Incidence and outcome of epilepsy syndromes with onset in the first year of life: A retrospective population-based study

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Summary

Objective: Population-based studies on infantile epilepsy syndromes are scarce. Our aim was to provide syndrome-specific data on the incidence and outcome of epilepsy in a population-based cohort of infants with epilepsy onset in the first year.

Methods: Included were all infants born in 1997 through 2006 whose epileptic seizures started before 12 months of age and who were residents of the Helsinki University Hospital district at the time of seizure onset. Patients were ascertained from hospital statistics, and all patient charts were reviewed. A reevaluation of the epilepsy syndromes, age at onset, etiology, and outcome at 24 months of age was based on data abstracted from the patient files.

Results: Inclusion criteria were fulfilled by 158 infants, of whom 92% were followed until age 24 months or death. The incidence of epilepsy in the first year was 124 of 100,000. An epilepsy syndrome recognized by the revised organization of epilepsies by ILAE was identified in 58% of the patients. The most common syndromes were West syndrome (41/100,000) and benign familial or nonfamilial infantile epilepsy (22/100,000). Etiology was structural-metabolic in 35%, genetic in 17%, and unknown in 48%. Early age at onset was associated with structural-metabolic etiology. Seven infants (4.4%) died before age 2 years. One infant with an SCN2A mutation died of sudden unexplained death in epilepsy (SUDEP). At 24 months, 58% of all children included in the cohort were seizure-free, and 46% had both seizure freedom and age-appropriate cognitive development. Age at onset was not associated with outcome when etiology was controlled for.

Significance: Benign familial and nonfamilial infantile epilepsy appears to be more common than previously suggested, second only to West syndrome. Early age at onset is not an independent risk factor for poor outcome.

KEY WORDS: Infantile epilepsy, Incidence, Epilepsy syndrome, Outcome.

Epilepsy has a high incidence in the first year of life compared to later childhood.1,2 Poor outcomes with only 21–38% of the children achieving normal development have been reported from hospital-based settings,3–6 whereas the only population-based study reporting outcome in this age group indicates a slightly better outcome (47% with normal cognition).7 Since 1978, several new epilepsy syndromes with onset in the first year have been identified,8–12 and all but one have been recognized by the new International League Against Epilepsy (ILAE) recommendation for terminology and organization of epilepsies.13 Apart from infantile spasms (West syndrome),14–16 very scarce population-based data are available on the incidence2,17 and prognosis7 of epilepsy syndromes in infancy.

Etiology has been shown to be the major determinant of prognosis in infantile epilepsy,7,18 but remains unknown for more than one third of patients in epidemiologic studies.2,17
Key Points

- A recognized epilepsy syndrome was identified in 58% of infants in a population-based cohort.
- After West syndrome, benign infantile epilepsy is the second most common syndrome with an incidence of 22/100,000.
- Seizure freedom with normal developmental outcome was observed in 46% of all patients at 24 months of age.
- Age at onset is not a significant predictor of outcome when etiology is controlled for.

As neuroimaging methodologies have advanced, developmental brain abnormalities have been increasingly revealed as causes of infantile epilepsy. Furthermore, the rapid development of next-generation sequencing techniques together with clinical genetic studies have led us to understand the importance of gene mutations in the etiology of epilepsy.

Our aim was to study the incidences of epilepsy syndromes and their etiology, applying the revised ILAE terminology and organization of seizures and epilepsies, to assess outcome at 2 years, and investigate if age at onset has an independent effect on outcome in a population-based cohort of infants with epilepsy onset in the first year of life.

Methods

We initially performed a search into Helsinki University Hospital patient statistics to identify all patients born in 1997 through 2006, who had been given a diagnosis of epilepsy (International Classification of Diseases, Tenth Revision [ICD-10]) codes G40.0 – 40.9) before the age of 24 months and who were residents at that time within the Helsinki University Hospital health care district (Helsinki, Vantaa, Espoo, Kauniainen, Kerava, and Kirkkonummi). All infants with seizures in this region are seen by a pediatric neurologist either at the Children’s Hospital in Helsinki or at the Jorvi Hospital in Espoo.

