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Correlation between temporal pole MRI abnormalities and surface ictal EEG patterns in patients with unilateral mesial temporal lobe epilepsy

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Summary

Objective: The objective of this retrospective study is to analyze ictal patterns observed during continuous Video-EEG monitoring in patients with temporal lobe epilepsy (TLE) due to unilateral hippocampal sclerosis (HS), and to correlate these EEG patterns to temporal pole abnormalities observed on magnetic resonance imaging exams.
Methods: We analyzed 147 seizures from 35 patients with TLE and unilateral HS. Ictal patterns were classified and correlated to signal abnormalities and volumetric measures of the temporal poles. Volume differences over 10% were considered abnormal.
Results: The most frequent type of ictal pattern was rhythmic theta activity (RTA), encountered in 65.5% of the seizures. Rhythmic beta activity (RBA) was observed in 11% of the seizures, localized attenuation in 8%, interruption of epileptiform

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discharges in 6%, repetitive discharges in 5.5%, and rhythmic delta activity (RDA) in 4%. Sixty-six percent of the patients presented signal abnormalities in the temporal pole that were always ipsilateral to the HS. Sixty percent presented significant asymmetry of the temporal poles consisting of reduced volume that was also always ipsilateral to HS. Although patients with RTA as the predominant ictal pattern tended to present asymmetry of temporal poles ($p = 0.305$), the ictal EEG pattern did not correlate with temporal pole asymmetry or signal abnormalities.

Conclusions: RTA is the most frequent initial ictal pattern in patients with TLE due to unilateral HS. Temporal pole signal changes and volumetric reduction were commonly found in this group of patients, both abnormalities appearing always ipsilateral to the HS. However, neither temporal pole volume reduction nor signal abnormalities correlated with the predominant ictal pattern, suggesting that the temporal poles are not crucially involved in the process of epileptogenesis.

Introduction

Temporal lobe epilepsy (TLE) is the most common type of focal epilepsy syndrome in adults.[1] Patients with TLE and hippocampal sclerosis (HS) diagnosed by magnetic resonance imaging (MRI) have greater probability of being refractory to clinical treatment than patients with other types of lesions.[2]

TLE due to HS constitutes the most frequent indication of epilepsy surgery in adults,[3] being unequivocally superior to clinical treatment with respect to seizure control and quality of life.[4] Most of the operated patients reaches considerable seizure control after surgery,[5] with probabilities of complete post-surgical seizure remission ranging from 70% to 90%.[6,7]

Video-electroencephalogram monitoring (Video-EEG) remains as one of the cornerstones of the presurgical evaluation of patients with refractory TLE. For most patients ictal scalp recordings can provide sufficient information for the identification of the epileptogenic temporal lobe, leaving invasive monitoring necessary for only few and selected cases.[8] Specific ictal patterns observed during scalp Video-EEG had already been correlated to the site of seizure onset,[9,10] and to the localization of the epileptogenic zone.[11]

Although the role of mesial temporal structures in the pathophysiology of TLE is already well recognized, the involvement of other temporal structures, particularly the anterior portion of the temporal lobes — the so-called temporal pole — has only recently driven the attention of investigators in the field. The influence of abnormalities of the temporal poles on the post-surgical prognosis still remains to be clearly defined as well.

We conducted a retrospective study aiming to analyze the clinical, electrophysiological, and radiological characteristics of a homogeneous group of patients with TLE and unilateral HS. We analyzed the role of ictal patterns captured during non-invasive Video-EEG, of signal and volume abnormalities of the temporal poles, and the correlation between these variables. We additionally discussed the involvement of the temporal poles in the seizures of patients with TLE due to HS, and the possible role of these structures in the pathophysiology of TLE.

Patients and methods

Thirty-five consecutive patients with medically refractory TLE were included in the study (Table 1). Diagnosis was established according to previously reported clinical and electrographic characteristics.[12] Patients who did not achieve seizure control despite of adequate use of at least two first-line antiepileptic drugs (AEDs), in monotherapy or polytherapy, and up to toxic levels, were qualified as medically refractory. The study was approved by the Ethics Committee of our institution.

All patients had extensive presurgical evaluation including high-resolution 1.5T MRI, prolonged non-invasive Video-EEG, and neuropsychological testing. All patients had unilateral hippocampal atrophy (HA) diagnosed on imaging. The exams were performed in 1.5T equipment (Siemens Somaton or Phillips Gyroscan), and blindly analyzed by a single neuroradiologist with expertise in epilepsy (Carrete Jr.). A standardized MRI protocol for imaging the temporal lobes was applied, including the following acquisition sequences: sagital spin-echo T1, 6 mm slices; axial spin-echo T2, 6 mm slices; coronal FLAIR (perpendicular to the longer axis of the hippocampus), 3 mm slices; coronal inversion-recovery (perpendicular to the longer axis of the hippocampus), 3 mm slices; coronal FFE-T1 volumetric acquisition of the whole hemispheres, 1.5 mm slices. Hippocampal atrophy and signal abnormalities of the temporal poles were assessed by visual analysis.

