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Correlation of Electroencephalography and Magnetic Resonance Imaging in Patients with Mesial Temporal Sclerosis

Duygu ÖZKAN[1], Yılmaz ÇETİNKAYA[2], Ayşegül ÖZYILMAZ[1], Havva Tuğba ÇELİK[3], Cemile Handan MISIRLI[2], Hülya TİRELİ[2]

[1]Düzce Atatürk State Hospital, Department of Neurology, Düzce, Turkey
[2]Haydarpaşa Numune Education and Research Hospital, Department of Neurology, İstanbul, Turkey
[3]Malkara State Hospital, Department of Neurology, Tekirdağ, Turkey

ABSTRACT

Introduction: To determine the lateralization of lesion by non-invasive methods through correlation of cranial magnetic resonance imaging (MRI) and electroencephalography (EEG) findings in patients with mesial temporal sclerosis (MTS).

Methods: This study included 40 patients (Age range, 19 to 55 years) among 1850 patients who were attending outpatient epilepsy clinic of Haydarpaşa Numune Hospital between the years 2000 and 2013. Exclusion criteria were surgery due to MTS, metabolic and systemic disorders, indefinite diagnosis, and inadequate MRI and EEG evaluations.

Results: EEG findings were within normal limits in 10 (25%) patients who had MTS on MRI. Of these, 5 patients had right MTS, 4 had left MTS, and one patient had bilateral MTS.

Conclusion: In this study, we used electrophysiological diagnostic methods together with MR imaging to determine epileptogenic localization in patients with MTS. We suggest that, when correlated with MRI, EEG is a non-invasive and easy method to identify lateralization findings.

Keywords: Epilepsy, mesial temporal sclerosis, EEG-MRI correlation

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INTRODUCTION

Epilepsy is a group of neurological disorders resulting from unprovoked seizures due to neuronal hyperexcitability in the brain. They stem from increased, fast, local electrical discharges in the grey matter, and clinically, sudden onset, short, transient, and stereotypical changes are observed in consciousness, behavior, perception, motion, and feelings. In general, other neurological dysfunctions do not accompany the idiopathic (spontaneous or arising from an obscure genetic cause) syndromes. Proceeding of developmental steps is normal; there is no underlying pathology; neurologic examination, and imaging are within normal limits. Family history is significant. Seizures are relatively rare, and respond well to treatment. Interictal electroencephalography (EEG) shows normal basic electrical rhythm. Idiopathic form usually has a good prognosis, and remission can be seen.

In contrast, symptomatic epilepsy shows an underlying brain disorder, neurological symptoms, and slow basic electrical rhythm on EEG. Medical history reveals a condition such as cental nervous system (CNS) disease, head trauma, infection, a growing tumor, or a degenerative disease that may lead to seizures. Response to treatment is variable, and the rate of spontaneous remission is low. The term "cryptogenic (unknown etiology) epilepsy" is used to define epileptic syndromes with cognitive impairment, and neurologic deficit; it is suspected that there is an underlying cause responsible but it is not visible (Lennox Gastaut Syndrome, Doose Syndrome, etc.).

According to the International Classification of Epileptic Syndromes (ILAE, 1989), temporal lobe epilepsy is a symptomatic epilepsy, and is divided into three subgroups as: I) Epileptic seizures originating from outside the temporal lobe, or distant regions from mesial part of the lobe. Ictal discharges show fast spreading, and lead to clinical findings related to mesial region; II) Epileptic seizures related to structural lesions located in or close to the mesial temporal lobe (hamartoma, glial tumor, arteriovenous malformations, cortical dysplasia, etc.); and III) HS-related mesial temporal lobe epilepsy (mTLE) (1). Although hippocampal sclerosis (HS) has been known for many years as the most important pathologic component of the temporal lobe epilepsy, it was not described in the previous classifications of ILAE (2). Accumulating evidence suggest that HS-related TLE is a distinct syndrome which is called mesial temporal lobe epilepsy with hippocampal sclerosis (mTLE-HS). These patients are mostly resistant to drugs, and early diagnosis and surgery may provide significant psychosocial improvement. A concensus meeting was held in Istanbul in 2002, and ILAE epileptic surgery commission declared that mTLE-HS is not a separate disease. Rather, it is a common subtype of mTLE. It may show primary, secondary and familial forms (3). Before high-resolution

Correspondence Address: Duygu Özkan, Düzce Atatürk Devlet Hastanesi, Nöroloji Bölümü, Düzce, Turkey • **E-mail**: duyzkan01@gmail.com
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MRI, characteristic patients without a significant lesion were diagnosed with cryptogenic TLE. However, surgical materials obtained from these patients showed HS findings, and the term "cryptogenic" turned into the term "lesional". For the pathological changes seen in HS, terms such as Ammon's horn sclerosis or MTS are also used interchangeably.

