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FULL-LENGTH ORIGINAL RESEARCH

Interictal regional polyspikes in noninvasive EEG suggest cortical dysplasia as etiology of focal epilepsies

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SUMMARY

Purpose: To evaluate the clinical significance of interictal regional polyspikes in focal epilepsies secondary to cortical dysplasia.

Methods: We performed a data search for the term "regional polyspikes" in the database of our epilepsy-monitoring unit. Patients with generalized epilepsies including Lennox-Gastaut syndrome were excluded. Regional interictal epileptiform discharges were recorded in 513 patients with noninvasive EEG.

Results: We identified 29 patients with interictal regional polyspikes and focal epilepsies. Another 484 patients showed regional epileptiform discharges other than polyspikes. The etiology of the epilepsy was significantly more frequently cortical dysplasia in the group of patients with regional polyspikes (35%, 10 of 29 patients) than in the patients with other regional epileptiform discharges (5%, 24 of 484 patients) ($p < 0.01$). The polyspikes were significantly more frequently localized to the extratemporal (72%; $n = 21$) than temporal (28%; $n = 8$) regions ($p < 0.01$). In contrast, regional epileptiform discharges other than polyspikes were significantly more frequently localized to the temporal lobe (75%; $n = 362$) than extratemporal regions (25%; $n = 122$) ($p < 0.01$). Eight of the 10 patients with focal cortical dysplasia had extratemporal polyspikes.

Discussion: Noninvasively recorded regional polyspikes suggest cortical dysplasias as etiology of predominantly extratemporal epilepsies.

KEY WORDS: EEG, Cortical dysplasia, Regional polyspikes, Epilepsy monitoring.

Malformations of cortical development are disorders of cortical formation (proliferation, migration, and differentiation) and are frequently associated with medically refractory epilepsy (Brodtkorb et al., 1998; Hashizume et al., 2000). In selected patients, particularly with focal cortical dysplasia (FCD), resective epilepsy surgery is an option. Results of epilepsy depend on the complete resection of dysplastic cortex (Edwards et al., 2000). Preoperative evaluation includes EEG-video recording, MRI, positron emission tomography (PET) and single photon emission computerized tomography (SPECT) to identify the epileptogenic zone (Rosenow & Luders, 2001). MRI typically underestimates the extent of the pathology, which tends to be larger in histological investigations (Yagishita et al., 1997; Tassi et al., 2001, 2002). Several authors have indicated that dysplastic cortex has intrinsic epileptogenicity and they have reported this especially with intracranial studies (Palmini et al., 1995; Avoli et al., 1999; Kuruvilla & Flink, 2002). Selected patients, in whom electrocorticography (ECoG) showed polyspikes had also polyspikes in noninvasive EEG recordings (Gambardella et al., 1996).

In the present study, we investigated the frequency of regional interictal polyspikes in noninvasive EEG recordings and identified the relation to FCD in an unselected consecutive sample of patients with different focal epilepsy syndromes who underwent EEG-video monitoring for differential diagnosis of epilepsy and planning of epilepsy surgery.

METHODS

We performed a data search for the term "regional polyspikes" in the database of our epilepsy-monitoring unit

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at the University of Munich between 1994 and 2003. Patients with polyspikes associated with generalized epilepsies including Lennox-Gastaut syndrome were excluded. Regional interictal epileptiform discharges (IEDs) were recorded in 513 patients with noninvasive EEG. The term regional is defined as EEG activity that is limited to a region of the scalp (Noachtar et al., 1999). All patients underwent EEG video monitoring for differential diagnosis or difficult to treat focal epilepsy for planning of epilepsy surgery.

Noninvasive EEG monitoring

All 513 patients underwent between 3 and 14 days of continuous noninvasive EEG-video monitoring with closely spaced surface electrodes using the international 10–10 electrode system with 32–64 channel EEG machines (Vanguard, Cleveland, OH, U.S.A.; XLTEK, Oakville, Ontario, Canada). IEDs were counted in randomly selected EEG periods of 2–10 min samples per hour during wakefulness and sleep. The localizations of all IED were defined and the relative frequency of each focus was calculated for the entire duration of recording. The EEGs were evaluated in daily monitoring conferences and at least two observers agreed on the classification and localization of the EEG findings.

