

Headache and dizziness: two independent symptoms or different spectros of the same disease

Abstract

Migraines, dizziness and vertigo are common in the general population, not uncommon, to be interrelated. The interrelation between migraine and vertigo, however, is complex, since they can coexist in the same individual at random, since they are common conditions in the population and, in addition, several syndromes in which vertigo and migraine are present are demonstrated. Vestibular Migraine (MV) is a recently described entity with a broad approach and difficult diagnosis. MV was defined as a nosological entity by IHS only in 2013, in its last consensus of neurological disorders. It is defined by the presence of episodes of headache classified as migraine concomitant to vertiginous symptoms of moderate to severe intensity lasting from 5minutes to 72hours. The most accepted diagnostic criteria were proposed by Neuhaser et al.,¹ which classify MV as definite or probable. It is well known that it may overlap with other entities, which makes it difficult to diagnose and indicate adequate therapy. Thus, in our study, a literature review was carried out using articles from the last 15years, which sought to evaluate whether exams such as magnetic resonance imaging of the inner ear, myogenic potential evoked by the vestibule by sound induction (VEMP's) and videonystagmography, may help in Differentiation and also the effectiveness of prophylactic treatment, from the change of the habits of life to the use of drugs indicated for this entity.

Keywords: headache, common migraine, vertigo, neurology, vestibular myogenic evoked potentials, magnetic resonance

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Introduction

Migraine, dizziness and vertigo are common in the general population, not infrequently interrelated.²

Migraine is defined by the IHS³ as a disabling, unilateral, recurrent, intermittent and pulsatile headache associated with nausea, photophobia and photosensitivity. Along with these typical symptoms, migraine is often associated with vertigo.⁴

Vertigo is caused by dysfunction of the vestibular apparatus and manifests itself as an illusion of movement, usually of the rotational type. Other symptoms may also be present, such as nausea, vomiting, tinnitus and hearing loss. It is differentiated from other types of dizziness, questioning the patient if he has the sensation that the objects or the environment are spinning around him (objective vertigo), or if he has the sensation of the head being turned (subjective vertigo).⁵

The interrelationship between migraine and vertigo, however, is complex, since they can coexist in the same individual at random, as they are common conditions in the general population and may be present in several syndromes, such as Benign Paroxysmal Positional Vertigo (BPPV), Ménière's disease (DM), motion-related diseases (such as motion sickness), cerebellar disorders, psychiatric disorders and also the recent entity called Vestibular Migraine.¹

MV was considered as nosological entity by IHS only in 2013 in its last consensus of neurological disorders. It is defined as the presence of episodes of headache classified as migraine while vertiginous symptoms of moderate to severe intensity lasting from 5minutes to 72hours.³ It is estimated that the occurrence of both symptoms (vertigo and migraine) may be present in 1.1% of the world population. In addition, this figure may be under diagnosed, with the probable number being 3.2%. MV affects at least 1% of the general population and 10% of the population with complaints of migraine and vertigo.¹

Meniere's disease (DM) is characterized by episodic vertigo, fluctuating hearing loss and tinnitus. While MV is characterized by episodic vertigo or dizziness, coexisting with headache and absence of hearing loss. Thus, it is difficult to distinguish these two entities, requiring more specific diagnostic criteria to differentiate them and treat their coexistence.⁶

Clinically, vestibular migraine may present as spontaneous seizures or positional vertigo lasting from seconds to days. Migraine headaches such as headache, photophobia, phonophobia or any other types of auras are common but not mandatory. Cochlear symptoms may be present, but most are mild and non-progressive. In acute attacks one can also observe central positional or spontaneous central nystagmus and less commonly unilateral vestibular hypo function. Because of the absence of control studies, MV treatment is guided by migraine spheres in order to avoid triggers, stress management and also pharmacotherapy for prophylaxis and acute attacks.¹

Objectives

Primary objectives: Establish whether patients presenting with both headache and dizziness have a single entity that causes their symptoms or if they only coexist coincidentally.

Secondary objectives: Establishing the entity responsible for the coexistence of symptoms and determine the most appropriate diagnostic and therapeutic approach.

Methodology

It has been proposed an evaluation of which methods may be used to distinguish MV from other entities causing dizziness or vertigo, in which pulsatile type headache can coexist. For this, a bibliographic review was carried out, selecting current articles from the last 15years and with great importance for health professionals. Fifteen articles were found, which contained relevant pathophysiological, diagnostic and therapeutic aspects.