Routine EEG of the patients with mTLE may show characteristic findings, or may be normal. The typical findings of interictal EEG may include both non-epileptiform and epileptiform abnormalities. Non-epileptiform abnormalities of mTLE are focal dysrhythmia/slowing, temporal intermittent rhythmic delta activity (TIRDA) whereas epileptiform discharges are spikes, and sharp waves. The anterior temporal spikes show maximum negativity over the temporal basal electrodes (4). These discharges may occur during transition from awake state to sleep, and NREM stage 1 and 2. They are suppressed during REM sleep. Williamson et al. (5) reported that 96% of the interictal EEG recordings of TLE patients had paroxysmal activity. Bilateral paroxysmal activity with a dominance on the seizure side was also observed. In addition, slowing during postictal period had significant lateralizing value (5).

The most important MRI finding of MTS is hippocampal atrophy and T2-signal hyperintensity. T1-T2-FLAIR-IR(fluid attenuated inversion recovery)-weighted sections may unveil hippocampal anatomy, and may reveal pathological changes (6–12). The most common MRI finding of hippocampal sclerosis is hippocampal atrophy (13). Qualitative comparison of hippocampi on both sides may show atrophy by using 1 to 2 mm sections at coronal plane. As distinguishing of hippocampal formation from amygdala at anterior regions is difficult, evaluation of atrophy may be difficult. Atrophy can be identified more easily at corpus level. Recently, computed volumetric MR measurements have also been performed recently besides visual assessment of unilateral hippocampal formation. Left and right hippocampal volumes are measured for quantitative evaluation. In general, variation difference may be present between both side hippocampal volumes. A correlation between hippocampal volume on MRI, and histopathologic neuronal density of surgical material was reported. In addition, it has been known that volume measurements are closely related to febrile convulsion history. If volumetric evaluation and EEG findings are lateralized to the same side, then the postoperative prognosis is good in 97% of the patients. Hippocampal volume loss can be seen in Alzheimer's disease, vascular dementia, and Parkinson's disease besides MTS. There are indirect secondary findings of MTS including widening of neighbour temporal horn, anterior hippocampal formation atrophy, ipsilateral collateral white matter loss, ipsilateral anterior temporal lobe, and parahippocampal gyrus atrophy, fornix and mamillary body atrophy.

Volumetric measurements of amygdala and hippocampus on MRI provided benefits in diagnosis of temporal lobe epilepsy. When volumetric MRI is combined with EEG, neuropsychiatric studies and other brain imaging methods, epilepsy patients can be diagnosed correctly, and costs of treatment can be lowered. Especially in bilateral ictal onset TLE, amygdala, and hippocampus volume measurements together with localization studies are beneficial for treatment. Thus, volumetric MRI may have additional value when combined with other brain imaging methods (14, 15).

METHODS

This retrospective study included 40 MTS patients (M/F, 21/19; mean age, 30 years; age range, 19 to 55 years) among 1850 patients who were attending outpatient epilepsy clinic of Haydarpaşa Numune Hospital between the years 2000–2013. Diagnosis was based on medical history, clinical examination, EEG, and cranial MRI findings. (Table 1)

MR images were obtained using different devices (0.5, 1.0 and 1.5 T) in different centers, including our hospital. T2 weigted sagittal and transverse sections were taken in all cases. The evaluation of the images was made by the same radiologist. The gold diagnostic criteria for MTS were as follows:

1. Hippocampal atrophy (Fig. 1a).
2. Increased signal intensity in hippocampus in T2-weighted MRI (Fig. 1b).
3. Abnormal morphology (dilatation of the ipsilateral temporal horn, loss of internal architecture).

Interictal scalp EEG recordings were made by using 10–20 electrode placement system, and EEG device (Nicolet One REF 2007) for 30 minutes. Exclusion criteria were surgery due to MTS, metabolic and systemic disorders, indefinite diagnosis, and inadequate MRI and EEG evaluations.

RESULTS

The mean age of first seizure was 13 years, and the average duration of the disease was 18 years. The frequency of seizures was once in 1 or 2 months. Seizure type was complex partial which sometimes turned into generalized tonic clonic. Nineteen patients had febrile convulsions, 4 patients had afebrile convulsions, 8 patients had trauma history, 4 patients had mental-motor retardation, and 1 patient had infection history.

