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Vestibular Migraine in Children: The Most Common Cause of Dizziness

Presenter: Soumit Dasgupta, MD

A warm welcome to all of you and thank you for joining us once again. Inventis is honored to present the third and final lecture in the special webinar series led by the internationally respected professors. We extend our heartfelt thanks to Professor Dasgupta for his generosity and passion in sharing his invaluable knowledge with us through these sessions. Today's webinar is titled Vestibular Migraine in Children the Commonest Cause of Dizziness. Although vestibular migraine is now recognized as the leading cause of dizziness in the pediatrics population, it remains underdiagnosed or misdiagnosed due to the complexity of evaluating children and the tendency to treat it as a diagnosis of exclusions.

We are also delighted to have with us Dr. Salvatore Martelucci from Latina here in Italy, a longtime friend and colleague of Professor Dasgupta and a member of the Vestibular Italian Society. As always, feel free to submit your questions using the dedicated section on your screen. And please don't forget to complete the short survey you'll receive after the session. Your feedback is precious and help us shape future academia content tailored to your needs.

Let's begin. Salvo, the floor is yours. Enjoy the webinar and see you later. Thank you. Thank you, Anna.

Good evening and welcome to all colleagues attending this academia meeting. And this will be a really precious lesson about an hot topic in vestibular medicine. Vestibular Migraine in Children by Professor Swamit Dasgupta, a true expert and a good friend of mine. Professor Dasgupta is director of the Audiology department at the Helder Hay Children's Hospital in Liverpool and the author of dozens of papers about vestibular disorder in pediatric age. But not only that, Sumit is a passionate historian of medicine and has a little but really valuable team of researchers including Professor Marco Magdala, Professor Guneri from Turkey, Professor Tarnow and Professor Bassim.

Altogether they published some exciting and extremely pleasant to read papers about the history of vestibular medicine. The last one I read is entitled the Pioneers of vestibular physiology in 19th century. I recommend you to read this paper. So it's a pleasure for me to introduce Professor Swami Dasgupta.

Thank you.

Thank you Inventis. Thank you, Anna. Thank you Salvo for the kind words. And this is the last in this year's series, as you know that I do three every year. So this is my ninth lecture to you guys and today I'm going to talk about vestibular migraine in children.

I must confess right at the beginning that of all these nine lectures that I've done, some of them quite complex, this is probably the most complex one. So you'll be quite surprised and curious that such a common disorder in children has got the most complexities, not only from understanding what the thing is all about, but also the diagnosis, which is a very intense and a complex process, quite different from what you would encounter in adults when you diagnose a vestibular migraine. And the reasons will become apparent. So I have taken some effort to explain why is vestibular migraine in children different from that in adults? What are the fundamental differences between a child's brain and an adult brain, which will make you easier for you to understand as to how does the phenotype differ?

So let's begin.

So, as you are all aware that migraine is a neurological disorder that is traditionally characterized from antiquity by pulsatile headache or a throbbing headache, which is episodic in nature, comes and goes, sometimes with identifiable triggers, sometimes no triggers can be identified, and they are usually self limiting within a certain period of time. The International Classification of Headache Disorders, which is kind of the reference standard for defining headache disorders, has defined this migraine with different variants and with different codes. For example, migraine with aura, migraine without aura, complicated migraine, probable migraine, episodic syndromes with migraine, etc. Etc. Etc.

Vestibular migraine actually finds its way into IHCD where the predominant symptom of headache in classical migraine is replaced by the predominant symptom of dizziness. Now, in some migraine with

aura, the dizziness or vertigo is categorized as an aura, which means it precedes the headache migrant spell. But now it has been recognized that vestibular migraine is a special variant of overall spectrum of migraine. It is a very simple condition in the sense that it's not dangerous, but it is a very distressing condition both in adults and especially in children. So the diagnosis is absolutely crucial and complex that someone does have a vestibular migraine.

And this is extremely important from the treatment point of view, because when managed properly, you can treat a vestibular migraine. The problem is in the diagnosis because there is so much overlapping all around. You must have a watertight process by which you can zero down on the diagnosis. Not helped by the fact that vestibular migraine does not have a biomarker. For example, you do not have classical VHIT results, you do not have classical VEMP results.

So how can you objectively prove someone has got vestibular migraine but there is a way out which I'm going to mention in this lecture. And needless to say, in children, management has a very rewarding outcome, which means it does give me a sense of fulfillment when I correctly diagnose a vestibular migraine in a child, treat it, cure it, and they are indebted to me with their parents for a very long time. I get phone calls from 18 plus teenagers who were teenagers when I treated their migraine in Christmas. They call me, call my secretary and wish me good, Happy New Year and good luck. So that is the biggest reward that a doctor can live for.

So, as Salvo very kindly said that we are a group of vestibular medicine historians who publish frequently once a year. So obviously my lecture cannot be without a brief introduction or a brief resume of how the whole concept of migraine evolved over the ages.

So the Ebers Papyrus in the Theban Necropolis in 3000 B.C. first mentioned an episodic syndrome where you get dizziness and vomiting. This was picked up by Hippocrates, the father of medicine, who also described a similar similar syndrome where he also mentioned that on top of all this, that is

headache, nausea, vomiting, dizziness, there is also an intolerance to light. Arataius from Cappadocia correctly identified a vestibular migraine syndrome which is strangely similar to the Barani criteria. And this was 2nd century AD.

Claudius Gallus dabbled in it. He also thought that there is a syndrome like this, which he ascribed could be coming from the stomach rashes. Al Mubarak or. I think not. I don't remember the exact name, but he was, and he was from the Caliphate, Baghdad.

Caliphate, Baghdad, Caliphate in Baghdad, who described not only vestibular migraine syndrome but also Meniere's disease as well. And Garyopontis in Arabia also did the same. Ibn Sina, who you also know as Avicenna from Persia, similarly thought that there is an episodic disorder where people get dizzy along with it. They hate to look into the light. So as you see, they all identified that there is some kind of a disorder or a pathology which resembles vestibular migraine that we define to this day.

However, it was not before 1960 that Shelby and Lance reported the association between a vascular headache and a vertigo. So they did a study where they found that in their cohort the subjects did not only present with headache but also with vertigo, which usually preceded the vertigo. Then Beaker staff codified basilar artery migraine, which is the classical migraine you all know of the first Time a systemic study was done was by Kayan and Hood. And I did know Alton Kayan. He retired when I started training.

But he was a giant of a man, not physically, but from the vestibular point of view, who first proposed that vestibular migraine is a predominant vestibular affliction and not a brain. They did not define much, but they correctly identified the typical phenotype or the typical features which are associated with the vestibular migraine. The term came into use by the pioneers Dietrich and Brandt from Munich. Neuhausen and Lempert in 2004 first proposed the diagnostic criteria and they called it migranous vertigo, quite distinct from classical migraine, because they were the ones who emphasized that

dizziness is the predominant symptom. And finally, in 2012, I think 2012, Barani Society published the diagnostic criteria, which has been now revised in 2022.

This was all very okay for adults. No one knew or no one thought that a similar condition can also happen in children. Baser in 1964 identified an episodic syndrome which he termed benign paroxysmal vertigo of childhood bpvc, where he found that children developed this brief period or a momentary period of dizziness and then recovered completely just after the spell. And this was confirmed to be a migraine variety later on. 2004 by Batson & Barani Society published the criteria for diagnosis of vestibular migraine in children in 2021.

Now I have. I will. I will kind of dwell a little bit elaborately on the epidemiology and pathogenesis of vestibular migraine, because as I said right at the beginning, it is a complex condition and understanding the pathogenesis will lead you for a better understanding of the condition. And why do you need a better understanding of the condition so that you can make a diagnosis correctly in a child?

So the reported prevalence in children presenting with dizziness is quite high. As you know that we have published this very frequently, that dizziness in children should not be ignored. Unfortunately, the awareness is not there. But dizziness in children is a significant cause of morbidity in children and the commonest cause of dizziness in children across all age groups, at least 25%, that is a quarter, is because of vestibular migraine. So it's a very common condition in children.

And the problem, once again, it's a diagnostic conundrum. You'll have to make a diagnosis of vestibular migraine and that is the most important aspect. It is more common in adults and the reason will become apparent and the main reason for this is cerebral plasticity is different. A child's vestibular system develops until the age of 16 when it becomes completely adult, like so, the neurotransmitter and synaptic circuitry keep on developing up to the age of 16 years. If you think that migraine is because of a problem in neuronal activity, then obviously this implies that a developing brain in a child will react to

a migranous process differently than in an adult.

BPVC is common in children under four years. That is once again because of this neural activity. Let me tell you, if you look into the development of the vestibular system, from the age of 4 years, the vestibular system becomes active. Up to the age of 4 years, the vestibular system is still in a hugely fast fluxed developing stage. From the age of four to eight it becomes more active and slowly starts to attain adult maturity.

