

An Investigation into the Correlation of Scalp Electrophysiological Findings with Preoperative Clinical and Imaging Findings in Patients with Focal Cortical Dysplasia

Zahide MAIL GURKAN¹, Ozge KAPAR², Seher Naz YENI³, Bilge BILGIC², Candan GURSES⁴

¹Istanbul University, Istanbul Faculty of Medicine, Departments of Neurology and Clinical Neurophysiology, Istanbul, Turkey

²Istanbul University, Istanbul Faculty of Medicine, Department of Pathology, Istanbul, Turkey

³Istanbul University-Cerrahpaşa, School of Medicine, Department of Neurology, Istanbul, Turkey

⁴Koc University, Department of Neurology, Istanbul, Turkey

Corresponding author: Zahide MAIL GURKAN ✉ zahidemailgurkan@gmail.com

ABSTRACT

AIM: To evaluate the patients who had epilepsy surgery and pathologically proven focal cortical dysplasia (FCD) in order to further classify and discuss electroencephalography (EEG) findings in different pathological subtypes.

MATERIAL and METHODS: This study included 19 refractory epilepsy patients who underwent surgery between 1999 and 2017 in the Istanbul Faculty of Medicine. Demographic data, preoperative examinations, scalp video EEGs, and postoperative outcomes were evaluated retrospectively.

RESULTS: In this study, 36.8% of the patients were female. The mean age was 21.89 ± 14.64 years. Rhythmic epileptiform discharges (RED) were observed in 31.6%. 37.5% of the patients with isolated intermittent spike/sharp waves were type I, 50% were type II, and 12.5% were type III. 100% of the patients with normal background activity were FCD type II. 67% of the patients with asymmetric slowing were FCD type I, 22% was FCD type II, 11% were FCD type III. 71% of the patients with symmetrical slowing were FCD type I, 29% were FCD type II. One patient had Frontal Intermittent Rhythmic Activity, one patient had Electrical Status Epilepticus in Slow Sleep, two patients had “burst suppression,” and one patient had a “switch of” sign. The frequency of focal epileptogenic activity was higher when there was an FCD lesion on magnetic resonance imaging.

CONCLUSION: The findings obtained in this study did not reveal any distinctive electrophysiological features in FCD and subgroups of FCD. The incidence of REDs did not differ between types. The frequency of isolated intermittent sharp/spike waves was higher in type II than I. Intermittent and continuous EEG slowing was more commonly seen among FCD Type I patients.

KEYWORDS: Focal cortical dysplasia, Epilepsy surgery, Scalp EEG, Pathological subtypes, Rhythmic epileptiform discharges

INTRODUCTION

Focal cortical dysplasia (FCD), first described by Taylor et al., is common etiology of epilepsy in patients with refractory epilepsy and is classified as malformations of cortical development (21,26). FCD is the most common etiology in childhood epilepsy and the third most common in adults worldwide. Prevalence of FCD is 20-25% in refractory

epilepsy (12,19,25) and 50% to 65% of FCD patients are rendered seizure-free after surgery (2). The prognosis after surgery may depend on the complete resection of the FCD and this is in accordance with the visibility of FCD on cranial magnetic resonance imaging (MRI) (27). Despite advanced techniques, FCD cannot be easily identified by cranial MRI (3). Electrophysiological investigations gain importance in

defining epileptogenic zones in FCD where imaging findings are insufficient. Scalp electroencephalographic findings associated with FCD have been described in previous studies. To our knowledge, there is no study classifying electrophysiological findings of the radiological features and subtypes in pathology. In this study, we aim to discuss the findings in scalp electroencephalography (EEG) of pathologically proven subtypes of FCD and their contribution to localization of the epileptogenic zone and diagnosis.

We included MRI, positron emission tomography (PET), and single positron emission tomography (SPECT) findings and surgical outcomes in the discussion.

■ MATERIAL and METHODS

Patients

In this study, 35 patients who were under follow-up in the Departments of Neurology and Neurosurgery at Istanbul Faculty of Medicine between 1999 and 2017 and underwent surgery because of refractory epilepsy due to FCD were evaluated. This is a retrospective observational study conducted in accordance with the ethical principles stated in the "Declaration of Helsinki" and was approved by the Istanbul Faculty of Medicine Ethics Committee on 24th November 2017 (No: 2017/1363).

Nineteen patients with a confirmed FCD by pathology and complete scalp EEG recordings available were included in the present study. Patients with incomplete and inadequate samples of EEG recordings in the archives were excluded. The socio-demographic characteristics of the patients, age, gender, age at seizure onset, epilepsy duration, and age at surgery were recorded. The epileptogenic zone and the area removed by the surgery were evaluated. The specimens obtained from patients during surgery were re-evaluated pathologically to confirm the diagnosis and identify the subgroups of FCD. The International League Against Epilepsy (ILAE) classification of systems for FCDs was used (4). The seizure semiologies of the patients were reviewed. Cranial MRIs of all patients were examined, markers of FCD were recorded, and accompanying additional pathologies were identified.

Electrophysiological Evaluation

Scalp EEG and simultaneous video recordings during wakefulness and sleep were analyzed retrospectively by two neurologists (CG, ZMG). Scalp EEG electrodes were placed according to the International 10–20 system. Recordings were evaluated in the referential and bipolar montage. The following EEG findings were identified; rhythmic epileptiform discharges (RED) and isolated intermittent spike/sharp waves (focal, regional, bilateral) as interictal epileptic discharges, continuous symmetrical and asymmetrical slowing as non-epileptiform discharges, Frontal Intermittent Rhythmic Delta Activity (FIRDA), Continuous Spikes, Waves During Slow Sleep (CSWS), burst suppression pattern and "Switch off" finding. Ictal activities were evaluated and were grouped according to whether they can lateralize or localize the epileptogenic zone.