EEG recording was within normal limits in 10 patients (6 female, 4 male), whereas it was pathologic in 30 patients (13 female, 17 male). Of the patients with normal EEG, 5 had right MTS, 4 had left MTS, and 1 had bilateral MTS. Seven patients showed slow wave activity on the MTS side, and 2 of these had theta activity on EEG (Fig. 1c). Of the patients, 83% had ipsilateral pathologic EEG with MTS. Six (15%) patients with bilateral MTS showed diffuse slowing on EEG which was sometimes lateralized to more severe lesioned side. Male gender was higher among epilepsy patients who were mostly young or middle aged. The mean age of male patients was 30 years whereas it was 31 for female patients. The mean age of first seizure was 11 for males, and 15 for females.

DISCUSSION

The most common pathological finding in temporal lobe epilepy is hippocampal sclerosis. Nevertheless, temporal lobe tumors or tumor-like space occupying lesions, vascular malformations, cortical developmental abnormalities, trauma, and infections may also form an epileptogenic focus (1). The onset of seizures is around the first decade, and these initial seizures respond well to antiepileptic treatment. During adolescence and adulthood, seizures become resistant to medical treatment. These may include complex febrile seizures or other triggering factors (16). The mean age of onset was 13 in our study group. In 1993, French et al. studied 67 patients, and the rate of convulsion history during infancy or childhood was 81%. Of these, 52 had febrile convulsions while 7 had central nervous system infection. Although other risk factors were rare, the rates of head trauma and labor trauma were 10% and 3%. Only 5 patients revealed no known risk factor. The onset of seizures occurred around 9 years of age; and, half of the patients had only complex partial seizures whereas the other half had complex partial, and secondary generalized seizures. Three patients had convulsive status epilepticus. Thirty-nine of the patients had the feeling of epigastric elevation which is the most common aural symptom of MTS (16).

The relationship of MTS and febrile seizures has been known for a long time, and verified in retrospective studies. Prospective studies remained controversial (16–18). In 1998, Vanlandingham et al. (17) investigated MRI findings of 27 infants following a complex febrile convulsion. Of the 15 infants with focal or lateralized febrile convulsion, 6 had significant MRI

Table 1. Clinical and laboratorial characteristics of 40 patients

Patient	Age (years)	Sex	First Seizure (years)	Seizure Frequency	Medical history	Seizure type	EEG findings	MR Findings (MTS)
1	46	M	22	4/per month	No	CPS	Normal	Right
2	33	F	27	2/per year	FC	CPS+GTCS	LTRED	Left
3	30	M	12	2–3/per month	Trauma	GTCS	Normal	Left
4	36	F	25	1/per month	FC	SPS	Normal	Left
5	40	M	20	1/per month	FC	CPS+GTCS	LTTA	Left
6	32	F	18	everyday	FC+MMR	CPS+GTCS	Normal	Bilateral
7	19	F	11	2/per year	No	GTCS	Normal	Right
8	19	F	13	1/per year	No	CPS	LTSA	Left
9	21	M	16	3/per month	No	CPS	LTSA	Left
10	38	F	5	1/per month	FC	CPS	Normal	Left
11	32	M	14	2–3/per month	Trauma	CPS+GTCS	BTDA	Bilateral
12	22	M	5	4/per year	FC+MMR	CPS+GTCS	LTRED	Left
13	26	F	0	2/per year	FC+AFC	CPS+GTCS	Normal	Right
14	24	M	3	Every 3 years	FC+AFC	GTCS	RTID	Right
15	25	M	13	4/per month	Infection	SPS+CPS	BRED	Right
16	20	F	11	6/per month	No	GTCS	RFTRED	Right
17	24	M	15	2/per year	No	GTCS	BRED	Right
18	25	F	17	2/per month	No	CPS+GTCS	RTTA	Right
19	37	F	25	3–4/per month	No	CPS+GTCS	BTRED	Right
20	13	F	22	1/per year	No	CPS	BTTA	Right
21	38	F	1	4/per year	FC	CPS+GTCS	LTID	Left
22	40	F	13	6/per year	FC	CPS+GTCS	LTID	Bilateral
23	41	M	10	2/per year	FC	SPS+CPS	Normal	Right
24	37	F	6	4/per month	FC	CPS+GTCS	RTRED	Right
25	26	M	2	6/per year	AFC	SPS+GTCS	LTRED	Left
26	33	M	9	1/per month	FC	SPS	Normal	Left
27	32	F	10	4/per year	AFC+Trauma	CPS+GTCS	LTTA	Left
28	30	F	0	6/per year	Trauma	GTCS	BTTA	Left
29	21	M	0	1/per year	FC+Trauma	CPS+GTCS	LTDA	Bilateral
30	19	M	12	4/per month	FC+Trauma	CPS+GTCS	BTID	Bilateral
31	27	F	18	1/per year	FC+MMR	CPS	BTID	Right
32	31	M	19	4/per month	FC	CPS+GTCS	LTRED	Left
33	31	M	0	2–3/per week	MMR	CPS	LTRED	Left
34	32	F	16	1/per year	No	CPS+GTCS	Normal	Right
35	37	M	0	4 per/year	FC+Trauma	GTCS	BTID	Bilateral
36	55	F	40	1/per month	No	CPS+GTCS	BTRED	Left
37	39	M	18	1/per month	No	CPS+GTCS	LTID	Left
38	29	M	18	1/per year	FC	CPS+GTCS	BTRED	Right
39	37	M	10	3/per week	No	CPS	BTRED	Right
40	19	M	10	1/per year	Trauma	CPS+GTCS	BTRED	Left

