Evaluation of partial epilepsy in Iran: role of video-EEG, EEG, and MRI with epilepsy protocol

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Abstract

Background: we evaluated the diagnostic value of Electroencephalography (EEG), video-EEG monitoring (VEM) and Magnetic resonance imaging (MRI) of the brain with epilepsy protocol in patients with complex partial epilepsy.

Methods: Forty-two consecutive patients underwent complete neurological examination, EEG, and MRI with a modified epilepsy protocol. A subset of these patients (n=29) also underwent VEM. Data were presented using descriptive statistics and were analyzed using Chi square and McNemar tests.

Results: Twenty-four women and eighteen men entered the study. The mean (±SD) age for patients, was 25.2(±10.1) and mean (±SD) age at onset was 10.9(±8.1). All patients had abnormal ictal or interictal EEG. Fifteen patients had normal MRI. Temporal lobe involvement was the most common involvement in both EEG (27 patients) and MRI (14 patients). Fifteen patients had normal MRI. Temporal lobe involvement was the most common involvement in both EEG (27 patients) and MRI (14 patients). Interictal EEG was abnormal in 81% of patients which showed epileptiform discharges in about half of the cases. In half of patients who had lateralized finding on MRI, site of the lesion was congruent between MRI and interictal EEG. Thirty-six patients had symptoms suggesting a specific lobe, of which interictal EEG was able to show the concordant lobe in 22 (61%) patients. McNemar test showed superiority of EEG over MRI in correct diagnosis of the involved lobe based on the clinical manifestations (P<0.01).

Conclusion: In our setting, both ictal and interictal EEG perform better than MRI in evaluating complex partial epilepsy. In addition, combination of these tools may increase the yield of showing abnormality to near 100% in patients with complex partial epilepsy.

Introduction

Partial epilepsy is the most common type of epilepsy with a prevalence of 100 to 190 per 100000 in developing countries. According to International League against Epilepsy, partial seizure is “a seizure whose initial semiology indicates, or is consistent with initial activation of only part of one cerebral hemisphere” [1]. Temporal lobe and limbic system are the most common affected parts and mesial temporal sclerosis is the most predominant pathology [2,3].

About 30% of patients with epilepsy do not respond to antiepileptic medications [4-6]. There is no consensus regarding the definition of intractable epilepsy; nevertheless, failure to respond to two or three antiepileptic drugs should prompt a referral to a tertiary epilepsy center. Significantly, around 2% of patients with epilepsy may need surgical management [7,8]. The assessment of a patient for surgery depends on localization...
of the cortical focus of seizure onset and mapping functions of the brain that may be influenced by surgical removal. Therefore, the key aim is to find the epileptogenic zone, the region of the cortex that is necessary for the generation of epileptic discharges [8].

Electroencephalography (EEG) is one of the most useful tests in the evaluation of partial epilepsy. In EEG, three kinds of discharges namely interictal epileptiform discharges (IEDs), periodic lateralized epileptiform discharges (PLEDs) and generalized periodic epileptiform discharges (GPEDs) are considered significant for diagnosing epilepsy [9].

Magnetic resonance imaging (MRI) of brain with epilepsy protocol is considered one of the most sensitive imaging modality for identifying the site of brain pathology in patients with partial epilepsy [10]. MRI is also the best neuroimaging method for diagnosing lesions that are responsive to surgical treatment [11]. Those patients who have localized and unilateral lesions on MRI have the best surgical prognosis. Both EEG and MRI are used to identify the site of the lesion and provide a guide for surgery [12]. However, in some cases, there is discordance in site of the lesion between MRI and EEG. In a study, three groups of patients underwent surgery. In the first group, EEG and MRI showed the same pathology; in the second and third group, the sites of the lesions were incongruent. Patients in the second group underwent surgery based on abnormal EEG and patients in the third group underwent operation based on abnormal MRI. Outcomes were worst in the second group whose lesions were resected based on abnormal EEG [13]. Both EEG and MRI are valuable tools for predicting the outcome of surgery; if one of either EEG or MRI findings is concordant with the site of the lesion, the possibility of good outcome is 60% to 65%. When findings of both EEG and MRI are congruent with the site of the lesion, the possibility raises to 95% [12]. Both EEG and MRI are parts of routine management in patients with partial epilepsy and as mentioned above, both of them are important in predicting the outcome of the patients after epilepsy surgery. There is a lack of evidence regarding the diagnostic utility of these methods in Iran. In the present paper, we seek to evaluate the value of ictal and interictal EEG and MRI with epilepsy protocol and their concordance with each other; in addition, with clinical symptoms in the Iranian patients with partial epilepsy referred to our center.

