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MADELINE HALEY:
Hi everyone! We will get started in a minute or two.

All right. There we go! Hi everybody! My name is Madeline Haley and I want to welcome you to the webinar, thank you for joining us today. Because of the number of participants, your audio will be muted for the call. It can submit questions at any point during the webinar, with the chat box. It is being recorded and will be available on the website, which I will drop in chat right now.

There will also be a short evaluation survey at the close of the webinar. We invite you to provide feedback on this webinar and suggestions for future openers. In the interest of time, let's get started. Went to acknowledge the health research and services Administration as the funding source for the AIR-P network.

Please join me in welcoming Doctor Rao.

DR LEKHA M RAO:
Thank you so much! I am a pediatric doctor at UCLA, thank you for being here today. Today I will start off with some basic definitions of what are seizures. What is epilepsy, and then we will talk about the prevalence of epilepsy and autism, and treatment strategies unique to epilepsy and autism. Have clinical trial support which is not relevant to anything I will discuss today.

And with pediatric epilepsy, most of what we discussed in terms of medication will be off label because it is very hard to randomized controlled trials in the pediatric populations so most of the time we are using medications that have an adult indication with the specific pediatric indication.

I will start out with an intro video.

(Video plays)

This is an example of what we call a seizure. The child has elevation of the extremities, stiffening followed by jerking, they're demonstrating proper seizure first aid by placing them on their side, providing comfort, nothing in the mouth, most of the times seizure stop on their own within a minute or two but you can see how these are scary to family members who witness them especially if they are unexpected.

So what is a seizure? Seizures are actually much more common than people realize. They affect an eight to 10% per population and one in 10 people will have a seizure in their lifetime. Not to scare anybody, but probably at least two or three are you here might have had a seizure or will go on to have a seizure. That includes provoked and unprovoked which I will go over what that means.

 

Seizures are very common in that they account for one to 2% of all emergency department visits and 1/4 of these will be a first time seizure.

What is a seizure? Seizures are defined as sustained, abnormal electric activity arising from the cerebral cortex with a discrete beginning and end.

How we defined this on this is we are looking for evolution. Meaning the abnormal electric activity starts, it grows, and it might become faster in frequency. And the appearance may change on the EEG.

What it looks like is a direct translation of what the electrical activity is like, where it comes from in the brain whether it is generalized like coming from all over the brain, and spreading, it will be seen like the child I showed you in the video of the whole body is involved. There is whole body stiffening and shaking versus focal meaning it comes from a portion of the brain. It starts in one place, and moves forward.

Depending on where that is, seizures can look very different, they can present with staring, chewing, rapid restless movements, depending on what part of the brain is affected.

What is a seizure versus what is epilepsy? Seizure is considered the event. In the moment, what is happening? Versus epilepsy is the disease that involves spontaneously reoccurring seizures. One pet peeve of many people like myself is on we hear somebody has a seizure disorder, either you have seizures and they are provoked, but if you have recurrent seizures you have epilepsy.

We do not like to say a disorder, we like to say whether or not they have epilepsy. Or they have seizures. Those things are distinct.

The traditional definition of epilepsy which has recently changed, it used to be a disorder characterized by two or more unprovoked seizures occurring more than 24 hours apart.

So, you could have one seizure in your lifetime, and then you have another seizure maybe three years later. That is enough to diagnose epilepsy. This means it is not something caused by infection, trauma, or anything else.

This was sort of a concise definition, it was easy to apply and could be broadly applied to many.

However, we know there are some people who have only one seizure but we know that they have epilepsy. I will explain what that means. This definition does not allow for the fact that some kids will outgrow their epilepsy. Epilepsy is a developmental condition in some circumstances, where it only happens in childhood. By puberty it resolves. This does not account for the fact that you can actually go on to not have epilepsy later on in life.

You could also have an epilepsy syndrome so, something where you have abnormal EEG that might

 

be interfering with your development but never have a seizure. This is not defined as epilepsy.

And those who have reflex seizures, things triggered by flashing lights for example are not included in this either.

So where this came from is looking at all the people coming into an emergency room, after you have a seizure your reoccurrence risk was 50%, but after two unprovoked seizure the risk by a third is 73%. If we look at the lower end of that confidence interval, 60% is the recurrence risk after having a second seizure.

