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Temporal lobe ictal behavioral patterns in hippocampal sclerosis and other structural abnormalities

Gulnihal Kutlu [a,*], Erhan Bilir [b], Atilla Erdem [c], Yasemin B. Gomceli [a], Alev Leventoglu [d], G. Semiha Kurt [e], Ayse Karatas [c], Ayse Serdaroglu [f]

[a] Department of Neurology, Ankara Training and Research Hospital, Ministry of Health, Ankara, Turkey
[b] Department of Neurology, Medical Faculty, Gazi University, Turkey
[c] Department of Neurosurgery, Medical Faculty, Ankara University, Ankara, Turkey
[d] Department of Neurology, Medical Faculty, Kirikkale University, Turkey
[e] Department of Neurology, Medical Faculty, Gaziosmanpase University, Turkey
[f] Department of Pediatrics, Medical Faculty, Gazi University, Turkey

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Abstract

Ictal behavioral characteristics may provide clues in determining the nature of the epileptic focus. We defined ictal behavioral characteristics in patients with intractable temporal lobe epilepsy (TLE) who underwent anterior temporal lobectomy (ATL) and lived seizure-free for 2 years of follow-up. Video/EEG data on 282 seizures observed in 48 patients who suffered from TLE and underwent ATL were analyzed. All patients were seizure-free after surgery. We divided the patients into two groups on the basis of the pathological examination. Two hundred and two seizures in 35 patients with hippocampal sclerosis (Group 1) and eighty seizures in 13 patients with other pathological findings, such as tumors, cavernoma, and hamartoma (Group 2), were analyzed. Ictal behavior characteristics were evaluated for each of the seizures recorded in the two groups. Behavioral arrest, bilateral hand automatisms, oral and leg automatisms, and ictal aggression were significantly more frequent in Group 2 ($P < 0.05$), whereas contralateral dystonia of the upper extremity ($P < 0.05$), ipsilateral hand automatisms ($P < 0.05$), ipsilateral hand automatisms in the presence of contralateral dystonia of the upper extremity ($P < 0.001$), contralateral forced head deviation ($P < 0.05$), and secondary generalization ($P < 0.05$) were more significant in Group 1. There was no significant difference in vocalization and ipsilateral nonforced head deviation between the two groups ($P > 0.05$). The number of seizures observed during ictal speech, crying, and postictal nose wiping was not large enough, so differences could not be analyzed. It was concluded that although ictal behavioral characteristics differed between the two groups, certain behavioral patterns may be helpful in differentiating between hippocampal sclerosis and other pathology.

Keywords: Temporal lobe epilepsy; Hippocampal sclerosis; Ictal behavior; Ictal EEG

1. Introduction

In patients with epilepsy, temporal lobe epilepsy (TLE) is observed mainly in adults (70%). Various lateralization signs are described during temporal lobe seizures by video/EEG monitoring [1–7]. These include contralateral dystonic posturing [5,8], unilateral hand automatisms [2,6], early ipsilateral head deviation [9], forced contralateral head deviation [2,5,9], ictal speech [10–12], ictal vomiting [13,14], unilateral eye blinking [4], postictal nose wiping [3], automatisms with preserved consciousness [15], and postictal dysphasia/aphasia [16]. However, the specificity of these signs is unclear;

* Corresponding author: Fax: +90 312 4663626.
E-mail address: drgkutlu@hotmail.com (G. Kutlu).

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some of these signs are only of limited value in lateralizing the side of the focus [2]. Most patients with TLE manifest none of these features [17].

In this study, we investigated which ictal behavioral patterns can be useful in estimating the pathological diagnosis of the lesion. We compared ictal behavioral characteristics of seizures in patients with hippocampal sclerosis and other underlying pathologies (tumors, vascular abnormalities, hamartoma, etc.).

2. Materials and methods

Four hundred and sixteen patients were evaluated at the Gazi University Medical Faculty Telemetry Center between October 1997 and January 2003. Seventy-three patients underwent anterior temporal lobectomy (ATL) with amygdalohippocampectomy (AH) in the Ankara University Medical Faculty Neurosurgery Department. Video/EEG data on 282 seizures observed in 48 patients who remained seizure-free at least 2 years after surgery were analyzed. Patients with dual pathology, patients who had a seizure after surgery, and patients who were recently operated (follow-up < 2 years) were excluded. This study included patients with bilateral hippocampal atrophy on cranial MRI when the unilateral temporal focus could be determined.

