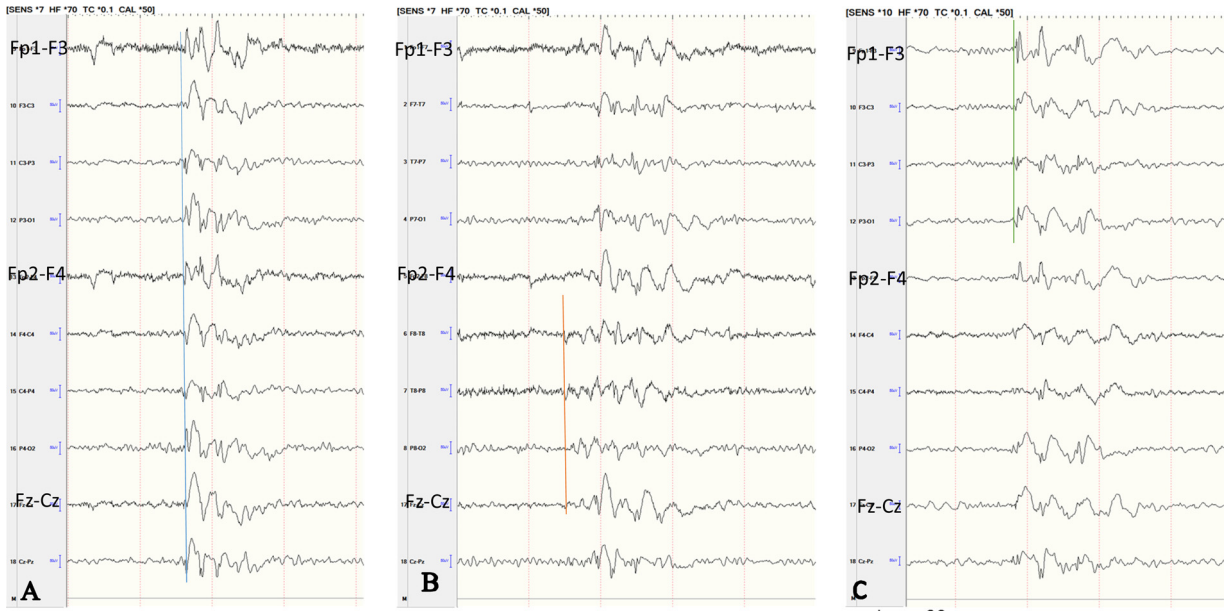

Fig. 5. Interictal *generalized* bursts of irregular spike and waves as recorded during EMU admission. (For interpretation of the references in colour in this figure, the reader is referred to the web version of this article.)
Bipolar montage with *three central* chains, Sensitivity 7μV, HF 70 Hz, TC 0.1 s, Cal bar 50 μV.
(Figure A) shows busts of generalized spike and wave. Blue vertical line indicate absence of a leading point. These abnormalities are sometimes *asymmetric*, with a lead in over the *right* hemisphere, indicated by the vertical orange line (Figure B) or over the *left* hemisphere, indicated by the vertical green line (Figure C).

feeling no abnormal sensation before or after an episode. He suddenly stopped what he was doing and mildly shook his head while being unresponsive with eyes open. There was no loss of postural muscle tone. These episodes lasted for 10–15 s. Immediately after an episode the patient resumed his activity as if there was no interruption. These events occurred four to five times daily.

Routine EEG showed generalized 3 Hz spike and wave complexes (Fig. 4A). In addition, there were *focal* epileptiform discharges with maximum negativity in the right and left frontal head regions (Fig. 4B). The patient was diagnosed with childhood absence epilepsy. He was started on ethosuximide that resulted in seizure freedom.