Next, we reviewed the hospital charts of these infants and included those whose first seizure occurred before the age of 12 months and whose epilepsy diagnosis could be confirmed after reevaluation of the data and consensus by two of the authors (ML and EG). Infants with only febrile seizures or only neonatal seizures were excluded.

The incidence rates per 100,000 were calculated by dividing the number of observed cases by the number of children born in the area in 1997 through 2006 (data from Statistics Finland, http://www.stat.fi/).

For the included infants, data on prenatal and perinatal periods, clinical characteristics, all investigations (including imaging, electroencephalography EEG/video-EEG, metabolic studies, and genetic analysis) performed before the age of 24 months and outcome for seizure freedom, survival, and neurodevelopment until 24 months of age were abstracted from the hospital charts (Table S1). Seizure freedom at 24 months was defined as no observed seizures after age 18 months, with or without antiepileptic drugs. Neurodevelopment at 24 months was categorized as normal if the child was documented to have skills at a minimum at the 18-month level (developmental quotient ≥75), or delayed if performance was below the 18-month level. Categorization of seizure outcome and developmental data was based on a consensus between two authors (ML and EG). Data on whether the child had cerebral palsy were recorded. Normal outcome was defined as seizure freedom and normal neurocognitive development as described above at 24 months.

All video-EEG recordings and most EEG studies had been performed at the Children’s Hospital in Helsinki and analyzed by clinical neurophysiologists experienced in pediatric EEG or experienced pediatric epileptologists. A small minority of EEG recordings had been performed at the Jorvi Hospital. All included infants had at least one sleep EEG within the first year. One or several video-EEG studies lasting for 4–8 h had been recorded to confirm seizure types of 95 infants (60%).

All magnetic resonance imaging (MRI) studies had been conducted at the Helsinki University Hospital Department of Radiology and analyzed by an experienced pediatric neuroradiologist. Magnetic resonance imaging (MRI) at 1.0 or 1.5 T had been performed for 134 infants (85%). No MRI was available for 24 infants, one of whom was an infant with benign myoclonic epilepsy. For the others, etiology was revealed through other available information as explained below in context with the different syndromes.

Data on cognitive outcome at 24 months of age were based on Bayley Scales of Infant and Toddler Development, Second (1993) or Third Edition (2005), in 99 children (63%). For the 39 children who continued in follow-up at 24 months but had no structured evaluation, estimation of outcome was based on the neuropsychiatrist’s notes documented in the hospital chart.

Based on the data that had accumulated by the end of the second year of life, a reclassification of the epilepsy type or syndrome for every infant was carried out by two of the authors (ML and EG). We conducted the reevaluation process according to the latest ILAE recommendations. A syndromic diagnosis was confirmed only for those patients on whose data a consensus was achieved.

West syndrome (infantile spasms) was defined by infantile spasm clusters as the main or first seizure type, with either hypersarrhythmia or multifocal spikes on interictal EEG.

Benign infantile epilepsy was defined by seizure onset at 3–11 months; no spasms, myoclonic seizures, or status epilepticus; normal interictal EEG; normal development...
prior to seizure onset; absence of relevant abnormalities on neuroimaging or metabolic studies, seizure freedom since age 18 months; and normal neurodevelopment at 24 months.²⁰ Benign familial infantile epilepsy was defined by the same criteria with the additional requirement of a positive family history for afebrile infantile seizures with no structural etiology in at least one first-degree relative.

We used the following clinical criteria for Dravet syndrome: (1) lateralized or bilateral tonic–clonic seizures associated with fever, infection, or vaccination and normal interictal EEG in the first year; (2) therapy-resistant febrile and afebrile tonic–clonic seizures in the second year; (3) myoclonias or ataxia in the second year; and (4) normal MRI before the age of 24 months.²¹

Focal epileptic seizures were identified according to motor semiology described in the patient charts, such as focal or unilateral tonic–clonic activity or head and eye deviation before bilateral motor behavior. Video-EEG confirmation showing focal ictal discharge was required for hypomotor and other equivocal semiologies. Myoclonic seizures were always confirmed by ictal video or video-EEG.