The following databases were searched: Pubmed, Scielo and Lilacs, using as Vertigo, Headache, Migraine, Vestibular Migraine, Vestibular (Lobby). The terms used as a key word were used in isolation or jointly.

The articles searched were limited to English and Portuguese. Of the 15 selected none were excluded from our review.

Results

Nakada et al.,⁶ performed a comparison of endolymphatic space in the vestibule and cochlea in seven patients with MV and in seven patients with vestibular DM, matched for age and sex, because endolymphatic hydrops (HE) was one Characteristic sign of vestibular DM, in an attempt to distinguish these similar entities. In order to evaluate endolymphatic space, patients underwent magnetic resonance imaging (MRI) with gadolinium contrast.

The results of endolymphatic space evaluation in patients with vestibular DM and MV are summarized in Tables 1 & 2, respectively.

Among the 14 ears of patients with MV, the number found with HE in the vestibule were: 11 without HE, 1 HE light and 2 HE significant. Contrary to this, among the 14 ears of the patients with vestibular DM, the number found with HE in the vestibule were: 0 without HE, 3 light HE and 11 with HE significant. Among the 14 ears of patients with MV, the number found with HE in the cochlea were: 5 without HE, 9 HE light and 0 HE significant. Among the 14 ears of the patients with vestibular DM, the number of HE in the cochlea was: 4 without HE, 6 with light HE and 4 with HE significant.

Kato Cited by Nakada⁶ evaluated images of endolymphatic space in 36 patients with cochlear DM and 28 patients with vestibular DM using intratympanic or intravenous gadolinium contrast MRI. In both groups of patients, HE in the vestibule was more common than in the cochlea, which strengthens the findings of the present study by Nakada et al. In their study, Gurkov et al.,⁷ aimed to investigate the presence of HE in patients with definite or probable MV, with hearing-related symptoms, to elucidate more clearly the relationship between MV and DM, due to the overlap of these entities.

The population studied was 19 patients with definite or probable MV diagnosis, according to Neuhauser et al criteria, with auditory symptoms (hearing loss, tinnitus or aural fullness). The mode used to evaluate the endolymphatic space was the MRI of the locally advanced inner ear (LEIM) and electrocochleography.

Of the 19 patients with definite or probable MV, four evidenced the presence of vestibular and cochlear HE in MRI. Three of these patients had been clinically classified with definite MV and one with probable MV.

Thirteen of the nineteen patients had a hearing loss of at least 20dB. This included three of the four patients with HE, the other patient had normal hearing. Tinnitus was the most common auditory symptom, being reported in almost all but one patient. Thirteen of the nineteen patients presented aural fullness, including the four patients with HE. In seventeen of the nineteen patients, the vertigo attacks lasted more than 20minutes.

Two of the four patients with HE presented photo and phonophobia associated with vertigo. Three of the four patients with HE also reported having visual aura. Of the four patients with HE, three presented clinical criteria for defined DM and one for probable.

Seven patients out of the nineteen selected had definite MV; of these seven, five also had definite DM, and of these 5 with both

diseases, presented both MRI and EcochG (electrocochleography). Interestingly, in this population studied, the EcochG showed evidence of HE in a larger number of patients (10 of 16), than RM LEIM, suggesting that MRI may be less sensitive when compared to EcochG. In the study by Baier et al.,⁸ myogenic potential evoked by the vestibule by sound induction (VEMP's) was performed in 63 patients, of whom 37 were women (mean age 42years) and 26 men (mean age 50years), With definite MV diagnosis (26 patients) and probable MV (37 patients). They were classified according to the Neuhauser diagnostic criteria, with the intention of determining if the saccular function is altered or not by the disease.

Patients who did not reach sufficient criteria for diagnosis, as well as patients with differential diagnoses, such as DM, BPPB or transient ischemic attack, were excluded from the study by physical examination, history and appropriate tests including electronystagmography, audiometry and doppler ultrasound Of the blood vessels (basilar artery). Also excluded were patients with hearing loss and mild headache. Thus, there were findings indicating that 2/3 of the patients presented reduction of the wave amplitude in the VEMP, concluding that the brainstem saccular pathways may be injured.