It is a fairly high number. This is why the old definition used to seizures as the reason for that definition. But it does not account for those who we know have known conditions that will make them predisposed to epilepsy.

What happens is periodically, there is a working group, the international league against epilepsy which one of our UCLA doctors, (Unknown Name), has been the leader of this working group. To redefine terminology, and definitions within epilepsy.

What they decided to do was sort of account for the circumstances. So now epilepsy is defined as either it could be two unprovoked, or reflex seizures within 24 hours, or one unprovoked plus probability of further seizures, occurring over the next 10 years. Or diagnosis of an epilepsy syndrome.

What this means is if you have a condition that is known to be associated with epilepsy, such as sclerosis, some of these other genetic syndromes I will talk about, you can be diagnosed with epilepsy after having only one seizure. You do not need to. This is accounting for an EEG abnormality which shows that you are just from that EEG alone, we can tell you have an epilepsy syndrome. We can make the diagnosis even if you only had one seizure.

This also allows for epilepsy to become resolved over time. This means that patients who have remained seizure free off medications for at least five years with no further seizures at 10 years, your epilepsy – we use the term outgrew, now it is considered resolved.

These are not epilepsy because there is usually a factor, Febrile seizures which are children between six months and six years of age have seizures, alcohol withdrawal seizures which can happen in adults, metabolic seizures things triggered by changes in electrolytes, glucose, these are provoked seizures as well as toxic seizures secondary to drug reactions or renal failure. Something called Convulsive syncope where you faint and have something that looks a lot like a seizure.

And acute concussive convulsion, and athletes who were football players, and were hit, seizures that occur within the first week of brain trauma, infection, or stroke which are called acute symptomatic seizures and they are not diagnosed as having epilepsy.

Epilepsy in a general sense or as a concept is considered to be a disorder of the brain characterizing

 

by an enduring predisposition, the definition of a epilepsy requires the occurrence of at least one epileptic seizure.

This does not include other syndromes where you could never have a clinical seizure. It does not account for something with abnormal EEG and I will discuss those things because they are relevant in autism.

How about epilepsy in patients with autism? The prevalence of epilepsy in the general population is .5 to 1%. There is a wide range in this prevalence depending on age, however, in individuals with autism, the prevalence of seizures is five to 40%.

The reason why there is such a big discrepancy between five and 40% is because this is pulled from a bunch of studies that have been going on. They draw from different sample types.

Some might use different definitions of autism. Some might use different definitions of epilepsy. Each study has some flaws to it. There was a recent meta-analysis from Mount Sinai that looked at all of the studies that have been published so far with a fair amount of evidence. They showed the overall median was 12.1%, if you exclude those that have a diagnosis of a syndrome epilepsy, that goes down to 11.2% and is generally higher in females.

The link with autism and epilepsy has been well known. It was first described in the 40s, but when it came to more of a highlight in the neurology community in the 1970s. There was an initial study in 1970 that showed a group of patients with autism, 30% had seizures and 80% actually had an abnormal EEG. There was a subsequent study in 1975 showing 64% had an abnormal EEG.

This might seem like high numbers, and I will go on to discuss that this is actually much higher than what we are seeing now.

Because in the past, abnormal EEG, that definition was very broad. That could include any change in frequencies that we might see secondary to medications and it might include changes in frequencies we see secondary to just – maybe, the brain is not moving as well as it should.

So it has what we called slowing on the EEG. That does not necessarily mean you have an abnormality that predisposes you to having seizures.

What about the role of intellectual disability and CPE? If you do not have an intellectual disability or cerebral palsy the risk of developing epilepsy is 2% by five years and 8% at 10 years. If you have a ID, that risk goes up.

7% at one year, 60% at five years, 27% at 10 years, with both ID and CPE that risk increases even more. 20% at one year, 35% by five years, 67% at 10 years.

Going back to that definition of epilepsy you would say if you have a patient with autism who has ID and SEP which means they probably have structural abnormality on their MRI, after one seizure might

 

end up treating the patient or diagnosing them because there recurrence rate is so high.

There was a study that looked at long-term follow-up of these patients out 20 years. Only 16% had remission of their seizures as adults. This is very different from the general epilepsy population with many childhood syndromes, about two thirds will resolve with age.