Polyspikes were defined as at least three consecutive spikes with a frequency of at least 10 Hz lasting at least 300 ms. Ictal EEG seizure pattern consisting of polyspikes, which typically lasted more than 4 s were excluded. Thus, we only included regional polyspikes, which were not associated with any ictal clinical change of behavior or sensation.

Imaging studies

All patients underwent cranial MRI evaluation. Each MRI includes axial, coronal, and sagittal planes T_1-weighted, T_2-weighted, proton-weighted and fluid-attenuated inversion recovery (FLAIR) images with a slice thickness of not more than 5 mm. (1.0/1.5 Tesla Impact/Vision/Symphony/Siemens). Additional coronal 3-mm T_1, T_2, and FLAIR images perpendicular to the long axis of the hippocampus were also performed. The acquisition of high-resolution T_1-weighted gradient echo sequence with an in-plane resolution and slides thickness of 1 mm was performed for detection of subtle FCD. FLAIR with 3-mm slice thickness was also performed. Contrast medium was used only if inflammation or tumors was suspected (Vollmar & Noachtar, 2004). Ictal brain perfusion SPECT with a technetium-99m-labeled ethylcysteinate dimer (^{99m}Tc-ECD, Neurolite; BMS Pharma, Brussels, Belgium) and interictal PET with fluorodeoxyglucose (FDG-PET) were performed mainly in selected extratemporal patients. The diagnosis of FCD in this study was based on the MRI results with the exception of one patient (Patient 23, Table 2), in whom histology of the resected specimen revealed FCD while MRI was normal.

Statistical analysis

Chi-square analysis or Fisher's exact test were used to evaluate the significance of relationship of regional polyspike localization and etiology of epilepsy, assuming significance at $p < 0.05$.

Results

We identified 29 patients with regional polyspikes and focal epilepsies out of 513 patients who underwent noninvasive EEG video monitoring. This comprises 5.7% of the study population (n = 513). Another 484 patients showed regional IED other than polyspikes (94.3%) such as spikes, sharp waves, spike-wave complexes (Noachtar et al., 1999). Three of the 29 patients with regional polyspikes showed only polyspikes and did not have any other IEDs. The etiologies of epilepsy of all patients are summarized in Table 1. Table 2 provides all data on the 29 patients with regional polyspikes. The duration of interictal regional polyspikes lasted between 0.5 s and 3 s. Sleep and wake periods had no effect on localization and frequency of the regional polyspikes (Table 2).

Patients with regional polyspikes had significantly more frequently cortical dysplasia (34%, 10 of 29 patients) than the patients with other regional nonpolyspike IEDs (5%, 24 of 484 patients; $p < 0.01$) (Fig. 2). Tumors were more commonly the etiology of epilepsy in patients with nonpolyspike IEDs than in the polyspike group (n = 3 of 29 vs. n = 79 of 484) (Table 1) ($p < 0.03$). Pure mesial temporal

Table 1. Etiology of epilepsy in patients with regional polyspikes and other regional interictal epileptiform discharges (IEDs)

	Regional polyspikes n = 29 (5.7%)		Other IEDs n = 484 (94.3%)		
Etiology	n	(%)	n	(%)	p
Unknown	12	(41)	167	(35)	n.s.
Tumor	3	(10)	79	(16)	0.03
Mesial temporal sclerosis	1[a]	(3)	75	(15)	–
Trauma	–	–	39	(8)	–
Focal cortical dysplasia (FCD)	10	(34)	24	(5)	0.01
Infection	–	–	19	(4)	–
Perinatal lesion	–	–	23	(5)	–
Congenital malformation	–	–	22	(5)	–
Other	4	(14)	36	(7)	–
Total	29	(100)	484	(100)	

The last column shows the statistical significance, with n.s. denoting a not significant result.

[a]Dual pathology in one patient (mesial temporal sclerosis and ipsilateral frontal FCD); not included in statistical analysis.

–, not included in statistical analysis.