From 8 to 16 it becomes adult like so, under the age of 4 years. Once again, the way vestibular migraine behaves will be different from a child who is roughly say aged 8, 10 years. And you'll find that out. And females, especially adolescents, are mostly affected. This is because of the well known association between hormonal changes in adolescence and migraine.

It is less common in Asians, probably because of genetic reason. And it is absolutely essential to diagnose as this whole condition, the vestibular migraine significantly affects the lives of a child and not only the child, but also its parents. There's huge loads of psychological consequences once again explained by a developing brain and of course the cognition or the cognitive processes which are associated with the developing brain. So let's have a brief look into the difference between adult and children brain function. So basically it's a huge topic and I have done some research on this and I published as well.

So I have narrowed this down to 4 main, which would be important for you guys for better understanding. So as I said right at the beginning, evolving neurotransmitters are implicated in migraine. That's the N acetylcysteine, the choline and the glutamate, which are migraine substrates. In adult they are very well established, but in children they are evolving. And because they are evolving and because they take part in the migranous process, classical or vestibular migraine, the effect is different.

It does explain the more intense distress in a child with vestibular migraine as compared to adults. And also the vestibular migraine spells, it might last the duration of the Spells are different from that in adults. Taurine is a very important inhibitory neurotransmitter. It's not an inhibition directly inhibitory, but it stimulates inhibitory neurotransmission. For example, glutamate that is slowly developing.

And these levels are low in a child. That means if you remove inhibitory stimulation, excitatory stimulation becomes overt. As a result, the spells that a child will have is far more intense than the adult. Cerebral blood flow is different. Metabolic rates, metabolic rate and oxygen demand is higher in a child, that obviously leads to a vascular change, which is, as you know, a vasodilation, or in other words, less, more oxygen demand will lead to a vascular compromise.

In a child's brain, that means that there will be a more global central phenotype. It goes pretty much all over the brain. So that's why the psychological effects and the cognitive effects following vestibular migraine are far more pronounced. And finally, of course, there is a maturing cerebral plasticity, and there is a continuous change in the balance between excitatory and inhibitory neurotransmission in a child, which also alters the way a child presents with vestibular migraine. So the phenotype of vestibular migraine in a child in terms of distress frequency, duration, intensity would be different from that in adults.

That is a very important consideration. However, this is a little bit too simplistic for the simple reason how will a child express the distress or the intensity of the distress? Because children on the age of 10 years, they will not be able to tell you much. Even teenagers can't tell you much. So let's look into the theories of pathogenesis of vestibular migraine.

There is a vascular theory, which is not the most important one. The most. The three most important ones of pathogenesis is the trigeminal vascular system, activation, tvs, cortical spreading depression,

and a genetically determined inherent hyperexcitability state. So these are the three most important substrates of vestibular migraine, which can act secondarily on the vasculature of the migraine focus. So trigeminal vascular system is a huge network of, let's say, nerve fibers which originate within the trigeminal ganglion in the petrous part of the temporal bone.

And then it goes up to the brain stem, where you get the classical trigeminal nucleus, the gasserian ganglion, and it goes to pretty much all over the brain. But what it does is it supplies the blood vessel, the intracranial blood vessels, and Also the anterior inferior cerebral artery, even the vestibulocochlear artery. So it's a very important neuronal control of the vasculature of the brain and the vestibular system.

That's the first theory. The second theory is cortical spreading depression. Now, this is a process which was first discovered, I think, 60 years back, and it appeared in nature, where there is a stimulus control, which we do not know what could be genetic susceptibility, where there is a random depolarization because of excitatory neurotransmission and with potassium. And what happens is they mess up the synaptic excitatory inhibitory balance. They stimulate the trigeminal vascular system and it spreads upwards.

So it's an upstream spread. So initially there is a period of depression. But then what happens is because the fine balance with inhibition excitation is gone, there is an excitation, a net result that the cells, they start firing. This classically explains the aura which you get in a typical migraine. So I've prepared this figure for you.

It's a complicated figure, but what you can see here is at the central is the TVS and cortical spreading depression and hyperexcitability state, the three important ones that I mentioned. So what that does is it leads to a process called neurogenic inflammation. This can be protective, by the way. So it's an inflammatory reaction activated by these triggers, which is actually protective. But in case of migraine,

it's not.

It's counterproductive. That might act in the intracranial blood vessels and that spreads to the brainstem areas. On the one hand, it spreads to the ICA territory. It may spread to the peripheral inner ear, and upstream it goes to the thalamus. The thalamus then sends the information to the somatosensory cortex in a number of regions in the brain.

And the final perception of vestibular migraine is in the thalamocortical centers with the different changes that you might find it also in its wake effects of limbic system. This red rectangle that you see, this is where the vestibular pathway for vestibular migraine and the migraine pathway from the classical migraine, they merge. So because there is involvement of all these regions in the vestibular system, what you get is a typical dizziness. It can be accompanied with a headache, but that needs to be present. In 50% of cases, there might be no headache.

So basically, this region is the most important region where the two of them merge. So Cortical, spreading depression, neurogenic inflammation, vestibular nuclei, and remember, two areas of the brain stem. One is the Raffy magnus, the classical migraine focus for a basilar artery migraine, and the peri aqueductum gray, which is the classical focus of a vestibular migraine. And the vestibular nuclei, it feeds heavily into the periaqueductal gray. This is important for us to remember because this has got a direct pharmacological connotation which I shall cover at the end.

Now, obviously you don't have to remember all this, but these are the vestibular areas participating in vestibular migraine, which is quite extensive, as you can see. Raphe nucleus is of course the classical and periaqueductal gray, as I've said. But as you can see, it's pretty much so much spread all over the brain stem, the cerebellum, the temporal lobe, cortical. So this focus actually spreads pretty much all over the brain, even though, even though it's dubbed as a vestibular migraine. So what are the different biomolecules in vestibular migraine?

There are quite a few neuropeptides which have been identified. The cgrp, as you know, has taken the adult world by storm because there is very good treatment now available with cgrp. Unfortunately it cannot be tried in kids as yet. Substance P is a pain mediating neurotransmitter, neurokinin, some pituitary adenate, cyclase. These are important neuropeptides which are all expressed in vestibular migraine neurotransmitter.

The most important neurotransmitters are the glutamate and the gaba. There's a fine balance between the two. And this balance is up, is upset and it goes towards the excitatory phase to explain the vestibular migraine focus. Serotonin is a very important neurotransmitter in vestibular migraine. Often vestibular migraine is called a low serotonin syndrome because low serotonin, what it does is it triggers up the migraine focus.

But once migraine is established, serotonin starts to flow and becomes increased in its level, which exacerbates further symptoms. So serotonin, low serotonin precipitates a migraine. High serotonin exacerbates a migraine and a dopamine, which is heavily, heavily expressed in the periaqueductal gray and in the vestibular nuclei. This is also an important migraine neurotransmitter for vestibular migraine. Quite a few biomolecules as you can see.

I'm not going to read them, but look at riboflavin, which can be used as a migraine treatment. And of course migraine has vestibular migraine, like classical migraine has got a very important relationship with sex steroid hormones, especially in females, estrogen and progesterone.

So you might ask that what happens to the cochlea? Because look, the same neurotransmitters, neuropeptides will also affect hearing as well the hearing cortex, because they're in the brain. And indeed 12.2% of vestibular migraine may have cochlear symptoms. So it's relatively rare. We have to explain why is it rare.

And that's because the trigeminal vascular system has got very limited connections in the cochlear circuitry. In fact it synapses with the dorsal cochlear nucleus and the superior colliculus and then pretty much disappears from the ascending tracks of the cochlea, which is, you know, all these Oliver, all the olivary complex, then the lateral lemiscus, etc. So that's why it somehow the cochlear excitatory spread or the cochlear cortical depression spread is arrested somewhere in the middle, but it can still happen. So upstream modulation is much less than vestibular connections. That's why you do not get cochlear symptoms that often, but you can still get it.

And the problem is if you get cochlear symptoms with vestibular migraine, there is always a chance that this might be a Menieres and associated endolymphatic high drop. So that distinction is also quite crucial to make. So you can already understand the complexity of the diagnosis. So it's not just a simple diagnosis, what we call an exclusion, a diagnosis by exclusion or some, some people say a garbage diagnosis. If you do not find anything, it's vestibular migraine, it's not the case.

You'll have to explain your case all the time. There are some risk factors or etiological factors, what I would call not exactly etiology, but comorbid conditions. So obviously one is classical migraine. Vestibular migraine can happen side by side. Genetic factors.

We have mentioned post traumatic vestibular migraine, kids with epilepsy, kids with Meniere's syndrome, neurodevelopmental disorders, for example autism, and there are a number of migraine variants and we shall visit them one by one.