Rhythmic epileptiform discharges are defined as stereotyped, rhythmic sequences of repetitive, sharp waves or spikes lasting >1 second. The relationship between FCD subtypes and scalp EEG findings was examined, and the effect of sleep was evaluated. To define the localization value of each EEG pattern, REDs, intermittent spike/sharp waves, and ictal activities were compared with the anatomical location of the structural cortical lesion on imaging.

Preoperative Examinations

Cranial MRI, PET, SPECT, and neuropsychological tests (NPT) performed before the operation were evaluated. All patients had cranial MRIs. MRI findings were grouped as normal MRI, only focal cortical dysplasia (OFCD), dual lesions (FCD and an additional lesion) and, lesions other than FCD. Dual lesions and lesions other than FCD were determined as contralateral hippocampal sclerosis, polymicrogyria, hypoplasia of corpus callosum, and sequelae tissue loss. PET results of 17 patients, ictal SPECT results of five patients, and NPT results of 12 patients were obtained. Cranial MRI findings, PET, SPECT findings, and the correlation between these and EEG findings were investigated. Their sensitivity in determining the epileptogenic zone was evaluated.

Surgery and Outcome

The process of determining the epileptogenic zone and the surgical approach to the lesion were examined. Postoperative outcome was evaluated using Engel's classification (9). Factors affecting outcomes were discussed.

Pathological Examination

All Hematoxylin-Eosin stained samples were retrospectively re-examined (BB, OK), and morphological diagnoses were determined according to the consensus recommended by the ILAE diagnostic methods commission for FCD (4). Neuronal nuclear antigen, Neurofilament-H antibodies were applied to some patients at the diagnostic stage.

Statistics

NCSS (Number Cruncher Statistical System) 2007 (Kaysville, Utah, USA) program was used for statistical analysis. While evaluating the study data, besides descriptive statistical methods (Average, Standard Deviation, Median, Frequency, Ratio, Minimum, Maximum), the Mann-Whitney U test was used to compare two groups of variables that did not show the normal distribution in the comparison of quantitative data. Fisher-Freeman-Halton test and Fisher's exact test were used to compare qualitative data. The patient group was divided into two groups as those with OFCD (n=9) according to their MRI scans and patients with normal MRI, dual lesions, or lesions other than FCD (n=10). Logistic regression analysis was used to determine the relationship between the presence of OFCD and the presence of rhythmic activity, isolated intermittent spike/sharp waves, and ictal activities. However, linear regression analysis was used to evaluate the relationship between the presence of OFCD and scalp interictal activity. Significance was assessed at least $p < 0.05$.

RESULTS

Clinical Data

In this study, 19 patients who were operated on for refractory epilepsy and who had FCD in pathological examinations of surgical materials were included. Seven (36.8%) of the patients were female and 12 (63.2%) were male. The ages of the patients ranged between four and 47, and the mean age was 21.89 ± 14.64 years. Age at onset of seizure ranged from one day to 13 years, with an average of 4.00 ± 4.54 years; duration of epilepsy varied between 0.6 and 46 years, with an average of 13.58 ± 12.36 years; the age at surgery ranged from 0.6 to 46 years, with an average of 17.46 ± 14.67 years (Table I). Seven (36.8%) of the patients had FCD Type Ia, four (21.1%) of patients Type Ib, 4 (21.1%) of patients Type IIa, 3 (15.7%) of patients Type IIb and 1 (5.3%) of patients Type IIIa. Type I was determined in 57.9% ($n=11$), type II in 36.8% ($n=7$), and type III in 5.3% of them ($n=1$) (Table II) (Figure 1).

There was no statistically significant difference between the age, the age of onset of seizures, duration of epilepsy, and the age of surgery of the patients with FCD type I and type II ($p > 0.05$). Among the patients with FCD type II, the ratio of female patients was statistically significantly higher than with FCD type I ($p=0.013$; $p<0.05$).

In the scalp EEGs, REDs were detected in 31.6% ($n=6$) of the patients. Among them, three patients had FCD Type I, and the other three patients had FCD Type II. Rhythmic epileptiform discharges in one of these patients were focal and localized to the lesion. Rhythmic discharges were common in one hemisphere in three of the patients, in both hemispheres in one patient, and the posteriors of both hemispheres in the other patient. When interictal activities were examined in scalp EEG, focal spikes were observed in eight patients, regional spikes in four patients, and bilateral spikes in seven patients.

Table I: Demographic Features

		n (%)
Age	Min-Max (Median)	4-47 (22)
	Average \pm Ss	21.89 \pm 14.64
Gender	Female	7 (36.8)
	Male	12 (63.2)
Age at epilepsy onset	Min-Max (Median)	1 day -13 years (1)
	Average \pm Ss	4.00 \pm 4.54
Duration of epilepsy	Min-Max (Median)	0.6-46 (15)
	Average \pm Ss	13.58 \pm 12.36
Age at surgery	Min-Max (Median)	0.6-46 (18)
	Average \pm Ss	17.46 \pm 14.67
Lateralization	Right	13 (68.4)
	Left	6 (31.6)
Localization	Frontal	8 (42.1)
	Parietal	5 (26.3)
	Temporal	4 (21.1)
	Occipital	2 (10.5)
Focal cortical dysplasia	I a	7 (36.8)
	I b	4 (21.1)
	II a	4 (21.1)
	II b	3 (15.7)
	III a	1 (5.3)
	Type I	11 (57.9)
	Type II	7 (36.8)
	Type III	1 (5.3)

