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Gelastic seizures: a retrospective study in five tertiary hospital centres

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ABSTRACT – *Aim.* This study aimed to characterize, clinically and neurophysiologically, a series of patients with gelastic seizures (GS), including both adults and children.
Methods. We retrospectively collected patients with GS from epilepsy clinics of five tertiary hospital centres within a single country. Patients were selected through relatives'/caregivers' descriptions, home video and/or video-EEG monitoring. GS were identified through ictal semiology.
Results. Thirty-five patients were enrolled; 62.9% had initial GS in infancy, 14.3% in adolescence and 22.8% at adult age. Twenty-six had abnormal MRI: eight presented with hypothalamic hamartoma (HH) and 16 non-HH lesions that included different structural aetiologies and genetic, metabolic and immune aetiologies. All patients with HH had their first GS in infancy or adolescence. For the remaining aetiologies, GS started in infancy in 59.3%, in adolescence in 11.1% and at adult age in 29.6%. Video-EEG data was available for analysis in 11 patients, including seven patients with a non-HH MRI lesion. The ictal onset topography on scalp video-EEG was usually concordant with the MRI lesion (in 6/7 patients) and the most frequent ictal onset was fronto-temporal. In two patients, both video-EEG and MRI suggested a parietal and occipital epileptogenic zone.
Conclusion. Aetiologies and patterns of affected topography unrelated to HH are common in patients with GS, and all age groups may manifest with this type of ictal semiology. This ictal manifestation has no lateralizing value and, despite a clear preponderance for hypothalamic, frontal and temporal lobe origins, other brain areas, namely the parietal and occipital lobes, should be considered.

Key words: gelastic seizures, hypothalamic hamartoma, video-EEG

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The first description of ictal epileptic laughter dates from 1873, by *Trousseau* (Trousseau *et al.,* 1873). Henceforth, seizures with this manifestation were named "gelastic seizures" (GS), from the Greek word "*gelos*", that means laughter. GS are characterized by recurrent bouts of paroxysmal stereotyped laughter or giggling, generally without mirth or appropriate affective tone (Gascon *et al.,* 1971; Chen *at al.,* 1973). It is a rare entity with an estimated prevalence between 0.4 and 0.8% (Kovac *et al.*, 2015). These seizures can consist exclusively of laughing or be associated with general autonomic arousal, motor automatisms or disturbed consciousness (Cerullo *et al.*, 1998).

GS are more likely to be diagnosed during childhood, classically associated with hypothalamic hamartoma (HH) (Striano *et al.,* 2009). In these cases, they are often refractory to pharmacological treatment, and precocious puberty and cognitive impairment are frequently associated (Nguyen *et al.*, 2003).

However, GS may also arise from other brain regions, including temporal, frontal or parietal lobes. In these cases, areas involved in the physiological network of smiling and laughter, connecting the hypothalamus with other regions, particularly the temporal and frontal lobes, should be considered (Wild *et al.*, 2003).

Most studies of GS are dedicated to the paediatric population. Patients with GS without HH have been mostly described in very few case reports, or case series, with small sample sizes (Tran *et al.*, 2014; Kovac *et al.*, 2015; Gutierrez *et al.*, 2016). These case series used video-EEG (VEEG) databases mostly from patients with refractory epilepsy based on pre-surgical work-up, hence patients were subjected to selection bias, as only severe epilepsy patients were included.

This study aimed therefore to characterize, clinically and neurophysiologically, a series of patients with GS collected from epilepsy clinics of five tertiary hospitals within a single country.

Material and methods

This retrospective study included patients with GS from epilepsy clinics from five Portuguese tertiary hospital centres: Hospital de Santa Maria (Centro Hospitalar e Universitário Lisboa Norte; Hospital de São José - Centro Hospitalar Universitário de Lisboa Central; Hospital Pediátrico de Coimbra; Centro Hospitalar Universitário de Coimbra; Centro Hospitalar do Hospital de São João).

All patients with seizures defined by the ILAE Operational Classification of Seizure Types, 2016 as GS were included, based on clinical and VEEG databases at the participating centres. Relatives'/caregivers' descriptions and home videos were accepted for ictal semiology description. Exclusion criteria were cases with absence of interictal EEG or cranial MRI, or limited access to detailed clinical data.

