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Childhood absence epilepsy and electroencephalographic focal abnormalities with or without clinical manifestations

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Outcome

Summary

Purpose: We studied the electroclinical features and evolution in patients with childhood absence epilepsy (CAE) associated with electroencephalographic findings similar to those of benign focal epilepsies (BFE) with or without clinical manifestations compatible with these focal idiopathic syndromes.
Methods: Between June 1994 and June 2002, we found 203 (3.6%) patients with typical electroclinical features of CAE among 8285 children with epilepsy. From this population of 203, we found 30 cases (14.7%) that also showed focal abnormalities of BFE on the EEG. Seven of these 30 cases also had clinical manifestations of BFE that preceded the onset of the absences.
Results: There were 20 (66.5%) boys and 10 (33.5%) girls. Age at onset of absences ranged from 2 to 10.5 years, with a mean age of 5.5 years. Of 30, 7 had focal clinical seizures as well. Three of seven had seizures characteristic of Panayiotopoulos syndrome (PS), and the other four had seizures compatible with childhood occipital epilepsy (COE) of Gastaut. The focal seizures started between 3 and 7 years of age. In all patients seizures were under control within 2–24 months (mean: 11 months) after onset. The focal discharges disappeared in 26 patients at a mean age of 8 years (range 4–13 years), 1 year after the typical absences had disappeared. In four patients the focal paroxysms are still present.

Abbreviations: CAE, childhood absence epilepsy; BFE, benign focal epilepsies; PS, Panayiotopoulos syndrome; COE, childhood occipital epilepsy.

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Conclusion: The association of two different idiopathic focal and generalized epilepsies in the same patient may be merely coincidental, but a close genetic relationship between both epileptic syndromes might be another hypothesis. Another explanation could be that our series of patients represent a subgroup of CAE.

Introduction

The latest proposed diagnostic scheme for people with epilepsy, the Classification of Epilepsies and Epileptic Syndromes of the International League Against Epilepsy,[1] defines childhood absence epilepsy (CAE) as an idiopathic generalized epilepsy characterized by typical absences associated with bilateral, synchronous and symmetric spike-and-wave discharges at 3 cycles per second. The proposal includes among the idiopathic focal epilepsies in childhood: benign childhood epilepsy with centrotemporal spikes (BCECTS), Panayiotopoulos syndrome (PS), and childhood occipital epilepsy (COE) of Gastaut.

The possible occurrence of generalized spike—wave discharges without clinical manifestations in patients with BCECTS has been well documented.[2–17] According to these authors, brief subclinical generalized spike—waves during sleep and wakefulness may occur in between 6.8 and 73% of the cases. The presence of these generalized paroxysms in patients with COE of Gastaut and less frequently in patients with PS has also been described.[18–25] However, the coexistence of focal EEG discharges similar to those found in idiopathic focal epilepsies in childhood associated with CAE has been more rarely documented.[26–30]

The co-occurrence of CAE and idiopathic focal epilepsies in the same child has occasionally been reported,[7,18,24,30–33] but the genetic linkage or a continuum between CAE and idiopathic focal epilepsies remains an open chapter and a debate in the literature.[11,14,17,33,34]

We studied the electroclinical features and evolution in patients with CAE associated with electroencephalographic findings similar to those found in idiopathic focal epilepsies with or without clinical manifestations compatible with these focal idiopathic syndromes.

Methods

Between June 1994 and June 2002, we found 203 (3.6%) patients with typical electroclinical features of CAE among 8285 children with epilepsy. The following criteria for CAE were considered [35]:

Inclusion criteria
- Age at onset between 2 and 10 years.
- Normal development and neuropsychological state.
- Pure typical absences lasting 5—25 sec with abrupt and clear impairment or loss of consciousness occurring several times a day.
- Ictal EEG discharges of generalized high-amplitude spikes and double (maximum three occasional spikes are allowed) spike and slow-wave complexes; rhythmic spike—waves at around 3 Hz with a gradual and regular slowdown from the initial to the terminal phase of the discharge. Varying duration of the discharges between 5 and 25 s.