Infants who had acute symptomatic seizures in the neonatal period followed by a seizure-free period and subsequent development of infantile spasms or other epileptic seizures in the first year were classified according to the symptoms and signs occurring after the acute phase.

Etiology was classified according to the ILAE recommendations¹³ into genetic, structural-metabolic, and unknown categories. Genetic etiology was defined by a known epilepsy-associated mutation with no metabolic or structural markers, or a family history of a first-degree relative with the same type of epilepsy (“familial”). Genetic testing was syndrome oriented. No gene panel testing or exome sequencing was done. For gene mutation and molecular karyotype analysis, we included results that had been obtained after the age of 24 months; all other etiologic information had been available before the age of 24 months. The unknown category was defined as normal findings on neuroimaging, metabolic, and genetic studies and negative family history. For the structural-metabolic group, we attempted to determine a specific etiology whenever possible.

Variable distributions between two groups were analyzed with use of the chi-square test. A stepwise regression analysis was done using Statistica software to assess the independent effects of age at onset and etiology on outcome. Analysis was done both forward and backward to check the stability of the model.

The study was approved by the ethics committee of the Helsinki University Central Hospital.

Results

The initial search into hospital statistics yielded 240 patients with a diagnosis of epilepsy before the age of 24 months and residency in the Helsinki University Hospital health care district. Review of the hospital charts confirmed the epilepsy diagnosis and seizure onset before the age of 12 months for 158 infants (58% male) who were included in the study. The number of infants at risk (i.e., those born in the study area in 1997 through 2006) was 127,730, giving an incidence rate of 124 of 100,000 for epilepsy in the first year of life.

The identified syndromes and specific structural-metabolic etiologies are presented in Tables 1 and 2.

West syndrome was confirmed in 34% of all infants, yielding an incidence of 41 of 100,000. One of these infants presented with Ohtahara syndrome at 7 weeks and evolved to West syndrome by the age of 3 months. Four infants developed infantile spasms after focal and two after unclassified seizures. Four infants, all with tuberous sclerosis, developed focal epileptic seizures after first having infantile spasms. In three infants, etiology was determined without MRI (all were imaged with computed tomography): neonatal group B streptococcal meningitis, severe hypoxic-ischemic encephalopathy, or 21 trisomy. The recognized genetic etiologies of West syndrome (5.7%) comprised of 21 trisomy (two patients) and of a deletion involving CDKL5 and SCML2 (one patient whose first seizure type was focal). All infants with West syndrome were treated with vigabatrin and 19 also with adrenocorticotropic hormone (ACTH).

Table 1. Electroclinical syndromes and etiologies based on the ILAE Commission Report 2005–2009¹³

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Structural-metabolic N (%)</th>
<th>Genetic N (%)</th>
<th>Unknown N (%)</th>
<th>Total N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>West syndrome²</td>
<td>31 (58)</td>
<td>3 (6)</td>
<td>19 (36)</td>
<td>53 (100)</td>
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<tr>
<td>Benign infantile epilepsy</td>
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<tr>
<td>Benign familial infantile epilepsy</td>
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<tr>
<td>Dravet syndrome</td>
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<tr>
<td>Epilepsy of infancy with migrating focal seizures</td>
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<tr>
<td>Myoclonic encephalopathy in nonprogressive disorders</td>
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<tr>
<td>Myoclonic epilepsy in infancy</td>
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<tr>
<td>Nonsyndromic epilepsy</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Focal seizures</td>
<td>22 (36)</td>
<td>11 (18)</td>
<td>28 (46)</td>
<td>61 (100)</td>
</tr>
<tr>
<td>Unclassified seizures</td>
<td>1 (20)</td>
<td>1 (20)</td>
<td>3 (60)</td>
<td>5 (100)</td>
</tr>
<tr>
<td>Total</td>
<td>55 (35%)</td>
<td>27 (17%)</td>
<td>76 (48%)</td>
<td>158 (100%)</td>
</tr>
</tbody>
</table>

¹³One infant presented with Ohtahara syndrome at 7 weeks and evolved to West syndrome by 3 months of age.

