J Autism Dev Disord (2014) 44:452–458
DOI 10.1007/s10803-013-1888-6

ORIGINAL PAPER

Incidence and Behavioral Correlates of Epileptiform Abnormalities in Autism Spectrum Disorders

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Published online: 20 July 2013

Abstract Autism spectrum disorders (ASD) are associated with an increased incidence of epilepsy and of epileptiform discharges on electroencephalograms. It is unknown whether epileptiform discharges correlate with symptoms of ASD. We completed a retrospective chart review of 101 patients with ASD who had overnight electroencephalograms. We looked for a relationship between epileptiform abnormalities and diagnosis, history of regression, communication skills, and other features associated with ASD. There was a higher incidence of epileptiform activity in children with stereotypies and aggressive behavior. The incidence of epileptiform abnormalities was significantly lower in Asperger's compared with more severe forms of autism. Results suggest that increasing severity of autistic symptoms may be associated with higher likelihood of epileptiform abnormalities. Whether treatment alters outcome is unknown.

Keywords Autism · ASD · Epilepsy · EEG · Behavior

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Introduction

Autism spectrum disorders (ASD) are a group of behaviorally defined disorders characterized by abnormal language and social interaction, as well as repetitive behaviors or inflexibility of interests. ASD can affect individuals in varying degrees and combinations, leading to a spectrum of related disorders which include Asperger syndrome, autism, and pervasive development disorder not otherwise specified (PDD-NOS) (American Psychiatric Association: Diagnostic and Statistical Manual IVth Edition 2000). While it is deficits in these core domains of behavior that characterize ASD, patients with these disorders may experience a large number of other types of problems including difficulty sleeping, and behavioral disturbances such as aggression, impulsivity, tantrums, and attention problems.

It is estimated that between 5 (Bryson et al. 1988) and 46 % (Hughes and Melyn 2005) of patients with ASD also have epilepsy, compared to a prevalence of 2–3 % in the general population (Tuchman and Rapin 2002). The significance of the relationship between epilepsy and ASD remains unclear. Some studies have shown a relationship between severity of intellectual deficiency and prevalence of epilepsy (Hara 2007; Hrdlicka et al. 2004) but a recent review points out that little is known about the relationship between ASD and epilepsy in the absence of intellectual disability (Berg and Plioplys 2012). There may also be a connection between developmental regression and epilepsy, but the results have been mixed on this topic as well (Spence and Schneider 2009).

Even individuals with ASD without a history of epilepsy may have abnormal electroencephalogram (EEG) activity, including epileptiform discharges. Estimates of the prevalence of epileptiform abnormalities in individuals with

ASD not diagnosed with epilepsy are variable, but range from 6.7 (Giovanardi Rossi et al. 2000) to 59 % (Kim et al. 2006). These epileptiform discharges are thought to be evidence of cerebral dysfunction, but whether or not they are responsible for autistic features is unknown. There is evidence that frequent epileptiform discharges in children with epilepsy correlates with transient cognitive impairment (Aldenkamp and Arends 2004). In addition, sleep-activated epileptiform activity with or without the presence of seizures is associated with loss of previously acquired language in Landau-Kleffner syndrome (LKS). Children with LKS may also have an autistic-like regression that extends to behaviors beyond language (Deonna and Roulet-Perez 2010). 30 % of children with ASD experience a regression of previously learned skills (Goldberg et al. 2003) and it is possible that in ASD, like LKS, epileptiform abnormalities may have an association with language deficits or regression.

There have been a few studies that looked at the prevalence of EEG abnormalities in different subsets of individuals with ASD. In regard to developmental regression, the results have been mixed with some studies reporting a significant correlation with the presence of epileptiform abnormalities (Baird et al. 2006; Tuchman and Rapin 1997) and others not (Chez et al. 2006). Though one study found a higher prevalence of epileptiform abnormalities in individuals with autism with a lower IQ (Yasuhara 2010), other studies have reported that intellectual deficiency is not associated with epileptiform abnormalities but is associated with a higher likelihood of seizures (Hrdlicka et al. 2004; Rossi et al. 1995). Another study looking at sleep showed that sleep disruption does not seem to be associated with epileptiform activity on EEG (Giannotti et al. 2008).

Few studies have attempted to correlate behaviors with epileptiform abnormalities. One study found no difference in behaviors or severity of autistic features in individuals with ASD with epileptiform abnormalities according to surveys, though those with seizures had significantly worse behaviors than those without seizures (Hartley-McAndrew and Weinstock 2010). In another recent study of patients with epilepsy and ASD, there was a non-significant trend for patients with severe EEG changes to have more neuropsychological problems in general (behavior problems, sleep disorders, ADHD, etc.) than those with less severe EEG changes (Lee et al. 2011).