Table II: Pathological Findings

Patient	Type	Pathological finding
Patient 1	Ia	Abnormal radial lamination - abundant microcolumnar organization (by more than 8 neurons aligned in vertical direction)
Patient 2	IIa	Abnormal lamination and the presence of dysmorphic neuron *
Patient 3	IIb	Abnormal lamination and the presence of balloon cells **
Patient 4	IIb	Abnormal lamination and the presence of balloon cells **
Patient 5	IIb	Abnormal lamination and the presence of balloon cells **
Patient 6	IIa	Abnormal lamination and the presence of dysmorphic neuron *
Patient 7	IIIa	Abnormal radial lamination and pyramidal cell loss in CA1 and CA4 regions of hippocampus
Patient 8	IIb	Abnormal lamination and the presence of balloon cells **
Patient 9	Ib	Abnormal tangential lamination- loss of 6-layer histological structure of the cortex
Patient 10	Ia	Abnormal radial lamination - abundant microcolumnar organization (by more than 8 neurons aligned in vertical direction)
Patient 11	Ia	Abnormal radial lamination - abundant microcolumnar organization (by more than 8 neurons aligned in vertical direction)
Patient 12	Ia	Abnormal radial lamination - abundant microcolumnar organization (by more than 8 neurons aligned in vertical direction)
Patient 13	Ib	Abnormal tangential lamination
Patient 14	Ia	Abnormal radial lamination - abundant microcolumnar organization (by more than 8 neurons aligned in vertical direction)
Patient 15	Ib	Abnormal tangential lamination- loss of 6-layer histological structure of the cortex
Patient 16	IIa	Abnormal lamination and the presence of dysmorphic neuron *
Patient 17	Ia	Abnormal radial lamination - abundant microcolumnar organization (by more than 8 neurons aligned in vertical direction)
Patient 18	Ib	Abnormal tangential lamination- loss of 6-layer histological structure of the cortex
Patient 19	Ia	Abnormal radial lamination - abundant microcolumnar organization (by more than 8 neurons aligned in vertical direction)

***Dysmorphic neuron:** Cells with abnormal cytoplasm and cytoplasmic appendages, in which the Nissl substance aggregated and concentrated towards the cell membrane, and in accumulation of neurofilament protein.

**** Balloon cells:** Large cells with a homogeneous eosinophilic cytoplasm and with an eccentric nucleus, where the Nissl body cannot be seen due to the accumulation of neurofilament proteins.

With the seizure activities recorded in the scalp EEG, in 68.4% (n=13) of the patients' lateralization, in 57.9% (n=11) localization was possible.

Background activity was normal in scalp EEGs of three patients. The asymmetric slowing was observed in nine patients and symmetrical slowing in seven patients. Three (n=3; 100%) of the patients with normal background activity had FCD type II. Six (67%) of the patients with asymmetric slowing had FCD type I, 2 (22%) had FCD type II, and 1 (11%) had FCD type III. Five (71%) of the patients with symmetrical slowing had FCD type I, and 29% (n=2) had FCD type II.

Frontal Intermittent Rhythmic Activity was observed in one patient. Continuous spikes and waves during sleep were

observed in one patient, and burst suppression patterns were observed in two patients. A "switch of" finding was detected in one patient. In the seizure recording, it was observed that the seizure starting from the left occipital rapidly passed to the left temporal. The incidence of RED did not differ between FCD types ($p>0.05$). There is not a significant difference between FCD type I and type II patients with respect to their scalp EEG findings allowing for lateralization and localization of the epileptogenic zone ($p>0.05$) (Table III).

When seizure semiology is examined, 26.3% of the patients (n= 5) had semiological findings compatible with lateralization and localization, 26.3% (n=5) compatible with lateralization only, and 26.3% (n=5) had findings compatible with localization