Materials and Methods
In 2009, 42 consecutive patients who were referred to the neurology department of Imam Khomeini hospital, Tehran, and had reliable history of partial seizure or a well-informed relative or a physician who had witnessed their attacks entered the study. Reliable history of seizure was considered as gold standard for diagnosing partial epilepsy in the present study. We included all patients with partial epilepsy, who were under 65 years of age, without history of any other neurological disorders and brain surgery. Because most of our patients were dependent to antiepileptic drugs and also most of these drugs do not affect the EEG, particularly if used chronically, most of the patients included were taking antiepileptic drugs. However, during the video-EEG monitoring (VEM) we tried to minimize the dose of the medication as much as possible. All patients underwent careful history (including neurological development) and physical examination as well as EEG and MRI. Some patients also underwent video EEG and VEM. Symptomatology was assessed based on the measures presented in Table 1 [14].

EEG: ScalpEEG was recorded on a 16-channel machine (Nihon Kohden Co., Tokyo, Japan) based on guidelines of American Clinical Neurophysiology Society available at www.acns.org. EEG Recording was done according to 10/20 system and with and without hyperventilation and light stimulus. A neurologist, who was most expert in the EEG interpretation, analyzed the recorded EEGs. Spikes, sharp-waves and spike-waves were considered epileptiform Abnormalities such as diffuse slowing, amplitude changes, or asymmetrical amplitudes were considered non-epileptiform abnormalities.

VEM: NicoletOneMachine (Viasys Healthcare, Mortara, United States) was used for VEM based on the last published guidelines of American Neurophysiological society available at www.acns.org. After admission in VEM ward, drugs were discontinued as much as possible and a trained VEM nurse was responsible for observing the patient. VEMwas continued until sufficient numbers of

| Table 1. Involved lobe based on symptomatology |
|---------------------------------------------|
| Lobe           | Medial temporal                      | Lateral temporal                          | Frontal                     | Parietal                  | Occipital                  |
| Clinical features | Oroalimentary automatism; contralateral dystonic posturing ipsilateral, nonversive head turning, sentences if right (non-dominant) Epigastric sensation, de ja vu | Auditory hallucination, facial twitching, rhythmic vocalization, vertigo | Abrupt posturing, bizarre gesturing, complex vocalizations contralateral versive head turning (fast) | Sensory aura with march, other features variable | Elementary visual hallucination, versive contralateral head turning (slow), nystagnus, blinking |

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ictal event recorded; however, sometimes because of lack of cooperation of the patient, or failure to record an event after four days or because of unaffordability of the patient, the VEM was discontinued without recording an event. During the attack, the nurse was responsible for checking the patients to assess their consciousness and memory. Whenever the patient felt aura, he could press a button to inform the nurse of the possible onset of the event. All patients who underwent VEM were under 24-hour observation, and all events were recorded simultaneously on EEG and video monitoring.

Medications were administered with the lowest possible dose to allow for occurrence of ictal event. VEM was recorded for 72 to 96 hours. Onset of seizure activity, attenuation or disappearance of previous interictal epileptiform activity and attenuation of previous background activity were used to localize the epileptogenic focus. Video-EEGs was assessed by an expert, and following clinical and electroencephalographic features were taken into consideration: Time of the event, obvious precipitating factor, activity of the patient at the time of occurrence, time and nature of the first clinical change, evolution, duration and end of the clinical event, postictal behavior and time of return to normal activity and functioning, status of the patient at the beginning of the EEG event, first alteration in EEG, first definite rhythmic activity together with its localization, evolution and termination, time of termination and postictal EEG pattern.