So, when does epilepsy develop in patients with autism? There tends to be a bimodal distribution similar to what we see to the general population, the vast majority of epilepsy is diagnosed under the age of 18.

Out of that, the majority is under the age of 15. This is not any different. In patients with autism. They develop anywhere between infancy to age 5, and there is a second peak in adolescence with the onset of puberty.

Similar to the study I just showed you, there is a higher risk with concurrent intellectual disability. With suggestion of increasing risk the more severe the impairment. There was one study that looked at patients with ID versus... 21.5% of patients with ID, versus 8% with that.

Patients with autism can have any form of seizure like generalized, focal, features of a benign epilepsy syndrome, really there is no specific kind of seizure and no specific epilepsy type is more associated with autism.

However, children with autism in a single non-Febrile seizures have a higher recurrence rate of seizures than anyone else in the general population. Many would consider autism a risk factor for epilepsy. Therefore in keeping with that new definition of epilepsy, many would it diagnose epilepsy after a first seizure in a patient with autism and treat after first seizure knowing it is a condition that predisposes to epilepsy. And has a higher recurrence risk.

So, why does epilepsy develop? We can look to keep clues from the epilepsy population. In epilepsy we have something called epileptic encephalopathy which causes an autism phenotype, and they may meet criteria for autism or may not meet criteria because they actually lack some of the social impairment. They may still be able to make eye contact, and have no issues with socialization, but they have issues with language and develop it.

Examples of this would be Lennox-Gastaut syndrome associated with multiple seizure types, and they have a characteristic pattern under EEG of the slow spike wave.

I tell the residence all the time, if you have sitting on your EEG that looks rhythmic or heartrate, that is never a good thing. When you have spikes like this this is not a seizure, this is just to look like when they're sitting there, you can imagine this is like having an electrical storm going on in your brain. You cannot interact with the world you normally would.

Same thing with another syndrome I will describe more in detail which is a sodium channel mutation, these patients also often have an autistic like progression with the onset of seizures, they often have

 

seizures with fevers because their mutation predisposes them to temperature, and there is EES which is a mouthful to handle, that is a phenomenon where kids might not even have clinical seizures and they have frequent spike wave discharges similar to this patient, all their sleeping. It is also been called Penelope syndrome because of any of you remember Greek mythology, Penelope was... (Indiscernible) she was really waiting for him to come back home, but what she would do if she would leave during the day come at that night she would leave it.

The Collett Penelope syndrome because kids will learn something during the day, go to sleep, wake up as if it had never happened. Many of these kids are normally developing and have a regression, sometimes this can mimic autism.

The other thing we observe in epilepsy is that sometimes in these (Unknown Name) if we treat the seizures and support discharges meeting the abnormal electric activity happening between seizures, that can actually lead to an improvement in their autism phenotype. We have had patient that met criteria for autism, improve the seizures, or they had epilepsy surgery and they no longer met criteria.

This is a phenomenon where the electrical activity may be influencing how their social and cognitive interactions are.

Epilepsy itself is also a risk factor for autism. If you look at children that have a genetic condition with a high prevalence of both autism and epilepsy, having the presence of epilepsy or the diagnosis actually increases your risk for autism spectrum disorder.

We see this in studies of children with epilepsy and this was a study out of the UK the look to children referred to in a large Centre for epilepsy, and they underwent neuropsychological testing. Out of these kids, none of them had a prior diagnosis of autism and through testing, they found 21% met criteria. 61% of those had intellectual disability and 28% were considered severe. They found interestingly as well that an additional 26% had features of autism based on parent and teacher questionnaires.

But they did not meet criteria. ID was a predictor of having autism. Epilepsy may play a role in the ID, thereby impacting the autism, not necessarily having a direct impact.

These children also have high rates of other neurobehavioural comorbidities which we see in epilepsy like OCD, tics, and ADHD. We know there is something to do with the neuronal connections and the network in epilepsy may also impact autism spectrum disorders.

The other thing we found is that in maternal registries, prenatal exposure to Valproic acid increases the risk of autism in children.

This might speak to some role of modulation of excitatory pathways during field development, there is something about suppressing some of that excitation that might lead to later development of autism. The other thing we know is infantile spasms has a risk of autism if they are treated, and that we found is the patients that have symptomatic infantile spasms, caused by something we know whether it is prenatal stroke or sclerosis or some underlying reason, they are more likely, even if you treat their

 

seizures early to go on to have autism.