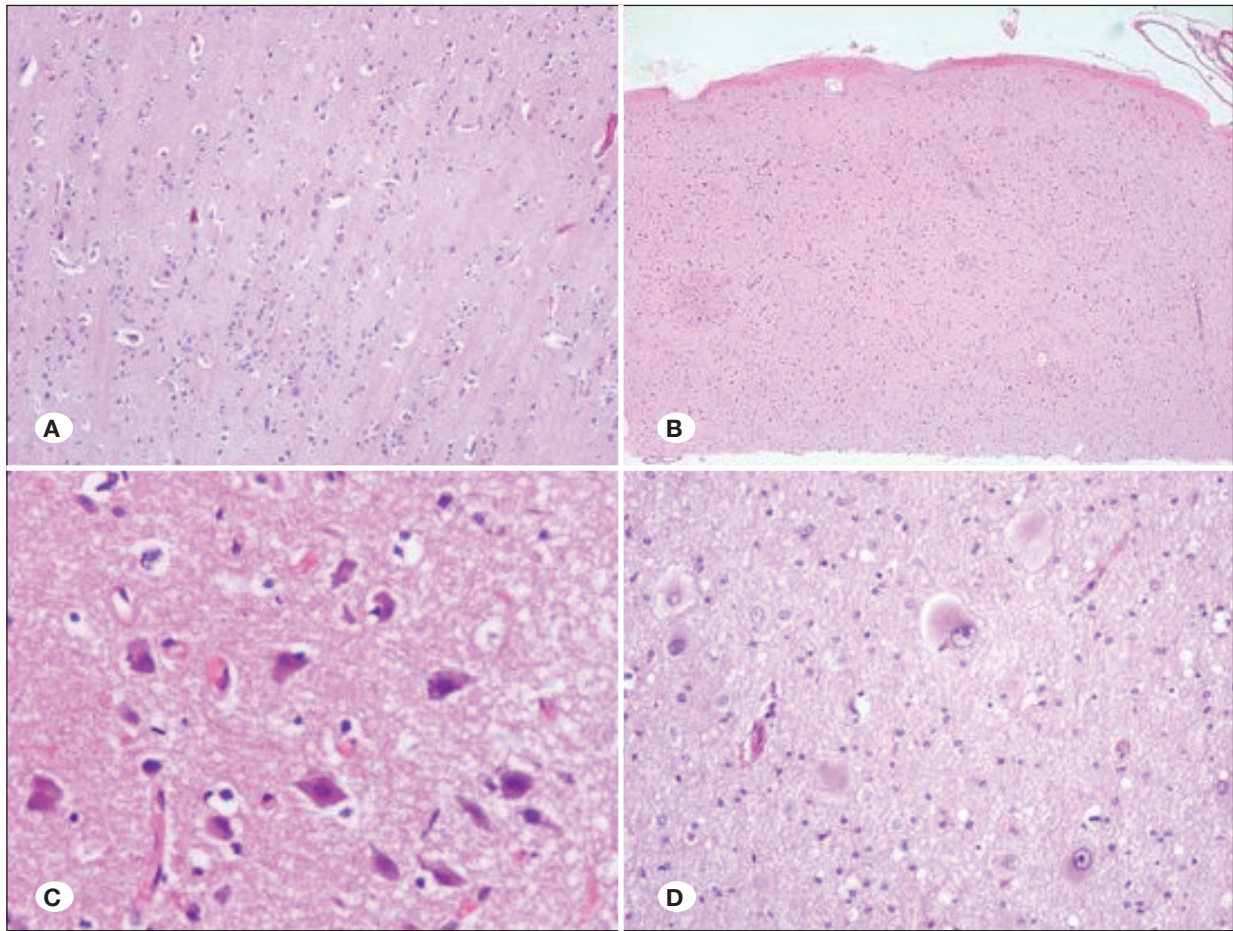


Figure 1: Pathological findings in FCD subgroups: **A)** Microcolumnar organization in FCD Type Ia, **B)** loss of 6 layer cortical lamination in FCD Type Ib, **C)** dysmorphic neurons in FCD Type IIa, **D)** balloon cells in FCD Type IIb.

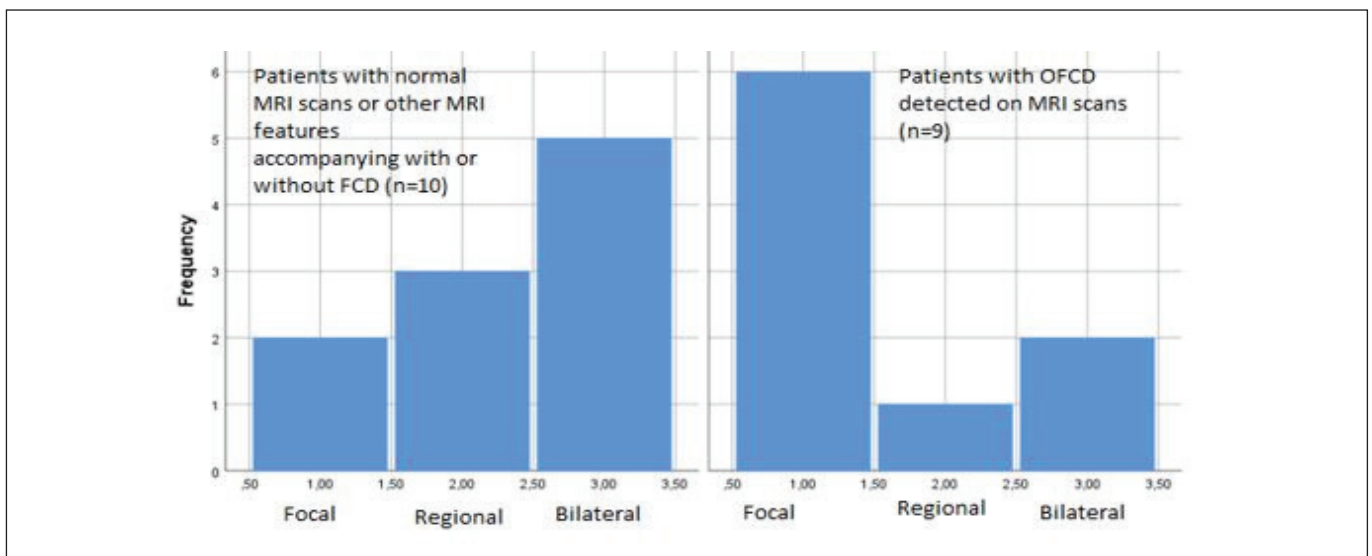


Figure 2: Interictal intermittent sharp /spike waves and MRI. *FCD: Focal cortical dysplasia; *OFCD: Only focal cortical dysplasia.

Table III: Scalp EEG Findings

Type 1 (n=11)		Focal Cortical Dysplasia			Total
		Type 2 (n=7)	Type 3 (n=1)		
RED*		3 (15.8%)	3 (15.8%)	-	6 (31.6%)
Interictal intermittent sharp / spike waves	Focal	3 (37.5%)	4 (50%)	1 (12.5%)	8 (42%)
	Regional	4 (100%)	-	-	4 (21%)
	Bilateral	4 (57%)	3 (43%)	-	7 (37%)
Non-epileptiform activities	Normal background activity	-	3 (100%)	-	3 (16%)
	Asymmetric slowdown	6 (67%)	2 (22%)	1 (11%)	9 (47%)
	Symmetric slowdown	5 (71%)	2 (29%)	-	7 (37%)
FIRDA**			1		
ESES***		1			
Burst suppression pattern		2 (1a+1b)			

*RED: Rhythmic epileptiform discharges, **FIRDA: Frontal intermittent rhythmic delta activity, ***ESES: Electrical status epilepticus in sleep.