From the patient clinical files, the following variables were extracted: sex, age at first GS (infancy [≤10 years old], adolescence [between 10 and 18 years old] and adulthood [≥18 years old]), GS as first manifestation of epilepsy, presence of other seizures and other conditions, aetiology and topography (based on cranial MRI), interictal and ictal (for patients with VEEG) EEG topography, therapeutics, epilepsy surgery, and follow-up after surgery.

All data were evaluated using descriptive statistics (SPSS 21).

Results (*table 1*)

Thirty-five patients with GS were obtained from the participating centres, 20 (57.1%) males and 15 females (42.9%). The first GS occurred between two months and 47 years of age, (median of six years). Twenty-two patients (62.9%) had a first seizure in infancy, five (14.3%) in adolescence and eight (22.8%) at adult age. In 15 patients (48.4%), GS were the first manifestation. The majority of patients presented also with other seizures besides GS. This information was available in 31 patients, of whom 27 (87.1%) had other seizures, such as generalized tonic-clonic seizures and focal seizures with motor or sensitive onset. Delayed psychomotor development was present in 58.1% of these 31 patients. In the HH group, all patients had concomitant psychomotor delay or cognitive deficits and one patient also had precocious puberty.

Diagnosis was made based on VEEG in 16 (45.7%) patients. For the remaining patients, ascertainment was based on descriptions by caregivers/relatives and direct clinical observation.

Twenty-six patients had abnormal MRI. Structural epilepsy was diagnosed in 24 patients: eight patients with HH and 16 with various aetiologies. One patient presented with genetic epilepsy (Niemann Pick C), and another had immune epilepsy related to Rasmussen disease. Lesions on MRI were either on the left (*n*=9) or on the right side (*n*=6). Nine patients had normal cranial MRI.

Regarding the eight patients with HH, seven (87.5%) had their first GS in infancy and one (12.5%) in adolescence. For the remaining aetiologies, 16 (59.3%) patients started having GS in infancy, three (11.1%) in adolescence and eight (29.6%) at adult age.

Interictal epileptiform activity was found in 31 patients (81.6%), including most patients with HH (7/8). Interictal epileptiform activity usually included either the frontal or the temporal lobes (*n*=26; 83.9%). However, five (16.1%) involved the parietal or the occipital lobe.

Table 1. Clinical characteristics of the all patients included in the study.

n	Sex	Age at onset	Method of seizure characterization	GS as first manifestation	Other seizures	Other associated conditions	Aetiology/ cranial MRI	Interictal epileptiform activity	Ictal onset (VEEG)	Refractory epilepsy	Therapeutic	Surgery	Follow-up after surgery
1	F	12 y	Clinical	Yes	Focal impaired awareness seizure with motor onset	No	Traumatic R parieto-temporo-occipital	R fronto-temporal	NA	Yes	LEV, ZNS, CBZ, LTG, TPM, PER	No	No
2	F	9 y	VEEG	Yes	GTCS	Delayed psychomotor development; severe cognitive deficit	Hypothalamic Hamartoma	None	R temporal	Yes	CBZ, VPA	Yes Lesionectomy	Engel IIb after 2 y
3	M	29 y	VEEG	No	GTCS	No	R occipital lesion MELAS	R occipital	R occipital	Yes	LEV, LCM, TPM, CZP	No	No
4	F	12 y	VEEG	No	GTCS; focal impaired awareness seizure with motor onset	No	L temporo-parietal DNET	None	L parietal	Yes	LTG, ESL, CLB	Yes Lesionectomy	Engel Ia after 19 m
5	M	30 y	VEEG	No	GCTS	No	No lesion	None	NA	Yes	LEV, VPA	No	No
6	F	21 y	VEEG	No	GTCS	No	L temporal arterial malformation	L temporal	L anterior temporal	Yes	LEV, TPM	No (refused)	No
7	M	20 y	VEEG	No	Focal impaired awareness seizure with motor onset	No	L frontal Taylor dysplasia	L frontal	L frontal	Yes	CBZ, LEV	Yes Lesionectomy	Engel Ia after 3 y

Table 1. Clinical characteristics of the all patients included in the study (*continued*).