Table 1 Clinical and laboratorial characteristics of the 35 patients

Patient	Age (years)	Onset (years)	Duration (years)	Febrile seizure	Hippocampal sclerosis	Temporal pole ABN/ASYM	Interictal discharges	Predominant pattern
1	40	20	20	N	L	Y/N	Unitemporal	RED
2	44	10	34	Y	R	N/N	Unitemporal	ID
3	34	12	22	N	R	Y/N	Unitemporal	RTA
4	53	14	39	Y	L	Y/Y	Unitemporal	RTA
5	33	15	18	N	R	Y/Y	Bitemporal	RTA
6	50	1	49	N	L	Y/N	Unitemporal	0
7	45	11	44	N	L	N/N	No discharges	RTA
8	29	18	11	Y	L	Y/Y	Unitemporal	RTA
9	39	3	36	N	L	Y/N	Unitemporal	RTA
10	51	23	28	N	R	NA/N	Bitemporal	RBA
11	42	14	28	N	L	Y/N	Unitemporal	RTA
12	29	7	22	N	L	Y/N	Unitemporal	RTA
13	49	19	30	Y	R	N/Y	Unitemporal	0
14	32	7	25	Y	L	Y/Y	Bitemporal	RTA
15	21	4	17	N	L	Y/Y	Unitemporal	RBA
16	37	2.5	34	Y	R	Y/Y	Unitemporal	RDA
17	29	10	19	N	L	Y/Y	Unitemporal	0
18	33	9	24	Y	L	Y/N	Unitemporal	ID
19	40	25	15	N	L	Y/N	Unitemporal	0
20	33	10	23	N	R	N/Y	Unitemporal	RTA
21	35	12	23	Y	L	Y/Y	Unitemporal	0
22	58	20	38	Y	L	N/Y	Bitemporal	0
23	43	8	35	N	R	NA/Y	Unitemporal	AT
24	42	17	25	N	R	Y/Y	Unitemporal	RTA
25	44	2	42	N	R	N/Y	Bitemporal	RTA
26	53	10	43	N	R	N/Y	Unitemporal	RTA
27	35	16	19	N	R	N/Y	Unitemporal	RTA
28	31	3	28	Y	R	N/Y	Unitemporal	RTA
29	36	18	18	Y	L	N/N	Unitemporal	0
30	35	2	33	Y	L	Y/Y	Bitemporal	RTA
31	20	7	13	N	L	Y/N	Unitemporal	0
32	43	3	40	Y	R	Y/Y	Unitemporal	RBA
33	40	35	5	N	L	N/N	Unitemporal	0
34	22	13	9	N	L	Y/Y	Unitemporal	0
35	37	13	24	N	R	Y/Y	Unitemporal	RTA

Y, yes; N, no; L, left; R, right; ABN, signal abnormality; ASYM, asymmetry; 0, no predominant pattern; AT, attenuation; ID, interruption of epileptiform discharges; RED, repetitive epileptiform discharges; RDA, rhythmic delta activity; RTA, rhythmic theta/alpha activity; RBA, rhythmic beta activity.

Volumetric measurements of the temporal poles was performed, and only asymmetry index between the two poles greater than 10% was considered abnormal.

Prolonged non-invasive Video-EEG monitoring was performed on 32-channel digital equipment (Biologic®, and Ceegraph® software). Electrodes were placed according to the 10-20 International System, plus intermediary temporal and sphenoidal electrodes. In order to record ictal events AEDs were tapered off or completely withdrew at physician's discretion (no standardized protocol for AED manipulation was applied).

Video-EEG recording was continuously monitored by technical staff. The frequency and location of interictal epileptiform discharges (IEDs) were visually assessed on 5 min EEG samples per hour, 24 h per day. Video-EEG analysis was blindly performed by board certified electroencephalographers with expertise in presurgical evaluation (EG and ACS).

For the purposes of this study, only seizures with loss of consciousness (complex partial and generalized seizures) were considered. Ictal EEG recordings were printed out for blind analysis in bipolar and referential montages (Pz reference whenever possible, or alternatively Cz reference). Digital filtering and gain were adjusted to optimize visual EEG analysis. Low frequency filter was set at 1 Hz, and high frequency filter at 70 Hz; whenever there were artifacts in excess, filters were adjusted to facilitate the analysis.

EEG samples contained no patient identification and were independently analyzed by two investigators (ACS, EG) who were asked to fill out an standardized EEG report. Both were blinded to all clinical and radiological data of the patients, as well as to the post-surgical seizure outcome. Whenever discordant categorization of the ictal patterns was present, agreement was reached by the two examiners in a second-round analysis.