First, a detailed medical history was taken from all patients. Each patient was then given physical and neurological examinations and neuropsychological tests. Long-term scalp video/EEG monitoring, cranial MRI, and the Wada test followed. Sphenoidal electrodes were used in scalp video/EEG monitoring of 15 patients. Eight of the patients were remonitored using subdural strip electrodes.

Video/EEG monitoring was performed with the Telefactor system, with up to 32 channels of EEG recorded continuously. The International 10–20 system was used to insert electrodes. Automatic seizure and spike detection programs were used, and all data were evaluated by an epileptologist, a pediatric neurologist, and two neurology researchers. All patients were monitored until at least two stereotypical seizures were observed.

Sphenoidal electrodes were replaced with anterior temporal electrodes. Strip electrodes were made from a single row of six electrodes in contact and of approximately 2-mm diameter at a fixed interelectrode distance of 10 mm. Strip electrodes could be inserted through burr holes in different directions in the subdural space on the basis of scalp ictal EEG findings.

We divided the patients into two groups: Group 1 patients had hippocampal sclerosis, and Group 2 patients had other pathology. Group 1 contained 35 patients (72.9%) and Group 2 included 13 patients (27.1%). All patients with a pathological diagnosis were operated by the same neurosurgeon in the Ankara University Neurosurgery Department. Surgical specimens were assessed by the same pathologist. ATL with AH was performed in all patients. All lesions of Group 2 patients were in the mesial temporal region. One patient in Group 2 had a colorless temporal area including mesial structures on macroscopic examination, but the pathological examination revealed neocortical gliosis. None of the patients in Group 2 had dual pathology on MRI or histopathologic analysis. Pathological examination revealed tumors in 8 patients (3 astrocytomas, 3 oligodendrogliomas, 1 ganglioglioma, 1 dysembryoplastic neuroepithelial tumor), and neocortical gliosis, hamartoma, cavernoma, arachnoid cyst, and cavernous hemangioma in the remaining 5 patients, respectively. Hippocampal sclerosis was defined in cases in which the hippocampus exhibited neuronal loss greater than 50%.

Ictal behavioral patterns were retrospectively evaluated for each patient in detail. These patterns were aura, behavioral arrest, oral automatisms, ipsilateral/bilateral hand automatisms, contralateral/bilateral dystonic posturing, ipsilateral automatisms in the presence of contralateral dystonic posturing, ipsilateral/contralateral versive/nonversive head and eye deviation, ictal vocalization, ictal speech, leg automatisms, unilateral eye blinking, automatisms with preserved responsiveness, aggression, ictal crying, postictal nose wiping, and secondary generalization.

Two hundred and eighty-two seizures were reviewed with respect to these characteristics by a neurologist who was not blind to the pathological diagnosis. χ^2 and Student t tests were used to analyze statistics.

3. Results

Forty-eight patients had a total of 282 seizures. Twenty-two (45.8%) were male and twenty-six (54.2%) were female. The mean age of the patients was 25.43 ± 8.21 (range: 7–51). Thirty patients (62.5%) underwent left ATL, and eighteen patients, right ATL. Interictal EEG, ictal EEG, MRI, and pathological findings for Group 1 and Group 2 patients are summarized in Tables 1 and 2, respectively. In 30 Group 1 patients the epileptogenic focus was localized to the mesial temporal region. Invasive monitoring using subdural strip electrodes was employed in the other five patients for whom the location of the epileptogenic focus had not been determined. In those five patients, invasive ictal EEG findings indicated that the focus was in the mesial temporal region. The mesial temporal region was determined to be the epileptogenic focus in 10 patients in Group 2 on scalp EEG monitoring. We could not determine the exact location of the focus in the other three patients, so they were reevaluated using subdural strip electrodes. The seizures of two of these patients origi-

Table 1
Interictal and ictal EEG and MRI findings for Group 1 (hippocampal sclerosis group)