n	Sex	Age at onset	Method of seizure characterization	GS as first manifestation	Other seizures	Other associated conditions	Aetiology/ cranial MRI	Interictal epileptiform activity	Ictal onset (VEEG)	Refractory epilepsy	Therapeutic	Surgery	Follow-up after surgery
8	M	6y	VEEG	No	Focal impaired awareness seizure with motor onset	No	R mesial sclerosis	R anterior temporal	R anterior temporal	Yes	CBZ, LTG, VPA	Yes Amygdalohippocampectomy	Engel Ia after 2 y
9	F	47 y	Clinical	No	Focal impaired awareness seizure with motor onset; GTCS	No	No lesion	L anterior temporal with contralateral spreading	L anterior temporal	Yes	VPA, ZNS, LTG, CLB	No	No
10	M	16 y	Clinical	NA	NA	NA	No lesion	L parieto-occipital	NA	NA	NA	NA	NA
11	M	32 y	Clinical	NA	NA	NA	R temporal low-grade glioma	R temporal	NA	NA	NA	NA	NA
12	M	20 y	Clinical	NA	NA	NA	No lesion	L anterior temporal	NA	NA	NA	NA	NA
13	F	6 y	Clinical	NA	NA	NA	Hypothalamic hamartoma	Frontal bilateral	NA	NA	NA	NA	NA
14	F	18 y	Clinical/ VEEG	No	Focal impaired awareness seizure with motor onset; GTCS	No	L temporal neocortical DNET	Left temporal	Bilateral temporal (+R)	Yes	VPA, CBZ, LTG	Yes, Lesionectomy	Engel Ia after 4 y

Table 1. Clinical characteristics of the all patients included in the study (*continued*).

n	Sex	Age at onset	Method of seizure characterization	GS as first manifestation	Other seizures	Other associated conditions	Aetiology/ cranial MRI	Interictal epileptiform activity	Ictal onset (VEEG)	Refractory epilepsy	Therapeutic	Surgery	Follow-up after surgery
15	F	1 y	Clinical/ VEEG	No	Focal impaired awareness seizure with motor onset; GTCS	No	L temporal cortical dysplasia and L mesial sclerosis	Bilateral temporal	Bilateral frontal (+L)	No	VPA	No	No
16	M	4 y	Clinical/ VEEG	No	GTCS	Cognitive deficit	Hypothalamic hamartoma	Left frontal	Bilateral frontal (+L)	Yes	TPM, LEV, VPA	No wait for radio-surgery	No
17	F	11 y	Clinical/ VEEG	No	Focal impaired awareness seizure with motor onset; GTCS	Puberty at 8 years	Hypothalamic Hamartoma	Bilateral frontal (+R)	Bilateral frontal (+R)	Yes	LEV, OXC, TPM, GBP	Yes Radiosurgery	Engel Ia after 14 m
18	M	2 m	Clinical/ VEEG	Yes	No	Delayed psychomotor development	Hypothalamic hamartoma	Bilateral frontal	NA	NA	NA	No	No
19	M	2 y	Clinical	Yes	GTCS	Delayed psychomotor development	Hypothalamic hamartoma	L anterior temporal	NA	Yes	ESL, VPA, CLB	No	No
20	M	1 y	Clinical	Yes	No	Delayed psychomotor development	Hypothalamic hamartoma	Multifocal	NA	Yes	NA	NA	NA
21	M	5 y	Clinical	Yes	Focal impaired awareness seizure with sensitive onset	Delayed psychomotor development	No lesion	R anterior temporal	NA	Yes	NA	No	No

Table 1. Clinical characteristics of the all patients included in the study (*continued*).

