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Incidental rolandic spikes: Long-term outcomes and impact of treatment

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ABSTRACT

We describe a group of 26 children with no prior history of seizures consistent with benign rolandic epilepsy who had rolandic spikes found coincidentally on EEG. A retrospective chart review as well as phone and email follow-ups with families were completed to assess long-term outcomes. A subset of this group (n = 7) with reported comorbid language or learning difficulties was then given an empiric trial of levetiracetam. Seven (27%) children eventually developed seizures, with a median of 14 months after the abnormal EEG. Of the 7 children ever treated with levetiracetam, 5 exhibited beneficial effects on learning, speech, or behavior. Side effects reported were mild and included irritability and headache. Incidental rolandic spikes may represent a discrete neurologic condition, with approximately one-quarter of the patients later developing epilepsy. Some of these children may experience improved intellectual functioning with levetiracetam.

1. Introduction

Benign rolandic epilepsy (BRE) is one of the most common pediatric epilepsy syndromes. It is characterized by hemifacial motor seizures and interictal centrotemporal (rolandic) spikes on EEG that are activated by drowsiness or sleep. These rolandic spikes are not specific to BRE, however, and can occur in 2–3% of healthy school-aged children, with fewer than 10% of them going on to develop seizures [1,2]. Additionally, these patients may exhibit cognitive deficits even without clinical epilepsy [3].

Rolandic spikes have also been found in 5–6% of patients with ADHD and in up to 20% of patients with autism spectrum disorders (ASDs) [4,5]. This subset is diagnosed at a younger age and has worse response inhibition scores on neuropsychological testing for patients with ADHD than for patients with ADHD without rolandic spikes [5]. The association appears to be true in the opposite direction as one study reported 65% of patients with BRE having ADHD [6]. For patients with ASDs, the presence of EEG abnormalities does not appear to play a role in autistic regression [4,7]. However, some data suggest that there may be a relationship between epileptiform EEG activity and isolated language regression [8]. Altogether, there is no definitive consensus on the relevance of probable incidental rolandic spikes in these different groups of patients or, more importantly, the management of these patients when they present to clinic.

It has been well described in patients with BRE that even those ASDs or ADHD are at a high risk of cognitive deficits, particularly involving language and memory [9,10]. In this patient population, neither the seizure frequency nor the severity nor the sidedness of the rolandic spikes clearly correlates with cognitive outcomes [3,9]. However, higher rolandic spike frequency has been associated with worse cognitive outcomes [3]. Although there is evidence that normalizing EEG parameters improves cognitive function in these patients, this is controversial [11].

The use of anticonvulsant therapy for patients with BRE is generally recommended if there is early onset of the disease, if there are multiple seizures at onset, or if there is a high seizure frequency [12]. However, management of patients with BRE who do not meet these criteria but exhibit cognitive deficits remains unclear. Historically, the most commonly used anticonvulsants for BRE have been carbamazepine and oxcarbazepine [13]. More recently, levetiracetam has been found to be both safe and effective for patients with BRE [14]. There is also evidence that not only does levetiracetam help control seizures, but it also helps a small cohort of patients with BRE improve auditory comprehension and verbal memory after a 12-month trial [15]. Additionally, preliminary data suggest that treating patients with rolandic spikes without epilepsy but with comorbid ADHD or learning disorders with sulthiame for 12 months improves attention and acquisition of words [16].

While both of these studies are small and preliminary, they raise the possibility that using anticonvulsants for children with cognitive deficits who have incidentally found rolandic spikes on EEG may be helpful. In the current study, we describe a group of children with no history of seizures consistent with BRE who had rolandic spikes found incidentally on EEG. A subset of this group who also had language disorders was offered a trial of levetiracetam.

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2. Methods

2.1. Chart review

Medical charts and electronic records of consecutive children with incidentally found rolandic spikes on EEG who were evaluated in child neurology clinics at The Johns Hopkins Hospital since 2009 were reviewed. These children were identified primarily from the epilepsy clinic of a single neurologist (EK) because of his interest in BRE and cognition. In addition, all sleep study EEGs at The Johns Hopkins Hospital, a common source of these incidental spikes, are currently read by this neurologist. None of the children, except one child (with 2 prior febrile seizures), had any preceding history of seizures. Twenty-six patients were included in the study. From each patient's chart, we noted the patient's age when the EEG was obtained, reason for obtaining the EEG, gender, family history, neuroimaging results, development, and medication use.

2.2. EEG criteria

Electroencephalogram results for all patients were reviewed by EK. The presence of rolandic spikes was determined by their shape, biphasic sharp waves, or spikes. They could be isolated or in clusters, unilateral, or bilateral. The discharges were seen predominantly in sleep. No patients had other epileptiform discharges, and all had an otherwise normal background.