Age/sex/side	MRI	Interictal EEG	Ictal focus	Ictal EEG pattern
28/F/R	R hippocampal atrophy	R temporal sharp waves	R mesial temporal	R mesial temporal focal spikes become bilateral after 8 s and rhythmic activity
18/F/R[a]	Bilateral hippocampal atrophy	R frontotemporal sharp waves	R fronto temporal	R frontotemporal spikes becoming bilateral after 10 s and rhythmic activity
18/F/L	L hippocampal atrophy	L mesial temporal sharp and slow waves	L mesial temporal	L mesial temporal rhythmic theta activity
29/F/L	L hippocampal atrophy	Normal	L mesial temporal	L mesial temporal rhythmic theta activity
25/M/R	R hippocampal atrophy	R temporal sharp waves	R mesial temporal	R mesial temporal rhythmic theta activity
20/F/R	R hippocampal atrophy	R frontotemporal slow waves	R mesial temporal	R mesial temporal focal spikes spread frontal region after 5 s and rhythmic activity
30/F/R	R hippocampal atrophy	R temporal sharp waves	R mesial temporal	R mesial temporal focal spikes spread frontal and contralateral temporal region after 7 s and arhythmic activity
29/M/L	Bilateral hippocampal atrophy	L temporal slow waves	L mesial temporal	L mesial temporal focal spikes become bilateral after 4 s and rhythmic activity
30/F/L	Normal	Normal	L mesial temporal	L mesial temporal focal spikes become bilateral after 8 s and rhythmic activity
20/F/R	R hippocampal atrophy	R temporal sharp and slow waves	R mesial temporal	R mesial temporal rhythmic theta activity
20/F/L	L hippocampal atrophy	Normal	L mesial temporal	L mesial temporal slowing become bilateral after 10 s and rhythmic activity
19/M/L	L hippocampal atrophy	L temporal sharp waves	L mesial temporal	L mesial temporal focal spikes become bilateral after 10 s and rhythmic activity
21/F/R	R hippocampal atrophy	R temporal slow waves	R mesial temporal	R mesial temporal rhythmic theta activity
17/F/L	L hippocampal atrophy	L temporal sharp waves	L mesial temporal	L mesial temporal focal spikes spread to frontal and contralateral temporal region after 10 s and rhythmic activity
20/F/L[a]	L hippocampal atrophy	L temporoparietal sharp waves	L temporo parietal	L temporoparietal rhythmic delta activity
20/F/R	R hippocampal atrophy	R temporal sharp waves	R mesial temporal	R mesial temporal focal spikes become bilateral after 5 s and rhythmic activity
32/M/L[a]	Bilateral hippocampal atrophy	L temporal slow waves	L fronto temporal	L frontotemporal focal spikes become bilateral after 5 s and arhythmic activity
31/M/L	L hippocampal atrophy	L frontotemporal slowing	L mesial temporal	L mesial temporal slow waves become bilateral after 4 s and rhythmic activity
24/M/L	L hippocampal atrophy	L temporal sharp waves	L mesial temporal	L mesial temporal rhythmic theta activity
28/F/R	R hippocampal atrophy	R temporal slow waves	R mesial temporal	R mesial temporal rhythmic theta activity
31/M/L	L hippocampal atrophy	L temporal slow waves	L mesial temporal	L mesial temporal focal spikes become bilateral after 3 s and arhythmic activity
23/F/L	L hippocampal atrophy	L temporal sharp and slow waves	L mesial temporal	L mesial temporal focal spikes become bilateral after 5 s and rhythmic activity
37/M/L	L hippocampal atrophy	L temporal slow waves	L mesial temporal	L mesial temporal rhythmic theta activity
25/M/R	R hippocampal atrophy	R temporal sharp waves	R mesial temporal	R mesial temporal focal spikes become bilateral after 3 s and rhythmic activity
30/F/L	L hippocampal atrophy	L temporal sharp and slow waves	L mesial temporal	L mesial temporal slow waves spread to frontal and contralateral temporal region after 4 s and rhythmic activity
49//F/R	R hippocampal atrophy	R temporal slow waves	R mesial temporal	R mesial temporal rhythmic theta activity
33/M/L	L hippocampal atrophy	L temporal sharp waves	L mesial temporal	L mesial temporal focal spikes become bilateral after 3 s and rhythmic activity
28/M/R	R hippocampal atrophy	R temporal slow waves	R mesial temporal	R mesial temporal slow waves spread to frontal and contralateral temporal region after 5 s and rhythmic activity
29/M/R[a]	R hippocampal atrophy	Bitemporal sharp and slow waves	R mesial temporal	Bitemporal focal spikes
24/F/L	L hippocampal atrophy	L temporal sharp waves	L mesial temporal	L mesial temporal focal spikes become bilateral after 3 s and arhythmic activity
23/M/L	L hippocampal atrophy	L temporal sharp waves	L mesial temporal	L mesial temporal focal spikes become bilateral after 2 s and rhythmic activity
20/M/R	Bilateral hippocampal atrophy	R temporal sharp waves	R mesial temporal	R mesial temporal continuous sharp waves
22/M/L	L hippocampal atrophy	L temporal sharp and slow waves	L mesial temporal	L mesial temporal focal spikes become bilateral after 4 s and rhythmic activity
29/F/L	L hippocampal atrophy	L temporal sharp waves	L mesial temporal	L mesial temporal focal spikes spread to frontal region after 3 s and rhythmic activity
22/M/R[a]	R hippocampal atrophy	R frontotemporal sharp waves	R frontotemporal	R frontotemporal sharp and slow waves