n	Sex	Age at onset	Method of seizure characterization	GS as first manifestation	Other seizures	Other associated conditions	Aetiology/ cranial MRI	Interictal epileptiform activity	Ictal onset (VEEG)	Refractory epilepsy	Therapeutic	Surgery	Follow-up after surgery
22	M	5 y	Clinical	Yes	GTCS; absence seizures	Delayed psychomotor development; autism	No lesion	Bilateral temporal (+L)	NA	Yes	FLB, VPA, CLB	No	No
23	M	7 y	Clinical	Yes	Focal impaired awareness seizure with sensitive onset	Delayed psychomotor development	No lesion	L central	NA	Yes	NA	No	No
24	F	2 y	Clinical	Yes	Absence seizures	Delayed psychomotor development; spastic tetraparesis	Niemann Pick C with frontal lobe atrophy	R centro-parietal	NA	Yes	NA	No	No
25	M	1 y	Clinical	No	GTCS	Delayed psychomotor development	Lennox Gastaut syndrome with midline malformation	R frontal	NA	Yes	VPA, CLB	No	No
26	M	2 y	Clinical/ VEEG	Yes	No	Delayed psychomotor development	L frontal Ganglioglioma	R frontal	NA	Yes	NA	Yes [1]	NA
27	F	8 m	Clinical	No	Absence seizures	Delayed psychomotor development	R hemisphere Lissencephaly	R central	NA	Yes	VPA, ZNS	No	No
28	F	2 m	Clinical	No	GTCS and myoclonic generalized seizures	Delayed psychomotor development	Methylmalonic aciduria No Lesion on MRI	Multifocal	NA	No	NA	No	No

Table 1. Clinical characteristics of the all patients included in the study (*continued*).

n	Sex	Age at onset	Method of seizure characterization	GS as first manifestation	Other seizures	Other associated conditions	Aetiology/ cranial MRI	Interictal epileptiform activity	Ictal onset (VEEG)	Refractory epilepsy	Therapeutic	Surgery	Follow-up after surgery
29	M	15 m	Clinical	No	GTCS	No	L hemispheric atrophy	L frontal	NA	No	NA	No	No
30	M	2 m	Clinical/ VEEG	Yes	GTCS and epileptic spasms	Delayed psychomotor development	L temporal medial Ganglioglioma	L temporal	NA	Yes	VPA, ESL	Yes [1]	Engel Ia*
31	F	4 m	Clinical/ VEEG	Yes	GTCS and epileptic spasms	Delayed psychomotor development	Diffuse lissencephaly	Hypsarrhythmia	NA	Yes	VGB, ZNS, CLB	No	No
32	F	6 y	Clinical/ VEEG	Yes	GTCS	Delayed psychomotor development	Dentatorubral-pallidoluysian atrophy	Occipital	NA	Yes	VPA, LEV, CLB	No	No
33	M	18 m	Clinical	No	GTCS; absence seizures	Delayed psychomotor development	Hypothalamic hamartoma	Bilateral frontal	NA	Yes	CLB, VPA	Yes [1]	Engel IV*
34	M	4 y	Clinical	Yes	EPC	Delayed psychomotor development	R hemisphere Rasmussen	R temporo-occipital	NA	Yes	NA	Yes [1]	Engel Ia*
35	F	10 y	Clinical	Yes	No	Delayed psychomotor development	Chromosomal disorder, no lesion on MRI	None	NA	Yes	VPA, CLB, ESL	Yes [1]	Engel IV*

Age at onset refers to the time of the first gelastic seizure manifestation; *No information about follow up time; 1No information about the surgery type; NA: not available; L: left; R: right; y: years; m: months; F: female; M: male; GTCS: generalized tonic-clonic seizures; CBZ: carbamazepine; CLB: clobazam; CZP: clonazepam; ESL: eslicarbazepine; FBM: felbamate; GBP: gabapentin; LCM: lacosamide; LEV: levetiracetam; LTG: lamotrigine; OXC: oxcarbazepine; PER: perampanel; TPM: topiramate; VPA: valproate; VGB: vigabatrin; ZNS: zonisamide.

VEEG data was available for analysis in 11 patients. Scalp EEG ictal onset was temporal in five patients, frontal in four, parietal in one, and occipital in one. Seven of these patients had non-HH lesions on cranial MRI. Among these, six had a VEEG concordant with the MRI lesion: frontal (one patient), temporal (three patients), parietal (one patient) and occipital (one patient) (*figure 1*).

In 30 patients, it was possible to evaluate the treatments prescribed. From those patients, 90% (*n*=27) had refractory epilepsy. All the treatments are described in *table 1*. Epilepsy surgery was performed in 36.7% (*n*=11) of patients. Only three patients with HH were submitted to surgery. In the HH group, only one of the three patients submitted to surgery remained seizure-free after 14 months of follow-up. In the non-HH group, on the contrary, most patients remained in Engel Class Ia, except for one patient with a genetic disorder, for whom surgery was ineffective.