Table 2. Data of the 29 patients with FCD[a]

Pat.	Age (y), Sex	Age of onset (y)	Localization of the epileptogenic zone	Etiology	MRI	Interictal polyspikes		Other IED		Ictal EEG	Epilepsy surgery outcome class (Engel et al., 1993)[b]
						Localization	%	Localization	%		
1	38 (m)	29	Lt. hemisphere	FCD Lt. hemisphere	FCD Lt. hemisphere	Lt. posterior temporal	20	1. Lt. temporal 2. Rt. temporal	22 58	Rt. frontal	ND
2	22 (f)	14	Rt. temporoparietal	FCD Rt. temporal and temporoparietal	FCD Rt. temporal and temporoparietal	Rt. post. temp.	34	1. Rt. post. temp. 2. Rt. temporal	15 51	1. Rt. post. temp. 2. Rt. central 3. Rt. centroparietal	ND
3	29 (f)	24	Lt. hemisphere	Unknown	Normal	Lt. frontocentral	100	None		Lt. frontocentral	ND
4	33 (m)	19	Lt. frontal	Unknown	Normal	Lt. frontal	13	1. Frontal nonlateralized 2. Lt. frontopolar	57 30	Frontal, nonlateralized	ND
5	42 (m)	34	Rt. frontal	Diffuse gliosis Rt. frontal	Normal	Rt. frontal	3	1. Rt. temporal 2. Rt. frontal	81 16	1. Rt. frontocentral 2. Rt. frontal	1a
6	36 (f)	13	Lt. temporal	Unknown	Normal	Lt. temporooccipital	67	1. Lt. temporooccipital 2. Lt. mesial temporal	27 6	1. Lt. temporal 2. Lt. temporooccipital	ND
7	22 (f)	12	Left paracentral	Unknown	Normal	Lt. central	79	1. Lt. central 2. Rt. central	20 1	1. Lt. central 2. Rt. central 3. Nonlateralized	ND
8	31 (f)	6	Lt. frontal	FCD lt. frontal	FCD lt. frontal	Lt. frontal	100	None		Lt. frontal	1a
9	51 (m)	9	Focal	Hypothalamic hamartoma	Hypothalamic hamartoma	1. Lt. temporal 2. Rt. temporal	39 14	1. Lt. temporal 2. Rt. temporal 3. Rt. frontal	38 8 1	Nonlateralized	3
10	9 (f)	6	Rt. parietal	Moderate gliosis Rt. parietal	Normal	Rt. parietal	12	1. Rt. temporal 2. Rt. frontal	67 21	Rt. parietal	3
11	22 (m)	7	Focal	Unknown	Normal	Lt. temporal	8	Lt. temporal	92	Lt. frontal	ND
12	19 (f)	9	Rt. frontal	FCD Rt. frontal	FCD Rt. frontal	Mid central	80	Mid central	20	Mid central	1c
13	22 (m)	1	Bilateral frontal	Calcified lesion Lt. frontocentral	Calcified lesion Lt. frontocentral	Lt. frontocentral	16	1. Lt. frontocentral 2. Rt. frontocentral 3. Lt. temporal	45 29 11	1. Lt. frontocentral 2. Rt. frontocentral	ND
14	30 (m)	11	Focal	Unknown	Normal	Lt. temporoparietal	75	Lt. temporal	25	Lt. temporal	ND
15	39 (f)	9	Lt. paracentral	Unknown	Normal	Central	100	None		1. Lt. central 2. Nonlateralized	ND
16	39 (f)	11	Focal	Hamartoma Rt. parietal	Hamartoma Rt. parietal	1. Lt. frontal 2. Rt. frontocentral	40 22	1. Lt. frontal 2. Rt. frontocentral	13 25	1. Lt. frontal 2. Rt. frontal 3. Nonlateralized	2
17	34 (f)	8	Rt. frontal	Unknown	Normal	Rt. frontocentral	34	1. Rt. frontocentral 2. Rt. temporoparietal 3. Lt. temporal 4. Lt. frontal	61 2 1 1	Rt. frontal	ND
18	33 (f)	16	Lt. parietooccipital	FCD Lt. parietooccipital > Rt. parietooccipital	FCD Lt. parietooccipital > Rt. parietooccipital	Rt. temporooccipital	29	1. Rt. temporooccipital 2. Lt. temporal-mesial 3. Rt. temporomesial	31 35 5	Rt. temporooccipital	ND