[a] These patients were reevaluated using subdural strip electrodes and the mesial temporal region was defined as the epileptogenic focus.

Table 2
Interictal and ictal EEG and MRI findings and pathological examinations in Group 2

Age/sex/side	MRI	Pathology	Interictal EEG	Ictal focus	Ictal EEG pattern
43/M/L	L temporal tumor	DNET[a]	L temporal sharp waves	L mesial temporal	L mesial temporal focal spikes become bilateral after 2 s and rhythmic activity
26/M/R	R temporal tumor	Oligodendroglioma	R temporal sharp waves	R mesial temporal	R mesial temporal focal slowing and spikes become bilateral after 4 s
7/F/L[b]	Bilateral (L > R) hippocampal atrophy	Hamartoma	Bitemporal sharp waves	L mesial/ bilateral temporal	L mesial temporal focal spikes become bilateral after 2 s and arhythmic activity
13/M/L[c]	Normal	Neocortical gliosis	L frontotemporal sharp waves	L fronto temporal	L frontotemporal continuous sharp and slow waves
30/M/R	R temporal cavernoma	Cavernoma	R temporal sharp waves	R mesial temporal	R mesial temporal rhythmic theta activity
30/M/L	Normal	Focal astrocytoma	L temporal sharp and slow waves	L mesial temporal	L mesial temporal focal slow waves become bilateral after 4 s and rhythmic activity
18/F/L	L temporal tumor	Low-grade astrocytoma	L temporal sharp waves	L mesial temporal	L mesial temporal focal slow and sharp waves become bilateral after 2 s and rhythmic activity
20/F/L	L temporal tumor	Oligodendroglioma	L temporal sharp waves	L mesial temporal	L mesial temporal rhythmic theta activity
21/M/L	L temporal cavernoma	Cavernous hemangioma	L temporal sharp and slow waves	L mesial temporal	L mesial temporal focal spikes spread to the frontal region after 3 s and rhythmic activity
51/F/L	L temporal tumor	Low-grade astrocytoma	L temporal sharp and slow waves	L mesial temporal	L mesial temporal focal slow waves become bilateral after 5 s and rhythmic activity
17/F/L	L temporal tumor	Ganglioglioma	L temporal PLEDS	L mesial temporal	L mesial temporal rhythmic theta activity
20/F/L	L arachnoid cyst	Arachnoid cyst	L temporal sharp waves	L mesial temporal	L mesial temporal focal spikes spread to the frontal region after 3 s and rhythmic activity
21/F/R[b]	Normal	Oligodendroglioma	R temporal slow waves	R frontotemporal	R frontotemporal continuous sharp waves

[a] DNET, dysembryoplastic neuroepithelial tumor; PLED: periodic lateralized epileptiform discharges.
[b] These patients were reevaluated using subdural strip electrodes, and the mesial temporal region was defined as the epileptogenic focus.
[c] This patient was reevaluated using subdural strip electrodes, and the temporal region (including posterior temporal area) was determined as the epileptogenic focus. Pathological examination revealed neocortical gliosis.

nated in the mesial temporal region. In the remaining patient, the epileptogenic focus was localized to the entire temporal region on ictal invasive EEG monitoring (patients with neocortical gliosis).