75% of children with vestibular migraine have a positive family history. It's not one of the important criteria, but it's a useful indicator that it still could be a vestibular migraine. So far, 12 definite and 134 probable loci have been identified. However, what I have seen is that a definite association with CACN1, which is a calcium channelopathy gene mutation, and these are the genes of which in my

clinical experience I've seen vestibular migraine because of the CACNA11A and TRPM7 gene, which can predispose to vestibular migraine.

Head injury is recognized as a cause for post traumatic vestibular migraine in about roughly 4% of cases because of changes in the vasculature following a head injury. But there have been hardly any studies in children which tell you that there could be a vestibular migraine. And the problem is that it's poorly correlated to the severity of the injury. In other words, child with a severe head injury might not develop vestibular migraine, whereas a child with a very trivial blow to the head might develop a vestibular migraine. And it's mainly because of, as I said, vascular changes and of extravasation of extracellular potassium, which once again tips up the balance between inhibition and excitation.

There might be a potential of neurogenic inflammation. As you know, head injuries will call, will generate a secondary inflammation in intracranium and they can actually potentiate the neurogenic inflammation which we talked about. And of course cerebral spinal fluid has a role to play. And because it is still in a state of flux in the child, in a growing brain, probably post traumatic vestibular migraine is far more intense in a child than in adult. But once again, let me clearly emphasize there is no dedicated study as yet.

I have seen in my clinical practice quite a few vestibular migraine after trauma, which are very, you know, very distressing conditions for the child. And they get ignored because in the grand scale of things, a child with a head injury, everybody is concerned with other things rather than the vestibular migraine.

Now, epilepsy and migraine, both of them are due to hyper excitable states, right? And some of them, or the pathways, the circuitry sometimes overlaps in the brain and merges together. For example, the brainstem is a very important area of epilepsy focus. So no wonder they might share common phenotypes for Example, vacant stairs, drop attacks, headache, dizziness, fatigue, and they are both

due to this random firing. So as I said, because of that they do exist side by side.

In fact, children with epilepsy might develop vestibular migraine and the other way around. But the main difference of the focus is whereas in epilepsy it's glutamate driven ampta, in migraine it is mainly the CGRP driven nmda. This is somewhat academic because we don't have pharmacological agents to specifically target these areas, but this is the fundamental molecular difference between the two. Cortical spreading depression is less widespread in epilepsy and the two of them merge together. And quite importantly, children, as I said, they develop an increased risk of epilepsy if they have vestibular migraine.

And you can make a distinction with EEG and with finding epilepsy. Focus Meniere's disease, the idiopathic variety is very uncommon in children. However, what happens is a Meniere syndrome, which is secondary endolymphatic hydrops. And in adults, 25 to 50% of vestibular migraine have also got a simultaneous Meniere's disease vestibular migraine subjects they can develop a Meniere's disease in adults and in children. I have seen about three or four kids with vestibular migraine presenting to me with secondary endolymphatic hydrops.

But as usual this has not been studied. In children the pathogenesis is different, different, as you all know. And the common pathway where the two of them merge is a central and peripheral vasospasm which leads to the typical spells. The Barani criteria, to their great credit, distinguishes between the two with different kinds of phenotype features, for example the duration and the presence or absence of autonomic symptoms. And herein comes the difficulty, because in BANI criteria, hearing loss or cochlear features predominantly would point out to an MD.

But as we have seen before, 12.2% of vestibular migraine might also have cochlear symptoms. Another very important distinguishing feature is the peripheral vestibular system. It might show weakness in chronic Meniere's disorder, but it rarely happens in vestibular migraine. In other words, in vestibular

migraine, finding a vestibular hyperfunction, you can find it, but it's not as common as that in many years disease. And of course classical migraine, the two of them might coexist.

In fact, Barani's criteria in children states number two, that 50% of the episodic disease spells in vestibular migraine in children need to be accompanied with a headache. But the main, most distinguishing point in classical migraine is the vertigo is actually before the headache, whereas in case of vestibular migraine, the dizziness is accompanied with a headache. Now, can you imagine how on earth would you get this history from a child, even from the parents? You know, if you ask the parent, did your child get the dizziness first and after that the headache, how would she know? Well, let me tell you, ladies and gentlemen, that there are ways which we dub as vestibular behavior by which you can indeed find out.

And I'm going to tell you all those different nuggets and tidbits when I come to the assessment of symptom part. So the dizziness in vestibular migraine is far more debilitating than the headache. The headache is a background pressure sensation, mainly unlike in classical migraine, where it's throbbing, can, you can be unilateral. But in vestibular migraine in children, the dizziness is the very debilitating, which can be accompanied with sickness. So the natural history is quite difficult.

You know, it's tenuous. And diagnostic criteria, which is still heavily based on symptoms, is a very simplistic approach and you might actually miss one from the other. And that's the import, that's the important thing to understand about symptom based diagnostic criteria, that they are not kind of foolproof. There's lots of lapses and lots of weaknesses in all these diagnostic criteria.

So neurodivergent conditions are also due to hyperexcitable state. And that is very important. And they have now been convincingly demonstrated to show low serotonin, same like vestibular migraine. So the two of them can be mutually inclusive of each other. In fact, children with autism, they possess a 2.5% risk of developing migraine and those with ADHD 1.75.

However, sensory dysregulation, which is sensory overload, which is basically kind of the child reacting to a very stressful sensory environment, is far more pronounced in neurodivergent condition than in vestibular migraine. So these kids, they are hyper tactile, they do not like smelling things, or they like smelling things the other way. So sensory dysregulation is very pronounced in neurodivergent conditions, but not as much as in vestibular migraine. So these are the different migraine variants which we have mentioned. So I'm not going to read this criteria to you, but look at cyclical vomiting, abdominal Migraine.

Did you know that abdominal migraine also presents with dizziness in a child? Dizziness and sickness and a tummy ache, stomach ache. So abdominal migraine is also important to know. And benign torticolis of childhood, where the head is fixed. This is also a migraine variety.

But I'd like to talk to you a little bit about benign paroxysmal vertigo of childhood. As you can see that at least five attacks fulfilling criteria B and C. The first one is vertigo occurring without warning, maximal at onset and resolve spontaneously and minutes to hours without loss of consciousness. What is important to understand here is how would a child describe this vertigo? So the way a child will describe the vertigo, if he is articulate enough, he'll tell you that, you know, mom, I'm feeling very dizzy.

But most often than not, they can't. So you ask the parents, what have you seen? And what will they tell you? They will tell you, my child is playing perfectly well, no problem at all. Remember, this child is under the age of four.

My child is playing perfectly well, no problem. Suddenly stops in his tracks, looks very vacant, with a very vacant expression in the eyes, and then suddenly drops on the floor and after a few seconds picks himself up as if nothing has happened. Sometimes he wouldn't be able to pick himself up in a minute. What does it tell you? A child will develop a strange facial expression if the child is in distress.

If there is a headache, the child will actually touch the head like that. In some instances, if the child is dizzy, he will also hold the head. But touching the head is most likely due to a headache. So he's not touching the head. A vacant expression in the face, and then suddenly drops.

Why would a child suddenly drop? That he loses his balance momentarily? Why would a child lose his balance? It could be a neurological problem, a dangerous one, we don't know. Or it could be due to a sudden whirlwind of swimming, kind of sensation or dizziness which makes him drop to the floor.

Now these are the histories which can be elicited by asking leading questions. But the most important history I'd like you to take home with you today is always ask the parents, what have you seen in your child's face? Another very interesting piece of history is when these things happen, what happens to the eyes? Mum do. Does your child close the eyes?

If the mum says yes, my child, when he goes like this, in a trance, shuts his eyes. That is an indication that the child doesn't like to look to the light. And what is that? Photophobia. So There are ways by which you can elicit history, but, you know, this doesn't come overnight.

It takes a lot of practice. You really have to work with children and their parents to develop this insight as to what is a right symptom and how to interpret what the mother tells you or the father tells you regarding the child. And everything is normal, by the way. Normal neurological examination, everything is normal. So, as I said, vestibular migraine does not come with frank vestibular hyperfunction in most cases.

And very importantly, you have to exclude other conditions. So why am I so keen on explaining BPVC to you?

In our Bharani's criteria in 2021, which my colleagues have written, they have redefined the term. They say BPVC should be removed from the lexicon, replaced by recurrent vertigo of childhood rvc. And one of the reasons they give is paroxysmia, by definition is for seconds. And vestibular migraine is. Does not last for sick, does not just last for seconds.

It has to be at least a minute. The point which I'll tell you today is a little bit different. You see, paroxysm in the English dictionary actually means seconds up to a minute. Multiple sclerosis can come in paroxysms, you know, and these paroxysms last for 50 seconds. 5, 0.