In the study by Brantberg et al.,⁹ 40 patients with recurrent vertigo episodes were selected and also met the IHS criteria for migraine. Patients with BPPV were excluded because they had positional vertigo attacks of less than 1minute. Other recurrent causes of vertigo were excluded through anamnesis, physical examination and audiometries.

A second group with 40 patients was selected, with a history of migraine in the family and recurrent episodes of vertigo. The groups presented female prevalence and half of the patients had visual aura and other symptoms. The group with defined MV presented fewer symptoms and of shorter duration when compared with probable MV, but the symptoms that accompanied the vertigo was similar in both groups, with almost all the patients presenting imbalance and nausea during the episodes. The most common triggers for vertigo attacks were stress and fatigue in both groups and women who were in the menstrual period reported a greater association of symptoms in both groups.

Furman et al.,¹⁰ performed a study including 15 patients, 5 of them with MV, 5 with migraine without dizziness and 5 controls (healthy). The mean age for the 3 groups was: with MV of 35years, with migraine without dizziness 31years and of the control group, 25years. In addition, 80% of the patients were female (4 women / 1 man in each group).

All patients were evaluated by a neurologist in order to establish the diagnosis of migraine, using the IHS criteria and the individuals with diagnosis of MV, according to Neuhauser et al criteria for diagnosis. Subjects who did not present active migraine (at least 2 episodes in the previous months) or control of headache were excluded from the selection process. Patients suffering from post-traumatic stress disorder, abusers of analgesics, patients with neurological complaints, or psychiatric disorders such as anxiety and depression were also excluded.

All the individuals were evaluated with vestibular tests when they were without manifestations of the symptoms and when exacerbation of the symptoms occurred. Among the vestibular tests were included: 1) Clinical examination otoneurological, 2) Videonystagmography, which consists of the evaluation of: A) Spontaneous nystagmus, B) Sacadic or semi-spontaneous nystagmus C) Ocular activity, D) Optokinetic nystagmus, E) Supine bearing F) Caloric test and 3)

electrooculographic recording of eye movement during the rotational test.

Videonystagmography results indicated that all patients with MV and all control patients were normal, had no changes in latency, accuracy and speed of the test; the ocular activity was normal, in addition, there was no asymmetry in the optokinetic nystagmus and none of them had spontaneous nystagmus. Two patients with MV and one patient with migraine without dizziness had low amplitude of persistent nystagmus, which did not exceed 4seconds in any position.

In the study by Baier et al.,¹⁰ we analyzed the effects of current prophylactic therapy for MV treatment, which aimed to compare duration, intensity and frequency of MV crises present in patients with And without treatment. A total of 100 patients were analyzed, of which 63 were women aged between 25 and 72years, and 37 men aged between 21 and 71years. All 100 patients were diagnosed according to Neuhauser criteria, with defined MV or probable MV. The criteria used for patient selection were 3: presence of vestibular symptoms of at least moderate intensity; History of past or current migraines that meets the criteria of the International Headache Society, and finally has one of the symptoms of migraine during 2 vertiginous attacks: throbbing headache, photophobia, phonophobia or visual aura.

Thus, 74 patients received prophylactic medication and 26 received non-drug treatment (Table 1). Of these, 14 received conservative

treatment and 12 had to change their lifestyle. Of these 74 patients, 10 exercised regularly, 8 had progressive muscle relaxation and 9 people changed their lifestyles. It is also worth noting that many of the patients studied used other medications that may interact with the therapeutic test medication: contraceptives, iodine, proton pump inhibitors, insulin, thyroxine, anticoagulant, hydrochlorothiazide, losartan and statins.

As a result, of the patients treated with the medication for 6months, 80% had decreased episodes frequency, 65% reported decreased duration of episodes and 68% reported decreased attacks intensity. As for the other 26 patients, who did not receive the drug treatment, 46% reported only improvement in the intensity of the symptoms.

In the study by Lepcha el al.,¹¹ the effect of flunarizine was investigated in patients diagnosed with vestibular migraine compared to its effect in patients with similar non-specific etiologies. A group of 48 individuals aged 18-75years diagnosed with vestibular migraine defined according to the Neuhauser criteria for a period of 12 weeks with patients separated into 2 groups: A and B. Group A was treated With acetaminophen (1000 mg for use in acute attacks), flunarizine (10mg once daily before bed) and beta-thistine (16mg three times daily). Group B, on the other hand, received only treatment with betaysteine (16mg) and paracetamol (1000mg). The groups had exactly the same number of people and it is worth considering that the medications were taken the same way by both.