M, Male; F, Female; FC, Febrile Convulsion; AFC, Afebrile Convulsion; MMR, Motor Mental Retardation; CPS, Complex Partial Seizure; GTCS, Generalized Tonic-Clonic Seizure; SPS, Simple Partial Seizure; LTRED, Left Temporal Repetitive Epileptiform Discharges; RTRED, Right Temporal Repetitive Epileptiform Discharges; LTTA, Left Temporal Theta Activity; LTSA, Left Temporal Spike Activity; BTDA, Bilateral Temporal Delta Activity; RTID, Right Temporal Interruption of Epileptiform Discharges; BRED, Bilateral Repetitive Epileptiform Discharges; RTTA, Right Temporal Theta Activity; BTRED, Bilateral Temporal Repetitive Epileptiform Discharges; BTTA, Bilateral Temporal Theta Activity; LTID, Left Temporal Interruption of Epileptiform Discharges; LTDA, Left Temporal Delta Activity; BTID, Bilateral Temporal Interruption of Epileptiform Discharges.

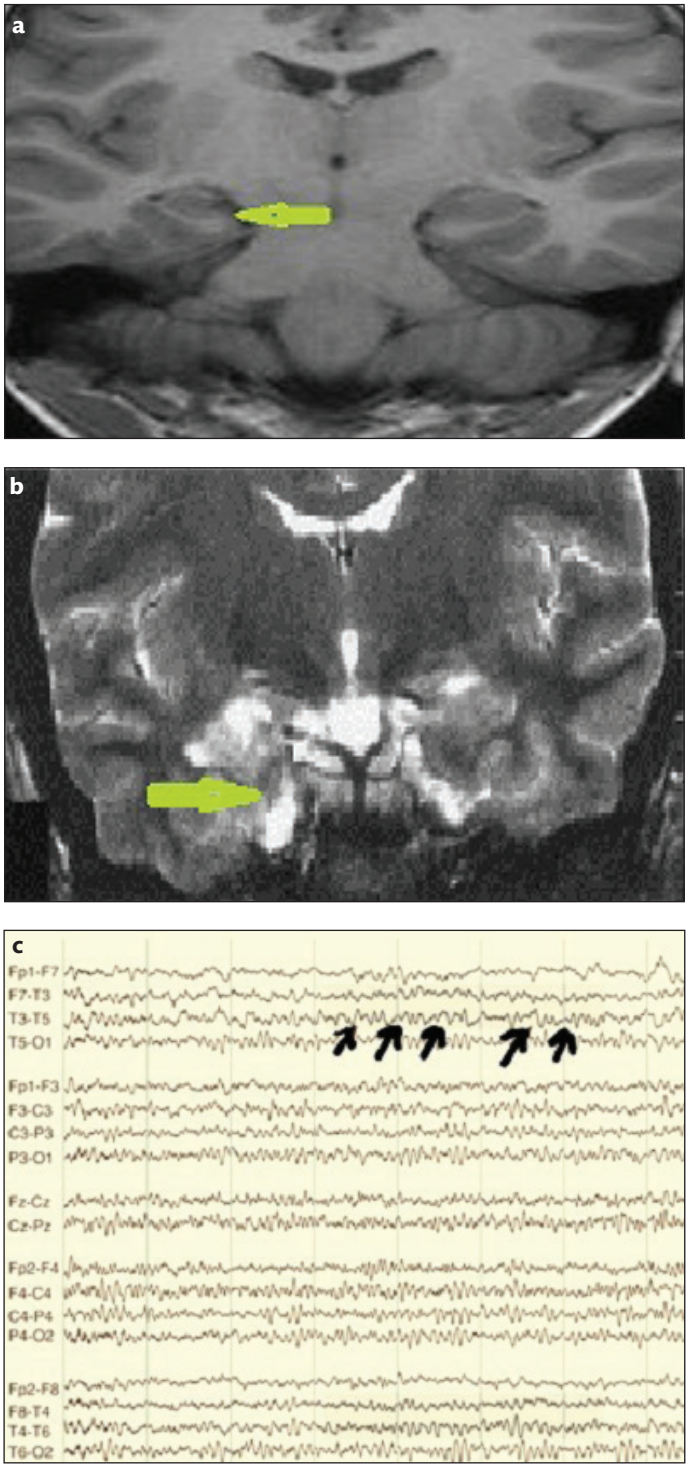

Figure 1. a-c. T1 weighted coronal image; right hippocampal atrophy (a). T2 weighted coronal image; increased signal intensity in right hippocampus (b). Theta activity in the left temporal lobe (c).

abnormalities. Twelve infants who had generalized febrile convulsion showed no abnormality. Of the 6 patients with MRI pathology, 2 had bilateral hippocampal atrophy which was considered to be related with perinatal trauma. The remaining 4 patients had prolonged seizures and MRI findings including T2 signal hyperintensity and edema at hippocampal regions of the hemisphere from which the seizure was originated. They showed that prolonged focal febrile convulsions may lead to acute hippocampal damage, and eventually hippocampal atrophy (17).

Tarkka et al. (18) investigated causal relationship between febrile convulsions and MTS. They followed up 3 patient groups for 12.3 years. Group 1 included 24 patients with prolonged first seizure, Group 2 included 8 patients with unprovoked seizure after the first febrile convulsion, and Group 3 included 32 patients with single simple febrile seizure. None of these study groups showed MTS during the follow up period. They failed to find a link between febrile convulsion and MTS (18). Conversely, in our study, 19 of 40 patients had febrile convulsion history.