Exclusion criteria
- Cases with atypical absences, brief absences and absences associated with motor manifestations such as generalized tonic—clonic seizures, or myoclonic jerks before or during the active stage of absences.
- Eyelid myoclonia, perioral myoclonia, rhythmic massive limb jerking, and single or arrhythmic myoclonic jerks of the head, trunk, or limbs. However, mild myoclonic elements of the eyes, eyebrows, and eyelids may be featured, particularly in the first seconds of the absence seizure.
- Brief EEG 3—4 Hz spike—wave paroxysms of less than 4 sec, multiple spikes more than 3 or ictal discharge fragmentations.
- Reflex seizures.
- Patients with absences that may be the result of secondary bilateral synchronies.

Among this population of 203, we found 30 cases (14.7%) that also showed focal EEG abnormalities of benign focal epilepsies (BFE) (a focal negative diphasic slow spike of medium to high voltage mostly over 100 μV followed by a slow wave).[36] Seven of these 30 cases also had clinical manifestations of BFE before the onset of the absences.

Our patients were monitored for 2 to 12 years (mean 5.5 years) with repeated clinical examinations and electroencephalograms during sleep and while awake. Morphology and topography of ictal and interictal paroxysms, and their reactivity to eye opening, hyperventilation, and photosensitivity were specially looked for. Family and personal his-

Table 1 Electroclinical features of benign focal epilepsy and CAE in seven children

Patients	Gender	Type of focal epilepsy	Age (years) at onset focal epilepsy	Age (years) at last focal seizure	Treatment	Age (years) at onset absences	Treatment	Age (years) at last absence	Age (years) at last focal spikes
1	M	PS	3	4	VPA	7	VPA	7.5	8.5
2	M	COE-G	5	6	VPA	8	VPA	9	12
3	F	COE-G	6	7.5	VPA	9	VPA	9.5	11
4	F	PS	4.5	5	—	6	ESM	8	10
5	F	COE-G	6	6	VPA	7	VPA	9	11.5
6	M	PS	4.7	5.5	VPA	8	VPA	10	12
7	M	COE-G	7	7.5	VPA	9	VPA	10.5	11.5

PS: Panayiotopoulos syndrome, COE-G: childhood occipital epilepsy of Gastaut, VPA: valproic acid, ESM: ethosuximide.

tories of epilepsy and febrile seizures were analysed, as well as age at onset, semiology, and duration and frequency of the seizures. Neurological examinations, brain CT scan, and magnetic resonance imaging were obtained in 15 and 7 patients, respectively.

Results

Number of patients and gender

There were 30 children, 20 (66.5%) boys and 10 (33.5%) girls, who met the inclusion criteria of CAE and who also had focal EEG abnormalities similar to BFE of childhood. Seven of them (23%) also had focal clinical manifestations (see Table 1).

Age at onset

Age at onset of absences ranged from 2 to 10.5 years, with a mean age of 5.5 years.

Family history of febrile seizures and epilepsy

A family history of epilepsy was found in four cases (13%): A brother of one patient had CAE without focal EEG abnormalities, the father of another patient had generalized tonic—clonic seizures, and a pair of siblings included in this series had CAE and COE of Gastaut. Febrile seizures were reported in five (16.5%).

Personal history of febrile seizures and epilepsy

Three children (10%) had had febrile seizures. Seven patients had electroclinical features of BFE before the typical absences started. Three of them previously had seizures characteristic of Panayiotopoulos syndrome (Figs. 1 and 2), and the other four seizures compatible with COE of Gastaut (Figs. 3 and 4). The focal seizures started between 3 and 7 years of age. Three of these last four children were referred to our center — and included in a previous study published by our group [24] — so this association does not represent a real prevalence in our series.

Ictal manifestations

The ictal events in CAE were characterized by typical absences with rhythmic clonic eye seizures in 10 cases, and automatisms and atonic components with autonomic signs in 6. The remaining 14 patients only had absences without any other signs.

Neurological examination and neuroradiological imaging

Neurologic and neuropsychological examinations were normal in all cases. Brain CT scan (performed in 15 cases) and MRI (7 cases) were normal as well.

Electroencephalographic findings

The background EEG rhythms were normal in all patients. Typical absences were associated with bilateral synchronous and symmetrical discharges of spike and waves at 3 cycles per second, spontaneously and during hyperventilation, and with more amplitude in anterior regions. The interictal EEG recordings documented spike and polyspike waves during wakefulness and in stages 1 and 2 of slow sleep in 25 patients. Nine patients exhibited a posterior symmetrical delta rhythm and sinusoidal activity at 3 cycles per second that was blocked by eye opening. Photoparoxysmal response on the EEG was positive in six children, and closing of the eyes did not activate absences in any of the children.