Now, in a child whose brain is developing under the age of four, as I showed you, all the different things where the system has not yet attained an adult maturity, far from it. A paroxysmal spell lasting for seconds does not exclude a vestibular migraine. So in my humble opinion, the term BPVC should be retained because it's a beautifully descriptive term. I hope I've been able to make myself clear, and I'm more than happy to take questions, questions. So defining inflexible duration of symptoms for a child is fraught with difficulties and ambiguity.

I mean, if you get even an adult coming, even a child at the age of six coming to you, where mom says it only lasts for a few seconds, the rest of the things are all typically vestibular migraine. Would you say that this is not a vestibular migraine because the duration doesn't fulfill the criteria? I won't do that. So once again, criteria driven diagnosis should be flexible and not something which is an absolute obligatory set in stone. That's a very important clinical aspect of diagnosing vestibular migraine.

So pinpointing the duration is often impossible in a child. It is possible in a child if the child is older. Fair enough. But in many children it's impossible. So if you are defining a disorder by the duration, you need to get an accurate history and that needs to be flexible.

It needs to tally with everything else to make you the diagnosis. But that is the point I'm trying to make. And in my opinion, the term BPVC should be retained after the age of four years when the system tries to. When the system starts to attain maturity like an adult neurotransmission is better established than under the age of four years. A child will behave as typically vestibular migraine, which has been set out beautifully in Varani's criteria.

In 2021, the pediatric Varani's criteria. So BPVC merges into vestibular migraine. So I wouldn't call it a recurrent vestibulopathy of childhood. I would call it a BPVC under the age of four years. So now that we know about the pathogenesis, about what happens, how does a vestibular migraine in child differ from that in adult?

Let's come to the diagnosis. Special considerations. This is difficult because there are no biomarkers, you can't confirm it. So diagnosis is based on the defined criteria which we have mentioned, which is a good guideline to go forward to. But always believe that a child is not a miniature adult.

An extrapolating adult criteria for VM vestibular migraine just by changing the age does not fit per. Does not fit the purpose and we miss the diagnosis because children sometimes can't speak back to you. And of course we have said that in detail and why and very importantly, you'll have to get surrogate history from the parents, that is vestibular behavior. And you need to be very skilled in the art of pediatric history taking and pediatric examination in order to do that. It's.

It is recommended that in order to do pediatric vestibular medicine, you spend some time in the pediatric ward to figure out what's going on, especially in acute pediatric medicine, that is that that will give you an immense insight and immense help as to what really goes on with pediatric medical disorders.

So I think of the Barani's criteria. If you get a vestibular migraine suggestion in a child, the most important criteria is criteria number four, exclusion of other conditions. That's absolutely crucial. Therefore full cochlear vestibular quantification. And yes, I do scans on all my kids presenting with vestibular migraine phenotype.

When I first started, I was told by many you are way over the top, this is migraine. Why do you have to do a scan. I have been caught out. I have been caught out when there are things going on in the brain which could have been picked up by the scan. So I have prevailed and I have managed to convince my colleagues that vestibular migraine in a child is not an easy thing to diagnose.

You would need a scan in all instances. Now you might disagree with me and I'm more than happy to enter into a discourse with you, but that's my clinical practice. Of course you need to do a full clinical examination for neurologic metabolic causes of dizziness. Because look, dizziness is a symptom which can be generated from a number of other systems, from just anemia, from a palpitation, from a heart block, from a neurological condition. So you'll have to be meticulously examining.

I still listen to my kids hearts with a stethoscope even now, although I'm a doctor for the year. And this is for all my vestibular patients, especially vestibular migraine. And imaging, once again, let me emphasize, is crucial. I'll show you a very interesting case today. You see the psychological reaction or the emotional reaction of vestibular migraine to children can be quite severe.

But it should not be that severe that the child becomes a complete mental wreck. That is when we question that there is a disproportionate emotional reaction to the actual condition. And in these instances, if you scan these kids, you'll find out something structural is explaining this severe reaction. And that's what I'm going to show you at the end. A very, very important case.

Especially if you get a lesion around the region of the vestibular nuclei or the corded nucleus or the amygdala, or in all these emotional centers, the limbic system, the perio equal gray, then the emotional reaction to the vestibular migraine would be huge. And this often gets ignored. And these kids, they end up with the mental health they can't explain. When we scan them, we explain them. We provide cognitive behavioral therapy with extremely good and rewarding outcomes.

So please, if you believe me what I saying, please consider scan for all your cases of vestibular migraine. And I stand my ground very, very firmly. You'll see the literature of the evidence which says scan is discretionary. Many of my colleagues, actually pediatric vestibular specialists, will tell you scan is discretionary. I'll tell you scan is compulsory and I've got a good reason for that.

So this is the Varani criteria. So I've given you three criteria Bharani Adult 2012, the Beta ICHD3 in 2018 and Bahrani Child in 2021. The ICADH Beta 2018 does not have any criteria for a probable vestibular migraine which Bharani has where you do not need the headache. But look at the Bharani child, which is pretty much the same as that of the adult. And to my mind, of course this would be same to that of an adult when the child is able to describe his symptoms.

But I've told you, in quite a few instances, in fact in 50%, I would say the child will not be able to tell you what's going on. So how on earth would you elicit all this? Will the child you my headache throbs, my dizziness, my headache throbs. Does, will the child tell you that I'm feeling very dizzy? Does the child will tell you that I'm having photophobia?

He won't. So that's why it's important to appreciate that these guidelines are applicable to children who can describe their symptoms. For children who can't describe their symptoms, you'll have to elicit the same history by asking the parents. Now look, the recurrent vertigo of childhood. It does not require current or past history of migraine or any of the c.

And the duration is 1 minute to 72 hours. BPVC, therefore, to my mind is an atypical vestibular migraine which is explained by the fundamental differences of migraine circuitry behavior under the age of four years and above the age of four years. And the fourth diagnosis is absolutely crucial, not beta accounted. So in other words, you have to exclude other conditions to prove migraine.

So we have just spoken that when a child is prelingual, you wouldn't be able to describe so vestibular behavior. And funny thing, and that's very important, I have seen many kids with BPVC who get treated for epilepsy because the epilepsy circuitry and vestibular migrant circuitry are pretty much similar. Right. So vacant stare, sudden drop to the floor, videoed and then started on epilepsy medication without any, without any success. They are in many instances bpvc.

And you treat BPVC or vestibular migraine? You might ask. There are some anti epileptic drugs which can be treated for, which can be used for vestibular migraine. But those come much later, as we shall see. Simple anti migraine medications will cure a bpvc.

In fact we did one only last week. Cyclical vomiting variant often ends up with gastroenterology who does everything, scopes everything, can't find anything, they get sent to us and yes, they can be very well treated.

So what are these symptoms? Encountered? Vacant stares, fright, child looks frightened, becomes pale, will not be able to walk, or walk with very great difficulty. Sudden drop attacks to the floor, fainting spells, but not fainting. Vestibular migraine is never associated with fainting.

Classical migraine might be, but not vestibular migraine. Clutching of head, that's the headache bit. Requiring silence and darkness. My child goes to a room which is to be switched off the light, no one can talk. Same thing the school tells you as well.

He struggles in school playground and he misses school. A travel sickness of recent onset. Sometimes triggers can be identified. For example stress, dehydration, significant physical exercise, challenging sensory environment or even periods. So head motion induced dizziness.

So these are typical vestibular migraine features.

Yeah, very true. Headache and dizziness might merge together because the child might catch the head in both instances. But usually in dizziness they don't catch the head like that. In headache they do a full comprehensive other history which we have covered before. But very importantly, performance in school and social circles is a crucial part of history.

Many a times I have to liaise with the school to tell them what to do when the child gets a vestibular migrant spell. You know, things do's and don'ts, for example, very complicated sensory environment, for example, playing in a maze, playing in a ball pool. That might trigger a migraine of. So school involvement is very, very crucial as well as significant others. That is the social, social, social circle.

So the emotional. There is a lot of emotional reaction which a child develops after a vestibular migraine and that is something which is far more pronounced in children than in adults. So full clinical examination, as I said, neurological, neurotological, audiological history, audiological examination I always do, because any dizziness is a vestibular thing. If it's a vestibular thing needs to be accompanied with a hearing test as well. But if you're absolutely sure there is no hearing loss, subjectively you might get away without doing it, but.

And then we do imaging studies in all cases and if I suspect a metabolic problem, for example, too much loss of blood in a female adolescent girl from periods, I would do blood tests often. Iron deficiency anemia will complicate the whole picture.

So once again, this is just a revision of just what I've told you. What happens to the vestibular function test? They are very variable. In fact, the literature is quite abundant. In adults, but in children it's not very well defined, but it is generally unremarkable up to 90%.