MRI: Patients underwent 1.5 Tesla MRI (General Electric Healthcare, United Kingdom) with epilepsy protocol to optimize the images. Sequences comprised of consecutive, thin (<1.5 mm) slices covering the whole brain. Sequences included Standard T1-weighted, T2-weighted fast spin-echo and gradient echo, and fluid-attenuated inversion recovery (FLAIR). All images were obtained in two orthogonal planes. Due to technical limitations, we could not obtain 3D images from our patients.

SPSS version 15.00 (Chicago, USA) was used for analysis. Chi-squared test was used for categorical data and Mann-Whitney U test was used to compare quantitative variables. McNemar test was used for comparison of two diagnostic methods. P-value of less than 0.05 was considered statistically significant.

Results

Twenty-four women and eighteen men entered the study. The mean (±SD) age for patients was 25.2 (±10.1). Mean (±SD) age at onset was 10.9 (±8.1). Twenty-nine patients had VEM but six patients had no clinical events on VEM. All patients with ictal event had either abnormal ictal or interictal EEG. Frequency of lateralized, non-epileptiform abnormalities and epileptiform discharges in interictal EEG were 59.5%, 42.9%, and 38.1% respectively. 73.9% of the patients had lateralized discharges in VEM. Twenty-seven patients had abnormal MRI. A summary of baseline and clinical findings is presented in table 2. Temporal lobe involvement was the most common involvement in both EEG (27 patients) and MRI (14 patients). Most common pathology on MRI was mesial temporal sclerosis (29.6%). A summary of findings in 42 patients are presented in table 3.

Table 2. Summary of data of the patients

| Variable                        | Data                              |
|---------------------------------|----------------------------------|
| Age (mean ± SD)                 | 25.21 (±10.14)                   |
| Age at onset                    | 10.92 (±8.11)                    |
| Gender (mean ± SD)              | 24 female, 18 male               |
| History of Febrile seizure      | 7 yes, 35 no                     |
| Family history                  | 8 Positive, 34 negative           |
| Interictal EEG                  |                                  |
| • Normal                        | 8 (19%)                          |
| • Epileptiform                  | 18 (42.9%)                       |
| • Other abnormalities           | 16 (38.1%)                       |
| Lateralization of interictal EEG|                                  |
| • Lateralized                   | 25 (59.5%)                       |
| • Epileptiform                  | 13 (31%)                         |
| • Other abnormalities           | 12 (28.5%)                       |
| • Generalized                   | 9 (21.4%)                        |
| • Epileptiform                  | 5 (11.9%)                        |
| • Other abnormalities           | 4 (10.5%)                        |
| • Normal                        | 8 (19.1%)                        |
| Ictal EEG (n=29)                |                                  |
| • Lateralized                   | 17 (58.6%)                       |
| • Generalized                   | 6 (20.6%)                        |
| • No event                      | 6 (20.6%)                        |
| MRI laterality                  |                                  |
| • Normal                        | 15 (35.7%)                       |
| • Right-sided                   | 9 (21.4%)                        |
| • Left-sided                    | 13 (31%)                         |
| • Bilateral                     | 5 (11.9%)                        |

MRI and interictal EEG

Interictal EEG was abnormal in 81% of patients and was epileptiform in about half of these cases. There were 15 patients with normal MRI and 22 (52%) with lateralized lesion in MRI. Thirty out of 42 patients had lateralized lesions in either EEG or VEM. Fifteen patients had either normal MRI or interictal EEG. Five additional patients had non-lateralized lesions on MRI. Excluding these patients, in 11 of 22 remaining patients (50%) patients, the site of the lesion is congruent between MRI and interictal EEG. Six additional patients who had discordant interictal EEG and MRI had congruent ictal EEG and MRI findings. In other words, total congruency, between EEG (ictal or interictal) and MRI was seen in 17 out of 42 patients. Excluding normal and non-lateralized MRI, 17 of 22 (77.2%) patients had congruent lesions between EEG and MRI.
Table 3. clinical data of the patients