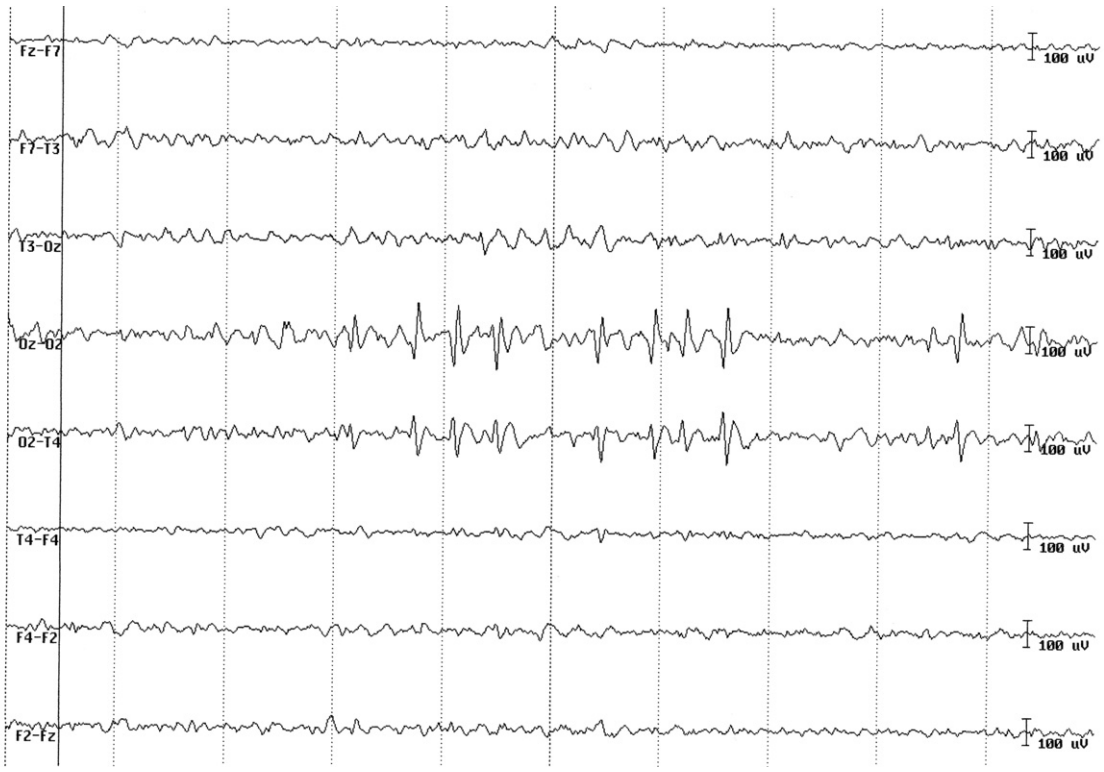

Figure 1 A 4-year and 6-month old boy with typical clinical features of PS. The awake EEG recording shows occasional right occipital spikes.