Table IV: Engel's Classification

Class 1 (n=10)		Engel's Classification		P
		Class 2+4 (n=7)		
Focal cortical dysplasia	Type I	3 (33.3)	6 (66.7)	**0.060
	Type II	6 (85.7)	1 (14.3)	
	Type III*	1 (100)	0 (0)	

*The number of people in the group was not statistically, they were not included in the assessment, **Fisher's Exact Test.

only. In 21.1% of the patients (n=4), there were no sufficient findings for lateralization and localization. Localization and seizure semiology according to the type of FCD did not differ statistically significantly ($p>0.05$).

Three different model were performed to determine relationship between presence of OFCD (independent variable) and rhythmic activity (dependent variable) ($p=0.41$, $B=-0.84$, 95% Confidence Interval=0.06-3.22), and localization during ictal activity (dependent variable) ($p=0.46$, $B=0.69$, 95% Confidence Interval = 0.31-12.84), and lateralization (dependent variable) ($p=0.87$, $B=-0.15$, 95% Confidence Interval = 0.12- 5.94) using logistic regression analyses. There was a relationship between interictal epileptogenic activities (dependent variable) and the presence of OFCD (independent variable), but it was not statistically significant ($p=0.07$, $B=-0.23$, 95% Confidence Interval = -0.49-0.26) (Linear regression analysis). Also, it was noteworthy that the frequency of focal epileptogenic activity was higher when there was an OFCD lesion on MRI (Figure 2).

MRI/PET/SPECT/NPT Findings

MRI was normal in 15.8% of the patients (n=3; FCD type Ia, Ib, IIa). 52.6% of the patients had lesions other than FCD on MRI (n=10), in three patients in this group, FCD lesions were observed in addition to different lesions. 47.4% (n=9) of the patients had lesions with OFCD on MRI (Figure 3). The different MR findings observed in patients were as

follows: contralateral hippocampal sclerosis, polymicrogyria, hypoplasia of corpus callosum, and sequelae tissue loss. Findings were sufficient to localize the lesion in 53.3% (n=8) of the patients who underwent PET and in 83.3% (n=5) of the patients who underwent SPECT. In 33.3% of the patients (n=4), findings compatible with the lesion were obtained in NPT. The number of antiepileptic drugs used varied between two and six, with an average of 2.95 ± 0.85 . The presence or absence of lesions on MRI did not show a statistically significant difference according to the type of FCD ($p>0.05$). There was no statistically significant difference between PET, SPECT, and NPT results of FCD type I and type II patients ($p>0.05$).

Surgery and Outcome

As for post-operative results according to the Classification of Engel, 58.8% of the patients (n=10) were class I, 11.8% (n=2) were class II and 29.4% (n=5) were class IV (Table IV). There was no statistically significant difference between FCD type I and type II patients according to Engel's classification ($p=0.060$; $p>0.05$), but postoperative results were more favorable in FCD type II patients.

According to the results of the surgery, the patients were divided into two groups as Engel 1 and Engel 2 + 4 (Table IV). No statistically significant difference was found in scalp EEG findings and lateralizing or localizing the lesion ($p>0.05$). There