| Patient number | Gender | Age | Involved lobe based on manifestations | Interictal EEG (abnormalities/involved area) | Ictal EEG | MRI |
|----------------|--------|-----|--------------------------------------|---------------------------------------------|-----------|-----|
| 1              | F      | 35  | Frontal                              | Epileptiform/left fronto-temporal           | No event  | Normal |
| 2              | M      | 24  | Right frontal                       | Epileptiform/bilateral                      | Left side | Normal |
| 3              | M      | 20  | Temporal                             | Epileptiform/bilateral                      | Left side | Normal |
| 4              | M      | 46  | Temporal                             | Non-epileptiform/left temporal              | Bilateral | Ischemic change in the anterior portion of the left frontal lobe. |
| 5              | M      | 30  | Frontal                              | Non-epileptiform/bilateral fronto-centro-temporal | Right fronto-temporal | Right hippocampal sclerosis |
| 6              | F      | 36  | Temporal                             | Non-epileptiform/bilateral fronto-temporal  | Left side | Left hippocampal sclerosis |
| 7              | F      | 20  | Frontal                              | Non-epileptiform/bilateral fronto-temporal  | Bilateral | Normal |
| 8              | F      | 12  | Temporal                             | Non-epileptiform/bilateral fronto-temporal  | No event  | Signal intensity in the right temporal region |
| 9              | M      | 21  | Medial temporal                      | Non-epileptiform/bilateral right fronto-temporal | Bilateral | Vascular malformation in the right temporo – occipital area |
| 10             | M      | 22  | Medial temporal                      | Non-epileptiform/bilateral right parieto-temporal | Right parieto-temporal | 10-mm lesion in medial aspect of the right temporal lobe |
| 11             | F      | 29  | Temporal                             | Non-epileptiform/bilateral left fronto-temporal | No event  | Ischemic changes in right centrum semiovale |
| 12             | F      | 31  | Medial temporal                      | Non-epileptiform/bilateral left fronto-temporal | No event  | Left hippocampal sclerosis |
| 13             | M      | 48  | Medial temporal                      | Epileptiform/ right fronto-temporal         | No event  | Left fronto-temporal |
| 14             | M      | 17  | Frontal                              | Epileptiform/bilateral                      | No event  | Small vessel disease |
| 15             | M      | 41  | Temporal                             | Non-epileptiform/bilateral right fronto-parieto-temporal | No event  | Left frontal lobe malacia with adjacent white matter edema. |
| 16             | M      | 24  | Medial temporal                      | Non-epileptiform/right centro-temporal      | No event  | Normal |
| 17             | F      | 37  | Unknown                              | Epileptiform/bilateral                      | No event  | Normal |
| 18             | F      | 21  | Right medial temporal                | Epileptiform/ right fronto-temporal         | Right side | Focal structural abnormality in the right posterior parietal lobe |
| 19             | F      | 22  | Frontal                              | Epileptiform/ right fronto-temporal         | Left side | Normal |
| 20             | F      | 22  | Medial temporal                      | Epileptiform/ right fronto-temporal         | Left fronto-parieto-temporal | Normal |
| 21             | F      | 21  | Lateral and medial temporal          | Epileptiform/bilateral fronto-cenro-temporal | Right temporal-central | Normal |
| 22             | M      | 35  | Unknown                              | Epileptiform/bilateral                      | Left temporal | Dandy Walker variant |