The most common MRI finding of hippocampal sclerosis is hippocampal atrophy. Besides visual evaluation of unilateral hippocampal formation atrophy, computed volumetric MR measurements have also been performed recently. In general, there is a variation difference between the two sides in terms of hippocampal volume. If volumetric evaluation and EEG findings are lateralized to the same side, then the postoperative prognosis is good in 97% of the patients (13).

Volumetric measurements of amygdala and hippocampus on MRI provided benefits in diagnosis of temporal lobe epilepsy. When volumetric MRI is combined with EEG, neuropsychiatric studies, and other brain imaging methods, epilepsy patients can be diagnosed correctly, and costs of treatment can be lowered. Especially in bilateral ictal onset TLE, amygdala, and hippocampus volume measurements together with localization studies are beneficial for treatment. Thus, volumetric MRI may have additional value when combined with other brain imaging methods (14, 15).

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Ictal EEG findings have been identified in detail (3, 5, 19, 20). Ictal scalp EEG may be normal in 60% of the patients. The start of ictal EEG shows disruption of interictal spikes, and diffuse flattening occurs. It may show localization or lateralization. Many seizure activities contain unilateral 5–7 Hz rhythmical discharges during the first 30 seconds of ictal EEG abnormality. Rhythmical crescendo theta activity is a typical finding. In our study, 30 out of 40 patients had abnormal EEG which indicated the side of morphological pathology, or the more severe side of the pathology.

In this study, we used electrophysiological diagnostic methods together with MR imaging to determine epileptogenic localization in patients with MTS. We suggest that when correlated with MRI, EEG is a non-invasive, and easy method to identify lateralization findings.

Ethics Committee Approval: Due to retrospective design of the study, no ethical approval has been taken..

Informed Consent: Due to retrospective design of the study, no informed consent has been taken.

Author Contributions: Concept - DÖ; Design - YÇ; Supervision - TÇ; Resource - CHM; Materials - HT; Data Collection and/ or Processing - DÖ; Analysis and/or Interpretation - AÖ; Literature Search - DÖ; Writing - DÖ; Critical Reviews - YÇ.

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