6.2. Case 2

A 26-year-old woman with diagnosis of epilepsy and anxiety presented to the epilepsy clinic. Seizures began in childhood. She denied any warning sensation, and she has no recollection of her seizures. After some episodes she may feel confused. After a “big one” she may feel sore in the shoulder, jaw, and other body parts. Occasionally, she may have difficulty eating for a few days due to a sore tongue after biting it during a seizure. Her husband indicated that she may stare and not respond for ~30 s to a minute. He recalled some instances in which the patient had repetitive chewing movements during her staring episodes. In addition, she had occasional seizures characterized by bilateral tonic posturing with ictal cry, “eyes rolled back”, blood tinted foaming at the mouth (sometimes), followed by bilateral jerking of limbs for approximately one minute. Postictal coma followed associated with loud breathing and gradual recovery. During these seizures, her head may turn to one side, but her husband could not recall which side. A routine EEG and brain MRI were both normal. She agreed to undergo an evaluation in the epilepsy monitoring unit (EMU) for characterization of her episodes and adjustment of AEDs.

She has tried multiple antiepileptic medications in the past. The patient was being treated with carbamazepine but continued to have at least one starring episode per month and two bilateral tonic clonic seizures per year. She was adherent to her AED and reported no side effects.

In addition to her epileptic paroxysmal events, she had “panic attacks” that are characterized by sensation of fear and difficulty breathing associated with dizziness lasting for minutes. These symptoms may occur on and off all day. She denied loss of consciousness during these episodes and these have never evolved to a bigger event. Her psychiatrist treated the “panic attacks” and currently are under good control.

6.3. Initial four-dimentional epilepsy classification after obtaining clinical history

Epileptic paroxysmal events
Ictal Semiology: Dialeptic (LOA)* → versive → bilateral tonic-clonic seizure
Epileptogenic zone: focal
Etiology: unknown
Comorbidities: anxiety
*LOA: loss of awareness

6.4. Comment 1, post interview

Based on the description of the events these are most likely epileptic. The initial classification of the epileptogenic zone was focal given the description of mouth automatisms and version during the bilateral tonic-clonic seizure.

During her EMU evaluation she was tapered off of carbamazepine. Her EEG showed burst of generalized spike and waves (Fig. 5). One non-epileptic panic attack and one epileptic seizure were recorded. Epileptic ictal semiology was characterized by: dialeptic (LOA) → left version → left face tonic → bilateral tonic-clonic seizure with an ipsilateral end-of-seizure arm clonus. EEG seizure pattern was generalized at the onset (Fig. 6A). The seizure evolution fifteen seconds after the onset of generalized electrographic seizure (Fig. 3A), monomorphic and regular bi-frontal rhythmic delta (~2 Hz) are better recognized in the first five seconds of the page that give way to theta-alpha frequency activity (resembling “saw-tooth”) in the right posterior head region (Fig. 6B). This activity spreads to both hemisphere toward the end of