2.3. Longitudinal follow-up

Families were contacted by phone or email for interval updates. If they agreed to participate in the study, questions were then asked about occurrence of seizures, headaches, medication use, new medical conditions, repeat EEGs and brain imaging, and school performance. We used a script approved by the Johns Hopkins institutional review board.

3. Results

3.1. Demographics

Twenty-six patients were included in our study. Demographic details are summarized in Table 1. The most common reason for obtaining the EEG was as part of a sleep study due to concerns for sleep apnea or due to snoring (10/26, 38%). The next most common indication was staring spells that turned out not to be actual seizures (9/26, 35%). See Table 2 for details of the remaining indications for EEG.

Table 1
Cohort demographics (n = 26).

Age when EEG was done, years (median, range)	6 (3–9)
Interval from EEG studies to clinic visit, months (median, range)	3 (1–24)
Male gender (%)	18 (69%)
Family history of seizures (%)	5 (19%)
Developmental delay (%)	14 (53%)
ADHD (%)	5 (19%)
Migraine headaches	1 (4%)
Neuroimaging	
Normal	14 (54%)
Abnormal[a]	3 (11%)
None	9 (35%)
Reason for obtaining the EEG	
Sleep study for apnea	10 (38%)
Staring spells	9 (35%)
Psychiatric disturbance	3 (11%)
Developmental delay workup	2 (8%)
Control in a research study	1 (4%)
Complex febrile seizures	1 (4%)

[a] No clinical correlation with EEG findings. Findings included the following: asymmetric lateral ventricles, absent septum pellucidum, and small white matter hyperintensities along the lateral ventricle.

3.2. Development of epilepsy

Our cohort was followed for at least 6 months after each patient's first abnormal EEG, with an average follow-up duration of 2.6 years (Table 2). Seven (27%) patients eventually developed seizures. Onset of seizures from the time of the initial abnormal EEG ranged widely in these 6 children, from 5 to 46 months, with a median of 14 months. Three patients developed seizures consistent with BRE (e.g., nocturnal hemifacial partial seizures), which occurred 15, 16, and 24 months after their abnormal EEGs. Of the additional 4 patients, one developed recurrent febrile seizures, one developed idiopathic generalized epilepsy, one developed idiopathic (not BRE) complex partial seizures, and one developed generalized tonic–clonic seizures in the setting of repeat EEGs concerning for electrical status epilepticus during slow-wave sleep. This EEG was significantly worse than EEGs with rolandic spikes years prior.

3.3. Anticonvulsant management

Eleven patients have been started on anticonvulsants to date. See Table 2 for case details. Four were started on anticonvulsants primarily because of the eventual occurrence of clear seizures. Of these, one patient with generalized epilepsy and significant psychiatric comorbidities has since been treated with oxcarbazepine, valproic acid, and lamotrigine (*Case 2*). The second patient was started on oxcarbazepine because of seizures consistent with BRE that were happening regularly (three times in one year) in the setting of sleep deprivation during travel (*Case 7*). The patient had no further seizures after oxcarbazepine was initiated. The third patient developed idiopathic complex partial seizures (not BRE) and has been treated with both oxcarbazepine and topiramate (*Case 8*). The fourth patient had recurrent febrile seizures that were controlled with levetiracetam and have not recurred (*Case 9*). Of note, this patient also had coexisting learning difficulties at school which is discussed further below.

A single patient was treated with valproate, which was started at an outside institution for staring spells and falls which were not seizures in retrospect, nor did they improve with treatment (*Case 25*). In addition, this child's autistic features did not improve either, and valproate was, therefore, discontinued at our center after 2 years of treatment without incident. Interestingly, 2 months after discontinuation of valproate, he had a classic BRE seizure (nocturnal, hemifacial, and brief), and valproate was, therefore, restarted. The remaining six patients without seizures were started on levetiracetam primarily for the purpose of attempting to improve cognitive function.

A total of seven patients were treated with levetiracetam (including the patient with febrile seizures noted above), of which 5 (71%) experienced a beneficial effect per parent report. One patient had improved school performance, better sleep, and decreased episodes of nocturnal enuresis (*Case 5*). One and a half years later, he was trialed off of levetiracetam with recurrence of symptoms, so it was restarted, and he has continued it ever since. The second patient saw improvement in her school performance in addition to cessation of her febrile seizures (*Case 9*). She remained on levetiracetam for 3 years before being tapered off and has not developed BRE to date. The third patient had improvement in her speech in addition to better sleep (*Case 12*). She was tapered off of levetiracetam after a year without worsening of symptoms. The fourth child had improvement in her speech delay (*Case 20*). Three years later she had convulsions and was found to have electrical status epilepticus of sleep. She was briefly transitioned off of levetiracetam and put on valproic acid; however, because of better language, she was placed back on levetiracetam, which she continues now, and her EEG has normalized. The fifth patient had rapid improvement in fatigue and school performance and remains on 20 mg/kg/day