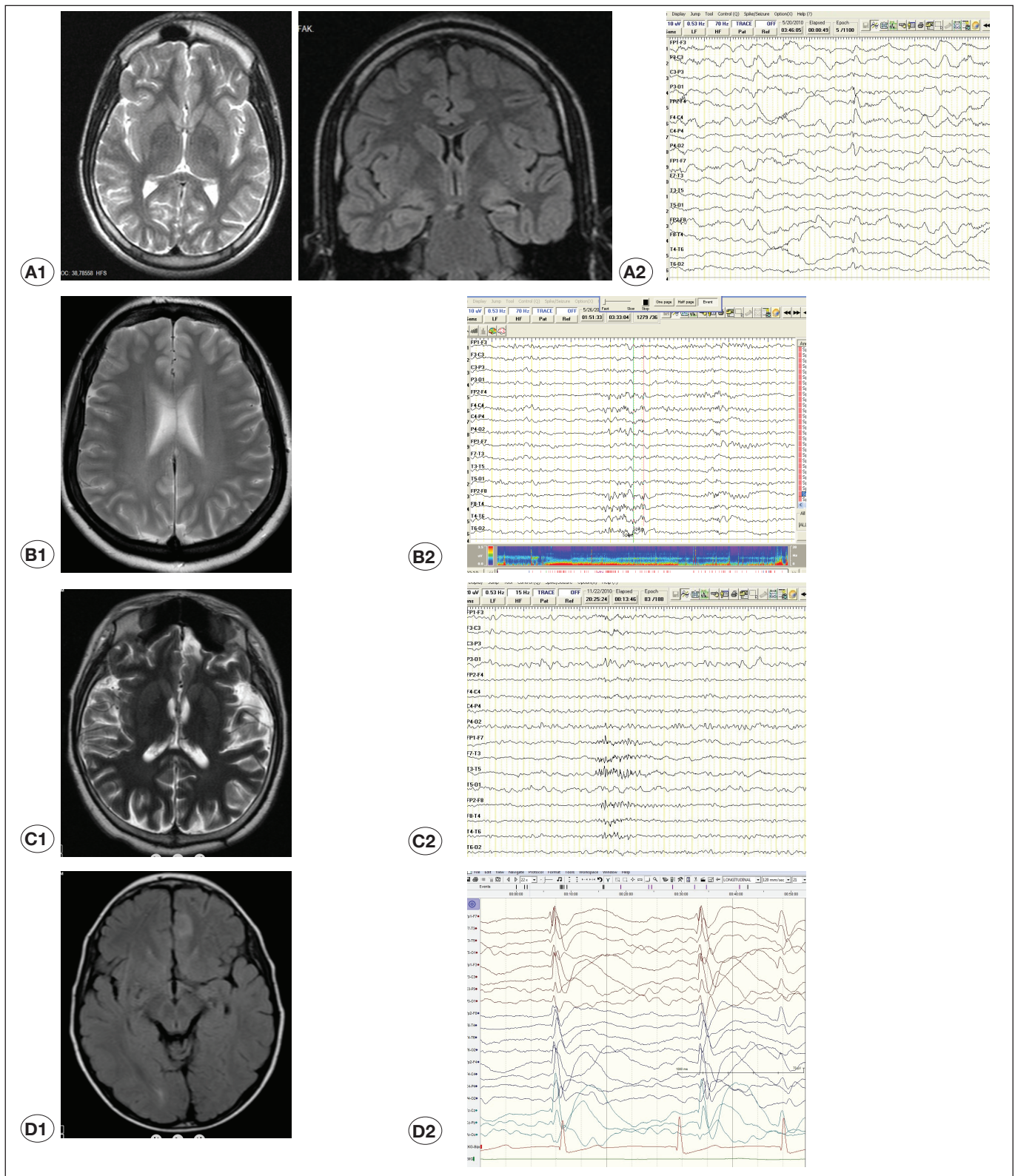


Figure 3: MRI and EEG Findings of the cases; **A1:** T2 image shows right occipital lesion compatible with FCD(A1a) and left HS on Flair images (A1b) of MRI, **A2:** Isolated intermittent spike/sharp wave activity on the posterior of the right hemisphere on scalp EEG, **B1:** Normal MRI, **B2:** REDs* in the fronto-centro-temporal-parietal on the right hemisphere on scalp EEG, **C1:** T2 image shows left frontopolar sequela cystic lesion on cranial MRI and cystic lesion with left temporopolar tissue loss, **C2:** REDs on the right frontotemporal, left temporal on scalp EEG, **D1:** Flair image shows interhemispheric lesion at the medial gyrus level in the left frontal, **D2:** NCSE**, *REDs: Rhythmic epileptiform discharges, **NCSE: Non convulsive status epilepticus.

was no statistically significant difference between the patients grouped as Engel 1 and Engel 2 + 4 according to the lesion detection associated with FCD on MRI ($p>0.05$). There was no statistically significant difference between the PET, SPECT, and NPT results of the patients who were grouped as Engel 1 and Engel 2 + 4 according to the results of the surgery ($p>0.05$).

■ DISCUSSION

In the present study, 19 patients who had FCD in pathological examinations of surgical materials and were operated on refractory epilepsy were included. Scalp EEGs of the patients were analyzed retrospectively. We aimed to discuss the findings of pathologically proven subtypes of FCD in scalp EEG and their contribution to localization of the epileptogenic zone and diagnosis. Among the FCD types, there was no difference in the incidence of REDs in scalp EEG. It was observed that the frequency of isolated intermittent spike/sharp waves was slightly higher in patients with Type II compared to type I. The frequency of focal epileptogenic activity was higher when there was an OFCD lesion on MRI. Epistashvili and et al. determined that REDs, continuous discharges, frequent rhythmic bursting epileptiform activity, polyspikes, and repetitive discharges were found in higher rates in patients with FCD compared to other etiologies of refractory epilepsies and suggested that these EEG patterns can be used as biomarkers of FCD (10). However, a study conducted among subgroups of FCD has not been identified.

Rhythmic epileptiform discharges are seen in approximately half of the patients with FCD (5,13). The spatial distribution of lesions is compatible with RED in 80% of patients (5). REDs can show the epileptogenic focus and are helpful for diagnosis. There is no significant difference between types, but it has an important place in the diagnosis. They also give an idea about the extent of the lesion during preoperative evaluation. Among our cases, REDs do not provide supportive information about spatial propagation, which indicates the need to evaluate more patients.

Isolated intermittent spike/sharp waves localize the lesion poorly and show compliance with only 43% (13). In FCD subtypes, there is no significant difference between patients regarding localization of the epileptogenic zone with scalp EEG (18,28). In our study, it is remarkable that the rate of focal activity was higher in patients with FCD Type II. Krsek et al. have found that this rate is higher in FCD type IIb, which could be associated with the presence of a more limited lesion area on MRI (17). In our study, we could not compare FCD Type II subtypes due to the small number of patients.

We observed a “switch of” finding in the scalp EEG of one patient. This finding was first reported by Steinhoff et al. and has been defined as the transition of lateralized ictal activity from one hemisphere to another in patients with temporal lobe epilepsy (23). Similarly, transition to another hemisphere region can be considered a “switch of.” This finding is known to be a negative prognostic indicator for surgical success. However, in the study conducted by Şirin et al., it has been reported

that this finding does not always show a poor prognosis in patients with temporal lobe seizures (22). In our patient with extratemporal lobe epilepsy with the switch of, complete seizure freedom was achieved after the surgery. Preoperative examinations are very important to increase the chance of success in epilepsy surgery. Based on this, we think that a comparison should be made between patients with temporal and extratemporal lobe seizures regarding the effects of “switch “ finding on prognosis.

Slowing in EEG can be observed in patients with FCD (5,18). This finding is important for the localization of the epileptogenic zone and can give an idea about hemispheric dysfunction. In our study, it was noted that intermittent and continuous EEG slowing were seen at a higher rate in FCD Type I patients. Similarly, in the study of Krsek et al., continuous EEG slowing was significantly higher in Type I FCD patients (18). Frontal Intermittent Rhythmic Activity was observed in one of the patients with focal EEG slowing in our study FIRDA is not a localizing finding regarding the epileptogenic zone (1). However, Şirin et al. pointed out that unilateral FIRDA seen in epileptic patients has a good lateralizing value for ipsilateral focal epileptic focus and focal lesion just as unilateral TIRDA (24). We should note that our case was completely seizure-free after the surgery.