This is an EEG of something called hips arrhythmia or the brain gets this very irritable pattern and looks like a child came and scribbled, but this is actually a child's brain waves. When this is happening, you can imagine it is like – again, it is an electrical storm.

Children cannot learn and develop and groves that many of these kids also have a regression, they are normally developing and have an onset of infantile spasms, if not treated early they will have severe intellectual disability. It is interesting to find even if you treat early they are more likely to develop autism.

This suggests there is a pathophysiological role for epilepsy in the development of autism. Also looking at family history, this was a study and review article out of Boston children's that looked at different studies with families with autism will stop the epilepsy rate was 12.8% but even with siblings without autism it was 2.3%. If you remember the general population, the incidence is 12.51% which is general of the population.

Clearly there is some genetic link between epilepsy and autism. Another study showed epilepsy was independently associated with broader autism phenotype, people felt like they had family members undiagnosed, this is another link. Reporting a family like with epilepsy was associated with an increased risk of those with autism.
Knowing there must be some genetic link we can look to
syndromes where we know there is autism and epilepsy as a core morbidity to look for clues of why this might happen.

Probably the best-known one is Down syndrome. Down ssyndrome has a risk of epilepsy in 8 to 13%, and autism in five to 9%. This is a chromosome disorder.

(Audio breaks up) we know that SHANK3 the gene is involved in receptors which are in excitatory neurotransmitter in the brain as well as in (Indiscernible) which is involved in learning in the brain, so, there is something to do with the excitation and connectivity here that can contribute to both. Fragile X is one of the most common inherited forms of autism and intellectual disability may have epilepsy in 10 or 12%, and these have spikes similar to what we see in a benign form of childhood epilepsy.

The thought is this might have some impact again on excitatory transmitters and excitatory receptors of the brain. And the most well-known in epilepsy world is sclerosis complex. The vast majority of these patients, 80 to 90% develop epilepsy, and autism as a high risk of 20 to 60%. This is a pathway demonstrating the pathways of these genetic mutations.

These are an overgrowth pathway, it might mean hyper connectivity causing both epilepsy and autism. P10 mutations is associated with microcephaly and the pathway associated with (Unknown Name) syndrome similar to TSC.

These are areas of the brain that can generate seizures. You are causing both the autism, and a focus

 

point for seizures. MECP2 syndrome or Rett syndrome, microcephaly, they have a loss of spoken language and hand movements. This is a high incidence of seizures in 50 to 90%. It is thought to be related to (Indiscernible) and there is a atypical Rett which is early-onset epilepsy, usually also females with microcephaly who often have no expressive language and have stereotyped hand movements.

FOXG1 prevents with infantile spasms and is a duplication on chromosome 14 which regulates patterning and neurogenesis and another one that even if the infantile spasms are treated early they often go on to develop autism.

And SCN2A is a sodium channel mutation as opposed to SC and 21 a – it was found in patients with autism then related to autism, there is a varying spectrum of autism in this which can go from benign neonatal syndromes to Ohtahara syndrome which is a catastrophic syndrome.

Why does epilepsy develop? We can also look to sibling studies, like you mentioned for the family history. Individuals with epilepsy are more likely to have siblings or first-degree relatives with ASD. There also might be a role for maternal information, from animal models we know that prenatal infection carries a high risk of later development of autism in the progeny as well as with epilepsy.

There might be some link between the two here. There is also a lot of theories about neuronal networks. Hyper- connectivity, we know from fMRI studies, autistic savants will have hyper-connectivity to areas like with music, or art, but hypo connectivity to the social areas of the brain.

Even though they can play songs from start to finish, they have trouble engaging in a conversation. There is also thought into whether the location of the abnormality is on their EEG might also be predictive of whether they develop epilepsy, or has to do with autism in general and there is research still ongoing with us to find biomarkers of who might develop epilepsy so it could be caught early.

The frontal brain is more involved with expressive speech, with higher executive functions. There is some thought that maybe this frontal location also has to do with difficulties with socialization.

There is also imaging studies that support this, I mentioned fMRI. SPECT studies have often been used for seizure localization in children with refractory epilepsy. This study is from Japan that I brought up here, looking at children who have refractory epilepsy who are ongoing evaluation.