Ictal pattern analysis

For categorization of ictal patterns we modified and adapted criteria previously proposed by Steinhoff et al.[13] At the onset of the seizures the ictal patterns were classified as: (a) AT: background attenuation (only if localized or lateralized); (b) ID: interruption of epileptiform discharges; (c) RED: repetitive epileptiform (spikes and/or sharp waves) discharges; (d) RDA: rhythmic delta activity; (e) RTA: rhythmic theta/alpha activity; (f) RBA: rhythmic beta activity. Topography of the ictal discharges was classified as follows: (a) temporal (left or right): amplitude ratio $\geq$2 when comparing the two sides in referential montage, and $\geq$2 when comparing temporal and parasagital leads in bipolar montage; (b) hemispheric (left or right): amplitude ratio $\geq$2 when comparing the two sides in referential montage, but $<$2 when comparing temporal and parasagital leads in bipolar montage; (c) bilateral, lateralized (left or right): bilateral ictal discharges with amplitude ratio $>$1 but $<$2 in favor of one side in referential and bipolar montages; (d) bilateral, non-lateralized; (e) extratemporal (left or right). In addition to the type and localization of the initial ictal pattern, each seizure was further analyzed regarding the Involvement of the contralateral hemisphere, and the presence of switch of lateralization from one hemisphere to the other. Following these criteria patients were divided in four separate groups, according to the localization of the ictal discharges: (a) patients with all seizures localized (temporal onset) or lateralized (one non-lateralized seizure allowed); (b) patients with bilateral seizures (bitemporal or bi-hemispheric); (c) patients with non-lateralized seizures (one lateralized seizure allowed); (d) patients with switch of lateralization (at least one seizure terminating with greater amplitude in the hemisphere contralateral to ictal onset). Patients who only had two seizures recorded, one lateralized and one non-lateralized, were included in the first group (lateralized seizures).

Surgical treatment

All patients were submitted to standardized corticoamygdalohippocampectomy (CAH). Surgical strategy was not influenced by the results of the present study.

Statistical analysis

All data were analyzed by X^2-test, or Fisher's exact test whenever necessary. p-Values < 0.05 were considered significant. SPSS 10.0 FOR Windows® was employed for statistical analysis.

Results

Thirty-five consecutive patients (19 women, 16 men) were included in the study and their age varied from 20 to 58 years (mean = 38.2). Thirteen patients (37%) had past history of febrile seizure occurring from 8 months to 4 years (mean = 2 years). Age at onset of epilepsy varied from 1 to 35 years (mean = 12 years), while duration of epilepsy from onset of seizures to surgery ranged from 5 to 49 years (mean = 26.6 years).

Interictal EEG

Twenty-eight patients (80%) had unilateral IEDs, six (17%) had bilateral temporal IEDs, defined as more than 20% of IEDs independently recorded over the temporal lobe contralateral to the epileptogenic temporal lobe, and one (3%) did not present IEDs during Video-EEG.

Ictal analysis

The entire group of patients presented a total of 164 seizures (including auras) during Video-EEG (2—16 seizures per patient, mean of 4.8 seizures). Out of these, 147 seizures fulfilled previously defined criterion (loss of consciousness) and were analyzed in the study. The final categorization of the ictal patterns was as follows: (a) AT (background attenuation): 12 seizures (8%); (b) ID (interruption of epileptiform discharges): 9 (6%); (c) RED (repetitive epileptiform discharges): 8 (5.5%); (d) RDA (rhythmic delta activity): 6 (4%); (e) RTA (rhythmic theta/alpha activity): 96 (65.5%); (f) RBA (rhythmic beta activity): 16 (11%). Forty-three percent of the patients presented a single ictal pattern at the onset of all seizures, while 57% presented more than one ictal pattern. In these patients, for the purpose of categorization of the ictal patterns, only the predominant ictal pattern was considered. The categorization was as follows: (a) AT (background attenuation): 1 patient (3%); (b) ID (interruption of epileptiform discharges): 2 (5%); (c) RED (rhythmic epileptiform discharges): 1 (3%); (d) RDA (rhythmic delta activity): 1 (3%); (e) RTA