In our study, one patient with FCD Type Ib had CSWS. There are studies reporting the association of polymicrogyria with CSWS (14,15). However, there are few studies about the association of FCD and CSWS. Apart from this, continuous epileptiform activity (status epilepticus) is seen in 9% of patients with FCD and this rate does not change between types (18). In our study, NCSE was seen in a patient whose pathology was compatible with FCD Type IIb.

Rare cases have been reported for concomitant FCD in patients with Lennox-Gastaut syndrome. In a reported case, the findings showed that the removal of the lesion of FCD was associated with a good prognosis (29). In our study, there was no change in seizure frequency observed after the surgery in two patients with burst suppression patterns. It was observed that these two patients developed Lennox-Gastaut syndrome in their subsequent follows up. Surgery had no significant effect on prognosis in our patients. However, it should be kept in mind that the treatment of concomitant etiologies in cryptogenic childhood epileptic encephalopathies may provide clinical benefits.

In the literature, it has been shown that patients with FCD type II have a better postoperative prognosis than FCD Type I (6,11,16,25). It has been reported that extratemporal location, undefined ictal EEG onsets, secondary generalized tonic-clonic seizures, use of intracranial electrodes and large resections are correlated with poor seizure control after surgery (20). In our study, although it was not statistically significant ($p=0.060$; $p>0.05$), similar to what is reported in the literature, it was remarkable that the success rate in FCD type II cases was higher than in FCD type I cases. The high rate of lesion detection on MRI in FCD Type II patients supported this finding, although it was not statistically significant. Cranial MRI is normal in some FCD patients (8). Especially in FCD Type I,

this rate is higher (7). Determining the boundaries of the lesion with MRI is associated with a good prognosis. In our study, however, no such relationship was found. In addition, the high frequency of focal activity in patients with OFCD lesions on MRI is worth highlighting concerning easily localizing the lesion and contributing positively to prognosis.

In cases with FCD, PET and ictal SPECT can detect the epileptogenic zone at a high rate. PET correlates approximately 75-90% and ictal SPECT correlates 50% with epileptogenic zone (20). In our study, very good results were obtained regarding showing the epileptogenic zone. However, it was not found predictive of prognosis.

Limitations

The limitation of this study is the small number of patients with FCD involved.

CONCLUSION

It seems that electrophysiological findings will help us determine FCD subgroups and reveal the relationship with the clinical and imaging features. Determination of the electrophysiological findings seen in FCD and their contribution to recognizing the subgroups of FCD will significantly contribute to the diagnosis of the disease with non-invasive methods and identify the treatment approaches. Although it was determined in this study that the characteristic features, such as EEG, MRI findings and clinical features, can help for diagnosing subgroups of FCD and may be associated with the postoperative outcome, supportive studies are needed. In addition, due to the different appearance of electrophysiology in different epilepsy etiologies, we believe the fields use electrophysiology and perspectives will change.

ACKNOWLEDGEMENTS

We thank all technicians and secretary electrophysiology laboratory, and Yildizhan Sengul for her contributions.

AUTHORSHIP CONTRIBUTION

Study conception and design: ZMG, CG

Data collection: ZMG, OK, BB

Analysis and interpretation of results: ZMG, OK, SNY, BB, CG

Draft manuscript preparation: ZMG, OK

Critical revision of the article: CG, SNY, BB

All authors (ZMG, OK, SNY, BB, CG) reviewed the results and approved the final version of the manuscript.