Table 1 Evaluation of endolymphatic space in patients with vestibular DM

No.	Age	Sex	HE in Vestibule		HE in Cochlea	
			D	E	D	E
1	71	F	2	1	1	1
2	26	F	2	2	1	1
3	46	F	2	2	2	2
4	27	F	1	2	2	1
5	57	M	2	2	0	0
6	60	M	2	2	0	0
7	42	F	2	1	2	1

Source: Nakada [14]

Table 2 Evaluation of endonlinal space in patients with MV.

No.	Age	Sex	HE in Vestibule		HE in Cochlea	
			D	E	D	E
1	72	F	0	0	1	1
2	23	M	0	1	1	1
3	46	F	0	0	0	1
4	25	F	2	2	1	1
5	53	M	0	0	1	0
6	61	M	0	0	0	0
7	43	F	0	0	1	0

Source: Nakada [14]

Table 3 Comparison of vertigo and characteristics of post-treatment headache in treatment and control

	A	B	Total	p Value
Frequency of Vertigo				
High Frequency	3	11	14	0,01
Low Frequency	22	12	34	
Total	25	23	48	
Improvement In Vertigo Severity				
Little Improvement	3	9	12	0.046
Little Improvement	3	9	12	0.047
Little Improvement	3	9	12	0.048

Table Continued...

	A	B	Total	p Value
Headache Frequency				
High Frequency at	8	10	18	0.38
Low Frequenciesa	17	13	30	
Total	25	23	48	
Improvement in the Severity of Headache				
Little Improvement b	6	10	16	0,22
Great Improvement b	19	13	32	
Total	25	23	48	

A is the treatment group and B is the control group A is high frequency (scores 1-4), low frequency (scores 5-6), where score 1 is > 5 episodes / week, 2 3-4 / week, 3 is 1 / week, 4 is 2-3 / Month, 5 is 2-3 in 3 months and score 6 is <2/3 months

B small improvement (scores 0-2), great improvement (scores 3-4), where 0 is no improvement, 1 is slight improvement, 2 is moderate improvement, 3 is excellent improvement and 4 patients are no longer symptomatic. Source: Lepcha [13]

For evaluation of improvement or worsening of the picture, an evaluation scale of 1-4 was set up for the symptoms in question, 0 - 2 being, none or a small improvement of the symptom, 3-4, significant improvement.

progression of the disease when compared to the patients of the group B. Some side effects indicated by the patients, such as gain of Weight in two patients, acne reported by an isolated patient and drowsiness in five patients (Figure 1).

Table 4 Classification of migraine associated with auditory vestibular dysfunction.

Classification of Migraine Associated

with Auditory-Vestibular Dysfunction

Defined

Episodes of vestibular symptoms, of moderate to severe intensity (rotational vertigo, positional vertigo, other sensations of intolerance to head movement)

Migraine according to the criteria of the International Headache Society, 2001

At least one of the following

symptoms of migraine during at least two dizziness attacks: Headache, photophobia, phonophobia, visual disturbances or other aura symptoms

Exclusion of other causes after

clinical investigation

Likely

Episodes of vestibular symptoms, of moderate to severe intensity (rotational vertigo, positional vertigo, other sensations of intolerance to head movement)

At least one of the following IHS

signs for migraine patients:

Migraine symptoms during at least

two vertiginous episodes;

Signs "triggers" for

migraine pictures like some foods;

Sleep irregularities

Hormonal changes

Positive response to anti-migraine drugs

Discard other defined causes of

dizziness after due investigation

"Light" vestibular

symptoms were defined as those that do not interfere

with the individual's daily activities, "moderate" when

interfering with daily activities, but does not

prevent them and "severe" when the

individual is unable to perform daily activities.

It is worth remembering that

non-vestibular dizziness such as

orthostatic hypotension was not included.