In the current study, the aims were to learn more about the prevalence of epileptiform abnormalities in patients with ASD, and whether or not specific behavioral characteristics correlate with the presence or absence of epileptiform abnormalities. The method was a retrospective chart review of all children with an ASD diagnosis who had undergone an overnight EEG at our institution. Although this approach sets limits on the type of information we can obtain, it is nonetheless a useful tool that may be important for both clinical counseling of families and also for consideration of certain types of interventions such as a trial of anti-epileptic medication.

Methods

Patients

Records from patients with an ASD seen by a single pediatric neurologist and who had a non-sedated overnight EEG done between July 2003 and July 2010 were reviewed retrospectively. The pediatric neurologist is board-certified in neuro-developmental disabilities, and her practice is almost exclusively focused on autism and related neuro-developmental disorders. Children are referred to this clinician for diagnostic purposes as well as for treatment recommendations. It is the practice of this clinician to obtain an overnight EEG on all new patients who have a diagnosis of autism spectrum disorder.

All patients were diagnosed to have an autism spectrum disorder according to DSM-IV criteria. Records were de-identified before information was entered in a database for analysis. This study was performed in accordance with the procedures of the Institutional Review Board, which gave ethical consent.

EEG Testing

All EEGs were performed at a single Children's Hospital and were either ambulatory (64 patients) or in-patient video EEGs (37 patients). The EEGs were recorded for at least 24 h, and all EEGs including an extended period of natural sleep (sleep was never induced). Ambulatory EEGs were done with the 1,800+ recording system and video EEGs used the XLTEK digital EEG and digital video recording system. All EEGs were read and interpreted clinically by board-certified pediatric electroencephalographers. Since the EEGs were conducted for clinical purposes, the electroencephalographers were not blinded to the diagnosis. The authors did not have access to the raw EEGs, but utilized the dictated EEG reports. The following information was extracted from the EEG reports and is summarized in Table 1: whether any antiepileptic drugs were being taken at the time of the EEG, whether the EEG was read as normal or abnormal, presence of abnormal slowing, and presence of epileptiform discharges. If epileptiform discharges were present, the location, frequency (rare, recurrent, or frequent), and timing (wakefulness, sleep, or both) were noted. In general, if epileptiform abnormalities were present in both wakefulness and sleep, they were more common in sleep.

Table 1 Variables recorded from the physician's note and EEG report

Pediatric neurologist notes	EEG report
General characteristics	Impression (normal or abnormal)
Age; sex; diagnosis	Abnormal slowing
Communication	Epileptiform discharges
Expressive language (very limited, some, fair); receptive language (none, some, good); echolalia; scripted language	Frequency (rare, recurrent, frequent); timing (sleep, wakefulness, or both); location
Social interaction	Medications
Eye contact; pretend play (none, emerging, some); interactive play (none, parallel, some interactive)	Antiepileptics; antipsychotics
Inflexible/repetitive behaviors	
Stereotypies; difficulty with transitions; picky eater	
Behavioral disturbances	
Aggression; impulsivity; attention problems	
Other	
History of seizures; sleep disturbances; regression	

Patient Characteristics

As summarized in Table 1, a variety of information was recorded from the physician's initial note and follow up notes including the age, sex, and diagnosis of the patient. Past medical history of seizures was noted and the presence of regression or the loss of any previously acquired skills (eye contact, pointing, language, etc.) was determined from the history. An index of the patient's expressive language abilities was recorded according to the following categories: very limited speech (no words or single words), some speech (2–3 word phrases or some sentences), or fair speech (normal or mostly normal). Patients with "mostly normal" speech included those with pragmatic language deficits. Receptive language was categorized in a similar way: none, some, or good. The presence or absence of echolalia and scripted language was also recorded if the information was available. Data about social interactions were recorded according to the history about eye contact (decreased or good), pretend play (none, emerging, some), and interactive play (none, parallel play only, some interactive play). Presence of stereotypies, problems with transitions or change of routine, tactile sensitivity, whether or not the patient was a picky eater were recorded as an indicator of the patient's inflexibility. The presence of behavioral disturbances such as aggression, impulsivity, and attention problems were recorded along with the presence of sleep disturbances. All characteristics were recorded only if it was specifically indicated in the physician's note and were left blank if there was no mention of a specific characteristic.