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Nine infants fulfilled the criteria described in Methods for benign infantile familial epilepsy and 19 infants those for benign infantile epilepsy. One additional infant who had only an ictal EEG showing a typical posterior discharge with bilateral spreading and only one cluster of seizures lasting for 5 days, was accepted in the nonfamilial group. MRI was not performed in 12 infants (6 familial) because of rapid remission of seizures and lack of any physical signs of intracranial pathology. The combined incidence for benign familial and nonfamilial infantile epilepsy was 22 of 100,000.

Six infants fulfilled the criteria for Dravet syndrome before age 24 months, giving an incidence of 4.7 of 100,000. All infants had been tested for SCN1A mutations and two turned out positive, yielding an incidence of SCN1A-mutation–positive Dravet syndrome of 1.6 of 100,000. One SCN1A-negative female patient had a PCDH19 mutation. The other three SCN1A-mutation–negative individuals were male.

Nonsyndromic epilepsy was observed in 42% of the infants. Most of them had focal seizures and unknown etiology (Table 1). There were eight infants without MRI: three with a positive family history but not fulfilling all criteria for benign familial infantile epilepsy at 24 months, one possible benign infantile epilepsy lost to follow-up, one with seizure onset before 2 months and excellent outcome, two with hypoxic-ischemic encephalopathy, and one with Wolf-Hirschhorn syndrome. Genetic etiologies included five familial cases, three SCN2A mutations, one CDKL5 mutation, one deletion involving SCN1A, SCN2A, and SCN9A, and one 21 trisomy. Seizure onsets in the infants with SCN2A mutations were 2 days (two infants) or 4 months (one).

The onset of epilepsy during the first year of life was most frequent in the first 7 months, especially at months 5–7 (42% of all onsets) (Fig. 1). Seizure onset at 8–11 months occurred in only 13% of the infants. We did not adjust age at onset for gestational age (7 children were born before 36 completed weeks of pregnancy). Structural-metabolic etiology accounted for 28 of (52%) 54 of the cases with onset during the first 4 months of life, while being less prevalent later in the first year (27/104, 26%) (p = 0.0012).

Outcome data for seizures and neurodevelopment until 24 months of age or death were available for 145 (92%) of 158 infants and are presented in Table 3. Of the 13 infants lost to follow-up, 8 with nonsyndromic (focal) epilepsy with unknown etiology had dropped out at age 12–21 months with excellent outcome when last seen. Five children had moved out of the health care area.

Seven patients (4.4%) died during the first 2 years of life. One patient with an SCN2A mutation died of SUDEP at 20 months after being seizure-free for 4 months. The other causes of death were pneumonia (two), a myocardial infarct associated with cardiomyopathy (one), or unknown (three infants with no autopsy).

Two infants underwent epilepsy surgery before age 2 years. One infant with perinatal middle cerebral artery infarct and infantile spasms became seizure-free after hemispherotomy at 18 months of age, but was developmentally delayed at 2 years. The other operation was a palliative
neocortical temporal lobe resection in an infant with tuberous sclerosis at 22 months. One additional infant had undergone surgery for a pilocytic astrocytoma.

The majority of infants (90/158, 57%) had normal cognitive development at 2 years and 48 (30%) were delayed (see Methods for definition). Cerebral palsy was observed in 24 (15%) of 158 children: 8 with hemiparesis, 5 with tetraplegia, 2 dyskinetic, and 9 undetermined. Three children (two hemiparesis, one ataxia) had normal cognition, 19 were delayed, and two had died before 24 months. Twenty infants (13%) had either died or were lost to follow-up (see above for details).

At 2 years, 91 (58%) of 158 children were seizure-free and 47 (30%) of 158 still had seizures (see Methods for definition of seizure freedom). In West syndrome, seizure freedom was achieved by all 19 children with unknown etiology. Normal cognitive development was more common in seizure-free children (73/91, 89%) than in children with continuing seizures (17/47, 36%) (p < 0.0001). Normal outcome was observed more rarely in infants with structural-metabolic etiology (12/52, 23%) compared to unknown or genetic etiology (61/93, 66% p < 0.0001), and in those whose seizure onset occurred in the first 4 months of life (16/54, 30%) compared to age at seizure onset after 4 months of age (57/104, 55%) (p = 0.0026). However, in regression analysis, etiology had a dominant effect on outcome, and age at onset was not a significant independent predictor of outcome (p = 0.8).