We know that many children undergoing epilepsy evaluation who have autism will show temporal hypo metabolism on PET, even if that is not whether seizures are coming from. This speaks to some difficulty with connections and aberrant hypo connectivity.

Including cerebral blood flow, areas of the hyper metabolism are probably areas that generate seizures versus hypo metabolism which are signs of deficient connectivity. SPECT often shows this in the temporal and frontal lobes. Some sign of aberrant signalling and aberrant conductivity contributing to the development of both autism and epilepsy.

How do we diagnose epilepsy and autism? Routine EEG is not considered standard of care, there are

 

things we have found to be standard of care but this is generally only indicated if there is some clinical suspicion procedures. Things like staring spells, or movements that do not look like regular behaviour, some consider getting an EEG if there is history of regression.

It should not be a routine thing. The reason being that, yes children with autism are at higher risk. However, there are challenges that are involved with getting an EEG in a patient with autism. They might have difficulty getting their head touched or might need to be restrained, this might call medical trauma or fear.

It is important these are done with pediatric trained technicians. We involved child life, sometimes in the outpatient setting, patients will bring their OT or therapist to try to help. I had a patient with autism the mom put together a booklet with pictures and diagrams, so she could explain to him exactly what we were doing and why we were doing this.

In a neurodevelopmentally normal child, getting a 30 minute EEG is not a big deal, but with children with autism it can be a struggle. We need to be conscious of that.

So 30 minutes versus a longer EEG for four hours, versus overnight and getting ambulatory EEG. Your likelihood of abnormalities being caught goes up to the study, again some clinics we all know of some out there they routinely get EEGs and that is fine.

If they are able to do that without significant impact to the patients. That only downside is financial. However, if you find abnormality is, what will you do with those abnormalities? That is something I will go over.

Finally, if the child is so uncooperative you have to sedate them, getting sedated EEGs is not ideal because the sedation itself will have an impact on the study and the frequencies that might obscure abnormality is. If you will sedate the child, you might as well sedate them for the placement, wake them up, then obtain your EEG to keep them overnight.

I do not like getting sedated EEGs just for the 30 minute setting. If you like it is a lot to put the child through. If you do not have a high suspicion for -- seizures.

EEGs might be useful to guide treatments, many psychiatrists like having a baseline EEG because psychiatric medications can lower the seizure threshold and they would want to know if the child is at risk for having seizures.

Some clinicians also like to use anti-seizure medications for behaviour. Then you start to say, OK, let's say you find an abnormality – are we treating the EEG or the patient?

There is suggestion of having higher Association of EEG abnormality's might have correlation with severity of behaviour in autism. Studies have been difficult to replicate, there have been many studies looking at this and they have not been able to replicate it.

 

We do not routinely recommend treating... (Audio breaks up) in the earlier studies the definition of an abnormality was broader but it is still a high rate. The likelihood you will find something is pretty high.

You can find focal discharges in 17 to 20% including spikes similar to those found in benign focal abilities in childhood, but there is no association between the localization of those discharges and presence of language progression. In the central temporal region, it is called the (Unknown Name) region which is involved in expressive and expressive language.

If you have abnormalities it might affect your speech, but the study did not find that. The other thing we know is that children might be diagnosed with autism, and cured once they are treated.

This is – I will not call it a well-known secret because any people in the neurology community know there is a certain celebrity who is a proponent of things vaccines cause autism and her child was cured. That child actually had one of these epileptic encephalopathies and was treated for that, and the autistic -like features improved.

So, what is this phenomenon of ESES? We know that regression is reported in 30% of patients with autism, usually between the ages of 18 to 24 months and that 15 to 18 month range is what is associated with the MMR vaccine, but that is really just a coincidence and its correlation not causation.

There is about 20% that will have an abnormal EEG which is referred to in the epilepsy literature as autistic regression with epileptic form EEG. But not ESES.

Some studies suggest higher rates of epilepsy in children with autism and regression, others with no correlation. Seizures are more likely in those who have language regression after age 3, so they were developing normally until age 3 and had regression is more likely to be epileptic encephalopathy. There was a prospective study that looked at people and brought them in for an overnight EEG at a 10 year period, and out of all of those children there were only 10 with ESES identified and only one had autism. This phenomenon is really rare.