The scalp ictal EEG findings for Group 1 demonstrated mesial temporal rhythmic theta activity in 9 (30%) patients, mesial temporal sharp and/or slow waves becoming bitemporal within 2 to 10 s in 14 (46.7%) patients, mesial temporal sharp and/or slow waves spreading to the frontal and/or contralateral temporal regions within 3 to 10 s in 6 (20%) patients, and mesial temporal continuous sharp waves in one (3.3%) patient. On scalp video/EEG monitoring of Group 2, rhythmic theta activity, mesial temporal sharp and/or slow waves becoming bitemporal, and mesial temporal sharp and/or slow waves spreading to frontal and/or contralateral temporal regions were observed in 3 (30%), 5 (50%), and 2 (20%) patients, respectively.

The mean duration of monitoring was 4.62 ± 2.08 days. Two hundred and two seizures were reviewed in Group 1 and eighty seizures were investigated in Group 2. We evaluated an average of 5.77 seizures per patient in Group 1 and 6.15 seizures per patients in Group 2. Risk factors of the patients are listed in Table 3. Febrile convulsions were reported by 18 patients in Group 1 and by only one patient in Group 2. Statistically, there were no differences between these two groups in terms of age, sex, side of surgery, seizure frequency, and antiepileptic treatment ($P > 0.05$).

The auras of the patients in the two groups are listed in Table 4. Epigastric sensation was the most commonly experienced type of aura (30.2% of the total seizures) in Group 1. Undefined sensation and palpitation (37.5% of total seizures) were observed in Group 2. In 75 seizures (31%) in Group 1 and 33 seizures (41.3%) in Group 2, there was no aura.

Table 3
Risk factors

	Group 1 ($n = 35$)	Group 2 ($n = 13$)
(1) Febrile convulsion	12 (34.3%)	1 (7.7%)
(2) Head trauma	4 (11.4%)	2 (15.4%)
(3) Anoxic birth	1 (2.9%)	—
(4) Meningitis	1 (2.9%)	—
(1) + (2)	3 (8.5%)	—
(1) + (3)	2 (5.7%)	—
(1) + (2) + (3)	1 (2.9%)	—
No risk factor	11 (31.4%)	10 (76.9%)

Table 4
Types of auras experienced by patients

	Group 1 ($n = 202$)	Group 2 ($n = 80$)
Epigastric sensation	61 (30.2%)	5 (6.3%)
Undefined sensation/palpitation	47 (23.3%)	30 (37.5%)
Fear	13 (6.4%)	—
Olfactory hallucination	6 (3%)	—
Abdominal pain	—	10 (12.5%)
Visual hallucination	—	2 (2.5%)
No aura	75 (37.1%)	33 (41.3%)

Table 5
Ictal behavioral characteristics observed during video/EEG monitoring in the two groups

	Group 1 ($n = 202$)	Group 2 ($n = 80$)
Behavioral arrest	62 (30.7%)	51 (63.8%)
Oral automatisms	68 (33.7%)	45 (56.3%)
Ipsilateral hand automatisms	156 (77.2%)	47 (58.8%)
Bilateral hand automatisms	17 (8.4%)	23 (28.8%)
Contralateral dystonic posturing	120 (59.4%)	13 (16.3%)
Bilateral dystonic posturing	22 (10.9%)	—
Ipsilateral hand automatisms in the presence of contralateral dystonic posturing	120 (59.4%)	13 (16.3%)
Ipsilateral nonversive head deviation	68 (33.7%)	35 (43.8%)
Contralateral nonversive head deviation	5 (2.5%)	—
Contralateral versive head deviation	35 (17.3%)	6 (7.5%)
Ipsilateral versive head deviation	1 (0.5%)	—
Vocalization	21 (10.4%)	13 (16.3%)
Ictal speech	18 (8.9%)	—
Leg automatism	24 (11.9%)	13 (16.3%)
Automatisms with preserved responsiveness	9 (4.5%)	—
Aggression	16 (7.9%)	11 (13.8%)
Secondary generalization	52 (25.7%)	10 (12.5%)
Postictal nose wiping	15 (7.4%)	6 (7.6%)
Ictal crying	4 (2%)	—