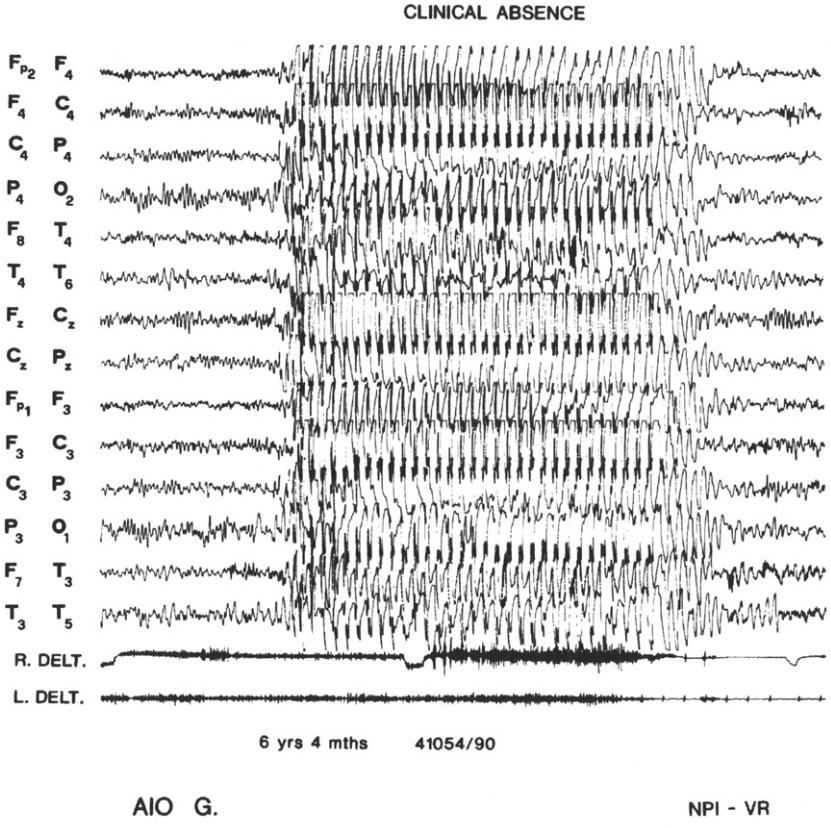

Figure 2 The same child, at 6 years and 4 months of age. The EEG recording shows bilateral, synchronous and symmetric spike-and-wave discharges at 3 Hz associated with typical absences.

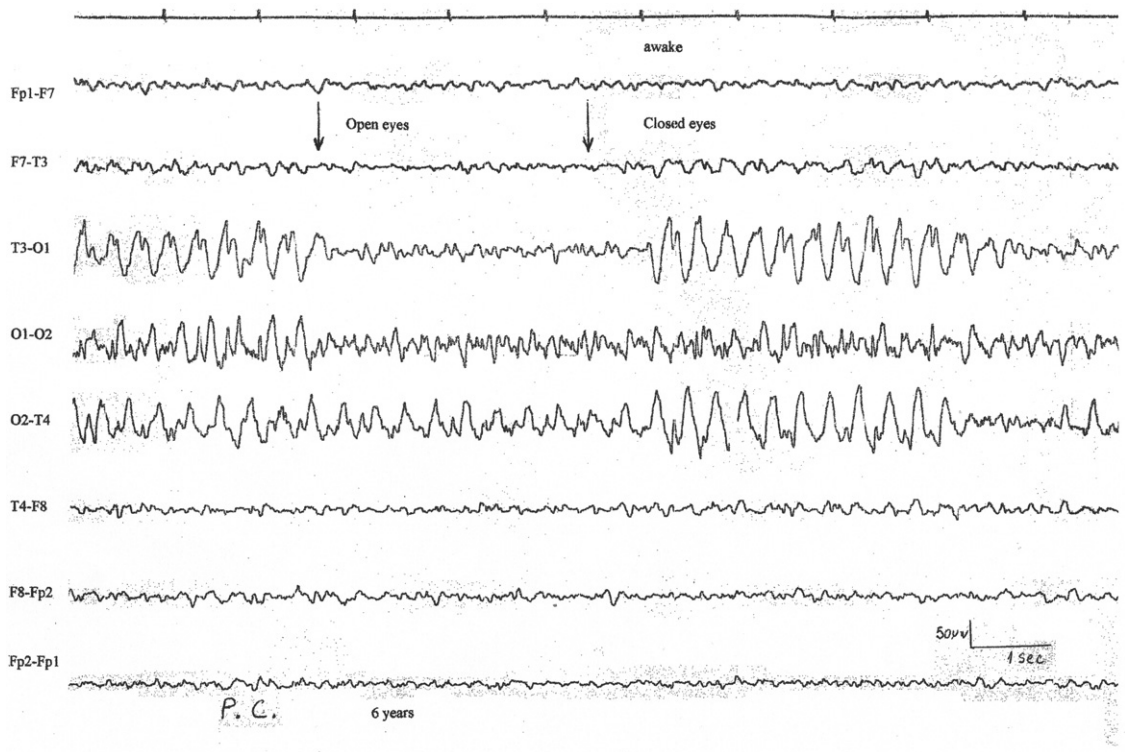

Figure 3 A 6-year-old girl with visual manifestations followed by hemiclonic seizures and migraine-like symptoms. The interictal EEG shows bilateral occipital spike—waves reactive to eye opening and closure.