Just because somebody had a developmental regression does not mean they have ESES, there is something else on top of it usually.

What do we do to treat these kids? Some medications that were used to treat seizures may actually have some benefit in terms of behavioural side effects, this is where some clinics will treat abnormal EEGs because if they use something to treat behaviour anyway, I might as well treat with a seizure medication. However, I do not advocate for trying to treat to normalize EEG, there is no evidence to support that. Although I do know there are clinicians out there who do that.

Things like Valproic acid it might be useful for mood stabilization, many kids with concurrent epilepsy also have migraines. This might be helpful for migraine prophylaxis as well, as well as (Unknown Name) used for OCD, and for mood stabilization. Lamothe Eugene, and Carbamazepine which is not recommended for generalized epilepsy, it could be mood neutral or in some ways stabilizing.

 

And Clobazam is used in Europe a lot for anti-anxiety, we will talk about these adverse effects as well. Something specific for epilepsy syndromes we use Ethosuximide which is mood neutral and should not have effect on mood, and Vigabatrin might go on to prevent development of epilepsy so there is some move in a trial to starting kids routinely on this before they develop infantile spasms. And Everolumus is an anti-inhibitor used in sclerosis, they had improvement in the seizure frequency and there was a trend towards improvement in their autism symptoms as well. However a lot of our medications may also have adverse effects.

So this medication is associated with agitation and irritability. Many people do not like using it in children with autism, however if you look at the literature that it only occurs in 15 to 20%, and if there is a component of epileptic encephalopathy, this kids having frequent seizures, these medications are good spike suppressants. I have had the reverse happened where had patients with autism become less irritable, and the parents said the teachers reported like, what did you do? Did you have a new therapist?

All of a sudden they are much more happy and interactive. It works as a spike suppressant, so do not discount this or not use it in autism because of this potential for agitation or irritability.

If the kid is irritable to begin with, maybe it lowers the threshold for them to get more agitated. But you agitation to the point it cannot be tolerated is really, really rare.

Valproic acid is used a lot as a mood stabilizer but it can cause easy bruising and bleeding, this is an issue if kids have behaviours that include banging, or headbanging, things like that. You have to worry about that. However it is also a spike suppressant.

If they have features of activation of spike wave discharges, it might be helpful in that respect. And Lamotrigine can also be activating, I have trouble with kids not sleeping, and Perampanel can be associated with agitation and aggression.

And Clobazam I mentioned to be beneficial for anxiety but can cause impulsiveness because it is in the nature of a benzo diazepam.

Some consideration to think about is that not everything that shakes is a seizure. This was a study that found they looked at all patients with non-epileptic seizures, these are seizures not coming from electrical – abnormal electrical activity in the brain, they screened 59 patients with non-epileptic seizures, and attentiveness patients – 17% of the patients actually had autism. 50% were undiagnosed at the time of referral and 50% were diagnosed subsequently, they found a high rate of concurrent neuropsychiatric disorders, like I said earlier, OCD, ADHD.

There is some thought this might be related to (Unknown Name) which is a fancy term for difficulty recognizing your internal emotional state. One of the series of non-epileptic seizures it is your body expressing something else it is feeling. Whether it is underlying anxiety, or manifestation of buried trauma, and the thought is in kids with autism is that this lack of social participation may lead them to

 

compensate during times of pressure, like a release mechanism for their body to have a non-epileptic seizure.

Clinicians who work with non-epileptic seizures should also screen for autism or this is something we should be on alert for as well.

In conclusion, children with autism are at high risk for developing epilepsy than the general population, it is something to keep an eye out for. There is no clear role for treating discharges, if they have a normal EEG and have never had a clinical seizure, I do not advocate using a seizure medication to suppress those spikes.

Some people will use them for other purposes such as mood and may have an added benefit of suppressing the spikes. Having regression does not mean there is an underlying epileptic process, but epilepsy can be a risk factor for developmental regression. Treatment can be specific for the epilepsy type but also keeping in mind the different side effects profiles. And it screening for autism or screening for non-epileptic seizures in autism is good as they may be at higher risk. Thank you!

MADELINE HALEY:
Thank you so much for this presentation. We do have some time for questions from the audience. I do not see any yet. But I will wait a little bit just to see if some people start submitting questions.