All ictal behavioral patterns observed during video/EEG monitoring of the two groups are summarized in Table 5. Ipsilateral hand automatisms ($P < 0.05$), contralateral dystonic posturing ($P < 0.05$), ipsilateral automatisms in the presence of contralateral dystonic posturing ($P < 0.001$), and contralateral versive head–eye deviation were more common in Group 1, whereas behavioral arrest ($P < 0.05$), ictal aggression ($P < 0.05$), and bilateral hand, leg, and oral automatisms ($P < 0.05$) were more common in Group 2.

There were no statistically significant differences between the two groups with respect to vocalization, ipsilateral nonversive head–eye deviation, postictal nose wiping, and leg automatisms ($P > 0.05$). Statistical analysis could not be performed in cases of ictal speech, ictal crying, automatisms with preserved responsiveness, and bilateral upper extremity dystonic posturing because of the limited number of seizures. As a result, ipsilateral automatisms in the presence of contralateral dystonic posturing were much more specific to the hippocampal sclerosis group.

4. Discussion

Several ictal behavioral characteristics have been defined in TLE. Although the value of these characteristics for lateralization is not specific, some of them are pecu-

liar to TLE [1,2]. Ipsilateral hand automatisms in the presence of contralateral dystonic posturing have been observed especially in complex partial seizures originating from the temporal lobe [2]. Our results are in accord with previous findings in which ipsilateral hand automatisms in the presence of contralateral dystonic posturing were an important lateralizing and localizing sign. This was much more specific to the hippocampal sclerosis group in our study.

Ipsilateral automatisms in the presence of contralateral dystonic posturing have not been studied with respect to pathology previous to our research. Our patients with hippocampal sclerosis also showed ipsilateral hand automatisms, contralateral dystonic posturing, contralateral versive head–eye deviation, and secondary generalization during seizures. On the other hand, bilateral hand, oral, and leg automatisms were seen especially in Group 2.

Kernan et al. studied 92 secondary generalized seizures in 29 patients with a specific lateralized focus using video/EEG telemetry. They found that 83 of 92 secondary generalized complex partial seizures had lateralized and sustained head deviations. The side of head deviation was contralateral in more than 90% of the seizures. They also found that nonforced head deviation was not of lateralizing significance [9]. In our study, ipsilateral nonversive head–eye deviation was observed in both groups, but was not specific to pathological diagnosis. Contralateral head deviation and secondary generalization were more common in the hippocampal sclerosis group.

Preserved responsiveness is defined as the ability of a patient to respond verbally or to follow motor commands throughout the seizure. Ebner et al. reported that seven patients had preserved responsiveness in the presence of automatisms [15]. They investigated 15 seizures. Ictal EEG was localized over the right temporal area in 9 seizures and over the right hemisphere in 5 seizures, and it was not localized in 1 seizure. They concluded that automatisms with preserved responsiveness are lateralized to the right side in TLE [15]. We have observed automatisms with preserved responsiveness in 9 seizures (two patients), all of which originated in the right temporal area (nondominant hemisphere).

Gabr et al reviewed video/EEG of 100 complex partial seizures in 35 patients who underwent ATL. They observed ictal speech manifestations in 79 seizures. They classified these speech manifestations into three groups as vocalization, normal speech, and abnormal speech. Normal speech occurred ictally in 34.2% of the patients, and seizures in 83% of these originated in the nondominant hemisphere. However, they observed normal speech in 12.5% of the patients with seizures originating in the dominant hemisphere [11]. We have seen ictal speech in 18 seizures, all of which originated in the right temporal area.