REFERENCES

- Accolla EA, Kaplan PW, Maeder-Ingvar M, Jukopila S, Rossetti AO: Clinical correlates of frontal intermittent rhythmic delta activity (FIRDA). *Clin Neurophysiol* 122:27-31, 2011
- Bast T, Ramantani G, Seitz A, Rating D: Focal cortical dysplasia: Prevalence, clinical presentation and epilepsy in children and adults. *Acta Neurol Scand* 113:72-81, 2006
- Bautista JF, Foldvary-Schaefer N, Bingaman WE, Lüders HO: Focal cortical dysplasia and intractable epilepsy in adults: Clinical, EEG, imaging, and surgical features. *Epilepsy Res* 55:131-136, 2003
- Blümcke I, Thom M, Aronica E, Armstrong DD, Vinters HV, Palmini A, Jacques TS, Avanzini G, Barkovich AJ, Battaglia G: The clinicopathologic spectrum of focal cortical dysplasias: A consensus classification proposed by an ad hoc task force of the ILAE Diagnostic Methods Commission. *Epilepsia* 52(1):158-174, 2011
- Chassoux F, Devaux B, Landré E, Turak B, Nataf F, Varlet P, Chodkiewicz JP, Daumas-Duport C: Stereoelectroencephalography in focal cortical dysplasia: A 3D approach to delineating the dysplastic cortex. *Brain* 123:1733-1751, 2000
- Chung CK, Lee SK, Kim KJ: Surgical outcome of epilepsy caused by cortical dysplasia. *Epilepsia* 46:25-29, 2005
- Colombo N, Tassi L, Deleo F, Citterio A, Brammerio M, Mai R, Sartori I, Cardinale F, Russo GL, Spreafico R: Focal cortical dysplasia type IIa and IIb: MRI aspects in 118 cases proven by histopathology. *Neuroradiology* 54:1065-1077, 2012
- Colombo N, Tassi L, Galli C, Citterio A, Russo GL, Scialfa G, Spreafico R: Focal cortical dysplasias: MR imaging, histopathologic, and clinical correlations in surgically treated patients with epilepsy. *Am J Neuroradiol* 24:724-733, 2003
- Engel J Jr, Van Ness P, Rasmussen TB, Ojemann LM: Outcome with respect to epileptic seizures. In: Engel J Jr, (ed). *Surgical Treatment of the Epilepsies*, 2nd ed. New York: Raven Press, 1993:609-621
- Epitashvili N, San Antonio-Arce V, Brandt A, Schulze-Bonhage A: Scalp electroencephalographic biomarkers in epilepsy patients with focal cortical dysplasia. *Ann Neurol* 84:564-575, 2018
- Fauser S, Bast T, Altenmüller DM, Schulte-Mönting J, Strobl K, Steinhoff BJ, Zentner J, Schulze-Bonhage A: Factors influencing surgical outcome in patients with focal cortical dysplasia. *J Neurol Neurosurg Psychiatry* 79:103-105, 2008
- Fauser S, Huppertz HJ, Bast T, Strobl K, Pantazis G, Altenmueller DM, Feil B, Rona S, Kurth C, Rating D: Clinical characteristics in focal cortical dysplasia: A retrospective evaluation in a series of 120 patients. *Brain* 129:1907-1916, 2006
- Gambardella A, Palmini A, Andermann F, Dubeau F, Da Costa JC, Quesney LF, Andermann E, Olivier A: Usefulness of focal rhythmic discharges on scalp EEG of patients with focal cortical dysplasia and intractable epilepsy. *Electroencephalogr Clin Neurophysiol* 98:243-249, 1996
- Guerrini R, Genton P, Bureau M, Parmeggiani A, Salas-Puig X, Santucci M, Bonanni P, Ambrosetto G, Dravet C: Multilobar polymicrogyria, intractable drop attack seizures, and sleep-related electrical status epilepticus. *Neurology* 51:504-512, 1998
- Guerrini R, Sicca F, Parmeggiani L: Epilepsy and malformations of the cerebral cortex. *Epileptic Disorders* 5:9-26, 2003

16. Kim D, Lee SK, Chu K, Park KI, Lee S, Lee CH, Chung CK, Choe G, Kim J: Predictors of surgical outcome and pathologic considerations in focal cortical dysplasia. *Neurology* 72:211-216, 2009
17. Krsek P, Maton B, Korman B, Pacheco-Jacome E, Jayakar P, Dunoyer C, Rey G, Morrison G, Ragheb J, Vinters HV: Different features of histopathological subtypes of pediatric focal cortical dysplasia. *Ann Neurol* 63:758-769, 2008
18. Krsek P, Pieper T, Karlmeier A, Hildebrandt M, Kolodziejczyk D, Winkler P, Pauli E, Blümcke I, Holthausen H: Different presurgical characteristics and seizure outcomes in children with focal cortical dysplasia type I or II. *Epilepsia* 50:125-137, 2009
19. Kuzniecky R, Murro A, King D, Morawetz R, Smith J, Powers R, Yaghai F, Faught E, Gallagher B, Snead O: Magnetic resonance imaging in childhood intractable partial epilepsies: Pathologic correlations. *Neurology* 43:681-681, 1993
20. Lerner JT, Salamon N, Hauptman JS, Velasco TR, Hemb M, Wu JY, Sankar R, Donald Shields W, Engel Jr J, Fried I: Assessment and surgical outcomes for mild type I and severe type II cortical dysplasia: A critical review and the UCLA experience. *Epilepsia* 50:1310-1335, 2009
21. Rowland NC, Englot DJ, Cage TA, Sughrue ME, Barbaro NM, Chang EF: A meta-analysis of predictors of seizure freedom in the surgical management of focal cortical dysplasia. *J Neurosurg* 116:1035-1041, 2012
22. Sirin NG, Yilmaz E, Bebek N, Baykan B, Gokyigit A, Gurses C: Unusual ictal propagation patterns suggesting poor prognosis after temporal lobe epilepsy surgery: Switch of lateralization and bilateral asynchrony. *Epilepsy & Behavior* 86:31-36, 2018
23. Steinhoff BJ, So NK, Lim S, Lüders HO: Ictal scalp EEG in temporal lobe epilepsy with unitemporal versus bitemporal interictal epileptiform discharges. *Neurology* 45:889-896, 1995
24. Sirin TC, Sirinocak PB, Arkali BN, Akinci T, Yeni SN: Electroencephalographic features associated with intermittent rhythmic delta activity. *Neurophysiol Clin* 49:227-234, 2019
25. Tassi L, Colombo N, Garbelli R, Francione S, Lo Russo G, Mai R, Cardinale F, Cossu M, Ferrario A, Galli C: Focal cortical dysplasia: Neuropathological subtypes, EEG, neuroimaging and surgical outcome. *Brain* 125:1719-1732, 2002
26. Taylor D, Falconer M, Bruton C, Corsellis J: Focal dysplasia of the cerebral cortex in epilepsy. *J Neurol Neurosurg Psychiatry* 34:369-387, 1971
27. Widdess-Walsh P, Kellinghaus C, Jeha L, Kotagal P, Prayson R, Bingaman W, Najm IM: Electro-clinical and imaging characteristics of focal cortical dysplasia: Correlation with pathological subtypes. *Epilepsy Res* 67:25-33, 2005
28. Widdess-Walsh P, Diehl B, Najm I: Neuroimaging of focal cortical dysplasia. *J Neuroimaging* 16:185-196, 2006
29. You SJ, Lee JK, Ko TS: Epilepsy surgery in a patient with Lennox-Gastaut syndrome and cortical dysplasia. *Brain and Development* 29:167-170, 2007