Discussion

In this study, we reported 35 adult and paediatric patients with GS and identified different aetiologies and affected topographies, including 27 patients without HH. Cases were obtained not only based on VEEG but also from other sources for seizure characterization, and patients with both laughter and giggling were included, providing a more comprehensive picture of GS in clinical practice.

In most patients, GS were accompanied by other seizures, including mostly generalized tonic-clonic seizures and focal seizures with impaired consciousness and motor or sensitive onset. These data are similar to those of other studies, which also concluded that patients with GS usually have other types of seizures (Tassinari *et al.*, 1991; Striano *et al.*, 1999, 2012). Most seizures are automotor seizures, that commonly originate in temporal or frontal regions, and involve the different areas involved in GS network and propagation of seizure activity. GS was the presenting symptom of epilepsy in only 15 of the 31 patients based on the information available. In both HH and non-HH, approximately half the patients had other types of seizures before GS were noted or reported.

The majority of previously published GS series have addressed HH related epilepsies (Nguyen *et al.,* 2003). Indeed, GS are commonly deemed a hallmark of HH, especially in early-age-onset cases (Kerrigan *et al.*, 2005). In this type of pathology, patients can present with other conditions, like delayed psychomotor development, severe cognitive deficit or precocious puberty (Tassinari *et al.*, 1991). This is congruent with our data regarding other associated conditions. In our study, we found eight patients (22.9%) with GS associated with HH, and in all of them, seizures started during infancy or adolescence, reinforcing this classic association.

In patients without HH, the age at the first GS varied significantly, and a high proportion started in adolescence and adulthood. This is in accordance with previously published series of non-HH GS in which seizure onset ranged from between months to 93 years of age (Tassinari *et al.*, 1991; Kovac *et al.,* 2015; Gutierrez *et al.,* 2016).

There are many different non-HH GS associated aetiolies. These include inflammatory, infectious, and dysplastic aetiologies, as well as mesial sclerosis and tumours of all cerebral lobes, the pituitary gland, mammillary bodies and the third ventricle (Tassinari *et al.*, 1991; Munari *et al.,* 1995; Kuzniecky *et al.*, 1997). Although mesial temporal lobe sclerosis was not suggested to be an aetiology of GS based on earlier studies, (Tassinari *et al.*, 1991), later series have shown a predominance of mesial sclerosis, ranging from 12.5 to 21% (Kovac *et al.,* 2015; Gutierrez *et al.,* 2016). In our series, the percentage was smaller (7.4%), suggesting that this preponderance in previous studies is most likely related to the reference bias associated with VEEG.

As previously mentioned, localization of the epileptogenic focus in GS is presumably related to the cerebral network of laughter that spreads from the hypothalamus to several cerebral areas, such as the inferior temporal, the mesial frontal and the parietal lobes (Umeoka *et al.*, 2008; Talvik *et al.,* 2012). Cortical stimulation studies showed widespread areas involved in that network (Caruana *et al.,* 2015). In our series, data from both cranial MRI and interictal and ictal EEG reinforces this claim. Indeed, interictal epileptiform activity and ictal EEG onset was located at fronto-temporal leads, respectively, in 83.9% and 71.4% of the patients. This is also in accordance with previous reports of GS (Tassinari *et al.*, 1991; Striano *et al.,* 1999; Tran *et al.*, 2014; Kovac *et al.*, 2015; Gutierrez *et al.,* 2016). In particular, Tassinari and co-workers (Tassinari *et al.*, 1991) described the largest series of GS published to date. These authors reviewed 60 patients with GS not associated with HH and 60 patients with HH, mostly from previously published cases. In accordance with our data, in non-HH patients, seizure foci were located in sites that included mostly temporal (61%), frontal (10%), and frontal and temporal (22%) areas. Localization of seizure onset was not possible in 7% of the patients. Cortical stimulation studies also show that the temporal lobe generates smiling manifestations (Arroyo *et al.,* 1997; Wild *et al.,* 2003). The frontal lobe has also been shown to be involved in the motor aspect of laughter, with a non-emotionally driven pathway involving the anterior cingulate, premotor frontal, and opercular areas projecting through