**Discussion**

The major strengths of our study are that it is population-based and that it includes a large number of patients compared to other population-based prospective or retrospective studies (Table 4). We estimate that almost all infants with epilepsy in the health district under study were identified, and that the data used to reevaluate the epilepsy types and etiologies are reliable as all infants were treated by neuropaediatricians at only two hospitals, and a consensus between two authors was reached for epilepsy syndrome, etiology, and outcome estimates. We also had a high rate of MRI and video-EEG studies, and were able to review all patient files and reports. Seizure outcome data are based on careful follow-up at the same hospital until 2 years of age, and almost two thirds had a structured evaluation of cognitive outcome. The major weaknesses of our study are the retrospective design and the short follow-up period. In addition, imaging may have missed some underlying lesions because of the short follow-up and the young age of the patients at the time of the MRI studies.

The overall incidence of epilepsy in the first year was somewhat higher than in most population-based studies (Table 4). Possible explanations for the higher detection rate in our study may be the homogeneous data sources, as explained earlier, good awareness of the neuropaediatricians at both hospitals for benign infantile epilepsy, and the fact that we included also infants whose epilepsy onset occurred in the neonatal period, unlike some other studies pointed out in Table 4. A high incidence was reported also from Iceland,1 where the explanation may be the inclusion of infants with isolated seizures, and from Germany,22 where only five infants in this age group were included in the study.

In 58% of our subjects, we could identify an infantile epilepsy syndrome acknowledged by the ILAE Commission on Classification and Terminology, 2005–2009.13 There are no...
previous studies applying the new recommendation on epilepsies with onset limited to the first year only for comparison. The proportion of infants and children with identified syndromes with onset within the first 2 years of life was lower than what we found in the first year. Perhaps more identifiable syndromes may have onsets in the first rather than in the second year or later.

To our knowledge, an incidence estimate (22/100,000) for benign familial and nonfamilial epilepsy of infancy has not been reported previously. These two syndromes were observed in 18% of our study population. Previously, the only population-based data come from a recent prospective United Kingdom study, where two nonfamilial cases were identified among 45 infants (4%). A Japanese hospital-based study estimated that up to 29% of their patients with epilepsy onset before 24 months of age had benign partial epilepsy of infancy, which probably is the same syndrome as benign infantile epilepsy in the new terminology. The diagnosis made at 24 months may be held reliable, as a longer follow-up study until 5 years of age has shown that 90% of children fulfilling the criteria for benign infantile epilepsy at 2 years continue to do so at 5 years. The true incidence may be slightly higher than found in our study, as eight infants with possible benign infantile epilepsy dropped out of follow-up while having normal outcomes at 12–21 months of age.

We chose to combine the familial and nonfamilial cases of benign infantile epilepsy for incidence calculations because the true proportion of familial cases remained uncertain. Our criterion for familial etiology was very strict (afebrile infantile seizures with no structural etiology in at least one first-degree relative) and we relied on the notes made in the patient files. The family history may not always be sufficiently documented for various reasons, and a systematic study of the pedigrees might reveal more familial cases.

The incidence and etiologies of West syndrome in our study are similar to what has previously been reported from Finland and the United Kingdom.

We identified six patients with the clinical characteristics of Dravet syndrome, but only two with SCN1A mutations, giving an incidence of SCN1A-mutation–positive Dravet syndrome of 1.6 of 100,000. This is lower than what was estimated in the United Kingdom (2.4/100,000) and in Denmark (4.5/100,000), perhaps because the children in our study were tested before the latest developments in gene-sequencing techniques. All of our patients with the Dravet phenotype and also the more severely affected patient with a deletion involving SCN1A, SCN2A, and SCN9A, benefited from the combination of stiripentol, valproate, and clobazam shown to be effective in Dravet syndrome, irrespective of their mutational status.

The incidence of epilepsy of infancy with migrating focal seizures (1.6/100,000) was higher than the estimate from a nationwide email-based query in the United Kingdom (0.26–0.55/100,000), possibly due to chance or our good coverage of all infants with epilepsy in our region.