Source: Cal [6]

According to Table 3, it can be observed that the patients in group A presented a considerable improvement of the symptoms and not

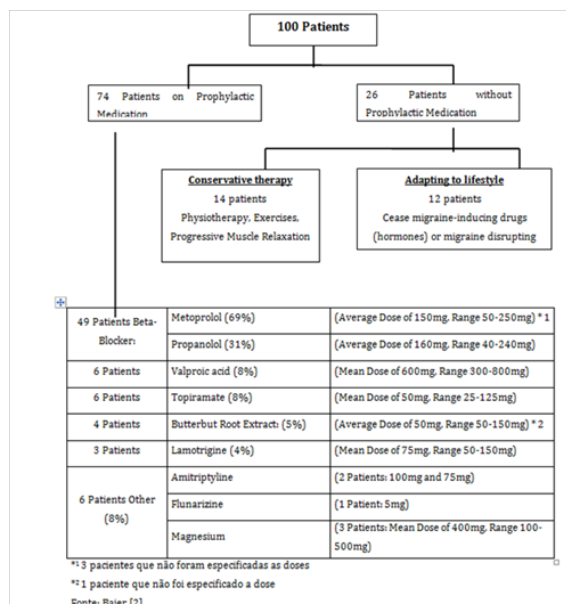


Figure 1 Treatment in 100 patients with MV.

Discussion

The link between migraine and vertigo was recognized by some of the first neurologists in the 19th century, but systematic studies of vertigo caused by migraine began only 100years later.¹ According to the analysis of the existing literature, the scarcity of data can be observed to arrive at an accurate diagnosis of MV. Often, the diagnosis can be confused with another etiology because the current criteria do not overcome the overlap with other entities and their clinical variability.⁷

The clinical and epidemiological association between migraine and vestibular signs and symptoms suggests that they may have common mechanisms. Connections between the vestibular nuclei, the trigeminal system and the thalamic pathways provide a rational basis for the development of a pathophysiological model of migraine related to vertigo.⁴

It is believed that the MV is under diagnosed due to several factors, including the great variability in the clinical presentation of patients and the lack of a widely accepted pathophysiological model that links migraine to vertigo.² For diagnostic purposes, the criteria currently

used are those proposed by Neuhauser (Table 4) and the search for more specific criteria has been a difficult for professionals working in this area. In the present study, the use of MRI of the inner ear, in addition to other tests such as myogenic potential evoked by the vestibule by sound induction (VEMP's) and videonystagmography.^{2,6}

Current treatment can be divided into prophylactic or acute attacks. The suggested drugs for prophylaxis are propranolol, tricyclic antidepressants, pizotifen and flunarizine and for acute attacks, triptans and vestibular suppressors, such as dimenhydrinate, promethazine and meclizine. Non-drug therapy that demonstrates efficacy in individual cases, such as diet, management of triggers (triggers), stress management, etiology clarification and vestibular rehabilitation¹ are also indicated. According to Gurkov et al.,⁷ the diagnosis of MV is difficult for three reasons: first, the attacks are associated in only 65% with typical symptoms of migraine; Second, vertigo attacks and headaches may appear simultaneously or independently; third, there is evidently an overlap between MV and DM.

Gurkov et al.,⁷ says that Neuhauser et al.,¹³ diagnostic criteria present a broad base as a starting point to help clinicians guide themselves. Neuhauser & Lempert¹ report that the pathophysiology of MV is still unclear, none of the possible pathophysiological mechanisms for MV have been investigated experimentally. Therefore, all of them are still highly speculative.

For Lempert & Neuhauser,¹ a familiar factor may occur that is not uncommon, probably based on an autosomal dominant paternal gene with low male penetration. Neuhauser & Lempert¹ believe that several neurotransmitters that are involved in the pathogenesis of migraine (the peptide of the calcitonin gene, serotonin, noradrenaline and dopamine) are also known to modulate the activity of vestibular neurons and may contribute to the Pathogenesis of MV.

The study by Nakada et al.,⁶ concluded that MRI results with gadolinium contrast showed that MV patients rarely present HE in the vestibule, whereas DM patients often present, suggesting that the pathophysiological process of vestibular DM occurs in the Inner ear, whereas MV arises in the vestibular nerve and / or CNS. Therefore, the image of the inner ear using gadolinium contrast MRI was useful in differentiating these two clinical entities.

Already for Gurkov et al.,⁷ MRI may be useful in the differential diagnosis of patients with auditory and MV symptoms, since it can detect HE in a proportion of these difficult cases. This has immediate implications for the differential diagnosis in vertiginous patients, for the pathophysiological understanding and clinical definitions of MV and DM as separate entities and for their therapeutics.