| 23             | F      | 20  | Unknown                              | Epileptiform/normal                         | Left side | Normal |
| 24             | F      | 12  | Unknown                              | Non-epileptiform/bilateral right parieto-temporal | No event  | Pachygyria and schizencephaly on right side |
| 25             | F      | 20  | Left frontal                        | Non-epileptiform/bilateral left frontotemporal | Left fronto-temporal | Mass with Low signal on T1 and high signal on T2 located deeply in the left medial temporal |
| 26             | M      | 23  | Frontal                              | Epileptiform/normal                         | Generalized | Normal |
| 27             | M      | 46  | Temporal                             | Non-epileptiform/bilateral left fronto-parieto-temporal | Left fronto-temporal | Normal |
| 28             | F      | 20  | Left lateral temporal                | Non-epileptiform/bilateral right parieto-temporal | Epileptiform/ left fronto-parieto-temporal | NA |
| 29             | F      | 7   | Medical temporal                     | Epileptiform/normal                         | NA | Left temporal lesion |
| 30             | M      | 17  | Medial temporal                      | Normal                                    | Left temporal | Left temporal lesion (possible mesial temporal sclerosis) |
| 31             | F      | 36  | Medial temporal                      | Normal                                    | Left temporal | Mass in the left hippocampal head and amygdala |
| 32             | M      | 19  | Unknown                              | Epileptiform/ left fronto-central            | NA | Normal |
| 33             | F      | 36  | Bilateral temporal                   | Epileptiform/left temporal                 | NA | Left hippocampal sclerosis |
| 34             | F      | 25  | Lateral temporal                     | Epileptiform/bilateral                      | NA | Normal |
| 35             | M      | 4   | Medial temporal                      | Epileptiform/bilateral                      | NA | Small arachnoid cyst of left temporal lobe |
| 36             | F      | 21  | Medial temporal                      | Epileptiform/ left fronto-temporal          | NA | Left frontal lobe lesion |
| 37             | F      | 25  | Unknown                              | Epileptiform/bilateral                      | NA | Multiple demyelinating plaques |
| 38             | F      | 15  | Temporal and occipital               | Epileptiform/ right temporal                | NA | Widening of right silvian fissure (possible mesial temporal sclerosis) |
| 39             | F      | 22  | Medial temporal                      | Epileptiform/bilateral                      | NA | Left temporal lesion |
| 40             | F      | 15  | Left frontal                         | Epileptiform/normal                         | NA | Normal |
| 41             | F      | 30  | Lateral temporal                     | Epileptiform/ right temporal                | NA | Right temporal atrophy with hydrocephaly |
| 42             | M      | 32  | Medial temporal                      | Non-epileptiform/bilateral                  | NA | Normal |
Fifteen patients had normal MRI, and only four of these patients had normal EEG. Four of these patients had lateralized findings on EEG. Twenty-five patients had lateralized abnormalities on interictal EEG, of which eighteen patients had focal abnormalities on MRI, five had normal MRI and two had diffuse MRI abnormalities. From 17 patients who had non-lateralized interictal EEG, 13 also had non-lateralized MRI findings while four had lateralized findings. Patients with normal MRI had significantly higher percentage of generalized EEG abnormality than those with abnormal MRI (54% vs. 13% P=0.01).