Statistics

Descriptive statistics were compiled from the database and included mean age at EEG, and the frequency and timing of epileptiform abnormalities. Chi square analysis was done to assess if there was any association between specific patient characteristics (e.g. history of seizures, expressive language function, etc.) and the presence and frequency of epileptiform abnormalities. In some cases, the numbers of individuals for which certain types of information was recorded were insufficient to do a comparison between groups in a Chi square analysis. For these characteristics (aggressive behavior, impulsivity, and attention problems), a one-sample Chi square goodness of fit test was done to test if that group was significantly different from the expected frequency of epileptiform abnormalities as determined implicitly from the overall frequency of epileptiform abnormalities in our population.

Results

One hundred one patient files were reviewed (78 males and 23 females; ratio 3.4:1). The age at the time of EEG ranged from 1.68 to 18.32 years (mean 7.06 $\pm$ 3.74 years). Overall, 66.3 % of patients with ASD had some type of EEG abnormality (e.g. slowing or epileptiform discharges), with epileptiform abnormalities occurring in 59.4 % and non-epileptiform abnormalities (i.e. slowing) in 21.8 % of patients. No individual in this study had electrical status epilepticus during sleep (*ESES*) or continuous spike-wave discharges during sleep (CSWS).

A history of seizures was present in 23.1 % of 91 patients for whom a history of seizures was recorded. When comparing individuals with ASD with and without seizures, patients with a history of seizures were significantly older at the time of EEG compared to patients without a history of seizures (Table 2). Epileptiform abnormalities occurred in 95.2 % of individuals with ASD with a history of seizures and 50 % of patients without a history of seizures ($p < 0.01$).

Information about the frequency of epileptiform abnormalities was present for 55 patients and showed that when present, epileptiform abnormalities were rare in 56.4 % of patients, recurrent in 27.3 %, and frequent for 16.4 %. Timing information was available for 56 patients with epileptiform abnormalities and of those, 58.9 % of patients had epileptiform abnormalities in sleep only, 37.5 % of patients had epileptiform abnormalities in both wakefulness and

Table 2 Characteristics and incidence of epileptiform abnormalities in patients with and without a history of seizures

	Overall (n = 101)	History of seizures (n = 21)	No history of seizures (n = 70)
Mean age at EEG (years ± SD)*	7.06 ± 3.74	9.57 ± 3.43	6.53 ± 3.88
Epileptiform abnormality**	59.4 %	95.2 %	50.0 %

* Represents significant difference $p < 0.01$ (independent t test); ** Represents a significant difference $p < 0.01$ (Chi square)

Table 3 Incidence of seizures and EEG abnormalities in patients grouped by diagnosis

* Represents significant difference $p < 0.01$ (Chi square)

	Asperger (n = 10) (%)	Autism (n = 75) (%)	PDD-NOS (n = 16) (%)
Seizures	0	22.1	40.0
Epileptiform abnormalities*	20.0	60.0	81.3
Abnormal slowing	20.0	21.3	25.0

sleep, and 3.6 % patients had epileptiform abnormalities that occurred in wakefulness only. When epileptiform abnormalities occurred in both wakefulness and sleep, they were generally more common in sleep than wakefulness. There was no predilection for specific locations of the epileptiform abnormalities. The most frequent location was frontal, though all locations were represented and many patients had multifocal or generalized epileptiform abnormalities.

Table 3 shows the prevalence of EEG abnormalities by diagnosis. There was a significant interaction between diagnosis and prevalence of epileptiform abnormalities, with Asperger's patients having the lowest prevalence of epileptiform abnormalities (20.0 %) compared to 60.0 % in autism and 81.3 % in PDD-NOS ($p < 0.01$). There was no difference in non-epileptiform abnormalities (focal or generalized slowing) associated with diagnosis.

The prevalence of epileptiform abnormalities (with or without clinical seizures) in association with specific symptoms of ASD is summarized in Table 4 and organized by domains of behavior including communication, social interaction, inflexibility of behavior, and other features of autism. A history of aggression was associated with a high incidence of epileptiform abnormalities (75 % compared to 0 %; $p < .05$), but not with the presence of clinical seizures. Motor stereotypies (hand flapping, spinning) were present in 69 of 80 patients for whom the information was recorded, with 60.9 % of patients with stereotypies having epileptiform abnormalities compared to 36.4 % of patients without ($p = .044$).

No other variables examined differed significantly relative to the presence or absence of epileptiform EEG abnormalities. These included language ability, social interaction, sleep problems, and history of regression. Furthermore, when a subset of children without a history of clinical seizures was analyzed, no differences were found beyond what had been seen in the entire group.

Six patients (5.9 %) were identified as having EEG features similar to those found in LKS with sleep-activated frequent to persistent bilateral centrotemporal epileptiform discharges. One patient had a history of seizures and none of these patients had a known history of regression. In general, most of these patients had some deficits in expressive and receptive language, stereotypies were present, and they had decreased eye contact.