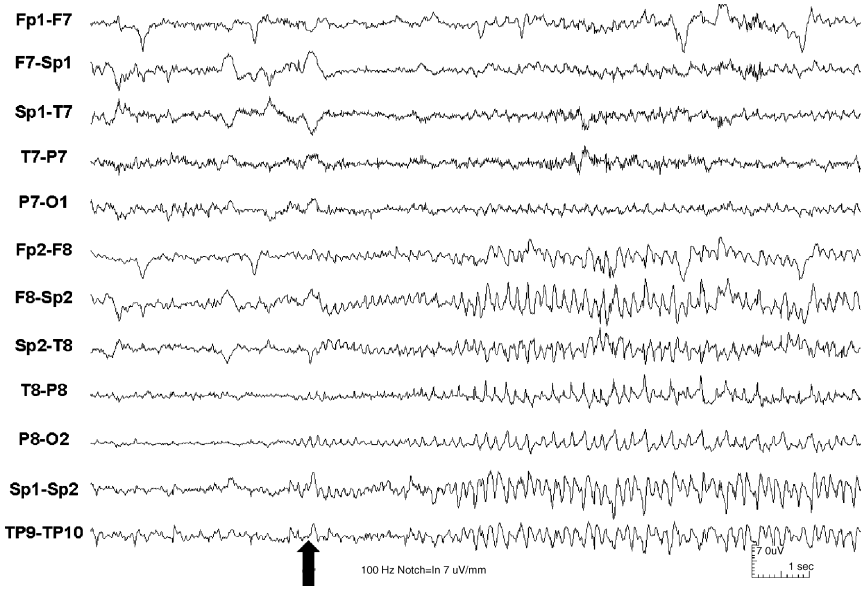

Figure 1 The arrow marks ictal onset characterized by rhythmic theta activity over the right temporal region, clearly visualized in the sphenoidal electrode.

(rhythmic theta/alpha activity): 17 (49%); (f) RBA (rhythmic beta activity): 6 (17%). Ten patients (30%) had no predominant ictal pattern. RTA was the most frequently encountered ictal pattern, as well as the most frequent predominant ictal pattern (Fig. 1). With respect to lateralization of the ictal pattern, patients were divided in the following groups: (a) lateralized seizures: 25 patients (71.5%); (b) non-lateralized seizures: 1 (3%); (c) seizures with switch of lateralization: 5 (14%); (d) bitemporal seizures: 4 (11.5%).

Neuroimaging data

As previously defined, all patients had unilateral hippocampal atrophy. Temporal pole MRI signal abnormalities were observed in 66% of the patients (21/32; three MRIs could not be evaluated due to suboptimal quality of FLAIR sequences). Except for one patient who had bilateral temporal pole abnormality, in all other cases the MRI findings were ipsilateral to the HS (Fig. 2). Regarding the volumetric assessment of the temporal poles that was performed for all patients, 60% of them (21/35) presented significant volume asymmetry between the two temporal poles. In all cases, the reduced temporal pole was ipsilateral to the atrophic hippocampus (Fig. 3). The MRI abnormalities observed in the temporal poles — signal changes and volume reduction — were correlated to the ictal patterns. For statistical comparison, due to the small size of each group of predominant ictal patterns, the patients were

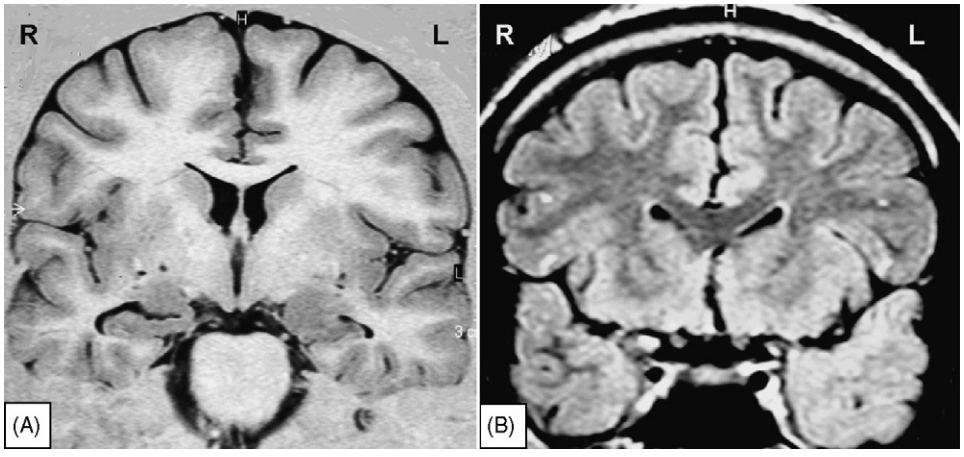

Figure 2 MRI coronal images. (A) IR sequence showing markedly atrophic left hippocampus and (B) FLAIR sequence revealing abnormal signal of left temporal pole, with loss of gray matter—white matter differentiation.