And if it's even it's present, it's very, very mild. It's nowhere as severe as in peripheral vestibulopathies. So you might get a mild unilateral caloric paresis and directional preponderance abnormalities. Might get a lobby organ, an absent wimp, subjective visual vertical tilt, some problems with rotatory chair, for example, the face or in the simple harmonic sinusoidal harmonic motion and central eye movement abnormalities. However, you might get them in at least 50% because at the end of the day, the common circuitry is at the fourth ventricle or the third ventricle.

So. But it's also important to remember that vestibular function test might show excitability, which means you might get a high threshold, you might get a high amplitude and a low threshold, just like a semicycle canal reheance in vestibular migraine, or you might get a high VOR gain. So that might also happen. So it might go either way. And not, not many studies have been done and it's very, very variable and heterogeneous.

Whole lot of differential diagnosis. Posterior fossa tumor, peripheral vestibular disorder, neurological conditions, idiopathic intracranial hypertension, gastrointestinal disorders, torticollis, orthostatic hypotension, temporomandibular joint syndromes, psychogenic dizziness is once again a diagnosis which has to be taken with great caution. In our series, it's less than 5% and it has been reported to be very high. But I don't buy that because most of the publications, they haven't been very comprehensive in their vestibular testing. So as you can see, these differential diagnosis, they need to be excluded.

And the most dangerous, this is what intracranial structural region for that imaging is supreme. So you have made a diagnosis. You are pretty confident it's a vestibular migraine. How do we treat this child? So this is a general algorithm.

You determine the phenotype, quantify the system, diagnose, and then go for different things. Not just treating the vestibular migraine with medications. Zap, boom, gone. No, the child matters, not the vestibular migraine. So what the vestibular migraine is doing to the child, that need to be addressed as well.

So what we call a holistic approach. The first step is. So just let me just summarize it before I go through. So lifestyle changes, identification of triggers, skipping diary Counseling, tell them what's going on. Reassuring psychological support.

Identify the different places where he might be predisposed for a vestibular migraine. Dietary modifications Dehydration is a very important cause of vestibular migraine. Often after exercises, a child gets a spell and that's because he has forgotten to drink during the exercises. You'll have to involve everybody around the child. So it's not simple, guys.

Vestibular migraine children is not a simple thing to treat. Pharmacological management in some instances where there is a documented objective vestibular weakness, you can try vestibular rehabilitation. And the condition is usually self limiting, but you'll have to treat it because of the distress it generates. So it's not a dangerous condition. Let me reemphasize.

It's not a dangerous condition, but it messes up the life so badly that as a physician you need to do something.

So first let me talk to you about the cognitive approach. So these guys, they often come to me after visiting many, many, many, not many, many, many consultants and many specialists without an answer.

So the key element to treat a child with the vestibular migraine is to understand how the child is reacting to the vestibular migraine. The child is asking him questions, the child is asking mom the question. The parents are very, very anxious. So what is going on through their minds? That's a key component of starting management.

So you need to first debunk the myth, tell them what's going on. So if the child is old enough, he'll understand. If the child is young, then the parents will understand and they'll try to regulate and modify lifestyles around the child. And a concrete diagnosis, in this instance, it achieves cognitive closure and that in itself achieves a huge sense of well being. I have seen parents and teenagers and just, you know, between 6 and 10, not 6 and 10, 10 and above, kids and parents burst into tears when I told them that this is a simple condition.

It's going to go, trust me and I can treat it. That in itself leads to a cognitive well being. You might think this is not exactly medicine. I would beg to differ. This is definitely medicine.

This is when you are seeing things beyond the actual pathology. And that's exactly how a human body and a human mind react. I mean, look, ask yourself if you get a common cold or if you get a cough which is not getting better. After four weeks, the first thing which hits your mind, am I having cancer? High.

Very, very huge uncertainty. You go to a respiratory physician who tells you the verdict? Yes, it's cancer, then you look forward to treating it. He says, no, it's not cancer. You get reassured.

What will it do to your mind? It will give you your mind, that peace of mind. And that's absolutely important to understand this cognitive illness, cognition of any disorder. And believe me, in kids and their parents, this leads to a significant improvement in quality of life. This is not intervention with medicines or surgery.

This is something quite a different at a very, very abstract and spiritual level. And I think it is very important for all doctors to practice this. And occasionally in severe psychological distresses which I have seen CBT might be tried, which I have tried successfully in a few, few kids. Lifestyle changes of course are very important. Ask them to maintain a diary, give them counseling of how to cope with stress, relaxation, coping with tiredness, coping with busy sensory surroundings, the right transport to travel, a transport which is easy and friendly so that it doesn't precipitate a spell.

So this list is not exhaustive. There are many but very importantly adequate physical exercises with a lot of water hydration.

So it is important to consider this life sizes because it does prevent spells. But at the same time, remember it must not come at the expense of childhood. So a school provides beautiful sensory stimulation with mazes, with different things. Everybody is playing and the child because of his vestibular migraine can't play. Now that is a severe dent on childhood.

So you'll have to mitigate this. How to mitigate this? Do you ask the child to avoid. No, you don't. You ask the school to take adequate precautions when such a thing happens to make it a more friendlier atmosphere or even before the child goes to play, give him some pharmacological intervention.

It can be mitigated. When I tell you this, I have done it, it can be mitigated. So do not deprive a child of his childhood unless absolutely you don't have a choice that's different. In that case, the pathology becomes paramount and not the child and not playground activities. Now this is crucial and the crunch of treating vestibular migraine, pharmacological management medicines.

And it is a quite a complex thing and it's a time consuming and almost like a trial and error. So there are two types of medications which is an, which is to abort the spell. A breakthrough drug which provides

relief during the spells. So a child, when he gets the spells with hugely distressed, you do give give a breakthrough pill. Now remember, if the spell is too short lasting, for example 2 minutes, there's no point in giving a pill, it's only for those spells which last for hours to days, and a prophylactic drug to prevent the spells from happening.

So you do not give prophylactic drugs for everybody. Because I don't believe in drugs drugs. All of drugs have called side effects, so I try to avoid them as much as possible and I'll only use it when absolutely indicated or pushed. So to me, for our alder hay, the indications for phylaxis include more than three attacks in a month, severe distress which is restricting the lifestyle of the child with withdrawal, and a female sex that is in adolescence or puberty. So we have to consider side effects and contraindications and regular updates every eight weeks.

When one drug doesn't work, you go to the next second one that doesn't work, go to the third one and try and carry on. So it's like a trial and error. And also one of the indicators that the drug is working is one of the vindications of your diagnosis. Vestibular migraine. That the drug is working is called a therapeutic trial, which means if the drug works, then you're diagnosis is very much tenable.

But research is very limited in vestibular migraine treatment in pediatric population. So this is the table that I prepared for you. These are different drugs. So as you can see right from the beginning, we start with breakthroughs. So procroperazine, triptans and rihergtamine in classical migraine triptans are extremely effective.

However, in vestibular migraine what I found, prochloprazine is more effective than the triptan. You know the reason why? Because the vestibular nuclei are heavily expressed with dopamine as a migraine trigger. And prochlorprazine acts on dopamine. So I would use as my first line of breakthrough abortion of a migraine spell a prochloperazine rather than a triptan.

And then of course you can use NSID and dihergotamine. I haven't used dihydroergetamine very much in children, but I've used nsaid. And. And then you come to the. We shall take the questions right at the end once.

So just keep your questions ready. We shall keep your questions right at the end. And someone has raised, raised her hand. So then comes the regular migraine treatment. So these are the different drugs and I have put them on the hierarchical fashion which I use.

So I start with the propranolol, provided there is, there is no contraindication, followed by bizartifen topiramate, cinerazine, flurazine, tcad, sodium, valproate, gabapentin and I might do some adjunct therapy with riboflavin, zinc and magnesium. I'm not going to read the different mechanisms of action but they all work at the trigeminal vascular level or on the cortical spreading depression. So as you can see out here, the most effective drug in my book are two One is flunazine because it has got multiple actions and the second one is topiramate increases GABA and inhibits glutamate. However, I can tell you that 80% of my children would settle with simple propranolol because propranolol is also an anxiolytic and there's a significant anxiety which is associated with which is associated with which is kind of which is with Estella migraine in children.

So how do you monitor outcome? Improvement of distress, frequency and duration. Ask mum or the child to keep a diary. Reception of daily activities the child who was unable to go to school. The child who was unable to mix with friends finds himself more and more engaging in these activities.