Only one patient (2.5%) had left temporal sclerosis on MRI in conjunction with non-epileptiform abnormalities in the right temporal lobe in EEG which thought to be related to kindling phenomenon. Of the 11 patients with normal MRI and abnormal EEG, 8 patients had bilateral findings, and three had lateralized findings; all of these lateralized findings were recorded from more than one lobe in EEG.

When we compared the capability of MRI and interictal EEG in the evaluation the focus of partial epilepsy using McNemar test, no significant difference was found (P=0.1). The results were in favor of EEG when we compared MRI findings with combined ictal and interictal EEG findings (P=0.01).

**MRI and ictal EEG**

58.6% of patients had lateralized discharges on ictal EEG. 73% of total 23 patients with event on VEM eight patients (34.7%) had congruency between ictal EEG and MRI findings. From 11 patients with normal MRI, only 2 had ictal EEG with no event and nine others had either generalized (three patients) or lateralized EEG findings.

Using McNemar test ictal EEG performed better than MRI in the diagnosis of abnormality in partial epilepsy (P<0.01).

Ictal and interictal EEG: All patients who had both interictal and ictal EEG recording had abnormalities in at least one of the EEG recordings (either ictal or interictal).

EEG, MRI, and clinical symptoms: Thirty-six patients had symptoms suggesting a specific lobe, of which interictal EEG was able to show the concordant lobe in 22 (61%) patients. Five patients with possible frontal lobe symptoms had non-specific interictal EEG findings of which only one patient showed involved lobe in both ictal EEG and MRI, and four others had nonspecific or normal MRI and interictal EEG findings. 27 patients with temporal lobe symptoms, 9 patients did not show evidences of temporal lobe involvement in interictal EEG. Together, EEG (ictal and interictal), and MRI were congruent with involved lobe (based on symptomatology) in 29 (80%) of 36 patients.

Of 27 patients with temporal lobe symptoms, only 12 (44%) had identifiable lesions of the temporal lobe in MRI. Of nine patients with frontal lobe symptoms, only one patient had a lesion of frontal lobe on MRI.

McNemar test showed superiority of EEG (ictal and interictal) over MRI in the correct diagnosis of the involved lobe based on the clinical manifestations (P<0.01).

**Discussion**

The present study aimed to compare two routinely used diagnostic methods (EEG and MRI with epilepsy protocol) in Iranian patients with complex partial epilepsy. Our results showed superiority of EEG over MRI in terms of abnormal findings as well as focus of the lesion. However, we also showed that these modalities are complementary in finding the focus of epilepsy.

In our study, 81% of the patients showed abnormal findings in interictal EEG. However, epileptiform discharges in interictal EEG were observed in less than half of the patients which was similar to other studies. Several authors have reported a frequency of 29 to 55% of positive interictal EEG in patients with partial epilepsy [9]. This proportion may increase in some circumstances; for example, Cascino et al. reported 159 patients with refractory temporal lobe epilepsy of which 129 (81%) had epileptiform abnormalities [15]; difference with their study can be explained in two ways: first, they recorded interictal EEG for two hours while period of recording was 45 minutes in our study; second, their study sample consisted of most intractable cases of epilepsy which is usually accompanied by more abnormal EEGs.

Frequencies of lateralized finding in interictal EEG, MRI and ictal EEG (excluding the normal ictal EEGs) were 59%, 52%, and 73.9% respectively. Several authors reached at different results. In a study of 184 patients with temporal lobe epilepsy frequencies of lateralized findings on interictal EEG, ictal EEG and MRI were 62%, 63.5% and 60.9% respectively [16]. In a study on 55 patients with complex partial epilepsy, Marks and colleagues showed lateralized abnormality in 82% and 65% of ictal EEG recordings and MRI of the brain respectively [17]. Serles et al showed a frequency of 49% of lateralized findings in interictal EEG in 59 patients with temporal lobe epilepsy [18]. Some authors however showed lateralization rate as high as 78% and 97% using MRI [19,20]. These differences can be explained in several ways. First, we did not use some electrodes such T1 and T2 & sphenoidal electrodes for recording EEG due to technical limitations. Second, period of recording in our study was less than other studies. Due to technical limitations, we were unable to obtain all MRI sequences required for an epilepsy protocol. In addition, criteria for excluding and including patients in the mentioned studies were different from ours; some studies had only included patients with temporal lobe epilepsy while others only included patients who required surgery and had pathology results.