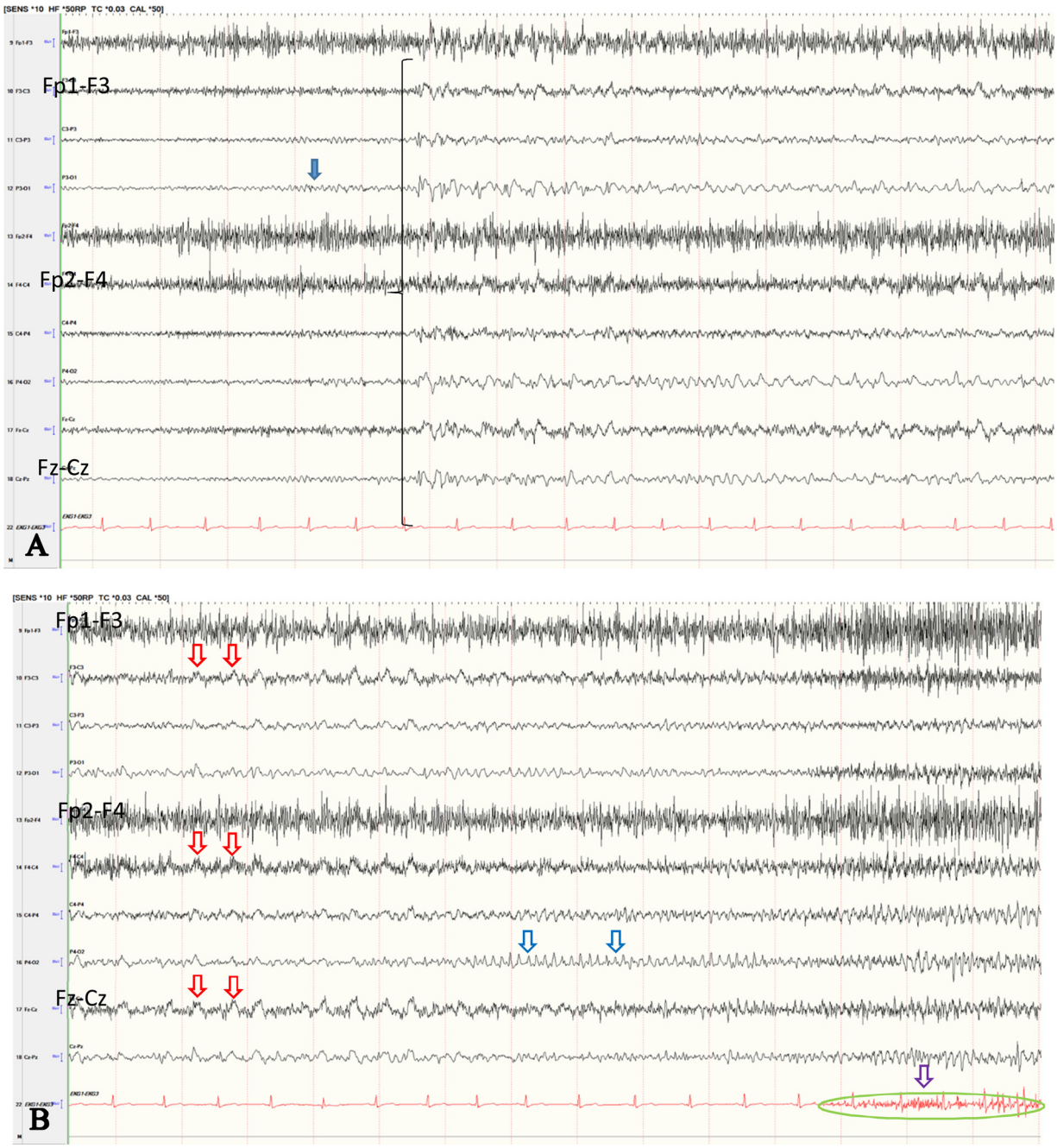

Fig. 6. Generalized seizure. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
(Bipolar montage with *three central* chains, Sensitivity 10 μV, HF 50 Hz, TC 0.03 s, Cal bar 50 μV).
A. Seizure onset
EEG seizure pattern is generalized at onset as indicated by the bracket. Posterior dominant rhythm is indicated by the blue arrow before the seizure onset.
B. Seizure evolution
Seizure evolution fifteen seconds after the onset of generalized electrographic seizure (Fig. 3A), monomorphic and regular *bi-frontal* rhythmic delta (~2 Hz) (red arrows) are better recognized in the first five seconds of the page that give way to theta-alpha frequency activity (resembling "saw-tooth") (blue arrows) in the right posterior head region. This activity spreads to both hemisphere toward the end of the page. In addition, EKG shows involvement of the muscles of the torso during the last three seconds of the page (green oval). Purple arrow indicates the moment when the head version starts.
C. End of the seizure
The end of the EEG seizure shows bilateral discharges (right hemisphere discharges indicated by the blue arrows) probably higher over the left hemisphere, which is likely producing the time-locked EMG artifact over the right hemisphere electrodes. The EEG electrodes on the right have overriding EMG artifacts of frontalis and occipitalis muscles. The unilateral EMG artifacts correlate with the asymmetric end-of-seizure clonic movements (clonus of right limb at the end of a GTC) (green box). After the end of the seizure, there is generalized suppression of background activity corresponding to postictal coma (blue box).

the page. In addition, EKG shows involvement of the muscles of the torso during the last three seconds of the page. At this point, the patient exhibited left version. The end of the EEG seizure shows bilateral discharges probably higher over the left hemisphere, which are likely producing a time-locked EMG artifact over the right hemisphere, correlating with the asymmetric end of seizure clonus (Fig. 6C).