Discussion

The primary goal of this study was to determine if there is a relationship between epileptiform abnormalities and specific behavioral characteristics of ASD. To ensure that results were not driven by individuals with ASD with a history of seizures, a subset of the population that only included individuals with ASD without seizures was also analyzed.

A secondary goal was to determine the prevalence of epileptiform abnormalities during prolonged non-sedated EEG recordings in a large population of patients with ASD. Similar to previous studies, we found a high incidence (23 %) of epilepsy in individuals with autism, and the majority of patients in the study (59.4 %) had epileptiform abnormalities. Those patients with seizures had almost uniformly (95 %) abnormal epileptiform EEGs, while half of the patients without a history of seizures had epileptiform abnormalities. Previous studies have documented epileptiform abnormalities in individuals with ASD without seizures, although the reported prevalence has varied widely between studies from 6.7 (Giovanardi Rossi et al. 2000) to 60.7 % (Chez et al. 2006). The high prevalence of epileptiform abnormalities in our study may be related to the fact that all of our patients had prolonged EEG recordings which captured wakefulness and a full night's sleep, as opposed to a sedated EEG or routine EEG. In fact, 58.9 % of all patients with epileptiform abnormalities had them in sleep only. Other studies that have used 24 h recordings

Table 4 Incidence of epileptiform abnormalities in core domains of ASD for all patients

Domain	Characteristic	Percent with epileptiform abnormalities
Communication	Expressive language	
	None or very limited (n = 37)	54.1
	Some (n = 34)	73.5
	Fair or normal (n = 30)	50.0
	Receptive language	
	None (n = 16)	56.3
	Some (n = 60)	63.3
	Good (n = 25)	52.0
	Echolalia	
	Present (n = 22)	59.1
	Absent (n = 30)	56.7
Social interaction	Eye contact	
	Decreased (n = 65)	56.9
	Good (n = 23)	65.2
	Pretend play	
	None (n = 26)	65.4
	Emerging (n = 21)	57.1
	Some (n = 9)	33.3
	Interactive play	
	None (n = 25)	64.0
	Parallel play (n = 29)	55.2
	Some interactive play (n = 12)	58.3
Inflexibility	Stereotypies	
	Present (n = 69)	60.9
	Absent (n = 11)	36.4
	Difficulty with transitions	
	Present (n = 19)	47.4
	Absent (n = 10)	60.0
	Picky eater	
	Present (n = 34)	58.8
	Absent (n = 23)	56.5
Other common features in ASD domain	Regression	
	Present (n = 39)	51.3
	Absent (n = 57)	62.6
	Sleep disturbances	
	Present (n = 39)	51.3
	Absent (n = 22)	63.6
	Aggression[a]	
	Present (n = 41)	75.6
	Absent (n = 7)	0
	Impulsivity[a]	
	Present (n = 16)	50.0
	Absent (n = 2)	0

The number of patients for which the information was available is indicated in parentheses

[a] analysis by Chi square goodness of fit

also found high prevalences of epileptiform discharges in patients without seizures (Kim et al. 2006; Chez et al. 2006). Our results, along with the data from these studies, suggest it may be necessary to use a longer recording time to truly assess the presence of epileptiform abnormalities in individuals with ASD.

A key finding of this study was that the prevalence of epileptiform abnormalities differed significantly by

diagnosis of ASD, the first time this type of difference has been described. Asperger's patients had the lowest prevalence of epileptiform abnormalities at 20.0 %, compared with much higher rates in autism (60.0 %) and PDD-NOS (81.3 %). One previous study found no difference between autism and PDD-NOS in 1 h EEGs, but did not include patients with Asperger's (Ekinci et al. 2010). Asperger's is often considered a "milder" form of autism due to the fact that these individuals have normal intelligence and normal propositional language. The low prevalence of epileptiform abnormalities in Asperger's patients may be a reflection of the general severity of disease rather than a specific cause-and-effect phenomenon. A previous study showed that patients with epileptiform abnormalities did not differ in scores on the Childhood Autism Rating Scale compared to those with normal EEGs; however, patients were not compared by diagnosis across the autism spectrum (Hartley-McAndrew and Weinstock 2010). Asperger's is differentiated from other ASDs by the preservation of normal propositional language. While we did not find any association between language and epileptiform abnormalities in this study, it is possible that a more quantitative measure of language might reveal a relationship with epileptiform abnormalities. In addition, some studies have shown that decreased intellectual ability is correlated with increased epileptiform abnormalities (Yasuhara 2010). Individuals with Asperger syndrome have intellectual levels in the normal and even superior range. While we were not able to evaluate this relationship in our study due to lack of information about IQ levels, it is possible that our differences in EEG findings by diagnosis could be explained by differences in cognitive functioning, and we cannot rule out this as an explanation.