Continued

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Table 2. Continued

Pat.	Age (y), Sex	Age of onset (y)	Localization of the epileptogenic zone	Etiology	MRI	Interictal polyspikes		Other IED		Ictal EEG	Epilepsy surgery outcome class (Engel et al., 1993)[b]
						Localization	%	Localization	%		
19	53 (f)	13	Focal	FCD Lt. frontal	FCD Lt. frontal & Lt. hippocampal sclerosis	Lt. frontal	13	1. Rt. temporal mesial 2. Lt. frontal 3. Lt. temporal	49 22 16	1. Rt. frontopolar 2. Lt. frontopolar	ND
20	17 (m)	2	Rt. frontal	FCD Rt. frontal	FCD Rt. frontal	1. Rt. frontal	2	1. Rt. frontal 2. Rt. frontocentral	96 2	Rt. frontal	ND
21	25 (f)	25	Focal	Unknown	Normal	1. Rt. occipital 2. Lt. occipital	13	1. Rt. occipital 2. Lt. occipital 3. Lt. frontal 4. Rt. frontal 5. Lt. temporal 6. Lt. parietal	70 3 2 3 7 2	Lt. temporal	ND
22	20 (m)	13	Focal	Unknown	Normal	Rt. mesial temporal	12	1. Rt. mesial temporal 2. Lt. mesial temporal	59 29	1. Lt. temporal 2. Rt. temp.-occ.	ND
23	32 (m)	17	Rt. frontal	FCD Rt. frontal	Normal	Rt. frontal	72	Rt. frontal	28	1. Rt. frontal 2. Rt. temporal 3. Nonlateralized	2
24	36 (m)	5	Rt. frontal	FCD Rt. mesial frontal	FCD Rt. mesial frontal	Rt. frontal	63	Rt. frontal	37	Rt. frontal	2
25	29 (m)	2	Focal E	Febrile seizures (amygdalo-hippocampectomy) 1983)	Status post amygdalo-hippo-campectomy	Rt. parietal	47	1. Rt. parietal 2. Rt. temporal 3. Lt. temporal	49 3 1	1. Rt. frontal 2. Rt. parietal	SUDEP
26	33 (m)	7	Rt. FLE	FCD Rt. frontal	FCD Rt. frontal	Rt. frontal	64	Rt. frontal	36	Rt. frontal	1a
27	43 (f)	9	Focal E	Cavernoma Lt. frontal and Rt. occipital	Cavernoma Lt. frontal and Rt. occipital	1. Lt. temporal 2. Rt. frontal	28	1. Lt. temporal 2. Rt. frontal 3. Lt. frontal 4. Frontocentral nonlateralized 5. Generalized	39 7 15 4 7	1. Lt. frontocentral 2. Lt. frontal 3. frontocentral nonlateralized 4. Rt. temporal	ND
28	24 (f)	21	Focal E	Unknown	Normal	Rt. frontocentral	40	1. Rt. posterior temporal 2. Rt. frontocentral	47 13	1. Rt. frontal 2. Rt. temporal	ND
29	27 (m)	12	Focal E	Unknown	Normal	Lt. anterior temporal	19	1. Lt. temp.-ant. 2. Lt. posterior temporal 3. Lt. frontopolar 4. Rt. posterior temporal	77 2 1 1	1. Lt. temporal 2. Rt. parietooccipital 3. Lt. parietooccipital	ND

[a]Values which are smaller than one have been rounded to one. Lt., left; Rt., right; focal, focal but not further localized; ND, not done; SUDEP, sudden unexpected death in epilepsy.

[b]Numbers in this column refer to the Epilepsy Surgery Outcome Classification as proposed by Engel et al., 1993.