In the meantime, you can always email us if you have any questions, I will also send the link for the feedback survey right now.

Additionally, here is the link to register for next month's webinar on November 30, which is also on the screen.

DR LEKHA M RAO:
I hope no questions means it was clear.

(Laughter)

MADELINE HALEY:
It seemed very clear to me, so... we did get a question. So he asked in the chat, how do stimulant medications interact with the epilepsy medications?

DR LEKHA M RAO:
I get this question a lot. You see in stimulants listed, like, risk of seizures as a side effect. What we see in patients who have epilepsy and they are sleep deprived, that can increase the risk of seizures.

What I usually say is if you can control that, the benefits of stimulant medication outweighs any risk of increasing seizure. It does not matter, you know, if their epilepsy is well-controlled if they cannot participate in school because of their ADHD is out of control. I advocate for whatever treats ADHD that is beneficial, there is no interaction between the medications, it is that the stimulants can cause sleep

 

deprivation or insomnia which can lower the seizure threshold.

MADELINE HALEY:
Thank you. We did get another question. How does epilepsy medication play a role in obesity among autistic patients?

DR LEKHA M RAO:
Good question. It depends on the specific medications. Valproic acid is commonly associated with increased appetite and can contribute to weight gain. As well as others, which are often used and can increase appetite. Otherwise most medications are considered weight neutral which do not increase appetite or increase in weight.

MADELINE HALEY:
Somebody asked in the chat as well, is there an interaction between ADHD and epilepsy medication? Somewhat similar to the stimulant one but I do not know if that was more specific.

DR LEKHA M RAO:
It is similar in that there is no interaction between the medications, but more of the theoretical lowering of the seizure threshold and we say it is better to treat their ADHD.

MADELINE HALEY:
There was a question about increased seizures with medications, they said their understanding is that these medications – something about antipsychotics with Klonopin, maybe the interaction with those two?

DR LEKHA M RAO:
It is known that those medications can lower the seizure threshold, however, we do have patience with epilepsy who need to be on these antipsychotics. I will work with a psychiatrist to let them know, this is what you need to do I will manage their epilepsy.

We often see patients with whom they had their first onset seizure and happened to be on that medication, a lot of times they have an epilepsy syndrome we diagnose on EEG. It is not that it precipitated this syndrome, they were going to have a seizure at some point.

That is what I would not routinely getting in EEG before starting these medicines unless they had a predisposing factor or a history of abnormalities and otherwise you can counsel them on it.

MADELINE HALEY:
There was a question about interventions to use for kids with epilepsy and autism versus just autism.

DR LEKHA M RAO:
If they mean by interventions like therapies, or medications? I think the main thing to keep an eye out for is that epilepsy has a high association with neuropsychiatric disorders. Epilepsy alone will increase your risk of anxiety, depression, ADHD, tics, OCD, and on top of that many of those features are found

 

in autism as well.

Often times it is a matter of trying to address both things at the same time or using seizure medications that might be beneficial from that respect. That is why he mentioned the seizure medications that were more likely to use in children with autism – you are killing two birds with one stone.. To try to take advantage of the side effects away.

MADELINE HALEY:
Are the long-term risk factors for autistic patients with epilepsy?

DR LEKHA M RAO:
In general they are less likely to outgrow their epilepsy, in terms of long-term, we think about people with epilepsy on certain medications, we need to watch the bone density. Women need to be ... Valproic acid increases the risk of autism, maybe that is not the best medication, it causes other things as well. The other thing in the long term is that epilepsy especially is generalized seizures, there is a risk of sudden unexplained death in epilepsy patients.

If seizures are not controlled, your risk goes up higher. That is the other thing a lot of patients with autism and epilepsy if they have that, I try to get this under control as best as possible.

MADELINE HALEY:
Thank you. Time for one more question? We do have a bit of time. I will wait a moment to see if any more questions come in.

DR LEKHA M RAO:
I can scroll back to any of these slides if you need me to.

MADELINE HALEY:
Thank you!

Alright, I think that will be it then. If you have any questions, feel free to contact us at the email on screen. And also, please provide your feedback with the survey and otherwise – thank you so much, again, Doctor Rao and we will see you next month in November!

Live captioning by Ai-Media

 