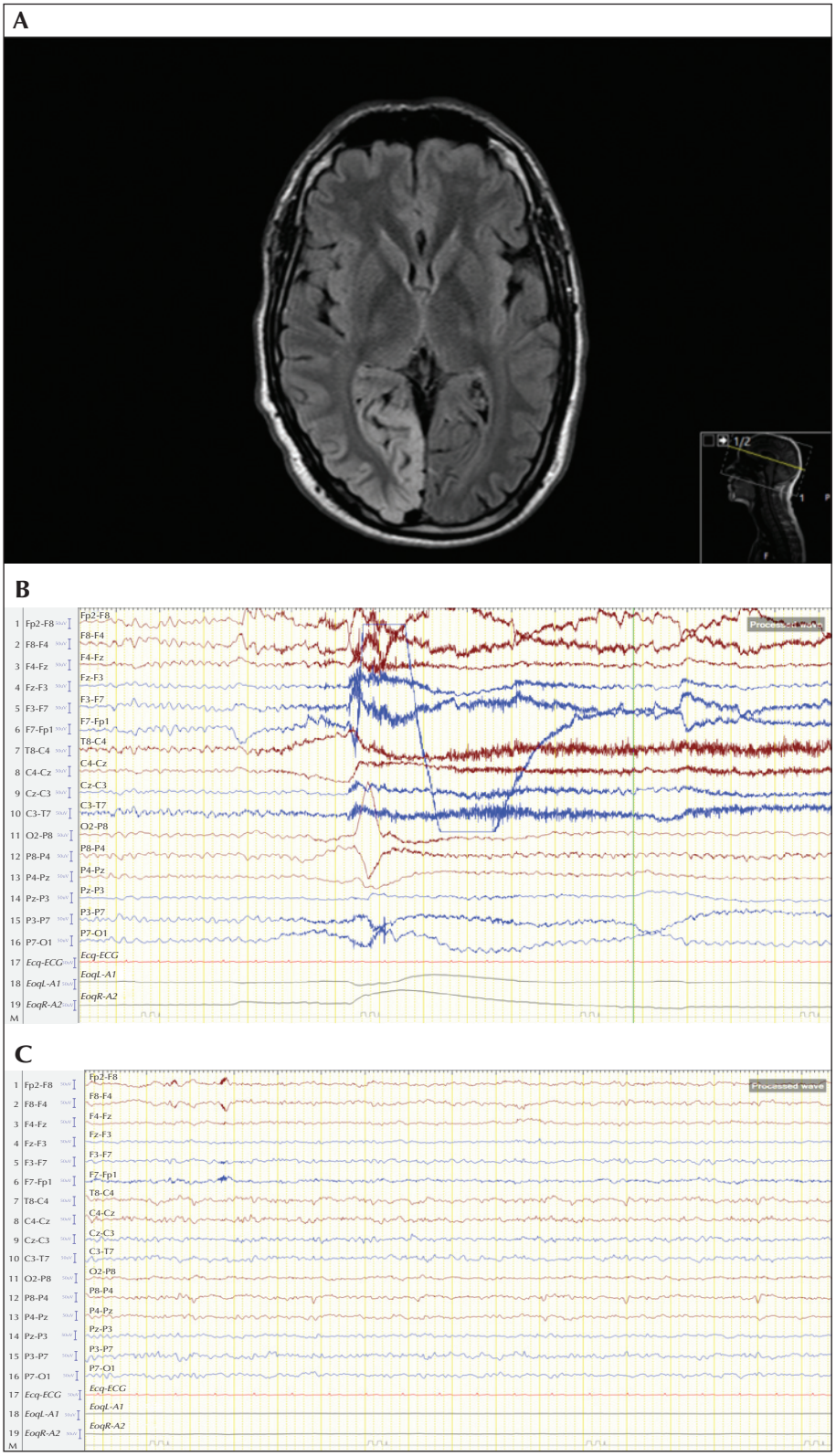

Figure 1. Patient with MELAS disease. (A) Cranial MRI-FLAIR- hypersignal in right occipital region; (B) Ictal EEG- Rhytmic theta in posterio-occipital leads, bilateral; (C) Interical EEG – Periodic discharges in right parietal- occipital regions.