Epilepsia, 49(6):1011–1017, 2008
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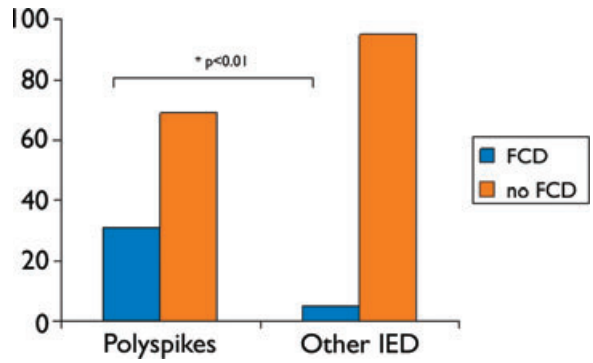

Figure 1.
Focal cortical dysplasia (FCD) is more common in patients with regional polyspikes (10 of 29) than other regional interictal epileptiform discharges (IEDs) (24 of 484).
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Table 3. Frequency of patients with regional polyspikes and other regional interictal epileptiform discharges (IEDs) (n = 513) and localization of the IEDs

	Total subjects n = 513		
Localization	Regional polyspikes n = 29 (5.7%)	Other regional IEDs n = 484 (94.3%)	Significance
Temporal	8 (28%)	362 (75%)	$p < 0.01$
Extratemporal	21 (72%)	122 (25%)	$p < 0.01$
Significance	$p < 0.01$	$p < 0.01$	

sclerosis only occurred in the nonpolyspike IED (Table 1). One patient with frontal polyspikes had a dual pathology with a frontal FCD and an ipsilateral mesial temporal sclerosis (Table 1).

The polyspikes were significantly more frequently localized to extratemporal (72%; n = 21) than temporal (28%, n = 8) regions ($p < 0.01$) (Table 3). In contrast, regional IEDs other than polyspikes were significantly more frequently localized to the temporal lobes (75%, n = 362) than extratemporally (25%, n = 122) ($p < 0.01$) (Table 3). Eight of the 10 patients with FCD had extratemporal polyspikes. The localizations of the regional polyspikes and the FCDs were consistent in 9 of 10 patients. In one patient with right frontal FCD the polyspikes were midcentral.

The regional polyspikes had a repetition rate of 10–22 Hz and occurred during wakefulness and sleep in patients with and without FCD. For the purpose of this study, we did not quantify the occurrence of regional polyspikes during sleep or wakefulness. There was no significant difference in the frequency and duration of the regional polyspikes in patients with and without FCD.

Discussion

This study shows that scalp-recorded interictal regional polyspikes are more commonly associated with FCD than other epileptiform discharges in epilepsy surgery candidates with poor antiepileptic drug (AED) control. To avoid the selection bias of former studies who primarily look at children with cortical dysplasia (Quirk et al., 1993) or patients who underwent invasive ECoG (Gambardella et al., 1996), we evaluated a series of unselected consecutive patients who all underwent noninvasive EEG-video monitoring. Our patient population was heterogeneous and reflects all patients referred to an epilepsy-monitoring unit for evaluation of possible epilepsy surgery and differential diagnosis of focal epilepsy. Thus, different etiologies of epilepsy are represented.

In invasive recordings (ECoG), FCD was associated with high-frequency spiking (polyspiking), and the prolonged epileptic activity in dysplastic tissue was considered a consequence of impairment of local inhibitory circuits (Palmini et al., 1995). Most common findings were recruiting/derecruiting spikes (48%), high-frequency rhythmic polyspikes (bursting pattern, 30%), and continuous/quasicontinuous rhythmic spiking pattern on intraoperative ECoG recordings (35%). In a retrospective analysis of the surface EEG of these patients, the occurrence of rhythmic epileptiform discharges on the noninvasive EEG and continuous epileptiform discharges on ECoG recordings were compared in patients who underwent resective epilepsy surgery (Gambardella et al., 1996). It was concluded that repetitive spiking/polyspiking was highly specific and a sensitive indicator for focal cortical dysplastic lesions. Autoradiography of surgical specimen of FCD revealed reduced density of GABA-A receptors as visualized preoperatively by flumazenil PET (Arnold et al., 2000). Although continuous spiking was also described in patients with gliosis after traumatic brain injury or brain tumors, it has been suggested that continuous spiking on preresection ECoG can predict the presence of coexisting cortical dysplasia in a high proportion of patients (91%) with a specificity of 96% (Ferrier et al., 2006). These results and our findings support that continuous spiking and regional polyspikes are seen significantly frequent in FCD. However, the specificity of these invasive EEG findings for cortical dysplasia has been questioned by others who found polyspiking in invasive recordings also in other etiologies such as tumors (Rosenow et al., 1998). In electrocorticographic recordings, continuous spiking has been seen in 55% versus 12% of patients with FCD and glioneural tumors (GNT), respectively, and the FCDs were more frequently localized extratemporally when compared to GNTs (Ferrier et al., 2006). In concordance with this invasive study, we found that regional extratemporal polyspikes in