In nonsyndromic epilepsy, the most common underlying etiologies were structural-metabolic (35%) and unknown (47%), in line with Eltze et al. As discussed earlier, it is possible that the unknown etiology group includes some children with benign infantile epilepsy but insufficient follow-up to confirm the diagnosis. The early clinical presentations of the three children with SCN2A mutations resembled benign familial neonatal-infantile seizures, not yet recognized as a syndrome in the new ILAE organization of epilepsies. Two of them described in more detail elsewhere presented with episodic ataxia during the second year of life.

The total proportion of structural-metabolic etiology was lower in our study (35%, see Table 1) than in a previous retrospective cohort with seizure onset before 1 year of age (54%). A recent prospective study also found more structural-metabolic etiology; 44% of infants with seizure onset
at 0–24 months. The differences may be due to a more comprehensive inclusion of infants with benign infantile epilepsies in our study.

The proportion of genetic etiology in our study was slightly less than the 22% reported by Wirrell et al. but higher than the 7% reported by Eltze et al. This is probably due to methodologic differences; Wirrell et al. included patients up to 17 years of age and classified etiology as genetic also in patients who had an epilepsy syndrome known to have a strong genetic contribution, regardless of family history, whereas Eltze et al. only reported identified chromosomal or gene abnormalities. The clinical presentation of the child with several deleted sodium channel genes was similar to cases described by Lim et al.

Overall mortality in our study (4.4%) was lower than what was observed by Moseley et al. and Datta and Wirrell (7%). This was probably due to inclusion of more children with benign epilepsies and a shorter follow-up period. Mortality in West syndrome before 2 years of age was similar to that reported by Riikonen before age 3 years. The only child with SUDEP in our study had an SCN2A mutation. SCN1A, SCN5A, and SCN8A mutations have been associated with an increased risk of SUDEP, but we are not aware of any previous reports implicating SCN2A mutations in sudden death.

To our knowledge, this is the first population-based study to report outcome of infantile epilepsy with seizure onset between birth and 12 months of age. In line with previous hospital-based series of infants with similar age at seizure onset, we found that structural-metabolic etiology is significantly associated with poor outcomes compared to unknown etiology.

Normal outcomes of West syndrome with unknown etiology were slightly more common (89%) in our study than in previous population-based studies from Finland (72%) and Sweden (67%). This difference is probably explained by better availability of MRI making the unknown etiology group more homogeneous in our study. In addition, all infants with suspected seizures in our study region were seen by a pediatric neurologist, probably leading to shorter diagnostic and therapeutic delays. In contrast, normal outcomes in the structural-metabolic (“symptomatic”) etiology group were observed in <20%, both in the older studies and the present study. The striking difference between the unknown and the structural-metabolic etiology groups suggests that the unknown etiology group may contain yet undiscovered genetic etiologies associated with good prognosis. This was suggested earlier by Dulac et al. and Vigevano et al., who identified a subgroup of “idiopathic” West syndrome characterized by positive family history for epilepsy or febrile seizures, lack of focal abnormalities on EEG, later development of genetic EEG traits, and a good prognosis.

It is important to note, however, that the assessment of cognitive level at age 24 months carries many uncertainties. Mild mental deficiency may be missed at that early age, and later deterioration may occur in children with drug resistant epilepsies such as Dravet syndrome.

Both Chevrier and Aicardi and the present study agree that the first seizure in infantile epilepsy occurs more often before the age of 6 months than later in the first year. Our results (Fig. 1) suggested that the frequency of seizure onsets continue on a high level through the age of 7 months and declines sharply after that age.

We and others have observed that onset of epilepsy within the first 3–6 months of life carries a worse prognosis than later onset in the first year. However, we also showed that onset in the first 4 months of life is significantly associated with structural-metabolic etiology, which is a known predictor of poor outcome, and that age at onset does not have a significant independent effect on outcome when etiology is controlled for.

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**Disclosure of Conflict of Interest**

Eija Gaily has received lecture honoraria from UCB Pharma, Eisai, and Orion Pharma and served in the scientific advisory board for Eisai. None of the other authors have any conflicts of interest to report. 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.

**References**


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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Table S1. Data collected from the hospital charts.