the motor cortex directly to the ventral brainstem (Wild *et al.,* 2003; Tran *et al.,* 2014). Tassinari described semiological differences between GS according to its origin and aetiology (Tassinari *et al.*, 1991). GS with temporal lobe origin could resemble normal laughter and usually occurred with a sense of mirth, pleasure or happiness. On the contrary, frontal lobe origin (six cases) led to "unnatural" and briefer periods of laughter without emotional content that were usually less frequent with long-lasting laughter. In HH, GS resembled natural laughter but lacked emotional content and were usually brief. These semiological differences are in accordance with this proposed role of the frontal lobe in the motor component of laughter, whereas limbic structures are mostly responsible for the emotional part of this behaviour. Unfortunately, in our study, given its retrospective nature, we were unable to analyse the motor and emotional characteristics of GS. In our study, as in other published series, non-HH GS may occur both in left and right hemisphere epilepsies and have no lateralizing value (Tassinari *et al.*, 1991; Tran *et al.*, 2014).

Of note, we have also identified two patients with concordant lesional and VEEG data for ictal onset in parietal and occipital lobes. Only one published study describes the involvement of the parietal lobe in the genesis of GS (Kovac *et al.,* 2015). In the series of Tassarini *et al*. (1991), there was also one patient in whom GS was elicited by fusiform stimulation. In one of the patients (Patient 4), a left temporo-parietal dysembryoplastic neuroepithelial tumour (around the parietal operculum) was removed and the patient remained seizure-free, confirming this area as the epileptogenic region. In is not possible to exclude, however, propagation to more anterior temporal areas. In Patient 3, only scalp EEGs were performed and we have to acknowledge that this more posterior EEG activity may be due to propagation from fronto-temporal origins. The ictal EEG, however, shows rhythmic activity in bilateral posterior regions (*figure 1B*) with later propagation to the bilateral anterior lead (not shown). Furthermore, the concordance between MRI (*figure 1A*) and VEEG (both ictal [*figure 1A*] and interictal [*figure 1C*]), and the fact that these posterior brain regions have also been described to participate in the cerebral networks of laughter, suggest that these areas should also be considered as possible ictal onset zones for HH. Taken together, our data and the data from other series in the literature suggest that gelastic manifestation in seizures may not have localizing value.

Most patients with GS presented with refractory epilepsy, as is usually the case in GS series (Striano *et al.,* 2009; Gutierrez *et al.,* 2016). In this study, we aimed to focus on patients with GS in clinical practice, avoiding the reference bias associated with VEEG data. The frequent occurrence of refractory epilepsy in our series, however, may still be related to reference bias rather than a specific association with GS. On the one hand, we recruited patients from tertiary care hospitals with frequent referrals for refractory epilepsy. On the other hand, it is possible that subtle gelastic features of seizures in patients with other types of ictal semiology were missed in patients with complete seizure remission under medication or with rare seizures, as is the case of non-refractory patients. Of note, patients submitted to epilepsy surgery had an overall good surgical outcome, emphasizing the importance of this treatment strategy in selected patients (Téllez-Zenteno *et al.,* 2010).

This study presents several limitations. First, it is retrospective and multicentric with possible selection bias, and included non-standardized patient evaluation. Secondly, the majority of the diagnosis of GS was made based on direct clinical observations or relatives'/caregivers' descriptions. Although this may have led to false-positive cases, it also makes our sample more comprehensive because it also included patients for whom VEEG was not necessary. Finally, due to the retrospective nature of our study, other semiological data that may have clinical or localizing value, such as the emotional content of GS (laughter with no mirth originating in anterior cingulate areas (Sperli *et al.,* 2006) and emotional content from more basal temporal areas (Tassinari *et al.*, 1991; Fried *et al.,* 1998), or the association with hormonal symptoms in HH, were not collected.

In conclusion, our study reinforces that, despite the strong association between GS and HH in children, other aetiologies and patterns of affected topography unrelated to HH are common in patients of all ages with these types of seizure manifestation. This ictal sign has no lateralizing value and, despite a clear preponderance for hypothalamic, frontal and temporal lobe origin, other brain areas, namely the parietal and occipital lobes, should be considered. Further studies are necessary to better characterize ictal laughter and its association with specific emotional content. □

Supplementary data.

Summary didactic slides are available on the www.epilepticdisorders.com website.

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TEST YOURSELF

(1) Provide the essential characteristics of a gelastic seizure?

(2) Are gelastic seizures always associated with hypothalamic hamartomas?

(3) Which other brain regions can trigger gelastic seizures?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".