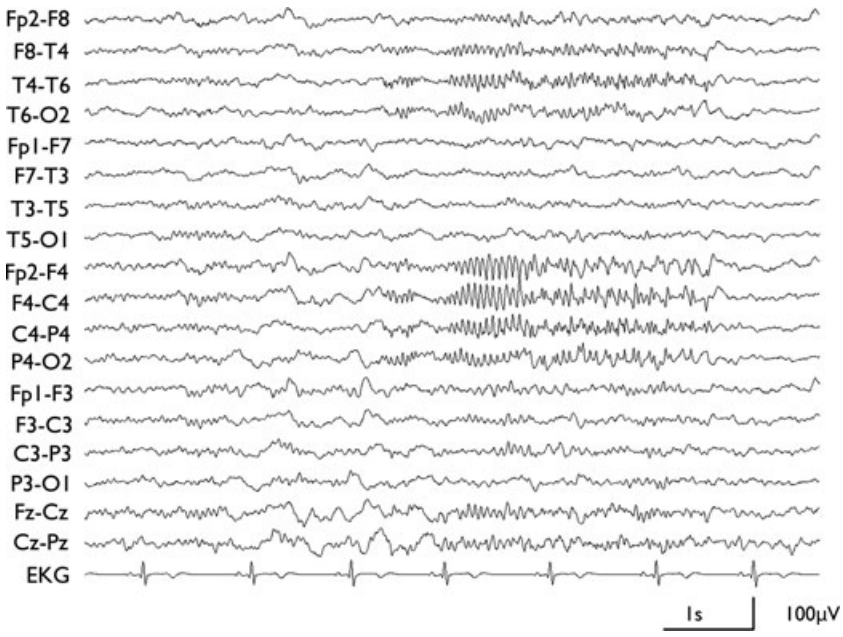

Figure 2.
Right frontal polyspikes during sleep EEG (longitudinal bipolar montage) in a 31-year-old patient with a right FCD.
Epilepsia © ILAE

noninvasive EEG are highly associated with cortical dysplasia (80%).

Cortical dysplasias used to be recognized only in the resected tissue during surgical treatment of patients with intractable epilepsy until the development of modern imaging techniques. CT has a low sensitivity for FCD but MRI enabled the recognition and classification of the different types of lesions (Andermann, 2000). High-resolution MRI using special techniques may reveal dysplastic cortex, which was not detected by standard MRI (Hakamada et al., 1979; Quirk et al., 1993; Palmini et al., 1995; Raymond et al., 1995; Raymond & Fish, 1996). However there are medically refractory epilepsy patients with normal MRI (Sisodiya, 2000; Tassi et al., 2002). In some of these patients, postsurgical histological examination helps detecting cortical dysplasia, which was not identified by MRI (Raymond & Fish, 1996; Yagishita et al., 1997; Tassi et al., 2001; Tassi et al., 2002).

Our study shows the diagnostic value of interictal regional polyspikes as a correlate of FCD, which was more significant in extratemporal localizations. We conclude that regional polyspikes, especially in extratemporal location, should lead the clinician to perform advanced MRI studies to detect cortical dysplasia.

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Conflict of interest: We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines. The authors report no conflicts of interest.

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