Ictal aggression and ictal crying are rarely observed as clinical behavioral patterns in epilepsy [18,19]. Ictal aggression was investigated by Delgado-Escueta et al., who concluded that its role in lateralization was limited [18]. In our study, ictal aggression was more common in Group 2. However, there was no statistically significant difference in the occurrence of ictal aggression when we excluded the patient with neocortical gliosis. Ictal crying has usually been reported in cases of complex partial seizures originating in the nondominant hemisphere [19–21]. We observed ictal crying in four seizures in one patient. Statistical analysis could not be performed in cases of ictal speech, ictal crying, and automatisms with preserved responsiveness because of the limited number of seizures.

Postictal nose wiping was investigated in 87 patients by Hirsh et al., who reported that postictal nose wiping was more common in unilateral TLE, particularly in mesial temporal lobe epilepsy. In that study, postictal nose wiping with 60 s of electrographic seizure offset occurred in 60% of patients and 43% of seizures [3]. In our study, postictal nose wiping was not specific to the pathology. Postictal nose wiping occurred in 21 seizures (6 patients). This behavioral sign was ipsilateral to the epileptic zone in 18 of the seizures (5 patients) and contralateral to the epileptic zone in 3 seizures (1 patient). Statistically there was no significant difference in the occurrence of postictal nose wiping between the two groups.

Saygi et al. observed the sequential ictal behavioral characteristics of seizures in patients with temporal lobe tumors and patients with hippocampal sclerosis. They analyzed 145 seizures in 33 patients with hippocampal sclerosis and 79 seizures in 22 patients with tumors. Ipsilateral hand automatisms were more common in the first 60 s in the hippocampal sclerosis group, and contralateral head turning was observed in the first 5 s only in the tumor group [1]. We have not evaluated sequential symptomatology in our research, because our aim in this study was to define the ictal behavioral characteristics in mesial temporal lobe onset seizures that were specific to the pathological diagnosis.

AH was added to ATL and lesionectomy in Group 2 patients because all the lesions of Group 2 patients were in the mesial temporal region. In patients with neocortical gliosis the AH was done because of the colorless temporal lobe including mesial temporal structures. Patients with lesions of the temporal lobe who did not have hippocampal atrophy and those patients who had a more extensive resection with mesial temporal structure had a more favorable outcome [22].

Mesial temporal lobe epilepsy (MTLE) patients were more likely to have a prior history of febrile convulsions, CNS infections, perinatal complications, and head injury [23]. In our study, these risk factors were more common in Group 1 patients. Ictal behavior characteristics

have been studied in mesial (MTLE) and neocortical (NTLE) temporal lobe epilepsy. NTLE seizures lacked the features commonly exhibited in MTLE including automatisms, contralateral dystonia, head movements, body shifting, hyperventilation, and postictal cough [23]. Facial grimacing and twitching occurred earlier in NTLE seizures [24]. In our study, ipsilateral hand automatisms, contralateral dystonic posturing, ipsilateral hand automatisms in the presence of contralateral dystonic posturing, and contralateral versive head deviation were more common in the hippocampal sclerosis group (Group 1). Behavioral arrest and bilateral hand, leg, and oral automatisms were more common in Group 2.

The mesial temporal region was determined to be the epileptogenic focus in 40 patients after scalp video/EEG monitoring; the remaining eight patients were reevaluated using subdural strip electrodes, and the focus was located in the mesial temporal region. Although ictal EEG findings were similar for both groups, behavioral characteristics during seizures differed. This is interesting. Primary MTLE that is not secondary to the structural lesion is a clinically distinct entity from secondary MTLE. The organization of seizures may be complex and different patterns exist.

Our study had some limitations. For instance, the number of patients in Group 2, which was a heterogeneous group in terms of pathological diagnosis, was insufficient. We could have defined specific ictal behavioral patterns separately for each pathology if the number of the patients had been larger. Moreover, all lesions were located in the amygdala, parahippocampus, and hippocampus in all patients in Group 2 except for a patient with neocortical gliosis.

Ipsilateral hand automatisms together with contralateral dystonic posturing were statistically more significant in the hippocampal sclerosis group. This finding may be helpful in evaluating patients. However, new studies are necessary to support this hypothesis.

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