You'll sometimes you'll have to take a school report and vestibular function test. I have seen in few instances where they show definite recovery after after starting on vestibular profile after starting vestibular migraine profile Axis vestibular rehabilitation should not be a blanket treatment and it depends on the lives of the children proper quantification of vestibular weakness. My practice is I did do not use vestibular rehabilitation exercises if there is an active vestibular migraine going on. But if this

migraine comes with vestibular weaknesses then I'll probably try this. The reason being any active condition in the inner ear or vestibular system does not respond that well to vestibular rehabilitation until you treat the active condition first.

And the rewarding thing is the majority of children VM is a self limiting condition and and pharmacological management with cognitive approach and lifestyle lead to a very very favorable outcome in children. The chances of recurrence are present when you treat go back to treating them again. Vestibular migraine in a child carries an increased risk of developing BPPV and we have identified BPPV which is mighty rare in children unless following a trauma with vestibular migraine slightly older kids. 3pd is rare in children and it can and in VM it can still happen but I have seen maybe about 2 or 33 pds following vestibular migraine in teenagers. This can be avoided by meticulous follow up and by choosing the right medication.

In other words, you do not let the vestibular migraine proceed to that stage. But there is an increased chance of these children, when they grow up as adults to be hit by a classical migraine. Studies are lacking. This is an offhand figure, but it can happen, which you might have to cancel because often they ask what's going to happen in the future. So let's come to a few examples.

Child with an acute disease spell followed by recovery, but persistence of episodic spells, typical, typical history. Vestibular function tests are beautifully normal, but the VR gain was high. And we picked this up with vng, which is a very classical upbeat nystagmus. So we scanned scan was completely normal. So this could come from a central nervous system problem, of course.

And we treated this teenager with propranolol with excellent results. She was a skier and she went back to skiing.

This kid was with undiagnosed proximal myopathy, severe ataxia, positional dizziness. So typical central symptoms along with vestibular migraine features. And we checked his systems. Vestibular battery was completely normal. Scans were normal.

Partial response to vestibular rehabilitation because of the central. But the migraine completely went with bisotifan. And when we looked into his eyes, we found a very clear gaze evoke nystagmus which was bi directional, huge movements with rebound. So we made a diagnosis of vestibular migraine as well as something going on in the neurological system with a severe psychological reaction. We did some genetic testing and this child was a CAC N1 episodic ataxia type 2 mutation.

He is pretty much out of his vestibular migraine. Child with an acute headache and diagnosed with third ventricle aqueduct of Sylvia stenosis. He had an emergency third endoscopic third ventriculostomy and presented with typical features of vestibular migraine with a severe, severe, severe emotional distress. In fact, he was so upset and so distressed that he refused to do anything. He was a teenager.

So vestibular function tests were beautifully normal, but the V organ was slightly low. And I picked up this beautiful nystagmus, which is a perinose nystagmus, which is upward convergence retraction. Nystagmus. Let me see. There it is.

There it comes. So convergence retraction with upward gaze, upbeat nystagmus. So we picked up an parino nystagmus and then we treated the migraine very successfully. And of course this is the Severe distressing physiological psychological reaction was because this lesion, the aqueduct of sidious lesion, was very close to the periaqueductal gray which is one of the top emotional centers. So we reacted.

I've picked up loads of some exotic pathologies. Heterotrophic calcification of the third ventricle leading to severe psychological distress. Corded nuclear cyst leading to severe psychological distress. All presenting the stella migraine which is very common. But the distress or the emotional reaction will tell

you if there is something going on in the central nervous system.

So a very quick word about our series which we have published in the archives. So we studied all our pe, all our kids with vestibular migraine for 12 months, total cohort 81. And there was. We, we picked up 1/5 of our children with dizziness with vestibular migraine. We followed them up and 38, 40% are vestibular migraine.

Probable VM was similar and BPVC was in about 15%. We also picked up cyclical vomiting and other type of migraine and these were pretty much based on Bahrani Society criteria. However, we were flexible in our duration as I have mentioned before, frequency of episodes, as you can see, it's pretty much heterogeneous. But the predominant was, you know, it comes every day, which really, really, really is very, very distressing. So in our series what we found was the VOR gain was uniformly high in most children.

So we picked up hyper excitability vestibular migraine rather than low one, except the one which I showed you before. And about, about 40% of our children had normal examination. But central neurological eye movements were found in quite a few cases. And 80% we tried profile acting midoctum medication when 84% improved. And that is a fantastic vindication of the algorithm which we have created.

And we also took them through situational counseling, cognitive counseling as well as lifestyle changes. And we involve the school and the parents. So this is a very rewarding outcome.

So as you now appreciate that, it's a very complex condition to diagnose and it demands a very high clinical set because you really have to elicit the history because vestibular migraine is a diagnosis based on history. So you'll have to ask the parents surrogate history. It has to be flexible and open because sometimes a vestibular migraine in a child might not fulfill the criteria which has been laid out.

So you must have the clinical acumen and clinical eye to make the correct diagnosis by putting two plus two together. And I don't think centuries and millennia will pass that AI will be able to replace this human wisdom.

No, it's your wisdom, it's your clinical judgment which is absolutely crucial. Vestibular function tests are mandated and indicated and they may show variable results. So it shouldn't be too much relied upon. But imaging is very, very important and the best thing is if you make the right diagnosis, treatment and the future is extremely bright for the people, for the, for the. For your patients and their carriers.

So I finished the lecture and you are most welcome to attend my pediatric vestibular course in October 2025, 20th and 21st of October. Please get in touch with me through Inventis. And many of you actually have come here last year and this is a beautiful city of Liverpool where I hosted in my hospital and you see the delegation last year and my vestibular lab and my hospital and the surroundings. So it's a very, very beautiful place. We have recently floated an international pediatric vestibular network.

Ten of us from all over the world and our website is running www.ipbn.net. please visit our website and we would be asking for memberships shortly. So this is the first initiative of its kind anywhere in the world and it looks very, very promising. We plan to organize a conference soon. Recently my fellow and now an established consultant with a lot of interest in pediatric vestibular medicine in Kuala Lumpur in Malaysia, Jayasakti.

She has edited a book published by Springer where we have contributed to different aspects of vestibular migraine. I recommend please either buy this book or, or read it and download it through your institution logins. This is a most comprehensive treatise on vestibular migraine in children that you can ever find. All written by pediatric vestibular specialists like Josien Vidashauvin from Maastricht, Sharon from Canada, Toronto, my colleague Dr. Adnaiki Jayasakti herself, myself, Jacob Brodsky from from Johns Hopkins and Harvard.

You know, they are all here. It's a very, very, very exhaustive with lots of evidence and really, really I would recommend that you read this book. And finally going back to vestibular history, medicine history again we published on Vincent van Gogh where we analyzed 903 of his letters, translated them from French and from Dutch and we showed that he fulfills Brahmani's criteria of vestibular migraine and adult. I have made this film for you just to tell you how intensely distressing this condition is. When it happened in Van Gogh, his art changed.

So I'm going to play a song. A very famous Song about Vincent van Gogh's son by Don Maclean. But in his paintings that you will see, you'll see all his figures are tilted to one side, to the left. And that is a subjective visual vertical tilt which a vestibular migrainer faces. Enjoy the song.

Paint your palette blue and gray, tilted. Look out on a summer day with eyes that know the darkness in my. So the inner turmoil that it produces. Vestibular migraine. So it's a very important diagnosis.

It's a complex diagnosis which has to be made in all cases, especially in children. Thank you very much.

Right. And as Longo finished. Oh, hello. Yeah, thank you. Thank you.

Thank you really for this marvelous lesson. I. I understood several aspects of super migraine children that I.

I didn't know. And I also understood that Bharani criteria have many shadows. So probably we don't go there. We don't go into controversy. Salvo?

No, no. Controversial, but too many unclear aspects. I have some questions for you, but Professor Dasgupta, I must read some question from our attendees. The first one by Maria Laura Esseveri from Argentina. I suppose in your experience, do you found useful the vestibular evoked biogenic potential in distinguishing vestibular migraine and many diseases?

Well, that's a good question. I don't think that there is much distinguishing because both might present with. Both might present with vamp reduction of amplitudes. The one difference which I found is latency in hyperexcitability vestibular migraine. The latency will actually be shortened in case of vestibular migraine and not in md.

And the other thing which I found in MD is this vamp tuning which you can do in md, but it's not indicated really in vestibular migrant. So overall I wouldn't kind of. I wouldn't kind of say this is an MD if I find an hyper excitable vestibular migraine. Let's put it like that.

Okay. And another question from Simon Mary, Simon Mary ask, are there any volumetric change noted on MRI scans correlated with the vestibular migraine children? I don't think so. And moreover, the other thing is important to understand is why do we need to see the volumetric changes? I am doing a scan not to confirm vestibular migraine, but I'm doing a scan to exclude neurological structural abnormalities which can range for anything between tumor to atrophic calcification.

So I'm not really interested in the volumetric changes. And as far as I know, in the pediatric population there hardly has been any study There is no study. So do you perform in any case scan also? This is scan. Also CT scan.