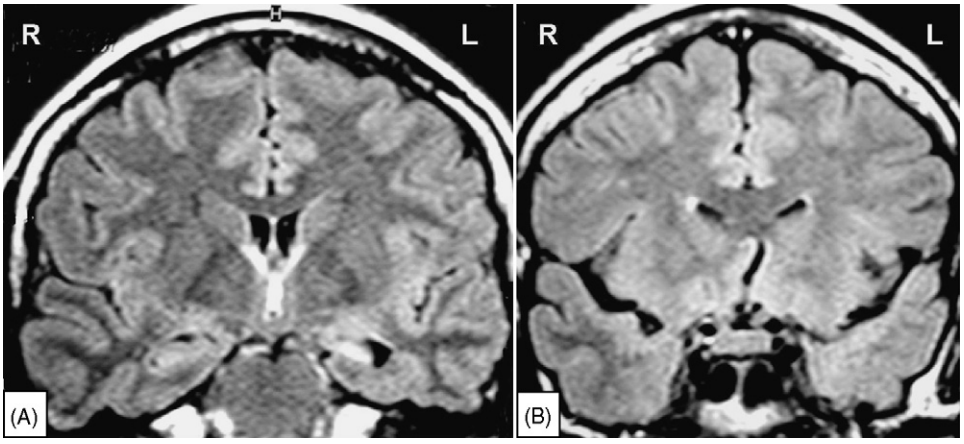

Figure 3 MRI coronal images. (A) FLAIR sequence showing atrophy and increased signal of the left hippocampus and (B) more anterior FLAIR sequence, revealing reduced left temporal pole.

divided in two separate groups: those with RTA as the predominant ictal pattern (RTA group); and those with predominantly other ictal patterns or with no predominant ictal pattern (others group). Patients with RTA as the predominant ictal pattern tended to have more temporal pole volumetric asymmetry but not temporal pole signal abnormalities (Table 2). Similarly, no clear association was observed when patients with lateralized seizures and those with non-lateralized seizures, seizures with switch of lateralization, and bitemporal seizures were compared taking in account data relative to abnormalities of temporal poles.

Discussion

In most patients with refractory TLE who are candidates to surgical treatment, non-invasive Video-EEG is sufficient for lateralization of the epileptogenic temporal lobe.[8] Optimization of non-invasive monitoring data is pursued in order to restrict invasive monitoring only to highly selected cases.[14] Detailed analysis of ictal patterns may provide useful information regarding lateralization of the epileptogenic temporal lobe as well as anatomic localization of the ictal activity.

Table 2 Correlation between predominant ictal pattern and volume asymmetries of temporal poles

Asymmetry	Predominant ictal EEG pattern		
	RTA	Other	Total
Yes	12 (57%)	9 (43%)	21 (100%)
No	5 (36%)	9 (64%)	14 (100%)
Total	17 (49%)	18 (51%)	35 (100%)

$p = 0.305$.

There may be concerns with respect to AED withdrawal during Video-EEG, since it might affect the patterns of ictal onset and/or propagation of ictal activity. Nevertheless AED withdrawal is routinely applied in most epilepsy surgery centers worldwide, and may in fact facilitate the occurrence of seizures during diagnostic monitoring.[15] However, it does not seem to modify the morphology of the initial ictal pattern or the time of propagation of ictal discharges.[16]

Following the classification adapted from Steinhoff et al.[13] in our series of patients with TLE and unilateral HS the most commonly observed ictal pattern was rhythmic theta activity (RTA). When patients were divided according to the predominant ictal pattern, near half of them showed this pattern as the predominant one. Thirty-one out of the 35 patients had at least one seizure with RTA as the initial ictal pattern. The marked predominance of this type of pattern is in accordance with previous data from the literature.

Steinhoff et al.[13] also found RTA as the initial ictal pattern in the vast majority of seizures in their patients with TLE who were seizure-free after anterior temporal lobectomy. Ebersole and Pacia[10] studied TLE patients with surface electrodes and concluded that rhythmic discharges with frequency from 5 to 9 Hz usually had onset in the hippocampus, while slower discharges had simultaneous onset in the hippocampus and temporal neocortex. These data were further confirmed by invasive recordings[17,18] when comparing ictal patterns presented by patients with TLE and unilateral HS and patients with neocortical TLE. RTA was the most frequent ictal pattern in the group of patients with HS, as opposed to repetitive epileptiform discharges, which predominated in patients with neocortical TLE. Giagante et al.[11] analysed ictal patterns from

26 patients with TLE and HS, and also found RTA as the most common pattern in 62% of the seizures.