Still in the study by Gurkov et al.,⁷ through electrocochleography, a greater number of HE was demonstrated in relation to that found by MRI, suggesting that audiovestibular function tests can detect physiological changes (eg, Basal membrane), caused by subtle or early degrees of HE, which may be too small to be visualized by imaging techniques, such as MRI.

Baier et al.,¹³ performed myogenic evoked potential (VEMP's) in patients with defined MV and in patients with probable MV. It was concluded that patients with vestibular migraine have low amplitudes on both sides (left and right) when compared to healthy patients. The myogenic potential is a cervical reflex from an auditory stimulus obtained through the vestibule that is diminished in patients with MV. It is a fact that contributes to the diagnosis of vestibular migraine, since the pathophysiology of the disease is not yet fully UNDERSTOOD.

This study has limitations. Time period between the tests and the last crises, as well as the duration and the frequency of the same ones vary, which can have an impact on the assessed physiological data. In addition, despite the fact that all patients are within the criteria of probable or definite vestibular migraine, this is a heterogeneous entity, whose differential diagnoses may also influence the electrophysiological data.

Furman et al.,¹⁰ evaluated tests such as videonystagmography and caloric tests in patients with MV and migraine without associated dizziness to verify if there was an alteration of the peripheral vestibular function. The study reported that patients with MV who have normal ocular motor function usually have normal caloric responses and occasionally may demonstrate persistent positioning nystagmus. Previous studies have suggested that MV patients often have nonspecific results, so it is difficult to diagnose them. The study showed that patients with MV usually have normal peripheral vestibular function, with a decrease on the reflex of the horizontal semicircular canal and an increase in the sensory oscillation.

During electro-ocular recording (OVAR), postural oscillation recordings in response to an optical stimulus suggest an increase in vision dependence for upright balance. Only the horizontal semicircular canal (caloric tests) and utricular function (OVAR) were evaluated and there were no caloric abnormalities in the group of individuals with MV, as there were a small number of patients tested.

In the study by Baier et al.,¹³ it was concluded that conservative therapy and lifestyle adaptations can contribute to the reduction of dizzying seizures in about 1/3 of the patients. Therefore, the response to prophylactic therapy as a diagnostic criterion for MV cannot be absolute. However, the response from patients, whose diagnosis is not clear, can serve as a guide for the diagnostic process.

For Lepcha et al.,¹¹ flunarizine (10 mg) is a useful drug in patients with MV, especially those with significant morbidity as a function of vestibular symptoms. The frequency and severity of headache in MV were dramatically reduced in patients taking flunarizine and there were no significant side effects reported, and are therefore indicated for the treatment of patients with MV and whose vestibular symptoms are considerable.¹⁴ Previous studies in patients who meet the criteria for MV have shown benefits with drugs such as beta-blockers, calcium channel blockers, tricyclic antidepressants, anticonvulsants and benzodiazepines, but betamethasone, a H1 receptor agonist and antagonist H3 receptor, was effective In the treatment of vestibular vertigo.

In the study by Lepcha et al.,¹¹ also, flunarizine, a calcium channel blocker, was successfully used in the treatment of migraine and vertigo.¹⁵ Experimental and clinical studies have shown encouraging effects on vestibular disorders and it has been shown that flunarizine objectively reduced vertiginous symptoms.

Conclusion

Dizziness and headache are two symptoms that can make part of the same disease. Vestibular migraine is a frequent entity in patients complaining of vertigo and may coexist with other diseases such as DM. The lack of a specific pathophysiological model, makes it difficult to reach the diagnose. Use of exams such as MRI of the inner ear and VEMPs may help to establish a more precise diagnostic classification. It is worth mentioning that Neuhauser's current diagnostic criteria may point to a large number of false positives due to its subjectivity and to the overlap of other comorbidities. Videonystagmography did not prove to be a promising diagnostic test.

Given the high prevalence of patients with MV, we performed an evaluation of the current prophylactic drug therapy and changes in lifestyle, which appear to influence the incidence of patients' complaints. MV prophylaxis could be applied with drugs or lifestyle changes, such as improving diet, performing physical exercises or evaluating stressors that act as triggers for seizures, for example. The drugs to be used prophylactically are mainly: Propranolol, Atenolol, Valproic Acid, Topiramate, Amitriptyline, Flunarizine. These drugs showed to be efficient in the decrease of frequency, intensity and duration of symptoms during the crisis.

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Conflicts of interest

Author declares there are no conflicts of interest.

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