Ct CT scan. I will not do to begin with, but if there is an obvious third window feature, for example, you know, vestibular migraine, the hyper excitable state with low thresholds and high amplitude, I probably, probably will exclude a third window. Probably. But usually it's always the Mr. Scan.

Because as I said, the objective of the Mr. Scan is not for a biomarker. The objective of the Mr. Scan is to exclude something more dangerous or something sinister going on. Yes, yes, I agree with you.

But in Italy, in Italy in particular, we have some difficulties in performing MRI on a child under 6 year old. Sure, it is possible, but with one day hospital stay, you know. Yeah, we do a general aesthetic. Yeah. In some case, in some games, maybe we can postponing the.

The MRI or not, in your opinion? No, I will do an Mr. Scan. The thing is, because under the age of four, they end up with neurology first. They already get an Mr.

Scan done before they come to me. So if I get a virgin case, let's say the first time that I'm seeing, right, with BPVC like picture, under the age of four years, then I'm going to. I'm going to treat that child from the BPVC point of view and monitor the response. If there is a response, then I'll defer the Mr. Scan.

I'll do it later, but I'll do it. No mistake. I'll do it, but I'll defer it. And if. If there is no response, then yes, I'm going to ask for an anesthetic.

And another question. I am. I'm reading the. The questions in order also by Simon Mary. Have you used a taurine?

Have you used a taurine? It's interesting. No, I haven't because the pediatric. You know, I keep on saying these are all adult things in pediatrics. There has been no trial.

No, no. So we can't use taurine. Taurine is taurine. Makes a lot of sense to me, but I can't use it. No.

So we can, we. Can you cure the vestibular migraine children with. Okay, we need studies. Not with the rebel. Another question by Sosan Abu Zaid.

I'm sorry, my friend, if I read your. Your name in a bad man. At which. At what age can we give children with vestibular migraine the supplements including magnesium and riboflavin. Riboflavine.

And what are their doses for children? Well, the thing is that magnesium mainly Riboflavin is licensed in the UK. Okay. For vestibular migraine at 400 milligrams. Right.

I would use it for teenagers, but I haven't used it for children under the age of 10 years. And to be very frank, I hardly use magnesium. I don't think I need to. But yes, you can use it. Okay.

I wouldn't. I don't. I usually don't use magnesium because I achieve a result before magnesium. But in some instances when it's very refractory, riboflavin does work. So the dose here is 400mg, if I'm not mistaken.

Offend. Yeah. A personal question. What prophylaxis. What prophylaxis would you recommend for a four year old child with very frequent episodes?

For a four year old child, four year. Yeah. So I will. I will use propranolol. Propanol.

Yes. And I would also use a breakthrough medication if the spells are longer lasting. Proclo. They are licensed in this country under the age of four years. Yes.

What dosage? So for under the age of four years, it depends on the body weight, but on an average you can go up to 10 milligrams twice a day. How many milligrams? 10, 10. 1 0.

10. 10 milligrams twice a day? Twice a day. Okay. You can use it from the age of two years actually.

Well, it depends on how he responds. If he doesn't respond, you'll have to go to another medication. Okay. Okay.

Another question.

Thank you. Thank you. A lot of compliments for you, Professor. That's good. Oh, thank you.

When I'm suspecting vestibular migraine children, what would you suggest is most helpful to include in my report to better guide physician? Particularly in encouraging consider consideration of other differential and prompting imaging if red flags are present. So I think, Yana, that's a very, very good point. I think what you need to tell your colleagues is Bahrani's fourth criteria. Has it been excluded or not?

So if you write that in your report that Bharani's fourth criteria is being excluded, normal vestibular function tests and everything, or not. If you can actually put it in your report. Actually, I think it would be helpful if you can put in your report all the four Bharani's criteria, you do a table and just fill it in as you go along, your colleagues will then pick it up.

In other words, write to them that you are suspecting vestibular migraine and why. Okay. Another question. If a patient has classic migraine Symptoms associated with dizziness and vestibular evaluation indicate both central and peripheral vestibular involvement. Should we diagnose it simply as vestibular migraine or as a vestibular migraine?

Second central involvement with concurrent peripheral vestibular dysfunction. Okay, so as I said, vestibular dysfunction. I'm not very sure exactly what you mean by vestibular dysfunction. Vestibular

dysfunction is a broad umbrella, which means that even a deranged VOR gain is vestibular dysfunction. So if you get V gain abnormalities only.

Right. With this kind of picture, then you can probably say that this is a vestal vestibular migraine. It's superfluous to make a distinction with central because vestibular migraine at the end of the day is also a central involvement. Vestibular migraine is just not limited to the periphery. So you can say vestibular migraine, that's it.

But if you find an obvious vestibular weakness, for example, the VOR gain accompanied with saccades, which is a weakness or a significant asymmetry in the WIMP test, then of course you'll have to figure out why this is the case. So you'll have to actually actively seek for different etiologies which can explain a vestibular weakness. And you'll be surprised. You will pick up a few. Now, if you are happy that there is absolutely no etiology found, then you'll put it down as one of those rare instances where vestibular migraine has actually generated a vestibular weakness.

So the diagnosis for you will still be a vestibular migraine with a peripheral vestibular without a peripheral vestibular weakness. Okay. Nilopat Dutta say you hello because he attended your course in 2022 in Liverpool and his question is, what is the duration? We use the migraine prophylactic medication in a child you as well before. But yeah, okay, so the duration is pretty much case by case basis, which means that initially you would put the child on a medication for eight weeks.

It's very important you take an update after eight weeks and you go through all the different parameters of a response, for example, the diary, the distress, the frequency and the duration. If there is a tangible change for the better, then you continue. You continue it for further eight weeks and at the end of three months, you take an update every two months. At the end of the third cycle of two months, you then see that the spells have completely disappeared. Then you wean, you taper the medication for a period of two months and Then finally, if the child is spell free for three months, you call it a day and discharge.

So overall treatment duration in the best possible case scenario is three, two, three, eight months. So you'll have to continue to follow up the child for eight months and that's quite long. Now Kovit has taught us that nowadays they don't come to me, I talk to them on the telephone. Easy to do. Easy to do.

Easy to do, yeah. Another question. In cases of intracranial pressure related headache, I think salvo, we have missed one. Would the treatment approach differ between BPVC and vestibular migraine? Oh, I'm sorry, I'm sorry, please, I can, I cannot.

Yeah, so this is a very good question because you see, you do not have the luxury of trying 12 different drugs for BPVC because many drugs are not licensed under the age of six. So yes, the principles are the same, but the drug choice is limited. For example, in chick kids you can use prochloperazine, you can use propranolol, you can use valproate, but topiramate and all the other drugs, they are not licensed, at least in this country. So principle is the same. But that might not have the luxury of so many drugs.

Okay, I'm sorry. I know, that's fine. Don't, don't worry. The question, a question, a difficult question by Mehmet Kadir Khan. How can we detect, understand the effect of vestibular migraine on the vestibular nuclear AI without any pathological vestibular function test results?

Okay, so let me tell you a very basic fundamental physiology. There is a thing called a physiological asymptote for both cochlea and vestibule. That is defined as the period of time where the system can restore its function even if assaulted to normal. And offhand, on an average this is between 16 to 48 hours for both vestibule and, and cochlear. Now imagine vestibular migraine focus which is hitting the vestibular system either in the vestibular nuclei or elsewhere.

And it doesn't last for this period of time. Then the system will restore and you will not have any pathological vestibular function test results. The important thing to understand is the vestibular nuclei is an important spot, an important trigger for the vestibular migraine. If it lingers on for more than 48 hours, then yes, you can get a vestibular hyper function or a weakness, but it seldom does it as a result. In fact, the criteria also tell you the same duration as a result you do not get a permanent damage.

Which is very clearly understandable because 90% of our vestibular migrant kids, they do not show any pathological vestibular function. Test result. Does that make sense? Albo? The system restores.

System restores. In other words, vestibular neuritis, for example, it goes on beyond 48 hours. Right. And the system will show a permanent damage. Frequent spells of many years will show a permanent damage.

But vestibular migraine, because it's short lasting, it doesn't lead to a permanent damage. However, I might tell you, Salvo, that we have seen kids during the spell where you do get vestibular weakness, probably in vestibular neuritis, in my opinion, the damage take the elevator and the recovery take the stairs, you know. Yeah. In vestibular migraine, the damage and the recover. Take the Levito.

That's right. But I have seen a few kids during vestibular migraine spell and there I have found out vestibular weakness. But that's not permanent. That's most important to know. That's not permanent.

It restores completely because it doesn't, it doesn't extend beyond the human asymptote. A question, a personal question. Do you ask the parents to record some video during the spells? I have asked them, but so far only two have. So they usually don't?