Predominant ictal pattern may correlate to pathological findings in TLE. Initial ictal discharges in the 4—7 Hz range are associated to marked HS in surgical specimens, while slower frequencies (lower than 4 Hz) are associated to mild HS or normal histology.[19] Further analysis with invasive electrodes confirmed this association.[20]

A significant majority of the seizures presented by our patients (71.5%) were considered lateralized with respect to ictal onset. Our sample comprehended patients with unilateral IEDs (80%) but also with bilateral IEDs (17%) who could express some degree of bilateral epileptogenicity,[21—23] and lead to the occurrence of seizures with bilateral features. In patients with unilateral HS and exclusively unilateral IEDs, the degree of lateralization of ictal discharges also seems to be greater.[24,25]

Although ictal patterns may provide information with respect to lateralization of the epileptogenic temporal lobe, characteristics of initial ictal discharges do not seem to correlate to volumetric measures of mesial temporal structures in patients with mesial TLE. Spanedda et al.[26] analyzed seizures from 23 patients with TLE, and found neither correlation between the precise site of ictal onset (hippocampus, amigdala, or both) and the region of greater atrophy, nor between the morphology of ictal discharges and the pattern of atrophy of mesial temporal structures.

In our study, ictal patterns were not compared to degree of atrophy of hippocampus or other mesial structures. However, we compared these patterns to MRI abnormalities of the temporal poles. Patients with RTA as the predominant ictal pattern showed a tendency to have more often volume asymmetry between the two poles; however, due to the small size of the group, no statistically significant correlation could be demonstrated. No correlation was observed between the predominant ictal pattern and signal abnormalities of the poles.

Patients with TLE may present structural abnormalities, which extend beyond the hippocampal atrophy. Recently, special attention has been given to the study of the anterior portions of the temporal lobes, or temporal poles. The frequency of temporal pole abnormalities vary among different studies. Pageot et al.[27] studied 100 consecutive patients with refractory partial epilepsy, 54 of whom had TLE. Among these patients with TLE, 15 (28%) presented temporopolar white matter signal abnormalities, and 19 (35%) had temporal pole atrophy ipsilateral to the epileptogenic temporal lobe. Coste et al.[28] found significant volumetric asymmetry between temporal poles in 23 (77%) of 30 patients with TLE, 19 of whom showed signs of HS ipsilateral to the reduced temporal pole. Chabardès et al.[29] encountered temporal pole signal abnormalities in 48% (12/25) of the patients with TLE. The authors correlated these abnormalities to the occurrence of fast activity at the ictal onset of seizures recorded with invasive monitoring, stressing the potential role of the temporal poles in the origin of seizures in patients with TLE. Meiners et al.[30] found signal abnormalities in 66% of the 80 patients with TLE; these abnormalities were always ipsilateral to the atrophic hippocampus, enhancing the power of MRI for detecting lateralized structural abnormalities in this group of patients. Among 36 patients with medically refractory TLE, Mitchell et al.[31] found signal abnormalities in 23 (64%) patients. They defined these abnormalities as loss of gray matter-white matter differentiation, associated to abnormal signal in T2, IR and proton density sequences.

The variable frequency of temporal pole abnormalities among different studies is probably due to different MRI techniques. In our study, all exams were made following a pre-established protocol for assessment of the temporal lobes, including coronal FLAIR sequences perpendicular to the longer axis of the hippocampus. Using these sequences, which are very sensitive for detecting abnormalities of the temporal poles, we observed signal abnormalities in two thirds of the patients. In addition, through volumetric measures of the temporal poles, significant asymmetry was detected in 60% of all cases. Quantitative analysis of the temporal poles, with volumetric measures of these portions of the temporal lobes, may help confirm the presence of structural abnormalities.[32] In our series, signal abnormalities and volume reduction were always ipsilateral to the HS, therefore helping in the lateralization of the epileptogenic temporal lobe.

Atrophy and signal abnormalities in the temporal poles may have the same pathological substrates of the alterations observed in the hippocampus of patients with mesial TLE.[31] These abnormalities of the temporal poles may also be correlated to more prominent hypometabolism in the ipsilateral temporal lobe (as detected in PET scans), earlier age of onset of epilepsy, better post-surgical outcome with respect to control of seizures, and to the presence of heterotopic neurons in histopathological studies.[33] Loss of myelin may add to the presence of heterotopic neurons as a possible explanation for the signal abnormalities observed in the temporal poles of patients with TLE.[30]

The observation of atrophy and signal abnormalities in the temporal pole may suggest a potential role for this structure in the pathophysiology of TLE.

Failure of seizure control after anterior temporal lobectomy (ATL) in a significant portion of patients with TLE also suggests that the epileptogenic zone may not be restricted solely to the mesial structures of the temporal lobes.[34,35] There is enough evidence pointing to the fact that in patients with TLE due to HS, pathological abnormalities may be found beyond the hippocampus, in other mesial structures and in the white matter of the temporal lobe.[36] Ectopic neurons and oligodendrocyte-like perivascular infiltrates are frequently observed in the white matter of the temporal lobes of patients with TLE submitted to ATL.[37] However, the actual role of these ectopic neurons in the pathophysiology of the epilepsy in this group of patients is still matter of debate.[38]

These pathological alterations may explain the finding of signal abnormalities in the temporal poles as detected by MRI exams, although conflicting data exist concerning this possibility.[39] Further studies correlating imaging and pathological findings may help elucidate this question.