MRI in our study showed abnormality in 64% of the patients and revealed possible epileptogenic foci in 50% of
the patients. Usual MRI protocol may not be suitable for diagnosing focus of the seizure, as several studies have found that frequency of abnormal standard MRI is less than 50%. For example Maillard et al [21] found a frequency of 33% for brain pathologies in patients with temporal lobe epilepsy using routine MRI. In one study, Von Oertzen et al compared standard MRI with MRI using an epilepsy protocol in 123 patients referred to an epilepsy clinic for surgery evaluation [22]. A focus was found in 39% and 91% of patients respectively showing an obvious superiority of the MRI with epilepsy protocol. Thus, capability of MRI with our modified epilepsy protocol in revealing the abnormality of the brain is something between that of standard MRI and MRI with a dedicated epilepsy protocol. One of the reasons for high yield of MRI with epilepsy protocol is using oblique coronal images; this minimizes partial volume effect which may obscure hippocampal sclerosis as the most common MRI abnormality in patients with complex partial seizure. In a study by Brooks et al [23], using 1.5 Tesla standard MRI, 79% of the patients with pathologically proved mesial temporal sclerosis showed nonspecific or normal findings. Nevertheless, MRI could diagnose tumoral lesions in 14 out of 15 patients with complex partial epilepsy. In another study, Heinz et al found that standard MRI was capable of diagnosis of abnormalities in 67% of all patients with temporal lobe epilepsy and about half of the patients with possible mesial temporal lobe sclerosis [24]. Inability to detect mesial temporal sclerosis was not the only reason for low yield of MRI in the present study since only four out of fifteen patients with normal MRI showed evidences of symptoms related to medial temporal lobe epilepsy. Higher technologies of MRI such as diffusion tensor imaging, MRI with higher field strength (such as 3-Tesla MRI), 3D imaging, and T2-mapping can improve recognition of epileptogenic lesions[25-29]. Nevertheless most of these modalities are not available in developing countries.

Congruent findings were seen in half of the patients regarding MRI and ictal EEG findings. When we considered interictal EEG, six additional patients showed congruency between EEG and MRI. Seventy-two percent of patients with lateralized lesions on MRI had congruency between EEG and MRI. Other studies obtained similar findings. In a paper by Gilliam et al [30], 61% of patients with mesial temporal lobe epilepsy had congruent lateralized findings on interictal EEG and MRI. Higher concordance in their study may be related to their selection criteria. They selected patients with mesial temporal lobe epilepsy who underwent surgery, while patients in our study had several types of complex partial seizures. In another study by Serles et al, a 54% concordance was observed between MRI and interictal EEG in patients with temporal lobe epilepsy [18]. Cascino et al evaluated 159 patients with temporal lobe epilepsy, they found a 61% concordance between side of the lesion in EEG and MRI [15]. We used semiology as a gold standard in our study, because of unavailability of pathology results. Marks and Laxer showed that many manifestations of patients with temporal lobe epilepsy may be of value in lateralizing the site of the lesion [17]. Serles et al [18] found that seizure semiology could lateralize 78% of the patients. Regarding symptomatology, more than half of the patients in our study showed concordance between interictal EEG and focus of the lesion suggested by symptoms. This concordance was increased when we took MRI and ictal EEG results into account. This is more than findings of Serles et al [18]. They found a congruency of 57% between seizure semiology and MRI, and a congruency of 45% between ictal surface EEG and MRI.

Our study had several strengths: we selected a diverse group of patients with partial epilepsy and this makes our study more generalizable. This study was the first to our knowledge, which evaluated Iranian patients with complex partial epilepsy using three different diagnostic modalities. Besides, MRI studies were done using epilepsy protocol with some modifications. This lead to better MRI performance in our study compared with other studies which used standard MRI protocols.

Present study had also some limitations. Due to technical limitations EEG and MRI with epilepsy protocol were performed with some limitations. Nevertheless, differences between results of our study and findings of others were small. We used the semiology rather than pathology as a gold standard for comparison, although this may have some pitfalls, semiology is considered a relatively reliable tool in diagnosis of the epilepsy. Nevertheless, it may be inappropriate to make the clinical judgment and classification solely on the basis of semiology. This study was done in a tertiary referral center with high expertise on interpretation of MRI and EEG. Therefore the results may not be generalizable to other settings in this regard.

To the best of our knowledge, this is the first study which addressed evaluation of complex partial epilepsy in our country. We demonstrated that both ictal and interictal EEG perform better than MRI in several aspects of evaluating complex partial epilepsy. We also showed that our limited epilepsy protocol performs better than standard MRI but still is far from the dedicated epilepsy protocol used in epilepsy centers in developed countries. Finally we found that a combination of these tools may increase the yield of the showing abnormality to near 100% in patients with complex partial epilepsy.
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