No, they usually don't.

They are nice and often in the. The main, the main proof of the vertigo spell. Nystagmus. Intercritical nystagmus.

Another question, a long question. In cases of intracranial pressure related headaches caused by improper venous drainage from brain, which potentially led to cochlear vestibular symptoms resembling endolymphatic hydro? Should the clinical focus be on identifying migraine or intracranial pressure related headache characteristics or should it primarily emphasize the assessment of cerebral blood flow dynamics? So if it's a case of intracranial pressure related headache because of impro. Improper proper improper venous drainage, then this is hardly likely to be stable migraine because it does not fulfill Ban's fourth criteria.

Remember, you need to exclude other conditions. So here your clinical picture suggests that this could be a problem with venous obstruction in the brain. So your entire investigation, including cerebral blood flow dynamics, will be geared towards that. So this is by definition not vestibular migraine because it does not fulfill Merani's fourth criteria. However, the two can coexist, of course.

Yeah. Another interesting question for me by Simone Shiraz. Simon Shirazi. Would you comment on your experience on Vestibular migraine in children already diagnosed with autism. This is a very difficult one because, you know, history is so difficult, even from the parents, you know, because it, you know, the behavior in autistic kids is so much variable and heterogeneous, you often do not know what's going on.

So eliciting a history is very difficult. But I have seen that in lower grade autisms, for example, in high functioning autisms, vestibular migraine can definitely be encountered. So as I told, as I said in that slide, autistic kids have more double incidence of vestibular migraine. So I have seen quite a few. But it's very, very, very difficult to diagnose because you won't be able to even examine the patient properly.

So, you know, it's like almost like an empirical diagnosis, but it can be done. You know, just have to keep your mind open really. Another question, by nil. Is it safe to use prochlorperazine as it has a lot of anticholinergic and dopaminergic effects? Good point.

The quick answer is not for prolonged period. But if you are using it for abortive attempts, then yes, you can use it for a continuous of five to seven days. After that the half life dictates it eliminates. But if you use it for more than a month regularly, then yes, you're in for trouble. Especially dystonic hand trema.

So dictum is do not use proclopirazine for more than five to seven days continuously. Okay. And if you are using it as a, as a, as an, as an, like, like a paracetamol or an ibuprofen, you know, just three a day only during the spells. Then you are absolutely safe. No problem.

Okay. Okay. So. So a week is the maximum week continuous. Maximum week, yeah.

Dr. Tapas Basu, I have a case. Five year old boy in spectrum having nystagmus hyperphagia. But the child is having an issue since the last three years. After eating food, he tries to vomit, but his BMI is at the 50 percentile.

Mother is having severe psychosis. Child has gone through trauma by parenting issue. They have visited so many pediatric specialists, but didn't work out. Currently by examination founded severe constipation, kidney stones. It can be abdominal migraine.

I would say no. Me too. But I need more information to give you an accurate diagnosis. But it's not abdominal migraine. I agree with you Salvo, but I need more information.

Especially I'll be very interested to find why this kid has an hystagmus does this kid really have an astigmas? If so, why? I think this is behavioral. Don't you think? Abdominal mother is having psycho says his BMI is 50th centile.

I don't know.

Oh, this is a nice question. Oh know nice question, but I. Oh, okay. What are the most triggers of vestibular migraine of children? Okay, I'll tell you.

The most common stress trigger for a vestibular migraine in children is dehydration. That's the most common one. And then of course comes in succession things like excessive physical activity, overloading of sensory stimulation, stress, hormonal changes and all these things. But in my experience, anecdotally at least the commonest trigger of vestibular migraine is dehydration. They don't drink enough.

They don't drink enough. No water or water.

Not the fuzzy drinks. Salvo. They are bad. Plain and simple. Water.

Okay. Role of C proeptadine in BPVC. So ciproeptadine can be used in bpvc provided there is an accompanying travel sickness. So I would use it not regularly. But if there is a travel sickness then yes, I will use cyprobeptadine in some instances is safe.

Well, so far I haven't found any side effect. I'll try to avoid it as much as possible possible. You see Salvo, I'm not very fond of drugs so I achieve very good results with just propranolol and prochloperazine in bpvc. No, I don't have to use pro pro because usually the paroxysms are from seconds. So we don't need to about that.

But propranolol completely turns the life. And remember BPVC is self limiting in the vast majority. It will go so and guarantee. Yeah, counseling the the best way. Absolutely.

Fat maragab. What about tricyclic amines? Now this is a very good drug in adults tricyclics and I have seen teenagers with tricyclics, not ripline. But my experience with tricyclic is not good because of its anticholinergic effects. So if other drugs don't work, I might try.

But so far I've been lucky. In my new numerous thousands of vestibular migrant kids I did not have to use tricyclic amines. It's my seventh or eighth line salvo. I've got seven other drug drugs to try before that and one of them works.

Okay, probably I have to choose some question because we are. We are in closure.

According to literature there is no Crossing error with thalamus in terms of vestibular pathways. Based on this information, could we assume that vestibular migraine symptoms are less severe if the origin of migraine lower than thalamic level? What I mean is, could we say that vestibular migraine symptoms more severe if the problems originates from hemispheric level? I'm not very clear about why this question is being asked about pediatric vestibular migraine. We have clearly seen that a subthalamic vestibular focus or a circuitry is in the developing state.

And that's where the vestibular migraine focus in a child is. And that's why the symptoms are so distressing, which I've clearly, clearly mentioned. And I'm also not sure as to what is meant by no crossing area. Do you mean crossing area from one side to other? Because the vestibule of thalam, the vestibular pathways are very, very strongly represented in the thalamus.

There are loads of vestibular thalamic pathway. So I'm not very clear as to what this question is meant. I can tell you from the pediatric point of view, the problem does originate from below the hemisphere at a region which is developing with neurotransmitter control. And if this region is upset from the excitatory and inhibitory balance, then the problem will be far more intense than an adult. If you're talking about adults, then it's a different topic altogether, which I'm more than happy to discuss later.

But from the pediatric point of view, I don't think that I would agree to this statement.

Okay. And by a pediatric point of view, this question is more practical. Is it possible to use fluoride design in children and how long and what doses? Yes, it's possible. What do this and how long, please?

This is which one? Fluorozine. Yeah. Fluorescent is a magnificent drug and it's not licensed under the age of 18 years in this country. But we as specialists can recommend it.

And usually we start at a very low dose. So under the age of 10 we would use 5 milligrams at tea time. And over the age of 10 we will use it as 10 milligrams at tea time. It's a magnificent drug. However, it's got lots of side effects.

Alvo. So it's not my first choice. Not. Yes. You prefer propanolol?

Yes, propanolol, topiramate, cinerazine and much later. Yeah. Benign torticolis. In childhood it is postulated to be a migraine variant. Yes, the Last question.

Benign torticollis in childhood. It's a migraine variant. Yeah, it is a migrant variant. At least it's postulated to be a migrant variant. Okay, now maybe the last.

Last or not? Not. Yes, the last one. Salvo. Thank you.

The last one. A lot of questions. A lot of questions. No, that's good. Keep the questions coming.

That's good. Absolutely. Professor, we have time for the last one. What about trip tons? Yeah, triptans, they would work if propy present fails because I.

As I've told you, the mechanism of action are different. So yes, trip tons can be used.

Okay, the last. And that's it. So what can I say? Thank you for attending. And so just one, just one thing, Anna.

There is a question about serotonin level. What is the normal value of serotonin level for children based on your clinical practice? Now, Mehmed, I think we met in Budapest. Is it clinically important or pertinent to know serotonin levels? Because it doesn't change anything.

But yes, it does change beyond the age of four years. But why do we need to know serotonin level in vestibular migraine? From the clinical point of view, how does it change? Are we going to give the kids serotonin supplements? Because excessive serotonin might mess up the whole thing.

So I'm not very clear as to what is the point of this question. Okay, I think that's it. I think that's it. Okay, thank you. Thank you.

So thank you all for attending. What an inspiring session to conclude this series. Professor Gupta and we are truly grateful for your contribution to invent this academia and. No, thank you. And to a wider audiology and neurotology community, thank you so much.

Thank you. So yeah, next, next year, similar. Yeah, sure, sure. Pretty sure. Thank you, Salvo.

We also thank Dr. For joining us today and enriching the discussion. So looking ahead, our next Academia webinar will take place on April 22nd featuring Dr. Vishal Pavar from Dubai. This will be the second in his series of three webinar on the whit titled the Fundamentals of a Video Head Inputs Test.

Lesson number two, Clinical application and Interpretation. So stay tuned for more updates by visiting the Academia section on our website and following us on LinkedIn and other social media channels. Thank you once again for being with us and see you at the next session. Thank you. Bye bye.