Structural abnormalities observed in the temporal poles may reinforce the evidences of involvement of these regions in seizures originated in the hippocampus.[29] This may also reflect the importance of the temporal poles in the origin or in the propagation of these seizures, but adequate evaluation of the temporal poles with appropriate MRI techniques might additionally contribute for the investigation of patients with suspected TLE.

In conclusion, in this study we observed that RTA is the most frequent initial ictal pattern in patients with mesial TLE due to unilateral HS. We additionally observed that temporal pole signal and volume abnormalities are frequent findings in this group of patients, occurring always ipsilateral to the atrophic hippocampus. This observation suggests that these findings might be of help in lateralizing the epileptogenic zone, especially in those patients whose MRIs do not show unequivocal evidences of temporal lobe structural lesion. Finally, we observed no correlation between temporal pole abnormalities and surface ictal patterns, suggesting that the temporal poles are not crucially involved in the process of epileptogenesis. However, caution must be exercised since TLE may include subgroups of patients with distinct pathophysiological substrates, and further analysis on larger groups of patients is probably needed to better assess these correlations.

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References

1. Foldvary N. Symptomatic focal epilepsies. In: Wyllie E, editor. *The treatment of epilepsy. Principles and practice*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001 . p. 467—74.
2. Semah F, Picot M-C, Adam C, Broglin D, Arzimanoglou A, Bazin B, et al. Is the underlying cause of epilepsy a major prognostic factor for recurrence? *Neurology* 1998;**51**:1256—62.
3. Najm IM, Babb TL, Mohamed A, Diehl B, Ng TC, Bingaman WE, et al. Mesial temporal lobe sclerosis. In: Lüders, Ho e Comair YG, editors. *Epilepsy surgery*. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 95—103.
4. Wiebe S, Blume WT, Girvin JP, Eliasziw M. A randomized, controlled trial of surgery for temporal lobe epilepsy. *New Engl J Med* 2001;**345**:311—8.
5. McIntosh AM, Wilson SJ, Berkovic SF. Seizure outcome after temporal lobectomy: current research practice and findings. *Epilepsia* 2001;**42**:1288—307.
6. Engel Jr J, Van Ness PC, Rasmussen TB, Ojemann LM. Outcome with respect to epileptic seizures. In: Engel Jr J, editor. *Surgical treatment of the epilepsies*. 2nd ed. New York: Raven Press; 1993. p. 609—21.
7. Engel Jr J. Current concepts: surgery for seizures. *New Engl J Med* 1996;**334**:647—52.
8. Rosenow F, Lüders HO. Presurgical evaluation of epilepsy. *Brain* 2001;**124**:1683—700.
9. Risinger MW, Engel Jr J, Van Ness PC, Henry TR, Crandall PH. Ictal localization of temporal lobe seizures with scalp/sphenoidal recordings. *Neurology* 1989;**39**:1288—93.
10. Ebersole JS, Pacia SV. Localization of temporal lobe foci by ictal EEG patterns. *Epilepsia* 1996;**37**:386—99.
11. Giagante B, Oddo S, Silva W, Consalvo D, Centurion E, D'Alessio L, et al. Clinical-electroencephalogram patterns at seizure onset in patients with hippocampal sclerosis. *Clin Neurophysiol* 2003;**114**:2286—93.
12. Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for classification of epilepsies and epileptic syndromes. *Epilepsia* 1989;**30**: 389—99.
13. Steinhoff BJ, So NK, Lim S, Lüders HO. Ictal scalp EEG in temporal lobe epilepsy with unitemporal versus bitemporal interictal epileptiform discharges. *Neurology* 1995;**45**: 889—96.
14. Diehl B, Lüders HO. Temporal lobe epilepsy: when are invasive recordings needed? *Epilepsia* 2000;**41**(Suppl. 3):S61—74.
15. Acharya JN, Lüders HO. Effect of anticonvulsant withdrawal on seizure semiology. In: Lüders, Ho e Comair YG, editors. *Epilepsy surgery*. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 451—6.
16. So N, Gotman J. Changes in seizure activity following anticonvulsant drug withdrawal. *Neurology* 1990;**40**:407—13.
17. Pacia SV, Ebersole JS. Intracranial EEG substrates of scalp ictal patterns from temporal lobe foci. *Epilepsia* 1997;**38**: 642—54.
18. Foldvary N, Klem G, Hammel J, Bingaman W, Najm I, Lüders H. The localizing value of ictal EEG in focal epilepsy. *Neurology* 2001;**57**:2022—8.
19. Vossler DG, Kraemer DLA, Knowlton RC, Kjos BO, Rostad SW, Wyler AR, et al. Temporal ictal EEG frequency correlates with hippocampal atrophy and sclerosis. *Ann Neurol* 1998;**43**: 756—62.