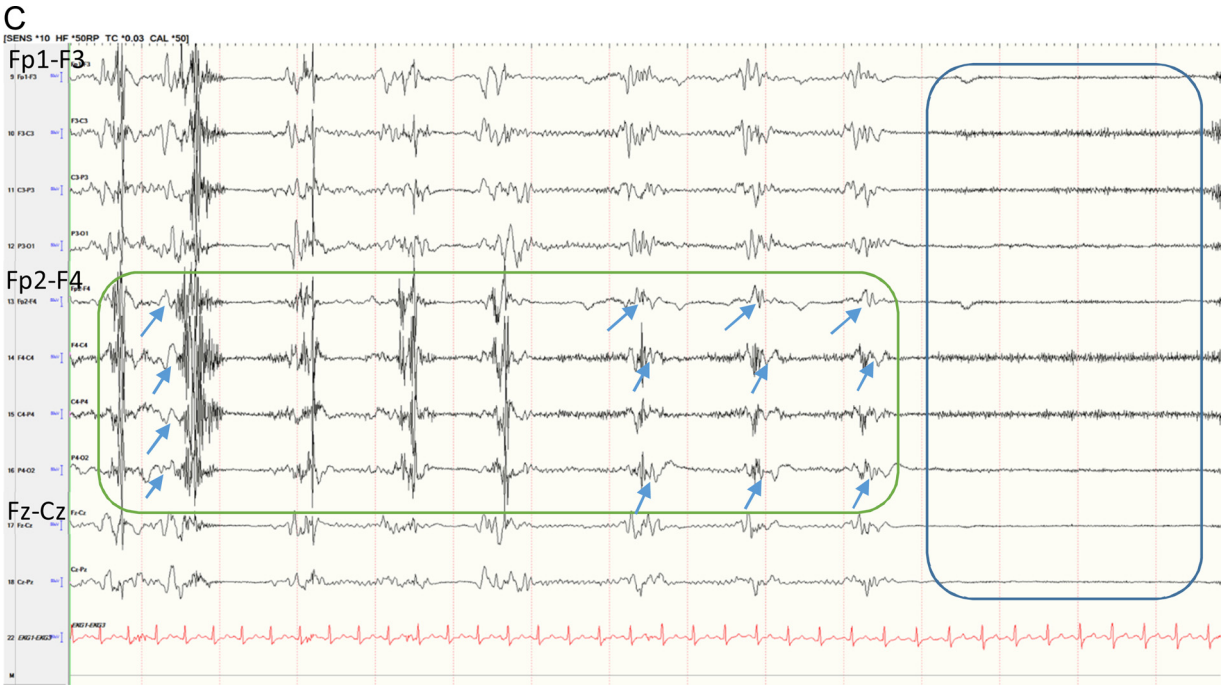

Fig. 6. (*continued*)

6.5. Comment 2, post EMU evaluation

The seizure semiology had features of focality as described. However, the onset of ictal EEG pattern was generalized (Fig. 6A). The left version occurred in the setting of generalized ictal EEG pattern. At the end of the seizure, both hemispheres showed ictal discharges (probably greater on the left than right). More intense ictal discharges on the left resulted in end-of-seizure right limb clonus. Note that her ictal semiology can mimic focal epilepsy arising from the right hemisphere. The patient was diagnosed with generalized epilepsy. She was switched to lamotrigine with subsequent good seizure control.

7. Conclusion

The presence of focal interictal abnormality and focal ictal signs and symptoms may mimic focal epilepsy in a patient who, indeed, has generalized epilepsy. Careful analysis of ictal EEG and its corresponding ictal semiology on video may help differentiate between focal and generalized epilepsy. Neuroimaging and genetic testing may provide additional layer(s) of data toward correct diagnosis to avoid iatrogenic complications, including an inappropriate consideration for epilepsy surgery.

Declaration of Competing Interest

Authors have no conflict of interest.

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