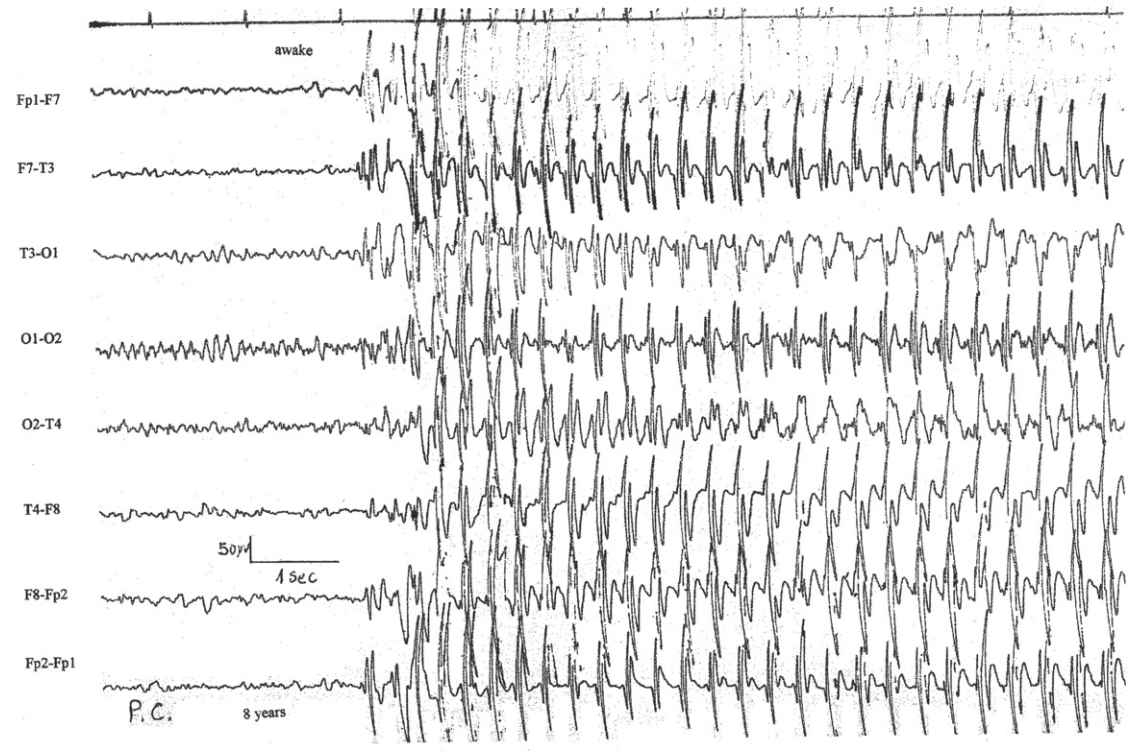

Figure 4 The same child of this figure, at 8 years of age. The ictal EEG shows bilateral, synchronous, and symmetric spike-and-wave discharges at 3 Hz associated with typical absences.

Focal EEG abnormalities found in typical idiopathic focal epilepsies were registered in all children. In 25 patients, the focal paroxysms were documented during sleep and wakefulness, and in 5 children the focal discharges only appeared during sleep. Eleven of 30 presented more than 2 independent foci. As typically occurs in focal idiopathic epilepsies, the paroxysms were activated during sleep.

Centrotemporal spikes were found in 10 patients, parietooccipital spikes in 9, centrotemporal and parietooccipital in 3, frontocentrotemporal and vertex in 3, frontocentrotemporal in 3, frontoparietooccipital, and vertex in 2 of the cases. In five children, similar focal discharges were evoked by finger tapping.

The focal paroxysms were already present at the onset of the typical absences in 23 patients and in 7 they occurred after onset.

Treatment

All patients received antiepileptic drugs: 21 patients were treated with valproic acid, 4 with ethosuximide, 3 with valproic acid associated with ethosuximide, and 2 with polytherapy. All patients with focal seizures received valproic acid, except one who was not treated. However, none of the patients was treated with valproate at the time of the diagnosis of CAE.

Evolution

The absences were under control within 2—24 months (mean: 11 months) after onset in all patients. Antiepileptic treatment was discontinued in 17 patients, 10 receive monotherapy, and 3 receive 2 antiepileptic drugs. All patients have remained seizure free over a period of 1—9 years of follow-up. The focal discharges disappeared in 26 patients at a mean age of 8 years (range 4—13 years), 1 year after the typical absences had disappeared. In four patients the focal paroxysms are still present despite the disappearance of the absences over a period of 2—24 months. One case presented with a continuous spike wave during slow sleep at 6 years of age, 10 months after the onset of typical electroclinical features of CAE. In this patient, the continuous spikes and waves during slow sleep disappeared spontaneously 10 months later.