Another key finding was that epileptiform abnormalities were associated with a higher incidence of aggression and of motor stereotypies. Although we found no reports of interictal abnormalities in children being related to aggression, there have been other reports of a link between aggressive behaviors and frontal lobe epilepsy (Sumer et al. 2007; Shih et al. 2009). Other studies have shown behavioral differences in children with cryptogenic epilepsy (e.g., Saskia et al. 2009), but aggressive behavior is not one of the more common issues cited in many of the epilepsy studies. However, since there is some evidence that aggression may respond to treatment with antiepileptic medications (Hollander et al. 2010), this finding warrants further investigation.

A small number of patients in the present study had EEG abnormalities that were similar to those seen in LKS (Deonna and Roulet-Perez 2010). These patients had sleep-onset persistent bilateral independent central and/or temporal epileptiform discharges, yet clinically resembled individuals with ASD. There was no history of regression in these patients and in general, they had deficits in language, social interactions, and inflexibility of behavior. There is a potential overlap between LKS and ASD as many children with LKS may also have an autistic-like regression that extends to behaviors beyond language (Deonna and Roulet-Perez 2010) and children with ASD may experience a regression of previously learned skills including language (Goldberg et al. 2003). While the significance of these types of EEG abnormalities is unclear, our finding that some individuals with ASD have EEG abnormalities similar to those in LKS suggests that there may be a subset of patients with ASD that have a similar mechanism of disease to LKS. It is unknown if the six patients identified in this study were subsequently treated with steroids or antiepileptic medications, though it is possible that this subset of patients may respond to such treatments that have been used with some success in LKS (Sinclair and Snyder 2005).

One of the limitations of this study is that quantitative data regarding diagnosis and symptoms of patients with ASD were not available. The diagnosis of ASD was determined clinically without the aid of diagnostic tools such as the Autism Diagnostic Interview or the Autism Diagnostic Observation Schedule (ADOS) and language skills were assessed generally during a clinical visit with a pediatric neurologist who specialized in neuro-developmental disabilities. This study would be strengthened by the addition of formal language testing to provide more sensitivity and specificity for language deficits which might show a clearer relationship with epileptiform abnormalities. In addition, formal IQ testing would provide information about intellectual ability which may explain the differences in prevalence of epileptiform abnormalities by diagnosis. While we did review medical records from a large number of patients, not every patient had recorded information about each characteristic of interest. As a result, our sample sizes for some analyses were smaller than the number of files reviewed, and our ability to detect differences may have suffered as a result. Future studies would benefit from a prospective study with more quantitative information such as the ADOS, formal language testing, and IQ testing to correlate with differences in EEG abnormalities. Quantification of the frequency of epileptiform discharges will also be important for future studies. Furthermore, we may have underestimated the prevalence of epileptiform abnormalities in the ASD population because of the younger age at which many children were tested. The children with seizures were tested at a higher mean age than those without seizures, and it is possible that epileptiform abnormalities may appear later in childhood for some children on the autism spectrum.

In conclusion, this study provides new information about the relationship between ASD and epileptiform abnormalities. Specifically, there was a high prevalence of epileptiform abnormalities in individuals with ASD with and

without a history of seizures, which suggests clinicians may need to rely on prolonged non-sedated overnight EEGs to assess for the presence of epileptiform abnormalities. In addition, there was an increased prevalence of epileptiform abnormalities in patients with stereotypies and aggression regardless of seizure history. Further, the prevalence of epileptiform abnormalities is significantly smaller in Asperger's patients compared to those with more severe forms of autism. While we did not find a relationship between epileptiform abnormalities and language skills in this study, the fact that Asperger's is distinguished from other ASDs by the preservation of language skills suggests that epileptiform abnormalities may have an adverse effect on language, or may reflect a more severe type of brain dysfunction that affects both language and neuronal excitability.

Acknowledgments This project was funded by NIH Clinical Research Training Grant T32 RR023254-05, by a grant from The Autism Tree Project Foundation, and by a grant from P.A.C.E. (Parents Against Childhood Epilepsy). The authors thank Amy Spilkin Ph.D. for help with statistical analyses and Lurenette Griffin for administrative support.

Conflict of interest The authors declare that they have no conflict of interest.

Ethical standards This study was performed in accordance with the procedures of the University of California, San Diego Institutional Review Board, which gave ethical consent.

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