20. Vossler DG, Kraemer DL, Haltiner AM, Rostad SW, Kjos BO, Davis BJ, et al. Intracranial EEG in temporal lobe epilepsy: location of seizure onset relates to degree of hippocampal pathology. *Epilepsia* 2004;**45**:497—503.
21. Morrell F. Secondary epileptogenesis in man. *Arch Neurol* 1985;**42**:318—35.
22. Janszky J, Fogarasi A, Jokeit H, Schulz R, Hoppe M, Ebner A. Spatiotemporal relationship between seizure activity and interictal spikes in temporal lobe epilepsy. *Epilepsy Res* 2001;**47**:179—88.
23. Janszky J, Rásonyi G, Clemens Z, Schulz R, Hoppe M, Barsi P, et al. Clinical differences in patients with unilateral hippocampal sclerosis and unitemporal or bitemporal epileptiform discharges. *Seizure* 2003;**12**:550—4.
24. Cascino GD, Trenerry MR, So EL, Sharbrough FW, Shin C, Lagerlund TD, et al. Routine EEG and temporal lobe epilepsy: relation to long-term EEG monitoring, quantitative MRI, and operative outcome. *Epilepsia* 1996;**37**:651—6.
25. Pataraia E, Lurger S, Serles W, Lindinger G, Aull S, Leutmezer F, et al. Ictal scalp EEG in unilateral mesial temporal lobe epilepsy. *Epilepsia* 1998;**39**:608—14.
26. Spanedda F, Cendes F, Gotman J. Relations between EEG seizure morphology, interhemispheric spread, and mesial temporal atrophy in bitemporal epilepsy. *Epilepsia* 1997;**38**:1300—14.
27. Pageot N, Ryvlin P, Isnard J, Mauguière F. Temporopolar MRI changes in drug-resistant partial epilepsy: a retrospective study in 100 patients. *Epilepsia* 1998;**39**(Suppl. 6): 95.
28. Coste S, Ryvlin P, Hermier M, Mauguière F. Temporo-polar atrophy in TLE: a quantitative volumetric MRI study. *Epilepsia* 1999;**40**(Suppl. 7):189.
29. Chabardès S, Kahane P, Hoffmann D, Munari C, Benabid AL. Role of the temporo-polar region in the genesis of temporal lobe seizures. *Epilepsia* 1999;**40**(Suppl. 7):78.
30. Meiners LC, Witkamp TD, DeKort GAP, van Huffelen AC, van der Graaf Y, Jansen GH, et al. Relevance of temporal lobe white matter changes in hippocampal sclerosis. *Invest Radiol* 1999;**34**:38—45.
31. Mitchell LA, Jackson GD, Kalnins RM, Saling MM, Fitt GJ, Ashpole RD, et al. Anterior temporal abnormality in temporal lobe epilepsy: a quantitative MRI and histopathologic study. *Neurology* 1999;**52**:327—36.
32. Coste S, Ryvlin P, Hermier M, Ostrowsky K, Adeleine P, Froment JC, et al. Temporopolar changes in temporal lobe epilepsy. A quantitative MRI-based study. *Neurology* 2002;**59**:855—61.
33. Choi D, Na DG, Byun HS, Suh YL, Kim SE, Ro DW, et al. White matter changes in mesial temporal sclerosis: correlation of MRI with PET, pathology and clinical features. *Epilepsia* 1999;**40**:1634—41.
34. Henessy MJ, Elwes RDC, Binnie CD, Polkey CE. Failed surgery for epilepsy. A study of persistence and recurrence of seizures following temporal resections. *Brain* 2000;**123**:2445—66.
35. Kahane P, Chabardès S, Minotti L, Hoffmann D, Benabid AL, Munari C. The role of the temporal pole in the genesis of temporal lobe seizures. *Epileptic Disord* 2002;**4**(Suppl. 1):S51—8.
36. Bocti C, Robitaille Y, Diadori P, Lortie A, Mercier C, Bouthillier A, et al. The pathological basis of temporal lobe epilepsy in childhood. *Neurology* 2003;**60**:191—5.
37. Kasper BS, Stefan H, Buchfelder M, Paulus W. Temporal lobe microdysgenesis in epilepsy versus control brains. *J Neuropathol Exp Neurol* 1999;**58**:22—8.
38. Kasper BS, Stefan H, Paulus W. Microdysgenesis in mesial temporal lobe epilepsy: a clinicopathological study. *Ann Neurol* 2003;**54**:501—6.
39. Ryvlin P, Coste S, Hermier M, Mauguière F. Temporal pole MRI abnormalities in temporal lobe epilepsy. *Epileptic Disord* 2002;**4**(Suppl. 1):S33—9.