At last examination (range 9—18 years), all patients had normal neurological and neuropsychological evaluations.

Discussion

In this study, the patients had a typical electroclinical picture and evolution of CAE as previously described in the literature.[37—41] All of them also had focal EEG abnormalities similar to those found in idiopathic focal epilepsies of childhood.[36]

From a prognostic point of view, the coexistence of these focal paroxysms and CAE in our patients was not different from the typical cases with CAE described in the literature. Neither did this EEG finding represent an increased risk to develop other types of seizures. Similar results were described in the literature.[26—30] In the majority of the patients the antiepileptic medications were discontinued before the onset of adolescence and before the spontaneous disappearance of BFE. In the seven cases that had both types of epilepsies the absences always started after the initiation of BFE and the contrary has never occurred yet.

Four of 30 had typical clinical manifestations of COE of Gastaut, and 3 of 30 had typical clinical features of PS. The incidence of the focal paroxysms associated with CAE was higher (30 of 203, or 14.7%) than that found in the literature up to now.[26—30,42] The presence of CAE in children who initially had BFE has been well documented, but is rare.[24,30—33,43,44] It is interesting to note that in our series of patients Rolandic spikes were the most frequent interictal focal abnormality and Rolandic epilepsy is recognized as the most frequent idiopathic focal syndrome in childhood. However, none of our patients with Rolandic spikes had clinical manifestations.

In our series of patients with CAE associated with focal abnormalities with or without clinical seizures, 20 (65%) of 30 are males, whereas in the total population of our series of cases with CAE there is a clear female predominance.

The cause of the existence of focal EEG discharges in cases with CAE has not been explained as yet. Berkovic [34] proposes that the focal and generalized EEG paroxysms may be an expression of common and shared genes in families with generalized epilepsy and families with BFE. This hypothesis may also explain the frequent incidence of generalized spikes and waves in patients with BFE.[2—4,7,9,10—12,14,16,17,45] It is even more difficult to understand the reason for the coexistence of two different types of epilepsy in the same patient. The occurrence of typical generalized spike-and-wave discharges of 3 cycles per second has led to speculation about a neurobiologic, pathophysiologic and genetic continuum between childhood absence seizures and BFE.[17,33] From pathophysiological point of view, another issue that could be involved is the theory stating that absence epilepsy might in fact have a focal onset. A cortical focus spreading to

the thalamus may induce the thalamus to elicit a bilaterally synchronous generalized spike—wave response.[35,39] In our group of cases, focal EEG abnormalities without clinical manifestations were found in 15%, which is more frequent than in the normal population (5%).

According to the literature, a benign course in patients with CAE occurs in only about 40—60%.[35,39] In our series of patients, the association between CAE and focal EEG abnormalities with or without clinical manifestations, predominantly in males, and with a uniformly benign prognosis may suggest the existence of a subgroup of CAE. This could be another argument for a genetic link. Some authors, however, believe that the rarity of the association of two types of idiopathic epilepsies makes this hypothesis less feasible.[30,32]

Recently, three patients with COE of Gastaut and a peculiar evolution were published. The patients had ictal events that were characterized by visual symptoms followed by typical absences. In two of them, the seizures continued despite AED treatment. [46] One may interpret that the appearance of typical absences in these three patients is due to the phenomenon of secondary bilateral synchronies rather than the occurrence of two different types of idiopathic epilepsies in the same patient. Sofue et al. [47] reported 12 patients who had focal seizures in early childhood that were later followed by absences. Two of them had focal seizures and absences at the same age, but not during the same seizures. The absences responded well to antiepileptic drugs, but the focal seizures were refractory in most of the cases. These patients had symptomatic or probably symptomatic focal epilepsy. The absences may also be due to secondary bilateral synchrony.

Conclusion

In patients with CAE, focal discharges characteristic of BFE may be present during an important period of the evolution. This finding in 30 cases (14.7%) of our series of 203 patients with CAE coincides with other series of patients with CAE published.

The association of two different idiopathic focal and generalized epilepsies in the same patient may be merely coincidental, but a close genetic relationship between both epileptic syndromes might be another hypothesis. Another explanation could be that our series of patients represent a subgroup of CAE.

Future genetic studies are necessary to define whether these two idiopathic epileptic syndromes are genetically linked.

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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.

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